



Foot-and-mouth disease in Africa

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Introduction

- The 7 serotypes are defined due to lack of cross protection
- Based on epidemiology, 2 different diseases?
- Africa:
 - Unique SAT types
 - More genetic and antigenic variation
 - Wildlife maintenance host with apparent co-evolution
 - New genetic and antigenic FMDV variants are generated
 - Unclear role of other cloven-hoofed wildlife in spreading and maintaining the disease
 - Various epidemiological patterns
 - Involvement of wildlife
 - Primarily livestock involvement
- SATs have caused very few outbreaks outside sub-Saharan Africa



Summary of SAT outbreaks outside sub-Saharan Africa

Country	Serotype	Year
Libya	SAT-2	2003
Egypt	SAT-2	1950
Saudi Arabia	SAT-2	2000
Yemen	SAT-1	1984
	SAT-2	1990
Jordan	SAT-1	1962
Israel	SAT-1	1962
Lebanon	SAT-1	1962
Turkey	SAT-1	1962-1965
Iraq	SAT-1	1962
Iran	SAT-1	1962-1964
Kuwait	SAT-1	1969-1970
	SAT-2	2000



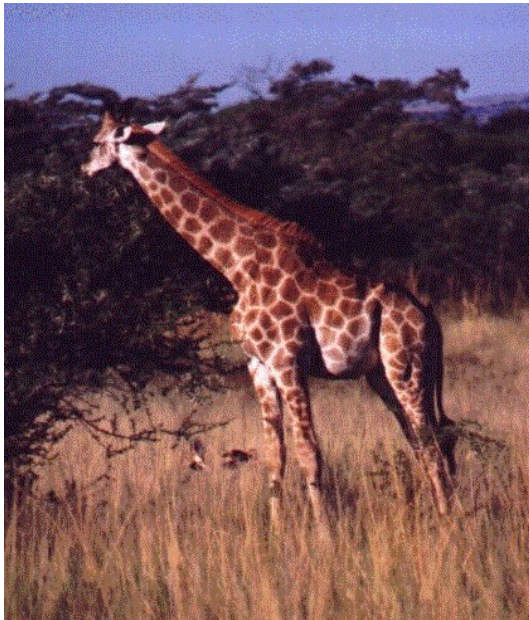
FMD in sub-Saharan Africa

- The SAT serotypes predominate in southern Africa, while serotypes O and A also occur in other parts of sub-Saharan Africa
- Different “patterns” of FMD occur - dependant on wildlife and/or on domestic animals





To control FMD in the face of increasing integration of land-use, we need to understand the behaviour of SAT viruses in domestic animals and wildlife and how FMD is transmitted from wildlife to livestock





