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Satisfying control options for influenza
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Drawing on real-life examples from past influenza outbreaks, particularly 2009 H1N1 pdm and 2013 H7N9, this talk will highlight the state of the science in influenza preparedness research, in mitigation of annual epidemics, the next pandemic and newly emerging outbreaks otherwise. It will draw on the multiple disciplines of ecology, evolutionary biology, virology, epidemiology, and mathematical sciences. A “One Health” approach that recognises the zoonotic driver of epidemics will be emphasised. Particular attention will focus on the multiple strands of global health initiatives contributing to the common goal of health and human security against influenza and its sequelae.

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Zoonotic diseases at the human-domestic animal - Wildlife interface in Southern and Eastern Africa
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Southern and East African Countries are rich in ecosystems where human, livestock and wildlife populations are in close proximity and serviced by the ecosystems services such as water, land and fauna resources. In the course of mingling there are possibility of sharing pathogens which consequently may lead to outbreaks of zoonotic agents in the concern populations. In Tanzania various studies were conducted in the past decade which were determining the presence of zoonotic agents, the burden in individual populations, the dynamics and drivers of disease transmissions at the human-livestock-wildlife interfaces.

Using serological and molecular biological techniques, a cross sectional studies were conducted in human and animal populations at an various ecosystems neighbouring wildlife conservation areas of Tanzania. The selected agents studied included bacterial and viral zoonotic agents.

Microscopic Agglutination Test (MAT) was carried out to test for leptospira antibodies in 1,351 livestock and 42 wildlife. The overall seroprevalence was 26.35% and 28.57% with serovars of Leptospira Interrogans; Hardjo, Hebdomadis, Grippotyphosa, Sokoine and Lora were common. Similarly, 30% of 267 human samples tested positive, for almost similar serovars. Sequencing alignment on 16S ribosomal DNA gene, suggested that serovars of Leptospira interrogans were common among human and animal populations. Using Rose bengal as a screen test, a total of 5.75% and 11.9% of sera from domestic animal and wild animals were found to be positive respectively. The IDEXX Q Fever ELISA for the detection of antibodies against Coxiella burnetii was employed and 40 of 587 (6.8%) cattle and 15 of 22 (68.2%) of wild animals were found to have antibodies against C. burnetii. RVF virus testing conducted IgG and IgM ELISA revealed, thirty two out of 800 (4%) and eight out of 42 (19%) from domestic animals and wildlife tested positive for IgG respectively. Of the 440 sera from domestic animal tested for IgM only 15 (3.4%) had IgM, while all wild animal samples were negative. Under the PREDICT Project protocol, a total of 268 wildlife animal species (Bats, Rodents and Non human primates) were subjected to molecular virology diagnostic tests and revealed the presence of 64 viruses including 48 novel viruses. The identification of the novel viruses is still underway to determine the peculiar genus and species. Using the geographical information system, the locations for infected animals and humans congregated at same coordinates putatively indicate cross infections between two populations.

Findings from the present studies are providing important insight on presence of zoonotic agents which potentially may cause febrile illness among persons in frequent contact with animals and their products in the poor resources rural communities not only of Tanzania but across the developing world.

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Diseases are emerging and remerging rapidly in different ecosystems and regions. Disease surveillance is an approach widely used for detecting new pathogens through event-based or indicator surveillance efforts. We should ask ourselves why, as a global community, do we not implement a robust global surveillance and early warning system capable of detecting early signals of disease emergence? In addition, why is it that we continue to incur high costs of crisis mitigation, as in outbreaks of H5N1 HPAI, Nipah, MERS-CoV, Zika, Ebola, etc., particularly in regions associated with poor indicators of development and high vulnerability?

A new mindset is required to change the way that the international community coordinates and manages disease emergencies. Lessons learned from HPAI H5N1 outbreaks indicate that many affected countries continue to suffer disease impacts because of failures in implementation of technical strategies, poor practices and inadequate policies for disease prevention and control. A better understanding of the drivers of disease emergence is needed to help identify prompt actions that will tackle the issues at their source. A multidisciplinary approach is required to build a strong network of institutions and coordinate incidents at the complex human/animal/ecosystem interface. This approach also necessitates local capabilities and networks with epidemiologists or public health specialists capable of conducting disease outbreak investigations with the support of national or regional laboratory networks. Strengthening local capacities in epidemiological analysis, and the use of open analytical tools and GIS platforms, and new technologies (e.g. mobile devices, rapid diagnostics) are opening a new window of opportunities to enhance the quality and speed on how disease information is reported, detected, verified and communicated.

Disease information is available and circulating every day from the media, social networks, informal surveillance systems and official systems. Health information should be recognized as a public good and we should all practice due diligence to share publicly our data and information collectively. The international community should no longer wait for official reports to respond to disease threats. This new approach, however, requires a coordinated and joint effort among governments, communities, donors and international networks to invest in prevention systems with capability to identify early signals for the emergence, spill over and spread of animal pathogens (livestock production dynamics, trade issues and markets, climate change, civil unrest, consumer behaviors, etc.). A global and intelligent early warning system is needed to capture, analyze and transform data and information that can be used
effective for early detection of signals related to the disease emergence, spillover and spread at the interface.

