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Reservoir and transmission of Visceral Leishmaniasis

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ABSTRACT

The domestic dog is the most important reservoir of visceral leishmaniasis (VL) in metropolitan areas. The dog develops an intense cutaneous parasitism, favoring infection of sandflies, thereby playing an important role in the epidemiological chain of VL. Importantly, most dogs remain asymptomatic for long periods of time, which contribute to the maintenance and transmission of the disease (Giunchetti, et al., 2006). In Europe, in addition to dogs, rodents are also identified as reservoirs, whereas humans and domestic cats are considered accidental hosts (Poli, et al., 2006). Moreover, leishmanial antibodies were also detected in 33 of 1,220 animals; in 31 of 867 goats (*Capra hircus*), 1 of 161 cattle (*Bos indicus*), 1 of 54 wild rats (*Rattus* sp.), but none of 106 chickens (*Gallus domesticus*), 26 sheep (*Ovis aries*), 3 water buffaloes (*Bubalus bubalus*), and 3 dogs (*Canis familiaris*). The parasite DNA was detected in 20 of 33 serologically positive blood samples; in 20 goats and 1 cow. This study indicated that goats are also the potential animal reservoirs of human VL in India (Singh, et al., 2013).

Keywords: DNA, VL, Leishmaniasis, Eosophagus, Promastigotes, Phagocytosed, Amastigotes

I. INTRODUCTION

Transmission of visceral leishmaniasis :

Transmission of VL generally takes place between the invertebrate vector (sandfly) and a mammalian host. A female phlebotominae sandfly vector is infected by ingesting amastigotes present in the dermis during the blood meal of a mammalian host. The amastigotes transform into promastigotes within the digestive tract of the insect within 24 to 48 hours after the blood meal. The promastigotes proliferate in the intestine and migrate to the eosophagus and pharynx, and are then regurgitated into the mammalian host during the blood meal (Kamhawi, 2006). Soon after inoculation the promastigotes are phagocytosed by macrophages and reside inside a vacuole that fuses with lysosomes, named the parasitophorous vacuole (Mosser and Rosenthal, 1993; Pearson and Sousa, 1996). The promastigotes then transform into amastigotes that replicate intracellularly eventually causing the rupture of the macrophage and thereby infecting other phagocytic cells (Handman and Bullen, 2002).

Although VL is largely recognized as an insect borne disease, there are several reports of transmission in the absence of vector. For instance, the transmission through blood transfusion in human patients has been reported (Otero, et al., 2000) as well as transmission by common use of needles among drug addicts (Bosch et al., 2002).Venereal transmission of VL has been documented between an infected man and his wife in an area completely free of the insect vector (Symmers, 1960).

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