Websites:
http://www.sciencepub.net/nature
http://www.sciencepub.net

Emails:
naturesciencej@gmail.com
editor@sciencepub.net

Nature and Science

MARSLAND PRESS
Multidisciplinary Academic Journal Publisher

QUALITATIVE AND QUANTITATIVE RISK ASSESSMENT OF BLUETONGUE VIRUS THROUGH IMPORT OF IMPROVED BREEDS OF SHEEP FROM SOUTH AFRICA TO ETHIOPIA

Nuru Mohammed Mekonnen

Debre Birhan Sheep Multiplication and Breed Improvement Center, P.O. Box 464, Debre Birhan, Ethiopia.
Email: nurum0686@gmail.com

Abstract: Bluetongue is an infectious, non-contagious disease of ruminants and camelids caused by bluetongue virus and transmitted by culicoides vector. The disease is no specific therapy but prophylactic immunisation and removal (prevention) of vector attacks can be used for control. Improved sheep imported to Ethiopia from restricted areas and certified under regulation carry a non negligible risk of introducing Bluetongue virus to Ethiopia. But failure of laboratory tests through false negative results, variance in interpretation of results between laboratories or human error the probability of virus is high. The presence of potential vector culicoides, favorable climatic condition and susceptible small ruminant population the probability of exposure for bluetongue virus is high. The consequence of bluetongue virus is high, that means direct loss of livestock, walfare implications, bans on export of live ruminants and movement restrictions. At 91% confident the risk of Bluetongue virus is between 5 animals/10,000 and 9 animals/10,000.

[Nuru Mohammed Mekonnen. **Qualitative and Quantitative Risk Assessment of Bluetongue Virus through Import of Improved Breeds of Sheep from South Africa to Ethiopia**. *Nat Sci* 2024,22(5):26-32]. ISSN 1545-0740(print); ISSN 2375-7167 (online). http://www.sciencepub.net/nature 04. doi:10.7537/marsnsj220524.04.

Keywords: Bluetongue, Risk, Sheep

1. Introduction

Bluetongue is an infectious, non-contagious disease of ruminants and camelids transmitted by *Culicoides* biting midges. It is caused by the bluetongue virus (BTV) and is placed in the Office International des Epizooties list of diseases. The manifestations of bluetongue range from an inapparent to a fatal outcome depending on the serotype and strain of the virus and the species, breed and age of the infected animal; older animals are generally more susceptible (Elbers*et al.*, 2008).

Bluetongue was first described in the Cape Colony of South Africa after merino sheep were introduced into the region in the late 18th century and was subsequently recognized in other parts of Africa, Europe, the Middle East and Indian subcontinent, Americas, Asia, Australia and several islands in the tropics and subtropics (Howell, 1979). Twenty four (likely 25) serotypes of bluetongue virus (BTV) are recognized globally and the virus has now been isolated on all continents except Antarctica (Hofmann *et al.*, 2008).

The distribution and intensity of infection is determined by the climate, geography and altitude, as these affect the occurrence and activity of the Culicoides vectors and the presence of susceptible mammalian hosts. It usually occurred where sheep industries have been established by the introduction of European fine wool and mutton breeds (Maclachlan*et al.*, 2009). Native sheep have a high level of innate resistance rendering most infections totally benign. Cattle and goats are involved in maintaining the virus wherever it occurs but disease is seldom seen in any breed. Although bluetongue does not appear to be a major economic constraint to African livestock breeders and it adversely affect the success of livestock improvement projects (Haresnape *et al.*, 1988).

The major production losses include deaths, thriftiness during prolonged convalescence, wool breaks, and reproductive losses. It causes vascular endothelial damage, resulting in changes to capillary permeability and subsequent intravascular coagulation. This results in edema, congestion, hemorrhage, inflammation, and necrosis. Some affected sheep have severe swelling of the tongue, which may become cyanotic (‘blue tongue”) and even protrude from the mouth. In many areas of the world, Bluetongue Virus infection in sheep, and especially in other ruminants, is subclinical. In endemic areas the infection is always present but clinical disease of the indigenous species is unusual. It can occur with new BTV strains and when non indigenous susceptible species are introduced to the area. Infection occurs in a number of animals but significant disease occurs only in sheep. Infection of

bluetongue is also seen in cattle but also recorded in elk, white-tailed deer, pronghorn antelope, camels and other wild ruminants. The disease is not contagious and is transmitted biologically by certain species of culicoides (DuToit, 1944).

