Epidemiological investigation into the introduction and factors for spread of Peste des Petits Ruminants, southern Tanzania

Article *in* The Onderstepoort journal of veterinary research · June 2012

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ONDERSTEPOORT
Journal of Veterinary Research

Supplement No. 3

ISSN: 0030-2495

Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011

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Vol. 79 No. 2 2012

Volume 79 ■ Number 2

Daddy's girl (photo: Yvonne Chamberlain)

ONDERSTEPOORT
Journal of Veterinary Research

ISSN: 0030-2495 (print)
ISSN: 2219-0635 (online)

Preface

Proceedings

This supplement no. 3 is brought to you in association with the Southern African Centre for Infectious Disease Surveillance

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Towards one Africa, one health: The Southern African Centre for Infectious Disease Surveillance One Health focus on infectious diseases

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How to cite this preface:
Rweyemamu, M.M., Paweska, J., Kambarage, D. & Namuba, F., 2012, 'Towards one Africa, one health: The Southern African Centre for Infectious Disease Surveillance One Health focus on infectious diseases', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #449, 2 pages. http://dx.doi.org/10.4102/ojvr.v79i2.449

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

The genesis of the Southern African Centre for Infectious Disease Surveillance (SACIDS) is rooted in the realisation that infectious diseases are and will continue to be a formidable challenge to human welfare and economic development in Africa over the horizon of 30 years from 2005, and thereby impede Africa's ability to meet the targets of the UN Millennium Development Goals (MDG). Several recent studies indicate that Africa probably has the highest burden of infectious diseases of humans and animals and yet the least capacity for their detection, identification, monitoring and risk management. Whilst climate change, changing in habitation and farming systems, globalisation of travel and trade are shared drivers for infectious diseases globally, certain other drivers have special relevance to Africa. These include the high human-livestock-wildlife interaction, land use and socio-economic settings.

The realisation of opportunities offered by new technologies for risk management of the conventional and emerging communicable diseases in humans and animals will require the integration of research across sectors (human, animal, environment) and disciplines (natural and social science) plus surveillance and disease control strategies that take account of cultural and governance settings. Such approaches will, increasingly, have to be based on ecological systems, which often transcend administrative or national boundaries.

It is against this background that African experts in infectious diseases of humans, animals and plants advocated in 2005 a Pan-African Vision for Infectious Disease Management as:

> A Pan-African concerted effort, shared by AU member governments, reflecting the needs of African society and supported by the international community, with the goal of a society protected from the ravages of dangerous infectious diseases that compromise either human health or livelihoods and agriculture and economic development.

They also recommended that such a vision was best developed through national and regional clusters.

The above considerations propelled academic and research institutions involved with infectious diseases of humans and animals (domesticated or wild, terrestrial or aquatic) in southern Africa to form the SACIDS (http://www.sacids.org), with the vision of:

> A Southern African society protected from devastating infectious diseases affecting the health of humans, animals, i.e. both terrestrial and aquatic, and plants, i.e. crop, forest and ornamental, thereby promoting livelihoods, socio-economic development including market access and the environment.

The *SACIDS Mission* is: to harness innovation in science and technology in order to improve southern Africa's capacity (including human, financial and physical) to detect, identify and monitor infectious diseases of humans, animals, plants and their interactions in order to better manage the risk posed by them.

From the standpoint of such a Vision and Mission, the SACIDS 'One Health' focus is to address infectious diseases in the endemic settings of sub-Saharan Africa, with a particular attention to southern, central and East Africa through a *collaborative effort between natural and social sciences to advance the understanding of interactions between humans, animals and the environment to improve public and animal health.*

Thus the SACIDS model is one of a Virtual Centre that links the core institutions in southern Africa with centres of Excellence in the 'North', bound by a common mission and focus on One Health and whose operation seeks to focus on intra-African as well as South-South-North collaboration.

Our research strategy is reflected in the themes of this conference, namely:

1. Climate dependent, vector-borne diseases.
2. Diseases with potential inter-species concern or spread between wildlife, livestock and humans.

3. Diseases of economic importance.
4. Bacterial rare diseases.
5. Dangerous emerging diseases.
6. Systems for disease surveillance and preparedness analysis.
7. Socio-economic approaches to One Health policy research.

The SACIDS philosophy is to work towards: One Africa, One Health. Accordingly, we seek to collaborate with other programmes, such as Afrique One, EAIDSNet, (East African Integrated Disease Surveillance Network) OHCEA (One Health Central and Eastern Africa), AFENET (African Field Epidemiology Network), RUFORUM (The Regional Universities Forum for Capacity Building in Agriculture) and others that share this philosophy through a One Health approach, irrespective of their specific strand of emphasis on 'One Health'. The new Strategic Framework of SACIDS to 2020 is:

> A Sub-Saharan African society protected from devastating infectious diseases affecting the health of humans, animals, i.e. both terrestrial and aquatic, and ecosystems, thereby promoting livelihoods, socio-economic development including market access and the environment Our convening of the First One Health conference in Africa at the National Institute for Communicable Diseases, Johannesburg is testimony to our commitment to this goal. Over the coming months and years we intend to collaborate with others of like mind to focus our attention on this Vision through the study and application of One Health approaches.

We trust that the readers of this special issue of the Onderstepoort Journal of Veterinary Research will appreciate the diversity of disciplines and expertise that is already beginning to collaborate effectively towards the goal of: One Africa, One Health through smart partnerships.

Acknowledgements

Southern African Centre for Infectious Disease Surveillance is funded primarily by grants from the Wellcome Trust, Rockefeller Foundation and Google Foundation. We are also grateful to the support of Sokoine University of Agriculture, and those sponsors, such as FAO (Food and Agriculture Organisation) of the United Nations, who have funded some targeted activities.

One Health: Towards safeguarding the health, food security and economic welfare of communities

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How to cite this preface:
Nabarro, D., 2012, 'One Health: Towards safeguarding the health, food security and economic welfare of communities', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #450, 3 pages. http://dx.doi.org/10.4102/ojvr.v79i2.450

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

It is a privilege for me to be invited to join this conference organised by young African scientists who are exploring the opportunities for integrated approaches to animal and human health. Thank you for inviting me.

In my early professional assignments I worked with communities in Southeast Asia and East Africa. I sensed that their food systems, patterns of agriculture, nutrition and health were inextricably linked. Their leaders often told me that dealing with a range of different professionals was intensely frustrating and what they needed was multi-skilled professionals who really understood the challenges people face. They need to appreciate how rural life and livelihoods vary from season to season, or change over the years as climatic conditions, access to infrastructure or new initiatives by government or civil society alter the environment in which people live and work.

In my presentation today I shall focus on ways in which the 'One Health' approach is contributing to food security and economic well-being of communities as well as helping to safeguard their personal health. I appreciate that you have been discussing these issues in depth in the last two days: I hope that you will share your observations with me.

One Health thinking

One Health is a way of thinking that reflects the reality of people's lives and livelihoods. That thinking encourages a focus on new kinds of outcomes, policies, actions and research agendas – different from those which will be pursued if we stay within our normal professional disciplines.

One Health thinking, outcomes, policies, action and research are important for:

- increasing the number of people who enjoy food security – who have stable access to nutritious foods for their needs
- ensuring bio-secure and ecologically sustainable production of safe food for people to eat
- encouraging accessible local markets and fair international trade in agricultural products.

The risk of disease emergence at the interface between animals, humans and ecosystems is at the current core of *One Health thinking*. It can include illnesses that have an indirect effect on human health through their impact on people's livelihoods … and the drivers that increase the risk of emerging diseases (climate change, resource depletion, land degradation, demand, lack of bio-security, contamination).

We should not try to define One Health thinking and action too tightly. Please do not see one health as a scientific discipline or international programme. Instead let us look at how different stakeholders – in government, amongst farmers and food processors, within consumer groups and civil society – use the concepts in practice.

A serious global food security situation

We face a global situation where an estimated 925 million people go hungry. The effects of recent food price increases are likely to deepen the vulnerability of those who spend between 50% and 80% of their family budget on food, mostly basic staples.

Efforts to respond to food insecurity, food safety and food trade challenges have addressed all four dimensions of food security namely, availability, access, utilisation and stability. Progress has been made at several levels such as:

- countries affected by food insecurity
- investors in countries' efforts to improve food security
- the organisations that support both countries and investors and
- the system for governance of international arrangements to assist affected countries (including the revitalised Committee on Food Security).

Rising prices present a great opportunity for farmers to respond to growing demand. But farmers need inputs and cash to do this. They need help to manage the risks associated with trying to produce more. And they need the infrastructure to market their food. Well-functioning food markets and trade have huge potential to increase small farmers' integration into value chains so as to increase the benefits they can capture from the trade in their products.

Drivers for animal disease

Farmers seeking to increase their livestock holdings have particular challenges. They can find themselves facing outbreaks of disease in their animals: these might contribute to the emergence of infectious disease emergence at the human-animal-ecosystem interface.

The livestock sector is an area in which One Health thinking and action can make a difference to lives and livelihoods. Estimates from the World Bank on the projection for the increase in meat production over the next 40 years indicate that most of this will occur in the developing world. Improving livestock production in a sustainable and humane manner that balances both carbon and water footprints is a major challenge facing the One Health community.

For the 75% of the world's poor that are rural and dependent on agriculture, disease outbreaks in livestock not only put at risk their immediate food source, but it also puts at risk their livelihoods and resilience capacity – and that affects their long-term food security.

Disease outbreaks which reduce the availability of live animals and livestock products can reduce household income, undermine the diets of household members, impair nutritional status *and increase risks to health*, especially of women and children. Outbreaks can also impair the wider market availability for those products. Chronic food insecurity also drives risky behaviours related to animals: no one who is well-fed would consider consuming the carcass of an animal that has died of disease.

Effects of animal disease extend to people who work in production and processing – including livestock and agri-food workers, transporters and sellers. *One Health* thinking helps us find ways to limit these risks and encourage resilient livelihoods.

Agricultural intensification and lack of biosecurity can also result in food borne disease: The WHO (World Health Organisation) estimates that in 1997, food contamination cost up to $35 billion in the United States alone due to medical costs and lost productivity.

Fear of losing business can also result in further disease spread in a clandestine way. The potential negative economic impacts of restricting the movement and sale of diseased animals or animal products (a common response to disease emergence) may unfortunately drive some linked to the industry to try to work around bans on sales and trade in order to maintain their income – and in the process, potentially extends a disease outbreak and makes it harder to control.

The World Bank has calculated that:

> The emergence of BSE, SARS, H5N1, and influenza A(H1N1) have caused over US$20 billion in direct economic losses over the last decade and much more than US$200 billion in indirect losses.

Better food security and food safety through One Health principles

In the UN (United Nations) system we seek coordinated responses to food insecurity and unsafe food through *movements* of multiple actors. These encourage a broad range of stakeholders to work in synergy as they pursue immediate and longer term food security outcomes. The work goes best if undertaken under the leadership of national authorities. We focus on multi-disciplinary and multi-stakeholder *movements*, anchored to institutions but not controlled by them.

We also encourage multi-stakeholder platforms where different groups work together and coordinate support particularly for smallholder-based food security initiatives and nutrition-sensitive agricultural development. We have developed a Comprehensive Framework for Action (CFA) for food security and a Road Map for Scaling up Nutrition (SUN) to guide the emergence of these movements.

What have we learnt over the past few years in applying one health thinking to food and nutrition security, food safety, markets and trade?

- *Firstly, the need for country leadership*: Effective action to ensure food security and safe food for all happens if led from within countries.
- *Secondly, the need for clarity on results*: In our work the emphasis has been on equitable and sustained improvements in people's lives, long-term health and resilient livelihoods.
- *Thirdly, the need for joint working*: by institutions which tend to function separately. These include government services – and their ministries – with separate responsibilities for animal and human health, the environment, as well as for agriculture and trade.
- *Fourthly, the value of being inclusive*: working with and responding to stakeholders outside government – particularly organisations of smallholder farmers and those who work in the livestock val ue chain, businesses, agriculture unions, consumers' organisations, local food producers.

Putting One Health thinking into practice

Government Ministers expect us bureaucrats and professionals to advise them on how best to avoid risk

associated with disease at the animal-human-ecosystem interface. We have made real progress in the area of H5N1 Highly Pathogenic Avian Influenza, and in relation to SARS and spongiform encephalopathy. But we have much more to do with regard to sustainable livelihoods of pastoral communities, rabies, or what some refer to as the forgotten zoonoses. I know that you have been discussing some of these in your meeting.

I am keen to see *One Health* thinking become a central feature for responsible national and international policy making. This means putting the lessons we have learnt into practice:

- Firstly, never forgetting that our primary clients are those most at risk of food insecurity or most likely to be affected by unsafe food or market failure.
- Secondly, working together for sensible and realistic policies to improve food and nutrition security, sustainable and bio-secure production, safe livestock products, functioning markets and fair trade.
- Thirdly, overcoming our tendency to work in our professional niches and bureaucratic silos, and instead sharing data and analyses, developing joint policies, doing research together, implementing joint investigations and being accountable for delivering results. It helps if we undergo training together, too.
- Fourthly, focusing on outcomes that have meaning to the business community, to human health, animal welfare and wildlife advocates, to politicians and to the media and then working hard to demonstrate and communicate our results.

Within the UN system our Secretary General advocates for comprehensive, and increasingly integrated responses to the current challenges we face in the health, food and agriculture, climate change and trade interface. He encourages broad-based partnerships that focus on results. He requires our different organisations to work together and to link up with governments, regional organisations, private companies, civil society and – most importantly – farmers' organisations. He wants us to explain the virtues of working in a joined up way and of breaking down professional silos. Only then can we contribute to safeguarding the health, food security and economic prospects of poor communities.

We have seen real progress in the African Union – especially with pastoral communities and in the work being done by FAO (Food and Agriculture Organisation), WHO and the OIE (Office of International Education). We are delighted to see the emergence of multi-disciplinary networks of professionals dedicated to supporting equity, reducing poverty and encouraging resilient livelihoods. We are pleased that young professionals like you are taking responsibility for working effectively at interfaces in ways that respond to people's realities. You listen to the people you serve, to each other, to politicians and other stakeholders and to your own instincts as you take this important work forward. You are helping to build trust and respect between people and professionals, between different professional disciplines and between departments and organisations. You are impatient for results so you try to work in synergy rather than in competition. You appreciate what matters to people rather than spending time telling them what they should do – or feel.

Much more needs to be done. Here are my requests to you today:

- Please continue to work in ways that reflect One Health thinking and action.
- Please look for opportunities to advocate for One Health – in national parliaments, regional assemblies and globally; with farmers' organisations, labour unions and civil society groups; with businesses and entrepreneurs.
- Please do what you can to inspire wider and stronger networks of people who are excited by the potential of effective one-health working that empowers communities to act in ways that improve health and livelihoods.
- Please contribute to the growing movement for One Health that works in the service both of poor communities and of the global good, brings together women and men, farmers and consumers as well as social and physical scientists, and engages all nations.

Ebola virus outbreaks in Africa: Past and present

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How to cite this proceeding:
Muyembe-Tamfum, J.J., Mulangu, S., Masumu, J., Kayembe, J.M., Kemp, A. & Paweska, J.T., 2012, 'Ebola virus outbreaks in Africa: Past and present', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #451, 8 pages. http://dx.doi.org/10.4102/ojvr.v79i2.451

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Ebola haemorrhagic fever (EHF) is a zoonosis affecting both human and non-human primates (NHP). Outbreaks in Africa occur mainly in the Congo and Nile basins. The first outbreaks of EHF occurred nearly simultaneously in 1976 in the Democratic Republic of the Congo (DRC, former Zaire) and Sudan with very high case fatality rates of 88% and 53%, respectively. The two outbreaks were caused by two distinct species of Ebola virus named *Zaire ebolavirus* (ZEBOV) and *Sudan ebolavirus* (SEBOV). The source of transmission remains unknown. After a long period of silence (1980–1993), EHF outbreaks in Africa caused by the two species erupted with increased frequency and new species were discovered, namely *Côte d'Ivoire ebolavirus* (CIEBOV) in 1994 in the Ivory Coast and *Bundibugyo ebolavirus* (BEBOV) in 2007 in Uganda. The re-emergence of EHF outbreaks in Gabon and Republic of the Congo were concomitant with an increase in mortality amongst gorillas and chimpanzees infected with ZEBOV. The human outbreaks were related to multiple, unrelated index cases who had contact with dead gorillas or chimpanzees. However, in areas where NHP were rare or absent, as in Kikwit (DRC) in 1995, Mweka (DRC) in 2007, Gulu (Uganda) in 2000 and Yambio (Sudan) in 2004, the hunting and eating of fruit bats may have resulted in the primary transmission of Ebola virus to humans. Human-to-human transmission is associated with direct contact with body fluids or tissues from an infected subject or contaminated objects. Despite several, often heroic field studies, the epidemiology and ecology of Ebola virus, including identification of its natural reservoir hosts, remains a formidable challenge for public health and scientific communities.

Introduction

The first Southern African Centre for Infectious Disease Surveillance (SACIDS) conference on 'One Africa, One Health' served as inspiration for this review to illustrate the concept through a typical emerging infection. Ebola haemorrhagic fever (EHF) is caused by any of five genetically distinct members of the *Filoviridae* family: *Zaire ebolavirus* (ZEBOV), *Sudan ebolavirus* (SEBOV), *Côte d'Ivoire ebolavirus* (CEBOV), *Bundibugyo ebolavirus* (BEBOV) and *Reston ebolavirus* (REBOV). *Côte d'Ivoire ebolavirus* has been associated with only one human case (Le Guenno *et al*. 1995). *Reston ebolavirus* has only caused disease in non-human primates (NHP) and was found in swine suffering from porcine reproductive and respiratory disease syndrome (Barrette *et al*. 2009). Zaire, Sudan and Bundibugyo Ebola viruses are responsible for most of the EHF outbreaks (Feldmann *et al*. 2005; Groseth, Feldmann & Strong 2007; Towner *et al*. 2008) but ZEBOV constitutes a particularly serious threat to both human and NHPs in sub-Saharan Africa. Ebola haemorrhagic fever has been associated with large human outbreaks, with case fatality rates for ZEBOV as high as 90%. The case fatality rate of EBOV in NHP is unknown but some ecological data suggest that EBOV has contributed to declines of up to 98% of local great ape populations in Gabon and the Republic of Congo (Walsh *et al*. 2003).

Currently there are no approved antiviral drugs or vaccines against filoviruses. The prevention of EHF requires improving our understanding of the epidemiology of the disease, especially the role of wildlife, including bats, in the transmission of Ebola virus to humans. In their exhaustive review on Ebola virus, Feldmann and Geisbert (2011) tackled different fundamental aspects of EHF outbreaks. Leroy, Gonzalez and Baize (2011) reviewed the major scientific advances in our understanding of the ecology, host interactions, and control of the Ebola and Marburg viruses. In the present review we report important features related to Ebola outbreaks in Africa based on previous findings and own observations during major outbreaks that occurred on the continent.

Documented human and non-human primate outbreaks in Africa

Ebola viruses constitute a serious threat to both human and wildlife health in the Congo and Nile basins. The first documented outbreaks were generally regarded as causing a mysterious

disease, so dramatic in its effect that it inspired novelists and film producers. In most of the cases, the disease has appeared suddenly out of the elusive natural environment and dissipated slowly during the outbreak. The first outbreaks of EHF occurred almost simultaneously in 1976 in southern Sudan (June) and northwestern Zaire (now Democratic Republic of the Congo, DRC) (September). Initially it was thought that the DRC outbreak was due to dissemination of the Sudan outbreak but, in fact, the outbreaks were caused by two antigenically and biologically distinct species named SEBOV and ZEBOV. The index case in Sudan was a worker in a cotton factory in Nzara who subsequently was the source of nosocomial transmission in Maridi hospital. The mortality rate amongst the 284 notified cases was 53% (WHO 1978a). The index case in the Zaire or DRC outbreak was a 44 year-old male instructor at the Yambuku catholic mission school who fell ill after extensive travels in northern Equateur Province. He bought fresh and smoked antelope and monkey meat on his way back to Yambuku. He was treated for presumptive malaria at the Yambuku hospital, where the outbreak emerged subsequently. In total 318 cases were recorded, with a case fatality rate of 88%. Close contact with an acute Ebola case and receiving an injection with a reused, unsterilised syringe at the hospital were the major risk factors for virus transmission in humans (WHO 1978b).

In 1979, Nzara and Maridi in Sudan were again hit by a small outbreak, with 34 cases and 22 deaths (Baron, McCormick & Zubeir 1983), whereas a single case was described in a child at Tandala hospital in DRC (Heymann *et al*. 1980). Apparently, none of the cases had contact with wild animals.

After an absence from 1980 to 1993, several independent foci of Ebola virus transmission were recorded. Most of them were caused by ZEBOV and SEBOV but some were caused by the newly discovered species, namely CIEBOV and BEBOV:

- Ebola haemorrhagic fever in Côte d'Ivoire (1994): A large Ebola virus outbreak occurred amongst chimpanzees living in the Taï National Park in Côte d'Ivoire. An ethologist was infected whilst performing an autopsy on a dead chimpanzee. The patient was treated as a presumptive malaria case in Abidjan hospital, without success. There were no secondary cases. This was the first documented outbreak of Ebola virus amongst NHP in nature and the first in West Africa. The outbreak led to the discovery of a new species of Ebola virus, namely CIEBOV (Le Guenno *et al*. 1995).
- Ebola haemorrhagic fever in Gabon (1994, 1996, 1997 and 2001–2002): Several viral haemorrhagic fever outbreaks, caused by ZEBOV, were associated with the hunting of NHP. The 1994 outbreak involved gold-diggers in the Minkebé Forest who had killed a sick gorilla for food; the illness was initially confused with yellow fever (Amblard *et al*. 1997; Georges-Courbot *et al*. 1997a, 1997b; Leroy *et al*. 2002).
- Ebola haemorrhagic fever in the Republic of the Congo (2001–2002, 2003 and 2005): The first recorded outbreak of Ebola occurred in 2001–2002. In 2003, ZEBOV re-emerged, affecting 143 individuals in Mbomo (17 cases) and Kellé (126 cases), and 128 deaths were recorded (Formenty *et al*. 2006). Three independent index cases were identified in relation to hunting episodes and contact with gorillas. During this outbreak, intra-familial transmission was more important than nosocomial transmission. However, three health care workers were infected. In the same year, another small Ebola outbreak, involving 35 cases and 29 deaths, had occurred. The last documented Ebola outbreak in the country was reported in 2005, with 11 cases and nine deaths (Nkoghe *et al*. 2011).
- Ebola haemorrhagic fever in the DRC (1995): ZEBOV reemerged in 1995 in the city of Kikwit, with 400 000 inhabitants, 1000 km south of the location of the 1976 outbreak. In total, 315 cases and 250 deaths were recorded. A 31-year-old female Ebola patient traveled during the early stage of her disease to Kinshasa, where she was isolated in a private clinic. No secondary transmission occurred (Khan *et al*. 1999). The main occupation of the index case was farming and preparing charcoal in one of the remnant forest areas near Kikwit but the exact cause of infection or exposure is not known. There were no great apes but a lot of bats and rodents were present in the region. The risk factors for secondary human-to-human infection were mainly working in Kikwit general hospital or preparing corpses for burial. Almost 20% of the 250 victims were health care workers (Guimard *et al*. 1999).
- Ebola haemorrhagic fever in the DRC (2007–2008, 2008–2009): A further outbreak occurred in 2007, in the Mweka health zone, West Kasai Province, involving 264 cases and 187 deaths with a case fatality rate (CFR) of 71%. Kampungu city was the epicenter of the outbreak with 47% of cases, followed by the city of Kaluamba (42% of cases). The index case was the chief of the village and a hunter. The outbreak was apparently associated with a massive fruit bat migration through this region (Leroy *et al*. 2007). During this outbreak, the fatalities amongst health care workers were fewer. However, several human-to-human transmissions had occurred in churches where patients had been taken for prayers and nursing (Doctors Without Borders, unpublished data).
- In the 2008 Ebola outbreak, Kaluamba was affected again, with 32 cases and 14 deaths (CFR of 43.8%). The index case was believed to be an 18 year old girl who had died from a post-abortion haemorrhage. However, the source of her exposure remains unknown. This outbreak was reported to the national and provincial health authorities 21 days after the disease onset, compared to a period of four months in the 2007 epidemic. The observed low CFR in Kaluamba outbreak is considered to be due to the early recognition of the disease and the prompt response of the national team.
- Ebola haemorrhagic fever in Uganda (2000, 2007, 2011): An outbreak of SEBOV occurred in Gulu in 2000 and spread to the cities of Mbarara and Masindi, with a total of 425 cases and 224 deaths (CFR of 52%) (Lamunu *et al*. 2004). This was the largest epidemic caused by SEBOV. The outbreak was recognised from a cluster of human cases and was amplified by nosocomial transmission. Uganda was again affected in 2007 when a new Ebola species,

BEBOV, killed 30 people out of 116 cases (CFR of 26%) (Towner *et al*. 2008). An isolated case of EHF caused by SEBOV was reported from Uganda in 2011 (WHO 2011).

- Ebola haemorrhagic fever in Sudan 2004: The epicenter of this small outbreak of 17 cases and seven deaths (CFR of 41.2%) was the town of Yambio, near to the two previous Ebola sites (Nzara and Maridi) (Onyango *et al*. 2007). The outbreak started with the admission of a 27-year-old man to Yambio hospital with fever and haemorrhagic manifestations. The onset of symptoms started on 15 April 2004. The outbreak was contained rapidly with the establishment of infection control measures, thanks to the early recognition and confirmation of the outbreak by the Kenya Medical Research Institute (KEMRI).

Etiology

Ebola virus belongs to the family *Filoviridae*, in the order Mononegavirales which includes *Rhabdoviridae* and *Paramyxoviridae*. The virion is pleomorphic, producing 'U'-shaped, '6'-shaped, or circular forms but the predominant forms of the virion most frequently seen by electron microscope are long tubular structures. It contains one molecule of linear, single-stranded, negative-sense RNA of $4.2x10^6$ Da.

The virus was first recognised in 1976 when two unrelated EHF outbreaks occurred 800 km apart in northern Zaire (Yambuku) and southern Sudan (Nzara or Maridi) (WHO 1978a, 1978b). It was given the name 'Ebola' after the small river near the catholic mission of Yambuku, the epicenter of the 1976 EHF outbreak.

Ebola virus is not restricted to Africa. A new species, REBOV, was described in cynomolgus monkeys (*Macaca fascicularis*) imported from the Philippines (Manila) to a quarantine facility in Reston, USA in 1989. Subsequently, REBOV has been re-isolated from cynomolgus monkeys and domestic pigs in the Philippines (Barrette *et al*. 2009).

Transmission

In most outbreaks, Ebola virus is introduced into human populations via the handling of infected animal carcasses**.** In these cases, the first source of transmission is an animal found dead or hunted in the forest, followed by person-to-person transmission from index case to family members or health-care staff. Animal-to-human transmission occurs when people come into contact with tissues and bodily fluids of infected animals, especially with infected nonhuman primates (Leroy *et al*. 2004). Transmission has been reported in Côte d'Ivoire where an ethologist was infected through handling an infected, dead chimpanzee in the Taï Forest (Le Guenno *et al*. 1995). It was confirmed that the deaths of chimpanzees were indeed due to Ebola virus. In Gabon and the Republic of the Congo, outbreaks in humans were associated with extensive deaths of chimpanzees and gorillas (Rouquet *et al*. 2005). In contrast, the animal source of infection during the DRC, Uganda and Sudan outbreaks has never been detected. However, when analysing the risk factors for primary transmission of EBOV from a broad anthropological point of view, it is noticeable that the increase in Ebola outbreak since 1994 is frequently associated with drastic changes in forest ecosystems in tropical Africa. The perturbation of these ecosystems due to extensive deforestation and human activities in the depth of the forests may have promoted direct or indirect contact between humans and a natural reservoir of the virus. EBOV infection has therefore been related to human economic activities like hunting (young hunters infected by a chimpanzee in the forest near Mayibout, Gabon in 1996), farming (the charcoal maker in the forest near Kikwit, DRC in 1995) and gold digging (in Minkebé Forest, Gabon in 1994). In some cases, as in Mweka (DRC), EHF outbreaks seemed to be linked to the hunting of bats for bush meat (Leroy *et al*. 2009). Rarely, scientific activities have resulted in primary EBOV infection, for example, in the case of the ethologist who was involved in wildlife studies in the Taï Forest in 1994. These examples show clearly that certain economic activities, which many populations depend on for their survival, are risk factors for EBOV infection. This finding should be taken into account when public health measures need to be implemented in EBOV-endemic areas. Regarding human-to-human viral transmission, infection occurs in community and hospital settings through direct contact with infected fluids (blood, secretions and excretions) or tissues of an acute patient or through direct contact with contaminated materials. Any clustering of deaths in the same family pointed to EHF during the larger outbreaks. In the community setting, new infections were related to the ministration of funeral rites, which involve ritual cleansing of the cadaver and removal of hair, finger nails, toe nails and clothing before burial. People visiting or taking care of infected persons in their homes or in hospitals also risk being exposed to Ebola infections.

However, we have observed major differences in EBOV transmission cycles between community-based outbreaks and hospital-based outbreaks. When Ebola virus is introduced into a village, the outbreak seems to end spontaneously with limited generations of cases. As shown for the DRC epidemics in Table 1, Ebola virus was introduced into 55 villages around Yambuku (1976) and 25 villages around Kikwit (1995). The majority of the affected villages reported less than ten cases each. Similarly, the chain of transmission of EHF in the village of Ekata in Gabon was very short after the exposure of four brothers to dead animals in 2001. In contrast, a hospital setting with low standards of hygiene and sanitation rapidly becomes a source of epidemic amplification, especially if barrier-nursing techniques and universal hygiene measures are not observed by health workers. As a consequence, these nosocomial outbreaks are characterised by a relatively high proportion of deaths amongst health care workers. Ironically, it was the excessive fatalities amongst health care workers in Kikwit hospital in 1995 that brought the outbreak to the attention of the public health authorities. From this observation, it is possible that EHF infections are not as rare as generally thought. Isolated cases may frequently happen in the community without being reported. In support of this hypothesis, several epidemiological sero-surveys reported high prevalence of Ebola antibodies in communities in the absence of reports of Ebola outbreaks (Becquart *et al*. 2010; Busico *et al*. 1999; Gonzalez *et al*. 2000). Because IgG

TABLE 1: Distribution of Ebola cases in the affected villages in Democratic Republic of the Congo.

Number of cases	Number of affected villages			
	Yambuku (1976)		Kikwit (1995)	
	Number	%	Number	%
1	17	30.9	15	60
2–5	18	32.7	10	40
6–9	12	21.8	0	–
10–14	4	7.3	0	–
15–19	1	1.8	0	–
20–29	1	1.8	0	–
30 +	2	3.7	0	–
Total	**55**	**–**	**25**	**–**

antibodies are known to cross-react amongst Ebola species (MacNeil, Reed & Rollin 2011), this high seroprevalence may be the outcome of exposure to yet unknown, less pathogenic or non-pathogenic variants of Ebola virus.

Sexual transmission has been suggested in humans since filoviruses can be found in semen (Bausch *et al*. 2007). Aerosol infection is questioned since people sharing the same space with infected persons do not contract the infection even though aerosol infection of NHP has been demonstrated in the laboratory (Leffel & Reed 2004).

Ecology

Tropical rain forests in Africa constitute a common ecosystem for Ebola virus emergence (i.e. the Western Congo Swamp Forests near Yambuku, Taï Forest in Côte d'Ivoire and Minkebé Forest in Gabon), providing rich animal biodiversity, and epidemics appear to be seasonal. Documented human and non-human EHF outbreaks occurred mainly during wet seasons, marked by fruit abundance. The index case of the 1995 EHF outbreak in Kikwit fell ill in January and the 1994 EHF outbreak amongst chimpanzees in the Tai forest occurred in November, at the end of the wet season.

The natural reservoir of infection remains unknown, but the virus clearly has a zoonotic origin. In some outbreaks where information is available, the human index cases have invariably had direct contact with gorillas, chimpanzees, antelope or bats.

The search for a reservoir of EBOV has been very aggressive. Although great apes are generally involved in EHF outbreaks, NHPs are not thought to be natural reservoirs but, rather, susceptible hosts (Table 2) based on the sudden sharp decline in populations of the great apes in Gabon and the Republic of Congo which coincided with EBOV outbreaks in humans (Pourrut *et al*. 2005). Several other animal and plant species have been investigated for susceptibility and to determine a natural reservoir of EBOV. During the 1976–1979 EHF outbreaks, several ecological studies were conducted in order to identify the reservoir of the virus in nature. Studies were conducted on plants eaten by guinea pigs (Tandala, DRC), on monkeys (Yambuku, DRC) and on bats (Nzara, Sudan) but without success. Further ecological investigations using modern technologies, were carried out during the subsequent episodes of EHF outbreaks (1994–2010) especially in Kikwit, where thousands of rodents, insects and birds were screened. These investigations have not been successful for various reasons, one being that they are usually implemented retrospectively, several weeks or months after the index case has been infected by a putative reservoir. It is possible that by that time the putative reservoirs may have moved to another site. A surveillance system capable of early detection of Ebola cases could allow animal reservoir studies in 'real time', which is not always easy in remote places in African forests.

Experimental studies provide a more convenient, alternative method to identify candidate animal reservoirs and need not rely on an actual outbreak. Studies on 33 varieties of 24 species of plants and on 19 species of vertebrates and invertebrates experimentally infected with Ebola virus gave the first evidence that both insectivorous and frugivorous bats can support the replication and circulation of EBOV (Swanepoel *et al*. 1996). This evidence along with reports of bat exposures for some of the Ebola index cases directed the research toward the bats as potential reservoirs. Indeed, an ecological survey revealed the presence of ZEBOV-specific antibodies in six bat species caught in the field (*Epomops franqueti*, *Hypsignathus monstrosus*, *Myonycteris torquata*, *Micropteropus pusillus*, *Mops condylurus* and *Rousettus aegyptiacus*) (Pourrut *et al*. 2005). Viral nucleic acid sequences of Ebola virus was also found in three species of fruit bat during the 2001–2003 outbreaks in Gabon and Republic of the Congo (Leroy *et al*. 2005). These studies were pre-dated by the ecological investigation of the 1998–2000 Marburg heamorrhagic fever outbreak in Durba village in northeastern DRC, which consisted of repeated occurrences of short transmission chains arising in workers in Goroumbwa Mine where large numbers of bats roosted (Swanepoel *et al*. 2007).

TABLE 2: Documented outbreaks of Ebola virus amongst non-human primates and swine (1980–2005) in Africa.

Year	Non-human primates or swine	Location	Comments
1989–1990	Macaques	Reston, VA, USA	Discovery of a new species: Ebola Reston (REBOV)
1992	Macaques	Sienna, Italy	
1996	Macaques	Alice, TX, USA	
2009	Swine	Philippines	
1994	Chimpanzees	Tai Forest, Côte dôlvoire	Discovery of a new species: Ebola Côte dôlvoire (CIEBOV)
1996	Chimpanzees	Mayibout 2, Gabon	Ebola Zaire (ZEBOV)
2001–2002	Gorillas	Mendemba, Gabon	Ebola Zaire (ZEBOV)
2003	Gorillas	Mbomo, Republic of the Congo	Ebola Zaire (ZEBOV)
2005	Gorillas	Mbomo-Kellé, Republic of the Congo	Ebola Zaire (ZEBOV)
		Etumbi, Republic of the Congo	Ebola Zaire (ZEBOV)

A hypothesis of recurrent introductions of infection into humans from a natural source was supported by the finding that multiple genetic lineages of virus circulated during the outbreak. Diverse genetic lineages of Marburg virus were detected in Egyptian fruit bats, *R. aegyptiacus*, and two species of insectivorous bat in the mine (*Rhinolophus eloquens*, the eloquent horse-shoe bat, and *Miniopterus inflatus*, the greater long-fingered bat) and, furthermore, these lineages corresponded to the ones isolated from the humans in Durba. Due to the complexity of laboratory testing of specimens collected from animals potentially infected with filoviruses and the necessity to conduct the testing under biosafety level four (BSL-4) conditions, the results of the study were only published seven years later but in the meantime had been presented at numerous international scientific meetings and significantly assisted in focusing filovirus ecology studies on bats as putative reservoir species.

In this context, an Ebola ecology expedition was organised in May 2011 (Figures 1 and Figure 2) to undertake a preliminary study in Luebo, DRC where recent outbreaks of Ebola haemorrhagic fever occurred, and where cases of the disease were linked to catching bats for human consumption (Leroy *et al*. 2009). The expedition aimed at testing the techniques and materials to be used during a larger international expedition planned to take place early the following year. Forty-four specimens of different bat species were sampled, including those which are strongly implicated as potential reservoirs of filoviruses (e.g. *H. monstrosus)*. Blood and tissues from various organs were collected and brought to the NICD or NHLS for laboratory testing in the recently commissioned maximum security laboratory (BSL-4), which had undergone extensive and protracted upgrading and refurbishment. This maximum security laboratory constitutes a strategically important research resource for preparedness training and response to outbreaks of dangerous pathogens, but will also greatly support efforts by African scientists to unravel the elusive nature of filovirus transmission cycles (Figure 3). Experimental inoculation studies have recently been conducted at this facility with filoviruses and Egyptian fruit bats.

Despite the finding of Ebola virus nucleic acid, antigen or antibodies in bats, EBOV has never been isolated from them or any putative animal reservoir so far**.** However, the isolation of replicative Marburg virus, another member of the filovirus family, from wild fruit bats (Towner *et al*. 2009), reinforces the assumption that bats are strong EBOV reservoir candidates. Based on this hypothesis, a model of virus dissemination has been proposed: bats can transmit the virus either directly to humans and NHPs or through an unknown vector as illustrated in Figure 4.

Clinical features

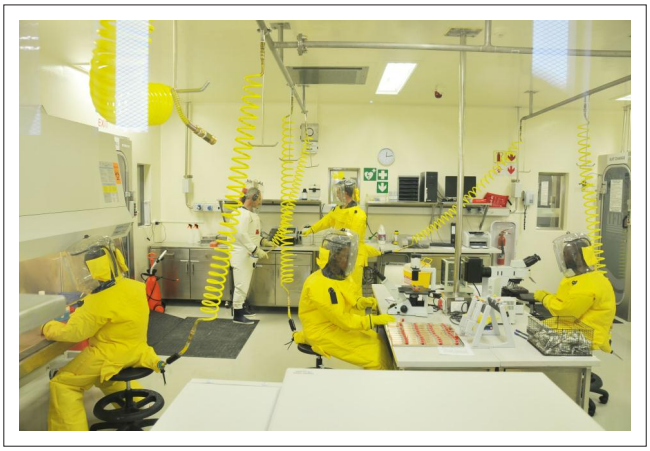
The onset of the disease is abrupt after an incubation period of two to 21 days. The clinical features can be divided into four main phases as follows, (1) Phase A. Influenza–like syndrome: The onset is abrupt with non-specific symptoms or signs such as high fever, headache, arthralgia, myalgia, sore throat, and malaise with nausea. (2) Phase B. Acute (day

Source: Photograph provided by authors
The expedition was supported by the World Health Organisation (WHO), the Southern African Centre for Infectious Disease Surveillance (SACIDS), the Institut National de Recherche Biomedicale (INRB, DRC), and the National Institute for Communicable Diseases of the National Health Laboratory Service (NICD/NHLS, RSA).
From left to right: Dr Justin Masumu (SACIDS), Dr Petrus Jansen van Vuren (NICD/NHLS), Prof. Janusz Paweska (NICD/NHLS), Mr David Francis (MAF pilot), Dr Jean-Luc Biampata (CPC Luebo hospital, host), Prof. Jean-Jacques Muyembe (INRB), Mr Kamal Aitikhlef (WHO) and Mr Alan Kemp (NICD/NHLS).

FIGURE 1: The Ebola ecology expedition team arriving in Luebo, Democratic Republic of the Congo, May 2011.

Source: Photograph provided by authors

FIGURE 2: Dissection of wild-caught bats. Field laboratory deployed during Ebola ecology study in Luebo, Democratic Republic of the Congo, May 2011.



Source: Photograph provided by authors

FIGURE 3: Processing and testing of blood and tissues from bats collected during the 2011 Ebola ecology expedition to Luebo, Democratic Republic of the Congo in biosafety level four facility in Sandringham-Johannesburg, South Africa.

1–6): Persistent fever not responding to antimalaria drugs or to antibiotics, headache, intense fatigue, followed by diarrhea and abdominal pain, anorexia and vomiting. (3) Phase C. Pseudo-remission (day 7–8): During this phase the patient feels better and seeks food. The health situation presents with some improvement. Some patients may recover during this phase and survive from the disease and (4) Phase D. Aggravation (day 9): In many if not most cases, the health status gets worse. The following symptoms may be observed:

- respiratory disorders: dyspnea, throat and chest pain, cough, hiccups
- symptoms of haemorrhagic diathesis: bloody diarrhoea, haematemesis, conjunctival injection, gingival bleeding, nosebleeds and bleeding at the site of injection consistent with disseminated intravascular coagulation (Figure 5)
- skin manifestations: petaechiae (not so obvious on black skin), purpura (morbiliform skin rash)
- neuro-psychiatric manifestations: prostration, delirium, confusion, coma
- cardio-vascular distress and hypovolaemic shock (death).

From these clinical manifestations it is obvious that EHF may mimic many other tropical diseases like malaria, typhoid fever or yellow fever at the start of the disease. In most outbreaks, recognition of the disease is delayed because physicians are not accustomed to this new illness and the symptoms are generally non-specific. Outside the epidemic context, it appears quite impossible to recognise the first Ebola case in an outbreak on clinical grounds only. Suspicion of EHF is only possible later during the aggravation phase.

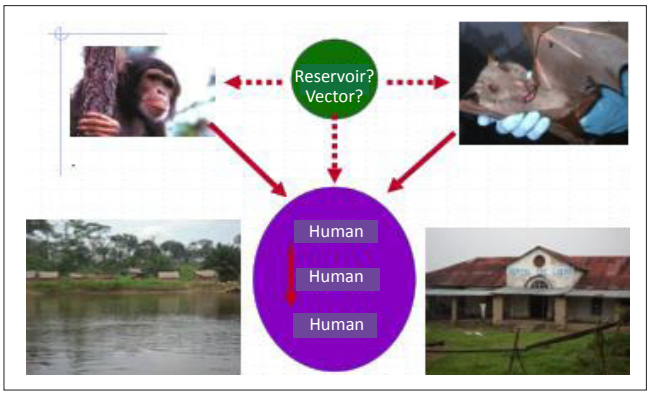
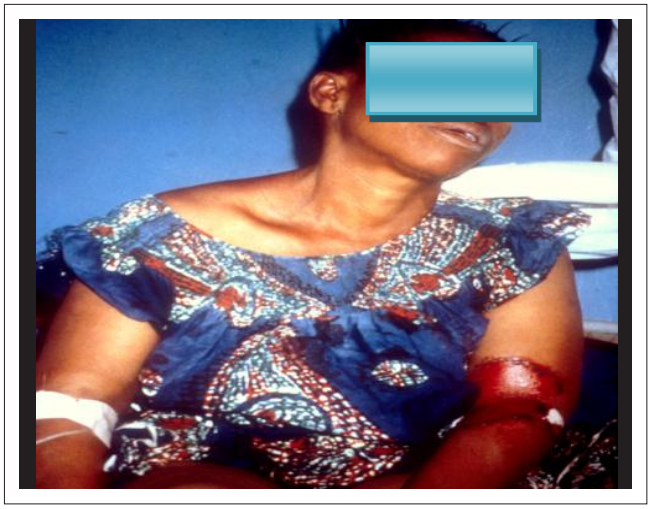


FIGURE 4: The potential chains of transmission of Ebola virus may be described as involving 3 stages, from primates or bats to humans (especially hunters) in the wild (index case), from index case to secondary cases (introduction into the domestic environment) and from patient to healthcare personnel in the clinical setting. Whilst primates and fruit bats are known to be sources of Ebola virus in nature, the reservoir has not yet been identified with any certainty.



Source: Photograph provided by authors

FIGURE 5: Ebola patient with haemorrhagic diathesis at transfusion and injection sites.

Diagnosis

Early laboratory confirmation of suspected clinical haemorrhagic fever cases is essential to implement appropriate control measures. Definitive diagnosis of suspected cases of EHF is usually made by PCR detection and virus isolation on Vero cells. As a class-4 pathogen, Ebola virus culture requires a maximum containment facility.

Additional laboratory diagnostic tests include ELISAs for the detection of Ebola IgG- and IgM-specific antibodies and virus antigens; more specialised molecular testing is also available but is not readily available in the usual clinical setting.

In Africa, laboratory confirmation of Ebola cases has been challenging and early recognition of the first outbreaks were severely hampered as a result. Because the disease was poorly known or rare, laboratory investigations were oriented towards the more common, endemic pathogens in the area.

Initially, *Salmonella typhi* and yellow fever virus were suspected to be responsible for the 1976 Yambuku outbreak. Blood samples collected for cultures (that remained negative), Widal tests and liver specimens presented for pathological examination showed inconsistent results: some specimens gave evidence of yellow fever whilst others were compatible with liver congestion. The Yambuku outbreak was finally confirmed by viral culture at the Institute of Tropical Medicine in Antwerp thanks to blood samples collected from a Belgian nun who fell ill in Yambuku and was transferred to Ngaliema hospital in Kinshasa. For the Kikwit cases in 1995, a nosocomial bloody diarrhea outbreak caused by *Shigella dysenteriae* or *S. typhi* was initially considered, as these diseases were endemic to Kikwit. Preliminary bacteriological investigations were conducted on 97 stool samples and nine blood cultures. Only four stool specimens tested positive for *Shigella* species and all blood cultures remained negative. The index case of this nosocomial outbreak was a 36-year-old laboratory technician who underwent a laparotomy in the Kikwit general referral hospital for suspected perforated bowel after a protracted febrile syndrome with headache, myalgia and asthaenia. Contact with his body fluids appeared to be the main mode of transmission for the subsequent secondary cases, suggesting that the disease was a viral haemorrhagic fever. To confirm this hypothesis, 14 blood specimens from acutely ill persons were collected and sent to the Institute of Tropical Medicine in Antwerp, Belgium. The evidence of acute Ebola infection was obtained in all specimens either by viral isolation, antigen-detection ELISA or RT-PCR.