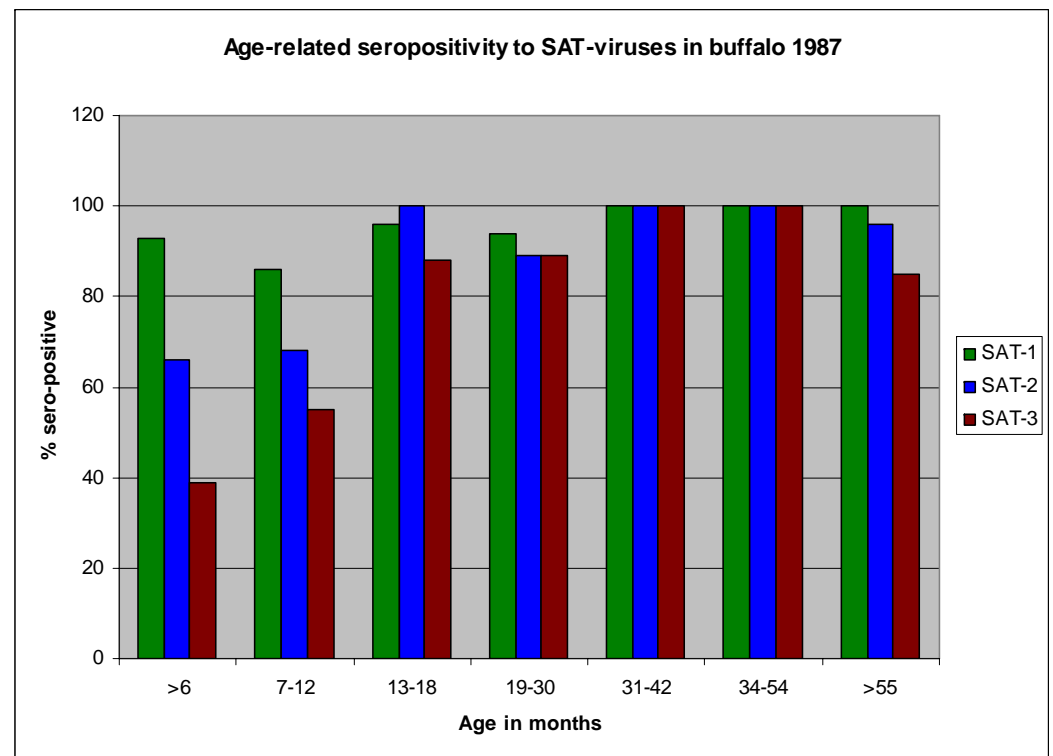
Role of African buffalo in the epidemiology of FMD in southern Africa

- The 3 SAT serotypes predominate in southern Africa
- They are maintained by African buffalo (*Syncerus caffer*) that can be a source of infection for susceptible livestock in close proximity
- Infection in buffalo is sub-clinical and normally occurs in calves as soon as maternal antibodies wane at 2-6 months of age





- Thereafter infection is rapid: in KNP > 80% develop antibody to SAT1-3 by 12 months of age
- VERY FEW animals, if any, develop clinical disease
- During acute infection, lasting about a week, there is considerable excretion of virus in all secretions





Features of the interaction between buffalo and the SAT type viruses

- During a monitored outbreak:
 - ~ 70% virus positive 1-4 months PI
 - 40% at 5 months PI
 - 17% after 6 months PI
- Carrier status may persist for 5 years or longer in a single animal and up to 24 years in an isolated herd