A preliminary diagnosis based on clinical signs, post-mortem findings and epidemiological assessment should be confirmed by laboratory examination (Afshar, 1994). Samples to be examined in the laboratory should include non-coagulated blood (use of ethylenediaminetetraacetic acid or heparin is preferred), blood serum, post-mortem tissue samples of spleen, lymph nodes, lungs, liver, bone marrow and, when indicated, heart and skeletal muscles; in addition, brain tissue is collected in foetuses (Tweedle and Mellor, 2002).

There is no specific therapy for animals with bluetongue. Symptomatic therapy includes gentle handling of affected animals, their stabling and, if indicated, administration of non-steroidal antiphlogistic drugs. An immediate ban on animal import from countries with bluetongue is the priority measure, followed by the monitoring of farms raising domestic ruminants which include clinical examination and serological and virological testing, and a monitoring of insect vectors. Prophylactic immunisation and the removal of vectors or prevention of vector attacks can also be used (Radostits*et al.*, 1994; Tweedle and Mellor, 2002).

The objective of this risk analysis is:

- ✓ To assess the qualitative and quantitative risk of bluetongue virus through importation of improved sheep from South Africa to Ethiopia.

2. Bluetongue in South Africa

Bluetongue is commonly seen in South Africa in late summer and autumn (between February to April), especially in areas with high rainfall and after good rains. In colder regions such as the southern Free State, the disease usually disappears with the occurrence of the first winter frosts, while it is probable that in warmer regions such as the KwaZulu Natal and Limpopo provinces the disease may be transmitted throughout the year. It has been suggested that the occurrence of BT in sheep in late summer and autumn in the country reflects the build-up of BTV in vector competent midge species during spring and early summer. A primary infection cycle may involve either wild antelope or cattle with sheep subsequently becoming infected as a result of "spill over" in a secondary infection cycle (Peter et.al. 2012).

Bluetongue is a notifiable disease in terms of the South African Animal Diseases Act of 1984. Adherence to the act is however poor and outbreaks are not always reported. Only wet seasons with a large number of outbreaks serve to raise any level of concern. No routine surveillance for BT is carried out and information regarding serotype prevalence is based on the retrospective analysis of samples that are sporadically submitted to the OVI, as well as a limited number of field surveys. These surveys have indicated that 22 of the known 26 serotypes are present in South Africa, with serotypes 20, 21, 25 and 26 being considered to be exotic. An analysis of 258 ovine samples that were submitted to the OVI between 1983 and 2003 from all provinces of South Africa indicated that low denomination serotypes (1–4) were isolated most frequently and that some serotypes were extremely rare not having occurred for a period of more than 20 years. However, the analysis of these samples also indicated the presence of serotype 17, which had up till then not been detected in the country (OIE, 2014). A single molecular epidemiological study using the NS3/A gene of BTV has been conducted in South Africa. In this study different serotypes that were collected from widespread regions throughout the country were analyzed. The phylogeny that was inferred from the nucleotide sequence data of the NS3/A gene indicated that BTVs in South Africa cluster into two distinct lineages irrespective of their serotype, geographic area and year of isolation, suggesting that the transmission of these lineages is not restricted to any particular vector species or area of the country. Interestingly BTV isolates from the United States clustered into both lineages, supporting the notion that more than one introduction of BTV from southern Africa may have occurred into the United States in the past (Elbers et.al.,2008). In South Africa multiple serotypes circulate each vector season however the occurrence of different serotypes is unpredictable and is most likely influenced by herd immunity. Monitoring of Culicoides spp. between 1978 and 1985 at various sites throughout South Africa indicated that 14 to 18 different serotypes were encountered every season, although at varying frequencies (Hofmann et.al. 2008). Usually three to five serotypes were isolated predominantly. These serotypes were replaced with other dominant serotypes the following season, just to become dominant again three to four years later. Serotypes which were most commonly encountered included 1–6, 8, 11 and 24. Serotypes 9, 10, 12, 13, 16 and 19 were encountered every season but at a much lower frequency, while serotypes 7, 15 and 18 were encountered only sporadically. Serotypes 1–6, 8, 11 and 24 are believed to have a high epidemic potential, whereas serotypes 1–6 and 10 which are more often associated with clinical disease in sheep are thought to have a high pathogenic index (Maclachlan et.al. 2009).