Since 1994, the incidence of Ebola outbreaks increased and, as a consequence, the awareness of the disease has improved and facilities capable of diagnosing EHV were established in Africa. National Public Health laboratories in endemic countries like Uganda (UVRI), Kenya (KEMRI) and Gabon

(CIRMF) have already developed capacities to diagnose EHF by ELISA and RT-PCR. South Africa is the only African country with a maximum containment, enclosed suit laboratory where all class-4 viral pathogens can be handled safely.

After the last Ebola outbreak in Kaluamba, DRC (2008–2009), the Ebola diagnostic technologies of ELISAs for the detection of antigens and IgM antibody, and RT-PCR have been transferred to the INRB in Kinshasa.

Treatment

Managing Ebola patients in the African setting was a major challenge because there was no effective antiviral drug and no specific vaccine available. Only supportive care could be administered, to sustain cardiac and renal functions with prudent use of perfusion. Oral rehydration was recommended but sometimes not realistic because of throat pain, vomiting and intense fatigue. The main objective was to provide optimal care to the patient with maximum protection of the medical and nursing staff. For that purpose, medical and nursing staff had been trained in donning and removing personal protective equipment (PPE) and applying barrier–nursing procedures.

In a clinical experiment conducted late in the 1995 Ebola outbreak in Kikwit, human convalescent blood was used for passive immunisation to treat patients that had been infected naturally with ZEBOV; seven out of eight patients who received blood transfusion from convalescent Ebola patients survived (Mupapa *et al*. 1999). Such experiments, unfortunately, have not been repeated in further outbreaks because *in vitro* studies showed that antibodies against Ebola had no neutralising activities. In addition, although monoclonal antibodies to the glycoprotein of Ebola virus showed protective and therapeutic properties in mice, they failed to protect NHP (Gupta *et al*. 2001; Oswald *et al*. 2007).

Since Ebola virus is generally considered as a potential biological weapon, it is urgent to develop effective antiviral drugs and vaccines. The ideal is to develop a candidate vaccine able to confer interspecies cross-protection against ZEBOV, SEBOV, BEBOV and unknown Ebola virus species.

Control measures

The corner-stone for controlling an outbreak of EHF is to interrupt the viral transmission chain. In order to reduce transmission, several strict public health measures need to be implemented as quickly as possible, including isolation of patients, barrier precautions and identification and tracking of all contacts. Most of the time, outbreaks are managed by a core structure called the International Committee on Scientific and Technical Coordination, under the aegis of the World Health Organisation (WHO). This committee is in charge of implementing control measure activities on a daily basis and has the following working subgroups:

- Co-ordination committee, which is responsible for all epidemic response activities, chair daily meetings and write reports for public health authorities and health partners.
- The patient management team is involved in the isolation of clinical cases in a quarantine ward, training of medical and relief personnel on the proper use of protective equipment (gloves, gowns, masks etc.), and providing medical care based on symptomatic therapy to maintain the vital respiratory, cardio-vascular and renal functions. The non-governmental organisation, Doctors Without Borders (MSF), has developed expertise in this field from involvement in outbreak response.
- The hygiene and sanitation team is in charge of disinfection and burial of all Ebola and non-Ebola dead bodies under safe conditions. Local Red Cross volunteers usually perform these activities.
- The epidemiological surveillance team is in charge of active and passive case finding, contact tracing and rumor-verification of suspect cases or deaths in the community.
- Social mobilisation and health education are critical for controlling an Ebola outbreak since resistance from the community to freely provide information on patients, deaths and contacts are commonplace. Ebola haemorrhagic fever outbreaks have many socio-cultural aspects that need to be studied deeply as communities can reject the anti-epidemic control measures imposed by the international scientific and technical committee. The existence of rumours and legends related to the outbreaks could obscure the viral nature of the disease. Sometimes the anti-epidemic control measures needed to be adapted to the local culture, for example, funeral practices as in the 2003 Ebola outbreak in Republic of the Congo (Hewlett *et al*. 2005). The members of this team should include medical anthropologists, local Red Cross volunteers and opinion leaders such as teachers, religious groups, et cetera, for public sensitisation, education and information.
- The logistic support team is in charge of providing any administrative, logistic and technical support to the other teams, such as coordination of secretariat, transport and communication.
- The laboratory and research team is in charge of collecting, storing and shipping of clinical samples for diagnostic confirmation. This team is also responsible for ecological studies to determine the origins of an outbreak.
- Psychosocial support for the affected family or families has been neglected during previous outbreaks, but this issue has become more and more important due to stigmatisation of survivors and their families by the community.

Conclusion

Formerly sporadic, with high case fatality rates (up to 90%), the deadly Ebola haemorrhagic fever outbreaks are becoming more and more frequent in Africa, mostly in relation to increasing contact with infected wildlife. Previous epidemics were detected after a long delay, especially because of the remoteness of the epidemic focus, the lack of laboratory facilities and the poor knowledge of the disease by doctors and nurses, who confused Ebola disease with malaria or typhoid fever.

The more recent epidemics in Yambio (2004) and Kaluamba (2008) resulted in low CFRs of 41.2% and 43.8%, respectively.

This is mainly related to the early detection of the outbreaks followed by a prompt and vigorous response from public health authorities and their partners.

Ebola haemorrhagic fever epidemics constitute a significant public health concern in Africa and an effective vaccine is needed urgently. Such a vaccine would primarily benefit doctors, nurses and field epidemiologists working in endemic countries. The second target group would be the scientists working with Ebola virus as well as veterinarians and those involved in wildlife conservation in endemic areas. Since its discovery in 1976, much is known about Ebola virology, physiopathology, clinical features and epidemiology, but the missing link certainly remains the virus reservoir in nature. The current research focused on bats as putative ZEBOV reservoirs has to be reinforced and extended to the reservoirs of other Ebola species.

References

Amblard, J., Obiang, P., Edzang, S., Prehaud, C., Bouloy, M. & Le Guenno, B., 1997, 'Identification of the Ebola virus in Gabon in 1994', *Lancet* 349, 181–182. http://dx.doi.org/10.1016/S0140-6736(05)60984-1

Baron, R.C., McCormick, J.B. & Zubeir, O.A., 1983, 'Ebola virus disease in southern Sudan: hospital dissemination and intrafamilial spread', *Bulletin of the World Health Organization* 61, 997–1003. PMid:6370486

Barrette, R.W., Metwally, S.A., Rowland, J.M., Xu, L., Zaki, S.R., Nichol, S.T. *et al*., 2009, 'Discovery of swine as a host for the Reston ebolavirus', *Science* 325, 204–206. http://dx.doi.org/10.1126/science.1172705, PMid:19590002

Bausch, D.G., Towner, J.S., Dowell, S.F., Kaducu, F., Lukwiya, M., Sanchez, A. *et al*., 2007, 'Assessment of the risk of Ebola virus transmission from bodily fluids and fomites', *Journal of Infectious Diseases* 196 (Supplement 2), S142–S147. http://dx.doi.org/10.1086/520545, PMid:17940942

Becquart, P., Wauquier, N., Mahlakoiv, T., Nkoghe, D., Padilla, C., Souris, M. *et al*., 2010, 'High prevalence of both humoral and cellular immunity to Zaire ebolavirus among rural populations in Gabon', *PLoS One* 5, e9126. http://dx.doi.org/10.1371/journal.pone.0009126, PMid:20161740

Busico, K.M., Marshall, K.L., Ksiazek, T.G., Roels, T.H., Fleerackers, Y., Feldmann, H. *et al*., 1999, 'Prevalence of IgG antibodies to Ebola virus in individuals during an Ebola outbreak, Democratic Republic of the Congo, 1995', *Journal of Infectious Diseases* 179 (Supplement 1), S102–S107. http://dx.doi.org/10.1086/514309, PMid:9988172

Feldmann, H. & Geisbert, T.W., 2011, 'Ebola haemorrhagic fever', *Lancet* 377, 849–862. http://dx.doi.org/10.1016/S0140-6736(10)60667-8

Feldmann, H., Geisbert, T.W., Jahrling, P.B., Klenk, H.D., Netesov, S.V., Peters, C.J. *et al*., 2005, 'Filoviridae', in C. Fauquet, M.A. Mayo, J. Maniloff, U. Desselberger & L.A. Ball (eds.), *Virus Taxonomy: VIIIth Report of the International Committee on Taxonomy of Viruses*, pp. 645–653, Elsevier/Academic Press, London.

Formenty, P., Leroy, E.M., Epelboin, A., Libama, F., Lenzi, M., Sudeck, H. *et al*., 2006, 'Detection of Ebola virus in oral fluid specimens during outbreaks of Ebola virus hemorrhagic fever in the Republic of Congo', *Clinical Infectious Diseases* 42, 1521–1526. http://dx.doi.org/10.1086/503836, PMid:16652308

Georges-Courbot, M.C., Lu, C.Y., Lansoud-Soukate, J., Leroy, E. & Baize, S., 1997a, 'Isolation and partial molecular characterisation of a strain of Ebola virus during a recent epidemic of viral haemorrhagic fever in Gabon', *Lancet* 349, 181. http://dx.doi.org/10.1016/S0140-6736(05)60983-X

Georges-Courbot, M.C., Sanchez, A., Lu, C.Y., Baize, S., Leroy, E., Lansout-Soukate, J. *et al*., 1997b, 'Isolation and phylogenetic characterization of Ebola viruses causing different outbreaks in Gabon', *Emerging Infectious Diseases* 3, 59–62. http://dx.doi.org/10.3201/eid0301.970107, PMid:9126445

Gonzalez, J.P., Nakouné, E., Slenczka, W., Vidal, P. & Morvan, J.M., 2000, 'Ebola and Marburg virus antibody prevalence in selected populations of the Central African Republic', *Microbes and Infection* 2, 39–44. http://dx.doi.org/10.1016/S1286-4579(00)00287-2

Groseth, A., Feldmann, H. & Strong, J.E., 2007, 'The ecology of Ebola virus', *Trends in Microbiology* 15, 408–416. http://dx.doi.org/10.1016/j.tim.2007.08.001, PMid:17698361

Guimard, Y., Bwaka, M.A., Colebunders, R., Calain, P., Massamba, M., De Roo, A. *et al*., 1999, 'Organization of patient care during the Ebola hemorrhagic fever epidemic in Kikwit, Democratic Republic of the Congo, 1995', *Journal of Infectious Diseases* 179 (Supplement 1), S268–S273. http://dx.doi.org/10.1086/514315, PMid:9988194

Gupta, M., Mahanty, S., Bray, M., Ahmed, R. & Rollin, P.E., 2001, 'Passive transfer of antibodies protects immunocompetent and imunodeficient mice against lethal Ebola virus infection without complete inhibition of viral replication', *Journal of Virology* 75, 4649–4654. http://dx.doi.org/10.1128/JVI.75.10.4649-4654.2001, PMid:11312335

Hewlett, B.S., Epelboin, A., Hewlett, B.L. & Formenty, P., 2005, 'Medical anthropology and Ebola in Congo: cultural models and humanistic care', *Bulletin de la Société de Pathologie Exotique* 98, 230–236. PMid:16267966

Heymann, D.L., Weisfeld, J.S., Webb, P.A., Johnson, K.M., Cairns, T. & Berquist, H., 1980, 'Ebola hemorrhagic fever: Tandala, Zaire, 1977–1978', *Journal of Infectious Diseases* 142, 372–376. http://dx.doi.org/10.1093/infdis/142.3.372, PMid:7441008

Khan, A.S., Tshioko, F.K., Heymann, D.L., Le Guenno, B., Nabeth, P., Kerstiens, B. *et al*., 1999, 'The re-emergence of Ebola hemorrhagic fever, Democratic Republic of the Congo, 1995', *Journal of Infectious Diseases* 179 (Supplement 1), S76–S86. http://dx.doi.org/10.1086/514306, PMid:9988168

Lamunu, M., Lutwama, J.J., Kamugisha, J., Opia, A., Nambooze, J., Ndayimirije, N. *et al*., 2004, 'Containing a haemorrhagic fever epidemic: The Ebola experience in Uganda (October 2000 – January 2001)', *International Journal of Infectious Diseases* 8, 27–37. http://dx.doi.org/10.1016/j.ijid.2003.04.001, PMid:14690778

Leffel, E.K. & Reed, D.S., 2004, 'Marburg and Ebola viruses as aerosol threats', *Biosecurity and Bioterrorism: Biodefense Strategy, Practice, and Science* 2, 186–191.

Le Guenno, B., Formenty, P., Wyers, M., Gounon, P., Walker, F. & Boesch, C., 1995, 'Isolation and partial characterisation of a new strain of Ebola virus', *Lancet* 345, 1271–1274. http://dx.doi.org/10.1016/S0140-6736(95)90925-7

Leroy, E.M., Epelboin, A., Mondonge, V., Pourrut, X., Gonzalez, J.P., Muyembe-Tamfum, J.J. *et al*., 2009, 'Human Ebola outbreak resulting from direct exposure to fruit bats in Luebo, Democratic Republic of Congo, 2007', *Vector-borne and Zoonotic Diseases* 9, 723–728. http://dx.doi.org/10.1089/vbz.2008.0167, PMid:19323614

Leroy, E.M., Gonzalez, J.P. & Baize, S., 2011, 'Ebola and Marburg haemorrhagic fever viruses: major scientific advances, but a relatively minor public health threat for Africa', *Clinical Microbiology and Infection* 17, 964–976. http://dx.doi.org/10.1111/j.1469-0691.2011.03535.x, PMid:21722250

Leroy, E.M., Kumulungui, B., Pourrut, X., Rouquet, P., Hassanin, A., Yaba, P. *et al*., 2005, 'Fruit bats as reservoirs of Ebola virus', *Nature* 438, 575–576. http://dx.doi.org/10.1038/438575a, PMid:16319873

Leroy, E.M., Rouquet, P., Formenty, P., Souquière, S., Kilbourne, A., Froment, J.M. *et al*., 2004, 'Multiple Ebola virus transmission events and rapid decline of central African wildlife', *Science* 303, 387–390. http://dx.doi.org/10.1126/science.1092528, PMid:14726594

Leroy, E.M., Souquiere, S., Rouquet, P. & Drevet, D., 2002, 'Re-emergence of ebola haemorrhagic fever in Gabon', *Lancet* 359, 712. http://dx.doi.org/10.1016/S0140-6736(02)07796-6

MacNeil, A., Reed, Z. & Rollin, P.E., 2011, 'Serologic cross-reactivity of human IgM and IgG antibodies to five species of Ebola virus', *PLoS Neglected Tropical Diseases* 5, e1175. http://dx.doi.org/10.1371/journal.pntd.0001175, PMid:21666792

Mupapa, K., Massamba, M., Kibadi, K., Kuvula, K., Bwaka, A., Kipasa, M. *et al*., 1999, 'Treatment of Ebola hemorrhagic fever with blood transfusions from convalescent patients', *Journal of Infectious Diseases* 179 (Supplement 1), S18–S23. http://dx.doi.org/10.1086/514298, PMid:9988160

Nkoghe, D., Kone, M.L., Yada, A. & Leroy, E., 2011, 'A limited outbreak of Ebola haemorrhagic fever in Etoumbi, Republic of Congo, 2005', *Transactions of the Royal Society of Tropical Medicine and Hygiene* 105, 466–472. http://dx.doi.org/10.1016/j.trstmh.2011.04.011, PMid:21605882

Onyango, C.O., Opoka, M.L., Ksiazek, T.G., Formenty, P., Ahmed, A., Tujkei, P.M. *et al*., 2007, 'Laboratory diagnosis of Ebola hemorrhagic fever during an outbreak in Yambio, Sudan, 2004', *Journal of Infectious Diseases* 196 (Supplement 2), S193–S198. http://dx.doi.org/10.1086/520609, PMid:17940949

Oswald, W.B., Geisbert, T.W., Davis, K.J., Geisbert, J.B., Sullivan, N.J., Jahrling, P.B. *et al*., 2007, 'Neutralizing antibody fails to impact the course of Ebola virus infection in monkeys', *PLoS Pathogens* 3, e9. http://dx.doi.org/10.1371/journal.ppat.0030009, PMid:17238286

Pourrut, X., Kumulungui, B., Wittmann, T., Moussavou, G., Délicat, A., Yaba, P. *et al*., 2005, 'The natural history of Ebola virus in Africa', *Microbes and Infection* 7, 1005–1014. http://dx.doi.org/10.1016/j.micinf.2005.04.006, PMid:16002313

Rouquet, P., Froment, J.M., Bermejo, M., Kilbourne, A., Karesh, W., Reed, P. *et al*., 2005, 'Wild animal mortality monitoring and human Ebola outbreaks, Gabon and Republic of Congo, 2001–2003', *Emerging Infectious Diseases* 11, 283–290. http://dx.doi.org/10.3201/eid1102.040533, PMid:15752448

Swanepoel, R., Leman, P.A., Burt, F.J., Zachariades, N.A., Braack, L.E.O., Ksiazek, T.G. *et al*., 1996, 'Experimental inoculation of plants and animals with Ebola virus', *Emerging Infectious Diseases* 2, 321–325. http://dx.doi.org/10.3201/eid0204.960407, PMid:8969248

Swanepoel, R., Smit, S.B., Rollin, P.E., Formenty, P., Leman, P.A., Kemp, A. *et al*., 2007, 'Studies of reservoir hosts for Marburg virus', *Emerging Infectious Diseases* 13, 1847–1851. http://dx.doi.org/10.3201/eid1312.071115, PMid:18258034

Towner, J.S., Amman, B.R., Sealy, T.K., Reeder Carrol, S.A., Comer, J.A., Kemp, A. *et al*., 2009, 'Isolation of genetically diverse Marburg viruses from Egyptian fruit bats', *PLoS Pathogens* 5, e1000536. http://dx.doi.org/10.1371/journal.ppat.1000536, PMid:19649327

Towner, J.S., Sealy, T.K., Khristova, M.L., Albariño, C.G., Conlan, S., Reeder, S.A. *et al*., 2008, 'Newly discovered ebola virus associated with hemorrhagic fever outbreak in Uganda', *PLoS Pathogens* 4, e1000212. http://dx.doi.org/10.1371/journal.ppat.1000212, PMid:19023410

Walsh, P.D., Abernethy, K.A., Bermejo, M., Beyers, R., De Wachter, P., Akou, M.E. *et al*., 2003, 'Catastrophic ape decline in western equatorial Africa', *Nature* 422, 611–614. http://dx.doi.org/10.1038/nature01566, PMid:12679788

WHO, 1978a, 'Ebola haemorrhagic fever in Sudan, 1976. Report of a WHO/International Study Team', *Bulletin of the World Health Organization* 56, 247–270. PMid:307455

WHO, 1978b, 'Ebola haemorrhagic fever in Zaire, 1976', *Bulletin of the World Health Organization* 56, 271–293. PMid:307456

WHO, 2011, 'Ebola in Uganda', *Global Alert and Response*, viewed 04 October 2011, from http://www.who.int/csr/don/2011_05_18/en/index.html

Zoonotic diseases and human health: The human influenza example

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How to cite this proceeding:
Schoub, B.D., 2012, 'Zoonotic diseases and human health: The human influenza example', *Onderstepoort Journal of Veterinary* Research 79(2), Art. #489, 4 pages. http://dx.doi.org/10.4102/ojvr.v79i2.489

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Over the past few decades a large number of new and emerging infectious diseases have been recognised in humans, partly because of improved diagnostic technologies and increased awareness and also, partly because of dynamic ecological changes between human hosts and their exposure to animals and the environment (Coker *et al*. 2011). Some 177 new pathogenic organisms have been recognised to be 'emerging', that is, have newly arisen or been newly introduced into human populations; almost three quarters of these, 130 (73%), have come from zoonotic origins (Cascio *et al.* 2011; Cutler, Fooks & Van Der Poel 2010; Taylor, Latham & Woolhouse 2001; Woolhouse & Gowtage-Sequeria 2005). One of the most prevalent and important human infectious disease is influenza, a disease responsible globally for a quarter million deaths annually. In the USA alone the toll from influenza is estimated at 36 000 deaths and 226 000 hospitalisations, and it ranks as the most important cause of vaccine preventable mortality in that country (CDC 2010). The epidemiological behaviour of human influenza clearly defines it as an emerging infectious disease and the recent understanding of its zoonotic origins has contributed much to the understanding of its behaviour in humans (Fauci 2006).

The classical view of influenza

Influenza is one of the most enigmatic of human viruses especially when one considers that it was the first human virus to be isolated (Smith, Andrews & Laidlaw 1933), that the vaccine was one of the first human vaccines to have been developed, in the mid-1940s, and that it is a virus which has been intensively studied since then in animal models, in human volunteers and since the early 1980s at an intense biophysical and biochemical level (Waterfield, Scrace & Skehel 1981). Yet this virus, despite its public health importance being responsible for causing such a massive burden of illness, is only now in recent times yielding up the secrets of its characteristics and its behaviour in human populations.

The classical view of influenza virus was that of a virus which behaved in one of two ways. Every winter human populations predictably experience seasonal influenza of varying degrees of intensity. Subtle changes in antigenicity as a result of mutational changes in the plastic ribonucleic acid (RNA) genome, termed antigenic drift, ensured that epidemics of influenza in temperate climates would be a feature of every winter. New strains of the virus therefore appeared regularly which would replace existing strains to which the population had built up immunity. On rare occasions, about two or three times per century, a more dramatic change in antigenicity, termed antigenic shift, would result from the introduction of a new gene or genes from an animal reservoir as a result of genetic reassortment of the fragmented genome of the virus. So arose the pandemics of 1918 (the Spanish influenza), 1957 (the Asian influenza) and 1968 (the Hong Kong influenza). With each pandemic a new subtype of influenza A virus would appear to replace its predecessor and it would then become the regular annual seasonal influenza virus. The first upset to this classical concept came with the reappearance of the H1N1 subtype in 1977 after an absence of over 20 years which did not replace the existing H3N2 subtype but circulated together with it and either or both of them would then be responsible for annual seasonal influenza outbreaks (Kilbourne 2006).

A prescient commentary by Scholtissek and Naylor in 1988 drew attention to the risks of future pandemics which could result from developments in aquaculture which promote contacts between humans, ducks and pigs (Scholtissek & Naylor 1988). In 1997 the first cases of human infections with H5N1 virus were reported from Hong Kong (Claas *et al*. 1998; Yuen *et al*. 1998) and subsequently in a number of publications over 50 sporadic and clustered cases of human infections with avian viruses of different subtypes resulting from direct, extended and intimate contact with human poultry were recorded (Alexander 2006; Lee & Saif 2009; Wong & Yuen 2006). The fundamental understanding of the ecology of influenza A viruses and the evolution of human viruses was now becoming apparent, namely, that the natural reservoir of influenza virus was avians, especially waterfowl (Subbarao & Katz 2000). From this reservoir new viruses or new

genes would periodically cross the species barrier and infect humans either directly or indirectly through an intermediate host such as the pig, or a novel gene or two could be transmitted through reassortment in an intermediate host such as the pig. A second important understanding was the concept that reassortment (the probable mechanism for the 1957 and 1968 pandemics) was not the only mechanism of how new human viruses arose from animal reservoirs but also direct viral transmission from an avian source to humans could play a role in the origin of human pandemics, as was thought to have occurred in the 1918 pandemic (Belshe 2005).

The influenza A (H5N1) outbreak in humans began in Hong Kong in 1997 with 18 cases, six of whom died (Claas *et al*. 1998; Yuen *et al*. 1998). The virus then apparently disappeared from human populations for five years re-emerging in 2003 in a Hong Kong family who had returned from a trip to mainland China (Peiris *et al*. 2004). Since then H5N1 disease in humans has spread to 15 countries in Asia, Europe and Africa (WHO 2011). As at 02 August 2011, 563 laboratory-confirmed cases have been reported to WHO, 330 of whom have died – an alarming case fatality of 59% (WHO 2011). The underlying clinico-pathological reasons for this extraordinarily high case fatality rate, far higher than even the 2.5% estimated case fatality of the notorious 1918 pandemic (Murray *et al*. 2006; Taubenberger & Morens 2006), is still not clear (Abdel-Ghafar *et al*. 2008; Korteweg & Gu 2008; Michaelis, Doerr & Cinatl 2009; Thanh, Van Doorn & De Jong 2008; Uyeki 2009). Fortunately very few cases of possible human-to-human transmission have been reported (Kandun *et al*. 2005; Ungchusak *et al*. 2005). Whether the virus will jump the species barrier and established itself in humans is a public health question of prime concern. A necessary, but not sufficient, first step would need to be a receptor switch from a haemagglutinin with a specificity for the α-2,3-galactose saccharide terminal prevalent in the gastrointestinal tract of birds to the α-2,6-galactose saccharide terminal prevalent in the upper respiratory tract of mammals including humans (Gabriel, Herwig & Klenk 2008; Ge & Wang 2011; Hatta *et al*. 2007; Uiprasertkul *et al*. 2005). Given the extremely high case fatality of these sporadic H5N1 cases, a pandemic caused by this virus would indeed be a most formidable prospect. Mortality rates rivaling, or even exceeding, those of the 1918/1919 pandemic are feared – it has been extrapolated from 1918 data that up to 62 million excess deaths could be expected (Murray *et al*. 2006). Fortunately the receptor switch and sustained crossing of the species barrier has not occurred in the 14 years since the first cases were observed in humans. The one positive outcome of the H5N1 alarm has been the mobilisation of energies to effectively prepare for a future inevitable pandemic (Breiman *et al*. 2007; Flahault *et al*. 2006; Uscher-Pines *et al*. 2006; Webby & Webster 2003). This pandemic preparedness planning did stand in good stead when the next unexpected pandemic arrived early in 2009.

Swine influenza H1N1

The swine influenza pandemic of 2009 took all by surprise. Its advent was not in south-east Asia as was usual for pandemics and from where the next pandemic was anticipated, but this time it arose in North America. The first intimation of an impending pandemic was an unusually high rate of hospitalisations of acute respiratory disease in Mexico in late March 2009 and the following month in California. These early observations alerted public health officials to infections with a novel H1N1 influenza A virus of swine origin (Chowell *et al*. 2011). Within several months the virus had rapidly spread throughout the world and on 11 June 2009 the World Health Organisation (WHO) raised the pandemic alert to level 6, that is, the formal declaration of a pandemic – the first influenza pandemic since 1968 (WHO 2009). The 2009 pandemic was also a first from a number of other respects (Leung & Nicoll 2010). Amongst the 'firsts', the 2009 pandemic was the first pandemic where intensive care facilities were available and this provided some intimation as to the severity of this pandemic. The mortality was in fact significantly less than preceding pandemics in most of the world and in many parts even less than the immediately preceding seasonal influenza epidemic. However, what was reported from many countries around the world was an unusually high rate of hospitalisation amongst young patients, many of whom were ostensibly healthy, as well as a heavy load on intensive care facilities (Lipsitch *et al*. 2009). The relative mildness expressed in terms of mortality rates, led to concerns being expressed, such as by the Council of Europe, as to whether the WHO had not been overhasty in declaring a pandemic (Social Health and Family Affairs Committee of the Parliamentary Assembly of the Council of Europe 2010). This also gave rise to a debate in the literature as to what exactly constitutes a pandemic (Doshi 2011; Morens, Folkers & Fauci 2009). If the criterion of rapid extra-seasonal spread and heavy involvement of young individuals who often appear to be otherwise healthy, are important elements defining pandemics, then certainly the 2009 pandemic fits into the pandemic definition. Another 'first' was that, for the first time, it was a pandemic not due to the advent of a new subtype of influenza but rather that of an existing seasonal subtype, H1N1, which had become antigenically distant enough from the pre-existing seasonal H1N1 influenza virus so as to behave like a characteristically pandemic virus, especially with respect to the rapidity of its spread worldwide through a largely non-immune population. How the new pandemic virus arose is a story of multiple reassortment events with frequent crossings of the species barrier between humans and animals, especially pigs. The new reassortants went largely undetected and their effects were largely unnoticed because of inadequate surveillance and awareness (Garten *et al*. 2009; Ilyushina *et al*. 2010; Trifonov, Khiabanian & Rabadan 2009). Evidence points to the introduction of influenza A (H1N1) into the pig population at about the same time as the 1918 pandemic in humans and it was then also responsible for widespread disease in pig herds (Zimmer & Burke 2009). An isolated outbreak of swine influenza A (HswN1) in humans in 1976 amongst military personnel in Fort Dix, New Jersey, resulted in 13 cases of severe respiratory disease and one death, but the infection did not spread further (Gaydos *et al*.

2006). A small outbreak of 12 cases of human infection due to a triple reassortant swine influenza virus with genes from swine, avian and human viral sources was detected in the USA between 2005 and 2009 (Dawood *et al.* 2009). The 2009 pandemic virus now designated as A/California/04/2009 pandemic (H1N1) probably arose from a subsequent reassortment event of this North American reassortant with a Eurasian lineage swine virus (Garten *et al*. 2009; Ilyushina *et al*. 2010; Trifonov *et al*. 2009). Antigenically this new virus had by now achieved a great antigenic distance from the human seasonal influenza A (H1N1) virus circulating at the time resulting in little cross-immunity between them. However the relative sparing of older persons in the 2009 pandemic was largely due to some degree from pre-existing immunity as a result of the structural and antigenic similarity between the haemagglutinin molecule of the pandemic virus and the H1N1 viruses which had circulated earlier in the 20th century and to which older persons had been exposed to (Xu *et al*. 2010). Direct transmission from humans to pigs has also been demonstrated (Pereda *et al*. 2010) and reassortment in pigs, which are susceptible to both human and avian viruses (often referred to as the the 'mixing vessel') pose an ongoing risk to human health. This again emphasises the crucial importance for public health of ongoing robust influenza surveillance of animals such as pigs (Mitka 2010; Vijaykrishna *et al*. 2010).

Conclusion

Our understanding of the dynamics of influenza epidemics and pandemics has improved by leaps and bounds since the avian and swine influenza events of the last decade. An essential component of preparedness for future pandemics by public health bodies must now involve a much greater focus on surveillance of animals and birds to avoid being caught out again by surprises such as occurred in 2009 with the swine flu pandemic. The concept of a one health framework to unify the sciences of human, veterinary and ecological health has come none too soon to deal with future emerging infectious diseases in humans (Coker *et al.* 2011; Kuehn 2010; Mazet *et al.* 2009). The importance of the one health perspective has been graphically illustrated by one of the most important of the emerging diseases in humans, influenza (Pappaioanou & Gramer 2010; Powdrill, Nipp & Rinderknecht 2010).

References

Abdel-Ghafar, A.N., Chotpitay Asunondh, T., Gao, Z., Hayden, F.G., Nguyen, D.H., De Jong, M.D. *et al*., 2008, 'Update on avian influenza A (H5N1) virus infection in humans', *New England Journal of Medicine* 358, 261–273. http://dx.doi.org/10.1056/NEJMra0707279, PMid:18199865

Alexander, D.J., 2006, 'Avian influenza viruses and human health', *Developments in Biologicals* (*Basel*) 124, 77–84.

Belshe, R.B., 2005, 'The origins of pandemic influenza-lessons from the 1918 virus', *New England Journal of Medicine* 353, 2209–2211. http://dx.doi.org/10.1056/NEJMp058281, PMid:16306515

Breiman, R.F., Nasidi, A., Katz, M.A., Kariuki Njenga, M. & Vertefeuille, J., 2007, 'Preparedness for highly pathogenic avian influenza pandemic in Africa', *Emerging Infectious Diseases* 13, 1453–1458. http://dx.doi.org/10.3201/eid1310.070400, PMid:18257986

Cascio, A., Bosilkovski, M., Rodriguez-Morales, A.J. & Pappas, G., 2011, 'The socio-ecology of zoonotic infections', *Clinical Microbiology and Infection* 17, 336–342. http://dx.doi.org/10.1111/j.1469-0691.2010.03451.x, PMid:21175957

CDC, 2010, 'Prevention and Control of Influenza with Vaccines', *Morbidity and Mortality Weekly Report* 59, 1–62. PMid:20075837

Chowell, G., Echevarria-Zuno, S., Viboud, C., Simonsen, L., Tamerius, J., Miller, M.A. *et al*., 2011, 'Characterizing the epidemiology of the 2009 influenza A/H1N1 pandemic in Mexico', *PLoS Medicine* 8, e1000436. http://dx.doi.org/10.1371/journal.pmed.1000436, PMid:21629683

Claas, E.C., Osterhaus, A.D., Van Beek, R., De Jong, J.C., Rimmelzwaan, G.F., Senne, D.A. *et al*., 1998, 'Human influenza A H5N1 virus related to a highly pathogenic avian influenza virus', *Lancet* 351, 472–477. http://dx.doi.org/10.1016/S0140-6736(97)11212-0

Coker, R., Rushton, J., Mounier-Jack, S., Karimuribo, E., Lutumba, P., Kambarage, D. *et al*., 2011, 'Towards a conceptual framework to support one-health research for policy on emerging zoonoses', *Lancet Infectious Diseases* 11, 326–331. http://dx.doi.org/10.1016/S1473-3099(10)70312-1

Cutler, S.J., Fooks, A.R. & Van der Poel, W.H., 2010, 'Public health threat of new, reemerging, and neglected zoonoses in the industrialized world', *Emerging Infectious Diseases* 16, 1–7. http://dx.doi.org/10.3201/eid1601.081467

Dawood, F.S., Jain, S., Finelli, L., Shaw, M.W., Lindstrom, S., Garten, R.J. *et al*., 2009, 'Emergence of a novel swine-origin influenza A (H1N1) virus in humans', *New England Journal of Medicine* 360, 2605–2615. http://dx.doi.org/10.1056/NEJMoa0903810, PMid:19423869

Doshi, P., 2011, 'The elusive definition of pandemic influenza', *Bulletin of the World Health Organization* 89, 532–538. http://dx.doi.org/10.2471/BLT.11.086173, PMid:21734768

Fauci, A.S., 2006, 'Emerging and re-emerging infectious diseases: Influenza as a prototype of the host-pathogen balancing act', *Cell* 124, 665–670. http://dx.doi.org/10.1016/j.cell.2006.02.010, PMid:16497575

Flahault, A., Vergu, E., Coudeville, L. & Grais, R.F., 2006, 'Strategies for containing a global influenza pandemic', *Vaccine* 24, 6751–6755. http://dx.doi.org/10.1016/j.vaccine.2006.05.079, PMid:16843574

Gabriel, G., Herwig, A. & Klenk, H.D., 2008, 'Interaction of polymerase subunit PB2 and NP with importin alpha1 is a determinant of host range of influenza A virus', *PLoS Pathogens* 4, e11. http://dx.doi.org/10.1371/journal.ppat.0040011, PMid:18248089

Garten, R.J., Davis, C.T., Russell, C.A., Shu, B., Lindstrom, S., Balish, A. *et al*., 2009, 'Antigenic and genetic characteristics of swine-origin 2009 A (H1N1) influenza viruses circulating in humans', *Science* 325, 197–201. http://dx.doi.org/10.1126/science.1176225, PMid:19465683

Gaydos, J.C., Top, F.H., Hodder, R.A. & Russell, P.K., 2006, 'Swine influenza a outbreak, Fort Dix, New Jersey, 1976', *Emerging Infectious Diseases* 12, 23–28. http://dx.doi.org/10.3201/eid1201.050965, PMid:16494712

Ge, S. & Wang, Z., 2011, 'An overview of influenza A virus receptors', *Critical Reviews in Microbiology* 37, 157–165. http://dx.doi.org/10.3109/1040841X.2010.536523, PMid:21438845

Hatta, M., Hatta, Y., Kim, J.H., Watanabe, S., Shinya, K., Nguyen, T. *et al*., 2007, 'Growth of H5N1 influenza A viruses in the upper respiratory tracts of mice', *PLoS Pathogens* 3, 1374–1379. http://dx.doi.org/10.1371/journal.ppat.0030133, PMid:17922570

Ilyushina, N.A., Kim, J.K., Negovetich, N.J., Choi, Y.K., Lang, V., Bovin, N.V. *et al*., 2010, 'Extensive mammalian ancestry of pandemic (H1N1) 2009 virus', *Emerging Infectious Diseases* 16, 314–317. PMid:20113569

Kandun, I.N., Wibisono, H., Sedyaningsih, E.R., Yusharmen Hadisoedarsuno, W., Purba, W., Santoso, H. *et al*., 2006, 'Three Indonesian clusters of H5N1 virus infection in 2005', *New England Journal of Medicine* 355, 2186–2194. http://dx.doi.org/10.1056/NEJMoa060930, PMid:17124016

Kilbourne, E.D., 2006, 'Influenza pandemics of the 20th century', *Emerging Infectious Diseases* 12, 9–14. http://dx.doi.org/10.3201/eid1201.051254, PMid:16494710

Korteweg, C. & Gu, J., 2008, 'Pathology, molecular biology, and pathogenesis of avian influenza A (H5N1) infection in humans', *American Journal of Pathology* 172, 1155–1170. http://dx.doi.org/10.2353/ajpath.2008.070791, PMid:18403604

Kuehn, B.M., 2010, 'Human, animal, ecosystem health all key to curbing emerging infectious diseases', *Journal of the American Medical Association* 303, 117–118, 124. http://dx.doi.org/10.1001/jama.2009.1896, PMid:20068200

Lee, C.W. & Saif, Y.M., 2009, 'Avian influenza virus', *Comparative Immunoloyg, Microbiology and Disease* 32, 301–310.

Leung, G.M. & Nicoll, A., 2010, 'Reflections on pandemic (H1N1) 2009 and the international response', *PLoS Medicine* 7.

Lipsitch, M., Riley, S., Cauchemez, S., Ghani, A.C. & Ferguson, N.M., 2009, 'Managing and reducing uncertainty in an emerging influenza pandemic', *New England Journal of Medicine* 361, 112–115. http://dx.doi.org/10.1056/NEJMp0904380, PMid:19474417

Mazet, J.A., Clifford, D.L., Coppolillo, P.B., Deolalikar, A.B., Erickson, J.D. & Kazwala, R.R., 2009, 'A "one health" approach to address emerging zoonoses: The HALI project in Tanzania', *PLoS Medicine* 6, e1000190. http://dx.doi.org/10.1371/journal.pmed.1000190, PMid:20016689

Michaelis, M., Doerr, H.W. & Cinatl, J. (Jnr.), 2009, 'Of chickens and men: Avian influenza in humans', *Current Molecular Medicine* 9, 131–151. http://dx.doi.org/10.2174/156652409787581565, PMid:19275623

Mitka, M., 2010, 'H1N1 influenza virus reassorting in pigs, poses unknown risk to human health', *Journal of the American Medical Association* 304, 626–627. http://dx.doi.org/10.1001/jama.2010.1056, PMid:20699449

Morens, D.M., Folkers, G.K. & Fauci, A.S., 2009, 'What is a pandemic?', *Journal of Infectious Diseases* 200, 1018–1021. http://dx.doi.org/10.1086/644537, PMid:19712039

Murray, C.J., Lopez, A.D., Chin, B., Feehan, D. & Hill, K.H., 2006, 'Estimation of potential global pandemic influenza mortality on the basis of vital registry data from the 1918-20 pandemic: A quantitative analysis', *Lancet* 368, 2211–2218. http://dx.doi.org/10.1016/S0140-6736(06)69895-4

Pappaioanou, M. & Gramer, M., 2010, 'Lessons from pandemic H1N1 2009 to improve prevention, detection, and response to influenza pandemics from a One Health perspective', *ILAR Journal J* 51, 268–280.

Peiris, J.S., Yu, W.C., Leung, C.W., Cheung, C.Y., Ng, W.F., Nicholls, J.M. *et al*., 2004, 'Re-emergence of fatal human influenza A subtype H5N1 disease', *Lancet* 363, 617–619. http://dx.doi.org/10.1016/S0140-6736(04)15595-5

Pereda, A., Cappuccio, J., Quiroga, M.A., Baumeister, E., Insarralde, L., Ibar, M. *et al*., 2010, 'Pandemic (H1N1) 2009 outbreak on pig farm, Argentina', *Emerging Infectious Diseases* 16, 304–307. PMid:20113566

Powdrill, T.F., Nipp, T.L. & Rinderknecht, J.L., 2010, 'One health approach to influenza: assessment of critical issues and options', *Emerging Infectious Diseases* 16, e1. http://dx.doi.org/10.3201/eid1608.100673, PMid:20678310

Scholtissek, C. & Naylor, E., 1988, 'Fish farming and influenza pandemics', *Nature* 331, 215. http://dx.doi.org/10.1038/331215a0, PMid:2827036

Smith, W., Andrewes, C.H. & Laidlaw, P.P., 1933, 'A virus obtained from influenza patients', *Lancet* ii, 66–68. http://dx.doi.org/10.1016/S0140-6736(00)78541-2

Social Health and Family Affairs Committee of the Parliamentary Assembly of the Council of Europe, 2010, *Hearing on The handling of the H1N1 pandemic: More transparency needed?*, viewed 12 August 2011, from http://www.assembly.coe.int/CommitteeDocs/2010/20100126_Statement%20Wodarg.pdf

Subbarao, K. & Katz, J., 2000, 'Avian influenza viruses infecting humans', *Cellular and Molecular Life Sciences* 57, 1770–1784. http://dx.doi.org/10.1007/PL00000657, PMid:11130181

Taubenberger, J.K. & Morens, D.M., 2006, '1918 Influenza: the mother of all pandemics', *Emerging Infectious Diseases* 12, 15–22. PMid:16494711

Taylor, L.H., Latham, S.M. & Woolhouse, M.E., 2001, 'Risk factors for human disease emergence', *Philosophical Trnasactions of the Royal Society B: Biological Sciences* 356, 983–989. http://dx.doi.org/10.1098/rstb.2001.0888, PMid:11516376

Thanh, T.T., Van Doorn, H.R. & De Jong, M.D., 2008, 'Human H5N1 influenza: Current insight into pathogenesis', *International Journal of Biochemistry and Cell Biology* 40, 2671–2674. http://dx.doi.org/10.1016/j.biocel.2008.05.019

Trifonov, V., Khiabanian, H. & Rabadan, R., 2009, 'Geographic dependence, surveillance, and origins of the 2009 influenza A (H1N1) virus', *New England Journal of Medicine* 361, 115–119. http://dx.doi.org/10.1056/NEJMp0904572, PMid:19474418

Uiprasertkul, M., Puthavathana, P., Sangsiriwut, K., Pooruk, P., Srisook, K., Peiris, M. *et al*., 2005, 'Influenza A H5N1 replication sites in humans', *Emerging Infectious Diseases* 11, 1036–1041. http://dx.doi.org/10.3201/eid1107.041313, PMid:16022777

Ungchusak, K., Auewarakul, P., Dowell, S.F., Kitphati, R., Auwanit, W., Puthavathana, P. *et al*., 2005, 'Probable person-to-person transmission of avian influenza A (H5N1)', *New England Journal of Medicine* 352, 333–340. http://dx.doi.org/10.1056/NEJMoa044021, PMid:15668219

Uscher-Pines, L., Omer, S.B., Barnett, D.J., Burke, T.A. & Balicer, R.D., 2006, 'Priority setting for pandemic influenza: an analysis of national preparedness plans', *PLoS Medicine* 3, e436. http://dx.doi.org/10.1371/journal.pmed.0030436, PMid:17048982

Uyeki, T.M., 2009, 'Human infection with highly pathogenic avian influenza A (H5N1) virus: Review of clinical issues', *Clinical Infectious Diseases* 49, 279–290. http://dx.doi.org/10.1086/600035, PMid:19522652

Vijaykrishna, D., Poon, L.L., Zhu, H.C., Ma, S.K., Li, O.T., Cheung, C.L. *et al*., 2010, 'Reassortment of pandemic H1N1/2009 influenza A virus in swine', *Science* 328, 1529. http://dx.doi.org/10.1126/science.1189132, PMid:20558710

Waterfield, M., Scrace, G. & Skehel, J., 1981, 'Disulphide bonds of haemagglutinin of Asian influenza virus', *Nature* 289, 422–424. http://dx.doi.org/10.1038/289422a0, PMid:7464912

Webby, R.J. & Webster, R.G., 2003, 'Are we ready for pandemic influenza?', *Science* 302, 1519–1522. http://dx.doi.org/10.1126/science.1090350, PMid:14645836

WHO, 2009, *World now at the start of 2009 influenza pandemic*, viewed 12 August 2011, from http://who.int/mediacentre/=news/statements/2009/h1n1_pandemic_phase6_20090611/en/index.htm1#

WHO, 2011, *Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to WHO*, viewed 12 August 2011, from http://www.who.int/csr/disease/avian_influenza/country/cases_table_2011_08_02/en/index.html#

Wong, S.S. & Yuen, K.Y., 2006, 'Avian influenza virus infections in humans', *Ches* 129, 156–168. http://dx.doi.org/10.1378/chest.129.1.156, PMid:16424427

Woolhouse, M.E. & Gowtage-Sequeria, S., 2005, 'Host range and emerging and reemerging pathogens', *Emerging Infectious Diseases* 11, 1842–1847. http://dx.doi.org/10.3201/eid1112.050997, PMid:16485468

Xu, R., Ekiert, D.C., Krause, J.C., Hai, R., Crowe, J.E. (Jnr) & Wilson, I.A., 2010, 'Structural basis of preexisting immunity to the 2009 H1N1 pandemic influenza virus', *Science* 328, 357–360. http://dx.doi.org/10.1126/science.1186430, PMid:20339031

Yuen, K.Y., Chan, P.K., Peiris, M., Tsang, D.N., Que, T.L., Shortridge, K.F. *et al*., 1998, 'Clinical features and rapid viral diagnosis of human disease associated with avian influenza A H5N1 virus', *Lancet* 351, 467–471. http://dx.doi.org/10.1016/S0140-6736(98)01182-9

Zimmer, S.M. & Burke, D.S., 2009, 'Historical perspective - Emergence of influenza A (H1N1) viruses', *New England Journal of Medicine* 361, 279–285. http://dx.doi.org/10.1056/NEJMra0904322, PMid:19564632

Bartonella spp. in human and animal populations in Gauteng, South Africa, from 2007 to 2009

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How to cite this proceeding:
Trataris, A.N., Rossouw, J., Arntzen, L., Karstaedt, A. & Frean, J., 2012, '*Bartonella* spp. in human and animal populations in Gauteng, South Africa, from 2007 to 2009', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #452, 8 pages. http://dx.doi.org/10.4102/ojvr.v79i2.452

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Bartonellae are highly adaptive organisms that have the ability to evade the host immune system and cause persistent bacteraemia by occupying the host's erythrocytes. *Bartonella* spp. is under-studied and health care professionals often misdiagnose *Bartonella*-related infections. The aim of this study was to investigate the carriage of *Bartonella* spp. circulating in human and animal populations in Gauteng using culturing and polymerase chain reaction (PCR) detection. A total of 424 human, 98 cat, 179 dog, and 124 wild rodent blood samples were plated onto specialised media and incubated for 7–21 days at 37 ºC in CO_2. Culture isolates morphologically similar to *Bartonella* control strains were confirmed by PCR and sequenced to determine species. Deoxyribonucleic acid (DNA) was extracted from all blood samples and tested by nested PCR. *Bartonella* could only be cultured from the cat and rodent specimens. Cat isolates were > 99% similar to *Bartonella henselae* URBHLIE 9, previously isolated from an endocarditis patient, and rat isolates were > 98% similar to either RN24BJ (candidus '*Bartonella thailandensis*') or RN28BJ, previously isolated from rodents in China. The PCR prevalences were 22.5% in HIV-positive patients, 9.5% in clinically healthy volunteers, 23.5% in cats, 9% in dogs and 25% in rodents. Findings of this study have important implications for HIV-positive patients.