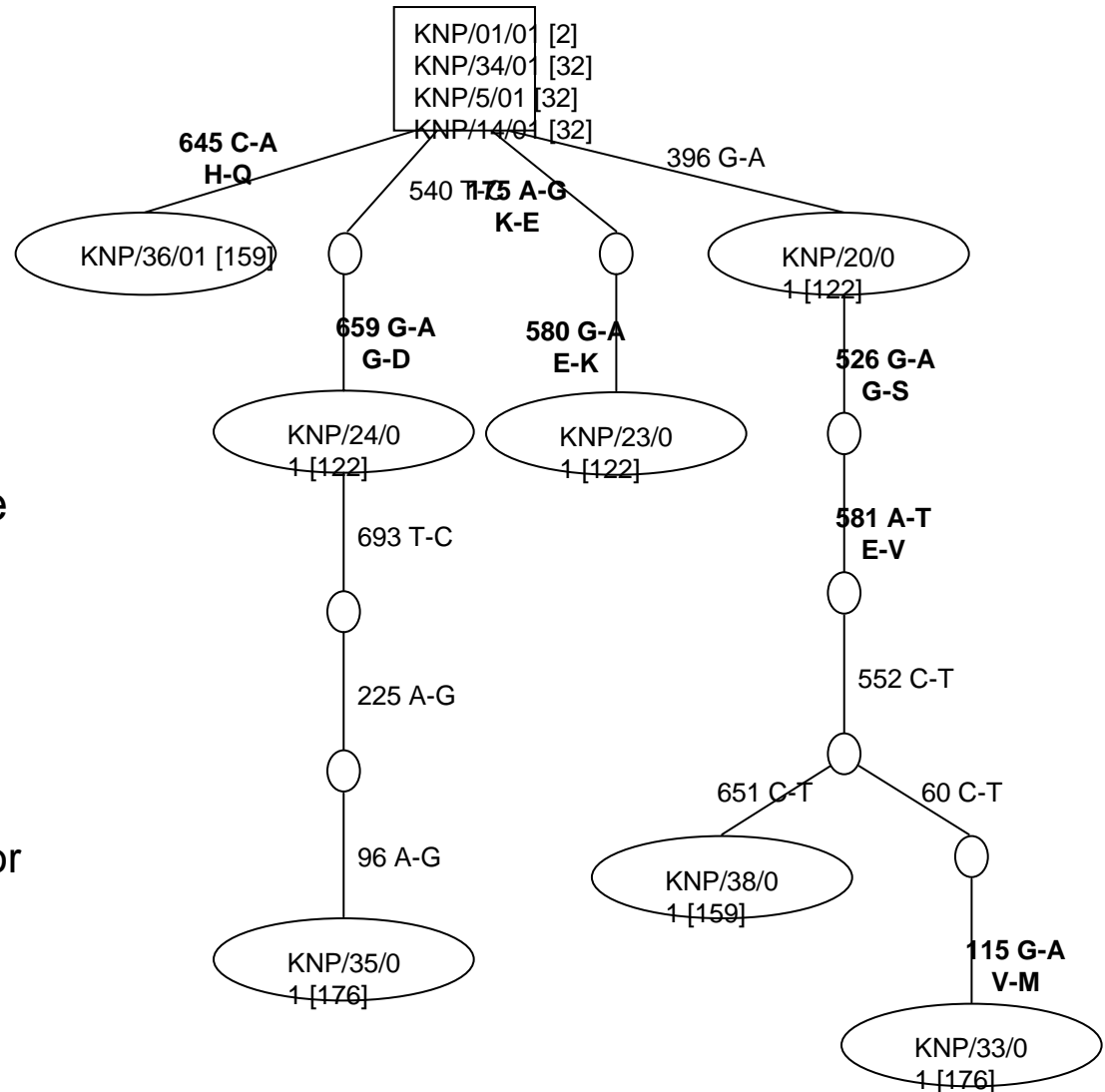
- Serological evidence indicates that more buffalo are infected by SAT-1 than by SAT-2, but historically SAT-2 outbreaks have occurred more frequently in other species
- Other wildlife species also become infected and their role in virus 'maintenance' is not clear, although they can transmit FMD to domestic animals in the acute phase of infection





Persistence as generator of genetic variants

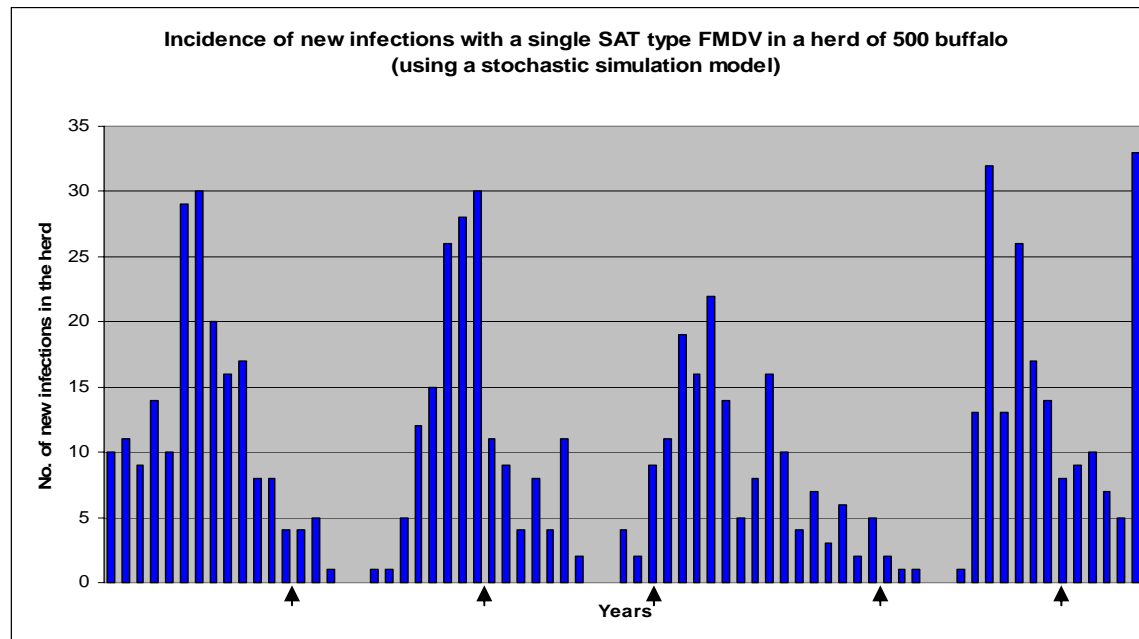
- FMD viruses change during persistent infection and may give rise to new antigenic variants as shown by at least 3 infections in buffalo (2 x SAT-1 and 1 X SAT-2)
- Molecular clock estimated to be 3×10^5 nt substitutions per site per day for SAT-1 infection in buffalo
 - Seven amino acid changes occurred 1D of which four occurred in hypervariable regions previously described for SAT-1