Bluetongue virus has been isolated from many different Culicoides species in South Africa, however only two species, Culicoidesimicola and

Culicoidesbolitinos, are recognized as significant in the transmission of BTV. Bluetongue virus has also occasionally been isolated from mosquitoes that may play a role in the mechanical transmission of the virus (Tweedle and Mellor, 2002). The total population of sheep in South Africa is approximately 28 million that are distributed across all 9 provinces of the country. Most indigenous African sheep breeds do not show clinical signs of BTV infection and therefore the disease is not considered to be of major concern in many sheep rearing rural communities in the country. A large population of susceptible improved European wool and mutton breeds in which outbreaks of clinical disease are common, are however commercially farmed in South Africa. From 1998 to 2000 the number of outbreaks reported per annum amongst sheep varied from 28 to almost 100, with the majority of outbreaks occurring during the wettest years. Most unvaccinated sheep are infected with BTV at an early age and BTV antibody-negative sheep are therefore not readily found. For example, a sero-prevalence survey for BTV-specific antibodies was conducted amongst Merino sheep in the high-lying regions of the Eastern Cape, where sheep are not vaccinated since BT disease is not recognized. This survey indicated that a large proportion of Merino sheep that were bled in early spring and autumn tested positive for antibodies against the virus. Sero-prevalences amongst Merino sheep varied on a monthly basis from 1% to 84%. A second survey was conducted in the wet, fairly tropical and low lying regions of KwaZulu Natal. In this survey a primarily unvaccinated rural/communal population of indigenous livestock or crosses thereof was targeted. In total 2852 animals were sampled of which a quarter consisted of sheep and the remainder consisted of goats. BTV antibodies were found in 63.7% of these animals (Peter et.al, 2012).

3. Risk Analysis

@RISK software was used to determine the probability distribution and the risk of each risk pathways. Iteration of 10,000 with a single simulation was done for each risk pathways.

4. Result and Discussion

4.1. Qualitative Risk Assessment

4.1.1. Importation of Infected Sheep

Improved sheep imported into Ethiopia from restricted areas, and certified under Regulation carry a non-negligible risk of introducing BTV to Ethiopia. Certification is based on such parameters as protection from vectors and results of laboratory tests (ELISA and/or PCR). The former may be mitigated somewhat by limiting animal movements to the Vector Free Period, strategic shipment to avoid times of greatest vector activity, use of appropriate housing, and use of repellents or insecticides. Failure of laboratory tests through false negative results, variance in interpretation of results between laboratories, or human error, could provide a means for BTV to be imported with infected sheep. Prevalence is high in imported sheep (from S-Africa; 63.7%), BTV is present in local sheep, cattle and wild ruminants as carrier and BTV has been found in imported sheep.

Conclusion of release assessment:

The total population of sheep in South Africa is approximately 28 million that are distributed across all 9 provinces of the country. Most indigenous African sheep breeds do not show clinical signs of BTV infection and therefore the disease is not considered to be of major concern in many sheep rearing rural communities in the country. A large population of susceptible improved European wool and mutton breeds in which outbreaks of clinical disease are common, are however commercially farmed in South Africa. From 1998 to 2000 the number of outbreaks reported per annum amongst sheep varied from 28 to almost 100, with the majority of outbreaks occurring during the wettest years. In total 2852 animals were sampled of which a quarter consisted of sheep and the remainder consisted of goats. BTV antibodies were found in 63.7% of these animals Pete et al., 2012. For the countries that do import improved sheep, the risk of a BVT incursion release due to sheep is considered high (Occurs very often). The uncertainty surrounding this estimate is, therefore, low.