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02.001
Teratogenic viral infections of the fetal central nervous system in animals: Timing and pathogen genetics are critical

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Congenital infections of domestic animals with viruses in several families, including Bunyaviridae, Flaviridae, Paroviridae, and Reoviridae, are the cause of naturally occurring teratogenic central nervous system defects. Congenital infections of ruminant livestock with bluetongue virus (BTV), a midge-transmitted arbovirus, have clearly shown the critical role of gestational age in determining outcome: specifically, fetuses infected prior to midgestation that survive congenital BTV infection are born with cavitating central nervous system defects that range from severe hydranencephaly to cerebral cysts (porencephaly). Generally, the younger the fetus (in terms of gestational age) at infection, the more severe the teratogenic lesion at birth. Fetuses infected after midgestation are often normal. Whereas congenital infection is characteristic of certain BTV strains, notably live-attenuated vaccine viruses that have been passaged in embryonating eggs, transplacental transmission is not characteristic of many field strains of the virus. Akabane and related teratogenic Bunyaviruses (e.g. Cache Valley, Aino, Schmallenberg and Rift Valley fever viruses, amongst others) also cause age-dependent teratogenesis in fetal ruminants but, in addition to cavitating central nervous system defects, affected fetuses are born with contracted limbs (congenital hydranencephaly/arthrogryposis syndrome). Pestiviruses are non-arthropod-transmitted members of the family Flaviridae that can cause teratogenic central nervous system defects in congenitally infected livestock, specifically bovine viral diarrhea virus in cattle and Border disease virus in sheep. Age-dependent virus infection and destruction of neuronal and/or glial cell precursors that populate the developing central nervous system is responsible for these naturally occurring virus-induced congenital defects of animals, thus lesions are most severe when progenitor cells are infected prior to their normal migration during embryogenesis. Although teratogenic viral infections of animals have been recognized for many years, much remains to be determined regarding the virological and animal host determinants of transplacental transmission of individual viruses. Importantly, in distinct contrast to primates, maternal immunoglobulins do not cross the umbilical placenta so that fetal ruminants are dependent on their own innate and acquired immune responses, which are acquired sequentially during gestation.

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02.002
Congenital Zika Syndrome

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The Zika virus (ZIKV) is a RNA virus in the family Flaviviridae, genus Flavivirus. ZIKV carries the name of a forest close to Kampala in Uganda, where it was first identified in Rhesus monkeys in 1947. In 1952 it was isolated in humans in Africa for the first time. In 2014, the ZIKV arrived in South America, notably in Brazil. Since then, a dramatic increase in cases of microcephaly was detected in several states, especially in the Northeast of Brazil. In April 2016, the causal relationship between microcephaly and Zika virus was proved.

After World Health Organization advised that the clusters of microcephaly and other neurological disorders and their possible association with microcephaly and Zika virus constitutes a Public Health Emergency of International Concern efforts are been made to describe and understand the syndrome.

A Congenital Zika Syndrome has as a main characteristic the brain impairment, with microcephalus, however it is still little known about this entity and its clinical spectrum that includes newborns with normal head circumference.

In addition to congenital microcephaly, a range of manifestations including craniofacial disproportion, spasticity, seizures, irritability, brainstem dysfunction such as swallowing problems, limb contractures, including arthrogryposis, hearing and ocular abnormalities, and brain anomalies detected by neuroimaging have been reported among neonates where there has been in utero exposure to Zikv virus.

The pattern of brain images abnormalities in congenital Zika syndrome has been fully described by Aragão et al (2016). Brain calcification and disorder of cortical development are the most frequent findings. Cerebellar atrophy and malformations of the brainstem may also occur. The pattern of calcifications at the junction between cortical and subcortical white matter, in addition to the cortical developmental disorders predominantly on frontal regions confers highly suggestive pattern of Zikv congenital infection.

The rare and unusual arthrogypotic joints did not result from abnormalities of the joints themselves and are likely to be of neurogenic origin, with chronic involvement of central and peripheral motor neurons, leading to intrauterine fixed postures and consequent deformities.

The ophthalmological abnormalities were already described by Paula Freitas et al and Ventura et al (2016) and Leal et al (2016) described sensorineural hearing loss associated with congenital zika syndrome.

There is a lack of studies regarding the consequence of congenital ZIKV infection at the third trimester of pregnancy. It is important to health professionals be alert to the changes in the neurodevelopment during the first years of life, especially if mothers describe record of cutaneous rash during pregnancy.

The complete neurological picture requires the central nervous system maturation and it will only become clear after, at least 18 months, so to a better definition of congenital Zika syndrome we need a longer follow-up.

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02.003
Mathematical models to elucidate the transmission dynamics and control of vector-borne disease

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Mathematical modeling offers a powerful toolkit to improve our understanding of infectious disease transmission and control. In particular, carefully calibrated mechanistic models of disease transmission can be used to forecast trajectories and likely disease burden of epidemics. This talk focuses on the key ingredients that need to be incorporated into mechanistic models in order...