Introduction

Background

Bartonella fall within the alpha-2 subgroup of the class Proteobacteria (Jacomo, Kelly & Raoult 2002). Recent studies have indicated that *Bartonella* species (spp.) have some degree of relatedness to other alpha-2 Proteobacteria including *Brucella* species, *Afipia* species, *Agrobacterium tumefaciens, Bradyrhizobium* species, and *Bosea* species (Duncan, Maggi & Breitschwerdt 2007; Greub & Raoult 2002; Houpikian & Raoult 2001; Jacomo *et al.* 2002; Pretorius, Beati & Birtles 2004; Rolain *et al.* 2004). Current knowledge suggests that there are more than 20 species and subspecies included within this genus (Márquez *et al.* 2008). Approximately 13 species have been associated with human diseases (Pérez-Martínez *et al.* 2009; Maggi *et al.* 2009; Pons *et al.* 2008) affecting both immunocompetent and immunocompromised individuals. At least six species affecting humans have been isolated from domestic cats and dogs (Chomel *et al.* 2006).

Bartonellae are pleomorphic, fastidious, oxidase-negative, Gram-negative bacilli (Jacomo *et al.* 2002; Maurin & Raoult 1996). *Bartonella* species growth is hemin-dependent (Wong *et al.* 1995), therefore the addition of hemin-rich rabbit blood or horse blood to the agar yields better growth than sheep blood (Jacomo *et al.* 2002). Growth occurs on enriched medium at 37 °C with 5% carbon dioxide (CO_2); however, growth also occurs in broth with fetal bovine serum, and in various tissue culture systems including cell lines (La Scola & Raoult 1999). On average primary isolates appear after 12 to 14 days (Jacomo *et al.* 2002), although it has been reported that primary isolation can take up to 45 days (Maurin *et al.* 1994). Subcultured colonies have been found to appear after only three to five days (Jacomo *et al.* 2002).

Objectives

In this study, human immunodeficiency virus (HIV) positive people, clinically healthy volunteers, impounded cats, impounded dogs, and commensal rodents were investigated for carriage of *Bartonella* spp. The objective was to determine the rates of infection in the study groups and to determine the species responsible for infection in the study populations.

Literature review

Various studies have been carried out globally to determine the prevalence of *Bartonella* spp. in humans and animals. There is very little data available for the culture or molecular prevalence of human *Bartonella.* The lowest prevalence reported in humans was 3% and the highest was 33.3%, in the USA (Breitschwerdt *et al.* 2007; Bonilla *et al.* 2009; Koehler *et al.* 2003). The prevalence in

cat populations was from as little as 2% in Canada (Kamrani *et al*. 2008) to as high as 53% in France (Heller *et al.* 1997), in dog populations from 0% in the United Kingdom (Birtles *et al.* 2002) to 16% in Korea (Kim *et al*. 2009), and in rodent populations from 6% in Indonesia (Winoto *et al*. 2005) to 43.5% in China (Ying *et al.* 2002).

The prevalence of *Bartonella* spp. is largely unknown in South Africa. There are some case reports of bacillary angiomatosis caused by *Bartonella* spp. in HIV positive patients (Frean, Arndt & Spencer 2002). A non-random pilot survey of outpatients of three Johannesburg HIV clinics has been done on a relatively small sample group (n = 188 patients). This study showed a 10% prevalence of *Bartonella henselae* in blood of HIV-positive patients, determined by nested polymerase chain reaction (PCR) (Frean *et al.* 2002). A prevalence study in domestic and wild felines in southern Africa published by Kelly *et al.* (1996) reported 23% (n = 171) prevalence. A study of rodents in South Africa showed a high rate of infection (44%) with a wide range of subtypes of bartonellae (Pretorius *et al.* 2004).

Materials and methods

Sample collection

Ethical considerations

Ethical approval for collection of samples from humans and animals was granted by the Human Research Ethics Committee (Medical) of the University of the Witwatersrand (Ethics Approval Number: M070637), the National Health Laboratory Service Animal Ethics Committee (Ethics Approval Number: 2007/112), and the University of the Witwatersrand Animal Ethics Screening Committee (AESC) (Ethics Approval Number: 2008/56/03).

Recruitment of participants and informed consent

A convenience sample of 382 HIV-positive patients was recruited from the Chris Hani Baragwanth Hospital (Soweto, Gauteng), HIV-clinic. HIV-status was known as the clinic only treats HIV-positive patients. Specimen collection started in September 2007 and was completed by the end of January 2008. Survey objectives and consent forms were explained to prospective participants by an available HIV-counselor employed at the clinic, a laboratory aid or by the phlebotomists drawing the blood samples. Patients were assured that the study was voluntary and results would not directly benefit nor disadvantage them in any way. Informed consent forms were completed by the participating patients before blood collection. The recruited volunteers were provided with an information sheet containing the contact details of the Special Bacterial Pathogens Reference Laboratory (SBPRL) at the National Institute for Communicable Diseases (NICD) to which queries were addressed.

Clinically healthy volunteers were recruited from the National Institute for Communicable Diseases and a local animal shelter for blood collection. Due to ethical considerations, the volunteers were not required to disclose their HIV status, and health was assumed on the basis of no observable signs of illness at the time of blood collection. These volunteers were also required to complete an informed consent form and participation was completely voluntary. Two 4 mL tubes of blood were collected from each volunteer, 1 tube containing the anticoagulant ethylenediaminetetra-acetic acid (EDTA) and the other a plain red-top tube in which the blood was allowed to clot. Blood specimens were stored at minus 20 °C until use.

Animal samples

Blood was collected from dogs and cats that were undergoing euthanasia, surgery or sterilisation procedures at a local animal shelter. Two 2-mL tubes of blood (as described above) were collected from each cat and dog. Samples were stored at 4 °C until use.

Rodents were prospectively sourced from a pest control company operating in the Ekurhuleni Metropolitan area. Rodents were trapped alive, anaesthetised using CO_2, and exsanguinated by cardiac puncture. Blood was collected using a syringe and needle from each rodent and was divided into two tubes, one red top and one purple top (as above). Specimens were stored at 4 °C until use.

Procedures

Culturing of *Bartonella* spp.

All blood samples were plated onto 5% rabbit blood-supplemented Columbia agar (hereafter referred to as *Bartonella* medium). Culturing was carried out in a class two biohazard safety cabinet to reduce the risk of contamination. Ethylenediaminetetra-acetic acid blood was predominantly used for culturing, unless the EDTA specimen was insufficient (especially for cat and dog samples), in which case some of the clotted blood (packed cells excluding serum) was used. Approximately 100 µL – 150 µL of each whole blood sample was inoculated onto fresh *Bartonella* media and spread over the surface using sterile glass spreaders. Inoculated media were incubated in 5% CO_2 at 37 °C for seven ± two days before being checked for growth.

If small dry colonies, pitting into the media were present, they were Gram stained, and plated out onto fresh media. Media with faint or no growth were re-incubated as above to a maximum of two months with periodic examinations every seven days. All blood culture isolates were confirmed by PCR and stored in tryptic soy broth (TSB) with 10% (v/v) glycerol at minus 80 °C.

Genus specific polymerase chain reaction amplification

All culture isolates and blood samples were tested by genus specific PCR amplification of the intergenic spacer (ITS) region between the 16S and 23S rRNA genes for evidence of *Bartonella* Deoxyribonucleic acid (DNA). Deoxyribonucleic acid extractions were carried out using the QIAamp DNA mini kit (Qiagen, Germany) as per kit protocol.

Published genus-specific primers were selected as shown in Table 1 (Roux & Raoult 1995; Seki *et al.* 2006). The reverse primer QHVE-14 (Seki *et al.* 2006) was modified by elongating the 5′ end by three nucleotides to decrease non-specific amplification.

Polymerase chain reaction confirmation of cultured isolates was carried out using primers QHVE-1 and QHVE-3, whereas the blood-extracted DNA required a nested PCR using the QHVE-12 and QHVE-14b inner primers. Table 2 shows the primer binding localities and product sizes of the six most commonly isolated human *Bartonella* spp., although other species within the genus are also detectable using these primers.

The single or first round PCR amplifications were carried out in a reaction mixture (50 µL) containing 20 ρmol of each primer (QHVE-1 and QHVE-3), 1 x Buffer II, 2 mM $MgCl_2$, 1.5 U AmpliTaq DNA polymerase (Applied Biosystems, USA), 200 µM of each deoxyribonucleotide triphosphate (dNTP) (Thermo Scientific, United Kingdom), and 1% (v/v) Triton-X 100 surfactant additive. The amount of DNA was used depended on the source of the DNA. For the culture DNA, 20 ng of template was included into the reaction, whereas 5 µL of the total blood extracted extracted DNA was included into the reaction. Polymerase chain reactions were performed on a VERITI Thermocycler (Applied Biosystems) under the following conditions: 2 min initial denaturation step at 94 °C, followed by 35 cycles of the following steps: denaturation at 94 °C for 30 s, primer annealing at 52 °C for 30 s, and elongation at 72 °C for 60 s. A final elongation step concluded the amplification at 72 °C for 6 min. Polymerase chain reaction products were maintained at 4 °C until being added to the reaction mixtures of the nested round. For the nested PCR reactions, a final volume of 50 µL consisted of 2 µL of first-round product, 1 x Buffer II, 1.5 mM $MgCl_2$, 30 ρmol of each inner primer (QHVE-12 and QHVE-14b), 200 µM of each dNTP, and 1.5 U AmpliTaq DNA polymerase. The reactions were amplified as previously described, with a variation in the annealing temperature (55 °C).

Amplicon analysis was performed on a 2% (w/v) Tris-acetic acid-EDTA (TAE) buffer agarose (WhiteSci, USA) gel, supplemented with 0.5 µg/mL ethidium bromide. Electrophoresis was carried out at 100 V in 1 x TAE for 40 min. Gels were visualised by ultraviolet illumination.

Deoxyribonucleic acid sequencing

The culture isolates were speciated by sequencing of the ITS region using the outer primers (QHVE-1 and QHVE-3). Amplicons were gel purified using the QIAquick Gel Extraction Kit (Qiagen, Germany) as per manufacturer protocol and sent to Inqaba Biotechnologies for direct sequencing.

Analysis

Sequences were aligned and analysed using BioEdit freeware (http://www.mbio.ncsu.edu/BioEdit/bioedit.html). Sequences were exported into the National Center for Biotechnology Information (NCBI) website's Basic Local Alignment Search Tool (BLAST) database for species identification of the isolates. A tree of relatedness was drawn using the neighbor-joining method (Saitou & Nei 1987) of molecular evolutionary genetics analysis (MEGA4) freeware (http://www.megasoftware.net/) (Tamura *et al.* 2007).

Results

Isolation of *Bartonella* from human and animal blood specimens

A total of 382 HIV-positive patients attending the HIV clinic volunteered to take part in this study; 246 (64%) were women and 136 (36%) were men. Ethylenediaminetetra-acetic acid blood samples (whole blood including plasma) were plated out after having been freeze-thawed from minus 20 °C. Blood cultures from this sample group yielded no *Bartonella* spp. isolates. Approximately a third (130/382) of the blood cultures were contaminated by fungal or environmental bacteria growth due to the prolonged incubation periods. In order to decrease the amount of contamination, amphotericin B was included in the blood culture technique. To assess the effect of amphotericin B on the growth of *Bartonella*, the *B. henselae* ATCC strain was inoculated into BHI broth with 5% (w/v) amphotericin B and plated out onto fresh *Bartonella* media. Upon assessment, it was observed that although amphotericin B does reduce the amount of fungal contamination, it also suppresses the growth of the control culture. The addition of

TABLE 1: Published genus-specific primers.

Name	Sequence	Bp	Reference
QHVE-1	5′ – TTC AGA TGA TGA TCC CAA GC – 3′	20	Roux and Raoult 1995; La Scola and Raoult 1999
QHVE-3	5′ – AAC ATG TCT GAA TAT ATC TTC – 3′	21	Roux and Raoult 1995; La Scola and Raoult 1999
QHVE-12	5′ – CCG GAG GGC TTG TAG CTC AG – 3′	20	Seki *et al.* 2006
QHVE-14a	5′ – CAC AAT TTC AAT AGA AC – 3′	17	Seki *et al.* 2006
QHVE-14b	5′ – CCT CAC AAT TTC AAT AGA AC – 3′	20	Unpublished

Note: Please see the full reference list of the article, Trataris, A.N., Rossouw, J., Arntzen, L., Karstaedt, A. & Frean, J., 2012, '*Bartonella* spp. in human and animal populations in Gauteng, South Africa, from 2007 to 2009', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #452, 8 pages. http://dx.doi.org/10.4102/ojvr.v79i2.452, for more information.

TABLE 2: Primer binding localities and product sizes of the six most commonly isolated human *Bartonella* spp.

Species	Outer primers			Inner primers		
	Bp	QHVE-1	QHVE-3	Bp	QHVE-12	QHVE-14
Bartonella henselae	723	318–337	1021–1041	568	448–467	1000–1016
Bartonella quintana	640	353–372	973–993	500	468–487	952–968
Bartonella vinsonii	661	336–355	977–997	481	491–510	956–972
Bartonella elizabethae	788	359–378	1135–1147	572	558–577	1114–1130
Bartonella clarridgeiae	711	313–332	1004–1024	573	425–445	982–998
Bartonella grahamii	736	311–330	1026–1046	487	538–557	1005–1024

amphotericin B was therefore discontinued. Due to the low circulating bacterial load often observed in natural infection, the risk of suppression, although slight, was not one worth taking. If a culture became contaminated with fungal growth, the culture was repeated. To ensure that *Bartonella* was not missed, any cultured organisms were Gram-stained, and any Gram-negative pleomorphic bacilli were sub-cultured and *Bartonella* was excluded by PCR.

A total of 42 clinically healthy volunteers were recruited to determine normal infection rates. Seven animal shelter staff and 35 National Institute for Communicable Diseases' staff members participated in this study. All specimens were freshly cultured onto the *Bartonella* media. Only one of the 42 blood specimens illustrated growth morphologically consistent with *Bartonella* spp. Unfortunately, the culture failed to grow on sub-culture.

The seven animal shelter volunteers had daily exposure to many different animals. *Bartonella* spp. infections have been associated with cats and dogs (Barnes *et al.* 2000; Ketring *et al.* 2004) and it was therefore expected that this group would be most likely to demonstrate *Bartonella* bacteraemia. This was not the case in this study, but the sample size was inadequate.

Dog (n = 179) blood specimens yielded no *Bartonella* culture isolates, but cat (n = 98) blood specimens yielded five (approximately 5%) *Bartonella* culture isolates that were confirmed by PCR. It was concerning that there were so few cultured isolates and that three of the five isolates obtained came from a single batch of seven feline blood samples received from the animal shelter, whereas preceding batches had not yielded culture isolates. To exclude the possibility of cross-contamination within this batch, the blood samples were re-cultured and subjected to PCR confirmation.

A total of 124 rodent samples taken from *Rattus norvegicus* and *Rattus rattus* were cultured on *Bartonella* medium. *Bartonella* was isolated from 16 (13%) rodents. All isolates were Gram-negative, pleomorphic bacilli with morphology consistent with *Bartonella*. Primary isolation occurred between five and nine days, with sub-cultures growing within four to five days. At least two different colony morphologies (Table 3) were observed for the rodent isolates, and these were later confirmed and identified by sequencing.

Molecular detection and confirmation of *Bartonella*

A total of 21 culture isolates were confirmed as *Bartonella* by PCR. Amplicon sizes varied between 728 and 809 bp.

Polymerase chain reaction of the HIV-positive population yielded a prevalence of 22.5% (86/382) (95% confidence; 18.5–27.1), whereas the clinically healthy group had a prevalence of 9.5% (4/42) (95% confidence; 3.1–23.5). This is a significant difference (p-value: 0.05; chi-square statistic: 3.818 with one degree of freedom) in the proportion of current infection for the two populations. This difference is unlikely to have occurred through mere chance, although the limited healthy volunteer sample size is a major limitation.

TABLE 3: Two different colony morphologies observed for the rodent isolates.

Morphology type 1	Morphology type 2
Tiny, pin-point, smooth, moist, and slightly metallic sheen	Tiny, pin-point , drier, rougher, and metallic sheen
Grew on surface of medium	Variable degrees of pitting into the medium
Highly self-adhesive, but easily scraped off the surface of the medium	More difficult to scrape off the medium due to pitting

The feline blood tested by PCR indicated a 23.5% (23/98) (95% confidence; 15.8–33.3) *Bartonella* prevalence. This was significantly different (p-value: 0.0002; chi-square statistic: 13.500 with one degree of freedom) to the culture prevalence (5%). Both test techniques test for current infection; however, due to the fastidious nature of the bacteria, PCR is the far more efficient method for detection of *Bartonella* spp. as it does not rely on the viability of the bacteria.

The dog PCR prevalence was 9% (16/179) (95% confidence; 5.4–14.4), which is significantly lower than the prevalences in felines (p-value: 0.0009; chi-square statistic: 11.053 with one degree of freedom) and rodents (p-value: 0.0001; chi-square statistic: 14.419 with one degree of freedom).

Rat bloods tested by PCR indicated 25% prevalence (31/124) (95% confidence; 17.9–33.7). There was a significant difference (p-value: 0.0151; chi-square statistic: 5.907 with one degree of freedom) between PCR prevalence and culture prevalence (13%). When the prevalence for rats was compared with that of the felines, there was no significant difference (p-value: 0.7918; chi-square statistic: 0.070 with one degree of freedom).

Deoxyribonucleic acid sequencing and analysis of *Bartonella* isolates

Comparison with GenBank (NCBI website) sequences showed that the rodent isolates ranged in percentage similarity from 97% – 99% to either the recently named novel species candidatus '*Bartonella thailandensis*' (RN24BJ;

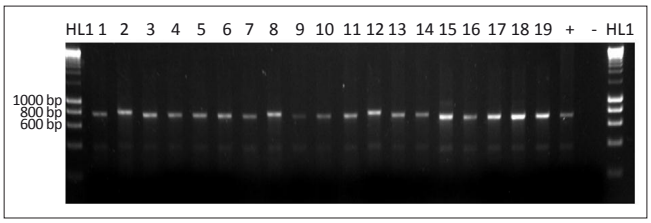

FIGURE 1: Molecular detection and confirmation of *Bartonella.*

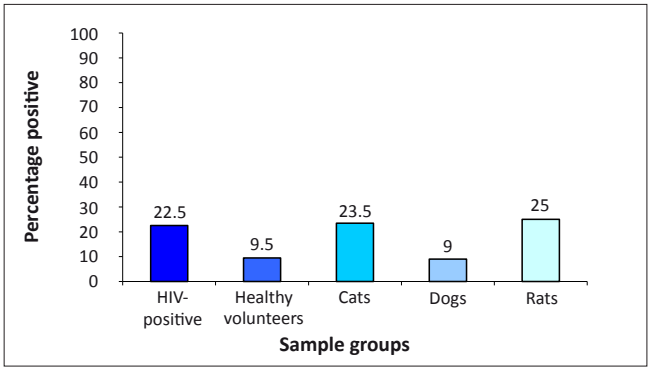

FIGURE 2: The positive percentage for the sample group.

accession number: EF190333.1) or RN28BJ (accession number: EF213776.1). Both isolates were described in a recent publication by Saisongkorh *et al.* (2009) where the rodents in Beijing, China were tested for bartonellae.

The rodent isolates were slightly more variable and a tree of relatedness was constructed (Figure 3). Contingent from the ITS data using parsimony and distance methods illustrated two well-supported (more than 90% bootstrap values) clusters within the isolates. The first cluster placed RN24BJ with 12 of the isolates (BART0272, BART0323, BART0268, BART0354, BART0379, BART0381, BART0312, BART0324, BART0359, BART0361, BART0357 and BART0358) and the second group clustered RN28BJ with the remaining three isolates (BART0271, BART 0355 and BART 0377). *Bartonella elizabethae* (GenBank accession number: L35103) and *Bartonella grahamii* (GenBank accession number: AJ269785) were used for comparison. *Bartonella elizabethae* was found to be most similar to the rodent isolates from this study.

The five cat isolates were 99% – 100% similar to *B. henselae* URBHLIE 9 (accession number: AF312496.1). Primers (QHVE1 & QHVE3) amplified a region consisting of 687 bp (excluding primers) for all the feline isolates. BART0480 and BART0483 were 100% identical to the *B. henselae* URBHLIE-9 strain, and had only one nucleotide difference from *B. henselae* Houston-1 (accession number: L35101) strain at position 98. BART0519 was 99% similar to URBHLIE-9 with a heterogenous nucleotide at position 285, where adenosine (A) or guanine (G) is equally expressed. BART0519 and BART0484 were identical to each other and 99% similar to URBHLIE-9. One nucleotide difference was observed at position 660.

Discussion

Bartonella remains one of the most difficult organisms to detect via blood or tissue culture. Frean *et al.* (2002) looked at the prevalence of *Bartonella* in HIV-positive out-patients at several hospitals in Johannesburg and found a 10% PCR prevalence of *Bartonella*. A conservative expectation for the present study was to find at least 10% culture-positive specimens for *Bartonella* spp.

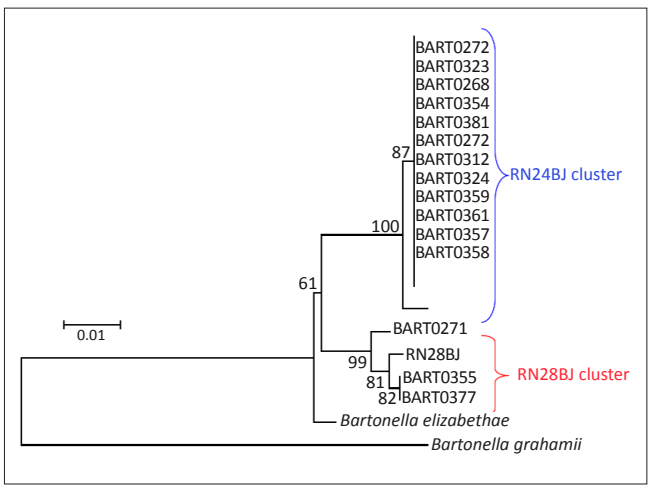

FIGURE 3: Tree of relatedness.

Bartonella henselae was first isolated from the bloodstream of an AIDS patient (Regnery *et al.* 1992a). Severely immunocompromised people are most at risk of contracting a *Bartonella* infection (Boulouis *et al.* 2005) and those with bacillary angiomatosis remain bacteremic for a number of weeks (Koehler & Tappero 1993). HIV-infected patients with CD4+ cell counts of less than 50/mm^3 are more likely to develop bacillary angiomatosis lesions (Boulouis *et al.* 2005; Koehler & Tappero 1993), and as a precaution, broad-spectrum antibiotic treatments are periodically prescribed for these patients. The effect of broad-spectrum antibiotics on *Bartonella* infections lacks clarity. Research has been conducted on *in vivo* and *in vitro* antimicrobial sensitivities of *Bartonella* to various drug classes. *In vitro* testing has shown bactericidal efficacy, but *in vivo* tests have demonstrated mostly bacteristatic activity, with the exception of aminoglycosides which are bactericidal (Florin *et al.* 2008). The bacteriostatic activity of the broad-spectrum drugs may be suppressing circulating bacteremia to undetectable levels for culture. It is probable that the HIV-positive patients we tested were on a range of broad-spectrum antibiotics as prophylaxis against opportunistic infections. Information regarding whether or not these patients were on the antibiotic treatment, the type of antibiotic treatment taken, the prescribed doses, and duration of treatment administered was not collected at the time of specimen collection and is thus unavailable. It was speculated that this would influence *Bartonella* isolation from the specimens.

Published methodologies were followed for culture, including freeze-thawing of the specimens (La Scola & Raoult 1999). The methodologies were later refined as it was discovered that inoculating fresh packed red-blood cell samples onto the *Bartonella* media yielded better results. Although culture is regarded as the gold standard for detection, it is limited and problematic. The lack of cultured *Bartonella* in human blood does not indicate that the prevalence is zero, since *Bartonella* spp. was detected by PCR.

This study has shown an even higher *Bartonella* prevalence (22.5%) in HIV-positive out-patients than the 10% previously reported (Frean *et al.* 2002). In immunocompromised individuals *B. henselae* infections are usually associated with exposure to cats and cat fleas (Boulouis *et al.* 2005; Koehler & Tappero 1993). The highest prevalences found in this study were for cats (23.5%) and rats (25%). These prevalences were not as high as some of the other reports published on the prevalence of bartonellae in animals. Dog and cat samples were stored in the fridge at 4 °C for up to three weeks before being processed. It is hypothesised that *Bartonella* bacilli (if present in the blood) became non-viable due to prolonged storage time. Supporting this hypothesis is the fact that isolates were obtained from blood specimens that were processed within one week of collection. This is further supported by the fact that PCR detected *Bartonella* DNA in the blood at a much higher rate than culture (23.5% vs 5%).

An interesting finding for the present study was the isolation of *B. henselae* URBHLIE9 from all five culture-positive cat isolates. This strain was previously isolated from the blood of a patient presenting with endocarditis and implies a strong

link between humans and cats as reservoirs for bartonellae (Houpikian & Raoult 2001).

Polymerase chain reaction results indicate that there is a high prevalence of bartonellae in human and animal populations in Gauteng Province, South Africa. More work is required to fully understand the extent of disease resulting from these *Bartonella* infections.

Limitations

Bartonella prevalence for HIV-positive patients in Gauteng was found to be much higher than previously reported. Based on the 22.5% (95% confidence) PCR prevalence, it is speculated that approximately 1 in 4 HIV-positive patients is infected with *Bartonella*. Although most patients are consistently subjected to broad-spectrum antibiotic treatment, the efficacy of treatment is not known since treatment efficacy varies from patient-to-patient. The fact that 22.5% of the tested samples were PCR positive for *Bartonella* indicates that the bacteria are present in the host. *In vitro* efficacy of antibiotic use against *Bartonella* has been limited (Florin *et al.* 2008). In most cases antibiotics have had an inhibitory effect rather than a bactericidal one. The lack of cultured isolates from HIV-positive human samples could be attributed to this inhibitory effect.

Treatment is onerous as it has to be administered for prolonged periods to suppress infections, such as *Bartonella* infections. There is limited information on the economic burden of *Bartonella* infections, since diseases caused by *Bartonella* are not notifiable. Furthermore, due to the difficulty in diagnosis of *Bartonella,* infections often go undetected and untreated.

Although there are various methodologies available for detection of *Bartonella*, each method has its own limitations. Most methods are only used for research purposes and highly-skilled laboratory staff. The first test dealt with in this study is culture. Most microbiology laboratories are proficient in this method and culture is generally regarded as the gold standard for most bacterial diagnoses. Culture of *Bartonella* differs from most conventional bacteria culture as it is more labor-intensive. Fresh EDTA blood must be centrifuged and the pellet is plated onto specialised media supplemented with rabbit blood. Isolation requires a prolonged incubation period from 7–21 days at 37 ºC under microaerophillic conditions. Most laboratories do not have the capacity to maintain these cultures for prolonged periods or prevent contamination. Assuming the above conditions are met, there still remains the task of confirmation of the culture as *Bartonella.* Bartonellae do not visibly metabolise compounds provided in rapid biochemical test panels. *Bartonella* spp. is oxidase and catalase negative and stain faintly as Gram negative, slightly curved, bacilli. Small diagnostic laboratories often do not have the means of confirming the culture isolate as *Bartonella.*

Polymerase chain reaction has become a relatively affordable option when testing for bacterial diseases, although affordability is relative. A skilled and competent operator performing a PCR may not have problems carrying out the test, particularly if it is a routine test, but if the operator does not routinely perform the test (especially for *Bartonella*) the method is relatively labour-intensive and prone to contamination. Much research has gone into developing nested PCR, high-tech real-time PCR and amplicon sequencing. These tests remain largely for research purposes only. Staff would have to be trained; primers and other reagents would have to be readily available and expensive equipment such as thermocyclers, electrophoresis power packs and tanks, UV light-boxes, software and computers would have to be purchased and maintained.

Recommendations

It is recommended that operator-friendly tests such as the IFA become more affordable for use in laboratories. Perhaps an ELISA as previously described (Bergmans *et al.* 1997; Vermeulen *et al.* 2007) would be a more cost effective method. Control culture is relatively easy to grow and the antigens of the culture can be sonicated from the growth and consequently be used to coat the microtitre wells. This method can be optimised and made 'in-house' thus mitigating the need for expensive commercially available kits. *Bartonella* testing is especially required for immunocompromised patients presenting with clinical symptoms and histories.

Training and out-reach programs directed towards doctors and the generally public should be carried out to ensure the indicative symptoms are recognised, and reported when presenting for medical assistance. Pest control and fleas control methods should be advised and implementable solutions should also be discussed.

Conclusion

In this study, results were within global trends for *Bartonella* prevalence in both human and animal populations. The objectives of the study were met and a better understanding of *Bartonella* prevelance in human and animal populations in Gauteng, South Africa is now available. This study has confirmed that the primary concern is human health, particularly for immunocompromised people who are at higher risk of contracting various opportunistic infections including *Bartonella*.

Bartonella is prevalent in human and animal populations in Gauteng, South Africa, and could be responsible for a number of unresolved or misdiagnosed diseases in immunocompromised patients. Although the broad-spectrum antibiotics offered to HIV-positive patients in particular maybe suppressing the infection, the treatment would have to be constant and there are possible side-effects that could cause the patient more discomfort. It is important to emphasise that immunocompromised patients be very careful when handling domestic animals.

Furthermore, attempts should be made to decrease the risk of rat exposure. By keeping food in sealed containers off

the floor, and ensuring proper sanitation in and around the home is followed, the household rodent population would be largely reduced.

Too little is known about *Bartonella* and further research is required to fully understand the extent of disease related to this emergent pathogen.

Acknowledgements

It is with gratitude that we would like to acknowledge the following; firstly the staff of SBPRL – CEZD for their support and assistance with the project, Mr Justice Matebula (Laboratory aid) who assisted with the translations and explanations of the informed consent in the recruitment of volunteer participants, the HIV-clinic councilors and phlebotomists for their assistance with translations, explanations and recruitment of HIV-positive volunteers as well as specimen collection, and to the staff of NICD and the animal shelter (wished to remain anonymous) for 'bleeding' for the cause. The animal shelter is furthermore thanked for assisting us with obtaining the cat and dog specimens. A special thank you is owed to Mr Lee Ashford and his enthusiastic staff at 'Scientific Supakill' pest-control company for providing us with the live rodents. And finally thank you to the NICD/NHLS for their support and use of facilities in this study.

Competing interests

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

References

Birtles, R.J., Laycock, G., Kenny, M.J., Shaw, S.E. & Day, M.J., 2002, 'Prevalence of *Bartonella* species causing bacteraemia in domesticated and companion animals in the United Kingdom', *Veterinary Record* 151, 225–229. http://dx.doi.org/10.1136/vr.151.8.225, PMid:12219899

Bonilla, D.L., Kabeya, H., Henn, J., Kramer, V.L. & Kosoy, M.Y., 2009, '*Bartonella quintana* in body lice and head lice from homeless persons, San Francisco, California, USA', *Emerging Infectious Diseases* 15, 912–915. http://dx.doi.org/10.3201/eid1506.090054, PMid:19523290

Boulouis, H.J., Chang, C.C., Henn, J.B., Kastenc, R.W. & Chomel, B.B., 2005, 'Factors associated with the rapid emergence of zoonotic *Bartonella* infections', *Veterinary Research* 36, 383–410. http://dx.doi.org/10.1051/vetres:2005009, PMid:15845231

Branley, J., Wolfson, C., Waters, P., Gottlieb, T. & Bradbury, R., 1996, 'Prevalence of *Bartonella henselae* bacteremia, the causative agent of cat scratch disease, in an Australian cat population', *Pathology* 28, 262–265. http://dx.doi.org/10.1080/00313029600169124, PMid:8912359

Breitschwerdt, E.B., Maggi, R.G., Duncan, A.W., Nicholson, W.L., Hegarty, B.C. & Woods, C.W., 2007, '*Bartonella* species in blood of immunocompetent persons with animal and arthropod contact', *Emerging Infectious Diseases* 13, 938–941. http://dx.doi.org/10.3201/eid1306.061337, PMid:17553243

Chomel, B.B., Boulouis, H.J., Maruyama, S. & Breitschwerdt, E.B., 2006, '*Bartonella* spp. in pets and effect on human health', *Emerging Infectious Diseases* 12, 389–394. http://dx.doi.org/10.3201/eid1203.050931, PMid:16704774

Duncan, A.W., Maggi, R.G. & Breitschwerdt, E.B., 2007, 'A combined approach for the enhanced detection and isolation of *Bartonella* species in dog blood samples: Pre-enrichment liquid culture followed by PCR and subculture onto agar plates', *Journal of Microbiological Methods* 69, 273–281. http://dx.doi.org/10.1016/j.mimet.2007.01.010, PMid:17346836

Florin, T.A., Zaoutis, T.E. & Zaoutis, L.B., 2008, 'Beyond cat scratch disease: Widening spectrum of *Bartonella henselae* infection', *Pediatrics* 121, 1413–1425. http://dx.doi.org/10.1542/peds.2007-1897, PMid:18443019

Frean, J., Arndt, S. & Spencer, D., 2002, 'High rate of *Bartonella henselae* infection in HIV-positive outpatients in Johannesburg, South Africa', *Transactions of the Royal Society of Tropical Medicine and Hygiene* 96, 549–550. http://dx.doi.org/10.1016/S0035-9203(02)90437-2

Greub, G. & Raoult, D., 2002, '*Bartonella*: New explanations for old diseases', *Journal of Medical Microbiology* 51, 915–923. PMid:12448674

Heller, R., Artois, M., Xemar, V., De Briel, D., Gehin, H., Jaulhac, B. *et al*., 1997, 'Prevalence of *Bartonella henselae* and *Bartonella clarridgeiae* in stray cats', *Journal of Clinical Microbiology* 35, 1327–1331. PMid:9163438

Houpikian, P. & Raoult, D., 2001, '16S/23S rRNA intergenic spacer regions for phylogenetic analysis, identification, and subtyping of *Bartonella* species', *Journal of Clinical Microbiology* 39, 2768–2778.

Jacomo, V., Kelly, P.J. & Raoult, D., 2002, 'Natural history of *Bartonella* infections (an exception to Koch's postulate)', *Clinical and Diagnostic Laboratory Immunology* 9, 8–18. PMid:11777823

Kamrani, A., Parreira, V.R., Greenwood, J. & Prescott, J.F., 2008, 'The prevalence of *Bartonella*, hemoplasma, and *Rickettsia felis* infections in domestic cats and in cat fleas in Ontario', *The Canadian Journal of Veterinary Research* 72, 411–419. PMid:19086373

Kelly, P.J., Matthewman, L.A., Hayter, D., Downey, S., Wray, K., Bryson, N.R. & Raoult, D., 1996, '*Bartonella* (*Rochalimaea*) *henselae* in southern Africa – evidence for infections in domestic cats and implications for veterinarians', *Journal of the South African Veterinary Association* 67, 182–187. PMid:9284029

Kim, C.M., Kim, J.Y., Yi, Y.H., Lee, M.J., Cho, M.R., Shah, D.H. *et al*., 2005, 'Detection of *Bartonella* species from ticks, mites and small mammals in Korea', *Journal of Veterinary Science* 6(4), 327–334. PMid:16293997

Kim, Y.S., Seo, K.W., Lee, J.H., Choi, E.W., Lee, H.W., Hwang, C.Y. *et al*., 2009, 'Prevalence of *Bartonella henselae* and *Bartonella clarridgeiae* in cats and dogs in Korea', *Journal of Veterinary Science* 10(1), 85–87. http://dx.doi.org/10.4142/jvs.2009.10.1.85, PMid:19255530

Koehler, J.E. & Tappero, J.W., 1993, 'Bacillary angiomatosis and bacillary peliosis in patients infected with human immunodeficiency virus', *Clinical Infectious Diseases* 17, 612–624. http://dx.doi.org/10.1093/clinids/17.4.612, PMid:8268340

Koehler, J.E., Sanchez, M.A., Tye, S., Garrido-Rowland, C.S., Chen, F.M., Maurer, T. *et al*., 2003, 'Prevalence of *Bartonella* infection amoung human immunodeficiency virus infected patients with fever', *Clinical Infectious Diseases* 37, 559–566. http://dx.doi.org/10.1086/375586, PMid:12905141

La Scola, B. & Raoult, D., 1999, 'Culture of *Bartonella quintana* and *Bartonella henselae* from Human Samples: A 5-Year Experience (1993 to 1998)', *Journal of Clinical Microbiology* 37, 1899–1905.

Maggi, R.G., Kosoy, M., Mintzer, M. & Breitschwerdt, E.B., 2009, 'Isolation of Candidatus *Bartonella melophagi* from Human Blood', *Emerging Infectious Diseases* 15(1), 66–68. http://dx.doi.org/10.3201/eid1501.081080, PMid:19116054

Márquez, F.J., Rodríguez-Liébana, J.J., Pachón-Ibáñez, M.E., Docobo-Pérez, F., Hidalgo-Fontiveros, A., Bernabeu-Wittel, M. *et al*., 2008, 'Molecular Screening of *Bartonella* Species in Rodents from South Western Spain', *Vector-Borne and Zoonotic Diseases* 8(5), 695–700. http://dx.doi.org/10.1089/vbz.2007.0257, PMid:18620508

Maurin, M., Roux, V., Stein, A., Ferrier, F., Viraben, R. & Raoult, D., 1994, 'Isolation and characterisation by immunofluorescence, sodium dodecyl sulfate-polyacrylamide gel electrophoresis, western blot, restriction fragment length polymorphism-PCR, 16srRNA gene sequencing, and pulsed-field gel electrophoresis of *Rochalimaea quintana* from a patient with bacillary angiomatosis', *Journal of Clinical Microbiology* 32, 1166–1171. PMid:7519628

Maurin, M. & Raoult, D., 1996, '*Bartonella* (*Rochalimaea*) *quintana* infections', *Clinical Microbiology Reviews* 9, 273–292. PMid:8809460

Pérez-Martínez, L., Venzal, J.M., González-Acuña, D., Portillo, A., Blanco, J.R. & Oteo, J.A., 2009, '*Bartonella rochalimae* and other *Bartonella* spp. in Fleas, Chile', *Emerging Infectious Diseases* 15(7), 1150–1152. http://dx.doi.org/10.3201/eid1507.081570, PMid:19624952

Pons, I., Sanfeliu, I., Nogueras, M.M., Sala, M., Cervantes, M., Amengual, M.J. *et al*., 2008, 'Seroprevalence for *Bartonella* spp. infection in HIV patients in Catalonia, Spain', *BioMed Central Infectious Diseases* 8, 58. http://dx.doi.org/10.1186/1471-2334-8-58, PMid:18452613

Pretorius, A., Beati, L. & Birtles, R.J., 2004, 'Diversity of bartonellae associated with small mammals inhabiting Free State province, South Africa', *International Journal of Systematic and Evolutionary Microbiology* 54, 1959–1967. http://dx.doi.org/10.1099/ijs.0.03033-0, PMid:15545418

Rampersad, J.N., Watkins, J.D., Samlal, M.S., Deonanan, R., Ramsubeik, S. & Ammons, D.R., 2005, 'A nested-PCR with an Internal Amplification Control for the detection and differentiation of *Bartonella henselae* and *B. clarridgeiae*: An examination of cats in Trinidad', *BioMed Central Infectious Diseases* 5, 63–68. http://dx.doi.org/10.1186/1471-2334-5-63, PMid:16098227

Regnery, R.L., Anderson, B.E., Clarridge, J.E., Rodriguez-Barradas, M.C., Jones, D.C. & Carr, J.H., 1992, 'Characterization of a novel *Rochalimaea* species, *R. henselae* sp. Nov., isolated from blood of a febrile, human immunodeficiency virus-positive patient', *Journal of Clinical Microbiology* 30, 265–274. PMid:1371515

Rolain, J.M., Locatelli, C., Chabanne, L., Davoust, B. & Raoult, D., 2004, 'Prevalence of *Bartonella clarridgeiae* and *Bartonella henselae* in domestic cats from France and detection of the organisms in erythrocytes by Immunofluorescence', *Clinical and Diagnostic Laboratory Immunology* 11(2), 423–425. PMid:15013998

Roux, V. & Raoult, D., 1995, 'Inter- and intraspecies identification of *Bartonella (Rochalimaea)* species', *Journal of Clinical Microbiology* 33, 1573–1579. PMid:7650189

Sander, A., Buhler, C., Pelz, K., Von Cramm, E. & Bredt, W., 1997, 'Detection and identification of two *Bartonella henselae* variants in domestic cats in Germany', *Journal of Clinical Microbiology* 35(3), 584–587. PMid:9041393

Seki, N., Sasaki, T., Sawabe, K., Sasaki, T., Matsuoka, M., Arakawa, Y. *et al*., 2006, 'Epidemiological Studies on *Bartonella quintana* Infections among Homeless People in Tokyo, Japan', *Japanese Journal of Infectious Diseases* 59(1), 31–35. PMid:16495631

Winoto, I.L., Goethert, H., Ibrahim, I.N., Yuniherlina, I., Stoops, C., Susanti, I. *et al*., 2005, '*Bartonella* species in rodents and shrews in the Greater Jakarta Area', *Southeast Asia Journal of Tropical Medicine and Public Health* 36(6), 1523–1529. PMid:16610656

Wong, M.T., Thornton, D.C., Kennedy, R.C. & Dolan, M.J., 1995, 'A chemically defined liquid medium that supports primary isolation of *Rochalimae* (*Bartonella*) *henselae* from blood and tissue specimens', *Journal of Clinical Microbiology* 33, 742–744. PMid:7538511

Yabsley, M.J., McKibben, J., Macpherson, C.N., Cattan, P.F., Cherry, N.A., Hegarty, B.C. *et al*., 2008, 'Prevalence of *Ehrlichia canis*, *Anaplasma platys*, *Babesia canis vogeli*, *Hepatozoon canis*, *Bartonella vinsonii berkhoffii*, and *Rickettsia* spp. in dogs from Grenada', *Veterinary Parasitology* 151, 279–285. http://dx.doi.org/10.1016/j.vetpar.2007.11.008, PMid:18160223

Ying, B., Kosoy, M.Y., Maupin, G.O., Tsuchiya, K.R. & Gage, K.L., 2002, 'Genetic and ecologic characteristics of *Bartonella* communities in rodents in Southern China', *American Journal of Tropical Medicine and Hygiene* 66(5), 622–627. PMid:12201602

The African buffalo: A villain for inter-species spread of infectious diseases in southern Africa

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How to cite this proceeding:
Michel, A.L. & Bengis, R.G., 2012, 'The African buffalo: A villain for inter-species spread of infectious diseases in southern Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #453, 5 pages. http://dx.doi.org/10.4102/ojvr.v79i2.453

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

The African buffalo (*Syncerus caffer*) is a large wild bovid which until recently ranged across all but the driest parts of sub-Saharan Africa, and their local range being limited to about 20 km from surface water. They are of high ecological value due to their important role as bulk feeders in the grazing hierarchy. They also have high economic value, because they are one of the sought after 'Big Five' in the eco-tourism industry. In Africa, buffaloes have been recognised for some time as an important role player in the maintenance and transmission of a variety of economically important livestock diseases at the wildlife and/or livestock interface. These include African strains of foot-and-mouth disease (FMD), Corridor disease (theileriosis), bovine tuberculosis and bovine brucellosis. For a number of other diseases of veterinary importance, African buffaloes may also serve as amplifier or incidental host, whereby infection with the causative pathogens may cause severe clinical signs such as death or abortion as in the case of anthrax and Rift Valley fever, or remain mild or subclinical for example heartwater. The long term health implications of most of those infections on the buffalo at a population level is usually limited, and they do not pose a threat on the population's survival. Because of their ability to harbour and transmit important diseases to livestock, their sustainable future in ecotourism, trade and transfrontier conservation projects become complex and costly and reliable diagnostic tools are required to monitor these infections in buffalo populations.

Introduction

The transmission and spread of infectious diseases amongst and between domestic and wild animals can occur directly through contact (foot-and-mouth disease [FMD], bovine tuberculosis and brucellosis), or indirectly through the agency of haematophagous arthropod vectors such as mosquitoes, tsetse flies and *Ixodid* ticks (Kock 2005). In addition, environmental contamination with infected ticks (theileriosis) or fomites (anthrax) can infect livestock even when the habitat is not shared at the same time. Some of these diseases can affect a variety of wildlife hosts, but only very few species play a decisive role in disease maintenance and transmission at the wildlife and/or livestock interface. The increasing competition for land available for livestock – based agriculture or wildlife ranching and conservation has highlighted the need for an integrated approach to sustainable livestock and wildlife health, use and management. In this debate the African buffalo features as a villain to those who wish to protect livestock populations from devastating diseases, as they undermine national and international disease eradication schemes, which have been implemented and executed with significant success, and at great cost in the past (Munag'andu *et al.* 2006). On the other hand, conservationists and wildlife ranchers take on a distinctly different attitude towards controlling livestock diseases transmitted by wildlife (Bengis, Kock & Fischer 2002).

In this overview the role of the African buffalo, both as a victim and asymptomatic carrier in the transmission of the most important livestock diseases at the wildlife and/or livestock interface, is reviewed. Links between disease ecology and buffaloes' ecology are examined in an attempt to reveal why buffaloes are successful maintenance hosts for several of those diseases.