4.1.2. Exposure Assessment

Spread of infection would depend to a great extent on the means of virus introduction. For example, vector spread of infected Culicoides across the Ethiopia could result in multiple foci of infection whereas the importation of a single infected ruminant could result in a more localized outbreak of disease. Spread of infection will depend on: Presence of a population of potential vector Culicoides, Climatic conditions favorable to midge survival and dispersal by wind and Susceptible small ruminant population.

In South Africa multiple serotypes circulate each vector season however the occurrence of different serotypes is unpredictable and is most likely influenced by herd immunity. Serotypes 1–6, 8, 11 and 24 are believed to have a high epidemic potential, whereas serotypes 1–6 and 10 which are more often associated with clinical disease in sheep are thought to have a high pathogenic index. Bluetongue virus has been isolated from many different Culicoides species in South Africa, however only two species, Culicoidesimicola and Culicoidesbolitinos, are recognized as significant in the transmission of BTV. Culicoidesimicola, of which its larvae primarily inhabit organically enriched soils, is the dominant vector of BT in regions throughout South

Africa, Bluetongue virus has also occasionally been isolated from mosquitoes that may play a role in the mechanical transmission of the virus.

Disease	Probability of Entry	Probability of Exposure
BTV	High	High

4.1.3. Consequence Assessment

The clinical presentation of BT can also vary widely amongst susceptible sheep and can range from sub-clinical infections in the majority of cases to severe disease and death of infected animals. The mortality among susceptible sheep ranges from 2-30% but can occasionally be as high as 70%.

Disease	Probability of Entry	Probability of Exposure	Consequences
BTV	High	High	High

The consequences of direct losses of livestock (illness in infected sheep, infertility, abortion and neonatal deaths, losses due to decreased production, lameness), welfare implications, bans on exports of live ruminants and germplasm to some destinations, and movement restrictions are likely to have severe economic impacts.

4.2. Quantitative Risk Assessment

4.2.1. Risk pathway

The pathway highlights the two key routes of entry; namely, the importation of infected animals and the windborne spread of infected midges. It is also important to note that we define disease entry as "the presence of a BTV positive animal in the Ethiopia," as opposed to the presence of an infectious vector. This is based on the assumption that an infectious vector reaching the Ethiopia is likely to infect the sheep and gives the 2 routes the same end points.

Risk path		Data	Risk variability			P
			Mn	Ml	Mx	
P1	Do the source population infected by BTV?	In total 2852 animals were sampled of which a quarter consisted of sheep and the remainder consisted of goats. BTV antibodies were found in 63.7% (1817/2852) of these animal (Peter.et al.,2012)				0.6370007Beta Distribution
P2	Do infected animals pas undetected during purchase?	The clinical presentation of BT can also vary widely amongst susceptible sheep and can range from sub-clinical infections in the majority of cases to severe disease and death of infected animals. The subclinical cases among susceptible sheep ranges from 2-30%, but can occasionally be as high as 70% .(Assumed)	0.25	0.3	0.35	0.3083333 Pert Distribution
P3	Do infected animals passed undetected by ELISA test?	Compared with SNT as the reference test, the sensitivity and specificity of the ELISA were 98 and 96%, respectively	0.98	0.96	0.035	0.03530639 beta binomial)

P4	Do infected sheep passed undetected during quarantine?	A large population of susceptible improved European wool and mutton breeds in which outbreaks of clinical disease are common, are however commercially farmed in South Africa. From 1998 to 2000 the number of outbreaks reported per annum amongst sheep varied from 28 to almost 100, with the majority of outbreaks occurring during the wettest years(Peter *et* al.,2012)	0.28	0.33	0.38	0.33 Triangular distribution
P5	Do infected sheep passed undetected during quarantine in Ethiopia?	A total of 90 serum samples were tested and 42 (46.67%) were positive for bluetongue virus antibodies(Woldemeskel el al.,2000)	.30	.467	.52	0.448 Pert
P6	Dose any estimated morbidity of BTV In Ethiopia?	Serological evidence of BTV infection was observed in 541 out of 784 ovines about 69.01%prevalence rate was recorded (Daniel Gizaw*et* al., 2016)	784	541	243	0.69005 Beta
The final risk of release of virus in the importing country (p final) = p1*p2*p3*p4*p5*p6						**0.0007073**

