Leptospirosis in Deer: Diagnosis and Production Responses to Vaccination

Introduction
Leptospiral infection in New Zealand farmed deer is common. Along with cattle and sheep, deer are maintenance hosts for Hardjo, with infection estimated to be endemic in 60-65% of herds. Deer are an accidental host for Pomona, with about 1 in 20 herds having evidence of infection at any point in time (Ayanegui-Alcerreca et al 2007). Other serovars are sporadically reported.

Clinical Disease
Clinical disease in deer is sporadic and most often associated with infection with the serovar Pomona. These acute cases are most often seen in deer between 3 to 9 months of age. Sudden death, lethargy, haematuria, jaundice, fever, mucosal petechiation, and rarely pneumonia are observed. Although not reported in deer; ataxia, salivation, and conjunctivitis associated with leptospiral meningitis should also be considered as presenting signs.

Post-mortem of acute cases often reveals enlarged kidneys (either dark red due to subcapsular haemorrhage or pale), liver and/or spleen, red urine, jaundice and petechial haemorrhage. PCR of urine or fluid from macerated kidneys can be useful. However, PCR does not determine the serovar involved. Culture of renal tissue and MAT of serum from heart blood should be performed. Renal, hepatic and CNS histology can also be useful to confirm pathology.

Subclinical Chronic Disease
Historically, leptospiral infection was thought to be an incidental finding in deer. The most common observation being kidney lesions (i.e. multiple white spots, capsular adhesions and cortex scarring (Wilson et al 1998) observed at slaughter. These are associated with serovars Hardjo and/or Pomona. However evidence from recent Leptavoid® vaccination trials suggest subclinical growth and reproduction effects are associated with either Pomona or Hardjo.

Diagnosis of herd Infection
Herd-level sero-diagnosis is made using MAT. As the within herd prevalence is high (Hardjo is usually >30% and ranges between 15-100% and Pomona is usually >15% and ranges from 5-100%) only a small number (e.g. n= 15,) of samples are required to detect herd infection. Herds with kidney lesions at slaughter are considerably more likely to be infected, however, there is no correlation between whites spots and seropositivity at the individual animal level (Ayanegui-Alcerreca et al 2005).
Production Response to Vaccination
Prevention of urinary shedding by Leptavoid was first demonstrated in a challenge study. In this study, 0/14 vaccinated deer and 38% (6/16) unvaccinated deer showed evidence of shedding following intraocular Hardjo challenge (I/SPAH, 1985). More recently, Ayanegui-Alcérreca (2006) demonstrated a marked reduction in shedding following vaccination in endemically infected herds, and Subharat and others (2008) demonstrated vaccination was 100% effective (i.e. 0/36 detected by either urine culture or PCR) in preventing infection in deer on 2/5 farms where current leptospiral challenge was demonstrated. While these data indicate vaccination will reduce the risk of infection to people who handle deer or process carcasses, the interesting finding was an increase in weaning percentage and growth rate in vaccinated deer.

Weaning Percentage
In the first study of its type in deer, Ayanegui-Alcérreca (2006) demonstrated in a herd with dual Hardjo and Pomona infection, rising two year old hinds randomly allocated to vaccination with Leptavoid 3 had a 9% higher weaning percentage, compared with those that were not vaccinated (i.e. 97 vs. 88%). In a repeat study across 4 farms, Subharat and others (2008) showed an average 5% difference (range 2 to 9%) in weaning percentage in pregnant hinds randomly allocated to either a vaccination with Leptavoid 2 in February to March compared with non-vaccinated controls. Interestingly, all 4 herds had serological evidence of a low level of Hardjo infection only.

Growth Rate
Following evidence that deer infected with either Pomona or Hardjo grew an average 3.7kg less between weaning and slaughter than non-infected deer (Ayanegui-Alcérreca et al 2007), Subharat et al (2008) demonstrated an average daily gain (ADG) between weaning and slaughter of 120g/day in the deer vaccinated at weaning compared with 110g/day in the non-vaccinated controls. In this study, no Pomona was found; all 5 farms had evidence of Hardjo only. On the one farm with a high seroprevalence, the difference in ADG between Leptavoid vaccinated and non-vaccinated groups was 27g/day. This equates to 6.5kg difference in live weight over the 8 months from vaccination to slaughter. The authors concluded:

“This is a substantial loss caused by Hardjo infection confirming that this serovar can cause subclinical production loss and that vaccination can prevent it.”

Vaccine programme
Besides the reduction in zoonotic risk, given the extremely high prevalence of infection, the potential production gains make vaccination of deer for leptospirosis cost effective.

In herds not currently vaccinating, all deer need a sensitiser and booster vaccine 4 to 6 weeks later. Following this initial programme, young deer should be vaccinated at about 3 months of age followed by a booster 4 to 6 weeks later. Rising 2 year old and adult deer should be boostered annually, the hinds 4 to 2 weeks prior to calving and the stags when convenient.

References
Ayanegui-Alcérreca MA. Epidemiology and control of leptospirosis in farmed deer in NZ., 2006.