Status of African buffaloes

Since the early days of European settlement in southern and eastern Africa, African buffaloes have attracted the admiration of hunters and have consequently been a highly sought after trophy animal for the past three centuries (Prins 1996). In addition, they gained extraordinary value for other ecotourism related purposes during the past decades (Van der Merwe, Saayman & Krugell 2004). The African buffalo (*Syncerus caffer*) is one of the most ubiquitous large herbivores which can tolerate a wide range of climatic conditions and habitats as long as it is provided with access to an abundant supply of water (Grimsdell 1969; Sinclair 1977). Ecologically, the African buffalo is a bulk grazer and occupies an important niche through opening up habitats that are preferred by short-grass grazers (De Vos & Bengis 1994). Currently the conservation status of the African

buffalo is satisfactory with no immediate threat of extinction (Friedmann & Daly 2004).

Infectious diseases transmitted by African buffaloes

African buffaloes have been recognised for some time for various roles in disease transmission amongst wildlife species and at the interface with livestock. Amongst the best known diseases are foot-and-mouth disease, caused by the African strains SAT 1, 2 and 3 (FMD viruses) and Corridor disease (theileriosis, caused by *Theileria parva*), owing to the disastrous, large scale clinical outbreaks they may cause in livestock (Anonymous 2007; Norval, Perry & Young 1992). However, African buffaloes have been implicated in a number of other epidemiological roles involving both indigenous and alien livestock diseases.

Diseases which originated in Africa and co-evolved with African wildlife species including African buffaloes are defined as indigenous. They generally do not pose a threat on the survival of their hosts' populations because of the evolutionary development of unique coping mechanisms. Well known indigenous diseases maintained by African buffaloes include FMD caused by the SAT strain viruses (Thomson 1995), Corridor disease and African trypanosomosis (Anonymous 2007, 2009; De Vos & Bengis 1994; Norval *et al.* 1992).

In contrast, foreign, so-called alien livestock diseases, such as bovine tuberculosis (*Mycobacterium bovis*) and bovine brucellosis (*Brucella abortus*), have been introduced and successfully established themselves in buffaloes which serve as wildlife maintenance host (De Vos *et al.* 2001). Rinderpest falls within the same grouping of alien diseases but its extreme pathogenicity and high mortality precluded the development of a wildlife reservoir (De Vos & Bengis 1994). The devastating effect of rinderpest on many cloven hoofed African ungulate species and African buffaloes (as an epidemic end host) in particular at a local or regional population level was more important ecologically and epidemiologically than their potential role in transmission of the disease to livestock. In 2011, rinderpest was officially eradicated from the world (Morens *et al*. 2011) and therefore its significance for interspecies transmission no longer exists.

Due to their wide geographical range and distribution, African buffaloes have been furthermore implicated as amplifiers or incidental hosts in the epidemiology of a number of other indigenous infectious agents, including those confined to specific localised geographic areas, for example trypanosomes is limited to certain savannah and forested areas endemic for tsetse flies, and buffalo are one of the preferred hosts for these flies and develop a symptomatic carrier state for this parasite (Molooa *et al.* 1999).

Anthrax, caused by *Brucella anthracis* is limited to certain anthrax endemic areas of the African continent (Anonymous 2009). For Rift Valley fever little is known about the precise mechanisms of virus maintenance and transmission. However, buffaloes have been associated with a possible sentinel role during epidemics and a possible maintenance role during interepidemic periods, respectively (Bengis *et al.* 2010; LaBeaud *et al*. 2011).

Factors related to African buffaloes in disease transmission

The epidemiology of infectious diseases is determined by factors related to the host, the environment and the causative pathogen. In the case of African buffaloes a number of intrinsic behavioural characteristics are instrumental to its role in hosting and transmitting livestock diseases. They are a highly social species living in large herds of up to 1000 animals, whose gregarious behaviour provides ideal conditions for direct pathogen transmission via aerosol and body secretions (Grimsdell 1969; Michel *et al.* 2006). Individual or small groups of buffaloes have also been shown to migrate over large distances within short periods of time, either in response to drought or as a result of the dispersal strategies for heifers or bachelor bulls. These events, like fission and fusion events driven by changes in the seasonal availability of grazing, take place on a regular basis and constitute a powerful vehicle for pathogen dispersal across herds (Cross, Lloyd-Smith & Getz 2005; Halley, Vandewalle & Taolo 2002).

Factors related to the pathogen

As regards the chronic, slow alien livestock diseases, such as bovine tuberculosis and bovine brucellosis, the lack of coping mechanisms in naïve African buffaloes may result in deleterious long term effect on certain buffalo populations.

Some pathogens exhibit a very strict and narrow host range, for example *T. parva*, whilst *M. bovis* is capable of causing disease in a very broad spectrum of domestic and wild animal species. Amongst its wildlife hosts, maintenance host potential has been confirmed for African buffaloes and is suspected for greater kudu and possibly others (Michel *et al.* 2006). Indirect transmission of pathogens via environmental contamination is more effective if the organism can survive for significant periods of time outside of its host(s). Whilst the survival of most viruses outside the host is very short, *M. bovis* has been shown to survive for between five days and four weeks in the environment (Tanner & Michel 1999) and the spore forming *Bacillus anthracis* bacterium is ideally equipped for long term (years) survival outside of a host (De Vos 2004).

Factors related to the environment

African buffaloes occur in many different habitats including woodlands, grasslands, swamps, floodplains and thickets, at a range of altitudes, provided they have access to an abundant supply of water and good quality grazing. This species' ability to survive in all but the more arid habitats, allows it to host a variety of pathogens under diverse environmental conditions. For example, the abundance of

the *Aedine* mosquitoes, the endemic vector of the Rift Valley fever virus, is influenced by wet climatic shifts which are frequently associated with increased virus transmission to domestic and wild ruminants (Bengis *et al.* 2010). In addition, African buffaloes spend a considerable percentage of their time in and near water (Ryan, Knechtel & Getz 2006) which further increases their risk of exposure to vectors. In addition, African buffaloes are a preferred host for certain ixodid tick species, the vectors of various protozoal and rickettsial parasites of bovids.

The modern wildlife industry grows at an average annual rate of 5.6% in terms of area exempted for game ranching and is mainly based on ecotourism, hunting and live game trade (Cloete, Taljaard & Grove 2007). This rapid growth rate in conjunction with the historical iconic role of the African buffalo as a member of the 'Big Five' for viewing, photographing and hunting has lead to an increase in the numbers and distribution of buffaloes on private land. In certain parts of South Africa the number of game farms registered for keeping buffaloes has equalled or exceeded that of livestock, resulting in an expansion of the wildlife and/or live stock interface and hence a risk in bi-directional disease transmission. High population densities and frequent translocation of African buffaloes between private game properties for commercial gain are contributing to cumulative risk of African buffalo to contract or transmit livestock diseases. For this reason, in South Africa buffaloes and cattle may not be kept together on shared rangeland. However, pathogens do not generally respect fences.

Against the background of the establishment of large wildlife conservation areas and transfrontier parks in southern Africa, the potential role of buffaloes in the transmission of infectious diseases at the wildlife and/or livestock interface cannot be ignored. High expectations have been linked to the creation of large conservation areas in terms of improved and sustainable livelihoods for the communities in and around those wildlife areas. However, increased numbers of livestock and buffaloes, in the absence of game deterrent fences and often a lack of effective livestock vaccines, will inevitably increase the risk of disease transmission, and make disease eradication from livestock virtually impossible (Kock 2005).

Impact of infectious diseases on African buffaloes and their environment

Amongst the infectious diseases, FMD and Corridor disease and African trypanosomosis are truly asymptomatic in African buffaloes, which act as biological reservoirs of infection in an endemic cycle. The FMD infection occasionally escapes into other sympatric wild cloven-hoofed species, and where livestock are in contact with African buffaloes, they may become infected resulting in devastating outbreaks with a high socio-economic impact (Vosloo *et al.* 2005).

Rift Valley fever, is a seasonal vector born disease and viremia and abortions have been reported in African buffaloes (Evans *et al.* 2008). Their exact role in the epidemiology of Rift Valley fever is however, still unknown but results of serological investigations in Kenya and South Africa indicated that African buffaloes become infected with the virus (Evans *et al.* 2008; LaBeaud *et al.* 2011).

Bovine tuberculosis in African buffaloes has been well documented and can be described as a chronic, progressively debilitating condition (De Vos *et al.* 2001). It has spread throughout the buffalo population of the Kruger National Park and spilled into more than a dozen other wildlife species. There is a continuous risk for spillback into livestock and an associated zoonotic risk for animal owners, which is undetermined (Berg *et al.* 2011; Michel, Müller & Van Helden 2010).

Anthrax is a sporadic, usually fatal disease affecting buffaloes in numerous endemic areas in sub-Saharan Africa (De Vos 1990; Mohan & Gotts 1970). Herbivores are known to be more susceptible to anthrax than omnivores and carnivores, and losses in African buffaloes, greater kudu, nyala and waterbuck have been significant in outbreaks of this disease in southern and eastern Africa (De Vos 2004). When buffaloes are affected by anthrax, they serve as highly effective multipliers of *B. anthracis*, which can contaminate the soil and run-off water, and carcasses become amplifiers for blowflies. Anthrax may be transmitted to neighbouring livestock farms and initiate an outbreak amongst cattle, and the converse has also been experienced when an outbreak in cattle spilled over into wildlife in an adjoining conservation area (Malilangwe – Zimbabwe) (De Vos 1990).

It has been speculated that African buffaloes are the species with the highest susceptibility to rinderpest, which decimated the buffalo populations all over Africa during the rinderpest epidemic between 1888 and 1899 (De Vos & Bengis 1994).

Diagnosis and control of infectious diseases in African buffaloes

Availability of diagnostic tests for wildlife species is very often the biggest limitation in diagnosing infectious diseases in target wildlife species which are taxonomically far removed from the closest livestock counterparts. The African buffalo falls into the subfamily Bovinae together with domestic cattle, which is helpful and provides a basis for adapting and developing diagnostic tests for relevant infections in buffaloes. However, it needs to be emphasised that all tests applied in buffaloes still require validation to prove *fitness for purpose* as stated by the World Organisation for Animal Health (OIE) (OIE 2009). The question whether infectious diseases in buffaloes require controlling is a contentious one and is best discussed per disease grouping. Since the causative agents of certain indigenous diseases, namely FMD viruses, trypanosomes and theilerias, are widely maintained by African buffaloes and generally do not pose a threat to these population's existence, diagnostic testing is usually applied for monitoring and surveillance activities at population level. For movement purposes and international

trade, the same tests (including direct pathogen detection for Corridor disease and trypanosomosis) are applied at individual animal level in conjunction with herd testing records. It is accepted that FMD can at best be contained in wildlife areas that have buffaloes, but eradication will not be achievable (Vosloo *et al.* 2005).

The situation is essentially the same for indigenous diseases detected less frequently in African buffaloes, such as Rift Valley fever and anthrax, where outbreaks are monitored strictly for epidemiological information and analyses.

A very different scenario is presented in the cases of bovine tuberculosis and bovine brucellosis, which are both alien diseases introduced into the buffalo population most likely through contact with infected cattle. Following their successful establishment in African buffaloes as their wildlife reservoir or maintenance host, the state veterinary and conservation authorities have been faced with a set of previously unknown challenges. Apart from the need to prevent spillover of the diseases to neighbouring livestock, these diseases may spread uncontrolled within infected conservation areas. Although our current knowledge on the impact of bovine tuberculosis and bovine brucellosis does not indicate an immediate threat on buffalo survival at the population level, it would be premature to make any prediction about the long term impact at this point in time. As no effective vaccine exists for bovine tuberculosis in animals and given the extremely broad host spectrum of the causative organism, the real threat goes beyond buffalo conservation but concerns severely affected sympatric species such as lions and kudus (Michel *et al.* 2009). It is fair to speculate that African buffaloes will not remain the only maintenance host in an endemically infected ecosystem such as the Kruger National Park and disease management and control strategies are urgently called for. Two effective vaccines are registered for the control of bovine brucellosis in cattle, but their efficacy and safety in African buffaloes has not been tested, as the disease is currently considered to have little effect on African buffaloes and the ecosystem (Godfroid 2004). Pre-movement testing of buffaloes for both bovine tuberculosis and brucellosis is an absolute requirement to minimise the risk of disease transmission to uninfected populations. In the case of bovine tuberculosis serological tests are not useful to detect infection and the intradermal tuberculin test (skin test) is applied in conjunction with the interferon gamma assay (Michel *et al.* 2011; Michel & Simoes 2009).

Conclusion

It is clear that disease transmission at the interface is bi-directional and African buffaloes are culprits or villains in respect of some diseases, whilst they have fallen victim to alien diseases transmitted from cattle. As the wildlife and/or livestock and/or human interface is rapidly expanding and gaining in intensity and complexity, there should be no room for a blaming game, but it must be appreciated that African buffaloes form an integral part of the indigenous fauna of Africa and are inseparable from their indigenous pathogens. A strictly separate approach is needed for the alien diseases which may directly affect the health of buffaloes or indirectly the conservation of wildlife by turning infected areas into 'conservation islands' and making them unavailable for conservation in the bigger sense.

To enable ecologists, veterinary researchers and conservation biologists to correctly assess and predict the long term dynamics of significant livestock diseases at this growing and intensifying disease interface, an understanding of the host-parasite interactions at population level in cattle and African buffaloes is crucial to finding solutions and to attempt achieve compatibility between traditional livestock farming and wildlife conservation-based ecotourism. It should also be emphasised that host-parasite interactions are not limited to the processes guiding infection and immunopathogenesis, but they must include the external determinants of the environment, including climate change, human interventions and other ecosystem drivers.

References

Anonymous, 2007, 'Institutional strategies and economic impacts of alternative FMD prevention and control strategies in Southern Africa', in B.D. Perry & K.R. Sones (eds.), *International Livestock Research Institute*, p. 56, Nairobi, Kenya.

Anonymous, 2009, *'African animal trypanosomiasis'*, Center for Food Security and Public Health, Iowa State University, Iowa.

Bengis, R.G., Kock, R.A. & Fischer, J., 2002, 'Infectious animal diseases: The wildlife/livestock interface', *Revue Scientifique et Technique* 21, 53–65.

Bengis, R.G., Swanepoel, R., De Klerk, L-M., Pienaar, N.J. & Prinsloo, G., 2010, 'Rift Valley fever: Current concepts and recent findings', paper presented at the 9th Annual Conference of the 'Southern African Society for Veterinary Epidemiology and Preventive Medicine', The Farm Inn, Pretoria, South Africa, 18–20 August.

Berg, S., Garcia-Pelayo, M., Müller, B., Hailu, E., Asiimwe, B., Kremer, K. *et al.*, 2011, 'African 2, a clonal complex of *Mycobacterium bovis* epidemiologically important in East Africa', *Journal of Bacteriology* 193, 670–678. http://dx.doi.org/10.1128/JB.00750-10, PMid:21097608

Cloete, P.C., Taljaard, P.R. & Grove, B., 2007, 'A comparative economic case study of switching from cattle farming to game ranching in the Northern Cape Province', *South African Journal of Wildlife Research* 37, 71–78. http://dx.doi.org/10.3957/0379-4369-37.1.71

Cross, P.C., Lloyd-Smith, J.O. & Getz, W.M., 2005, 'Disentangling association patterns in fission-fusion societies using African buffalo as an example', *Animal Behaviour* 69, 499–506. http://dx.doi.org/10.1016/j.anbehav.2004.08.006

De Vos, V., 2004, 'Anthrax', in J.A.W Coetzer & R.C Tustin (eds.), *Infectious diseases of livestock*, n.p., Oxford University Press, Cape Town.

De Vos, V., 1990, 'The ecology of anthrax in the Kruger National Park, South Africa', *Salisbury Medical Bulletin* 68, 19–23.

De Vos, V., Bengis, R.G., Kriek, N.P., Michel, A., Keet, D.F., Raath, J.P. & Huchzermeyer, H.F., 2001, 'The epidemiology of tuberculosis in free-ranging African buffalo (*Syncerus caffer*) in the Kruger National Park, South Africa', *Onderstepoort Journal of Veterinary Research* 68, 119–130. PMid:11585089

De Vos, V. & Bengis, R.G., 1994, 'The disease status of African buffalo in South Africa', in W. Van Hoven, H. Ebedes & A. Conroy (eds.), *Wildlife ranching: A celebration of diversity Proceedings of the 3rd International Wildlife Ranching Symposium*, Centre for Wildlife Management, University of Pretoria, Pretoria, October, 1992, n.p.

Evans, A., Gakuya, F., Paweska, J.T., Rostal, M., Akoolo, L., Vuren, P.J. *et al.*, 2008, 'Prevalence of antibodies against Rift Valley fever virus in Kenyan wildlife', *Epidemiology and Infection* 136, 1261–1269. http://dx.doi.org/10.1017/S0950268807009806

Friedmann, Y. & Daly, B., 2004, *Red Data book of mammals of South Africa: A conservation assessment*, Conservation Breeding Specialist Group Southern Africa, South Africa.

Godfroid, J., 2004, 'Brucellosis in wildlife', in J.A.W. Coetzer & R.C. Tustin (eds.), *Infectious Diseases of Livestock*, Oxford University Press.

Grimsdell, J.J.R., 1969, 'Ecology of the buffalo, *Syncerus caffer* in western Uganda', PhD thesis, Cambridge University.

Halley, D.J., Vandewalle, M.E.J. & Taolo, C., 2002, 'Herd-switching and long-distance dispersal in female African buffalo (*Syncerus caffer*)', *African Journal of Ecology* 40, 97–99. http://dx.doi.org/10.1046/j.0141-6707.2001.00336.x

Kock, R.A., 2005, 'What is the infamous wildlife/livestock disease interface? A review of current knowledge', Southern and East African Experts Panel on Designing Successful Conservation and Development Interventions at the Wildlife/Livestock Interface: Implications for Wildlife, Livestock, and Human Health. AHEAD Animal Health for the Environment And Development Forum. IUCN/SSC Veterinary Specialist Group, Southern Africa Sustainable Use Specialist Group *et al*., IUCN, Gland, Switzerland and Cambridge, UK, 14–15 September 2003.

LaBeaud, A.D., Cross, P.C., Getz, W.M., Glinka, A. & King, C.H., 2011, 'Rift Valley fever virus infection in African buffalo (*Syncerus caffer*) herds in rural South Africa: Evidence of interepidemic transmission', *American Journal of Tropical Medicine and Hygiene* 84, 641–646. http://dx.doi.org/10.4269/ajtmh.2011.10-0187, PMid:21460024

Michel, A.L., Bengis, R.G., Keet, D.F., Hofmeyr, M., De Klerk, L.-M., Cross, P.C. *et al*., 2006, 'Wildlife tuberculosis in South African conservation areas: Implications and challenges', *Veterinary Microbiology* 112, 91–100. http://dx.doi.org/10.1016/j.vetmic.2005.11.035, PMid:16343819

Michel, A.L., Coetzee, M.L., Keet, D.F., Mare, L., Warren, R., Cooper, D. *et al*., 2009, 'Molecular epidemiology of *Mycobacterium bovis* isolates from free-ranging wildlife in South African game reserves', *Veterinary Microbiology* 133, 335–343. http://dx.doi.org/10.1016/j.vetmic.2008.07.023, PMid:18786785

Michel, A.L., Cooper, D., Jooste, J., De Klerk, L.-M. & Jolles, A., 2011, 'Approaches towards optimising the gamma interferon assay for diagnosing *Mycobacterium bovis* infection in African buffalo (*Syncerus caffer*)', *Preventive Veterinary Medicine* 98, 142–151. http://dx.doi.org/10.1016/j.prevetmed.2010.10.016, PMid:21122932

Michel, A.L., Müller, B. & Van Helden, P.D., 2010, *'Mycobacterium bovis* at the animal-human interface: a problem, or not?', *Veterinary Microbiology* 140, 371–381. http://dx.doi.org/10.1016/j.vetmic.2009.08.029, PMid:19773134

Michel, A.L. & Simoes, M., 2009, 'Comparative field evaluation of two rapid immunochromatographic tests for the diagnosis of bovine tuberculosis in African buffaloes (*Syncerus caffer*)', *Veterinary Immunology and Immunopathology* 127, 186–189. http://dx.doi.org/10.1016/j.vetimm.2008.09.025, PMid:19007999

Mohan, R.N. & Gotts, M.G., 1970, 'Diseases and parasites of the African buffalo (*Syncerus caffer*)', *Veterinary Bulletin* 40, 157–166.

Molooa, S.K., Orindab, G.O., Sabwaa, C.L., Minjaa, S.H. & Masakea, R.A., 1999, 'Study on the sequential tsetse-transmitted *Trypanosoma congolense, T. brucei brucei* and *T. vivax* infections to African buffalo, eland, waterbuck, N'Dama and Boran cattle', *Veterinary Parasitology* 80, 197–213. PMid:9950344

Morens, D.M., Holmes, E.C., Davis, A.S. & Taubenberger, J.K., 2011, 'Global rinderpest eradication: lessons learned and why humans should celebrate too', *Journal of Infectious Diseases* 204, 502–505. http://dx.doi.org/10.1093/infdis/jir327, PMid:21653230

Munag'andu, H.M., Siamudaala, V.M., Nambota, A., Bwalya, J.M., Munyeme, M., Mweene, A.S. *et al*., 2006, 'Disease constraints for utilization of the African buffalo (*Syncerus caffer*) on game ranches in Zambia', *Japanese Journal of Veterinary Research* 54, 3–13. PMid:16786973

Norval, R.A.I., Perry, B.D. & Young, A.S. (eds.), 1992, *The epidemiology of theileriosis in Africa*, American Press, London.

OIE, 2009, 'Standard Operating Procedure (SOP) for OIE Validation and Certification of Diagnostic Assays', Version 2.1.

Prins, H.H.T., 1996, 'Ecology and behaviour of the African buffalo - Social inequality and decision making', PhD thesis, Chapman & Hall, London.

Ryan, S.J., Knechtel, C.U. & Getz, W.M., 2006, 'Range and habitat selection of African buffalo in South Africa', *Journal of Wildlife Management* 70, 764–776. http://dx.doi.org/10.2193/0022-541X(2006)70[764:RAHSOA]2.0.CO;2

Sinclair, A.R.E., 1977, 'The African buffalo - a study of resource limitations of populations', PhD thesis, University of Chicago Press.

Tanner, M. & Michel, A.L., 1999, 'Investigation of the viability of *Mycobacterium bovis* under different environmental conditions in the Kruger National Park', *Onderstepoort Journal of Veterinary Research* 66, 185–190. PMid:10631708

Thomson, G.R., 1995, 'Overview of foot and mouth disease in southern Africa', *Revue Scientifique et Technique* 14, 503–520. PMid:8593386

Van der Merwe, P., Saayman, M. & Krugell, W., 2004, 'Factors that determine the price of game', *Koedoe* 47, 105–113.

Vosloo, W., Bastos, A.D.S., Sahle, M., Sangare, O. & Dwarka, R.M., 2005, 'Virus topotypes and the role of wildlife in foot and mouth disease in Africa', Southern and East African Experts Panel on Designing Successful Conservation and Development Interventions at the Wildlife/Livestock Interface: Implications for Wildlife, Livestock, and Human Health. AHEAD Animal Health for the Environment And Development, Forum. IUCN/SSC Veterinary Specialist Group, Southern Africa Sustainable Use Specialist Group *et al*., IUCN, Gland, Switzerland and Cambridge, UK, 14–15 September 2003.

Towards One Health disease surveillance: The Southern African Centre for Infectious Disease Surveillance approach

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How to cite this proceeding:
Karimuribo, E.D., Sayalel, K., Beda, E., Short, N., Wambura, P., Mboera, L.G. *et al*., 2012, 'Towards One Health disease surveillance: The Southern African Centre for Infectious Disease Surveillance approach', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #454, 7 pages. http://dx.doi.org/10.4102/ojvr.v79i2.454

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Africa has the highest burden of infectious diseases in the world and yet the least capacity for its risk management. It has therefore become increasingly important to search for 'fit-for-purpose' approaches to infectious disease surveillance and thereby targeted disease control. The fact that the majority of human infectious diseases are originally of animal origin means we have to consider One Health (OH) approaches which require inter-sectoral collaboration for custom-made infectious disease surveillance in the endemic settings of Africa. A baseline survey was conducted to assess the current status and performance of human and animal health surveillance systems and subsequently a strategy towards OH surveillance system was developed. The strategy focused on assessing the combination of participatory epidemiological approaches and the deployment of mobile technologies to enhance the effectiveness of disease alerts and surveillance at the point of occurrence, which often lies in remote areas. We selected three study sites, namely the Ngorongoro, Kagera River basin and Zambezi River basin ecosystems. We have piloted and introduced the next-generation Android mobile phones running the EpiCollect application developed by Imperial College to aid geo-spatial and clinical data capture and transmission of this data from the field to the remote Information Technology (IT) servers at the research hubs for storage, analysis, feedback and reporting. We expect that the combination of participatory epidemiology and technology will significantly improve OH disease surveillance in southern Africa.

Introduction

Although there have been some recent advances in the diagnosis and management of human infectious diseases, they still are a significant impact and burden on global economies and public health (Jones *et al.* 2008). Infectious diseases are responsible for a quarter of all human deaths worldwide (King *et al.* 2006). Most of these are as a result of emerging infectious diseases (EIDs), defined as infections that have newly appeared in a population or have existed but are rapidly increasing in incidence or geographic range (Morse 1995). There are various drivers for the occurrence of EIDs including socio-economic, environmental and ecological factors. Analysis of origins of EIDs for longer than six decades concluded that over 60% are zoonotic of which about 72% originate in wild animals (Jones *et al*. 1978). It has also been observed that when considering spatial distribution of origins of infectious diseases, the majority are prevalent in and affect developing countries in the tropics, particularly in Africa and South-East Asia.

Effective disease surveillance is required to ensure freedom from EIDs or else timely intervention in order to reduce risks and impact on animal and human populations. Disease surveillance is commonly defined as an ongoing systematic collection, analysis and interpretation of data essential to the planning, implementation and evaluation of disease management practice, closely integrated with the timely dissemination of these data to those who need to know (Mboera, Rumisha & Kitua 2001; Thacker & Berkelman 1988). Although the purpose and objectives of disease surveillance may differ between different health sectors, it is generally agreed that surveillance is useful for rapid detection of new and/or foreign diseases, provides evidence of freedom from diseases within a defined geographic area or population, accurately delineates the distribution and occurrence of diseases relevant to disease control and provides evidence required to assess progress and success of disease control or rededication (FAO 2004).

Most developing countries have limited disease surveillance capacity and so need to ensure optimal use of available resources. However, previous studies indicated that both animal and human health sectors are poorly resourced in terms of both clinical and public health service provision (James & Muchiri 2006; Jones 2011; Karimuribo *et al.* 2011). Considering that most emerging infectious disease conditions in public health sectors, especially in tropical countries, are of animal (wildlife and domestic) origin (Jones *et al*. 2008; Wolfe, Dunavan & Diamond 2007),

it is important to encourage collaboration between the animal and human health sectors in order to minimise risks associated with such infections. The purpose of this work is to share findings based on our experience in southern Africa focusing on the OH surveillance strategy developed by the Southern African Centre for Infectious Disease Surveillance (SACIDS).

Materials and methods

One Health surveillance

In this paper, One Health (OH) surveillance is defined as collaborative efforts between the human and animal (wildlife and domestic) health sectors to conduct systematic collection of disease event data, analysis of this data and a timely dissemination of surveillance results to guide interventions aimed to prevent or control disease in human and animal populations.

Study design

This study involved two phases. Phase 1 involved conducting a baseline survey to assess the performance of current disease surveillance in animal and human health sectors. Lessons learnt from these exercises were used to improve the design of the OH surveillance system. The second phase involved designing an OH surveillance system suitable for the southern African region.

Baseline study on assessment of surveillance systems

A rapid situation analysis was carried out in May 2010 to understand the structure and requirement of surveillance systems in the animal and human health sectors in Tanzania. This was followed by assessing the performance of the disease surveillance systems in the two health sectors by visiting animal and human health facilities in Ngorongoro district, carried out in November 2010. During field visits, individuals responsible for detection and reporting of disease events were interviewed using a structured questionnaire. A total number of 14 wards of Ngorongoro district were visited where all resident ward livestock field officers (LFO) were interviewed. The study also collected data from 13 health facilities, representing approximately 62% of all health facilities in Ngorongoro district. The exclusion of an individual health facility was based on lack of access to key personnel or disease surveillance records during the field visit. Respondents were asked to specify frequency of collection and submission of disease surveillance reports, diseases reported and constraints and challenges faced during disease surveillance in animal and human health sectors. Field data collection was complemented by visiting the District Veterinary Office (DVO) and District Medical Office (DMO) at the Ngorongoro District Council headquarters where disease surveillance reports submitted for the period from 2005 to 2010 were retrieved and analysed.

The assessment of disease surveillance in both animal and human health sectors was based on measuring the completeness and accuracy of surveillance reports focusing on a period of five years (2005–2010) before the study. Other factors that might have influenced the timely submission of disease reports such as the distance from a health facility to the district headquarters were also recorded.

Design of Southern African Centre for Infectious Disease Surveillance One Health surveillance strategy in southern Africa

A participatory approach was used at an initial project inception workshop organised to agree on study sites and surveillance approaches. During the workshop, held in January 2010, it was agreed to develop and pilot OH surveillance in three ecosystems namely Ngorongoro, Kagera River basin and Zambezi River basin. The workshop recommended initial pilot activities to be carried out in one ecosystem (Ngorongoro) and then up-scaling the approach to the other two project sites.

Implementation of the project activities in Ngorongoro ecosystems required a collaborative approach coordinated by SACIDS and National Centre for Infectious Disease Surveillance (NatCIDS). Other key collaborators in the development of the OH surveillance strategy in Tanzania were: the Ministry of Health and Social Welfare (Epidemiology unit), the Ministry of Livestock and Fisheries Development (Epidemiology section) and two academic institutions responsible for veterinary (Sokoine University of Agriculture) and medical (Muhimbili University of Health and Allied Sciences) training in the country. The project also partnered with collaborators from UK, namely the Royal Veterinary College (RVC) and Imperial College London, who assisted in sharing with the southern colleagues their experiences in infectious disease surveillance and the use of appropriate technologies and tools to support OH disease surveillance. Recommendations of the UK Foresight report on infectious diseases (Brownlie *et al.* 2006) were also considered whilst devising OH surveillance strategy in southern Africa.

Study sites

The current project on OH disease surveillance adopted three study sites namely Ngorongoro, Kagera River basin and Zambezi River basin ecosystems. The first ecosystem represents an area of maximum human-wildlife-domestic animal interactions. The ecosystem is located in a remote area in the Arusha region which borders Kenya in the north and the Tanzanian Serengeti ecosystem in the west. The ecosystem is predominantly inhabited by the Maasai pastoral communities who keep cattle, goats and sheep and are in close proximity with wild animals in the wildlife protected areas of Ngorongoro Conservation Area (NCA). The other two sites (Kagera River basin and Zambezi River basins) represent cross-border ecosystems where OH surveillance could be potentially effective in diagnosing and managing infectious diseases across borders. Kagera River basin is an ecosystem located in the Great Lake Region of eastern Africa which links Uganda, Rwanda, Burundi and Tanzania. The ecosystem has a relatively high incidence of communicable

diseases with cross-border spread and is considered to be a high risk potential entry-point for haemorrhagic fever (especially Ebola and Marburg) which occur in the Democratic Republic of Congo (DRC). Zambezi River basin is located in the southern Africa and is the fourth largest basin in Africa after the Congo, Nile and Niger River basins. The Zambezi River has its source in Western Province of Zambia and flows through eastern Angola, Namibia (Eastern Caprivi strip), northern Botswana and through Victoria falls (shared between Zambia and Zimbabwe) before entering Lake Kariba. The project site focused on two districts of Zambia (Kazungula and Sesheke) which share international borders with Zimbabwe, Botswana, Namibia and Angola in the Zambezi River basin ecosystem.

Data analysis

Data collected during baseline study on assessment of the performance of disease surveillance systems were summarised and where applicable, descriptive statistics computed. Data trends by ward and health facilities were demonstrated using graphical presentation of the results. The names of health facilities were coded to ensure a blind contribution in disease reporting to comply with ethical clearance conditions.

Results

Baseline study on the performance of surveillance system in the animal and human health sector in Tanzania

Disease surveillance structure

The surveillance structure between the animal and human health sectors in Tanzania was found to be similar (Table 1). The initial detection of disease events in both health sectors starts in the communities where sick individuals are detected by community-based reporters. The current systems use official cadres who are the LFOs (in animal health sectors) and the health facility in-charge or Integrated Disease Surveillance and Response (IDSR) focal person (in human health sectors) to prepare and submit disease surveillance reports to the higher levels. The central coordinating level for disease surveillance and response is at the district level (DVO and DMO). The two offices are responsible for transmitting reports to higher authorities through the intermediate (zonal VICs and RMO) or sometimes directly to the central level in the ministry responsible for animal health and human health, respectively. The similarities in surveillance structures of the two health sectors offer opportunities for increased collaborations between veterinary and medical professionals with regards to disease surveillance and response.

With respect to frequency of reporting, the animal health sector demands submission of disease reports on a monthly basis using field and abattoir surveillance reports. In the case of notifiable diseases, the officials are required to report disease events immediately. Under the IDSR system, officials are required to report diseases under surveillance on weekly (epidemic-prone conditions) and monthly (epidemic-prone, diseases of public health importance and those targeted for eradication) basis.

Performance of surveillance systems

A total number of 13 human health facilities (dispensaries, health centres and hospitals) were visited and participated in this study. The facilities were located between 1 km and 237 km from the Ngorongoro District Council headquarters (Table 2). More than 69% of these facilities are owned by the local government authority, the Ngorongoro District Council. It was also found that organisations such as the Roman Catholic and the Evangelical Lutheran Churches of Tanzania run some health facilities that, in addition to offering health services, are involved in the capture and reporting of disease events in the study area.

The performance of disease surveillance in animal and human health sectors, defined by the number of surveillance reports received every month is shown in Figure 1. Overall, the reporting was better in the human health sector than in the animal health sector. There were some years such as 2008 and 2009 when the DVO did not receive a single disease report. Similarly the DMO did not receive monthly reports from human health facilities throughout 2005. Generally, some wards and health facilities performed better in submitting disease surveillance reports compared to others (Figures 2a and Figure 2b). It was clear that those wards and facilities close to the District Council headquarters submitted reports more regularly than the distant wards and facilities.

One Health surveillance strategy in southern Africa

The OH surveillance strategy was developed as a result of a participatory and consultative process summarised in Table 3.

TABLE 1: Similarities between disease surveillance structure in animal and human health sectors in Tanzania.

Level	Sector	
	Animal health	Human health
Peripheral (Community)	Farmers and community-based animal health workers	Community based health workers or community-owned resource persons
Peripheral (Village and wards)	Livestock Field Officers and Ward Agriculture Extension Officers	In-charge or focal points for IDSR at health facilities (dispensaries, health centres or hospitals)
Intermediate (District)	District Veterinary Officers or District Agriculture and Livestock Development Officers	District Medical Officers
Intermediate (Region or zone)	Zonal veterinary investigation centres	Regional Medical Officers
Central (Ministry)	National epidemiology section, Ministry of Livestock Development and Fisheries	Epidemiology and disease control section, Ministry of Health and Social Welfare
Regional or international	Regional or international bodies (e.g. AU/IBAR, SADC, EAC, OIE)	Regional or international bodies (e.g. EAC, WHO)

IDSR, Integrated Diseases Surveillance and Response strategy.

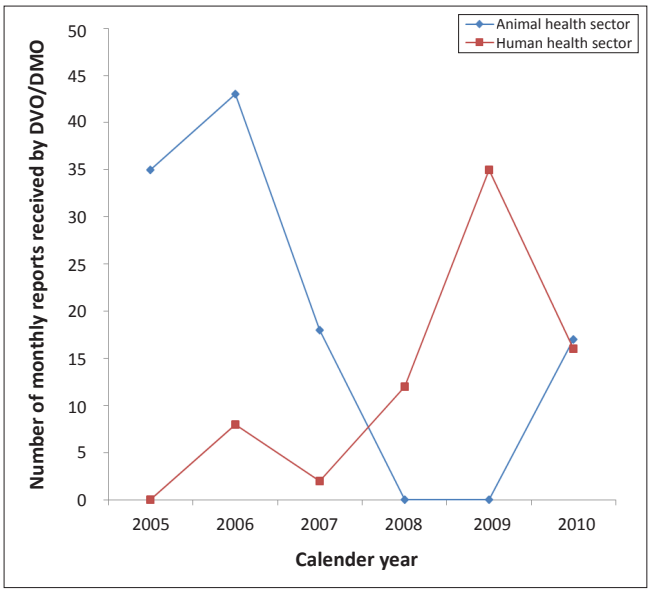
Before project implementation, applications were submitted to the appropriate bodies responsible for ethical clearance in the National Institute for Medical Research (NIMR) and the Tanzania Wildlife Research Institute (TAWIRI). Holding an inception workshop for key stakeholders interested in OH surveillance was helpful in selecting study sites as well as identification of appropriate mobile technologies and tools to assist surveillance. The subsequent meetings of the NatCIDS and the Joint Technical Committee (JTC) in August and September 2010 respectively, defined the OH surveillance strategy to be adopted in Ngorongoro and later in two other project sites. The meetings agreed that the strategy should consist of two complementing systems namely:

- Community-based Active Surveillance (CAS) system which was designed to actively capture disease events in animal and human populations using simple case definitions of symptoms and syndromes occurring in communities. It was also agreed the CAS system would use community-based health reporters who would actively screen for the occurrence of disease events in human, wildlife and domestic animal populations. Data on these events would be recorded and transmitted through Android mobile phones using the Epicollect data capture application in near to real time.
- District-based Passive Surveillance (DPS) system uses existing surveillance strategies in animal and human (IDSR) health sectors with enhanced performance through application of mobile technologies in transmission of near to real time data in the two health sectors.

Collaborating with other institutions in the United Kingdom (Royal Veterinary College and Imperial College London) as well as those in South-East Asia (BIOPHICS, Ministry of Public Health Thailand, MBDS and InsTEDD, Cambodia) assisted in the improvement of the OH surveillance system developed by SACIDS. The two systems (CAS and DPS) are linked together at the data analysis point. Data collected through CAS and DPS systems from pilot sites located in Tanzania are stored centrally on a server located at SACIDS headquarters. Southern African Centre for Infectious Disease Surveillance acts as a custodian and stores data on behalf of the Ministry of Livestock and Fisheries Development and the Ministry of Health and Social Welfare who own the data. At SACIDS, data are analysed and summarised as reports that are shared with the two ministries and field-based disease management units at district headquarters. A similar model is proposed for dealing with handling data collected in Zambezi River basin when data storage and analysis is expected to be the role of the University of Zambia Veterinary School (UNZA Vet) on behalf of respective ministries responsible for animal and human health. There is still on-going discussion on an appropriate model to adopt for collecting, storage and analysis of data from the Kagera River basin which is under the auspices of the East African Community (EAC).

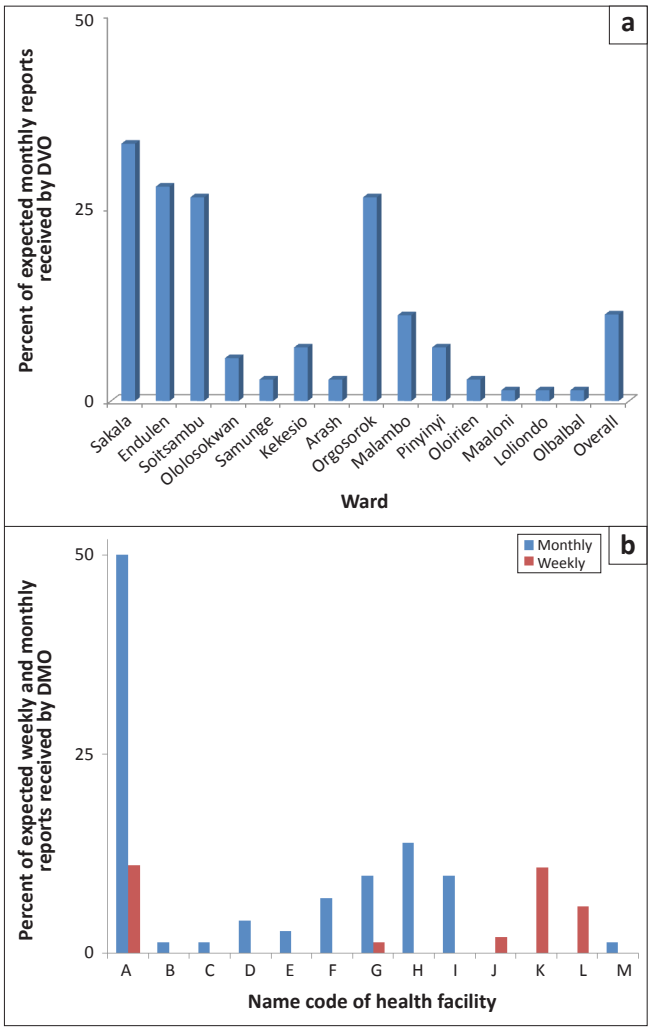
Discussion

Similarities in the current disease surveillance structure in animal and human health sectors provide opportunities for



DVO, District Veterinary Office; DMO, District Medical Office.

FIGURE 1: Efficiency of monthly disease reporting in the animal and human health sectors in Ngorongoro district from 2005 to 2010.



DVO, District Veterinary Office; DMO, District Medical Office.

FIGURE 2: Completeness of disease reporting in the (a) animal and (b) human health sector.

TABLE 2: Health facilities and wards visited during assessment of surveillance systems in Ngorongoro district.

Name of facility	Category	Owner	Location, ward	Catchment area population	Distance to the district headquarters, km
Arash	Dispensary	Faith-based Organisation	Arash	4291	52
Endulen	Hospital	Faith-based Organisation	Endulen	35 168	166
Kakesio	Dispensary	District Council	Kakesio	3945	237
Loliondo	Health centre	District Council	Orgosorok	5569	5
Malambo	Health centre	District Council	Malambo	8816	92
Nainokanoka	Dispensary	District Council	Nainokanoka	29 682	226
NCAA	Dispensary	Parastatal Organisation	Ngorongoro	4977	178
Ngarasero	Dispensary	District Council	Pinyinyi	1360	122
Oldonyosambu	Dispensary	District Council	Oldonyosambu	3927	93
Sakala	Dispensary	District Council	Sakala	1455	3
Samunge	Dispensary	District Council	Digodigo	5463	65
Sero	Dispensary	District Council	Soitsambu	2805	43
Wasso DDH	Hospital	Faith-based Organisation	Orgosorok	37 345	1.5

TABLE 3: Timeline of events that contributed to the development of Southern African Centre for Infectious Disease Surveillance One Health surveillance strategy in southern Africa.

Date	Event	Actors†	Achievement
January 2010	Inception workshop held at Ngurdoto Mountain Lodge, Arusha Tanzania	Representatives from DRC, Rwanda, Tanzania, Zambia and UK. Also attended by institutions involved in surveillance such as InstEDD, TRACNET Rwanda, P4H, FAO DPT, Imperial College London-EpiCollect, RVC	Agreed on 3 pilot sites and technologies to enhance OH surveillance in southern Africa
July 2010	Discussion with EAC Health desk on project implementation in Kagera river basin	Representatives from SACIDS and EAC-EAIDSNet	Agreed on project coordination arrangements
August 2010	In-country meeting to discuss project implementation in Ngorongoro	NatCIDS members from MUHAS, NIMR, SUA, MoHSW and MoLFD	Appointed a JTC to supervise project implementation
September 2010	JTC meeting	Members of JTC from SACIDS, SUA, MUHAS, NIMR, MoHSW and MoLFD	Defined and prioritised diseases and syndromes to focus on under OH surveillance. Agreed pilo OH surveillance data to be stored at SACIDS server on behalf of MoHSW and MoLFD
September–October 2010	Visiting UK by SACIDS Postdoc and ICT specialist	Members from SACIDS, RVC, Imperial College London and IoE	Received inputs on how to improve OH surveillance in Africa and use of technology (Android phones powered by Epicollect) to enhance OH surveillance. Hands on practice on use of the Android phones or Epicollect
November 2010	Setting up data storage system at SACIDS and field-testing of the Android phones-Epicollect in Tanzania	Team members from SACIDS and Imperial College London	Developed local capacity to use Epicollect and also assessed performance of Android phones-Epicollect in Ngorongoro
January–February 2011	Development of OH surveillance guidelines	SACIDS team at SUA	First draft of the surveillance guidelines developed
February 2011	Visiting South-East Asia	SACIDS team (Postdoc fellow & ICT specialist) visited Thailand (BIOPHICS, Epidemiology Bureau of the Ministry of Public Health & MBDS) and Cambodia (InsTEDD)	Learnt on opportunities and challenges with respect to surveillance and technologies used in South-East Asia
March 2011	Discussion on project implementation in Zambezi river basin	SACIDS team and NatCIDS Zambia team at UNZA Vet	Agreed on project implementation in Zambezi river basin ecosystem
March 2011	JTC meeting	Members of JTC from SACIDS, SUA, MUHAS, NIMR, MoHSW and MoLFD	Improved OH surveillance guidelines
June 2011	Exploratory visit, Kagera river basin	SACIDS team and EAC	Discussed and agreed on how to launch project activities in Kagera river basin ecosystem
June–July 2011	Procurement of Android phones and server	SACIDS & RVC	Agreed on procurement plan to initiate OH surveillance in southern Africa

JTC, Joint Technical Committee; EAC, East African Community; SACIDS, Southern African Centre for Infectious Disease Surveillance; ICT, information and communications technology; OH, One Health; DRC, Democratic Republic of the Congo.
†, Different institutions from Tanzania (MUHAS, Muhimbili University of Health and Allied Sciences; NIMR, National Institute for Medical Research; MoHSW, Ministry of Health and Social Welfare; MoLFD, Ministry of Livestock and Fisheries Development; SUA, Sokoine University of Agriculture; SACIDS, Southern African Centre for Infectious Disease Surveillance headquartered at SUA and its national chapter, NatCIDS), Zambia (UNZA Vet, University of Zambia Veterinary School), UK (RVC, Royal Veterinary College) and the East African Community (EAC-EAIDSNet, East Africa Integrated Disease Surveillance Network) were involved.

collaboration between the two sectors. For instance, under the current IDSR strategy, emphasis on disease management is placed in hands of district health facility levels (Franco, Setzer & Banke 2006). Similarly, the MoLFD demands the DVO to be in-charge of managing disease epidemics in animal populations. As both the DMO and DVO work under the umbrella of the District Executive Officer, it is logical to work together in the management of disease epidemics in animal and human populations in their respective districts. This has been happened in some instances during Rift Valley fever and anthrax outbreaks in Ngorongoro district between 2006 and 2009 (B.M. Miran, pers. comm., 2010). It was also found that sometimes animal and human health officials in Ngorongoro district do share vaccine storage facilities during surge demand of resources. This usually happens during disease vaccination campaigns when teams of vaccinators camping in remote areas require storage facilities for proper handling of vaccines. This experience is not new to resource-challenged remote areas as reported in other countries where sharing resources such as transport logistics and equipment

reduces costs (Schelling *et al.* 2007). The current OH strategy designed by SACIDS where one person (community-based health reporter) actively searches for occurrence of disease events is another good example of optimising the use of limited resources.

