



Elements of the FMD control problem in Southern Africa: 2

Gavin Thomson

Commodity-based trade of beef and enhanced market access: The vital role of the Department of Veterinary Services

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Issues to be covered

- The effects and consequences of reduced pathogenicity of SAT viruses for cattle
 - Viral excretion → rate of transmission
- Antigenic variation specific to SAT viruses

Interaction between SAT viruses & different susceptible species

Species	Susceptibility to infection	Maintenance hosts	Pathogenicity	Persistent infection	Evidence for carrier trans.
African buffalo	++++	++++	-/+	++++	+
Cattle	++++	+	++	+++	-
Pigs	++	-	++++	-	-
Sheep	++	-	+	+	-
Goats	++	-	+	+	-
Impala	+++	-	+	-	-
Kudu	++	-	+	+	-

Southern Africa has a long tradition of considering cattle carriers a vital element in the control of FMD – but <u>no</u> objective scientific evidence for that

Occurrence of mild and subclinical FMD in cattle



KwaZulu/Natal, 2011



Mahembo West, 2008

- First reported in 1932 that FMD in what is now Zimbabwe was different from 'European' FMD (PJ du Toit)
 - inefficient transmission described in detail
- Subsequently:
 - Botswana ('occult FMD', Falkner) & Zimbabwe
- The South African 'event' in northern kwaZulu-Natal reported to OIE as <u>completely subclinical</u> (>100 000 cattle involved comprising many outbreaks!)
- Publications in peer-reviewed journals reporting evidence in this connection, in SE Zimbabwe & NE RSA (SAT3 – Jori et al., 2014; Lazarus et al., 2017) & Uganda (SAT1 – Dhikusooka et al., 2016)
- Mahembo West, 2008

Occurrence of mild and subclinical FMD in cattle (cont.)

Summary of FMD 'disease events' reported to OIE: 2011-2015

FMD viral lineage	No of events	AMR >10% (overall %)	Average AMR (%)
Eurasian (World-wide)	51	30 (58.8)	35.4
SAT (Southern Africa)	43	3 (7)	3.3

This has major implications for surveillance directed at SAT infections of cattle in southern Africa

What explanation is there for slow & inefficient spread of SAT infections in cattle populations in southern Africa?

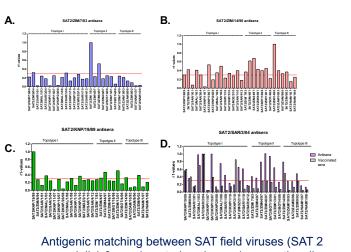
- Charleston et al, 2011 (Pirbright, UK) showed that quantities of 'infectivity' required to transmit the infection to other cattle is dependent on development of disease, i.e. diseased cattle more efficient transmitters than sub-clinically infected individuals
 - Likely that animals with mild disease are also less efficient transmitters than animals with fullblown disease
- Contributory factor: In southern Africa cattle raised in extensive systems → low density & low absolute numbers → lower amounts of infectivity released into the environment → inefficient transmission, i.e. slow & limited spread (what we observe)
- However, if animals are moved long distances 'artificially' (e.g. transport in vehicles) this
 won't apply (example: occurrence of FMD in Bulawayo introduced by cattle trucked from
 Mwanezi in Matabeleland South in 2015

An important issue

- The major source of SAT virus infections for cattle is buffalo (breeding herds) in the close vicinity; throughout southern Africa
- If cattle are infected frequently over protracted periods of time, SAT viruses can adapt to long-term survival in cattle populations (i.e. they can become independent of buffalo & behave like Eurasian serotypes)
 - This is the situation in East Africa for SAT1 and SAT2
- Question: Could this already have happened in Zimbabwe because outbreaks in cattle have been occurring continuously since about 2000?
 - No conclusive evidence yet one way or the other
 - However, if that happens cattle-adapted SAT viruses are likely to spread around the southern African region

SAT intra-typic variation

- Due to fragmentation of buffalo populations, SAT viruses have evolved independently in different locations resulting in different genotypes being distributed differently (topotypes)
- No clear subtypes with SAT serotypes complication for ensuring matching of outbreak viruses & vaccine strain
- Intra-typic variation greater within the SAT lineage than Eurasian lineage → further complicates 'matching'
 - e.g. SAT 2 virus consists of 14 genotypes
 - so control of SAT FMD using vaccine is more complicated (& expensive) than for Eurasian serotypes



especially) & vaccine strains viruses is exceptionally complicated

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Implications of major factors peculiar to SAT serotype FMD

Factor	Positive implications	Negative implications
Wildlife involvement	Carrier transmission is not a factor in livestock which reduces the need for extended quarantine & stand-still measures (widely applied in southern Africa)	Surveillance in wildlife populations difficult & expensive Movement of wildlife difficult to manage – fences are not completely effective Role of most cloven-hoofed wildlife unknown (lack of objective information)
Reduced pathogenicity for cattle	 Direct impact of the disease is reduced Reduced excretion of infectivity – favours most control measures because it reduces rate of transmission 	Physical inspection an unreliable surveillance tool, i.e. severely complicates surveillance
Rate of transmission	 Slow (except where long-distance transportation is involved) Further reduces need for long-lasting quarantine/stand-still periods 	
Extensive antigenic variation		Renders control through vaccination more problematic, less effective & expensive