Features of the interaction between buffalo and the SAT type viruses

- Mode of transmission between buffalo and susceptible animals is not known
- Theories: childhood infection in young calves





Features of the interaction between buffalo and the SAT type viruses

- Theory: sexual transmission
 - 20 bulls were tested, semen and sheath wash one 3.5 year old seropositive bull from the (no probang taken)
- Between 1996-98 a total of 94 bulls were tested (sperm, sheath, testis and epididymis washes)
- Not all samples were taken from each individual
- In addition, one more 3 year bull tested pos (testis wash)
- Virus could be isolated in ~ 2% of the adult buffalo
- {Samples from the sexual tracts of 56 buffalo cows (3 months - 12 years) yielded no virus}



Re-infection rates in adult buffalo

- Most buffalo become infected when maternal antibodies wane between 2-6 months
 - For how long do they stay protected from reinfection?
 - How solid is immunity to new viral variants?
- SAT-1 buffalo outbreak in captivity, all animals had high titres to all 3 serotypes, yet SAT-1 outbreak occurred and 27/29 animals harboured detectable virus
- In the adult buffalo sampled for the sexual study, 13% aged between 3 and 9 years old were sero negative
- Therefore, within buffalo herds, susceptible animals of all ages are present
- Evidence of sexual transmission from persistently infected buffalo is tenuous



The role of stress in activating virus shedding from persistently infected buffalo

- Both published clinical outbreaks were in captive buffalo
- Circumstantial evidence is strong that buffalo can infect livestock with whom they get into close contact
- Buffalo that escape from the KNP are mostly adult animals and not young calves that could be actively infected
- Field observation is that young heifers transmit disease
- Factors that could induce virus shedding need to be further investigated

It is not known whether buffalo in East Africa are carriers of the O and A type viruses



The role of impala in the epidemiology of FMD in Africa

- Outbreaks in impala are derived from buffalo herds as demonstrated by sequence analysis of viruses isolated from outbreaks in impala and persistently infected buffaloes





The role of impala in the epidemiology of FMD in Africa

- Between 1967 and 1998 23 outbreaks in impala were recorded in the KNP
 - 65% SAT-2
 - 30% SAT-1
 - 5% mixed SAT-1 and SAT-2



Period	No of outbreaks
1967-79	8
1980s	10
1990s	5
Since 1999	No clinical outbreaks



Changes in FMD seroprevalence over a ten-year period in impala in the KNP

- Study sites
 - Three geographically distinct areas of the KNP, where dense impala populations occur and the history of recent FMD outbreaks in impala were known, were selected for this study during 1997
- A total of 40 impala were sampled once every three months in each area
- Impala were bled and clinically examined



