CONTROL OF BLUE TONGUE IN SHEEP THROUGH VACCINATION

Baltus Erasmus
Deltamune (Pty) Ltd. Lyttelton, Pretoria
(Former Head : Onderstepoort Biological Products)

Most farmers in Southern Africa are reasonably familiar with bluetongue in sheep. But it is not generally known that cattle and almost all indigenous game are also susceptible to blue tongue virus although these animals seldom show any clinical signs of infection. On the other hand exotic animals such as deer and desert sheep as well as a few other animal species such as alpacas are very susceptible to bluetongue virus with high mortalities in certain cases.

Although cattle and African antelopes show no clinical signs of infection, they however do play an important role in the multiplication and maintenance of bluetongue viruses.

Bluetongue in sheep is known since Merino sheep were brought to South Africa in 1789. Dr. Duncan Hutcheon described the disease for the first time in 1880 but the first reasonably complete description of the disease and the post mortem lesions were described by Dr. Spreull here in South Africa. It is however highly probable that bluetongue virus circulated unseen in game for many centuries and that the imported Merino sheep merely served as indicator of the disease.

Various sheep breeds vary in clinical susceptibility to bluetongue. Indigenous sheep breeds can be infected with bluetongue virus, but as with the African antelopes they show no signs of the disease. Foreign sheep breeds however, especially British mutton breeds are exceptionally sensitive to bluetongue. Cross-breeds between indigenous and foreign breeds (eg. the Dorper) have a reasonable resistance against the disease and mortalities are not so common.

Bluetongue is caused by viruses belonging to the genus *Orbivirus*. At least 24 different bluetongue virus serotypes exist of which 21 serotypes are prevalent in South Africa. All the viruses in a specific serotype afford complete cross protection against each other, but antibodies against one serotype will not completely neutralise viruses of other serotypes. A sheep that has recovered from bluetongue will therefore be immune only against the serotype it contracted and not against the other 23 serotypes.
Bluetongue does not spread through contact and the virus is biologically transmitted by blood sucking midges of the genus *Culicoides*. Biological transmission means the virus, after being ingested from the blood of an infected animal into the intestine of the midge, multiplies and spreads to the salivary glands of the midge where further multiplication takes place. When this midge takes another blood meal 7-10 days later it injects the virus with the saliva into the animal.

The most important vector (transmitter) of bluetongue in Africa is *Culicoides imicola* but other species such as *C. variipennis* in the USA and *C. obsoletus* as well as *C. pulicaris* in Europe can also act as vectors.

The fact that bluetongue is an insect-transmitted disease explains why the prevalence of the disease is much higher in areas and circumstances that are favourable for the hatching of large numbers of insects. Bluetongue is a seasonal disease that is abundant during the months of summer and autumn, especially after good summer rains. In very cold areas the disease usually disappears approximately two weeks after the first frost but in areas with mild winters the disease may occur throughout the year although with a peak during summer and autumn.

**Clinical signs and lesions of bluetongue in sheep**

The primary lesion of blue tongue infection is the damage to endothelial cells especially of the small blood vessels. This leads to the leaking of serum into tissue and body cavities as well as a tendency to petechial (pin point) haemorrhages on the mucous membranes and in tissues.

Clinical signs of this tissue damage includes fever as well as swelling of the lips and other parts of the face (sometimes even the ears). Other distinctive lesions include bleeding in the papillae of the lips, the muzzle and the ocular mucous membranes. Erosions of the superficial epithelium are visible on the muzzle, edges of the nostrils and the oral mucous membranes (especially opposite the incisor and the molar teeth). The tongue may also enlarge due to the swelling and may even protrude from the mouth. **Due to the poor blood circulation the tongue appears bluish purple, hence the name bluetongue.** (But only a small percentage of sheep display a definite blue tongue!)

Deep and very painful sores develop where the swollen tongue comes into contact with the sharp molars. The tissues of the lower jaw and throat may also be swollen. The mouth lesions are very painful and the sheep will not be able to graze normally, resulting in serious weight loss.

Another very important sign of blue tongue is coronitis. It is observed as a purple red band where the hoof joins the skin of the leg. Often only the heel is affected but sometimes the band extends around the hoof. This is caused by extremely painful bleeding of the horn channels of the hoof. Affected sheep may be lame or remain
recumbent. This painful condition may last for 3-7 days during which the affected sheep is unable to reach feed or water resulting in serious emaciation. A break in the hoof is formed and the damaged hoof is sloughed by the new hoof tissue after 3-4 months.

The skeletal muscles may also be seriously affected with literally melting away of larger muscles. This muscle degeneration contributes greatly to the dramatic weight loss observed in some sheep. These sheep usually take as much as six months to regain lost mass, in spite of good nutrition. A small percentage of sheep may also develop torticollis (wry) resulting in sheep not being able to keep their balance. In the end they have to be slaughtered or fall prey to predators.

During the acute stage of blue tongue the skin may be very red (dermatitis). As a result of the inflammation of the wool follicles the wool fibre formed during the disease is thinner and weaker than normal wool fibre. When this weaker part of the fibre grows out of the follicles approximately 3-4 weeks later, parts of the fleece or the entire fleece may be lost due to the break in the wool. Even in cases where the break is not severe enough to lose the whole fleece, the fleece will still be inferior and should not be mixed with the rest of the shearcings.

Mortality due to bluetongue may vary (0-20%) and depends on the sheep breed, the virus strain but especially also environmental conditions. Sunlight (ultra violet rays) aggravates the disease. When sick animals are exposed to cold and very wet conditions a high percentage may die due to pneumonia.

