

FOCUS ARTICLE: THE IMPORTANCE OF WEST NILE VIRUS LINEAGE

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Since 1996 West Nile Virus (WNV) has re-emerged as an arbovirus of both public health and veterinary concern. WNV is a non-contagious viral disease transmitted predominantly by *Culex* mosquitoes in a bird and ornithophilic mosquito enzootic amplification cycle. Incidental infections occur in horses, humans and other mammals as a result of bites from haematophagus bridging vectors.

Though initially thought to be two lineages, phylogenetic analysis of West Nile Virus has defined five distinct lineages that differ from each other by 20-25% at the complete genome level.

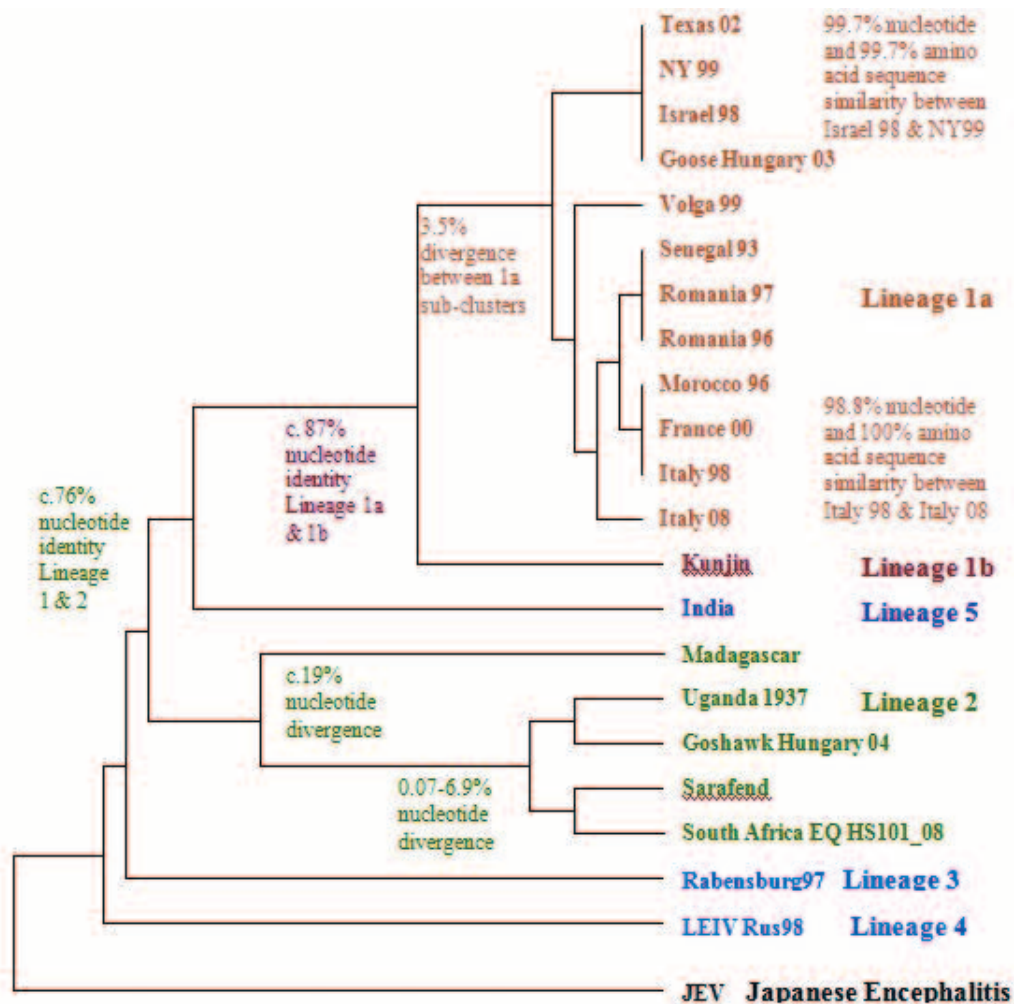


Figure 1: Phylogenetic analysis of West Nile Virus lineages (E-protein genome)

Lineage 1 is the most geographically widespread (1a Europe, Africa, Americas & Asia; 1b Kunjin Australia). Lineage 2 has been found in Africa and recently related to avian mortality in Central Europe (Hungary 2004 & 2005, Austria 2008). Lineages 3 and 4 have been identified in Russia and lineage 5 (formerly classified as 1c) in India.



