

SUMMARY

- » Dogs are susceptible to leptospiral serovars Copenhageni, Pomona and Hardjo, and now there are cases indicating infection with Tarassovi.
- » Dogs are infected as a result of contact with livestock and/or with rodents.
- » Infection may be more common in wet, warm conditions.
- » The current canine vaccine is designed for protection against Copenhageni, and there is no information as to cross-protection against the other serovars, including those infecting livestock.
- » Serology against all serovars is needed to make a definite diagnosis because non-specific titres to non-infective serovars are common. Unless all serovars are tested, the actual infective serovar may be completely missed.
- » Dogs that are azotaemic and/or have hepatobiliary disease may be infected with leptospirosis, particularly if they are residing in farming environments.

LEPTOSPIROSIS IN DOGS: *is infection increasing, and is there a need to consider multiple serovars?*

Janice Thompson, of Gribbles Veterinary, Palmerston North, argues that definitive diagnosis may now require considering all potential infective serovars.

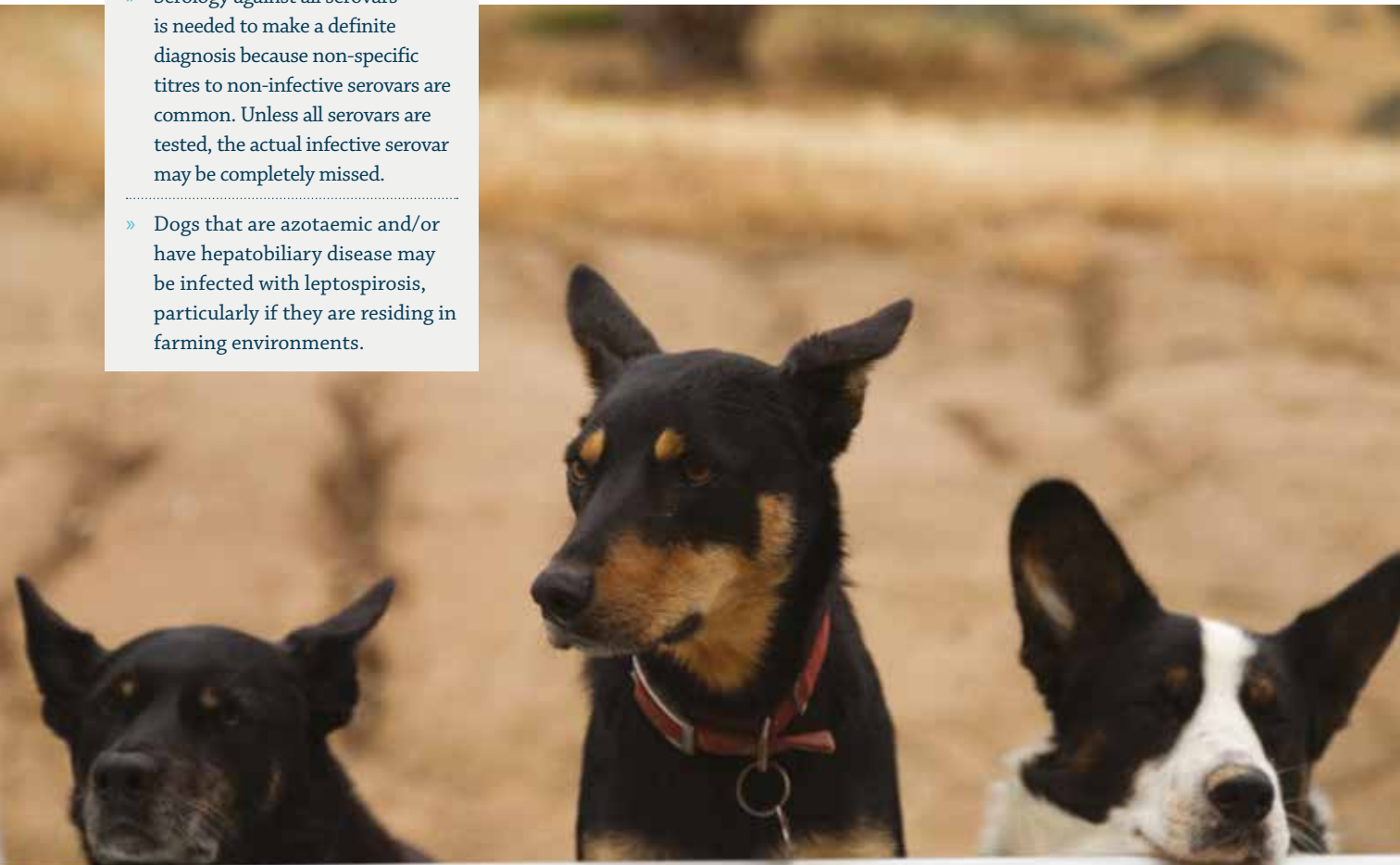


TABLE 1:
Summary of relevant information from dogs residing on farms

| Breed | Age | Liver | Kidney | Pomona | Hardjo | Copenhageni | Tarassovi |
|------------------------|--------|---|--|----------|----------|-------------|-----------|
| Beardie | 5 yrs | ALP 139 ALT 141 | Creatinine 267 Urea 59 | ≥1,600 | ≥1,600 | ≥1,600 | 1/100 |
| Huntaway | 6 yrs | Normal | Creatinine 176 Urea 13 | ≥1,600 | 1/200 | 1/800 | 1/100 |
| Bull Terrier | 7 yrs | ALP 1166 ALT 9378 Bili 6 | Creatinine 304 Urea 7.6 | Negative | Negative | 1/100 | 1/50 |
| Fox Terrier | 6 yrs | Normal | Creatinine 220 Urea 12.5 | 1/50 | 1/400 | Negative | Negative |
| Huntaway | ? | In clinic biochemistry diagnosis - normal | In clinic diagnosis of azotaemia. Urine SG 1.015 | 1/50 | ≥1,600 | Negative | Negative |
| Huntaway | 7 yrs | Liver normal | Creatinine 328 Urea 27 | 1/400 | ≥1,600 | 1/50 | 1/200 |
| Huntaway [✦] | 6 mths | Not done. Litter mate to puppy below | Not done. Litter mate to puppy below | Negative | ≥1600 | Negative | 1/50 |
| Huntaway ^{✦✧} | 6 mths | ALP 325 | No azotaemia | Negative | 1/200 | Negative | 1/1,600 |