Findings of the baseline survey agree with previous findings of poor performance of disease surveillance in animal and human health sectors in Tanzania (Allport *et al.* 2005; Mboera *et al.* 2001). The situation is made worse with the delayed reporting of sick individuals at health facilities where disease events are normally captured. A study by Shayo *et al*. (2003) indicated that the majority of rural-based individuals stay at home or consult traditional healers before visiting health facilities to seek medical services. Similar findings have been reported in the animal health sectors where sick animals are usually managed by farmers or community-based animal health workers in remote areas before intervention of veterinarians (Karimuribo & Swai 2006).

The baseline study also reported a significant variation in completeness of surveillance report in both animal and human health sectors. Although some wards and health facilities seem to do better than others, the overall picture confirms poor surveillance coverage. In-depth interviews of district officials responsible for animal and human health confirmed that the lack of human resources to supervise and manage surveillance systems may seriously affect the performance of the system. This is confirmed, for example, by at the fact that there was no DVO between 2006 and 2009 in Ngorongoro when there was sharp decline in the number of surveillance reports submitted to the district headquarters. This situation was reversed in 2010 after recruiting a veterinarian to head the veterinary section in the district. Other examples were noted where poor reporting was associated with times when key IDSR staff responsible for submitting weekly or monthly reports were away from their work stations. This experience had also been reported previously by Rumisha *et al*. (2007) when poor disease reporting under IDSR was attributed to staff being on annual leave.

Although quantitative data on timeliness was not collected in the current study, interview with officials responsible for disease surveillance in the animal and human health sectors indicated that there is always delayed reporting, a problem which is more critical in the animal than in the human health sector. For instance, monthly reports in the animal health sector can be delayed by six to nine months before being received by the Epidemiology section (F. Kivaria, pers. comm., 2010). In the human health sector, the timely submission of weekly and monthly reports have been reported to be only 8% and 24%, respectively (Rumisha *et al.* 2007). The problem of poor timely reporting is mainly attributed to the paper-based transmission of data coupled with challenging infrastructure and communication networks especially in rural areas.

Given the challenges of surveillance in the animal and human health sectors, the SACIDS designed a 'fit-for-purpose' OH surveillance strategy. The strategy is considered appropriate for southern Africa as it has taken into consideration the situations and challenges prevailing on the ground. Key considerations include the relatively higher proportion of patients receiving treatment at home from traditional healers before visiting health facilities, lack of proper diagnostic facilities at community and village levels, limited human diagnostic and mobility resources in remote areas as well as poor infrastructure for efficient communication between rural communities and district or ministry headquarters (Strasser 2003). The concept of collaborative efforts in managing infectious diseases in Tanzania and other southern African countries is not new. The emergence of innovative and appropriate technologies, approaches and tools for participatory epidemiology and disease surveillance (Hussain *et al*. 2005; Jost *et al.* 2007), such as the use of mobile technologies (Aanensen *et al.* 2009; Despont-Gros *et al.* 2005) is likely to mean that infectious disease surveillance can be improved in future. This will contribute to better public health and economic and social stability in Africa. It is also anticipated that the OH surveillance will foster stronger collaborative links between the animal and human health professionals and consequently improve management and control of infectious diseases in animal and human health sectors.

Conclusion

The approach designed by SACIDS on OH surveillance is considered suitable for detecting and containing infectious diseases in animal and human populations in countries with limited resources such as those in southern Africa. Adoption of mobile technologies and appropriate surveillance approaches will improve timely and complete capture of events that would have been otherwise have been missed using routine surveillance systems in the animal and human health sectors. It is concluded that the OH surveillance strategy is timely and relevant to sub-Saharan Africa.

Acknowledgements

We would like to acknowledge support and cooperation received from various individuals in the Tanzanian Ministry of Livestock and Fisheries Development, Ministry of Health and Social Welfare, officials at Ngorongoro district council working with the DVO and DMO offices who assisted retrieval of disease surveillance reports. We are grateful to clinical officers, in-charge of different health facilities and wards visited in Ngorongoro district as well as community members who participated in this study. We thank our colleagues at Imperial College London particularly Dr David Aanensen and Mr Chris Powell for their valuable support in capacity building related to setting up, field-testing and use of smartphones running the EpiCollect application in southern Africa. This study was funded by the Rockefeller Foundation through a research support grant 2009 DSN 305.

References

Aanensen, D.M., Huntley, D.M., Feil, E.J., Al-Own, F. & Spratt, B.G., 2009, 'EpiCollect: Linking smartphones to web applications for epidemiology, ecology and community data collection', *PLoS ONE* 4, e6968. http://dx.doi.org/10.1371/journal.pone.0006968, PMid:19756138

Allport, R., Mosha, R., Bahari, M., Swai, E. & Catley, A., 2005, 'The use of community-based animal health workers to strengthen disease surveillance systems in Tanzania', *Revue Scientifique et Technique* (OIE) 24, 921–932. PMid:16642762

Brownlie, J., Peckham, C., Waage, J., Woolhouse, M., Lyall, C., Meagher, L. *et al.*, 2006, *Foresight Infectious diseases: Preparing for the future threats,* Office of Science and Innovation, London.

Despont-Gros, C., Boeuf, C., Geissbuhler, A. & Lovis, C., 2005, 'The Digital Pen and Paper Technology: Implementation and use in an existing clinical information system', *Connecting Medical Informatics and Bio-Informatics* 116, 328–333.

FAO, 2004, 'Surveillance and zoning for aquatic animal diseases', FAO Fisheries Technical Paper 451, Rome.

Franco, L.M., Setzer, J. & Banke, K., 2006, 'Improving performance of IDSR at district and facility levels: Experiences in Tanzania and Ghana in making IDSR operational', PHRPlus (unpublished report).

Hussain, M., Malik, M.A., Fatima, Z. & Yousuf, M.R., 2005, 'Participatory surveillance of livestock diseases in Islamabad capital territory', *International Journal of Agriculture and Biology* 7, 567–570.

James, J. & Muchiri, S., 2006, 'HR mapping of the health sector in Kenya: The foundation for effective HR management', Technical Brief (Unpublished report), viewed n.d., from http://www.hlsp.org/LinkClick.aspx?fileticket=eky0oOnoNQA%3D&tabid=1715&mid=3388

Jones, B., 2011, 'Analysis of human and animal health system response capacity to epidemic and emerging infectious disease in Tanzania', MSc thesis, Royal Veterinary College, London.

Jones, K.E., Patel, N.G., Levy, M.A., Storeygard, A., Balk, D., Gittleman, J.L. *et al*., 2008, 'Global trend in emerging infectious diseases', *Nature* 451, 990–993. http://dx.doi.org/10.1038/nature06536, PMid:18288193

Jost, C.C., Mariner, J.C., Roeder, P.L., Sawitri, E. & Macgregor-Skinner, G.J., 2007, 'Participatory epidemiology in disease surveillance and research', *Revue Scientifique et Technique* 26, 537–49. PMid:18293603

Karimuribo, E., Wambura, P., Mounier-Jack, S., Sonoiya, S., Short, N., Aanensen, D. *et al*., 2011, 'Contrasting features and opportunities for "One Health" infectious disease surveillance system in Tanzania', *EcoHealth* 7, S49.

Karimuribo, E.D. & Swai, E., 2006, 'Impact of community-based animal health workers on animal health and extension services: A case study of smallholder dairy farming areas of East and West Usambara, Tanzania', *Tanzania Veterinary Journal* 23, 57–67.

King, D.A., Peckham, C., Waage, J.K., Brownlie, J. & Woolhouse, M.E.J., 2006, 'Infectious diseases: Preparing for the future', *Science* 313, 1392–1393. http://dx.doi.org/10.1126/science.1129134, PMid:16959992

Mboera, L.E.G., Rumisha, S.F. & Kitua, A., 2001, 'Strategic approach for strengthening national and regional disease surveillance system: The East African example', *Tanzania Health Research Bulletin* 3, 6–9. http://dx.doi.org/10.4314/thrb.v3i2.14211

Morse, S.S., 1995, 'Factors in the emergence of infectious diseases', *Emerging Infectious Diseases* 1, 7–15. http://dx.doi.org/10.3201/eid0101.950102, PMid:8903148

Rumisha, S.F., Mboera, L.E.G., Senkoro, K.P., Gueye, D. & Mmbuji, P.K., 2007, 'Monitoring and evaluation of Integrated Disease Surveillance and Response in selected districts in Tanzania', *Tanzania Health Research Bulletin* 9, 1–11. http://dx.doi.org/10.4314/thrb.v9i1.14285

Schelling, E., Bechir, M., Ahmed, M.A., Wyss, K., Randolph, T.F. & Zinsstag, J., 2007, 'Human and animal vaccination delivery to remote nomadic families, Chad', *Emerging Infectious Diseases* 13, 373–379. http://dx.doi.org/10.3201/eid1303.060391, PMid:17552089

Shayo, E., Mboera, L.E.G., Mmbuji, P., Rumisha, S.F., Senkoro, K.P. & Mwami, A.J., 2003, 'The role of community and traditional healers in communicable disease surveillance and management in Babati and Dodoma districts, Tanzania', *Tanzania Health Research Bulletin* 5, 48–55.

Strasser, R., 2003, 'Rural health around the world: Challenges and solutions', *Family Practice* 20, 457–463. http://dx.doi.org/10.1093/fampra/cmg422, PMid:12876121

Thacker, S.B. & Berkelman, R.L., 1988, 'Public Health surveillance in the United States', *Epidemiologic Reviews* 10(1), 164–190.

Wickramage, K. & Nellapalli, P., 2008, 'Community participatory methods in disease surveillance and public health in war-affected camps, and its potential contribution to peace building', *International Electronic Journal of Health Education* 11, 95–108.

Wolfe, N.D., Dunavan, C.P. & Diamond, J., 2007, 'Origins of major human infectious diseases', *Nature* 447, 279–283. http://dx.doi.org/10.1038/nature05775, PMid:17507975

Electronic Integrated Disease Surveillance System and Pathogen Asset Control System

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How to cite this proceeding:
Wahl, T.G., Burdakov, A.V., Oukharov, A.O. & Zhilokov, A.K., 2012, 'Electronic Integrated Disease Surveillance System and Pathogen Asset Control System', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #455, 5 pages. http://dx.doi.org/10.4102/ojvr.v79i2.455

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Electronic Integrated Disease Surveillance System (EIDSS) has been used to strengthen and support monitoring and prevention of dangerous diseases within One Health concept by integrating veterinary and human surveillance, passive and active approaches, case-based records including disease-specific clinical data based on standardised case definitions and aggregated data, laboratory data including sample tracking linked to each case and event with test results and epidemiological investigations. Information was collected and shared in secure way by different means: through the distributed nodes which are continuously synchronised amongst each other, through the web service, through the handheld devices. Electronic Integrated Disease Surveillance System provided near real time information flow that has been then disseminated to the appropriate organisations in a timely manner. It has been used for comprehensive analysis and visualisation capabilities including real time mapping of case events as these unfold enhancing decision making. Electronic Integrated Disease Surveillance System facilitated countries to comply with the IHR 2005 requirements through a data transfer module reporting diseases electronically to the World Health Organisation (WHO) data center as well as establish authorised data exchange with other electronic system using Open Architecture approach.

Pathogen Asset Control System (PACS) has been used for accounting, management and control of biological agent stocks. Information on samples and strains of any kind throughout their entire lifecycle has been tracked in a comprehensive and flexible solution PACS.

Both systems have been used in a combination and individually. Electronic Integrated Disease Surveillance System and PACS are currently deployed in the Republics of Kazakhstan, Georgia and Azerbaijan as a part of the Cooperative Biological Engagement Program (CBEP) sponsored by the US Defense Threat Reduction Agency (DTRA).

Problem statement

Infectious diseases in the twenty-first century continue to cause economic and social disruptions becoming more severe due to the emerging diseases often of a zoonotic nature, active travel increasing potential of regional and international epidemics and pandemics, and potential bioterrorist threats.

To adequately address these growing threats countries have to strengthen and improve their capability to perform early detection and rapid reporting of infectious disease situation and outbreaks, to timely and accurately verify presence or absence of high-consequence pathogens, and to comprehensively and rapidly respond to care for infected patients and reduce exposure of the wider population, and the accidental and/or deliberate release of high-consequence pathogens.

There have been a number of attempts to improve detection, reporting and respond capabilities with the modern information and communications technology. However, most of the advances in this field tend to either concentrate on isolated vertical segments (e.g. HIV, TB or Malaria), separately address human or veterinary areas, and be isolated from diagnostics (laboratory) and clinical data sources, be disintegrated with the international information systems (World Health Organisation [WHO] and World Organisation for Animal Health [OIE]), or be tailored only for certain environment and disease surveillance priorities without an ability to adjust.

Thus there is a need for a comprehensive information and communications technology solution, which would overcome the challenges of existing solutions and facilitate in early detection, rapid reporting and response. The creation of this comprehensive solution would require significant effort from the epidemiology and diagnostics experts society as well as investment in development and implementation of the solution.

Methods and approach

For the 10 years the United States Defense Threat Reduction Agency (DTRA) has been actively implementing the Cooperative Biological Engagement Program (CBEP), which goals closely match infectious disease threats most of the countries face:

- combat bioterrorism and prevent the proliferation of biological weapons-related technology, pathogens and expertise
- enhance host governments' disease surveillance systems to detect and report bioterrorism attacks, epidemics and potential pandemics.

To address these goals two information and communications technology solutions were created, (1) Electronic Integrated Disease Surveillance System (EIDSS) and (2) Pathogen Asset Control System (PACS). Electronic Integrated Disease Surveillance System facilitates in early detection, rapid reporting and response to disease outbreaks, whilst PACS improves security of dangerous pathogens stored in the laboratories.

Method 1: Electronic surveillance using Electronic Integrated Disease Surveillance System

Electronic Integrated Disease Surveillance System is an electronic system intended to facilitate in collecting, notifying, sharing and analysing surveillance data. It consists of several modules listed below together with a specification of tracked data:

- Human module
  - demographic data
  - disease-specific clinical data based on standard case definitions
  - epidemiological investigations
  - sample and laboratory tests tracking linked to a specific case
  - aggregate cases.
- Veterinary module
  - avian and livestock cases
  - farm or owner information
  - disease-specific clinical data based on standard case definitions
  - epidemiological investigations
  - sample and laboratory tests tracking linked to a specific case
  - penside tests
  - aggregate cases
  - active surveillance data.
- Laboratory
  - samples, tests assignment, tests results and batch tests
  - aliquots and derivatives
  - transfer in or out operations
  - links to clinical case data.

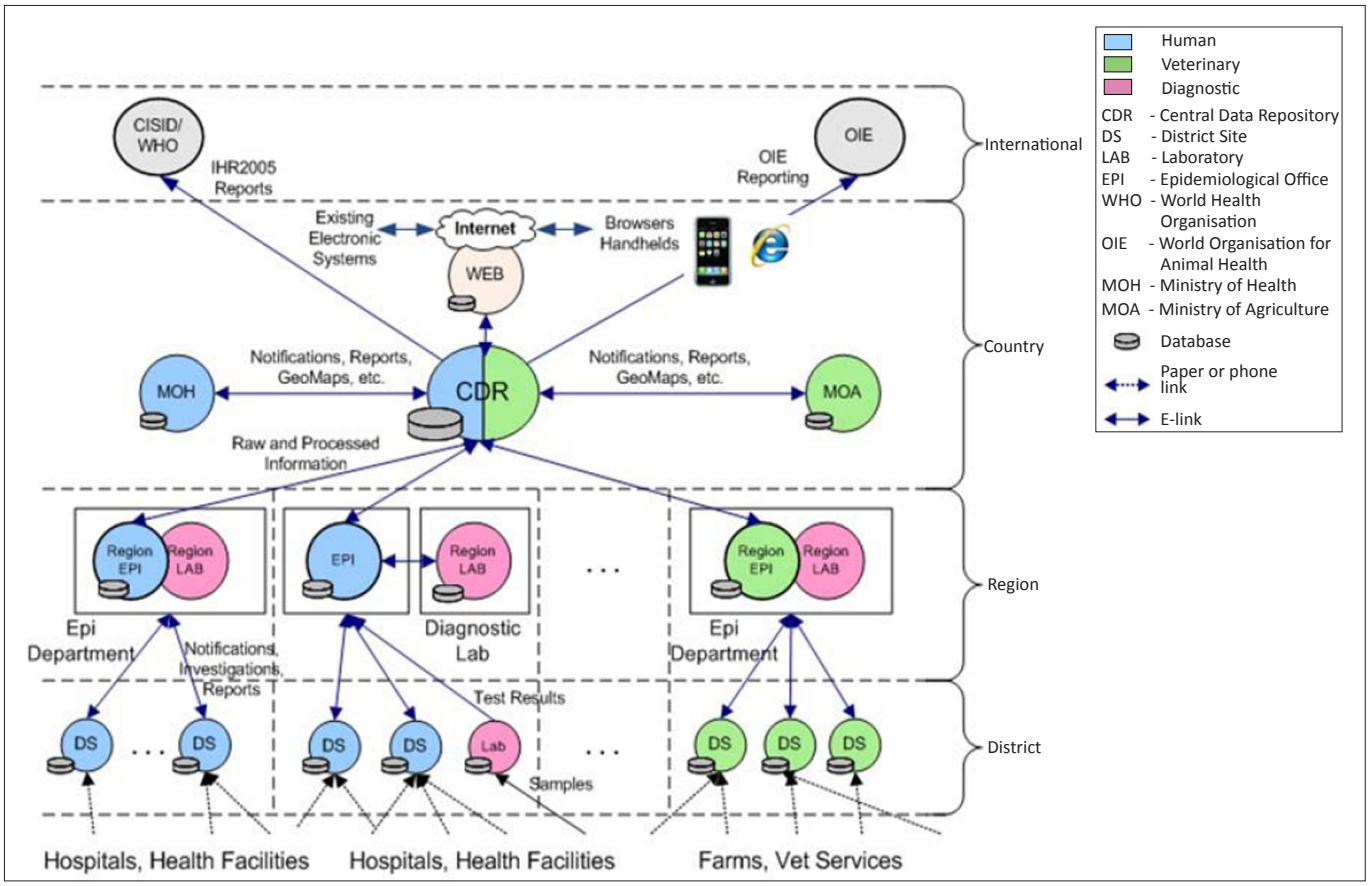

FIGURE 1: Typical Electronic Integrated Disease Surveillance System architecture.

- Analysis
  - customised and predefined reports
  - statistic analysis
  - different ways of data representation: grids, charts, maps.

Information can be collected and shared through these modules in several ways providing a near real-time information flow that can be then disseminated to the appropriate organisations in a timely manner. Figure 1 represents typical EIDSS hierarchy covering different administrative levels: district, region, country.

The first way to collect information is through a number of distributed nodes which are continuously synchronised amongst each other. Let us say a veterinary case is registered in a district. Once this information is entered to the system, replication starts and transfers the case to the region level site, which in turn send it to the Central Data Repository (CDR), then it goes other sites which are supposed to get such information. Bi-directional regular data exchange amongst sites guaranties that all participants get actual and up-to-date information. If a node gets off-line it keeps working in disconnected mode and will be synchronised with other nodes once it gets on-line.

The EIDSS Web server is the other way to get information into the system (Figure 2). Data entered through the web site is distributed to other nodes of the system and available for review and editing according to users' permissions.

Access to EIDSS from handheld devices is the third entry point (Figure 2). Web portal designed for handheld devices provides simplified interface and support a variety of devices. Any combination of these methods can be used depending on infrastructure, organisational and other issues. In a combination with using of commercial 'off-the-shelf' readily available generic hardware it makes EIDSS suitable for environments with different challenges.

Methods and features

Following One Health approach EIDSS integrates human and veterinary surveillance data along with a laboratory information. For example, it is possible to create an outbreak record in the system which ties together several human and veterinary cases. One can also link several veterinary cases together. Adding a laboratory piece gives the opportunity diagnose a case with a most recent laboratory data. For instance, case entered in the veterinary service is transferred to the laboratory and available through the EIDSS lab module. As soon as test results are registered for this case in the laboratory they will be transferred to the veterinary service site and can be used for further investigations.

Flexibility and scalability is one of the fundamental principles of the EIDSS. One example of such flexibility is that cases can be tracked with different levels of specification: case-based tracking with a specific case definitions and investigation information, when every case is registered as individual record, can be used for especially dangerous diseases, case-based tracking with just an emergency notification information can be used for other important diseases, aggregate reporting which is supposed to track just summary data from district or regions for a certain period of time. Electronic Integrated Disease Surveillance System also allows to switch between different approaches depending on the current situation. Case definitions used in the system also can be adjusted according to the specific requirements.

Electronic Integrated Disease Surveillance System supports different types of surveillance: passive surveillance (case-based and aggregate) is available for human and veterinary diseases, active surveillance is supported for veterinary disease, vector surveillance is planned to be released in the next version.

Comprehensive analysis modules give users various capabilities to investigate and present collected data. Using either predefined reports which duplicates officially-approved paper forms or customisable reports where one can build a report as needed, users are able to get access to any variables in the database and explore human, veterinary and lab. The GIS component of the system allows attaching geo-coordinates to cases and map data in addition to charts and grid representations (Figure 2).

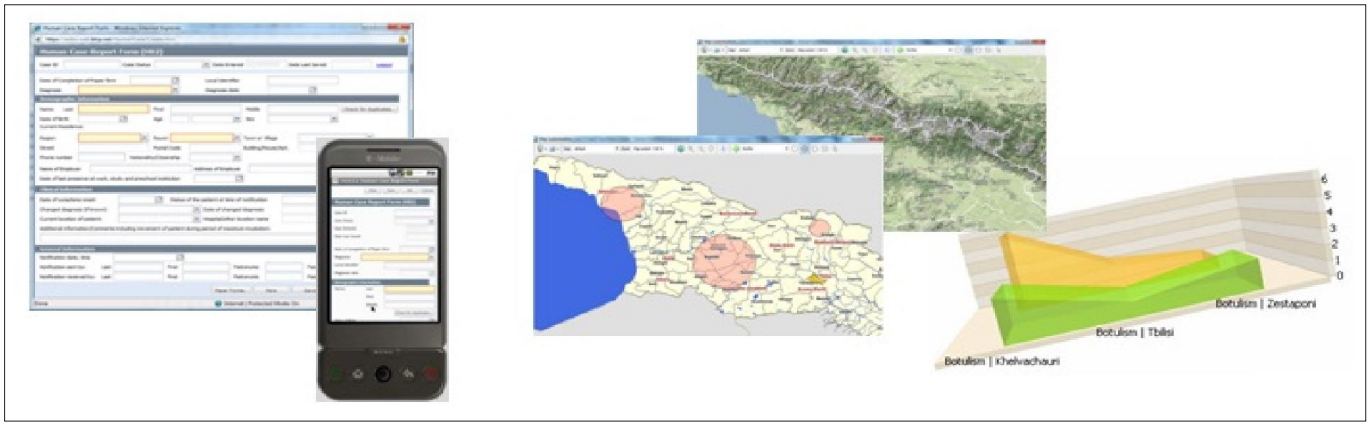

FIGURE 2: Web and/or handheld access to Electronic Integrated Disease Surveillance System: data analysis.

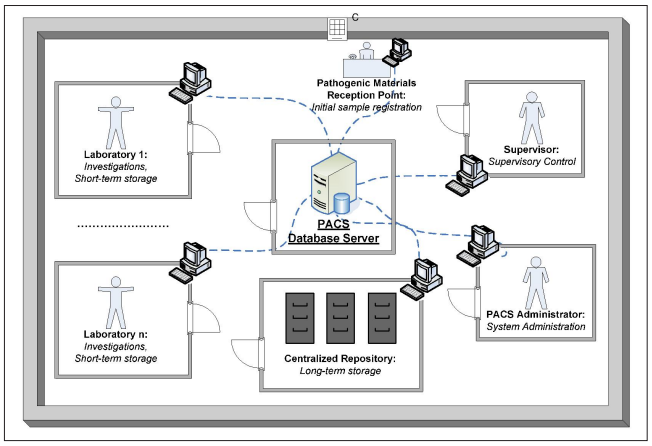

FIGURE 3: Pathogen Asset Control System conceptual schema.

FIGURE 4: Repository inventory audit.

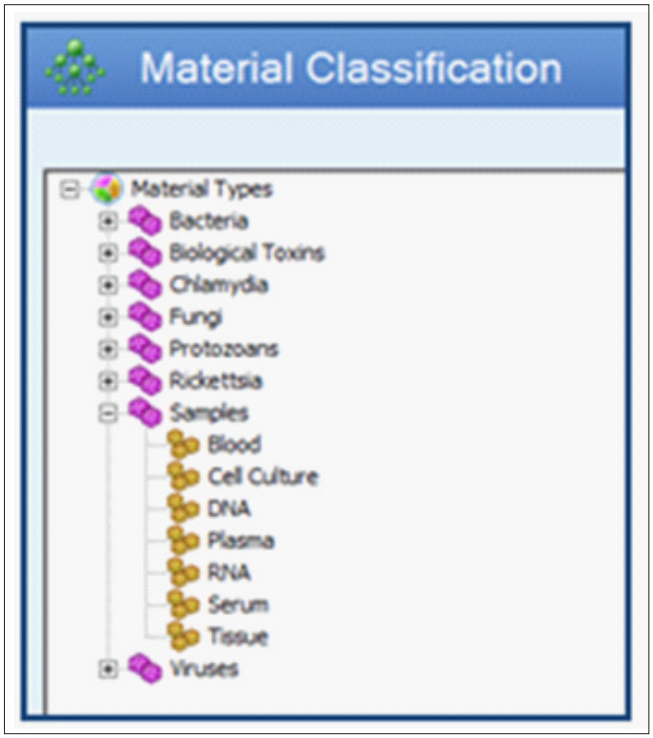

FIGURE 5: Material classification.

Electronic Integrated Disease Surveillance System provides means for integration with other local or international systems: the WHO module can transfer approved reports to Computerised Information System for Infectious Diseases (CISID) assisting in International Health Regulations (IHR) compliance, open architecture approach supported by EIDSS allows to establish communications with existing local electronic systems. Regional cooperation between neighbouring countries is also possible on the base of EIDSS.

Full localisation of the system which includes electronic forms, reports and manuals, unified design and staff training make it easy to operate for non-experienced users. Turn-key deployment and support contribute to the sustainability of the solution.

Results

Electronic Integrated Disease Surveillance System is being developed since 2005 in collaboration with different institutions including the Centers for Disease Control and Prevention (CDC), US, the Walter Reed Army Institute for Research (WRAIR), US, Ministries of Health and Veterinary Departments of Kazakhstan, Uzbekistan, Azerbaijan and Georgia, Ukraine and Armenia and numerous international medical and veterinary experts. Following iterative approach EIDSS went through the number of expertise (more than 75 000 hours) and revisions.

Currently version 3 is deployed in Azerbaijan (90 installations), Kazakhstan (more than 150 installations) and Georgia (more than 120 installations). The initial phase of deployment in Armenia and Ukraine. Electronic Integrated Disease Surveillance System has been officially recognised in Azerbaijan in 2010 and recently in Kazakhstan and Georgia.

Electronic Integrated Disease Surveillance System is a solution that improves the capacity to detect, diagnose and report bioterrorism attacks and potential pandemics, and supports bioresearch. It strengthens both regional and global disease surveillance with plans for expansion into African and Asian regions and globally.

Method 2: Biological pathogens tracking using Pathogen Asset Control System

Pathogen Asset Control System helps to track and control pathogens that are collected, investigated and stored in biological laboratories. It allows monitoring of the whole life-cycle of the pathogens with necessary levels of detail and appropriate security.

Pathogen Asset Control System can be implemented within different facilities, from small repositories with one centralised storage area to big multi-discipline institutions with distributed structure and numerous laboratories. Server-client architecture allows immediate access to all necessary information for every user who has appropriate rights, which includes principal investigators, facility management, IT administrators and other laboratory personnel.

Freezer 1 [HSDO-1303].Shelf 1.Box 1

	Container ID	Pos	Material #	Container Type	Microorganism / Sample type
1	C100061	4	M100008	Ampoule	Brucella suis
2	C100062	5	M100008	Ampoule	Brucella suis
3	C100066	18	M100008	Ampoule	Brucella suis

FIGURE 6: Freezer content report.

Pathogen Asset Control System utilises Barcode technology and Radio-frequency Identification (RFID) to uniquely track all individual vials with highly pathogenic materials and allows error-free fast data input and retrieval.

Handling dangerous biological materials and processing information about them requires certain level of safety and security, and PACS provides invaluable assistance in this area. Separated access control to data, detailed audit trails, inventory audit function and other security features of the application help the owner to comply with international and local regulations in Biosafety and Biosecurity.

Considering that different organisations might have different requirements on what information must be tracked on biological materials and how, PACS allows several options for system customisation and adoption to local requirements. That includes designing barcode labels, managing repository configuration, creating different data entry forms depending on material type customise data entry forms and output reports. Being flexible and adoptable to the owner's requirements PACS employs all common operations with biological materials, such as registration, transfer, destruction, splitting and reculturing. All operations are supplied with printable reports and acts.

Pathogen Asset Control System provides wide opportunities to utilise information entered into the system for biological investigation support. A variety of different user defined fields and information in them, supplemented with the capability of creating custom reports provides an opportunity to output data in ways so it can be used for analysis of different parameters.

Results

Pathogen Asset Control System is an important part of the overall Biosecurity solution and can be very efficient in every biological laboratory where implemented. It allows the necessary levels of tracking and control over dangerous pathogens and complies with strengthening or enforcing regulations in Biosafety.

Being implemented in numerous laboratories in 6 different countries PACS proved its reliability and effectiveness. Pathogen Asset Control System is currently undergoing Certification and Accreditation (C&A) process according to United States Department of Defense standards and should be offered to many government agencies as a system for tracking biological agents once C&A is completed. Pathogen Asset Control System is a robust electronic system that competently helps to solve the important task of managing dangerous materials and will be in demand by biological laboratories.

Acknowledgements

Mr Tom G. Wahl is a project manager for EIDSS and PACS product. He prepared the overview of the EIDSS and PACS and introduction. Dr Aleksey V. Burdakov made contribution to all sections of the article, including problem statement. Mr Andrey O. Oukharov wrote EIDSS section of the article. Mr Azamat K. Zhilokov wrote PACS section.

References

Burdakov, A.V., 2005, 'Implementation of the communications and information technology part of the International Program on Treat Agent Detection and Response in the Republic of Kazakhstan', *Environment and Population Health* 1, 37.

Wahl, T. & Burdakov, A.V., 2007, 'Electronic Integrated Disease Surveillance System (EIDSS)', *Advances in Disease Surveillance* 2, 171.

New technologies to diagnose and monitor infectious diseases of livestock: Challenges for sub-Saharan Africa

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How to cite this proceeding:
King, D.P., Madi, M., Mioulet, V., Wadsworth, J., Wright, C.F., Valdazo-González, B. *et al.*, 2012, 'New technologies to diagnose and monitor infectious diseases of livestock: Challenges for sub-Saharan Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #456, 6 pages. http://dx.doi.org/10.4102/ojvr.v79i2.456

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Using foot-and-mouth disease (FMD) as an example, this review describes new tools that can be used to detect and characterise livestock diseases. In recent years, molecular tests that can detect and characterise pathogens in a diverse range of sample types have revolutionised laboratory diagnostics. In addition to use in centralised laboratories, there are opportunities to locate diagnostic technologies close to the animals with suspected clinical signs. Work in this area has developed simple-to-use lateral-flow devices for the detection of FMD virus (FMDV), as well as new hardware platforms to allow molecular testing to be deployed into the field for use by non-specialists. Once FMDV has been detected, nucleotide sequencing is used to compare field strains with reference viruses. Transboundary movements of FMDV are routinely monitored using VP1 sequence data, while higher resolution transmission trees (at the farm-to-farm level) can be reconstructed using full-genome sequencing approaches. New technologies such as next-generation sequencing technologies are now being applied to dissect the viral sequence populations that exist within single samples. The driving force for the use of these technologies has largely been influenced by the priorities of developed countries with FMD-free (without vaccination) status. However, it is important to recognise that these approaches also show considerable promise for use in countries where FMD is endemic, although further modifications (such as sample archiving and strain and serotype characterisation) may be required to tailor these tests for use in these regions. Access to these new diagnostic and sequencing technologies in sub-Saharan Africa have the potential to provide novel insights into FMD epidemiology and will impact upon improved strategies for disease control.

Effective control of infectious diseases is reliant upon accurate diagnosis of clinical cases using laboratory tests, together with an understanding of factors that impact upon the epidemiology of the infectious agent. A wide range of new diagnostic tools and nucleotide sequencing methods are used by international reference laboratories to detect and characterise the agents causing outbreaks of infectious diseases. In the past, high costs (initial capital expenses, as well as day-to-day maintenance and running costs) and complexity of the protocols used to perform some of these tests have limited the use of these methods in smaller laboratories. However, simpler and more cost-effective formats are now being developed that offer the prospect that these technologies will be even more widely deployed into laboratories particularly those in developing regions of the world such as sub-Saharan Africa.

Foot-and-mouth disease

This short review focuses on foot-and-mouth disease (FMD) and highlights new diagnostic approaches that can be used to detect and characterise pathogens causing livestock diseases. Foot-and-mouth disease is a trans-boundary viral disease that is endemic in sub-Saharan Africa, much of south Asia (Middle East, Indian sub-continent and Southeast Asia) and parts of South America. The causative agent, FMD virus (FMDV), is a picornavirus (genus: *Aphthovirus*) with a positive-sense RNA genome of approximately 8300 nucleotides in length. Foot-and-mouth disease virus exists as seven genetically discrete serotypes, five of which are currently endemic in sub-Saharan Africa. This virus causes an acute disease in cloven-hoofed animals (i.e. cattle, sheep, goats, pigs and buffalo) that is associated with the development of vesicles on epithelial surfaces of the mouth and feet. Foot-and-mouth disease virus infection also generates a transient viraemia in infected animals that typically lasts for approximately five days (Figure 1; Alexandersen *et al*. 2003).

Tests that exploit these clinical windows in an infected animal form the basis of laboratory approaches currently used to diagnose FMD. These assays aim to detect FMDV in epithelium and fluid from vesicles, as well as in blood and swabs from mucosal surfaces (oral and nasal swabs). In addition, FMDV-specific antibody responses in exposed animals can be detected using serological assays; however these methods are not discussed in this review. In general terms, the virological assays utilise three different strategies: propagation of FMDV, detection of viral antigenic proteins,

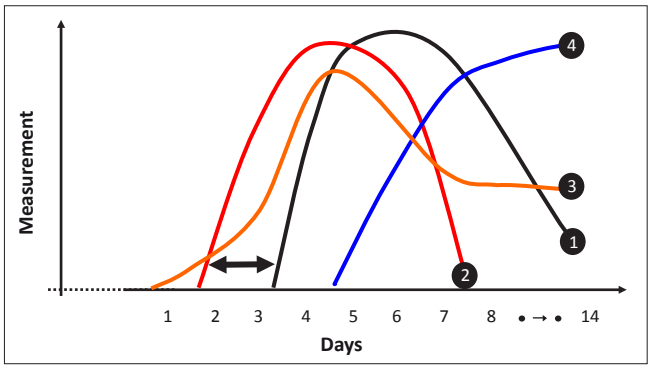

FIGURE 1: Diagnostic windows for FMD showing temporal distribution of FMDV in different biological samples ([1-black] vesicular epithelium; [2-red] blood and [3-orange] mucosal swabs) and (4-blue) FMDV-specific antibodies in sera. The dotted axis-line represents incubation period that is dependent upon infectious dose, virus strain, species and other host determinants. This figure was generated using representative 'in contact' experimental cattle data from previous studies (Alexandersen *et al.* 2003) and unpublished data from IAH.

or use of molecular assays to amplify specific RNA sequences. The earliest versions of these tests exploited approaches that were largely used to propagate FMDV for vaccine production (Brown 2003). Although initially developed during the 1920s and used up until the 1950s, the obvious disadvantages of these systems placed limitations on their use for wide scale FMD diagnostics. Indeed, these systems were superseded by improved highly sensitive *in-vitro* cell cultures used to propagate FMDV, such as primary bovine thyroid cells (Snowdon 1966), permanent pig kidney cultures (IB-RS-2; De Castro 1964) and more recently a goat tongue cell line (Brehm *et a*l. 2009). Virus isolation approaches can be highly sensitive (depending upon the cell culture system used) although it can be slow taking between one and four days to generate a result. Propagation of FMDV in cell cultures is still widely considered to be the 'gold-standard' test for FMD diagnosis.

The development and establishment of cell culture systems has been paralleled by improvements to immunological (enzyme-linked immunosorbent assays: ELISA) assays designed to detect FMDV antigen using polyclonal antisera (Ferris *et al.* 1988) or characterised monoclonal antibody reagents (Ferris *et al*. 2011). Capture ELISAs are more rapid than virus isolation, but they have lower analytical sensitivity and are also inappropriate for use with certain sample types such as blood, milk and swabs. More recently, lateral-flow devices (LFDs, also referred to as immuno-chromatographic strip tests) have been developed for the detection of FMD viral antigen. These simple-to-use and rapid tests utilise FMDV specific antibody reagents (normally monoclonal antibodies) in a format similar to the sandwich capture ELISA used for laboratory diagnosis. Positive test signal is generated by the diffusion of coloured, antibody-coated latex beads or colloidal gold particles through a membrane towards an immobilising band of trapping antibody. An LFD has been developed for the detection of all seven FMDV serotypes which uses a pan-serotypic monoclonal antibody (Ferris *et al*. 2009). In addition, sample preparation in field conditions can be achieved using simple disposable tissue homogenizers for preparing epithelial suspensions. In terms of diagnostic sensitivity and specificity, the overall performance of this LFD is similar to laboratory-based antigen ELISA, although the diagnostic sensitivity of the current test is lower for SAT 2 field strains (Ferris *et al.* 2009). This LFD and a simple homogenising kit is now commercially available (via Boehringer Ingelheim Vetmedica Svanova, Uppsala, Sweden). Data from the field illustrates the potential for the LFD to be used in locations close to animals to provide rapid support to veterinarians in their clinical assessment of suspected FMD cases. The simplicity and stability of the LFD may be important features for FMD diagnosis in sub-Saharan African countries. In particular, ability to rapidly recognise FMDV in clinical material may improve the selection of diagnostic samples that are shipped to reference laboratories for subsequent isolation and/or strain characterisation. In addition, recent results have also indicated that it is possible to recover RNA from FMDV positive LFDs that can be used in rRT-PCR assays and sequencing studies. Further LFD assays are now in development and future formats, such as the SAT 2 device that has recently been evaluated (Ferris *et al.* 2010), may also allow rapid serotyping of field strains.

Polymerase chain reaction technologies for foot-and-mouth disease virus detection

Amplification of specific nucleic acid sequences using reverse transcription polymerase chain reaction (RT-PCR) is now widely used for the laboratory detection of FMDV. These molecular assays are suitable for the diverse range of different samples that might be submitted for laboratory investigation (tissues, blood, swabs, oesophageal or pharyngeal (OP) scrapings, faecal samples and milk). Over the past 15 years, improvements have been made to RT-PCR protocols used for the detection of FMDV. Initially, assays that targeted conserved regions of the genome (3D: Meyer *et al*. 1991; Rodrıguez *et al.* 1994 and 5′ untranslated region [5′ UTR], Reid *et al*. 2000) utilised agarose gel electrophoresis for the detection of amplified products. However, these labour intensive procedures have a high risk of generating false positives due to carry-over of chain reaction (PCR) amplicons and are therefore not generally considered ideal for routine testing of large numbers of samples. Real-time RT-PCR (rRT-PCR) assays have now largely replaced agarose gel based assay formats. These more rapid fluorescence-based approaches are highly sensitive enabling simultaneous amplification and quantification of FMDV specific nucleic acid sequences. In addition to enhanced sensitivity, the benefits of these closed-tube rRT-PCR assays over conventional endpoint detection methods include a reduced risk of cross-contamination, their large dynamic range, an ability to be scaled up for high-throughput applications and the potential for accurate target quantification. Several assays have been developed to detect FMDV that use 5′-nuclease assay (TaqMan®) system to detect PCR amplicons (Callahan *et al.* 2002; Oem *et al.* 2005; Reid *et al*. 2002). Other formats exploited for FMDV-specific rRT-PCR assays include the use of modified minor groove binder (MGB) probes (McKillen *et al.* 2011; Moniwa *et al*. 2007), hybridisation probes (Moonen

et al. 2003), Primer-probe energy transfer (PriProET: Rasmussen *et al.* 2003) and RT-linear-after-the-exponential PCR (LATE PCR: Reid *et al.* 2010). In order to minimise human operator errors and increase assay throughput, these assays can be automated using robots for nucleic acid extraction (Reid *et al.*; Moonen *et al.* 2003). Together with the implementation of quality control systems, these improvements have increased the acceptance of the rRT-PCR assays for routine diagnostic purposes.

In addition to use in centralised laboratories, there are opportunities to deploy rRT-PCR technologies close to the animals with suspect clinical signs. These test formats may be particularly suitable for use in FMD-endemic areas such as countries within sub-Saharan Africa where the time taken to collect and dispatch samples to a laboratory for disease investigation can be protracted. Work in this area has explored the use of new hardware platforms to allow PCR testing to be deployed into the field for use by non-specialists (Callahan *et al.* 2002; Hearps, Zhang & Alexandersen 2002; King *et al.* 2008). The focus of current work is the development of hardware platforms (Madi *et al.* 2011) incorporating a simple-to-use and robust template extraction process such that all the steps of the assay can be performed without user intervention. These steps include (1) nucleic acid extraction, (2) PCR set-up, (3) amplification and (4) unambiguous calling of results. The use of homogeneous systems has previously been recognised an important aspect for the implementation of molecular methods for field detection of FMDV (Hearps *et al.* 2002). Currently, there is only limited access to these technologies outside of Europe and North America. In addition to the performance of these equipment and assays, the availability and cost of consumables, as well as mechanisms to locally service the machines (in the event of equipment failure) will be important factors for the routine use of these tests in countries within sub-Saharan Africa.

Isothermal amplification

The recent development of portable equipment for PCR has made molecular diagnosis of FMD in the field an achievable goal. However, this approach relies on precision thermocycling requiring instrumentation which can be fragile, prohibitively expensive and that will require decontamination when transferred from one site to another. As an alternative to PCR, isothermal (single temperature) amplification methods for the detection of FMDV have been developed. Since the specific amplification step for both nucleic acid sequence based amplification (NASBA) and reverse-transcription loop-mediated amplification (RT-LAMP) formats occurs at a constant temperature, there is less reliance upon expensive equipment and there is obvious potential to use of these assays as the basis of an inexpensive (or even disposable) molecular test. Two different isothermal strategies: NASBA and RT-LAMP are discussed in this review.

Nucleic acid sequence based amplification technology is a continuous, isothermal and enzyme-based method to amplify single stranded RNA and is therefore particularly suited to the detection of viruses with RNA genomes such as FMDV (Compton 1991). Assays employ three enzymes: T7 RNA polymerase, reverse-transcriptase and ribonuclease-H, a set of target-specific forward and reverse oligonucleotide primers and two types of detection probes. The forward primer has a 5′ extension containing the promoter sequence for T7 bacteriophage DNA-dependent RNA polymerase, while the reverse primer has a 5′ extension containing a complementary binding sequence for a DNA oligonucleotide detection probe. Using an FMDV specific NASBA assay (Collins *et al.* 2002), concordant results between NASBA and rRT-PCR were generated for 87% of FMD samples (Lau *et al.* 2008). Simple enzyme-linked oligonucleotide capture NASBA formats using ELISA plate readers have also been evaluated and these show promise for use in laboratories where more expensive equipment may not be available (Lau *et al.* 2008).

Reverse-transcription loop-mediated amplification is an isothermal autocyling strand-displacement DNA synthesis technique which utilises four specific primers to recognise six regions of the target genome (Notomi *et al.* 2000). Formation of loop structures enables explosive polymerase-based enzymatic amplification, which generates double-stranded, multi-sized amplicons. Pan-serotypic RT-LAMP assays have been designed for FMDV (Dukes, King & Alexandersen 2006; Li *et al.* 2009). Validation data indicates that RT-LAMP has equivalent analytical sensitivity to rRT-PCR and may be less sensitive to inhibition by problematic sample matrices such as OP fluids and faecal samples. Reverse-transcription loop-mediated amplification products are generated in abundance and can be detected using equipment to monitor turbidity, agarose gels or real-time PCR machines. Furthermore, it is also possible to visualise dual-labelled LAMP amplicons using novel lateral flow devices (James *et al.* 2010). In addition, changes in free-cation (Mg^{2+}) concentration in positive RT-LAMP reactions can be visualised using a colour change of a dye indicator such as hydroxynaphthol blue (Bearinger *et al.* 2011). Together with simple methods to prepare template RNA, these simple readouts for RT-LAMP may prove useful in field settings.

Using genome sequence data to define trans-boundary movements of foot-and-mouth disease virus

Once FMDV has been detected, nucleotide sequencing is a useful tool used to compare field strains with reference viruses and allows important phenotypic characteristics to be elucidated, such as antigenic determinants present on the viral capsid. Genetic characterisation of foot-and-mouth disease virus routinely uses VP1 sequence data generated by RT-PCR. This region of the FMDV genome is approximately 630 nucleotides in length and encodes a protein (1D) that comprises an important component of the FMD viral capsid. These sequences are used to categorise field strains into discrete sub-groups (or topotypes) which frequently show geographical clustering based on the historical distribution of

the virus (Di Nardo, Knowles & Paton 2011). These analyses provide evidence for the transboundary movements of FMDV and provide critical support to regional and country-level programmes to control FMD. Within sub-Saharan Africa, three main epidemiological pools of FMDV have been recognised (Paton, Sumption & Charleston 2009): West Africa comprising serotypes O, A, SAT 1 and SAT 2; East Africa comprising serotypes O, A, SAT 1, SAT 2 and SAT 3 and Southern Africa with serotypes SAT 1, SAT 2 and SAT 3.