Evidence of sub-clinical infection

- No pos animals were ever found in Shingwedzi
- On at least 3 occasions, mixed SAT-1 and SAT-2 infections were possible in the Orpen area
- Six SAT-2 outbreaks occurred in the Crocodile Bridge area in the 10 year sampling period
- Where mixed infections occurred, the serological response lasted longer (more than 3 years in the Orpen area)



Ecological reasons for differences in sero-prevalence

- Shingwedzi: unsuitable grazing, lower densities of impala
- Crocodile Bridge: suitable grazing that supports high densities of impala, sustainable water in river, less frequent animal contact
- Orpen: grazing supports high densities of impala, water mostly at bore holes and dams, more animal interaction





Risk factors for sero-prevalence

- Summer and autumn were highest risk factors for sero-positivity, but clinical infection were mostly observed at the end of the dry season
- More females and adults were sero-positive
- Impala do not become carriers, but long term infections can be maintained between herds
- Various complex factors play a role in the epidemiology of FMD in impala

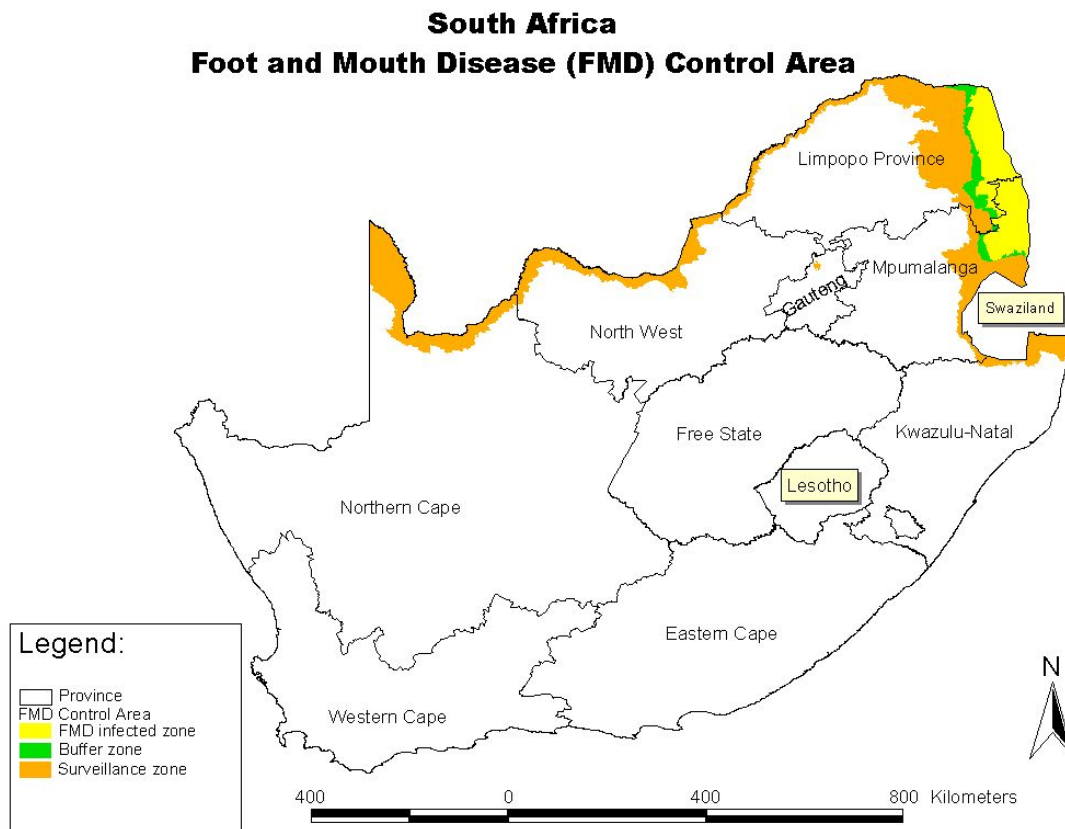


Evidence of impala infecting cattle

- The 1D gene of historical SAT-1 isolates obtained from impala and cattle between 1971 to 1981 were sequenced
- Five outbreaks occurred in impala between 1971 and 1981 of which four could be directly linked to cattle
- For three of the epizootics, disease was reported in cattle 5-6 months after the first impala isolates were made, confirming the direction of spread to be from wildlife to cattle
- One outbreak in cattle on the border of the KNP grouped with buffalo viruses occurring in the Park
- Since then, no outbreak in cattle could be linked to outbreaks in impala