| Spaniel | 1 yrs | ALP 127 ALT 827 | No azotaemia | Negative | 1/50 | Negative | 1/200 |
| Huntaway [✦] | 8 yrs | Normal liver enzymes Hyperglobulinaemia Hypoalbuminaemia present | No azotaemia Urine SG 1.014 | Negative | 1/400 | Negative | Negative |

LEGEND:

- ✧ PCR negative on blood
- ✦ PCR negative on urine
- ✦✧ Two puppies are litter mates

REFERENCE INTERVALS:

- Creatinine:** 48-109 umol/L
- Urea:** 2.5-9.0 mmol/L
- ALP:** 0-87 U/L
- ALT:** 0-88 U/L
- Bilirubin:** 1-3 umol/L

BACKGROUND

Leptospirosis in livestock in New Zealand has been the subject of several articles in recent months. Discussions have included the serovars present, the increasing incidence of Tarassovi in cattle and increasing incidence among meat workers. Tarassovi is not included in the three-way vaccine for cattle in New Zealand (Virbac vaccine product information). In the past, Copenhageni was considered the most important serovar in dogs and followed contact with infected rats. There is no information on cross-protection for dogs vaccinated with the canine leptospirosis vaccine that contains the serovar Icterohaemorrhagiae that protects against Copenhageni (Nobivac vaccine product information).

Recently, there has been media discussion of increased numbers of human cases of leptospirosis in wet winter months. Dogs are also susceptible to leptospirosis and work in the same environmental conditions as their human owners, are lower to the ground and therefore in closer contact with urine and puddles of contaminated water, and are without protective gear. Conditions causing an increased incidence of human leptospirosis are also likely to cause leptospirosis in dogs. Outbreaks among dogs overseas have been associated with warm temperatures, slow-moving and stagnant water and following periods of high rainfall. Anecdotally, the New Zealand 2017 winter was warmer and wetter than usual.

It cannot be assumed that the epidemiology and clinical syndromes present overseas are the same for the infective serovars here, because New Zealand has different serovars, maintenance hosts and environmental conditions. When considering the epidemiology of leptospirosis, factors such as whether the infected animal is an accidental host or a maintenance host, its ecology and the environmental conditions are also needed to understand the spread of a specific serovar.