Horses are susceptible to infection by both lineage 1 and 2, however it is not the individual WNV lineage, but the neuroinvasiveness of the individual virus serotypes that influence the development of clinical signs.

WNV presentation is a function of lesion location and produces a neurological syndrome characterised by a combination of clinical signs. The majority of WNV cases are subclinical or present with non-specific signs such as depression, anorexia and stiffness that resolve within 24-48hrs. Mild transient pyrexia (38-39oC) may occur, but is often undetected. Serological investigations in a number of WNV outbreaks indicate that many horses produce antibodies but few develop clinical signs or mortality. WNV infection can however be life threatening to clinically affected horses.

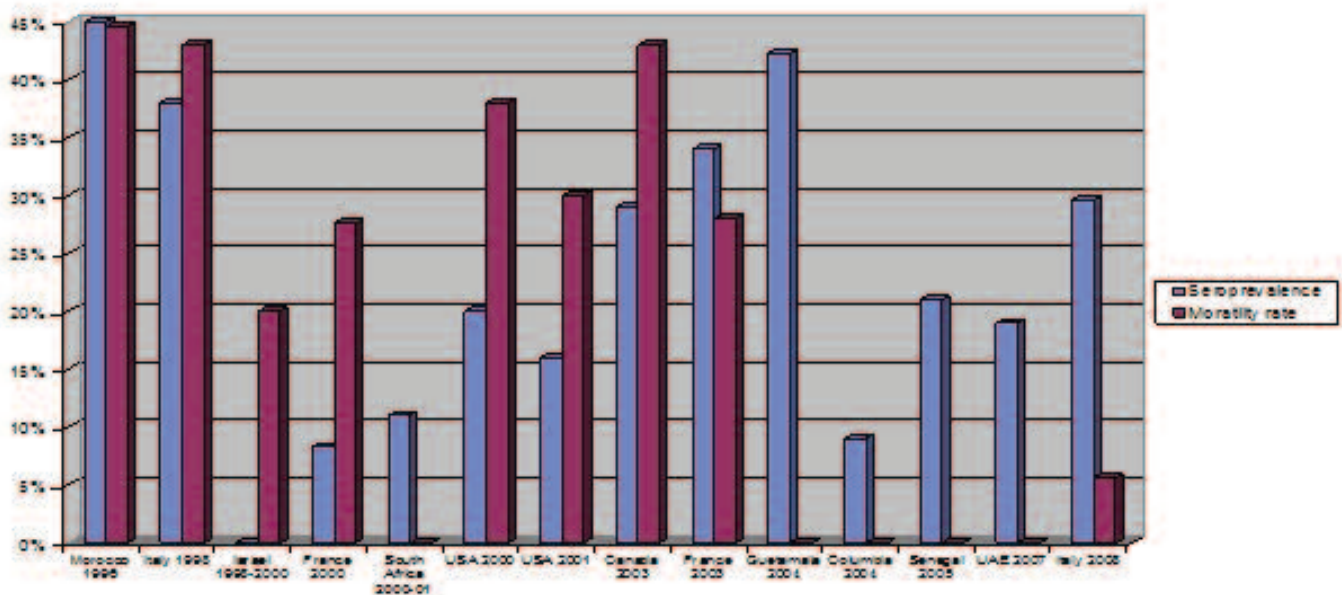


Figure 2: Comparison between observed seroprevalence and mortality rates for WNV lineage 1 and lineage 2 (South Africa 2000-01).

Mortality and recovery rates differ both between and within lineages. In the Italy 1998 lineage 1a outbreak the mortality rate was 43%, but all of the horses that did recover did so fully and within a 5-15 day clinical course. This contrasts with the North American lineage 1a WNV outbreaks, where mortality rates ranged up to 43%, but had mean clinical courses >21 days and recovery rates of only 79-93%. This North American lineage 1a WNV serotype then spread into the Caribbean in 2004, with a seroprevalence of 9-42.3% in the absence of clinical disease. A neuroinvasive Lineage 2 strain in South African 2008 killed 5/7 clinically affected horses. The two surviving horses did fully recover after protracted rest over several months. Wider seroprevalence of this strain in South Africa is unknown, however in endemic regions equine WNV may reach high seroprevalence rates. In Romania lineage 1a WNV seroprevalence was reported at 56% in the absence of clinical signs, while maximum seroprevalence of African lineages has been reported at 75-86% with an annual infection rate of 11-21%.