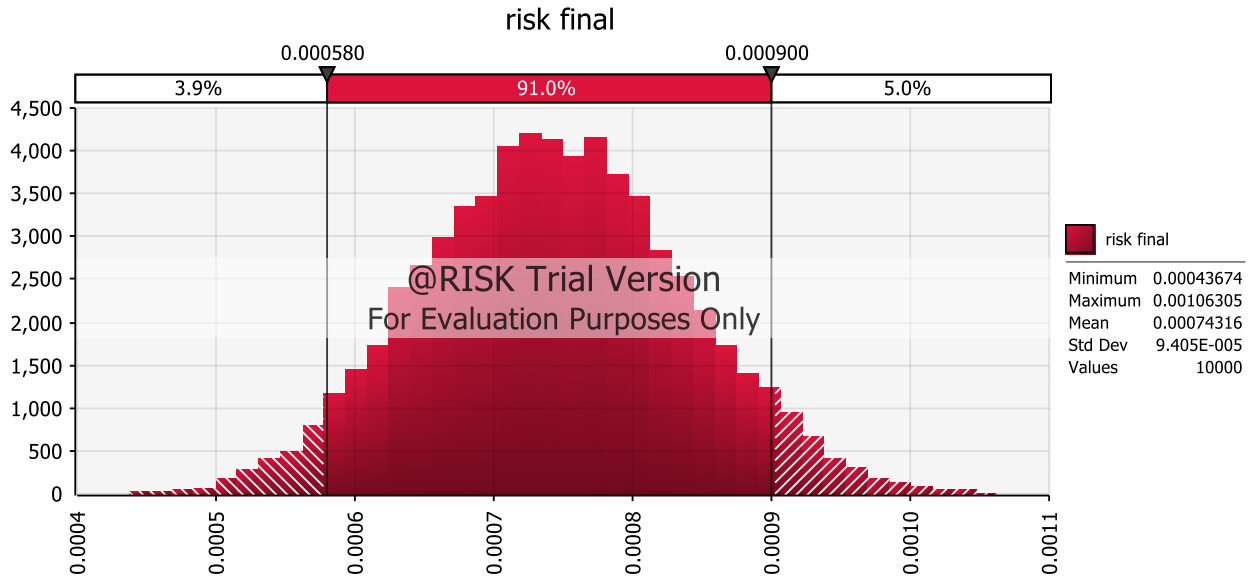

Fig.1.probablity distribution of risk

Result interpretation: The above graph shows that at 91% the risk is between 0.00580 and 0.0009 or at 91% confident the risk is between 5 animals/10,000 and 9 animals/10,000.

