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West-Nile in the Caribbean

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Since its discovery in Uganda (1937), West Nile (WN) fever historically remained confined in Africa and Middle East with sporadic incursions in Southern Europe. However, it has expanded in the last decades, and is now one of the most widespread arboviruses in the world. Nowadays WN remains present over six continents.

The disease is produced by a virus (WNV, genus *Flavivirus*) and transmitted by mosquitoes among susceptible hosts, usually birds. The virus can also affect dead-end hosts, like humans and equines. The identification of the drivers for WN emergence and spread is difficult. At local scale, WN transmission cycle can occur through different ecosystems with involvement of different species of vectors and hosts. High viral genetic variation and wide range of vectors and hosts makes WN a complex arthropod-borne disease. Recently, up to nine different WNV lineages have been proposed. WNV has been detected in more than 60 mosquito species in 11 genera. However species in the *Culex* genus are considered the main WNV vectors worldwide. Major amplifying hosts are birds, with more than 300 species of birds supporting infection. Mammals are generally considered as dead-end hosts as they are not efficient WNV amplifiers. Nevertheless, multiple mammalian species, amphibian and reptiles are susceptible to WNV infection.

WNV emerged in the New World in New York, 1999. Since then the virus provoked in the USA the major WNV epidemics ever recorded globally. Disease burden was high, causing significant morbidity and mortality in birds, horses and humans. The disease further spread northward (Canada) and southward. The southern spread of WNV into the Caribbean, Central and South America was apparently silent. In contrast to USA and Canada, WNV has caused no or very limited health impact on animal and human populations in the Caribbean. The apparent absence of bird mortality and clinical manifestations among humans or equines makes difficult to track WNV spread in the region. Thus, evidence for WNV circulation is mostly based on serological evidences in a region with other antigenically cross-reacting viruses potentially co-circulating. In the Caribbean, the disease was recorded for the first time in October 2001 in Cayman Islands, on a patient without previous history of travel. First serological investigations were implemented by 2001/2002 across the Caribbean Sea in Mexico, in the Greater Antilles (Dominican Republic, Jamaica and Puerto Rico) and in the Lesser Antilles (Guadeloupe). Such early wave of activities serologically enabled to detect WNV circulation among birds and equines in the Caribbean. Since then, other serological studies supported evidence for consistent WNV circulation in the Caribbean region, Central and South America, including records of sporadic human and equine cases.

The Great Caribbean region is very diverse and heterogeneous. Wide environmental and climatic variation is found along its number of islands but also some continental countries/territories. The Greater and Lesser Antilles are situated at the Carrefour of North and South America, along the "Mississippi and the Atlantic migratory flyways". The most likely way of WNV introduction in the region is through infected wild birds flying from North to South America. What remains unclear is whether endemic WNV cycles were established or whether detection follows regular introduction by wild birds. Also in some islands (Martinique at least, Guadeloupe's sister island) several serological investigations have been conducted in horses. However these investigations never succeeded in evidencing WNV circulation suggesting heterogeneous distribution of WNV in the region due to

(still) undetermined factors. Also, the diversity of climate and environments in the Caribbean (Greater Antilles vs Lesser Antilles) suggests different epidemiological cycles.

Unfortunately, information on mosquitoes and hosts is scarce and heterogeneous, and viral isolations have been much more challenging than expected. Therefore the dynamics of WNV in the Caribbean still remains puzzling. Identification and characterization of viral strains circulating, enzootic and bridge vectors and potential amplifying hosts remains a key issue. Similarly, the marked difference in epidemiological patterns and significant differences in morbidity between North and Southern continent remains unexplained. Ongoing activities on WNV in the region aim at reducing knowledge gaps on WN fever in Caribbean ecosystems.

Avian influenza in Belize

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Belize implemented its active surveillance programme for avian influenza (AI) in 1999. This was as a result of the high threat arising from the avian influenza H5N2 outbreaks which started in 1994 in the neighbouring country of Mexico. When Guatemala and El Salvador reported low pathogenic avian influenza H5N2 in 2000 and 2002, respectively, the active surveillance for avian influenza was strengthened. In 2009, through the implementation of the Belize Poultry Improvement Plan (BPIP), avian influenza monitoring of chicken broiler and layer breeder flocks commenced with breeder flocks being tested three times in their life. 2014 was no different than other years in avian influenza surveillance activities including active and passive surveillance and monitoring of breeder flocks.

Blood samples collected from active and passive surveillance for avian influenza in 2014 all tested negative. Blood samples collected under BPIP in 2014 also all tested negative to avian influenza except for samples collected in Spanish Lookout, Cayo District from an 8000 chicken broiler breeder flock 39 weeks of age, in early December, 2014. The sera that tested positive as well as swab samples from AI antibody positive birds were sent to the National Veterinary Services Laboratory in Ames, Iowa, USA, a reference laboratory for the World Organisation for Animal Health (OIE). Thus, by the 22 January 2015 the Belize Agricultural Health Authority (BAHA) had confirmation that there was an exposure to avian influenza H5N2. As there was great uncertainty as to the nature of the exposure particularly as antibody-positive birds were not showing any clinical signs of disease, sentinel birds were placed in known exposed flocks. PCR confirmation was obtained on the 14 February 2015. The virus was sequenced as: North American LPAI H5N2 98.8% similar to A/CK/Mexico/55-12/2012 H5N2

The Belize viruses are highly similar to Low pathogenic avian influenza (LPAI) H5N2 viruses isolated in Mexico. The Mexico LPAI viruses have circulated in poultry in Mexico since 1995 and are well adapted to poultry. Virus characterization results received the 12 March 2015 identified the virus as low pathogenic avian influenza by cleavage site analysis as well as in vivo assay. There have never been any clinical signs associated with the LPAI H5N2 virus in Belize; producers are, in fact, reporting better performance of their poultry but this is probably due to the enhanced management and biosecurity measures implemented.

BAHA responded swiftly to the serological detection of avian influenza by the immediate implementation of quarantine and movement control, enhanced biosecurity at farm and community level and the testing of all long lived poultry in Spanish Lookout, surrounding villages and communities considered high risk. Country wide surveillance has been strengthened with at risk communities having commercial poultry being periodically tested.

The epidemiological surveillance showed that the outbreak was localized in a hot zone in Spanish Lookout and in two nearby villages, Buenavista and Billy White. Once the avian influenza virus was characterized as LPAI H5N2 and virus circulation confirmed, additional control measures were implemented: stamping out of infected flocks and cleaning and disinfection. Vaccination was considered as a control measure but it has not been approved. Movement control at six designated checkpoints considerably reduced the movement of risk poultry and poultry products. Security surveillance in the area has led to confiscations of spent hens smuggled out of the infected