Recent FMD outbreaks in the United Kingdom (during 2001 and 2007) have highlighted the limitation to which VP1 sequence data can be used to discriminate sequences from field cases of disease. VP1 is relatively short (~8% of the genome length) and as a consequence, phylogenetic trees generated from viral sequences recovered within outbreaks clusters are typically flat with poor resolution. Furthermore, limiting sequence analysis to only VP1 reduces the ability to identify broad-scale recombination events that may drive step-changes in the generation of new genetic and antigenic variants. Identifying the sources of FMD outbreaks can play an important role in disease control: however, this can be confounded by incomplete epidemiological evidence and the numerous routes by which the virus can spread (via aerosols, movements of infected animals or their products, and spread of fomites on contaminated persons and objects). Recent advances in laboratory methodologies allow rapid sequencing of complete FMDV genomes (Abdul-Hamid *et al.* 2011; Cottam *et al.* 2009a). For the first time, this opens up the potential for using genome sequencing to reconstruct virus transmission trees with extremely high resolution and to reveal and identify the origin of unresolved transmission events within discrete infection clusters quickly.

Using full FMDV genome sequences determined from field samples collected from the 2001 FMD outbreak in the UK, it was shown that transmission events at the level of farm-to-farm spread could be reconstructed (Cottam *et al.* 2006, 2008b). Known patterns of spread of the virus were be reproduced by statistical parsimony-based analyses of this data. However, in some cases, these genetic data supported transmission histories different from those suggested by conventional contact tracing studies (Cottam *et al.* 2006). During the 2007 FMD outbreak in the UK, full genome sequencing was used in real-time to support epidemiological investigations (Cottam *et al.* 2008a). Analyses of these data have revealed the most likely chain of transmission events, and predicted undisclosed infected premises prior to their discovery by serological surveillance. Thus, for the first time, results indicate that full-genome sequencing can be used for fine-scale epidemiology to reveal and identify the origin of FMDV causing outbreaks.

Foot-and-mouth disease viral evolutionary dynamics

Foot-and-mouth disease evolves rapidly due to its large population size, high replication rate and poor proof-reading ability of its RNA-dependent RNA polymerase. Within cells, FMDV exists as heterogeneous populations comprising similar but non-identical genomes. Consensus (Sanger) sequencing identifies the predominant sequence present in a sample, but does not provide any information regarding the structure of minority variants that are present. Next-Generation Sequencing (NGS) techniques offer an unprecedented 'step-change' increase in the amount of sequence data that can be generated from a sample to reveal nucleotide substitutions present in only a small fraction of the population. Using NGS performed on a Genome Analyzer platform (Illumina), the viral populations within bovine epithelial samples (foot lesions) have been determined (Wright *et al.* 2011). This approach revealed the fine polymorphic sub-structure of the viral population, from nucleotide variants present at just below 50% frequency to those present at fractions of 1%. Some of the higher frequency polymorphisms identified encoded changes in the heparan sulphate binding site revealing intermediate stages in the evolution of a tissue-culture adapted viral genome upon replication within a mammalian host.

Conventional Sanger sequencing and NGS methods can provide powerful datasets that have the potential to revolutionise our understanding of the patterns of viral evolution and factors that impact upon FMDV circulation in endemic countries. Capital equipment and running cost to undertake sequencing studies are decreasing all the time. Regional and national centres of molecular and sequencing expertise are now becoming established in some African countries to provide support for research into the epidemiology of livestock diseases such as FMD. Although currently limited to use in specialised molecular facilities, there is a prospect that new more affordable formats of NGS technologies will become available soon.

Diagnostic challenges for sub-Saharan Africa

This short review describes new tools that have recently been developed to support the diagnosis and epidemiology of FMD. The driving force for these improvements has largely been influenced by the priorities of developed countries in Europe and North America with FMD-free (without vaccination) status. However, it is important to recognise that these technologies also show considerable promise for use in FMD-endemic countries, although further modifications may be required to tailor these tests for use in these regions. The presence of multiple serotypes of FMD in the majority of sub-Saharan African countries can influence local disease diagnostic priorities. Rather than pan-serotypic FMDV assays that are developed in FMD-free countries, a particular priority for endemic countries is the development of serotype-specific (and lineage-specific) typing assays that can be used rapidly to monitor incursions of exotic and emerging FMDV lineages into new geographical regions. The high genetic diversity of circulating FMDV strains (particularly SAT serotypes) provides a constant challenge for diagnostic laboratories in

FMD endemic countries in Africa, and it is important that on-going validation is undertaken to ensure tests remain fit for purpose and are able to detect new viruses as they continue to evolve. Furthermore, it is critical that robust protocols for sample collection and archiving in the field are adopted (Belsham *et al*. 2011). Our understanding of FMD epidemiology in Africa is patchy and currently suffers from a lack of good quality laboratory and sequence data. The global control of FMD strategy currently being developed jointly by OIE and FAO is expected to rely heavily on the use of these next generation tools for more accurate and rapid diagnosis of FMD at the local level. However, without improvements to the quality and coverage of samples collected from field outbreaks, it is unlikely that the epidemiological picture of the disease in the region will become clear enough to make a significant impact upon disease control.

Conclusions

These new diagnostic tools can play a critical role in our ability to detect and monitor the spread of FMD in endemic regions of the world. However, it is also important to recognise that effective monitoring and control of FMD is reliant upon adequate resources, these are principally financial but also include availability of trained field personnel and a strong supporting laboratory infrastructure. In addition, international cooperation, transparency between different countries, sharing of epidemiological data and ownership of disease are also key factors in the control of important trans-boundary disease such as FMD.

Acknowledgements

The authors of this article are supported by grant funding from UK Department of Environment, Food and Rural Affairs (Defra SE1124, SE1126, SE2938 and SE2937), EU NoE EPIZONE (Contract No. FOOD-CT-2006-016236) and the Combating Infectious Diseases of Livestock for International Development (CIDLID) project funded by DFiD/BBSRC/ Scottish Government (*Towards the strategic control of foot-and-mouth disease in Africa: New techniques for a neglected problem*). The authors also acknowledge Southern African Centre for Infectious Disease Surveillance (SACIDS) for providing travel support (to DK) to present this paper at the 1st One Health Conference in Africa, Johannesburg, July 2011.

References

Abdul-Hamid, N.F., Fırat-Saraç, M., Radford, A.D., Knowles, N.J. & King, D.P., 2011, 'Comparative sequence analysis of representative foot-and-mouth disease virus genomes from Southeast Asia', *Virus Genes* 43, 41–45. http://dx.doi.org/10.1007/s11262-011-0599-3, PMid:21479678

Alexandersen, S., Quan, M., Murphy, C., Knight, J. & Zhang, Z., 2003, 'Studies of quantitative parameters of virus excretion and transmission in pigs and cattle experimentally infected with foot-and-mouth disease virus', *Journal of Comparative Pathology* 129, 268–282. http://dx.doi.org/10.1016/S0021-9975(03)00045-8

Bearinger, J.P., Dugan, L.C., Baker, B.R., Hall, S.B., Ebert, K., Mioulet, V. *et al*., 2011, 'Development and initial results of a low cost, disposable, point-of-care testing device for pathogen detection', *IEEE Transactions on Biomedical Engineering* 58, 805–808. http://dx.doi.org/10.1109/TBME.2010.2089054, PMid:21342806

Belsham, G.J., Jamal, S.M., Tjørnehøj, K. & Bøtner, A., 2011, 'Rescue of foot-and-mouth disease viruses that are pathogenic for cattle from preserved viral RNA samples', *PLoS One* 6, e14621. http://dx.doi.org/10.1371/journal.pone.0014621, PMid:21298025

Brehm, K.E., Ferris, N.P., Lenk, M., Riebe, R. & Haas, B., 2009, 'Highly sensitive fetal goat tongue cell line for detection and isolation of foot-and-mouth disease virus', *Journal of Clinical Microbiology* 47, 3156–3160. http://dx.doi.org/10.1128/JCM.00510-09, PMid:19656987

Brown, F., 2003, 'The history of research in foot-and-mouth disease', *Virus Research* 91, 3–7. http://dx.doi.org/10.1016/S0168-1702(02)00268-X

Callahan, J.D., Brown, F., Osorio, F.A., Sur, J.H., Kramer, E., Long, G.W. *et al*., 2002, 'Use of a portable real-time reverse transcriptase-polymerase chain reaction assay for rapid detection of foot-and-mouth disease virus', *Journal of the American Medical Association* 220, 1636–1642.

Collins, R.A., Ko, L.S., Fung, K.Y., Lau, L.T., Xing, J. & Yu, A.C.H., 2002b, 'A method to detect major serotypes of foot-and-mouth disease virus', *Biochemistry Biophysics Research Communication* 297, 267–274. http://dx.doi.org/10.1016/S0006-291X(02)02178-2

Compton, J., 1991, 'Nucleic acid sequence-based amplification', *Nature* 350, 91–92. http://dx.doi.org/10.1038/350091a0, PMid:1706072

Cottam, E.M., Haydon, D.T., Paton, D.J., Gloster, J., Wilesmith, J.W., Ferris, N.P. *et al*., 2006, 'Molecular epidemiology of the foot-and-mouth disease virus outbreak in the United Kingdom in 2001', *Journal of Virology* 80, 11274–11282. http://dx.doi.org/10.1128/JVI.01236-06, PMid:16971422

Cottam, E.M., Wadsworth, J., Shaw, A.E., Rowlands, R.J., Goatley, L., Maan, S. *et al*., 2008a, 'Transmission pathways of foot-and-mouth disease virus in the United Kingdom in 2007', *PloS Pathogens* 4, e1000050. http://dx.doi.org/10.1371/journal.ppat.1000050, PMid:18421380

Cottam, E.M., Thébaud, G., Wadsworth, J., Gloster, J., Mansley, L., Paton, D.J. *et al*., 2008b, 'Integrating genetic and epidemiological data to determine transmission pathways of foot-and-mouth disease virus', *Proceedings of the Royal Society B* 275, 887–895. http://dx.doi.org/10.1098/rspb.2007.1442, PMid:18230598

Cottam, E.M., Wadsworth, J., Knowles, N.J. & King, D.P., 2009a, 'Full sequencing of viral genomes: practical strategies used for the amplification and characterization of foot-and-mouth disease virus', *Methods in Molecular Biology* 551, 217–230. http://dx.doi.org/10.1007/978-1-60327-999-4_17, PMid:19521878

De Castro, M.P., 1964, 'Behaviour of the foot-and-mouth disease virus in cell cultures; susceptibility of the IB-RS-2 cell line', *Archivos do Instituto Biologico, São Paulo* 31, 63–78.

Di Nardo, A., Knowles, N.J. & Paton, D.J., 2011, 'Combining livestock trade patterns with phylogenetics to help understand the spread of foot-and-mouth disease virus in sub-Saharan Africa, the middle East and Southeast Asia', *Scientific and Technical Review of the OIE* 30, 63–85.

Dukes, J.P., King, D.P. & Alexandersen, S., 2006, 'Novel reverse transcription loop-mediated isothermal amplification for rapid detection of foot-and-mouth disease virus', *Archives of Virology* 151, 1093–1106. http://dx.doi.org/10.1007/s00705-005-0708-5, PMid:16453084

Ferris, N.P. & Dawson, M., 1988, 'Routine application of enzyme-linked immunosorbent assay in comparison with complement fixation for the diagnosis of foot-and-mouth and swine vesicular diseases', *Veterinary Microbiology* 16, 201–209. http://dx.doi.org/10.1016/0378-1135(88)90024-7

Ferris, N.P., Hutchings, G.H., Reid, S.M., King, D.P., Ebert, K., Paton, D.J. *et al*., M., 2009, 'Development and laboratory validation of a lateral flow device for the detection of foot-and-mouth disease virus in clinical samples', *Journal of Virological Methods* 155, 10–17. http://dx.doi.org/10.1016/j.jviromet.2008.09.009, PMid:18848845

Ferris, N.P., Nordengrahn, A., Hutchings, G.H., Paton, D.J., Kristersson, T., Brocchi, E. *et al*., 2010, 'Development and laboratory validation of a lateral flow device for the detection of serotype SAT 2 foot-and-mouth disease viruses in clinical samples', *Journal of Virological Methods* 163, 474–476. http://dx.doi.org/10.1016/j.jviromet.2009.09.022, PMid:19815031

Ferris, N.P., Grazioli, S., Hutchings, G.H. & Brocchi, E., 2011, 'Validation of a recombinant integrin αvβ6/monoclonal antibody based antigen ELISA for the diagnosis of foot-and-mouth disease', *Journal of Virological Methods* 175, 253–260. http://dx.doi.org/10.1016/j.jviromet.2011.05.026, PMid:21635921

Hearps, A., Zhang, Z. & Alexandersen, S., 2002, 'Evaluation of a portable Cepheid SmartCycler real-time PCR machine for the rapid diagnosis of foot-and-mouth disease', *Veterinary Record* 150, 625–628. http://dx.doi.org/10.1136/vr.150.20.625, PMid:12046786

James, H.E., Ebert, K., McGonigle, R., Reid, S.M., Boonham, N., Tomlinson, J.A. *et al*., 2010, 'Detection of African swine fever virus by loop-mediated isothermal amplification', *Journal of Virological Methods* 164, 68–74. http://dx.doi.org/10.1016/j.jviromet.2009.11.034, PMid:19963011

King, D.P., Dukes, J.P., Reid, S.M., Ebert, K., Shaw, A.E., Mills, C.E. *et al*., 2008, 'Prospects for rapid diagnosis of foot-and-mouth disease in the field using reverse transcriptase-PCR', *Veterinary Record* 162, 315–316. http://dx.doi.org/10.1136/vr.162.10.315, PMid:18326844

Lau, L.T., Reid, S.M., King, D.P., Lau, A.M., Shaw, A.E., Ferris, N.P. *et al*., 2008, 'Detection of foot-and-mouth disease virus by nucleic acid sequence-based amplification (NASBA)', *Veterinary Microbiology* 126, 101–110. http://dx.doi.org/10.1016/j.vetmic.2007.07.008, PMid:17728080

Li, J., Chen, Q., Xiong, W. & Fang, X.E., 2009, 'Establishment of RT-LAMP for rapid detection of foot-and-mouth disease virus', *Bing Du Xue Bao* 25, 137–142. PMid:19678569

Madi, M., Hamilton, A., Squirrell, D., Mioulet, V., Evans, P., Lee, M. *et al*., 2011, 'Rapid detection of foot-and-mouth disease virus using a field-portable nucleic acid extraction and real-time PCR amplification platform', *Veterinary Journal* (submitted). PMid:21668486

McKillen, J., McMenamy, M., Reid, S.M., Duffy, C., Hiertner, B., King, D.P. *et al*., 2011, 'Pan-serotypic detection of foot-and-mouth disease virus using a minor groove binder probe reverse transcription polymerase chain reaction assay', *Journal of Virological Methods* 174, 117–119. http://dx.doi.org/10.1016/j.jviromet.2011.03.008, PMid:21419170

Meyer, R.F., Brown, C.C., House, C., House, J.A. & Molitor, T.W., 1991, 'Rapid and sensitive detection of foot-and-mouth disease virus in tissues by enzymatic RNA amplification of the polymerase gene', *Journal of Virological Methods* 34, 161–172. http://dx.doi.org/10.1016/0166-0934(91)90096-I

Moonen, P., Boonstra, J., Van der Honing, R.H., Leendertse, C.B., Jacob, L. & Dekker, A., 2003, 'Validation of a light Cycler-based reverse transcriptase polymerase chain reaction for the detection of foot-and-mouth disease virus', *Journal of Virological Methods* 113, 35–41. http://dx.doi.org/10.1016/S0166-0934(03)00220-9

Moniwa, M., Clavijo, A., Li, M., Collignon, B. & Kitching, P.R., 2007, 'Performance of a foot-and-disease virus reverse transcription-polymerase chain reaction with amplification controls between three real-time instruments', *Journal of Veterinary Diagnostic Investigation* 19, 9–20. http://dx.doi.org/10.1177/104063870701900103, PMid:17459827

Notomi, T., Okayama, H., Masubuchi, H., Yonekawa, T., Watanabe, K., Amino, N. *et al*., 2000, 'Loop-mediated isothermal amplification of DNA', *Nucleic Acids Research* 28, E63. http://dx.doi.org/10.1093/nar/28.12.e63

Oem, J.K., Kye, S.J., Lee, K.N., Kim, J.Y., Park, J.H., Joo, Y.S. *et al*., 2005, 'Development of a light cycler-based reverse transcriptase polymerase reaction for the detection of foot-and-mouth disease virus', *Journal of Veterinary Science* 6, 207–212. PMid:16131823

Paton, D.J., Sumption, K.J. & Charleston, B., 2009, 'Options for control of foot-and-mouth disease: knowledge, capability and policy', *Philosophical Transactions of the Royal Society London B Biology Science* 364, 2657–2567. http://dx.doi.org/10.1098/rstb.2009.0100, PMid:19687036

Rasmussen, T.B., Uttenthal, A., De Stricker, K., Belak, S. & Storgaard, T., 2003, 'Development of a novel quantitative real-time RT-PCR assay for the simultaneous detection of all serotypes of foot-and-mouth disease virus', *Archives of Virology* 148, 2005–2021. http://dx.doi.org/10.1007/s00705-003-0145-2, PMid:14551821

Reid, S.M., Ferris, N.P., Hutchings, G.H., Samuel, A.R. & Knowles, N.J., 2000, 'Primary diagnosis of foot-and-mouth disease by reverse transcription polymerase chain reaction', *Journal of Virological Methods* 89, 167–176. http://dx.doi.org/10.1016/S0166-0934(00)00213-5

Reid, S.M., Ferris, N.P., Hutchings, G.H., Zhang, Z., Belsham, G.J. & Alexandersen, S., 2002, 'Detection of all seven serotypes of foot-and-mouth disease virus by real-time, fluorogenic reverse transcription polymerase chain reaction assay', *Journal of Virological Methods* 105, 67–80. http://dx.doi.org/10.1016/S0166-0934(02)00081-2

Reid, S.M., Grierson, S.S., Ferris, N.P., Hutchings, G.H. & Alexandersen, S., 2003, 'Evaluation of automated RT-PCR to accelerate the laboratory diagnosis of foot-and-mouth disease virus', *Journal of Virological Methods* 107, 129–139. http://dx.doi.org/10.1016/S0166-0934(02)00210-0

Reid, S.M., Pierce, K.E., Mistry, R., Bharya, S., Dukes, J.P., Volpe, C. *et al*., 2010, 'Pan-serotypic detection of foot-and-mouth disease virus by RT linear-after-the exponential PCR', *Molecular and Cellular Probes* 24, 250–255. http://dx.doi.org/10.1016/j.mcp.2010.04.004, PMid:20433917

Rodríguez, A., Núñez, J.I., Nolasco, G., Ponz, F., Sobrino, F. & De Blas, C., 1994, 'Direct PCR detection of foot-and-mouth disease virus', *Journal of Virological Methods* 47, 345–349. http://dx.doi.org/10.1016/0166-0934(94)90030-2

Snowdon, W.A., 1966, 'Growth of foot-and mouth disease virus in monolayer cultures of calf thyroid cells', *Nature* 210, 1079–1080. http://dx.doi.org/10.1038/2101079a0, PMid:4288087

Wright, C.F., Morelli, M.J., Thébaud, G., Knowles, N.J., Herzyk, P., Paton, D.J. *et al*., 2011, 'Beyond the consensus: dissecting within-host population diversity of foot-and-mouth disease virus using next-generation sequencing', *Journal of Virology* 85, 2266–2275. http://dx.doi.org/10.1128/JVI.01396-10, PMid:21159860

Epidemiological investigation into the introduction and factors for spread of Peste des Petits Ruminants, southern Tanzania

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How to cite this proceeding:
Muse, E.A., Karimuribo, E.D., Gitao, G.C., Misinzo, G., Mellau, L.S.B., Msoffe, P.L.M. *et al*., 2012, 'Epidemiological investigation into the introduction and factors for spread of Peste des Petits Ruminants, southern Tanzania', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #457, 6 pages. http://dx.doi.org/10.4102/ojvr.v79i2.457

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

A study was carried out to confirm and identify sources and elucidate factors associated with the introduction of Peste des Petits Ruminants (PPR) in southern Tanzania. This study was conducted in Tandahimba and Newala districts of Mtwara region following suspected outbreak of PPR in the area. Qualitative data were collected using semi-structured questionnaires and in-depth interviews of key informants who included goat and sheep owners with suspected cases of PPR and animal health service providers as well as local administrative authority. Additionally, 216 serum samples and 28 swabs were collected for serological and virological laboratory disease confirmation. The results show that PPR was first introduced in Likuna village of Newala district in February 2009 through newly purchased goats from the Pugu livestock market located about 700 km in the outskirts of Dar es Salaam city. Factors which contributed to spread of PPR included communal grazing and the cheap prices of sick animals bought by livestock keepers for slaughtering in other villages. Laboratory findings confirmed presence of PPR in the area by RT-PCR and serological analysis revealed that seroprevalence was 31%. These findings have confirmed, for the first time, introduction of PPR in southern Tanzania. The presence of PPR poses high risk of southward spread of the disease to other southern African countries in the SADC region thus calling for concerted and collaborative efforts in prevention and control of the disease to avoid losses. Further elaborate studies on the spread, prevalence and risk factors associated with the disease should urgently be investigated.

Introduction

Peste des Petits ruminants is an acute, highly contagious infectious disease of small domestic ruminants and small wild ruminants, such as antelopes, impala and gazelles (Abu Elzein *et al.* 2004; Nussieba *et al.* 2009). The disease is caused by Peste des Petits Ruminants (PPR) virus (PPRv). PPRv belongs to a Morbillivirus genus of Paramixyoviridae family. PPRv is a single serotype that is differentiated into four (I-IV) lineages (Forsyth & Barrett 1995; Couacy-Hymann *et al.* 2002). The geographical distribution of PPR lineages varies as lineage I and II have been commonly reported in West Africa (Ashley *et al.* 2010), lineage III has been reported eastern Africa except for Sudan which has been found to harbour lineage IV in addition to lineage III (Khalafalla *et al.* 2010). On the other hand, lineage IV has been reported in Central and North Africa, Asia and China (Ozkul *et al.* 2002; Wang *et al.* 2009; Awa *et al.* 2000; Ayari-Fakhfakh *et al.* 2010; Balamurugan *et al.* 2010; Khalafalla *et al.* 2010).

The disease is transmitted by direct contact involving secretions or excretions from infected animals to healthy animals in close proximity. Clinically, PPR is characterised by sudden onset of depression, fever, lacrimation, sores in the mouth, dyspnoea and coughing, foul smelling diarrhoea and death. Post-mortem findings, normally restricted to the alimentary tract, consist of extensive erosive stomatitis and heamorrhagic gastro-enteritis, and often include streaks of congestion along the folds of the mucosa resulting in the characteristic 'zebra-striped' appearance (Chauhan *et al.* 2009). Secondary bronchopneumonia is common.

The disease is characterised by high morbidity and mortality (50% – 80%) in naive sheep and goats populations, impacting negatively on the livelihoods, food security and socio-economic activities of livestock keepers in affected areas. The disease also impacts negatively on the local and international livestock trade markets. In Tanzania the disease limits the efforts of farmers and government in attaining the millennium development goal for eradicating extreme poverty and hunger.

In Tanzania, PPR was first confirmed in 2008 in the northern areas (Kivaria *et al.* 2009; Swai *et al.* 2009), where it was confined until recently when it was suspected to have been introduced

in southern areas in 2010 (FAO 2010). The presence of PPR in southern Tanzania would pose a high risk for spread to the whole of the Southern African Development Community (SADC) threatening to devastate the livelihoods and food security of millions of small herders and agro-pastoralists (FAO 2010). Until the current study was designed, no official confirmation of PPR in southern parts of the country had been carried out. The objectives of this study were:

- to confirm the presence of PPR in goats and sheep in Newala and Tandahimba districts, Mtwara region
- to identify sources and factors that contributed to the introduction and spread of the PPR in southern Tanzania
- to describe the epidemiological factors and losses in the affected villages.

The hypotheses were to confirm if suspected cases seen in goats and sheep in southern Tanzania were caused by PPRv and to find out which factors facilitate the spread of the disease in this zone.

Material and methods

Study area

The study was carried out in eight selected villages in two districts of Mtwara region, Newala and Tandahimba districts, in southern Tanzania between March and May 2011 (Figure 1). The estimated census for humans, goats and sheep in these districts are shown in Table 1. The study area was purposively targeted following reports of suspected PPR outbreak that decimated small ruminants in the districts. This area borders Mueda district of the Cabo Delgado province of Mozambique with a small ruminant population that is naive to PPR. The study area lies between 2° 11′ and 6° 14′ S, and 35° 11′ and 38° 26′ E at an elevation of 100 m – 800 m above sea level. The rainy season starts in November and/or December to April and/or May with an average annual precipitation of 893 mm – 1001 mm. The mean monthly temperature varies from 23 °C to 27 °C and relative humidity varies from 79% to 87%.

Study design and data collection

The current study employed a cross-sectional study design where selected villages were visited once between March 2011 and May 2011. Purposive sampling of animals was carried out in villages with suspected PPR cases based on opinion from local veterinary officials and leaders. Upon identification of household with suspected cases, the researcher (EAM) conducted detailed interviews with owners of goats and sheep. This was complemented with random selection of five animals, either goats or sheep, which were subjected to detailed clinical examination as well as sample collection.

Questionnaire survey

A semi-structured questionnaire was developed and field tested on a few households keeping goats and sheep in the two districts. The interviews focused on collection of information on flock size, species, age and sex, health and vaccination status, and management. Age was approximated and classified as kids or lambs (≤ 3 months), weaners (> 3 to ≤ 9 months) and adults (> 9 months). Health status data was collected by recording history of disease outbreak or occurrence, its clinical signs, overall number of sick animals (used to compute morbidity) as well as overall and specific deaths associated with observed clinical cases (used to compute crude and case fatality of PPR). Flock management data included access to animal health and extension services (presence, type and frequency of services), action taken after the outbreak, live animal market visiting frequency and addition of new animals, and suspected source of the infection.

In-depth interviews of key informants were conducted to obtain opinion from local livestock field officers (LFOs) as well as from local government officials in affected villages and wards. The district veterinary officer (DVO) of Newala District and veterinary officers at Mtwara Veterinary Investigation Centre (VIC) were also interviewed to ascertain where and when PPR was first introduced into southern Tanzania and which disease control measures were instituted before the current study.

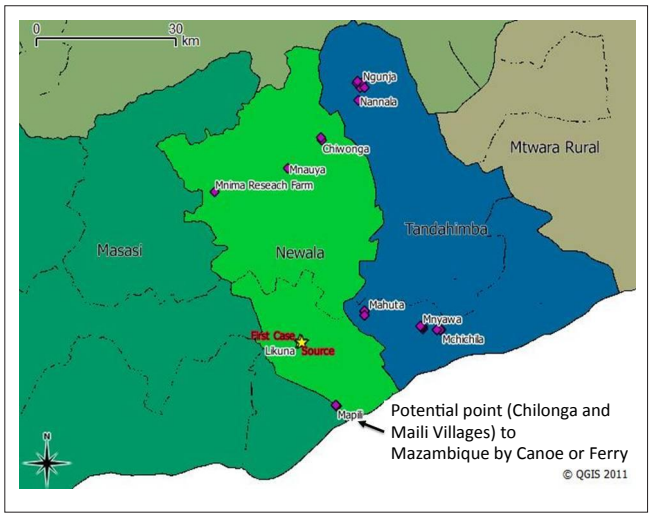

FIGURE 1: Showing sampling points and introduction of Peste des Petits Ruminants in Newala (star).

TABLE 1: The proportions of households, morbidity, mortality, chemotherapy, vaccination and statistics for goats, sheep and people.

Parameter†	Newala (*n* = 31)	Tandahimba (*n* = 48)	Overall (*n* = 79)
Households affected	19.4	81.3	57.0
Morbidity	4.8	73.1	48.9
Mortality (Crude)	4.4	37.4	25.7
Case fatality	92.9	51.2	52.6
Households treated (Chemotherapy)	22.6	43.8	35.4
Households vaccinated	0.0	0.0	0.0
Population statistics‡			
Goat	105 174	149 945	255119
Sheep	2085	2348	4433
Human	183 344	203 837	387181

†, Parameters are measured in percentage.
‡, Statistics are based on figures available in respective District Council Offices in Newala and Tandahimba districts during the study.

Samples collection for Competitive Enzyme Linked Immunosorbent Assay Analysis

A total of 216 serum samples were collected from goats and sheep in Newala and Tandahimba districts. Initially, blood samples were collected from the jugular vein of each animal using plain Vacutainer tubes. The samples were labelled accordingly to allow identification of each animal and flock sampled and kept in a slanted position overnight to allow serum separation from clotted blood samples. Serum was decanted and aliquoted into 1.5 mL cryovials before being transported and stored temporarily at the VIC, Mtwara. Finally the serum samples were shipped in a cool box chilled on ice packs to the Laboratory at the VIC, Arusha where serological analysis was carried out.

A monoclonal antibody (MAb) based competitive Enzyme Linked Immunosorbent Assay (cELISA) (Diallo *et al.* 1995) was used for the detection of antibodies in sera to PPRv using approved competitive ELISA kit as described by Singh *et al.* (2004a) and Swai *et al.* (2009). According to the manufacturer, the sensitivity and specificity (for both animal and flock levels) of this cELISA are 99.4% and 94.5%, respectively. Briefly, the ELISA plates were coated with PPR antigen; the unbound antigen was washed away using buffer then samples were added; Rabbit antimouse-horseradish peroxidase (HRPO) conjugate was added and incubated with constant agitation in each stage. Substrate solution (O-phenylenediamine dihydrochloride containing H_2O_2) was added allowing for a colour reaction to develop which was halted with the addition of an equal volume of 1 M H_2SO_4. The ELISA micro plates were read with an immunoskan reader (Flow laboratories, UK) with an inference filter of 492 nm and connected to a computer loaded with ELISA Data Information (EDI) software for automated reading and calculation of the percentage inhibition (PI) values.

Samples collection for Reverse Transcriptase Polymerase Chain Reaction

Samples (n = 28) from nasal and eye discharges as well as saliva from oral ulcers were collected from clinically sick sheep and/or goats using sterile swabs which were placed in a viral transport media containing antibiotics and antifungals. The samples were transported on ice to Sokoine University microbiology laboratory for further analysis.

Virological analysis employed Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) to confirm involvement of PPR in suspected cases. Briefly, the test was carried out as follows: The RNA extraction from samples was done using commercial RNA extraction kit (Qiagen®); the RNA was converted to cDNA using a reverse transcriptase enzyme (Superscript III Platinum One-Step Quantitative, Invitrogen®). The cDNA was amplified using PPRv specific NP3 and NP4 primers as previously described by Couacy-Hymann *et al.* (2002). PCR products were analysed by electrophoresis and visualised in a UV transilluminator.

Statistical data analysis

Villages, farms and individual animal data were stored in Microsoft Excel 2007 (version 12). Descriptive statistics for the animal and flock level explanatory variables examined in the study were computed using Microsoft Excel. Proportions were calculated for seroprevalence and factors that included animal species, sex and age, location of the flock, health status, management and veterinary services provision. The statistical significance for the proportions was compared using Chi-square test in Epi Info software version 5 (Centre for Disease Control and Prevention). A confidence limit of less than 5% was used to indicate a significant level. Separate statistical analyses were performed for the data from the two species because previous studies have indicated that virus infection rate and epidemiology may be very different in the two species (Waret-Szkuta *et al.* 2008). Study village maps were created using Quantum GIS (QGIS 1.4.0 version) Enceladus (Open Source Geospatial Foundation).

Results

Disease prevalence

The presence of the PPR infection in southern Tanzania was confirmed by the RT-PCR test whereby 53.6% of the samples were positive from both sheep and goats.

The overall PPR seroprevalence was 31% (95% CI = 24.9% – 37.6%) in the two districts (Figure 2). Tandahimba district recorded higher seroprevalence (55.5%) compared to the seroprevalence in 5.7% in Newala district, the difference which was also statistically significant ($p < 0.001$). Mnyawa village recorded highest seroprevalence followed by Nannala, Ngunja, and Mchichira villages of Tandahimba district (Figure 2). Mnima Research Farm had high seroprevalence followed by Chiwonga, Mapili and Mnauya village in that order in Newala district. There was no statistical difference in the seroprevalence recorded in goats (35.3%) compared to that in sheep (30.7%). Similarly, there was no statistical difference in the PPR seroprevalence in female (36.8%) compared to that recorded in male (29.8%) animals sampled in this study. Age-wise, 32.1% of the adults and 22.7% of the kids or lambs were seropositive, however, there was no statistical difference.

The PPR prevalence based on reported cases observed by interviewees is shown in Table 1. Overall, 57% of 79 households visited had experienced suspected PPR cases in their flocks. Again, a significantly higher proportion of households in Tandahimba (81.3%) had PPR cases than those in Newala district (19.4%; $p < 0.001$). A similar trend was also observed with respect to morbidity of PPR when Tandanimba recorded higher morbidity (73.1%) compared to that in Newala district (4.8%; $p < 0.001$). Clinically, a significantly higher proportion of goats (90.9%) were reported to be sick compared to only 9.1% sheep which were sick ($P < 0.001$). A similar trend in mortality was recorded when the proportion of goats and sheep which died was 93.4% and 6.6%, respectively ($P = 0.059$). A good number of

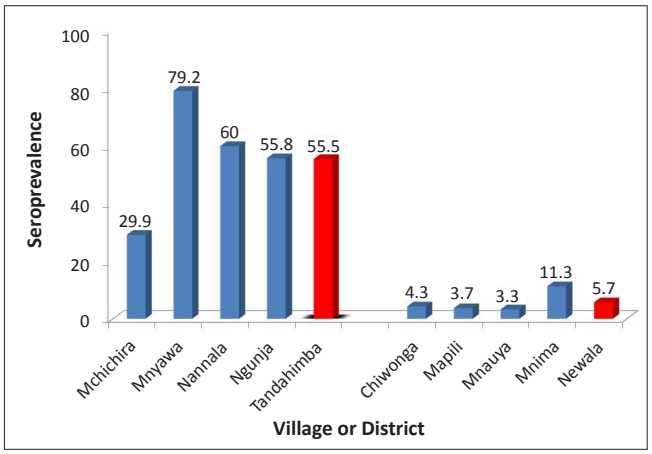

FIGURE 2: Seroprevalence by villages or district.

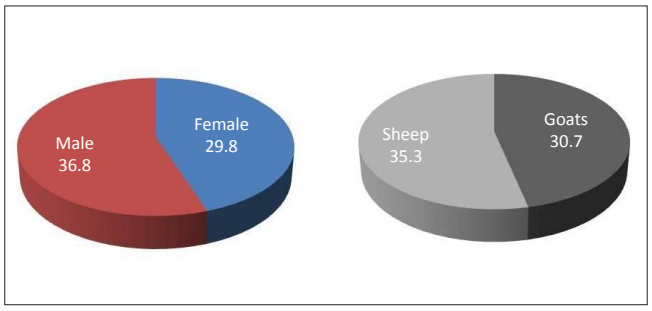

FIGURE 3: Proportions (%) for seroprevalence by sex (left) and species (right).

small ruminant owners also reported to treat their animals using antibiotics as shown in Table 1.

Management practices

It was observed that goats and sheep in the two districts are managed in two different ways. The first group keeps animals in small group in small houses on a raised floor at night and graze in a community land during day time. The second group manage animals collectively by keeping them together in groups in relatively large animal houses on ground floor and utilise communal grazing. Goats and sheep are kept for meat and for sale in order to generate income for the household.

There were more male respondents interviewed (90.0%) in comparison to female respondents (10.0%; Table 2). Only a few respondents in Newala district tethered their animals at night (10.1%) and most (94.9%) farmers kept their animals in a small raised house at night. All (100%) of the respondents utilised communal grazing areas (Table 2).

Introduction of Peste des Petits Ruminants in southern Tanzania

The in-depth interview revealed that PPR was introduced in the southern Tanzania for the first time in goats and sheep in February 2009. The current study managed to trace a village where sick animals started which was Likuna village of Newala district. It was observed that PPR introduction was through newly purchased goats from the Pugu livestock market located about 679 km in the outskirts of Dar es Salaam city. These animals were brought in about one week prior to the disease outbreak. Other villages in close proximity of this village that reported outbreak of PPR in the same month were Kikuyu, Makote, Namiyonga, Lidumbe and Mkunya. It was confirmed that these five villages had also received some of these goats from Pugu Livestock Market. The disease spread to neighbouring Tandahimba District through buying sick and cheap animals from Newala district with the intention of selling them at Tandahimba live animal markets and butcheries.

Observed clinical signs

Different clinical signs were reported by livestock keepers in suspected PPR cases. The signs included diarrhoea, lacrimation, nasal discharges, respiratory distress, oral ulcers and skin nodules (Figure 4).

Economic losses

The economic loss due to mortality in two districts was 25.7% and case fatality rate was 52.6% (Table 1). Case fatality rate was higher in Newala district (92.9%) compared to (51.2%) in Tandahimba district ($P < 0.000$).

TABLE 2: Proportions of respondents' sex, animal management, awareness of the disease, source and spreading of the infection and veterinary extension services.

Parameter†	Newala (n = 31)	Tandahimba (n = 48)	Overall (n = 79)
Household head sex			
Male	32.9	57.0	89.9
Female	5.1	5.1	10.1
Animal management			
Animal tethering	10.1	0.0	10.1
Boma	36.7	58.2	94.9
Communal grazing	39.2	60.8	100
Livestock keepers' awareness			
Outbreak awareness	3.8	60.8	64.6
Seen affected animals	11.4	57.0	68.4
Own animals affected	7.6	49.4	57.0
Knowledge of what disease it was	1.4	1.4	2.9
Seen similar disease in past	1.3	5.1	6.5
Extension worker present	17.8	41.1	58.9
Regular animal inspection	17.6	35.3	52.9
Frequency of inspection			
At least once a month	15.4	24.6	40.0
Once in several months	3.1	10.8	13.8
No visit at all	24.6	9.2	33.8
Visit when called	0.0	12.3	12.3
Source of infection and spread			
Unknown cause	0.0	36.7	36.7
Change of weather	0.0	20.0	20.0
Communal grazing area	10.0	33.3	43.3

†, Parameters are measured in percentage.

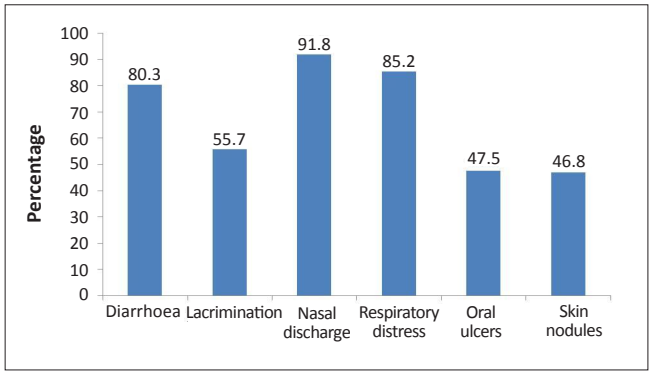

FIGURE 4: Clinical signs presented by Peste des Petits Ruminants disease.

Disease awareness and vaccination

Over 60.8% of respondents in Tandahimba were aware of the disease outbreak and 49.4% had their own animals affected (Table 2). Very few livestock keepers (2.9%) knew what disease it was and only (6.5%) though that they had seen it in the past. During the outbreak, animal owners (35.4%) from the two districts treated their animals using chemotherapy whereas 22.6% were from Newala and 43.8% were from Tandahimba (Table 1). More than half (58.9%) had veterinary extension workers in their villages but half (52.9%) of the farmers reported regular health inspection for their animals, although the frequency of inspection varied (Table 2). All (100%) respondents had not vaccinated their animals (Table 1). The source of the infection and spread was suspected by farmers to be communal grazing and change of weather conditions, with more animals being affected during the rainy season.

Discussion

This study has confirmed the aetiology of the disease outbreak that occurred in the districts of Tandahimba and Newala in southern Tanzania to be PPR in March 2011. It has also identified the village where the first introduction and consequently, the source of PPR spread in southern Tanzania happened. Although it took only one year from first official confirmation of the disease in northern Tanzania to introduction in southern Tanzania, it has taken about two years to confirm this disease through efforts of the current study. This is consistent with the findings by Karimuribo *et al.* (2011) of delayed confirmation following introduction of PPR in the country. These findings point to the importance of having efficient disease surveillance and diagnostic capacity especially for emerging and re-emerging fatal diseases affecting animal populations.

In this study it was found out that the source of the disease was through the introduction of new animals purchased from live animal market. Similar sources of the disease have been implicated before (Singh *et al*. 2004a; Muhammad *et al.* 2009).

The proportions of sero-converted animals examined in the area under study was low (31.0%) compared to the reports from northern Tanzania (45.5%) (Swai *et al.* 2009) but at the flock level the prevalence was high (48.9%). Ozkul *et al.* (2002) in Turkey found comparable findings whereby the overall prevalence was low based on antibody test and higher based on clinical signs. Low PPR seroprevalence have also been reported in Tunisia (Ayari-Fakhfakh *et al.* 2010).

Although the seroprevalence was low in this study it has been shown elsewhere that seroprevalence can be as high as 45.5%, 78% and 92.5%, as reported in Cameroon, Nigeria and Sudan respectively (Ekue *et al.* 1992; Obidike *et al.* 2006; Osman *et al.* 2008) using neutralisation and haemagglutination tests. When the competitive ELISA method was used the seroprevalence was reported to be 51% and 50% (Khan *et al.* 2008; Misbah *et al.* 2009) in Pakistan. The inconsistency in the seroprevalence of antibodies to PPRv in different areas is attributed to variations in a number of factors including the management system, levels of immunity, diagnostic test, sampling procedures used and technical know-how of the researchers (Singh *et al.* 2004b; Waret-Szkuta *et al.* 2008). In this study, goats were reported to show an acute form of the disease whilst sheep showed a sub-acute or chronic form; this finding is supported by other studies (Obi, Rowe & Taylor 1984; Swai *et al.* 2009).

Although, the differences observed between males and females was not significant Swai *et al.* (2009) observed sex differences in sheep whereby males were more affected compared to females. With respect to age category, the highest prevalence of PPR was observed in adults compared to other age category. This result is in agreement with other finds observed in Ethiopia and India (Singh *et al.* 2004a; Waret-Szkuta *et al.* 2008) where they reported high prevalence in adults. Association with seasonal (weather) changes observed by farmers has been reported elsewhere (Singh *et al.* 2004a; Muhammad *et al.* 2009).

Even though the PPR was introduced in Newala, it was more prevalent in Tandahimba district. The reasons are thought to be first selling of sick animals from Newala to Tandahimba animal markets and butcheries and secondly the livestock keepers in Tandahimba district managed their animals by utilising more (60.8%) communal grazing compared to Newala district (39.2%) (Table 2).

This study revealed that more than half of the farmers have little or no access to veterinary services. Lack of appropriate veterinary services and inadequate infrastructure especially in the local live animal markets in the country, may facilitate disease transmission. Extra attention from the government to upgrade the handling and penning facilities of these market places could result in reduced level of disease transmission. Efforts should be make to increase livestock and public awareness with respect to this new disease in the area. The current ongoing government efforts to perform PPR vaccination of goats and sheep in unaffected villages should be encouraged.

Conclusion

This study has confirmed, for the first time, the presence of the PPR in southern Tanzania. In addition, the study has identified the source of introduction of PPR to be newly purchased animals from Pugu Livestock Market. Given that no vaccinations had been carried out against PPR, our result confirms natural transmission of PPR virus under field conditions in the southern Tanzania. The spread of this disease to southern Tanzania poses a high risk of the disease spread to southern countries (SADC countries including Mozambique, Zambia and Malawi) with naïve goat and sheep populations. Further studies on virus isolation, disease status in wildlife and temporal trends events are required to define the epidemiology of PPR in large area of the southern Tanzania. National, regional and international collaborative efforts are required to contain and control the disease.

Aknowledgement

We would like to thank the project of RUFORUM (RU 2009 GRG 17TADs) for supporting this work. We also extend our appreciations to the staff at Mtwara Veterinary Investigation Centre, field officers and farmers for their invaluable contributions in terms of their time and cooperation. In addition, we value VIC Arusha SUA technician for laboratory technical assistance.

References

Abu Elzein, E.M.E., Housawi, F.M.T., Bashareek, Y., Gameel, A.A., Al- Afaleq, A.I. & Anderson, E.C., 2004, 'Severe PPR infection in Gazelles kept under semi-free range conditions in Saudi Arabia', *Journal of Veterinary Microbiology B* 51, 68–71. http://dx.doi.org/10.1111/j.1439-0450.2004.00731.x, PMid:15030603

Ashley, C.B., Satya, P., Carrie, B., Chris, O., Olivier, K. & Genevieve, L., 2010, 'Global distribution of peste des petits ruminants virus and prospects for improved diagnosis and control', *Journal of General Virology* 91, 2885–2897. http://dx.doi.org/10.1099/vir.0.025841-0, PMid:20844089

Awa, D.N., Njoya, A. & Ngo Tama, A.C., 2000, 'Economics of prophylaxis against peste des petits ruminants and gastrointestinal helminthosis in small ruminants in north Cameroon', *Tropical Animal Health and Production* 32, 391–403. http://dx.doi.org/10.1023/A:1005233703331, PMid:11147279

Ayari-Fakhfakh, E., Ghram, A., Bouattour, A., Larbi, I., Gribaa-Dridi, L., Kwiatek, O. *et al*., 2010, 'First serological investigation of peste-des-petits-ruminants and Rift Valley fever in Tunisia', *Veterinary Journal* (In Press).

Balamurugan, V., Sen, A., Venkatesan, G., Yadav, V., Bhanuprakash, V. & Singh, R.K., 2010, 'Isolation and identification of virulent peste des petits ruminants viruses from PPR outbreaks in India', *Tropical Animal Health and Production* 42, 1043–1046. http://dx.doi.org/10.1007/s11250-010-9527-0, PMid:20204510

Chauhan, H.C., Chandel, B.S, Kher, H.N., Dadawala, A.I. & Agrawal, S.M., 2009, 'Peste des petits ruminants virus infection in animals', *Veterinary World* 2, 150–155.