WNV infections have been confirmed in horses ranging from four months to 38 years. Age prevalence does not seem to be lineage related, though a number of equine case studies have shown an age-related incidence in WNV lineage 1a infection in the 6-10 and 10-16 year age brackets. This may not be a true risk but a factor of equine use and mosquito exposure, or biased dependent on the economic or emotional value of the equines and whether reporting of suspect clinical signs is compulsory in that country.



Concurrent disease does not seem to play a major role in the pathogenesis of lineage 1a WNV infection in horses; however co-infection in South Africa with African Horse Sickness, even in vaccinated horses, or Equine encephalosis may facilitate the central neuroinvasive quality of WNV lineage 2 leading to a greater case prevalence of blindness and seizures.

Ataxia and weakness are the major presenting clinical signs of WNV. Ataxia and muscle fasciculation may indicate a better prognosis than paresis, paralysis or central nervous signs. Though this is predominantly a function of diffuse lesions and less severe neuronal damage, recumbent horses have a poor prognosis due to intensive nursing requirement and associated secondary problems.

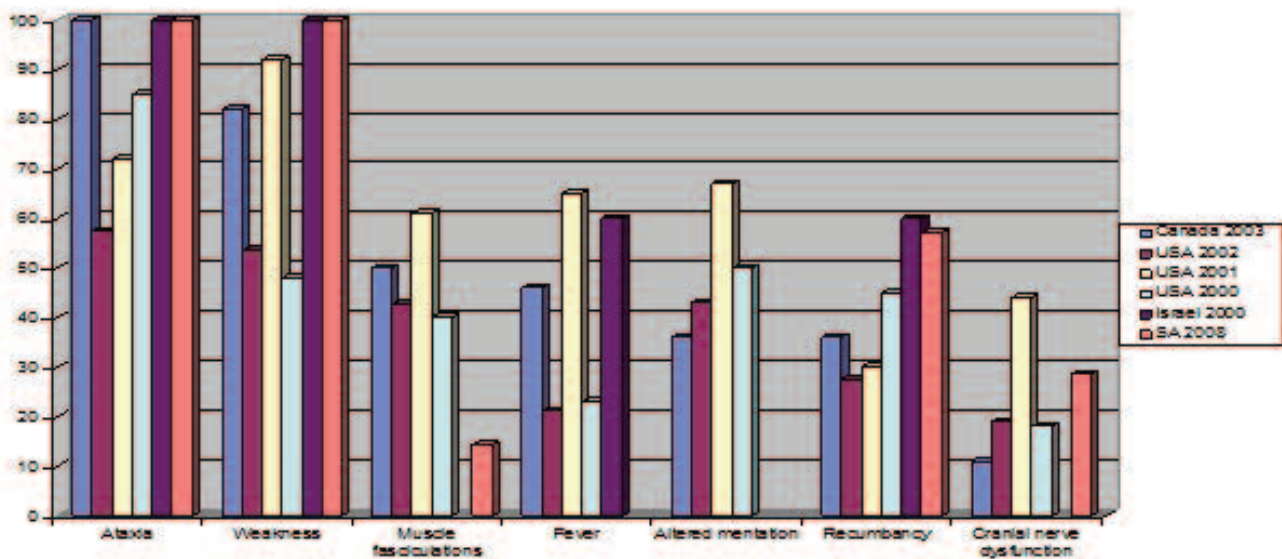


Figure 3: Clinical presenting signs associated with lineage 1 and lineage 2 (South Africa 2008) outbreaks

The risk of WNV introduction into the UK by horses remains very low (please see Defra-webpage for published risk assessment). Syndromic surveillance and the timely reporting of equine neurological disease can help detect any initial WNV case, regardless of WNV lineage, and provide a vital early warning of WNV circulation in the UK. Timely reporting and diagnosis also allows for more accurate assessment of the geographic distribution of WNV and will guide public health and veterinary preventative control measures with the aim of minimising any subsequent morbidity and mortality.

In the UK WNV is a notifiable disease. Any suspicion of WNV should be reported immediately to your local Animal Health office.

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