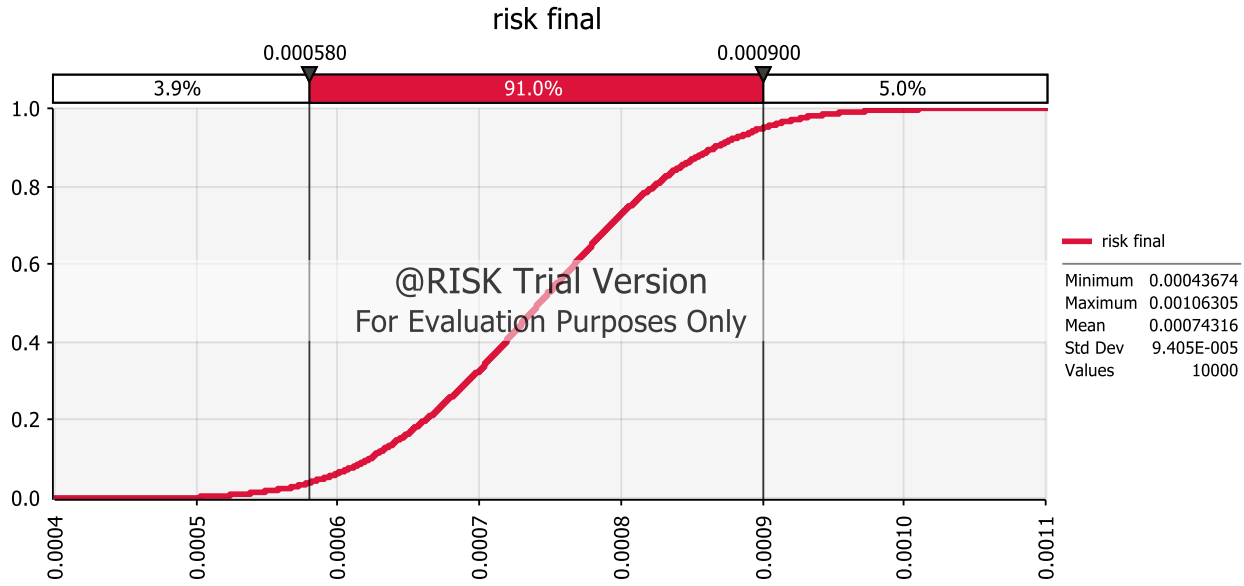

Figure 2. Cumulative distribution of risk

Result interpretation: The above graph shows that the cumulative risk, that means cumulative at 95% the risk is less than 0.0009 or there is a 5% chance that the risk is greater than 0.0009.

5. References

[1]. Afshar, A. (1994): Bluetongue: laboratory diagnosis. Com¬parative Immunology, Microbiology and Infectious Diseases **17**: 221–242.

[2]. Daniel Gizaw, DemekeSibhat, BrehanAyalew and MesfinSehal. (2016): Sero-prevalence study of bluetongue infection in sheep and goats in selected areas of Ethiopia. Ethiopian Veterinary Journal, **20 (1):**105-114.

[3]. Davies, F. G. and Walker, A. R., (1974): The distribution in Kenya of bluetongue virus and antibody, and the Culicoides vector. J. Hyg., 72, 265-272.

[4]. Du-Toit, R.M. (1944): The transmission of bluetongue and horse sickness by Culicoides. J. Vet. Anim. Ind., **19**: 7–16.

[5]. Elbers ARW, Backx A, Mintiens K, Gerbier G, Staubach C, Hendrickx G, van der Spek A (2008): Field obser¬vations during the bluetongue serotype 8 epidemic in 2006 II. Morbidity and mortality rate, case fatality and clinical recovery in sheep and cattle in Netherlands. Preventive Veterinary Medicine **87**: 31–40.

[6]. Haresnape, J.M., Taylor, W.P. and Lunggu, S.A.M. (1988): The epidemiology of bluetongue in Malawi. Epidem.Infect, **100**: 493-499.

[7]. Hofmann, M.A., Renzullo, S., Mader, M., Chaignat, V., Worwa, G. and Thuer, B. (2008): Genetic characterization of Toggenburg Orbivirus, a new bluetongue virus from goats, Switzerland. Emerg. Infect. Dis., **14**: 1855–1861.

[8]. Howell, P.G. (1979): The epidemiology of bluetongue in South Africa. In: Proceedings of the 2nd Symposium on Arbovirus Research in Australia (ed. T. D. St. George and E. L. French), pp. 117-123.

[9]. http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/documents/bluetongue_technical.PDF (accessed July 28, 2011).

[10]. Maclachlan, N.J., Drew, C.P., Darpel, K.E. and Worwa, G. (2009): The pathology and pathogenesis of bluetongue. J .Comp. Pathol, **141**: 1–16.

[11]. OIE (2014): Bluetongue. OIE: Territorial Manual, Office International des Epizootics (OIE). Paris, France. Pp. 1-18.
[12]. Peter Coetzee1, Maria Stokstad3, Estelle H Venter1, Mette Myrmel2 and Moritz Van Vuuren (2012): Bluetongue: a historical and epidemiological perspective with the emphasis on South Africa., Coetzee et al. Virology Journal.
[13]. Tweedle N, Mellor PS (2002): Technical review – bluetongue: The virus, hosts and vectors. Version 1.5. Report to the Department of Health, Social Services and Public Safety U.K. (DEFRA), Pp25.

5/5/2024