Couacy-Hymann, E., Roger, F., Hurard, C., Guillou, J.P., Libeau, G. & Diallo, A., 2002, 'Rapid and sensitive detection of peste des petits ruminants virus by a polymerase chain reaction assay', *Journal of Virology Methods* 100, 17–25. http://dx.doi.org/10.1016/S0166-0934(01)00386-X

Diallo, A., Libeau, G., Couacy-Hymann, E. & Barbron, M., 1995, 'Recent developments in the diagnosis of rinderpest and peste des petits ruminants', *Veterinary Microbiology* 44, 307– 317. http://dx.doi.org/10.1016/0378-1135(95)00025-6

Ekue, N.F., Tanya, V.N., Ndi, C. & Saliki, J.T., 1992, 'A serological survey of antibodies against peste des petits ruminants (PPR) in small ruminants in Cameroon', *Bulletin of Animal Health and Production in Africa* 40, 49–53.

FAO (2010), 'Deadly animal virus Peste des Petits Ruminants threatens to spread to souther Africa', *Goal Geology*, viewed 16 January 2011, from -animal-virus-peste-des-petits-ruminants-threatens-to-spread-to-southern-africa/8302/

Karimuribo, E.D., Loomu, P.M., Mellau, L.S.B. & Swai, E.S., 2011, 'Retrospective study on sero-epidemiology of peste des petits ruminants before its official confirmation in northern Tanzania in 2008', *Research Opinions in Animal and Veterinary Sciences* 1, 184–187.

Khalafalla, A.I., Saeed, I.K., Ali, Y.H., Abdurrahman, M.B., Kwiatek, O., Libeau, G. *et al.*, 2010, 'An outbreak of peste des petits ruminants (PPR) in camels in the Sudan', *Acta Tropica* 116, 161–165. http://dx.doi.org/10.1016/j.actatropica.2010.08.002, PMid:20707980

Khan, H.A., Siddique, M., Sajjad-ur-Rahman, Abubakar, M. & Ashraf, M., 2008, 'The detection of antibody against peste des petits ruminants virus in sheep, goats, cattle and buffalo', *Tropical Animal Health and Production* 40, 521–527. http://dx.doi.org/10.1007/s11250-008-9129-2, PMid:18716909

Kivaria, F.M., Kwiatek, O., Kapaga, A.M., Geneviève, L., Mpelumbe-Ngeleja, C.A.R. & Tinuga, D.K., 2009, 'Serological and virological investigations on an emerging peste des petits ruminants virus infection in goats and sheep in Tanzania', paper presented at the 27th Tanzania Veterinary Association Scientific Conference, Arusha, Tanzania, 04–06 December.

Misbah, A., Muhammad, A., Rehana, A., Shamim, S. & Qurban, A., 2009, 'Prevalence of peste des petits ruminants virus (PPRV) in Mardan, Hangu and Kohat District of Pakistan; Comparative analysis of PPRV suspected serum samples using competitive ELISA (cELISA) and agar gel immunodiffusion (AGID)', *Veterinary World* 2, 89–92.

Muhammad, A., Syed, M.J., Muhammad, J.A., Manzoor, H. & Qurban, A., 2009, 'Peste des petits ruminants virus (PPRV) infection: Its association with species, seasonal variations and geography', *Tropical Animal Health and Production* 41, 1197–1202. http://dx.doi.org/10.1007/s11250-008-9300-9, PMid:19130284

Nussieba, A.O., Ali, A.S., Mahasin, E.A.R. & Fadol, M.A., 2009, 'Antibody seroprevalences against peste des petits ruminants (PPR) virus in sheep and goats in Sudan', *Tropical Animal Health and Production* 41, 1449–1453. http://dx.doi.org/10.1007/s11250-009-9333-8, PMid:19798772

Obi, T.U., Rowe, L.W. & Taylor, W.P., 1984, 'Serological studies with Peste des petits ruminants and Rinderpest viruses in Nigeria', *Tropical Animal Health and Production* 16, 115–118. http://dx.doi.org/10.1007/BF02239855, PMid:6485096

Obidike, R.I., Ezeibe, M.C.O., Omeje, J.T.N. & Ugwuomarima, K.G., 2006, 'Incidence of peste de petits ruminants in farm and market goats in Nsukka, Enugu State, Nigeria', *Bulletin of Animal Health and Production in Africa* 54, 148–150. http://dx.doi.org/10.4314/bahpa.v54i2.32742

Osman, N.A., Mahasin, E., Rahman, A., Ali, A.S. & Fadol, M.A., 2008, 'Rapid detection of peste des petits ruminants (PPR) virus antigen in Sudan by agar gel precipitation (AGPT) and haemagglutination (HA) Testes', *Tropical Animal Health and Production* 40, 363–368. http://dx.doi.org/10.1007/s11250-007-9106-1

Ozkul, A., Akca, Y., Alkan, F., Barrett, T., Karaoglu, T., Dagalp, S.B. *et al*., 2002, 'Prevalence, distribution, and host range of peste des petits ruminants virus in Turkey', *Emerging Infectious Diseases* 8, 708–712. PMid:12095439

Singh, R.P., Saravanan, P., Sreenivasa, B.P., Singh, R.K. & Singh, B., 2004a, 'Prevalence and distribution of peste des petits ruminants virus infection in small ruminants in India', *Revue Scientifique et Technique* (OIE) 23, 807–819. PMid:15861876

Singh, R.P., Sreenivasa, B.P., Dhar, P., Shah, L.C. & Bandyopadhyay, S.K., 2004b, 'Development of monoclonal antibody based competitive ELISA for detection and titration of antibodies to peste des petits ruminants (PPR) virus', *Veterinary Microbiology* 98, 3–15. http://dx.doi.org/10.1016/j.vetmic.2003.07.007, PMid:14738776

Swai, E.S., Kapaga, A., Kivaria, F., Tinuga, D., Joshua, G. & Sanka, P., 2009, 'Prevalence and distribution of peste des petits ruminants virus antibodies in various districts of Tanzania', *Veterinary Research Communications* 33, 927–936. http://dx.doi.org/10.1007/s11259-009-9311-7, PMid:19705291

Wang, Z., Bao, J., Wu, X., Liu, Y., Li, L., Liu, C. *et al*., 2009, 'Peste des petits ruminants virus in Tibet, China', *Emerging Infectious Diseases* 15, 299–301. http://dx.doi.org/10.3201/eid1502.080817, PMid:19193278

Waret-Szkuta, A., Roger, F., Chavernac, D., Yigezu, L., Libeau, G., Pfeiffer, D.U. *et al*., 2008, 'Peste des petits ruminants (PPR) in Ethiopia: analysis of a national serological survey', *BMC Veterinary Research* 4, viewed 16 January 2011, from http://www.biomedcentral.com/1746-6148/4/34

One Health - 'joining the dots'

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How to cite this proceeding:
Dockrell, H.M., 2012, 'One Health - "joining the dots"', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #458, 3 pages. http://dx.doi.org/10.4102/ojvr.v79i2.458

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Discussion

To achieve the goals of the One Health agenda, multi- and interdisciplinary approaches are essential. It is now common to bring people from different backgrounds together to work on a single disease problem, with a large number of multi-partner consortia currently funded by the European Commission, the European and Developing Countries Clinical Trials Partnership (EDCTP), the Bill and Melinda Gates Foundation and others. Within institutions, mechanisms to increase interactions across disciplines include common seminars, retreats, and the formation of themed virtual centres. Despite the willingness of most scientists to cooperate in research towards a common goal, the existence of different professional cultures has been identified as a barrier in achieving One Health objectives. Cooperation, complementarities and convergence are required to achieve our goals. More effective communication, better teaching to increase awareness of One Health issues and more meetings that bring both animal and human health experts together are needed in order to join the dots across the disciplines in One Health.

A 'One Health' approach to improving human and animal health worldwide is a priority, as about two-thirds of human infectious diseases and about three-quarters of emerging infectious diseases are thought to be zoonoses. In addition, changes to farming methods and to more intensive farming in some parts of the world, together with greater travel and contact with wildlife, all increase the risk of infectious spread from animals to humans. A pertinent example of what can happen when human and animal health agencies do not communicate well is illustrated by the outbreak of Q-fever in the Netherlands (Enserink 2010). Q-fever is caused by *Coxiella burnetti*, an organism that can infect many species but it causes abortions and stillbirths in pregnant goats. In 2007, 182 human cases were diagnosed in the Netherlands, however this increased to 1000 cases in 2008 and 2361 cases in 2009. Due to fears of potential loss of income, the presence of infections in goats had not been communicated to human clinicians. In addition to the burden of human disease that resulted, there were 6 deaths in 2009, and the spread of the infection within intensively farmed goat herds resulted in the decision to cull all pregnant goats in order to control the epidemic. Although it is still unclear whether the epidemic resulted from a particularly virulent strain of *C. burnetti*, or from the high intensity farming of goats in a highly populated country, it is clear that there are both human health and economic risks from such outbreaks.

Multi- and interdisciplinary approaches are required to deal with such zoonotic threats to human health. Environmental factors include the production system in use, host abundance, host species diversity, interactions both within and between species, and selective pressures such as the use of antibiotics (Coker *et al*. 2011). Biological pressures will include both the diversity of the pathogenic species, and its mode and dynamics of transmission. The health impact will also be affected by the transmissibility of the infection, and the risk of disease emergence and spread. Finally there will be economic and social factors that include not just the economic impact of infection but the cultural, environmental and socioeconomic context. Thus Coker and colleagues have advocated that in order to achieve One Health, many disciplines will need to collaborate and work together (Coker *et al*. 2011).

People working on the same disease, or with a common purpose, are usually more than willing to collaborate. A successful example of interdisciplinary collaboration from the London School of Hygiene and Tropical Medicine is that of the Gates Malaria Partnership (n.d.) and its successor, the Malaria Capacity Development Consortium (n.d.), funded by both the Gates Foundation and the Wellcome Trust.

The Gates Malaria Partnership (2001–2009) was able to fund research projects for PhD students and postdoctoral scientists in 14 African countries as well as China, Bolivia and Pakistan, in disciplines ranging from laboratory science to entomology, intervention studies and health economics – yet all these projects became part of a network. The Malaria Capacity Development Consortium (2008–2013) is now continuing the same joined-up approach to research and training in malaria. There are other examples of successful consortia in the tuberculosis field. The European Commission FP6-funded TBVAC Consortium had 32 partners in 9 European and 4

African countries, all united by the common goal of a new vaccine against tuberculosis (TuBerculosis Vaccine Initiative n.d.a). This consortium included the discovery of new antigens, testing in animal models, work on delivery systems and adjuvants, on correlates of protection, and the early steps of testing the new vaccines – thus bringing together scientists with a range of disciplinary backgrounds. Again, successful progress has led to a new consortium, aptly titled NEWTBVAC (TuBerculosis Vaccine Initiative n.d.b), with European Commission FP7 funding, and the establishment of a foundation to facilitate European efforts towards the global development of new TB vaccines, the TuBerculosis Vaccine Initiative (TBVI) (TuBerculosis Vaccine Initiative, n.d.c).

Many other research consortia carry out large multicentre studies, such as the Gates Grand Challenge funded Biomarkers for TB in Africa (Biomarkers for TB n.d.), where cohorts of subjects at 7 African sites have been recruited and followed longitudinally for the development of disease, in order to identify biomarkers that predict the development of, or protection from, disease. Working across different countries can bring additional insights; for example, when BCG vaccinated infants were compared for the immune responses induced three months post BCG vaccination, in the United Kingdom (UK) and Malawi, UK infants were found to have stronger Th1 cytokine responses than Malawian infants (Lalor *et al*. 2009).

However, the use of multiplex bead array assays revealed that the Malawian infants were not merely poor responders – instead they made stronger responses in terms of other Th2 and down-regulatory cytokines, thus illustrating the impact of environment on such immunity (Lalor *et al*. 2011). Sometimes more than one infection can be studied in a consortium, as in the IDEA consortium which is studying the effect of helminth co-infection on immunity against TB, HIV and malaria (IDEA n.d.). On occasion the spectrum of disciplines and backgrounds in such consortia becomes even wider, such as in the Innovative Vector Consortium which combines scientists from academia and from industry (Innovative Vector Consortium n.d.). To be successful, such projects need sufficient funding, good leadership, and regular meetings (preferably at a remote location where all those present have to focus on the business of the meeting).

Partnerships across countries and diseases are also a feature of current capacity building efforts. These include the European and Developing Countries Clinical Trials Network (EDCTP n.d.) with its regional nodes of excellence, and the 7 Wellcome Trust-funded African Institutions Initiative capacity building consortia (Wellcome Trust n.d.). Again, despite different backgrounds and levels of research expertise, such consortia can work together well, sharing experience and identifying new opportunities for collaborative research.

Institutions such as the London School of Hygiene & Tropical Medicine (LSHTM) also have their challenges in maximising the benefit of the breadth of disciplines and infections present within the institution. One approach taken by the LSHTM has been to set up cross-faculty virtual Centres, that can be disease specific (such as the Malaria Centre, or the newer Tuberculosis Centre), or topic specific such as the MARCH Centre that works on maternal, reproductive and child health, linking over 100 researchers who have research interests in some aspect of this topic (London School of Hygiene & Tropical Medicine n.d.). Within the Bloomsbury area of London, a major cross-institution centre has been established, to break down barriers and allow innovative approaches to research and teaching in the area of international development. The London International Development Centre (London International Development Centre n.d.) is a partnership of six colleges situated in the Bloomsbury area – the Royal Veterinary College, Birkbeck College, the School of Pharmacy, the Institute of Education, the School of African and Oriental Studies and the LSHTM. LIDC facilitates the cooperation between LSHTM and the Royal Veterinary College on One Health research and teaching, including the UK support for the Southern African Centre for Infectious Disease Surveillance (SACIDS n.d.).

The outcome of all these linkages and consortia is that scientists, far from working in isolated silos, are becoming so well linked to others in their field that there is a danger that individual research innovation may suffer at the expense of harmonised multicentre studies. Partnerships and consortia are essential if we want to achieve our One Health goals, but these need time, effort and adequate funding, and good management and planning are essential. Together, the partners can make greater progress than they would on their own, but there still needs to be a place for original research ideas (Dockrell 2010).

If consortia and centres that are by definition cross disciplinary can work well, and if institutions can link researchers from different disciplines to work together, this raises the question why a recent publication by Meisser and colleagues (Meisser, Schelling & Zinsstag 2011) identified different professional cultures as the most important barrier to delivering on One Health. Jeff Waage of the London International Development Centre has proposed that in order to make an inter-disciplinary approach to One Health work, *Cooperation* – where working together is necessary to achieve a particular goal, *Complementarity* – where one party benefits from the other's strengths or resources, and *Convergence* – where societal change leads to common tools and agendas, are all needed (J. Waage, pers. comm., n.d.). For example, cooperation is needed on cooperative projects on zoonotic diseases which link veterinary and public health surveillance and management activity. The increasing threat of anti-microbial resistance is one subject that is a clear public health priority and that requires a co-operative approach. Complementarity may sometimes be less obvious, but can usually be found. For example, public health can make use of veterinary health's superior rural delivery systems, whilst public health systems can have superior financing sources and mechanisms that could be exploited by those working in veterinary health. New technologies and tools can also drive convergence, such as with the cassette based point of care

devices and genome sequencing tools that show promise as diagnostic tools for both human and animal infections.

So the challenge for One Health is to identify and overcome the barriers that prevent animal and human health professionals from working together. To forge cooperation, teams need to be built and the barriers that prevent interdisciplinary work broken down. Complementarity could be achieved by educating medical students and veterinary students about each others' activities so that both groups can 'borrow' good ideas. New teaching courses in One Health would facilitate this. And to drive convergence, we need to identify drivers of change and to be proactive, thinking beyond zoonoses. The recent review by Coker *et. al.* (2011) proposes a framework in which research to inform One Health policy can be conducted. What is certain is that more effective communication, better teaching and more meetings that bring both animal and human health experts together are needed in order to join the dots across the disciplines in One Health.

Acknowledgments

The author would like to thank Richard Coker and Jeff Waage for providing material for this review.

References

Biomarkers for TB, n.d., *The GC#6-74 Consortium*, viewed 09 May 2012, from http://www.biomarkers-for-tb.net/consortium/the-consortium

Coker, R., Rushton, J., Mounier-Jack, S., Karimuribo, E., Lutumba, P., Kambarage, D. *et al.*, 2011, 'Towards a conceptual framework to support one-health research for policy on emerging zoonoses', *Lancet Infectious Diseases* 11, 326–331.

Dockrell, H.M., 2010, 'Presidential address: the role of research networks in tackling major challenges in international health', *International Health* 2, 181–185.

Enserink, M., 2010, 'Questions abound in Q-fever explosion in the Netherlands', *Science* 327, 266–267.

European and Developing Countries Clinical Trials Partnership, n.d., *European and Developing Countries Clinical Trials Partnership*, viewed 09 May 2012, from http://www.edctp.org

Gates Malaria Partnership, n.d., *The Gates Malaria Partnership*, viewed 09 May 2012, from http://gmp.lshtm.ac.uk/

IDEA Consortium, n.d., 'Dissecting the Immunological Interplay between Poverty Related Diseases and Helminth infections: An African-European Research Initiative' in *European Commission Research & Innovation - Health*, viewed 09 May 2012, from http://ec.europa.eu/research/health/infectious-diseases/neglected-diseases/projects/014_en.html

Innovative Vector Control Consortium, n.d., *Innovative Vector Control Consortium*, viewed 09 May 2012, from http://www.ivcc.com/

Lalor, M.K., Ben-Smith, A., Gorak-Stolinska, P., Weir R.E., Floyd, S., Blitz. *et al.*, 2009, 'Population differences in immune responses to Bacille Calmette-Guerin vaccination in infancy', *Journal of Infectious Diseases* 199, 795–800. http://dx.doi.org/10.1086/597069, PMid:19434928

Lalor, M.K., Floyd, S., Gorak-Stolinska, P., Ben-Smith, A., Weir, R.E., Smith, S.G. *et al.*, 2011, 'BCG vaccination induces different cytokine profiles following infant BCG vaccination in the UK and Malawi', *Journal of Infectious Diseases* 204, 1075–1085. http://dx.doi.org/10.1093/infdis/jir515, PMid:21881123

London International Development Centre, n.d., *New approaches towards international development*, viewed 09 May 2012, from http://www.lidc.org.uk/

London School of Hygiene & Tropical Medicine, n.d., *Research centres and groups*, viewed 09 May 2012, from http://www.lshtm.ac.uk/research/centres-groups/

Malaria Capacity Development Consortium, n.d., *Malaria Capacity Development Consortium*, viewed 09 May 2012, from http://www.mcdconsortium.org/

Meisser, A., Schelling, E. & Zinsstag, J., 2011, 'One health in Switzerland: A visionary concept at a crossroads?', *Swiss Medical Weekly* 141, w13201.

Southern Africa Centre for Infectious Disease Surveillance (SACIDS), n.d., *Southern Africa Centre for Infectious Disease Surveillance*, viewed 09 May 2012, from http://www.sacids.org/kms/frontend/index.php?m=3

TubeBerculosis Vaccine Initiative (TBVI), n.d.a, *TBVAC*, viewed 09 May 2012, from http://www.tbvi.eu/projects/tbvac.html

TubeBerculosis Vaccine Initiative (TBVI), n.d.b, *NEWTBVAC*, viewed 09 May 2012, from http://www.tbvi.eu/projects/newtbvac.html

TuBerculosis Vaccine Initiative, n.d.c, *TuBerculosis Vaccine Initiative* (TBVI), viewed 09 May 2012, from http://www.tbvi.eu/home.html

Wellcome Trust, n.d., *African Institutions Initiative*, viewed 09 May 2012, from http://www.wellcome.ac.uk/Funding/International/WTX055734.htm

A foresight vision for infectious diseases in Africa

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How to cite this proceeding:
Brownie, J., 2012, 'A foresight vision for infectious diseases in Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #459, 4 pages. http://dx.doi.org/10.4102/ojvr.v79i2.459

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Introduction

In 2005, the United Kingdom (UK) through their Foresight programme, within the Government Office for Science (formerly the UK Office for Science and Innovation) launched a project to consider the future risks from infectious diseases for humans, animals and plants (http://www.bis.gov.uk/foresight/our-work/projects/published-projects/infectious-diseases). Whilst defining these risks, Foresight also explores the very best science that may be brought to transform our ability to manage the future challenges. A Report of the future threats was published in 2006 (Brownlie *et al.* 2006)

Following the published Report, we highlighted (King *et al.* 2006) that infectious diseases account for a quarter of all human mortality, and a similar fraction of morbidity (World Health Organisation 2005). Infectious diseases of crops and livestock cost the global economy uncounted billions of euros every year. On top of this, sudden epidemics of infectious diseases can deliver humanitarian and economic shocks on a scale difficult to absorb. According to the World Bank, the 2003 severe acute respiratory syndrome (SARS) epidemic, which killed fewer than 1000 people, was responsible for an estimated 2% fall in GDP across East Asia; an influenza pandemic could kill millions of people and cost 700 billion euros globally in a single year (European Union 2005). In recent years, there have been numerous outbreaks of livestock and crop diseases costing individual countries billions of euros, for example foot-and-mouth disease (FMD) in Taiwan and the UK; bovine spongiform encephalopathy (BSE) in the UK; Classical swine fever (CSF) in Netherlands; soybean rust in Brazil; southern corn blight in the United States (US); and most recently, avian influenza in Egypt. The UN Millennium Development Goals, as well as having explicit targets for reducing the burden of human diseases (particularly HIV and/or AIDS, tuberculosis and malaria), also have targets for reducing poverty and hunger, but these are compromised by crop and livestock diseases. In most developing regions, where the impacts of infectious disease are greatest, there is now little hope of meeting any of the Millennium Development Goals by 2015 (United Nations 2005).

It was clearly evident that outbreaks of disease can move rapidly and spread across a country, across regions and, in some cases, become global. The best strategy was to stop the disease 'in its tracks' as early as possible. For this to happen, we needed to be able to undertake extremely rapid detection and accurate identification of the pathogens; this would facilitate the correct control measures to be put in place, whether antimicrobials, vaccination or a culling policy. In those early stages, and thereafter, understanding where the outbreak is and its progress through the region is critical for control; thus effective monitoring surveillance and directed epidemiology provides the authorities with key information to target appropriate control measures.

The global nature and threat posed by many diseases is self-evident. For any foresightful look at their potential risk, account must be taken of their global prevalence and impact. With this in mind, the Project enlisted over 300 experts in some 30 countries for consultation and used a variety of methods, including Delphi studies (which use formal methods to generate forecasts from groups of experts), expert reviews, workshops, mathematical modelling and commissioned research.

The emerging and future risks from infectious diseases

At the conclusion of the final analysis, eight future categories of infectious diseases were identified as high risk and where improved detection systems would make a difference over the next 10–25 years.

- **New and emerging diseases**, such as SARS and BSE, and novel variants, such as H5N1 subtype influenza A, are anticipated to continue threaten nations.
- **Infections becoming resistant to treatment**, including antibiotic-resistant bacterial infections, such as tuberculosis and methicillin-resistant *Staphylococcus aureus* (MRSA) and those where mutation overcomes present vaccine protection, such as influenza.

- **Zoonoses**, that is, infections transferring to humans from animals, which can be associated with livestock, pets and, in many cases, with wildlife, for example, SARS, avian influenza, plague, Lyme disease and anthrax. This category includes foodborne infections such as *Escherichia coli* O157 or salmonella.
- **HIV and/or AIDS, tuberculosis and malaria**, the 'Big Three' tropical diseases covered by UN Millennium Development Goal 6.
- **Epidemic plant diseases**, such as cassava mosaic virus, coffee wilt disease and banana blight, currently of concern in East Africa.
- **Acute respiratory infections**, a category that covers pandemic influenza and a variety of other viral and bacterial infections.
- **Sexually transmitted infections (STIs)**, including but not only HIV and/or AIDS, which are increasing in incidence in many parts of the world.
- **Transboundary Animal diseases**, such as FMD, CSF and Newcastle disease, which remain amongst the most important barriers to international trade in livestock and livestock products.

International dimension of foresight and infectious diseases

It is obvious that global disease needs a global vision. To this end, the Project looked at infectious disease risks not just within the UK but also across sub-Saharan Africa and in China.

In sub-Saharan Africa, it was clear that the importance of infectious disease could not be greater (Rweyemamu, Otim-Nape & Serwadda 2006). It carries the heaviest burden of infectious disease of any region; the impact of HIV and/or AIDS, malaria and tuberculosis cannot be over-emphasised whilst the so-called 'neglected diseases' are ever-present and must not be forgotten. The seriousness of plant diseases is quickly seen on whole communities who solely rely on them for their own food and for nutrients for their livestock. The ultimate consequence of crop failure is starvation, rural migration, de-population of whole tracts of agricultural land but, at worse, it is death.

For livestock, the situation in some areas and some times, is not much better. Twelve of the 15 transboundary livestock diseases, formerly considered by the OIE as the most contagious (and which, in many countries, are notifiable to their government), are to be found in Africa. In the UK, they are all exotic. The situation in Africa has worsened in recent years with the further spread of diseases such as contagious bovine pleuropneumonia (CBPP), of Peste des petits ruminants virus (PPRV) and Rift Valley fever virus (RVFV).

This scourge of animal disease cuts deeper where there is poor governance and reduced infrastructure for the livestock extension services and limited diagnostic services. To compound the problem, these highly infectious diseases close all-important regional and global livestock markets that are so valuable to provide income back into the further development of agricultural communities.

The recent advances in technology have promoted rapid pen-side tests that have the potential to help with the diagnosis of infectious diseases. For sub-Saharan Africa, these tests, to be effective, will need to operate across a number of different countries, diverse systems of culture and governance and be cost effective; the question of global equity then becomes a critical issue.

For those of us involved with the sub-Saharan Africa Foresight project, it became clear that to have any long-term value or sustainability, a new paradigm for an integrated approach to control infectious diseases was needed. This gave rise to the 'pan-African Vision and Strategy for the management of infectious diseases' (Rweyemamu *et al*. 2006).

The mission statement, at that time, for a pan-African Vision and Strategy for the management of infectious diseases was:

> A pan-African concerted effort, shared by AU member governments reflecting the needs of the African society and supported by the international community, with the goal of society protecting from the ravages of dangerous infectious disease that compromise human, and animal and plant health, improved livelihoods, agriculture and economic development.

The birth of Southern African Centre for Infectious Disease Surveillance

The Foresight 'Detection, Identification and Monitoring of Infectious Diseases (DIM)' Project provided a number of findings which were taken up by a number of governments, agencies and organisations across the world. However, one of the outcomes that was pertinent only to sub-Saharan Africa, was the creation of a Centre for infectious diseases. In time, it was hoped that this would provide the African continent with a capability for diagnosis and identification of any new and emerging diseases; a capability similar to that provided by the Centres for Disease Control and Prevention (CDC) in Atlanta for the USA. Such a Centre would give greater 'ownership of disease' to African countries and thereby promote an African capability that would underpin the national and international control programmes, some of which were of only African interest.

To create such a Centre, we needed to start 'small' (in relative terms, for such an enormous continent), establish a local grouping of countries, find a vision and obtain some start up funds that would be independent of individual Government sponsorship.

The initial project was to join medical and veterinary institutions in Tanzania in a 'smart partnership' with relevant UK institutions (together to focus on human and animal diseases in a virtual Centre incorporating a small but selected number of Institutes, organisations and universities from the

two countries. The proposal *('Piloting African-UK Partnership through 'One- Medicine' Surveillance and Detection of Infectious Diseases in Tanzania')* was submitted for funding to the Wellcome Trust in the UK in 2008 with the Royal Veterinary College (Principal Investigator = Professor Joe Brownlie) as the UK Institution and with the Veterinary School at the University of Sokoine, Tanzania. This proposal failed to obtain funding but did start a series of actions both within the Wellcome Trust and within our Tanzanian collaborators – most notably Professor Mark Rweyemamu. The reasons for failure included the need for African leadership, wider country involvement and better 'take-up' by the medical sciences.

The invitation from the Wellcome Trust to reconsider the proposal required a deeper and more extensive African involvement. At this point, an inspired African leader took the reins and rode the proposal through and over the difficult cross-country obstacle race to win the race and be awarded the invaluable prize of Wellcome Trust funding. That leader was Professor Mark Rweyemamu and the ultimate prize was the creation of the Southern African Centre for Infectious Diseases and Surveillance (SACIDS) with 5 Southern African countries involved (Republic of South Africa, Tanzania, Zambia, Botswana, Mozambique, Democratic Republic of Congo) and a number of Institutions from London, UK (The Royal Veterinary College, the London School of Hygiene and Tropical Medicine and the London International Development Centre). The proposal to Wellcome Trust was entitled SACIDS.

The importance of Southern African Centre for Infectious Disease Surveillance

The main thrust of SACIDS is create a virtual Centre within Southern African countries with the capacity to provide effective diagnostic capability and research of international standard on infectious diseases, both medical and veterinary, and to provide training to the next generation of young African scientists with the long term ambition to populate the Centre and the relevant Institutions within the Southern African countries. This requires both new graduate courses in the molecular sciences and epidemiology to be designed, approved and initiated in SACIDS member universities and to rapidly incorporate the new technologies that would underpin national diagnosis and surveillance.

Returning to the Foresight programme for a moment (King *et al.* 2006), we can see that 'a wide range of technological advances from remote sensing to nanotechnology were reviewed and ultimately four were selected for detailed consideration. Novel information technologies for the capture, analysis and modelling of data are already being developed: these will allow data to be collected electronically from hand-held devices, or from remote sensors, and analysed and modelled in real time. Genomics and post-genomics approaches will allow the rapid characterisation of pathogens. Mass screening of people, animals and plants in transit should become feasible through non-invasive detection systems for volatile organic chemicals or atypical electromagnetic profiles. Portable devices will become available for diagnosing infections in individual patients, animals or plants, satisfying a growing demand for cheap, quick, easy-to-use, over-the-counter products, perhaps resembling today's home pregnancy test kits. Some of these technologies will be generic, such as 'lab-on-a-chip' screening for a range of infectious agents, non-specific diagnostics based on detecting activated immune responses, or simple tests to differentiate between viral and bacterial infections to aid the appropriate prescription of antibiotics.

Better disease detection capability is vital but will present challenges as well as opportunities. New technologies must be embedded within functional national or international surveillance systems. In practice, while the widespread use of self-administered tests, for example, could provide valuable data, it is not clear whether or how government agencies would gain access to it. Other kinds of information e.g., records from mobile phones or traffic cameras to follow movements, are potentially valuable for disease control purposes, particularly in outbreak situations, but the public might not accept their use for this purpose. There is always the danger that disease data could be used to discriminate against individuals without providing any benefit or compensation. Similarly, technologies deployed by developed countries could disadvantage developing countries by restricting travel and trade on the basis of the presence or even the suspicion of an infectious disease. Finally, everywhere in the world, better disease data might raise public alarm and expectations of effective action, whether or not this is realistic. This requires that surveillance is operationally linked to an appropriate response. For example, the Global Plan to Stop TB relies on the combination of case detection and directly observed therapy (DOTS) (World Health Organisation 2006) and plans to combat influenza involve surveillance, drug delivery and vaccine production (Fouchier *et al.* 2005).

Southern African Centre for Infectious Disease Surveillance has the real opportunity to co-ordinate the most important and impressive programme across the Southern African countries to combat infectious diseases in humans and Livestock. It needs to foster interdisciplinary approaches to infectious disease research that transcend both national and also traditional intellectual boundaries, such as those between medicine and veterinary medicine or between virology, bacteriology, mycology and parasitology. A better understanding of patterns of infectious disease also needs input from disciplines as diverse as anthropology, economics and climatology. Quantifying these relationships and understanding their dynamics requires inputs from statistics and mathematics to generate credible models. Health systems research is needed to understand how new technologies can be used most effectively, and must include consideration of the needs, expectations, capabilities and sensitivities of end users and other stakeholders (King *et al.* 2006).

SACIDS has already done what a couple of years ago would have been considered highly unlikely; with a little more effort over the next few years, it could achieve the impossible – a fully functional SACIDS that has morphed into a continental

sub-Saharan Centre for Infectious Disease Surveillance and Control.

References

Brownlie, J., Peckham, C., Waage, J., Woolhouse, M., Lyall, C., Meagher, L. *et al*., 2006, Foresight. Infectious Diseases: Preparing for the future Future Threats. Office of Science and Innovation, London, p. 1–83.

European Union, 2005, *Impact Assessment Avian Influenza*, viewed n.d., from http://archive.defra.gov.uk/

Fouchier, R., Kuiken, T., Rimmelzwaan, G. & Osterhaus, A., 2005, 'Global task force for influenza', *Nature* 435, 419–420. http://dx.doi.org/10.1038/435419a, PMid:15917779

King, D.A., Peckham, C., Waage, J.K., Brownlie, J. & Woolhouse, M., 2006, 'Infectious Diseases – Preparing for the future', *Science* 303, 1392–1393. http://dx.doi.org/10.1126/science.1129134, PMid:16959992

Rweyemamu, M., Otim-Nape, W. & Serwadda, D., 2006, *Foresight. Infectious Diseases: Preparing for the future, Africa*, Office of Science and Innovation, London, p. 1–120.

United Nations, 2005, *The Millennium Development Goals Report 2005,* UN, New York.

World Health Organization, 2006, Stop TB Strategy to fight the global tuberculosis epidemic, viewed n.d., from http://www.who.int/tb/features_archive/stop_tb_strategy_launch/en/index.html

From 'two medicines' to 'One Health' and beyond

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How to cite this proceeding:
Zinsstag, J., Meisser, A., Schelling, E., Bonfoh, B. & Tanner, M., 2012, 'From "two medicines" to "One Health" and beyond', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #492, 5 pages. http://dx.doi.org/10.4102/ojvr.v79i2.492

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

We first review historic and conceptual background to integrative thinking in medicine. Lacking a general theory of 'One Health', we provide an operational definition of 'One Health' and its leverage as: any added value in terms of human and animal health, financial savings or environmental benefit from closer cooperation of human and animal health sectors at all levels of organisation. Examples of such added value of 'One Health' are given from the fields of health systems, nutrition and zoonoses control in Africa and Asia.

'One Health' must become main-stream rather than a new discipline or new association; it should just become normal that practitioners and professionals in the health, animal and environment sectors work together as closely as possible. Current and future challenges in financing clean energy, migration flows, food security and global trade further warrant rethinking of human and animal health services. A conceptual outlook relates health as an outcome of human-environment systems called 'health in social-ecological systems'. The paper ends with an outlook on the operationalisation of 'One Health' and its future potential, specifically also in industrialised countries.

Introduction

The present paper is summarising and extending an invited key note, given as a video presentation to the first African 'One Health' conference, held in Johannesburg on July 14 and 15, 2011 by the first author (http://www.sacids.org/kms/frontend/?m=103). The human and animal health research unit in the department of Epidemiology and Public Health at the Swiss Tropical and Public Health Institute (http://www.swisstph.ch) entertains partnerships with countries in East–Africa, Central–Africa and West–Africa, Central Asia and Switzerland. Here we account for conceptual and practical research work on collaborative efforts between human and animal health, between developing and industrialised countries involving disciplines as diverse as epidemiology, anthropology, cultural sciences, sociology, geography, molecular biology, statistics and mathematics. Initially the focus of the research group was on the provision of health care to mobile pastoralists, who are nearly devoid of health services. However, soon questions on the control of zoonoses, diseases transmissible between animals and humans, like bovine tuberculosis, rabies, brucellosis, anthrax and avian influenza, came in the focus of attention.

The research group is part of several larger international research networks. It is the health partner in the National Centre of Competence in Research North-South (http://www.north-south.unibe.ch), together with seven Swiss research institutions and their partners on all continents except Australia. The network covers research partnerships on natural resource management, conflict transformation, governance, water and sanitation, livelihood, urban planning and health and is jointly funded by the Swiss National Science Foundation (SNSF) and the Swiss Agency for Development and Cooperation (SDC). In this network, health research is a component of a larger development research approach for which we currently work on integrated methods involving inter- and transdisciplinary approaches. The unit is also part of the European Union funded network on integrated control of neglected zoonoses in Africa (http://www.iconzafrica.org). It involves twenty European and African partner institutes. Further, the unit collaborates in Wellcome Trust funded projects on bovine tuberculosis in Africa which evolved into an African Capacity Building program 'Ecosystem and population health: bridging the frontiers in health' (http://www.afriqueone.net). The last network is the Consortium One Health Next Generation OH-NEXTGEN with a strong focus on training young fellows to change the mindset in health system considering health problem at the human, animal and environment interface in different eco-zones. Networks have proven essential for reassembling critical mass, inter- and transdisciplinary collaboration, North-South, South-South and East-West exchanges and comparisons, yielding typically a higher output of research outcomes as could be achieved if each partner worked alone.

We provide first a brief historic and conceptual background to integrative thinking in medicine. We will then provide an operational definition of 'One Health' and its leverage. Examples of the

leverage of 'One Health' are given from the fields of health systems, nutrition and zoonoses control in Africa and Asia. Prior to further conceptual extensions the unfinished 'One Health' agenda will be described. The paper ends with an outlook on how to make 'One Health' operational and its future potential, specifically also in industrialised countries.

Brief history of integrative thinking in medicine and health

We are often asked what is new about 'One Health'. Actually nothing, close interactions between human and animals are longstanding but vary by their historical intensity and cultural background. We provide here examples from a more detailed account (Zinsstag *et al.* 2011). In the ancient Egyptian culture humans and their animals were seen as belonging to one 'flock of God'. Transformation from humans to animals (metempsychosis) are known from India and Africa. Fulbe pastoralists in Africa, in their myths of creation, see cattle as being an integral part of their society (Louanges à la femme 1966). The Zhou Dynasty in China (11th century – 13th century) maintained the first integrated public health system including medical doctors and veterinarians. The Chinese Scholar Xu Dachun stated already in the 18th century, 100 years earlier than Rudolf Virchow that, 'the foundations of veterinary medicine are as comprehensive and subtle as those of human medicine and it is not possible to place one above the other' (Driesch & Peters 2003). Whilest human medicine became a faculty in the medieval European universities (Rüegg 2004), veterinary medicine remained in the hand of equerries, the persons in charge of the horses for warfare, until Claude Bourgelat, founded the first veterinary school in Lyon (1761). The end of 19th century with the advent of cellular pathology and microbiology was a period of very close interaction of human and animal health as comparative medicine. One of its protagonists, Rudolf Virchow, stated in an address to the Prussian government on bovine tuberculosis, 'between animal and human medicine there is no dividing line – nor should there be'. The object is different, but the experience obtained constitutes the basis of all medicine. 'In the twentieth century veterinary and human medicine evolved in a way as to specialise into more and more sub-disciplines and the influence of comparative medicine decreased. The American epidemiologist Calvin Schwabe, influenced by his work with Dinka pastoralists in Sudan coined the term 'One Medicine' in the 1960s. It means that, there is no difference of paradigm between human and veterinary medicine. Both sciences share a common body of knowledge in anatomy, physiology, pathology, on the origines of diseases in all species. We can thus conclude that the modern formulation of 'One Medicine' has African roots. In the past decades, 'One Medicine', addressing more and more public and environmental health issues became 'One Health'(Zinsstag *et al.* 2005) and has seen unprecedented revival at the level of international organisations, national governments and academia (Zinsstag *et al.* 2009b) after the outbreaks of major diseases (SARS, Avian Flu, Swine fever …).

Operational definition of 'One Health' and its leverage

The scholarly statements on 'One Medicine' mentioned above have been replaced by an ongoing debate on contemporary definitions and delimitation of what has become 'One Health'. In the past conferences, in particular at the first 'One Health' conference in Melbourne in February 2011, many presenters limited themselves to recognising the interdependence of humans and animals and their environment. In our view, this is a necessary component of 'One Health', but only part of it. We lack a modern and internationally acknowledged theory of 'One Health', which may require an in-depth epistemological assessment of all involved disciplines. We propose here a pragmatic operational definition of 'One Health' as any added value in terms of human and animal health, financial savings or social and environmental benefits from closer cooperation of professionals in the health, animal and environment sectors at all levels of organisation. Claiming a 'One Health', in our view, requires the demonstration of added value to what human and animal health working alone can achieve. Specifically a 'One Health' approach is capable of identifying points of leverage of health of humans and animals from a systemic analysis.

Examples of the leverage of 'One Health'

The presented examples are published as case studies on zoonoses epidemiology, nutrition and public health services. Most often zoonoses are investigated either in humans or animals. In the case of zoonotic diseases that are transmissible between humans and animals, integrated study designs investigating health status in humans and animals simultaneously allow an instantaneous identification of the source of a zoonotic disease. For example, in Chadian pastoralist human Q-fever to camels (Schelling *et al*. 2003).

Human brucellosis can be eliminated by interventions in animals. From a public health point of view, mass vaccination of livestock to prevent human brucellosis is not profitable in Mongolia. But if societal benefits are summed up, including benefits for private households and the livestock sector, the intervention is largely profitable. If costs of brucellosis mass vaccination are shared between the health and livestock sector proportional to their benefits, brucellosis control becomes highly cost-effective (Roth *et al.* 2003). Similarly, the cumulative cost of dog rabies mass vaccination and human post-exposure treatment (PET) in N'Djaména, Chad reaches break-even with the cumulative cost of PET alone after six years (Zinsstag *et al.* 2009a). Such comparative assessments can only be made if human and animal health is investigated as a single social-ecological disease system. A shared veterinary laboratory to diagnose brucellosis in febrile patients has brought the collaborating physician in Mali to include brucellosis testing as a differential diagnosis to malaria and typhoid fever in an area where raw milk consumption is still prevalent (Steinmann *et al.* 2005).

Pastoralists in Africa depend highly on milk from their animals for their nutrition and the vitamin A status of mobile pastoralist women and children in Chad depends directly on the vitamin A levels in the milk of their cows. In the same way a study on the vaccination status of mobile pastoralist children, women and their animals showed that the vaccination coverage of livestock was much higher than that of children and women. Joining the vaccination campaigns between the veterinary and public health services reduced the logistic cost by 15% and improved vaccination coverage of children and women, who have otherwise no access to health care (Bechir *et al*. 2004; Schelling *et al.* 2005). Work with pastoral communities heavily relayed on collaboration with cultural scientists, who lived for example with Kel Tamacheq communities in North Mali. Fluency in local languages and coranic literacy were critical for creating a trustful relationship. Informations and data on mother and child health seem to be more accurate from participant observation than from clinical surveys by a medical doctor (Münch *et al*. 2007). In this way a 'One Health' approach recognises the need for collaboration between medical and cultural sciences.

The unfinished 'One Health' agenda

The above examples clearly show an added value of closer cooperation between human an animal health for the understanding of the human animal linkage by taking more a societal perspective rather than a public health point of view only. It shows how interventions become profitable or public and animal health status can be improved. Much of this dynamic has been taken up but there remain still a huge unfinished agenda (Zinsstag *et al.* 2009b).

A recent outbreak of Q-fever in the Netherlands (Enserink 2010a & 2010b) has shown the current limitations of communication between the animal and public health surveillance system. There are obvious reasons why surveillance systems of communicable diseases for humans and animals should be coupled in a single cooperative surveillance system, which informs on outbreaks in all different species simultaneously to the whole system. This would, as the Dutch Q-fever example shows, reduce time to detection and time to intervention significantly. The control of Rift Valley Fever, another epidemically occurring zoonosis, would largely benefit from joint contingency planning where roles of each sector, how information flows and cost-sharing schemes are jointly decided on based on evidence before an outbreak. Similarly there are great public health opportunities in merging human and animal cancer registries (O'Brien *et al.* 2000). Geo-referenced detection of cancer incidence in one species could reveal environmental exposure for the other species. Canada is spearheading such approaches by its joint surveillance of antimicrobial resistance (CIPARS, http://www.phacaspcgc.ca/cipars-picra/index-eng.php) or the integrated surveillance of enterobacteriacea (C-enternet). The human-animal bond has far reaching consequences in the case of non-communicable disease like depression or obesity (O'Haire 2010). Systemic approaches, well known from pastoral counseling (Van Katwyk 2005) or family therapy could be extended to health care for humans and their pets or pets and their holders (the human-animal bond as an entity). Respiratory problems of a dog may be associated to smoking behavior of the dog holder (Reif *et al.* 1992). Obesity of a pet may be associated to a health problem of its owner, and hence the owner's care determines the pet animal's health. New ways of communication between clinical veterinarians and family doctors require a dialogue and negotiation as to when an interaction makes sense and may lead to improved health of animals and their humans.

To prevent fears of institutes being absorbed by larger ones, public and animal health systems should cooperate as equal rights partners respecting each others technical field of competence.

'One Health' as a mindset, must become mains-stream, rather than a new discipline or institution, it must become normal that professionals throughout all relevant disciplines (e.g. physicians, veterinarians, social scientists and ecologists) work together as closely as possible. Current and future challenges in financing, clean energy, increasing migration flows, food insecurity and global trade further warrant rethinking of human and animal health services (see below). The above 'unfinished' agenda is also incomplete and warrants each actor's imagination as to how interactions between the two medicines can yield better health for all.

Conceptual extensions of 'One Health' towards systemic approaches

The closer cooperation between human and animal health has been extended since over a decade by including ecological and eco-systemic aspects, known as eco-system health (http://www.ecohealth.net), recognising inter-dependency of health of humans and animals and the integrity of eco-systems (Forget & Lebel 2001; Lebel 2002). Systems biology, previously concerned mainly with complex processes at cellular and sub-cellular level recognise extensions at higher scales up to populations, for example for explaining the development of persistent infections and phylo-geographic lineages in tuberculosis (Gagneux & Small 2007; Young, Stark & Kirschner 2008). Environmental sciences and work on natural resource management use conceptual approaches called Social-Ecological Systems (Ostrom 2007) or Human-environment systems, which can easily be applied to a systemic approach to health e.g. in the management of Bovine Spongiform Encephalitis (BSE) in Switzerland (Scholz 2011). Studying Health in Social-Ecological Systems (HSES) opens new ways addressing complex, multivariable, nonlinear, cross-scale and dynamic factors determining the health of humans and animals (Zinsstag *et al.* 2011). HSES formally include social sciences and humanities in health research but require further work on epistemological bridges between humanities, economics and natural sciences.