FOOT AND MOUTH DISEASE CONTROL IN SOUTHERN AFRICA



- Successful control relies on the use of fences, zoning, movement control and vaccination
- Vaccines have serious deficiencies
 - Custom made vaccines for different regions
 - Cold chain nearly impossible to ensure
 - Cost of vaccine prohibitive
 - Incorrect application



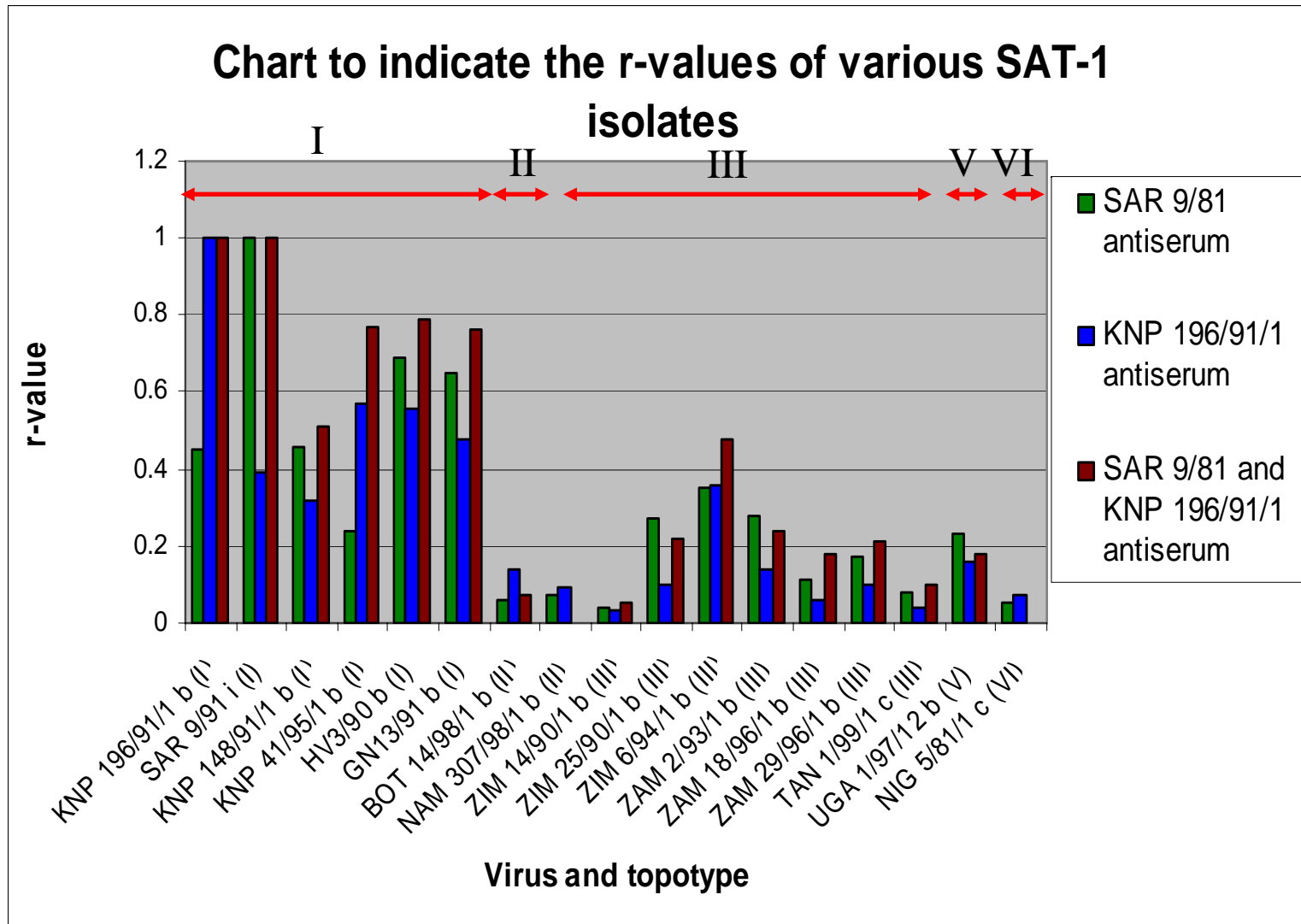
Molecular epidemiology of FMD in Africa

- For all serotypes occurring in Africa, geographically distinct genotypes / topotypes occur
- The genetic and antigenic variation have implications for vaccination policies





Antigenic variation between various topotypes





Evidence of increased number of FMD outbreaks

- **Summary of outbreaks in South Africa since 2000**
 - Previous outbreak in FMD-free zone 1957 and in control zone 1983
 - Since 2000: 6 outbreaks in cattle (2000, 2001, 2003, 2004 and 2006)
 - 1 SAT-1
 - 3 SAT-2
 - 1 SAT-3
 - 1 O





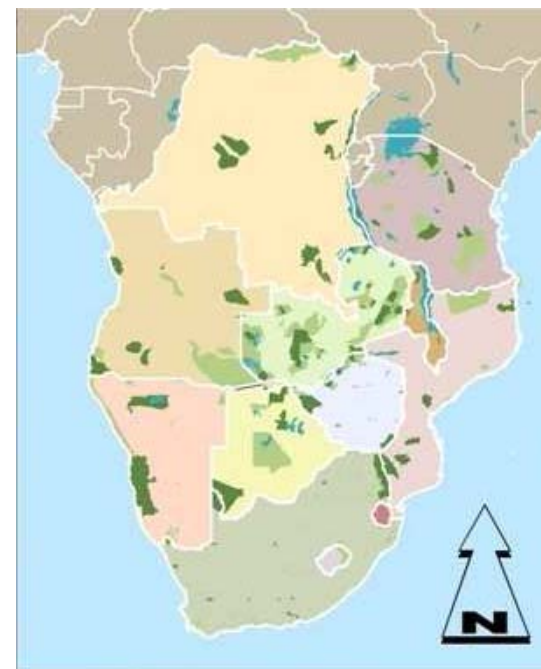
Possible reasons for the increase in outbreaks in southern Africa

- Floods of 2000 severely damaged the KNP fence
- Increase in elephant populations that damage the fences
- Unnatural water points removed in KNP with more pressure from elephants
- Reaction time to remove stray buffalo has increased
- Repair of fences not always up to requirements
- Staff morale
- Political instability
- Impact of vaccination
 - Changes in vaccination zones
 - Efficacy of vaccination
 - Quality of vaccine
 - Maintenance of cold chain
 - Efficiency of vaccination campaigns



Current developments that will impact on FMD control in Africa

- The establishment of TFCAs
 - Pressure to remove fences
 - Human encroachment into wildlife areas
 - Increased wildlife migration
 - Introduction of novel FMD virus topotypes and impact on vaccines
- Political instability (Zimbabwe, Kenya, Sudan, etc.)
- Lack of funds in the face of more important needs (human health, education)





FMD in Africa – a different ‘disease’?

- Possibly the origin of FMDV
- 5 of the 7 serotypes occur (serotype C?)
- Marked genetic and antigenic variation, especially in the SAT types
- Wildlife involvement and maintenance
- Eradication is probably not possible, control is challenging
- Africa will remain a constant threat to the livestock industries rest of the world