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# A Short Note on Bluetongue Disease

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## Editorial Note

Bluetongue disease is a noncontagious, insect-borne, viral disease of ruminants, mainly sheep and less frequently cattle, yaks, goats, buffalo, deer, dromedaries, and antelope. It is caused by Bluetongue virus. The virus is transmitted by the midges and other culicoids. In sheep, BTV causes an acute disease with high morbidity and mortality. BTV also infects goats, cattle and other domestic animals as well as wild ruminants (for example, blesbuck, white-tailed deer, elk, and pronghorn antelope). Major signs are high fever, excessive salivation, swelling of the face and tongue and cyanosis of the tongue. Swelling of the lips and tongue gives the tongue its typical blue appearance, though this sign is confined to a minority of the animals. Nasal signs may be prominent, with nasal discharge and stertorous respiration. Some animals also develop foot lesions, beginning with coronitis, with consequent lameness. In sheep, this can lead to knee-walking. In cattle, constant changing of position of the feet gives bluetongue the nickname 'The Dancing Disease'. Torsion of the neck (opisthotonos or torticollis) is observed in severely affected animals. Not all animals develop signs, but all those that do lose condition rapidly, and the sickest die within a week. For affected animals which do not die, recovery is very slow, lasting several months.

The incubation period is 5-20 days, and all signs usually develop within a month. The mortality rate is normally low, but it is high in susceptible breeds of sheep. In Africa, local breeds of sheep may have no mortality, but in imported breeds it may be up to 90 percent. In cattle, goats and wild ruminants infection is usually asymptomatic despite high virus levels in blood. Red deer are an exception, and in them the disease may be as acute as in sheep. Bluetongue is caused by the pathogenic virus, Bluetongue virus (BTV), of the genus Orbivirus, of the Reoviridae family. Twenty-six serotypes are now recognised for this virus. The virus particle consists of ten strands of double-stranded RNA surrounded by two protein shells. Unlike other arboviruses, BTV lacks a lipid envelope. The particle has a diameter of 86 nm. The structure of the 70 nm core was determined in 1998 and was at the time the largest atomic structure to be solved. The two outer capsid proteins, mediate attachment and penetration of BTV into the target cell. The virus makes initial contact with the cell with VP2, triggering receptor-mediated endocytosis of the virus. The low ph within the endosome then triggers BTV's membrane penetration protein VP5 to undergo a conformational change that disrupts the endosomal membrane. Uncoating yields a transcriptionally active 470S core particle which is composed of two major proteins VP7 and VP3, and the three minor proteins VP1, VP4 and VP6 in addition to the dsRNA genome. There is no evidence that any trace of the outer capsid remains associated with these cores, as has been described for reovirus. The cores may be further uncoated to form 390S subcore particles that lack VP7, also in contrast to reovirus. Subviral particles are probably akin to cores derived in vitro from virions by physical or proteolytic treatments that remove the outer capsid and causes activation of the BTV transcriptase.

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