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Brief Communication

BORDER DISEASE IN NORWAY

Border disease (BD) is a prenatal infectious disease in sheep caused by a togavirus. Clonic spasms and abnormally coarse and straight birthcoat — "hairy shakers" — in neonatal lambs are typical features. Barrenness and abortion may also occur. There are several strains of the virus which is antigenically related to the bovine virus diarrhoea/mucosal disease (BVD/MD) virus. BVD/MD-virus may cause typical BD experimentally (*Barlow et al.* 1980). BD has been reported from many parts of the world, but has until now not been diagnosed in lambs in Norway. This paper describes a disease in 5 lambs with findings in accordance with those characteristic of BD.

The 5 affected lambs all of the Dala breed were born in the spring of 1979 in 2 different flocks of about 120 and 150 breeding ewes respectively. Most animals in both flocks were of the Dala breed with some individuals of the Ryggja and the Old Norwegian breeds. BD-like symptoms had not been observed previously in any of the 2 flocks between which there had been no contact.

In the one flock some lambs were born weak with brachygnathia and died a few days later whereas in the other flock there were some abortions and weak-born lambs. The ewes showed no clinical symptoms. The previous year mucosal disease had been diagnosed in a calf from one of the farms, but there had been no direct contact between cattle and sheep during the indoor season.

Three of the 5 lambs submitted were triplets born by a 5-year old ewe, and the other 2 were twins of a hogg.

The triplets showed severe body tremor from birth onward and were unable to get to their feet. They were of normal size and shape, had a strong teat seeking activity and sucked vigorously when assisted. The fleece was rather coarse and straight, but did not differ markedly from that normally seen in that breed. At rest there were fine tremors especially of the head and neck. When the lambs were stressed or tried to rise, the condition was aggravated to a convulsive stage appearing as rhythmic clonic spasms of the whole body. One of the triplets was killed and necropsied on the second day of life. The symptoms in the

remainder declined so much during the following week that the lambs were able to rise, but they were still shaking severely and showed spasm-like ataxic movements. The intensity of the symptoms continued to weaken during the following weeks. At the age of 10 weeks when another of the triplets was necropsied, tremors were still obvious at rest and increased so much when the lambs rose that normal movements were impeded. The third triplet gradually recovered, and by 6 months of age no abnormalities were evident.

The twins showed similar tremors which became more intense by handling. Although they were undersized with poor vitality, the twins were able to rise and walk with ataxic movements soon after birth. The fleece seemed to be normal. One of the twins was killed and necropsied at the age of 2 days, whereas the other which showed retarded growth was killed when 8 weeks old as chronic pneumonia supervened.

Neutralizing antibodies to BVD/MD virus in sera were estimated at The National Veterinary Institute, Oslo. Six months after birth sera from the 2 ewes with the 5 affected lambs, and from 3 other ewes with mummified/weak-born offspring had titres of $\geq 1/64$. Three affected lambs, 10 weeks old, had titres of $< 1/2$ (*Krogsrud* 1980, personal communication).

Gross lesions in the 4 necropsied lambs were restricted to purulent pleuropneumonia in one of the twins. No bacteria were isolated from the organs. Histopathological examination of CNS from 3 affected lambs were performed on formalin-fixed sections stained with H. & E., Luxol fast blue and OTAN (*Barlow & Dickinson* 1965). Lesions consisted of an increase in stab cells and interfascicular glia, many with swollen nuclei and a variable deficiency of stainable myelin and in OTAN stained sections, aggregations of fine lipid droplets between the white matter fascicles. These were most evident in the cerebellum of the 10 week old triplet but were also present to a lesser extent in the spinal cord of the only twin histologically examined. The myelin defect was rather patchy, but this would be quite consistent with the age of the animals.

It is noticeable that all the 5 lambs examined had an apparently normal birthcoat. However, this breed has a medium-coarse fleece type, and *Orr & Barlow* (1978) have demonstrated that BD has no effect on the fleece of lambs of a coarse-fleeced breed. Furthermore birthcoat changes in field outbreaks of BD

may be difficult to interpret (*Barr* 1964). The clinical findings, the histopathological lesions of the affected lambs and the existence of ewes with abnormal progeny/reproductive failure are consistent with a diagnosis of BD (*Barlow & Dickinson*). The ewes were most probably infected during the first half of pregnancy (*Vantsis et al.* 1979), as the serological findings indicate that the lambs had not responded serologically to transplacental infection (*Barlow et al.*).

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