Correspondence

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The dog in the transmission of human leptospirosis under tropical conditions: victim or villain?

To the Editor:

We read with interest the paper on the epidemiology of leptospirosis in rodents and dogs in Trinidad and Tobago [1] regarding the zoonotic potential of canine leptospirosis. We would like to share our 20 years' experience with leptospirosis in dogs from Rio de Janeiro, Brazil.

Leptospirosis is more frequent in tropical than in temperate regions; this is due mainly to longer survival of leptospires in warm, humid environmental conditions. Moreover, in most tropical countries socio-economic conditions, population density, climatic and environmental conditions, as well as behavioural and occupational factors, provide greater opportunities for exposure of the human and animal population to infected reservoirs [2].

Almost every known species of mammal can act as reservoirs of leptospires, depending on the serovar. Rats are major sources of Icterohaemorrhagiae serogroup infection and the role of urban rodents, particularly *Rattus norvegicus*, in the transmission of Icterohaemorrhagiae serogroup (which includes serovar Copenhageni) infection to humans and dogs is well known [1, 3].

Dogs also are well known sources of human leptospirosis, since they may harbour leptospires in their kidneys; however, their role as a reservoir is limited to Canicola strains [4]. In contrast, in Brazil as well as in other tropical countries, human leptospirosis is mainly caused by members of Icterohaemorrhagiae serogroup, with serovars Copenhageni in Brazil [5], Icterohaemorrhagiae in Tanzania [3] or Lai in China [6].

In the acute clinical form of canine leptospirosis, members of Icterohaemorrhagiae are most commonly reported as serovar Copenhageni in Trinidad [1] and Australia [7] or Icterohaemorrhagiae in Japan [8] and Brazil [9].

Consequently, considering that the most frequent and virulent agents of human leptospirosis (Icterohaemorrhagiae and Copenhageni) are not the serovars maintained by dogs (Canicola), it is reasonable to assume that the role of the dog in human infection is limited, if it exists. In contrast, dogs are a lot more exposed to the infection than humans, since they have free access to contaminated environments; they hunt rats and often have their food or water contaminated by urine of rats.

Usually, an outbreak of canine leptospirosis in a specific region is a sign that the agent is circulating in the environment and that the rodent population is not controlled in that area. Hence, sanitary authorities should consider veterinary information as valuable in flagging a risk, so that planning of specific local activities for human leptospirosis control can be undertaken.

In our opinion dogs are far more frequently the victims of leptospirosis of rodent origin (determined by serovars Icterohaemorrhagiae and Copenhageni) than the villains in infecting the human population.

Declaration of Interest

None.

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The authors reply

It is well established that leptospirosis is one of the most common zoonotic diseases transmitted to humans through contaminated water or direct exposure to the urine of infected animals [1, 2]. Rodents are also known to be the most important reservoir for Leptospira spp. although other animals particularly dogs have been reported to be carriers of the pathogen; clinical leptospirosis with associated mortalities [3, 4] can occur. Distribution of serovars of Leptospira spp. has been shown to vary with geographical locations and animal species [5]. In several countries, serovars Canicola and Icterohaemorrhagiae have been reported to be most important as causative agents of clinical leptospirosis; thus earlier vaccines produced to prevent canine leptospirosis contained only these serovars [5]. The potential for dogs to serve as reservoirs and a

potential source of human infection has been documented [5-7].

Based on our studies in the Caribbean island of Trinidad [8], Martins *et al.*'s statement that 'it is reasonable to assume that the role of the dog in human infection is limited, if it exists', in the above letter, may not be accurate or supported by published data. Some of the reasons for our opinion are as follows:

- Serological studies on leptospirosis in dogs (suspect clinical cases, stray, farm and hunting) in Trinidad indicated that the prevalent serovars belonged to serogroup Icterohaemorrhagiae [9]. Serovar Canicola which is one of the two serovars in the vaccine used locally was not detected in the sera tested.
- In a more recent serological survey of dogs (stray and suspect clinical cases of leptospirosis) and rodents, serovar Copenhageni (also of the serogroup Icterohaemorrhagiae), and not serovar Canicola, was most prevalent [10].
- In the same environment the prevalent serovar detected in rodents by culture was also Copenhageni and not Canicola [8] which may suggest that rodents are sources of the serovar for the dogs although there is no proof of this.
- In a study of serovars of *Leptospira* spp. implicated in humans in Trinidad and Tobago serovar Copenhageni was also implicated as being predominant (Adesiyun *et al.*, unpublished data).

Although these data suggest that rodents in our country may be reservoirs of leptospirosis for both humans and dogs, the evidence is not conclusive bearing in mind that serovar Copenhageni has been detected in both apparently asymptomatic and symptomatic cases of leptospirosis. There are plans to conduct molecular studies on our isolates recovered from rodents and dogs in order to characterize them and possibly establish their relatedness. The authors' conclusion that 'dogs are far more frequently the victims of leptospirosis of rodent origin (determined by serovars Icterohaemorrhagiae and Copenhageni) than the villains in infecting the human population' would only be valid if dogs are determined to be not important as reservoirs of serovars of the Icterohaemorrhagiae serogroup. We are therefore of the view that until it is established that dogs cannot serve as reservoirs of serovars other than Canicola, it is premature to accept the conclusion of the authors of the letter.

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