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Effect of Blue Tongue Virus in Animals

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Opinion Article

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DESCRIPTION

Bluetongue illness is a noncontagious, insect-borne viral condition that affects ruminants such as sheep, goats, buffalo, deer, dromedaries and antelope. The virus that causes it is Blue Tongue Virus (BTV). *Culicoides imicola*, *Culicoides variipennis* and other culicoids are the vectors of the virus. BTV causes an acute disease in sheep that is associated with a high rate of morbidity and mortality. Goats, cattle and other domestic animals, as well as wild ruminants are all infected with BTV (for example, blesbuck, and white-tailed deer, elk and pronghorn antelope).

High temperature, profuse salivation, swelling of the face and tongue and cyanosis of the tongue are the most common symptoms. Swelling of the lips and tongue gives the tongue its characteristic blue colour but this is only seen in a small percentage of the animals.

Nasal symptoms such as nasal discharge and stertorous breathing may be present. Some animals have foot lesions. The frequent shifting of the feet of cattle has earned bluetongue and the nickname was "dancing sickness". In seriously damaged animals, neck torsion (opisthotonos or torticollis) is observed. Not all animals show symptoms, but those that do quickly deteriorate and the sickest will die within a week. Recovery will exceed gradual for injured animals that do not die will take several months.

The incubation period is 5–20 days with full symptoms appear within a month. The mortality rate is typically low but in sensitive sheep breeds which is very high. Local sheep breeds in Africa may have zero mortality whereas imported breeds may have up to 90% mortality. Despite high virus levels in the blood, infection in cattle, goats and wild ruminants is frequently asymptomatic. Red deer are an exception and the sickness in them can be as severe as it is in sheep. The pathogenic virus Blue Tongue virus (BTV), of the genus *Orbivirus*, of the *Reoviridae* family, causes bluetongue. This virus presently has twenty-six different serotypes. The viral particle is made up of ten double-stranded RNA strands encased in two protein shells. BTV does not have a lipid envelope like other arboviruses. The particle's diameter is 86 nanometers. The structure of the 70 nm core was solved in 1998, and it was the biggest atomic structure ever solved at that time.

BTV attachment and penetration into the target cell are mediated by the two outer capsid proteins VP2 and VP5. The virus makes first contact with the cell *via* VP2, starting virus endocytosis *via* receptor-mediated endocytosis. BTV's membrane penetration protein VP5 undergoes a conformational shift in response to the low pH within the endosome, disrupting the endosomal membrane. Uncoating results in a transcriptionally active 470s core particle that contains the dsRNA genome as well as two major proteins, VP7 and VP3, and three minor proteins, VP1, VP4, and VP6. As with reovirus, there is no evidence that any trace of the outer capsid remains connected with these cores. As with reovirus, there is no evidence that any trace of the outer capsid remains connected with these cores. In contrast to reovirus, the cores may be further uncoated to create 390s subcore particles that lack VP7. Cores generated in *vitro* from virions by physical or proteolytic treatments that remove the outer capsid and activate the BTV transcriptase are likely to be similar to subviral particles. In BTV-infected cells, three Non-Structural (NS) proteins, NS1, NS2 and NS3 (and a related NS3A) are synthesised in addition to the seven structural proteins. The NS3/NS3A subunit is important in the progeny virus's escape.

Bluetongue is a disease that has been found in Australia, the United States, Africa, the Middle East, Asia and Europe. During winters, viral survival and vector lifespan are observed. The ability of *C. obsoletus* and *C.pulicaris* to acquire and transmit the pathogen both of which are widely distributed throughout Europe has played a crucial role in the northward expansion of bluetongue illness. The original *C. imicola* vector, on the other hand, was only found in North Africa and the Mediterranean. The relatively new vector has aided a significantly more rapid spread than basic habitat extension northward due to global warming.

Quarantine, inoculation with a live modified virus vaccine and control of the midge vector including are all used to prevent the disease. Serotype-specific protection is provided by Live Attenuated Vaccines (LAVs). Neutralizing antibodies against unincluded serotypes can be induced by multiserotype LAV and successive vaccinations with three distinct pentavalent LAV can result in wide protection. These pentavalent cocktails contain a total of 15 serotypes: serotypes 1 through 14 as well as serotypes 19 and 20. However, vaccination with any of the existing vaccinations prevents further serological surveillance of affected cow populations. A concern that could be remedied with next-generation subunit vaccines.