

*Brief Communication*

NOSEMATOSIS (ENCEPHALITOOZONOSIS) IN A LITTER  
OF BLUE FOXES AFTER INTRAUTERINE INJECTION OF  
NOSEMA SPORES

During recent years, nosematosis (syn. encephalitozoonosis) has caused serious losses among young blue foxes (*Alopex lagopus*) in Norway; the causative organism is considered to be *Nosema* (syn. *Encephalitozoon*) *cuniculi*. Descriptions of clinical symptoms and patho-morphological changes on which the diagnosis is based have been given in previous papers (*Nordstoga* 1972, *Nordstoga et al.* 1974, *Nordstoga & Westbye* 1976). The disease, as a rule, is restricted to certain litters, in which several or all pups are affected, a pattern which indicates a congenital infection. In a previous report we presented experimental evidence for transplacental transmission, after oral infection of the dam (*Mohn et al.* 1974). Although we feel that oral exposure, resulting in subclinical infection, is the most common way in which the dams are infected, field observations indicate that the organisms, in rare cases, may be transmitted from the male during mating.

In order to elucidate this possibility, we carried out an experiment in which 2 vixens were artificially inseminated, with semen from a healthy male, in addition to natural mating; a few minutes after the artificial insemination they were injected intra-uterinely with 0.5 ml exudate from mouse peritoneum, containing about  $4 \cdot 10^5$  nosema spores per ml. The gonads of both sexes are frequently involved in disseminated nosematosis in blue foxes (*Nordstoga & Westbye*), but this procedure was chosen to ensure a large number of organisms; the insemination was carried out according to a technique previously described (*Fougner et al.* 1973).

None of the vixens delivered in the same season, and at the time of expected birth, both vixens were serologically negative for nosematosis, when examined by the indirect fluorescent antibody test (*Mohn & Ødegaard* 1977). One of the vixens was killed the same year in which the inoculation took place, whereas the second vixen was mated naturally next season, and delivered 2 dead and 9 live-born pups; all 9 pups were normal at birth and

during the first few weeks of their life. Eight of them developed, however, at the age of 6—8 weeks, clinical signs indicative of nosematosis; the diagnosis was confirmed serologically, by the indirect immunofluorescence technique, as 2 of the pups had antibody titres of 800 and a third  $\geq 1600$ ; at this time their dam also revealed a titre of  $\geq 1600$ . All pups were necropsied and had patho-morphological lesions characteristic for fox nosematosis (Nordstoga & Westbye).

The experiment was carried out at the Research Station for Fur-Bearing Animals, Heggedal, where "spontaneous" nosematosis had never occurred, although experiments referred to above had been performed at the station (Mohn *et al.*). The experimental animals had been kept in isolated sheds, and it seems very unlikely that the vixen had been exposed to the agent accidentally. Hence, it seems a reasonable conclusion that the pathogen most probably had survived in utero from the previous year, as a local and latent infection. It is also probable that nosematosis in this respect resembles toxoplasmosis in certain other species (Garcia 1968, Werner & Egger 1969, Waldeland 1977).

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