Observations on the relationship between Malignant Catarrhal Fever, Sheep and Fallow Deer

Michael Hindmarsh

Sheep associated malignant catarrhal fever (SA-MCF) is the most serious viral disease of deer within Australia and New Zealand. High mortalities attributed to SA-MCF have been recorded in Red deer (*Cervus elaphus*) (Wilson 1984), Rusa deer (*Cervus timorensis*) (Denholm and Westbury 1982), Chital deer (*Axis axis*) (English 1981) and Sika deer (*Cervus nippon*) (Wilson 1984). Minor mortalities have been observed in Sambar deer (*Cervus unicolour*) (Slee 1984), Sambar - Rusa hybrids (M. Hindmarsh personal data) and Fallow deer (*Dama dama*) (Anon 1980). Four deaths of fallow deer with histopathological lesions indicative of SA-MCF have been recorded in both South Australia and Victoria.

The first case was in a 2 year old menil doe which died at Mount Gambier in the lower south east of South Australia. (See Table 1). The death occurred suddenly after being separate from the herd for 2 days. The post mortem investigation revealed swollen lymph nodes and a haemorrhagic enteritis. Histopathological examination showed a fibrinoid necrosis and vasculitis of the small intestine wall.

The second case at Myrtleford, in north-eastern Victoria, was a yearling buck found comatose while an older buck and two does were dead. There were no sheep within 2km and 2 Angora goats grazed outside the deer paddock. The post mortem examination of the young buck revealed haemorrhages over the pleura, peritoneal adhesions, creamy white areas over the liver and 1-5 cm yellow centred and red ringed lesions on the serosal surface of the rumen. Histopathological study found fibrinoid necrosis and vasculitis in the rumen wall, abomasum diaphragm and omasum. The liver had massive periacinar necrosis and thrombi formation.

The third case involved fallow deer doe yearlings near Benalla dying with MCF-like lesions within a period of 12 days in September/October 1988. The first doe died without a struggle and the second had no pupillary light reflex, appeared deaf, did not respond to touch and was dehydrated. The post mortem of both showed multiple petechial haemorrhages throughout the carcase, renal cortex, mesenteric lymph nodes and the adrenals. Histopathological investigation revealed severe fibrinoid necrosis and mononuclear cell infiltrate of blood vessel walls and thrombosis in their lumens. The skeletal muscles had extensive areas of necrotic myofibres, while the kidneys had a myoglobinuric nephrosis. The biochemistry test results on a sera collected before death revealed high creatinine kinase (15450 U/L) and creatinine of 641 μM/L indicative of the muscle damage and kidney failure, respectively. Bacteriological culture of the liver, small intestines and faeces were negative for *Salmonella spp.* and *Yersinia spp.*

The author's personal experience with the deaths of 112 cattle in the Upper-South East of South Australia over a 12 year period indicates how flocks of weaner sheep can cause high mortalities of MCF, rather than the accepted transmission from lambing ewes. Five of the six outbreaks on this property had associations only with weaner sheep. Only two groups of hoggets were sold from this property. The first was purchased by a local grazier who always pastures the sheep after the cattle and he observed no outbreaks of MCF. The second sale was to a dairyfarmer who mixed the sheep with cattle and 13 cattle died over a 5 month period from SA-MCF. Nineteen sera from aged ewes on the original property were positive for antibodies to the *Alcephaline-herpesvirus - 1* (AHV - 1 WC 11 Strain) (Rossiter 1981) using the indirect immuno-fluorescent test. Sheep from this property have developed a unique association with this herpesvirus as no other reports of endemic MCF had been reported in South Australia (R. Vandergraaf personal communication). Some groups of sheep appear to be highly infective for cattle, Snowdon (1985) and this is probably due to quantitatively high levels of virus excretion. This example indicates, the infectivity of weaner sheep and care should be taken in separating sheep from deer especially when the herpesvirus status is unknown.

A high mortality of 35 Rusa deer from SA-MCF occurred after 1200 Border Leicester cross Merino ewes grazed outside their paddock. The ewes lambed from February to mid-March and the first deer mortality was on the 1st May 1987. This mortality was recorded in 1987 near Shepparton in the Goulburn Valley of Victoria. These Rusa deer came from Australia's first deer farm at Fish Creek in Gippsland which carried no sheep. There were 11 sporadic cases of MCF in the Rusa deer over a 12 year period (Presidente 1982). During the period of high mortality at Shepparton, 6 post mortems were diagnosed as positive for SA-MCF. This herd was then all sold and transported by road to Cairns in far North Queensland with a further loss of only 2 deer. There has been no subsequent losses from MCF and there are no sheep on this farm or within the area.

The susceptibility of deer to SA-MCF is related to their evolution in the absence of carrier sheep. This accounts for the high mortalities of the Indo-asiatic deer, cattle and buffalo to SA-MCF eg Rusa, Chital, Sika and Pere David's deer, water buffalo (Bubalus bubalis), Balinese cattle (Bos javanicus) which have evolved away from sheep. Fallow deer have originated from West Asia and Europe with the potential of contact with sheep. This explains why Fallow deer had no mortalities on a large deer farm in Gippsland, when 11 cases of MCF have been recorded in Rusa deer pastured with them. (Presidente 1982). The low mortality rates of Fallow deer attributed to SA-MCF from South Australia and Victoria eg 6% at Benalla are responsive to higher virus excretion from the sheep/goats. The example of low mortality rate MCF at Gippsland in Rusa deer are contrasted to the severity of the outbreak when the same deer were exposed to lambing ewes. This must be related to higher rates of virus excretion. Therefore, Fallow deer with a higher resistance to SA-MCF can also be susceptible when exposed to larger doses of this herpes virus. The role of goats as the reservoir host for MCF is not fully understood but research at Moredun Institute, Scotland has found goats with low levels of antibody to the wildebeest associated -MCF virus (Reid 1988). Care must be taken to separate Fallow deer from sheep/goats if deaths from SA-MCF are to be prevented.

TABLE 1 **MCF IN FALLOW DEER**

CASE	AREA	DEER MORTALITY	OTHER CONTACT ANIMALS	HISTOPATH POSITIVE LESIONS
1	MOUNT GAMBIER	1 - DOE	SHEEP	1 +
2	MYRTLEFORD	2 - BUCKS 2 - DOES	GOATS	1 +
3	BENALLA	2 - DOES	SHEEP	2 +

References

Anon (1980). Surveillance. MAFNZ 7: 13.
Denholm,L.J. and Westbury,H.A. (1982). AVJ 58: 81.
English,A.W. (1981). In "Wildlife Diseases of the Pacific Basin", M.E. Fowler, University California, Davis, California.
Presidente,P.J.A. (1982). The Deerbreeder. No.21: 6
Reid,H.W. (1988). In MCF in Asian Livestock, ACIAR, Canberra. p100.
Rossiter,P.B. (1981). J.Comp.Path. 91: 503
Slee,K. (1984). Proceedings No.72. PGCVSc. p563.
Snowdon,W.A. (1985). In "Veterinary Viral Diseases". Academic Press, Australia. p455.
Wilson,P.R. (1984). Proceedings No.72. PGCVSc. p518.