

LETTER TO THE EDITOR

A Note on the Co-Infection Dynamics of Lumpy Skin Disease (LSD) and Bovine Haemopprotozoan Parasites

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Dear Editor,

Arthropod-borne diseases pose a serious threat to livestock health and significantly impact the economic stability of the livestock sector. Among these, Lumpy Skin Disease (LSD), caused by the Lumpy Skin Disease Virus (LSDV) from the family Poxviridae, is a significant concern. Primarily affecting cattle, LSD is widespread in Asia and results in substantial economic losses. The disease is mainly transmitted by hematophagous vectors like stable flies -*Stomoxys* spp., *Haematobia* spp.^[1], mosquitoes^[2], and ticks- particularly those of the *Hyalomma* spp.^[3] and *Rhipicephalus* spp.^[1,4]. Likewise, ticks serve as vectors for several hemopprotozoan pathogens such as *Theileria* spp., *Babesia* spp., and *Anaplasma* spp., which cause theileriosis, babesiosis, and anaplasmosis, respectively. In recent years, reports of co-infections involving LSD and hemopprotozoan parasites from endemic areas have increased. The overlapping ecology and shared tick vector populations facilitate the simultaneous transmission of LSDV and hemopprotozoa. These co-infections exacerbate clinical signs, delay recovery, and complicate diagnosis, leading to additional economic losses in affected herds.

Previous research has shown that LSD often coexists with haemopprotozoan infections such as babesiosis^[1], theileriosis^[1,4], and anaplasmosis^[4]. Histopathological links between LSD and theileriosis have also been observed^[5]. LSDV DNA in tick salivary glands reinforces the idea that ticks play a role in transmitting the virus^[4]. The involvement of *Hyalomma anatomicum anatomicum* ticks in spreading LSD, theileriosis, and anaplasmosis is well

established^[4]. Likewise, the association of *Rhipicephalus* spp. ticks, that is a common vector for bovine babesiosis, also spread LSD^[1,4].

Breed susceptibility also plays a key role. Holstein Friesian cattle have been reported to develop more severe forms of LSD compared to indigenous breeds^[6]. Similarly, exotic breeds are more prone to haemopprotozoan infections such as theileriosis. The higher prevalence of coinfections can be linked to the common vector -the tick- which transmits LSDV and haemopprotozoa^[3]. Abas et al.^[7] found a strong correlation between LSD outbreaks and haemopprotozoan infections, showing a significant difference ($P<0.05$) in parasitemia levels between LSD-positive and LSD-negative cattle. This difference was due to the immunosuppressive effect of LSDV.

In haemopprotozoan infections, parasitaemia levels are closely linked to the phagocytic activity of leukocytes, which becomes significantly impaired during LSD infection. Typically, parasite invasion triggers an innate immune response through chemokine release and recruitment of phagocytic cells^[8-10]. This defence mechanism is particularly effective during acute infections with high parasitaemia, helping to control the infection^[9]. However, during LSD infection, this immune response is disrupted, resulting in altered parasitaemia levels and more severe disease progression. Additionally, animals with tropical theileriosis experience dysfunction in key immune cells -macrophages, neutrophils, B cells, and T lymphocytes (CD4+ and CD8+)- which collectively maintain immune balance^[11,12]. Any disturbance in their activity predisposes



animals to secondary viral infections, including LSDV [5]. Consequently, once coinfection occurs, both diseases worsen due to the host's weakened immune system.

Future investigations should focus on molecular and immunological methods to better understand the dynamics of these concurrent infections. The immunosuppressive effect of LSDV can make animals more vulnerable to secondary haemopprotozoan infections or trigger latent parasitic infections. Conversely, previous haemopprotozoan infections may weaken immune function, increasing susceptibility to LSDV. Real-time field and experimental studies are necessary to determine whether LSD predisposes animals to haemopprotozoan infections or the other way around. The possibility that carrier or sub clinically infected animals may become more susceptible to LSD also needs further study.

From a diagnostic standpoint, coinfection can mask typical clinical signs, resulting in underdiagnosis or misdiagnosis. Thus, a thorough diagnostic strategy that includes clinical evaluation, blood smear analysis, and molecular testing is vital for precise identification. Prompt diagnosis and immediate treatment are key to lowering morbidity, avoiding economic costs, and enhancing recovery in cases of coinfection.

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