Serological surveys have been carried out on a wide range of dogs throughout New Zealand. The animals were tested for Pomona, Hardjo, Copenhageni and Ballum, but Tarassovi was not included. Testing included serology on apparently

normal dogs and blood sourced from dogs at the Massey University Veterinary Teaching Hospital. In these surveys, breeds used as working dogs tended to have higher numbers of positive Hardjo titres compared with other breeds. Clinical cases of Pomona have also been described in dogs in New Zealand.

ROUTINE CASES RECEIVED, AUGUST AND SEPTEMBER 2017

Increased numbers of sick and azotaemic dogs from farm environments were noted in canine blood samples submitted to Gribbles Veterinary, Palmerston North for routine biochemistry over the later winter months of 2017 (when compared to earlier in 2017, and the same period in the previous year). Where azotaemia/renal disease and/or hepatobiliary disease were found, leptospirosis was noted as one of the differential diagnoses. The laboratory results for the 10 rural dogs with azotaemia and/or abnormal hepatic enzymes in August and September are summarised in Table 1. The dogs were mainly working dogs, plus three were pet dogs residing on farms.

The original Gribbles Veterinary leptospirosis panel of three serovars (namely Hardjo, Copenhageni and Pomona) was expanded during this period to include Tarassovi (free of charge) to obtain more information.

The majority of dogs were azotaemic, with probable renal disease; fewer suffered from hepatobiliary disease. One of the azotaemic dogs also had marked increases in liver enzymes. Dogs that were either azotaemic and/or had hepatobiliary disease had leptospiral serology requested. All dogs had titres to one or more of the leptospiral serovars. The results are summarised in Table 1. The majority of dogs were infected with Hardjo, fewer with Pomona, and one was infected with Tarassovi and another with Copenhageni. Smaller non-specific titres were also measured.

Polymerase chain reaction (PCR) testing was requested on either blood or urine from two of the Palmerston North cases; both were negative (see Table 1).

DOGS ARE SUSCEPTIBLE TO LEPTOSPIROSIS, AND WHERE AZOTAEMIA AND/OR HEPATOBILIARY DISEASE ARE SEEN IN DOGS IN CONTACT WITH FARMED LIVESTOCK AND RODENTS, LEPTOSPIROSIS SHOULD BE CONSIDERED.

DISCUSSION AND CONCLUSION

Dogs are susceptible to leptospirosis, and where azotaemia and/or hepatobiliary disease are seen in dogs in contact with farmed livestock and rodents, leptospirosis should be considered. Farm dogs exposed to livestock may be infected with Pomona, Hardjo or Tarassovi. In addition to the azotaemic dogs and dogs with hepatobiliary disease, there were cases of dogs in contact with livestock that showed signs of severe illness (including pyrexia) but that had no obvious biochemical abnormalities and may also have been infected with leptospires. However, serology and/or PCR were not requested for these cases.

To make an accurate diagnosis, all potential infective serovars need to be considered because livestock in New Zealand may be infected with Pomona, Hardjo or Tarassovi. Non-specific titres to non-infective serovars may be obtained. If practitioners select one serovar to test and do not test the others, it is possible that any titre obtained may be a non-specific reaction to a non-infective serovar and the infective serovar may be completely missed. Cross-reactions to non-vaccinal serovars may also occur, and there may be higher titres seen if

there is ongoing exposure to field strains following vaccination. Where low titres are measured, it is necessary to repeat the serology 10-14 days later in order to make a definite diagnosis.

Animals are leptospiraemic and leptospiruric until antibody develops. In accidental hosts, once antibody develops leptospires are cleared first from the blood, then later from the kidney. Hence, early in infection, animals with leptospiral infection are serologically negative but PCR-positive. Exact times for the development of antibody are variable and depend on infective dose and individual animal susceptibility. Both serology and PCR may be needed to make a diagnosis, but identification of the actual infective serovar requires serology. The PCR test available at Gribbles Veterinary identifies pathogenic leptospires, but does not identify the specific serovar. Culture is not useful for diagnosis of clinical cases because leptospires require special culture media and may be difficult to grow, in addition to being very slow growing. ^(vs)

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