The post mortem lesions of bluetongue include the signs already seen in the live animal as well as internal lesions. The latter usually includes liquid fluid in the thoracic cavity and pericardial sac, possible pulmonary oedema (with froth in the air ways) or even signs of pneumonia (dark collapsed areas resembling liver tissue). A very characteristic lesion of bluetongue is bleeding in the wall of the artery leading to the lungs (pulmonary artery). Skeletal muscles usually are pale and watery and may display greyish white areas of necrosis, sometimes with calcification.

**Treatment**
The best treatment for sheep with bluetongue is good nursing with feed, water and shade. Sick sheep must be taken from the flock as soon as possible and placed in a small enclosure with shelter against the sun and fresh water. Soft green feed must be available but sometimes their mouths are so painful that they cannot even chew soft feed. The best solution is to feed them high quality feed cubes.

The sick sheep will often take the cube into its mouth and wait for the cube to be softened by saliva before it swallows the cube without chewing. In this manner the animal will ingest enough food to survive.
Where there is a great risk of contracting pneumonia a broad spectrum antibiotic may be administered.

**Control**
The only practical and effective measure in preventing bluetongue in sheep is preventive immunisation that should be administered correctly and in time before the onset of the bluetongue season.

Due to the huge economical impact of bluetongue on successful sheep farming it was already realised early in the nineteenth century that immunisation should offer the only real solution against bluetongue.

Sir Arnold Theiler made several efforts to develop a vaccine but it was only in 1946 after bluetongue virus was successfully cultivated and attenuated in embryonated eggs that the first reasonably successful bluetongue vaccine was developed.

By 1948 the existence of numerous bluetongue virus serotypes was proven. This explained why the existing vaccine sometimes was effective but sometimes failed.

During the fifties bluetongue virus was successfully cultivated in cell cultures. This made it possible to serotype bluetongue viruses through neutralisation. By 1960 12 different serotypes were described. As new serotypes were discovered the viruses could be weakened (attenuated) by repeated passage into embryonated eggs and then to be included in the multiple strain (polyvalent) bluetongue vaccine.

By 1963 the vaccine already contained 14 live attenuated virus strains. It soon became clear that sheep cannot react to so many different serotypes in a vaccine. A live attenuated virus strain must be able to replicate in order to establish effective immunity. With 14 strains in one vaccine you have competition and the sheep only develop antibodies against the two or three most dominant strains.

To further complicate the situation widespread outbreaks of bluetongue occurred in 1976, also in sheep that were vaccinated regularly. It was suspected that a new serotype was involved and this was confirmed when two new serotypes (BT18 and BT19) were isolated. Type 19 had a widespread occurrence and was unrelated to any of bluetongue virus strains in the vaccine – that explains the numerous cases of bluetongue that occurred in vaccinated sheep.

It was clear that Type 19 should also be included in the vaccine that already contained 14 strains. Research work showed in the meantime that sheep develop antibodies against only three of the strains, irrespective of how many strains in the vaccine. It can be compared to a race in which many athletes take part, but only the first three receive prizes. Because it would be absurd to include 15 strains in one
vaccine is was decided to construct three penta-valent vaccines (each containing five strains). The logic behind this decision may again be demonstrated by competing athletes. Should 15 athletes take part in separate groups of five each in three different races, nine prizes would be awarded instead of only three had they all been in one race.

The three penta-valent vaccines (marked A, B and C) contain the following bluetongue virus strains: Bottle A: Types 1, 4, 6, 12 and 14; bottle B: Types 3, 8, 9, 10 and 11 and bottle C: Types 2, 5, 7, 13 and 19. The three vaccines should be administered with a minimum of three week intervals although intervals of four weeks or more yielded better results.

It is admitted that five live bluetongue strains in one vaccine is still too many. The ideal would be to prepare five vaccines each containing only three strains but this would be totally unacceptable to sheep farmers, even if better results could be achieved.

It was very disappointing that farmers who demanded a better vaccine after the losses they suffered in 1976, immediately after the launching of the more effective threefold vaccine displayed great resistance to the three vaccines, claiming it is too much effort to immunise sheep three times.

The result was that farmers did not follow instructions when using the vaccine. Some simply mixed the three vaccines knowing full well that the single vaccine with 14 strains available before 1979 was not effective. Another general practice was to mix bottle A and B and dispose of bottle C. The rumour was spread that the strains in bottle C do not occur in South Africa – this while bottle C contained Type 2, the most prevalent serotype in South Africa over many years.

The central idea is that the three vaccines form a unit and that is why they are supplied together. Broad protective immunity depends on the correct administration of all three combinations. It is particularly important that young sheep be correctly vaccinated as soon as they are six months old. Vaccination at this age is still not complicated by breeding programmes and young sheep are in any case handled more often to treat worm infestation and administering other vaccines. To save labour bluetongue vaccinations can be combined with these other activities. Bluetongue vaccines may be administered together with any inactivated vaccine but not together with a live virus vaccine.

Vaccination of pregnant ewes is only problematic when giving the first bluetongue vaccinations. If young ewes receive the three separate vaccines before breeding they can in following years safely be vaccinated even if they are pregnant. If farmers for whatever reason do not want to vaccinate three times, A, B and C may be
mixed as a single combination provided the ewes received the three separate vaccines previously. The ideal is still the separate administration of each bottle.

Fortunately there are farmers who administer the threefold vaccine strictly as prescribed and they literally have no problems with bluetongue in their flocks. This serve as proof that the disease can be successfully controlled by using the present vaccine correctly.