As an example on health in a social-ecological system, we can mention the interactions of the socio-political and ecological

changes from a planned economy to a market economy in Mongolia in 1990, causing the breakdown of public health and veterinary disease control systems. In the same time the privatised livestock production led to a sharp increase of livestock numbers, pasture degradation and animal disease like foot-and-mouth disease (FMD) and brucellosis, which is transmissible to humans. Effective reduction of brucellosis incidence in humans requires interventions in livestock. Understanding livestock demographic dynamics becomes a key for the planning of sustainable pasture management and the planning of animal health interventions. Seeking a stabilisation of the Mongolian livestock population in Mongolia will be an important element to preserving pastures. Potential freedom of important zoonotic and transboundary diseases will provide market access and help stabilising livestock population by increasing offtake. All these complex social-ecological processes determine the health of humans and animals but depend on political and societal forces engaging their respective interests. Further work aims at demonstrating added value of a systemic approach on overall societal burden and cost of disease and its control, while preserving ecosystem services and social stability using transdisciplinary approaches.

Operationalisation of 'One Health'

We have shown that 'One Health' is well integrated into broader conceptual thinking like 'Ecosystem health' or 'Health in Social-Ecological Systems'. Whilst research can disentangle the complex interactions between health, society and ecosystems, demonstrating central points of leverage for future interventions, governments and international agencies aim primarily at making 'One Health' work in practice.

There is not a blueprint for making 'One Health' operational and there are multiple actors involved and require stakeholders' involvement, long term partnerships, capacity-building, but also local champions. Most of the activities in industrialised countries are mirrored by the 'One Health' initiative website (http://www.onehealthinitiative.org). The World Bank for example engages in the study of structural savings from institutional planning by joining surveillance or laboratory capacity. Academic curricula teaching 'One Health' are developed by several universities, for example the University of Calgary in Canada and imply the development of methods for 'One Health' research (Zinsstag *et al.* 2009b). Research for development shows that operational models of 'One Health' require transdisciplinary processes (Schelling *et al*. 2007). Academic research extends processes to improved understanding of the interactions by involving stakeholders like communities, authorities and experts for the identification of locally acceptable and adapted health interventions. All processes need to be negotiated between actors since each context is different (Meisser, Schelling & Zinsstag 2011; Schelling *et al.* 2005; Schelling *et al.* 2007). Contextual solutions will address the importance of cultural determinants of the human animal relationship. As an example we can mention the dog, which in some cultures has mainly an emotional value with strong human-dog bonds and in others a commodity with commercial value as food supply. Researchers and planners aiming at making 'One Health' operational require high level self reflexive capacity in recognising inter-cultural aspects of the human-animal relationship. Key outcomes of a closer cooperation of human and animal health will remain economic savings, health benefits for humans and animals and ecological benefits. Not all outcomes such as improved communication and information flows between sectors with subsequent e.g. earlier detection of a disease or appropriate measure can be easily quantified, but can still be captured with qualitative attributes.

The potential is endless and potential savings go in the billions, if doctors and veterinarians communicate and interact in a closer way. But we are also very much aware that those in charge of national planning would want to be re-assured by examples of cost saving potential, if not from their own country, at least from a country in their region.

Acknowledgment

We acknowledge the collaboration of all members of our research unit and the collaborators at Swiss TPH. We gratefully acknowledge our partners in Chad, Ethiopia, Mali, Mauritania, Côte d'Ivoire, Kyrgyzstan and Mongolia. Lisa Crump thanked for language editing. This paper has been supported by NCCR North-South, EU FP7 ICONZ Africa and the consortium Afrique One funded by the Wellcome Trust.

References

Bechir, M., Schelling, E., Wyss, K., Daugla, D.M., Daoud, S., Tanner, M. & Zinsstag, J., 2004, 'An innovative approach combining human and animal vaccination campaigns in nomadic settings of Chad: Experiences and costs', *Medecine Tropicale (Marseilles)* 64, 497–502.

Driesch A. & Peters J., 2003, *Geschichte der Tiermedizin* [History of Veterinary Medicine}, Stuttgart, Schattauer.

Enserink, M., 2010a, 'Infectious diseases. Humans, animals--it's one health. Or is it?', *Science* 327, 266–267. http://dx.doi.org/10.1126/science.327.5963.266-a

Enserink, M., 2010b, 'Infectious diseases. Questions abound in Q-fever explosion in the Netherlands', *Science* 327, 266–267. http://dx.doi.org/10.1126/science.327.5963.266-a

Forget, G. & Lebel, J., 2001, 'An ecosystem approach to human health', *International Journal of Occupational and Environmental Health* 7, S3–38.

Gagneux, S. & Small, P.M., 2007, 'Global phylogeography of Mycobacterium tuberculosis and implications for tuberculosis product development', *Infectious Diseases* 7, 328–337.

Lebel, J., 2002, *Health: An ecosytem approach*, Ottawa, Canada.

Louanges à la femme / 'Dieu a des richesses, j'ai des vaches!' [Praise be to the woman / 'God of wealth, I have cows!], 1966, In Sow, A. I., ed. La Femme, la Vache, la Foi - Ecrivains & Poètes du Foûta-Djalon. Julliard, 285–335.

Meisser, A., Schelling, E. & Zinsstag, J., 2011, 'One health in Switzerland: A visionary concept at a crossroads?', *Medical Weekly* 141, w13201.

Münch, A., Diallo, B., Bonfoh B., Mohamed, E.A., Doumbia, S., Diop, S.T. & Zinsstag J., 2007, 'The meaning and representation of illness, health and curing among the Kel Tamasheq in northern Mali', *Tropical Medicine & International Health* 1(12), 204.

O'Brien, D.J., Kaneene, J.B., Getis, A., Lloyd, J.W., Swanson, G.M. & Leader, R.W., 2000, 'Spatial and temporal comparison of selected cancers in dogs and humans, Michigan, USA, 1964-1994', *Preventive Veterinary Medicine* 47, 187–204. http://dx.doi.org/10.1016/S0167-5877(00)00168-9

O'Haire, M, 2010, 'Companion animals and human health: Benefits, challenges, and the road ahead', *Clinical Applications and Research* 5, 226–234.

Ostrom, E, 2007, 'A diagnostic approach going beyond panaceas', *Proceedings of the National Academy of Sciences* 104, 15181–15187. http://dx.doi.org/10.1073/pnas.0702288104, PMid:17881578

Reif, J.S., Dunn, K., Ogilvie, G.K. & Harris, C.K., 1992, 'Passive smoking and canine lung cancer risk', *American Journal of Epidemiology* 135, 234–239. PMid:1546698

Roth, F., Zinsstag, J., Orkhon, D., Chimed-Ochir, G., Hutton, G., Cosivi, O., Carrin, G. & Otte, J., 2003, 'Human health benefits from livestock vaccination for brucellosis: Case study', *Bulletin World Health Organisation* 81, 867–876. PMid:14997239

Rüegg W., 2004, *A history of the University in Europe*, Cambridge University Press, Cambridge.

Schelling, E., Diguimbaye, C., Daoud, S., Nicolet, J., Boerlin, P., Tanner, M. & Zinsstag, J., 2003, 'Brucellosis and Q-fever seroprevalences of nomadic pastoralists and their livestock in Chad', *Preventive Veterinary Medicine* 61, 279–293. http://dx.doi.org/10.1016/j.prevetmed.2003.08.004, PMid:14623412

Schelling, E., Wyss, K., Béchir, M., Moto, D.D. & Zinsstag, J., 2005, 'Synergy between public health and veterinary services to deliver human and animal health interventions in rural low income settings', *British Medical Journal* (331), 1264–1267. http://dx.doi.org/10.1136/bmj.331.7527.1264, PMid:16308393, PMCid:1289333

Schelling, E., Wyss, K., Diguimbaye, C., Bechir, M., Taleb, M.O., Bonfoh, B., Tanner, M. & Zinsstag, J., 2007, 'Toward Integrated and Adapted Health Services for Nomadic Pastoralists and their Animals: A North-South Partnership', in G. Hirsch Hadorn, H. Hoffmann-Reim, S. Biber-Klemm, W. Grossenbacher, D. Joye, C. Pohl, U. Wiesmann & E. Zemp (eds.), *Handbook of Transdisciplinary Research. A Proposition by the Swiss Academies of Arts and Sciences*, pp. 277–291, Springer, Heidelberg.

Scholz, R.W., 2011, *Environmental Literacy in Science and Society: From Knowledge to Decisions*, Cambridge University Press , Cambridge UK.

Steinmann P., Bonfoh, B., Farah, Z., Peter O., Schelling E., Traore M. & Zinsstag, J., 2005, 'Seroprevalence of Q-fever in febrile individuals in Mali', *Tropical Medicine & International Health* 10(6), 612–617. http://dx.doi.org/10.1111/j.1365-3156.2005.01420.x, PMid:15941426

Van Katwyk, P., 2005, *Spiritual care and therapy: Integrative perspectives,* Wilfried Laurier University Press, Waterloo ON, Canada.

Young, D., Stark, J. & Kirschner, D., 2008, 'Systems biology of persistent infection: tuberculosis as a case study', *Nature Reviews Microbiology* 6, 528. http://dx.doi.org/10.1038/nrmicro1919, PMid:18536727

Zinsstag, J., Durr, S., Penny, M.A., Mindekem, R., Roth, F., Menendez Gonzalez, S., *et al*., 2009a, 'Transmission dynamics and economics of rabies control in dogs and humans in an African city', *Proceedings of the National Academy of Science* 106, 14996–15001. http://dx.doi.org/10.1073/pnas.0904740106, PMid:19706492, PMCid:2728111

Zinsstag, J., Schelling, E., Bonfoh B., Fooks, A.R., Kasymbekov, J., Waltner-Toews, D. & Tanner, M., 2009b, 'Towards a "one health" research and application tool box', *Veterinaria Italiana* 45, 121–133. PMid:20391395

Zinsstag, J., Schelling, E., Waltner-Toews, D. & Tanner, M., 2011, 'From "one medicine" to "one health" and systemic approaches to health and well-being', *Preventive Veterinary Medicine* 101, 148–156. http://dx.doi.org/10.1016/j.prevetmed.2010.07.003, PMid:20832879, PMCid:3145159

Zinsstag, J., Schelling, E., Wyss, K. & Mahamat, M.B., 2005, 'Potential of cooperation between human and animal health to strengthen health systems', *Lancet* 366(9503), 2142–2145. http://dx.doi.org/10.1016/S0140-6736(05)67731-8

A socio-economic approach to One Health policy research in southern Africa

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How to cite this proceeding:
Kayunze, K.A., Kiwara, A.D., Lyamuya, E., Kambarage, D.M., Rushton, J., Coker, R. *et al.*, 2012, 'A socio-economic approach to One Health policy research in southern Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #460, 8 pages. http://dx.doi.org/10.4102/ojvr.v79i2.460

One-health approaches have started being applied to health systems in some countries in controlling infectious diseases in order to reduce the burden of disease in humans, livestock and wild animals collaboratively. However, one wonders whether the problem of lingering and emerging zoonoses is more affected by health policies, low application of one-health approaches, or other factors. As part of efforts to answer this question, the Southern African Centre for Infectious Disease Surveillance (SACIDS) smart partnership of human health, animal health and socio-economic experts published, in April 2011, a conceptual framework to support One Health research for policy on emerging zoonoses. The main objective of this paper was to identify which factors really affect the burden of disease and how the burden could affect socio-economic well-being. Amongst other issues, the review of literature shows that the occurrence of infectious diseases in humans and animals is driven by many factors, the most important ones being the causative agents (viruses, bacteria, parasites, etc.) and the mediator conditions (social, cultural, economic or climatic) which facilitate the infection to occur and hold. Literature also shows that in many countries there is little collaboration between medical and veterinary services despite the shared underlying science and the increasing infectious disease threat. In view of these findings, a research to inform health policy must walk on two legs: a natural sciences leg and a social sciences one.

Introduction

Infectious diseases which occur in an epidemic or explosive form attract national, regional or international attention because of their propensity for causing high morbidity and rapid transboundary spread across national borders or even across continents and because of their potential for causing high mortality in affected populations and national and/or international socio-economic impacts. Those that occur in endemic form or cause chronic disease tend to attract less public attention, although locally they might even be of a higher socio-economic impact (Maudlin, Eisler & Welburn 2009). This latter category includes many of what have been referred to as 'neglected' or 'lingering' zoonoses. In general most of the newly recognised emerging or re-emerging infectious diseases of humans or animals have tended to be of the epidemic or transboundary type (World Bank 2010). An emerging disease may be defined as one 'that is newly recognized or newly evolved, or that has occurred previously but shows an increase in incidence or expansion in a geographical, host or vector range' (FAO/OIE/WHO 2004). The majority (i.e. about 60%) of all infectious diseases of humans and most (i.e. about 75%) emerging infectious diseases of humans have been shown to have an animal origin, and thereby of zoonotic nature (Jones *et al.* 2008; Taylor, Latham & Woolhouse 2001; Otte *et al.* 2007; Woolhouse & Gowtage-Sequeria 2005, cited by Shaw 2009). Examples of such emerging diseases include Ebola, avian influenza, pandemic influenza, human immunodeficiency virus (HIV) and AIDS, bovine spongiform encephalitis (BSE) and the Nipah virus. Examples of the so-called neglected or lingering zoonoses include anthrax, bovine tuberculosis (TB), brucellosis, cysticercosis and neurocysticercosis, cystic echinococcosis or hydatid disease, rabies, zoonotic sleeping sickness or human african trypanosomosis (HAT), and food-borne zoonoses, including *Salmonella* (salmonellosis), *Campylobacter* (campylobacteriosis), and *Escherichia coli* (colibacillosis) infections of animal origin affecting millions of people annually.

In order to address the infectious disease burden effectively – especially in developing countries – there is, therefore, an increasing body of opinion that advocates not only an enhanced collaboration between the human and veterinary medical sectors (i.e. the so-called one medicine, Schwabe 1969) but also inter-sectoral collaboration across the public, animal and environmental health sectors, involving both the natural and social sciences (Coker *et al.* 2011; Zinsstag *et al.* 2011). In this regard

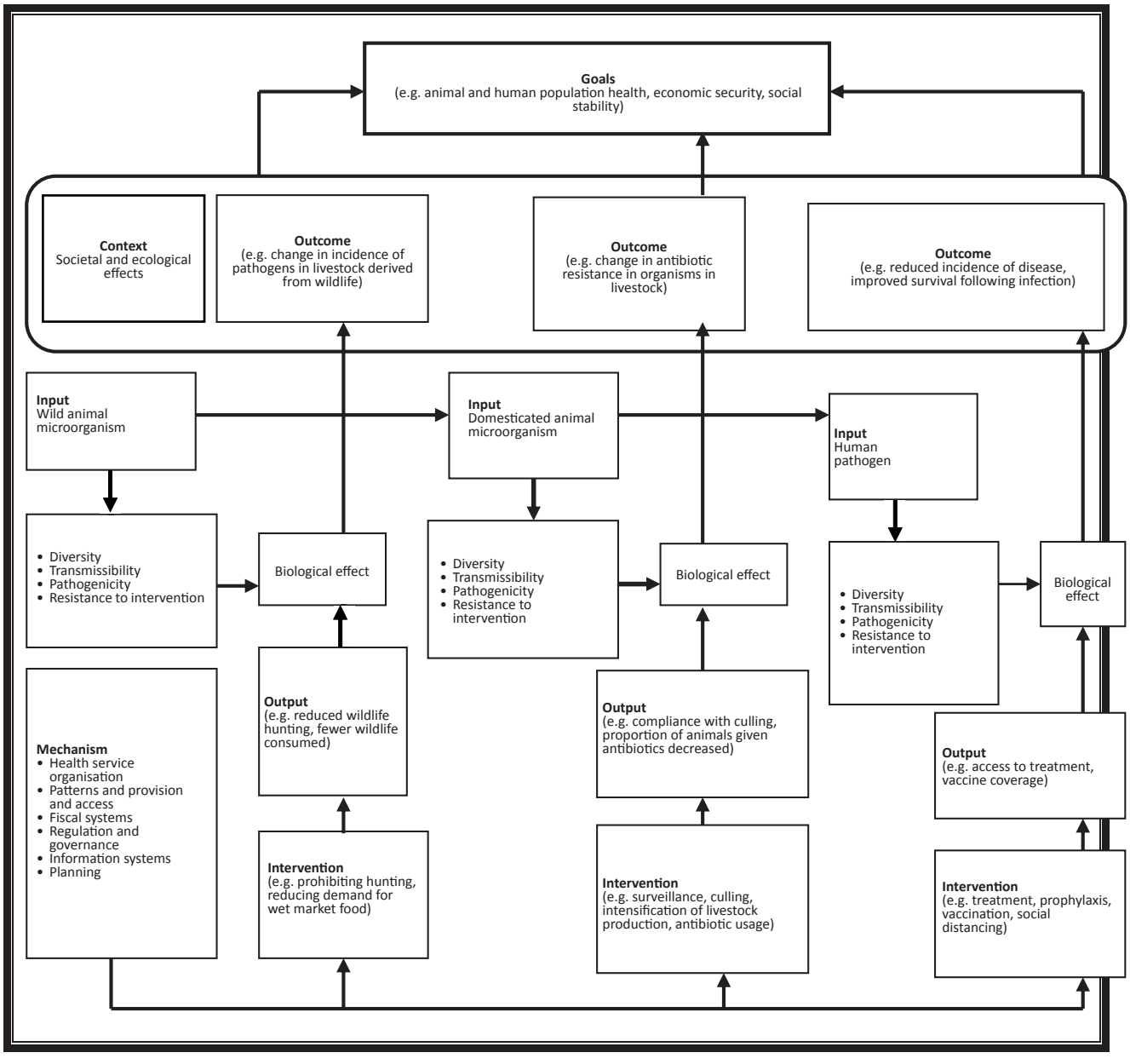
Note: Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

current health policies, whether for human or animal health, need some re-examination as to their fitness-for-purpose for the One Health approaches. As part of an effort to generate evidence to inform policy development for re-examining how existing health systems are structured, resourced, and managed to create synergies between animal and human health, and in the process to reduce the effect of zoonotic disease burdens, the SACIDS smart partnership of human health, animal health and socio-economic experts published, in April 2011, a conceptual framework to support one-health research for policy on emerging zoonoses (Coker *et al*. 2011).

The present paper builds on this framework to examine factors that have been reported to affect the burden of disease and how such a burden affects socio-economic well-being so that research projects, especially in sub-Saharan Africa, could be geared towards analysing some of the linkages documented in literature. The specific objectives were to, (1) analyse how interactions amongst wildlife, livestock, and humans lead to occurrence of infectious diseases, (2) explore the application of One Health approaches to controlling infectious diseases, (3) investigate how One Health approaches, theoretical factors, health policies, and socio-economic factors explain burden of disease and (4) illustrate the impact of burden of disease on poverty. The findings were intended to generate empirical information on which advocacy might be based for more collaboration, and how to realise it, for more effective control of lingering and emerging infectious diseases.

Materials and methods

The starting point for this paper was the SACIDS conceptual framework described by Coker *et al*. (2011), presented in Figure 1. The main assumption for the framework is that wild



Source: Coker, R., Rushton, R., Mounier-Jack, S., Karimuribo, R., Lutumba, P., Kambarage, D. *et al.*, 2011, 'Towards a conceptual framework to support one-health research for policy on emerging zoonoses', *The Lancet Infectious Diseases* 11, April, 326–331. http://dx.doi.org/10.1016/S1473-3099(10)70312-1

FIGURE 1: A framework for research to inform One-Health policy.

animals are reservoirs of pathogens and that the pathogens can spread to humans directly or indirectly. As seen in Figure 1, biological interventions can be applied at the following three levels: prevention of pathogens against crossing from wild animals to livestock; prevention of pathogens which are in livestock from crossing to humans and once the pathogens are in humans, controlling them to reduce morbidity and mortality in humans. These interventions can result in biological improvements at three levels of:

- a change in incidence of pathogens in livestock derived from wildlife
- a change in antibiotic resistance in organisms in livestock
- reduced incidences of disease and improved survival following infection.

Success in those interventions is expected to contribute to attainment of the highest level of success, which is having healthy animal and human populations, economic security, and social stability. However, that highest level of success can hardly be attained without active participation of other stakeholders. That is why in the SACIDS framework such stakeholders have been identified, as seen under the titles 'Context' and 'Mechanism'. The stakeholders are other natural scientists (besides veterinary and medical personnel), ecologists and agricultural scientists to address environmental and agricultural issues; socio-economists to address societal issues including human behaviour and economic issues and policy-makers to address health service organisation issues, patterns and provision and access, fiscal systems, regulation and governance, information and planning aspects.

Findings from literature

Wild Animals-Livestock-Humans Interactions and Disease Occurrence

Infectious diseases can spread directly or indirectly from one person to another, one animal to another, or from animals to persons and vice versa. Wild animals are known to be reservoirs of pathogens some of which may not affect them due to their genetic make-up and adaptation to wild conditions, albeit some of the pathogens can cause disease in livestock and in humans. But infectious diseases can also cross from either humans to wild animals (e.g. human TB) or from livestock to wildlife (e.g. bovine TB). A major transboundary animal disease of cattle that used to cause heavy mortality in wild ungulates, whose elimination from the Maasai eco-system of Tanzania and Kenya resulted in a progressive increase in the population of wildlife, notably the wildebeest in the Serengeti (Kock 2003) and whose global eradication depended on concerted action only in the cattle population, was rinderpest (FAO & OIE 2011).

Interactions amongst wildlife, livestock and humans can favour the spread of the pathogens either directly to people through contact with wild animals harbouring the pathogens, contact with contaminated wild products, or consuming wildlife products, including bush-meat. These interactions are best explained with the aid of a diagram like the one in Figure 2 by Institutes of Medicine (2009, in WHO 2010). A major inference from Figure 2 is that interactions amongst the members of the natural ecosystems (e.g. human encroachment and land use, etc), food and agriculture systems (e.g. expanding agricultural production, etc.), and human living environments (including increasing population density and growth, etc.) can lead to disease occurrence or infection spread. In their analysis of interaction trends amongst wildlife, livestock and humans, Jones *et al.* (2008) concluded that the majority of emerging infectious diseases of humans (71.8%) originate from wildlife.

The interactions in Figure 2 are also explained by two theories:

- the Island biogeography theory
- the parasite-stress theory of human sociality.

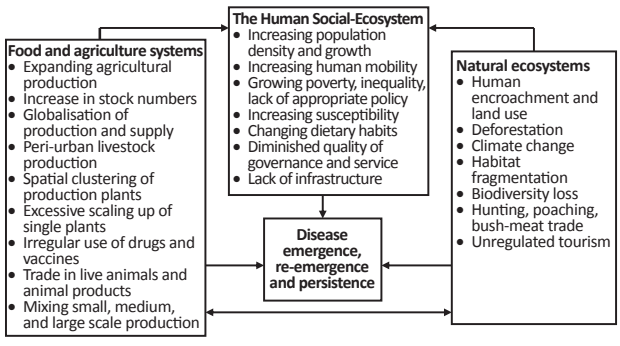
The former states that pathogens that lead to disease occurrence are identified at three levels of:

- interactions within species sources of pathogens
- interactions between recipient host species and species sources of pathogens
- interactions within recipient host species (MacArthur & Wilson 1967).

The latter states that humans' ontogenetic experiences with infectious diseases as well as their evolutionary historical interactions with these diseases exert causal influences on human psychology and social behaviour (Thornhill 2010). This theory emphasises the causal role of non-zoonotic parasites, which are characteristic of disease transmission from one person to another one, rather than zoonotic parasites which transmit diseases from vertebrate animals to humans.

Burden of disease in humans and animals

Burden of disease is a measure of financial cost, mortality, morbidity, or other indicators for humans and animals at the individual, community, herd or flock, farm, national, or global level due to diseases. It is normally measured in terms of Disability-Adjusted Life Years (DALYs). A DALY is equivalent to the loss of one year of 'healthy life' and allows the burden of disease in a population to be measured as the gap between current health and an ideal situation where everyone lives to old age, free of disease and disability (Mathers *et al.* 2001). This definition is from a medical point of



Source: Adapted from Institutes of Medicine (2009, cited by WHO 2010)

FIGURE 2: Interactions that may lead to disease occurrence.

view; it is in human medicine that burden of disease started being measured, and the measurement is described below.

Measurement of burden of disease in humans

Burden of disease in humans is measured in various ways using various indicators, which are presented in Table 1.

Although WHO publishes annual tables showing how many DALYs a year different diseases are estimated to cost, the zoonotic component of infectious diseases is largely missing from the league of tables (Coleman 2002, in Shaw 2009). In some works, total burden of disease is calculated; where this is done, conventionally, direct burden of disease in people is measured in DALYs, and all the other components (direct losses in animals and costs of prevention and treatment in people and animals) are measured in monetary terms (Brazier *et al*. 2007; Drummond *et al*. 2005; Shaw 2009).

Burden of disease in animals

Unlike in humans where assigning monetary values to people's losses of life complicates calculation of burden of disease, in animals the calculation is straightforward because most direct losses due to illness and due to death have objective monetary values (Shaw 2009). However, the calculation is complicated by the presence of many animal species which have various roles in the human society. In spite of the differences and complexities in calculating burdens of disease in humans and animals, Table 2 gives the ways of how to do the calculations.

Linkage between burden of disease and other factors

Collaborative efforts of many disciplines and experts in those disciplines to deal with infections diseases to reduce the burden of diseases in humans and animals is one thing; there are other factors which can enhance or constrain the pace towards controlling the diseases. In this paper, the other factors considered are health policies, the practice of health care services on the ground, theoretical factors, and socio-economic factors, which are discussed below.

Linkage between burden of disease and health policies

Health policy means different things to different people, but its compressive definition which is widely acceptable is given by Walt (1994) as a set of statements stipulating courses of action that affect the set of institutions, organisations, services, and funding arrangements of the health care system and goes beyond health services by including actions or intended actions by public, private and voluntary organisations that have an impact on health. She adds that health policy is concerned with environmental and socio-economic effects on health as well as with health care provision. However, many books on health policy focus narrowly on the health care system only. Therefore, some scholars, for example Nancy (1987, in Walt 1994), prefer talking about health public policy in order to differentiate the broader definition from the narrow one.

Health policies are affected by related international polices and by other policies which have nothing to do with health care or services for example, (1) environmental pollution, (2) insecurity and instability (whether caused by employment or violence), (3) economic regulation and deregulation and (4) contaminated water and poor sanitation, all of which increase morbidity and mortality. In view of this, the implementation of health policies may be constrained or enhanced by these other policies. Besides these, also cost-sharing affects health policies. For example, in Africa, it is widely known that the policy of cost sharing in both animal health and human health since the 1980s has complicated access to the services. This situation is well explained by Rushton and Leonard (2009) as follows: before the 1980s, particularly from the late 1940s, animal health had been regarded as a predominantly public service and thus was predominantly provided by governments. But since the 1980s the provision of the services has been increasingly opened to market institutions. However, animal health, like human health, is subject to market failures, and there remains a role for the state in their correction, through the provision of selected goods and services, the setting and monitoring of regulations and taxes and subsidies. In the human health sector cost-sharing has been characterised by people contributing for health

TABLE 1: Indicators for computing burden of disease in humans.

Direct losses due to ill health or death		Costs of treating and caring for those affected and costs of prevention	
Non-monetary losses	**Monetary losses**	**Households**	**Medical service**
DALY: The most widely used indicator to measure BoD, which includes two components: YLD and YLL	A proportion of patients' earned income or the value (opportunity cost) of their contribution to the running of their household is lost during illness	At the household level, patients and their families incur costs: • whilst seeking treatment and correct diagnosis • during the course of illness and follow-up	Medical service costs can usually be estimated relatively straightforwardly in terms of medical practitioners' time, costs of medicines, diagnostics, hospital days, etc.
YLD: Years of life lived with disability, both during illness and after recovery	Premature death leading to the loss of patients' whole future income and contribution to their household	These costs may be financial (transport to health facilities, cost of medicines, medical care), or they may be opportunity costs of time spent by family members accompanying and caring for the patient	Often it is necessary to work out the share of resources going to a particular group of patients for a particular disease
YLL: Years of life lost due to disease if it leads to premature death Age-weighting can be applied, whereby economically active adults' years receive a higher weighting	-	Households often also bear some of the costs of prevention (usually travel and time)	Costs of routine prevention (e.g. immunisation) can also be calculated

Source: Shaw, A.P.M., 2009, 'The economics of zoonoses and their control', in J. Rushton, *The Economics of Animal Health & Production*, pp. 161–167, CAB International, Oxfordshire & Massachusetts
DALY, Disability-Adjusted Life Years; BoD, burden of disease; YLD, Years Lived with Disability; YLL, Years of Life Lost.

TABLE 2: Items for calculation of burden of disease in terms of Disability-Adjusted Life Years.

Affected animals	Direct losses due to ill health or death		Costs of treating and caring for affected animals and costs of prevention	
	Non-monetary losses	Monetary	Animal keepers	Veterinary services
Livestock	Society values farming and the presence of livestock, particularly breed and species diversity	The steps involved in calculating the losses due to disease in livestock (valuing mortality and the components of morbidity)	Livestock keepers' costs consist of expenditure on veterinary pharmaceuticals, veterinary care and of, often very substantial, investments of livestock keepers' time	Veterinary public health services are involved in diagnosis, treatment, prevention (e.g. vaccination) and in food hygiene (e.g. abattoir inspections). Such costs are usually recorded
Companion animals	People derive psychological and health benefits from keeping companion animals, some of which could be quantified in DALYs	• Some companion animals, such as guard dogs, actually fulfil an economic role which could be quantified • Companion animals are bought and sold, and so have an economic price	In affluent countries substantial sums of money are spent on caring for companion animals. Owners' time and costs could thus be estimated	Public services are involved in dealing with zoonoses in companion animals. Such costs are usually recorded
Wildlife	Society values wildlife and places a particularly high value on rare and endangered species	There is a growing literature on how to value wildlife. Approaches include estimating their value to tourism and 'contingent valuation' whereby members of society are asked what value they place on defined wildlife resources or what they would be prepared to pay to conserve them	Wildlife parks and game reserves will devote some of their resources to caring for sick animals	Veterinary services will be called in to deal with zoonotic disease outbreaks in wildlife

Source: Shaw, A.P.M., 2009, 'The economics of zoonoses and their control', in J. Rushton, *The Economics of Animal Health & Production*, pp. 161–167, CAB International, Oxfordshire & Massachusetts
DALYs, Disability-Adjusted Life Years.

services but ending up not getting the services, for example prescription for medicines to buy instead of being given the medicines. This connotes high burden of disease, especially in rural areas where people cannot afford paying for health services in private health facilities, which have proliferated concomitantly with the rise in cost-sharing in public health facilities.

Linkage between burden of disease and the practice of health care services

With respect to the practice of health care services, delivery of health services is indicated by a number of variables, which should be applicable to individuals, households, communities, populations, nations, regions, and globally so that comparisons in the levels of the services can be possible. For human diseases, WHO (2010) gives a number of indicators, which are divided in the following categories: (1) health service coverage, (2) risk factors, (3) health workforce, infrastructure and essential medicines, (4) health expenditure ratios and per capita health expenditures and (5) health inequities. Under each of those categories there are a number of indicators. The indicators are not reproduced here for saving space, but they are readily available on the Interment. Better health services result into less burden of disease, and vice versa.

About health service practice for animals, as in human health, health indicators are very important in order to prevent, control and treat animal diseases effectively. According to European Commission (2007), simple and reliable performance indicators help to measure progress towards animal health, guide policy, inform priorities, and target resources. The indicators can be divided into hard indicators of animal health (e.g. disease prevalence, number of animals eliminated) and softer indicators tracking the confidence, expectations and perceptions of citizens. In rural areas of developing countries, like for human health, animal health statuses and services are poor vis-à-vis urban areas, mainly due to fewer animal health facilities and fewer animal health personnel. As a result, according to WHO (2006), zoonoses typically affect isolated rural livestock keeping communities and those living in urban slums.

Linkage between burden of disease and psycho-social theories of health care

Psycho-social theories of health care seeking behaviour are amongst various factors that explain burden of disease. The theories explain the determinants of behaviour that lead people to accessing and utilising health services. Some of the determinants are common in seeking health care services for humans and for animals, and these include local people's experiences with diseases, availability of traditional versus modern treatment, knowledge and beliefs about diseases, decision process for seeking health services, and parochial versus cosmopolitan outlook of diseases. The behavioural aspects that are practised in turn determine the extent to which health services are accessed. Some of the prominent theories are Parsons' sick role, Mechanic's general theory of help seeking, Suchman's stages of illness and medical care, Theory of Reasoned Action (TRA), and Theory of Planned Behaviour (TPB). Besides the theories, there are models for health care seeking behaviour. The difference between the theories and the models is that the former consider decision points or stages of health care seeking, but the latter can be regarded as containing sets of interacting variables (Rebhan 2009). One of the prominent models is the Health Belief Model (HBM). In view of this model, if individuals do not perceive the illness as serious, they will not seek treatment or preventive measures for themselves, for their household members or for their livestock (Rosenstock, Strecher & Becker 1994; Sheeran & Abraham 1995, in Hausmamm-Muela, Ribera & Nyamongo 2003). In the interest of saving space, only some of the theories and models are described here.

The TRA and the TPB are closely associated as the latter is an extension of the former. By TRA, Fishbein and Ajzen (1975) argue that attitude and subjective norm are the primary determinants of behaviour. However, no behaviour is specified in both of the theories. Therefore, various researchers in medical and agricultural systems (including livestock production) have applied the two theories in various situations and found them applicable to explaining correlations between certain factors (drivers of behaviour) and behaviour (good or bad) that people express. For example, both TRA and TPB have been used to study

farmers' conservative behaviours, and TRA has been used to study attitude towards buying feeds for livestock, and adoption of olive oil in British kitchens (Jackson *et al*. 2006). If the behaviour is assumed to be the way how people act in choosing sources of health care services that people prefer for themselves, for their household members, or for their livestock, the two theories can be used in the same way as the Health Care Utilisation Model (HCUM), which was developed by Andersen (1968) looking at three categories of determinants of choosing where to seek health services, (1) predisposing characteristics (demographics, position within the social structure, and beliefs of health services benefits), (2) enabling characteristics (resources found within the family and the community) and (3) need-based characteristics, including the perception of need for health services, whether individual, social, or clinically evaluated perceptions of need (Wolinsky 1988, in Rebhan 2009). The model has undergone modifications a number of times, but its current form centres specifically on treatment selection – whether people go for traditional healers, modern healers, drug sellers, self-treatment, or no treatment.

Linkage between burden of disease and some socio-economic factors

It is widely known that the most basic social services are education, health, water supply, and communication services. Health services, which are amongst other social services, are affected by the other services. Some sophisticated social services in view of current technologies like mobile phones and the Internet influence access to health services in various ways. The linkage between social aspects and health are detailed in the Report of the WHO's Commission on Social Determinants of Health (WHO 2008). The report gives three overarching recommendations for improving health:

- improving daily living conditions
- tackling the inequitable distribution of power, money, and resources
- measuring and understanding the problem of health inequity and assessing its impact of action.

Burden of disease and poverty

Poverty is pronounced deprivation in well-being (World Bank 2001). It is a multidimensional phenomenon whose comprehensive definition would have to include all of its indicators, which are innumerable. Accordingly, no attempt is made to present its definition here, but it is well known that its indicators include deficiencies in basic needs, especially food, shelter, and clothes; and in social services like education, healthcare, and water supply. It is also indicated by vulnerability, exposure to risk, voicelessness, powerlessness, and capabilities that a person has (Sen 1999; World Bank 2001). There are various ways of measuring poverty, but they are avoided in this short paper. However, at least it is worth mentioning that income is a poor indicator of well being since it is volatile and some people having much income may not use it to obtain important needs. This view is supported by Sen (1999) who argues that resources are imperfect indicators of well-being and Alkire, Qizilbash and Comim (2010) who contend that income is a fuzzy measure of poverty. Therefore, non-monetary indicators are preferable to monetary ones, either to supplement the latter or alone. The preference for using non-monetary indicators grew in the 1990s after Sen (1999) came up with the capability approach to poverty measurement, which is linked with the human development perspective and is now fashionable in measuring poverty.

Empirical information shows that healthier people are more productive and that wealthier people can obtain things that make them healthier. For example, studies which were conducted in Colombia, Peru, and Nicaragua in the mid-1990s showed that reduced exposure to disease is associated positively with the health of adults and also with greater individual income-generating capacity (Savedoff & Schultz 2000). With respect to animal health, the burden of disease affects not only livestock keepers but also consumers of livestock products like meat, milk, and eggs. For livestock keepers, there may be losses of income. It is known that about 42% of the poor worldwide are dependent on livestock as their livelihoods but that imperfect or missing markets often trap them in low income equilibriums, preventing them from benefiting from the increased demand for animal protein (Otte & Pica-Ciamarra 2009). Besides the problem of market imperfections, diseases affect much the ability of livestock keepers through low productivity and mortality of their livestock. Accordingly, it is obvious that amongst livestock keepers infectious diseases contribute to impoverishing them. In view of this, the same authors (Otte & Pica-Ciamarra 2009) contend that if poverty alleviation is a policy goal, policy makers should identify, design and implement public actions that allow poor livestock producers to take advantage of the increasing demand for meat, milk and eggs.

Conclusion and recommendations

We have seen in the literature reviewed that the occurrence of infectious diseases is driven by causative agents (viruses, bacteria, parasites, etc) and mediator conditions (social, cultural, economic or climatic) which facilitate the infection to occur and aid spread or transmission of the infection. The causative agents are best understood through the natural sciences whilst the mediator conditions are best understood through the social sciences. Accordingly, the research framework that recognises the contribution that socio-economists can play in collaboration with biological scientists to harness innovation in science and technology in order to improve the capacity to detect, identify and monitor infectious diseases of humans and animals and their interactions in order to better manage the risk posed by them is one that is likely to provide the type of evidence-based policy impact.

The practice of One Health approaches is long overdue. Adopting them should not be debatable, but the modalities of how to adopt and practise them should be the discussion, in view of the factors that enhance and those that constrain them. Accordingly, factors that constrain the adoption and

practice of One Health should be curbed and those that promote it should be fostered.

Using social research methods (e.g. questionnaire-based interviews, key informant interviews, life histories, PRA, FGD, ethnography, grounded theory, probing, and prompting), and tools (e.g. questionnaires, checklists of items for discussion, Likert-type scales, index scales, and differential semantic scales) can add value to explanation of disease emergence, re-emergence, and persistence as well as burden of disease; the tools are good at studying attitudinal and behavioural aspects, which cannot be studied biologically. Moreover, economics of controlling diseases facilitate quantification of burden of disease, for example by determining burden of disease in monetary terms and using the separable costs method of cost-effective analysis to determine equitable sharing of costs amongst various sectors working collaboratively to control diseases.

The One Health driven policy research framework to examine the extent to which One Health approach can help to streamline health policies in the public, animal, and wildlife health sectors in such a way that they facilitate more collaboration between natural and social scientists so as to increase the effectiveness of interventions to stem infectious diseases for better socio-economic well-being is more focused on health policies. However, there may be other factors apart from health policies constraining the collaboration, which may include health service provision on the ground, psycho-social theories of health care seeking behaviour, and socio-economic factors. Accordingly, such a framework should include an assessment of such factors as determinants of collaboration amongst various stakeholders, to find which of them are more associated with enhancing or deterring the collaboration and reduction of the burden of disease.

Infectious diseases increase the burden of disease in humans and animals, and the burden in turn aggravates poverty, especially amongst rural and sub-urban people whose economy mainly depends on livestock. Therefore, there should be equitable control of human and animal diseases in rural and urban areas.

Literature shows that the majority of infections, new or old, either do or have the propensity to move across species (human and animals) or may have originated from animals before assuming the human-to-human transmissibility. One Health approaches seem to be the logical strategy. But one should not under-estimate the challenges by the current organisational systems; even for the shared problem of zoonoses between animal and human health; FAO (2006) has observed the challenges.

Acknowledgements

This work has been supported by Wellcome Trust Grant WT087546MA to the Southern African Centre for Infectious Disease Surveillance (http://www.sacids.org) and its collaborating UK partner institutions, namely the London School of Hygiene and Tropical Medicine (http://www.lshtm.ac.uk), the Royal Veterinary College (http://www.rvc.ac.uk) and the London International Development Centre (http://www.lidc.org.uk).

References

Alkire, S., Qizilbash, M. & Comim, F., 2010, 'Introduction', in F. Comim, M. Qizilbash & S. Alkire (eds.), *The capability approach: Concepts, measures and applications*, pp. 1–25, Cambridge University Press, Cambridge.

Andersen, R., 1968, *A behavioral model of families' use of health services,* Research Series No. 25, Center for Health Administration Studies, University of Chicago, Chicago, IL.

Brazier, J., Ratcliffe, J., Salomon, J.A. & Tsuchiya, A., 2007, *Measuring and valuing health benefits for economic evaluation*, Oxford University Press, Oxford.

Coker, R., Rushton, R., Mounier-Jack, S., Karimuribo, R., Lutumba, P., Kambarage, D. *et al*., 2011, 'Towards a conceptual framework to support one-health research for policy on emerging zoonoses', *The Lancet Infectious Diseases* 11, April, 326–331. http://dx.doi.org/10.1016/S1473-3099(10)70312-1

Drummond, M.F., Sculpher, M.J., Torrance, G.W., O'Brien, B.J. & Stoddart, G.L., 2005, *Methods for the economic evaluation of health care programmes,* 3rd edn., Oxford University Press, Oxford.

European Commission, 2007, *A new animal health strategy for the European Union (2007–2013),* computer software, viewed 29 May 2011, from http://ec.europa.eu/food/animal/diseases/strategy/animal_health_strategy_en.pdf

FAO & OIE, 2011, 'Joint FAO/OIE Committee on Global Rinderpest Eradication Final Report', computer software, viewed 30 August 2011, from http://www.oie.int/fileadmin/Home/eng/Media_Center/docs/pdf/Final_Report_May2011.pdf

FAO/OIE/WHO, 2004, *The Global Framework for the Progressive Control of Trans-Boundary Animal Diseases (GF-TADS),* computer software, viewed on 8 June 2011, from http://www.oie.int/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/GF-TADs_approved_version24May2004.pdf

Jackson, E.L., Quaddus, M., Islam, N. & Stanton, J., 2006, 'Hybrid vigour of behavioural theories in the agribusiness research domain: Is it possible?', *Journal of International Farm Management* 3, 1–15.

Jones, K.E., Patel, N.G., Levy, M.A., Storeygard, A., Balk, D., Gittleman, J.L. *et al*., 2008, 'Global trends in human emerging infectious diseases', *Nature* 451, 990–993. http://dx.doi.org/10.1038/nature06536, PMid:18288193

Kock, R.A., 2003, 'What is this infamous "Wildlife/Livestock Disease Interface?" A review of current knowledge for the African continent', viewed 30 August 2011, from ftp://ftp.fao.org/docrep/nonfao/lead/x6198e/x6198e01.pdf

MacArthur, R.H. & Wilson, E.O., 1967, *The theory of island biogeography*, Princeton University Press, New Jersey.

Mathers, C.D., Vos, E.T., Stevenson, C.E. & Begg, S.J., 2001, 'The burden of disease and injury in Australia', *Bulletin of the World Health Organization* 79, 1076–1084. PMid:11731817, PMCid:2566696

Maudlin, I., Eisler, M.C. & Welburn, S.C., 2009, 'Neglected and endemic zoonoses', *Philosophical Transactions of the Royal Society* B 364, 2777–2787, viewed 30 August 2011, from http://rstb.royalsocietypublishing.org/content/364/1530/2777.full

Otte, J. & Pica-Ciamarra, U., 2009, 'Livestock policy and poverty reduction: Experiences from the Developing World', in. J. Ruston (ed.), *The economics of animal health & production,* pp. 267–288, CAB International, Oxfordshire & Massachusetts.

Otte, J., Roland-Holst, D., Pfeiffer, D, Soares-Magalhaes, R., Rushton, J., Graham, R. *et al*., 2007, 'Industrial Livestock Production and Global Health Risks, Pro-Poor Livestock Policy Initiative: A Living from Livestock', in Research Report Number 07–09, June, viewed 30 August 2011, from http://www.fao.org/AG/AGAINFO/programmes/en/pplpi/docarc/rep-hpai_industrialisationrisks.pdf

Rebhan, D. P., 2009, 'Health Care Utilization: Understanding and applying theories and models of health care seeking behavior', viewed 23 May 2011, from http://www.cwru.edu/med/epidbio/mphp439/healthcareutil.pdf

Rosenstock, I.M., Strecher, V.J. & Becker, M.H., 1994, 'The health belief model and HIV risk behavior change', in R.J. DiClemente & J.L. Peterson (eds.), *Preventing AIDS: Theories and methods of behavioral* interventions, pp. 5–24, Plenum Press, New York, NY.

Rushton, J. & Leonard, D.K., 2009, 'The new institutional economics and the assessment of animal disease control', in J. Rushton (ed.), *The Economics of Animal Health & Production*, pp. 144–148, CAB International, Oxfordshire & Massachusetts.

Rushton, J., 2009, *The economics of animal health & production,* CAB International, Oxfordshire & Massachusetts.

Savedoff, W.D. & Schultz, T.P., 2000, *Wealth from health: Linking social investments to earnings in Latin America*, Inter-American Development Bank, Washington, DC.

Schwabe, C.W., 1969, *Veterinary medicine and human health,* 2nd edn., The Williams & Wilkins Company, Baltimore.

Sen, A., 1999, *Development as Freedom*, Oxford University Press, Oxford.

Shaw, A.P.M., 2009, 'The economics of zoonoses and their control', in J. Rushton, *The Economics of Animal Health & Production*, pp. 161–167, CAB International, Oxfordshire & Massachusetts.

Taylor, L.H., Latham, S.M. & Woolhouse, E.J., 2001, *Risk Factors for Human Disease Emergence,* computer software, viewed 31 July 2011, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1088493/pdf/TB010983.pdf

Thornhill, R., Fincher, C.L., Murray, D.R. & Schaller, M., 2010, 'Zoonotic and non-zoonotic diseases in relation to human personality and societal values: Support for the parasite-stress model', *Evolutionary Psychology* 8(2), 151–169, viewed 13 May 2011, from http://www.epjournal.net/filestore/EP08151169.pdf

Walt, G., 1994, *Health policy: An introduction to process and power*, Witwatersrand University Press, Johannesburg & Zed Books, London and New Jersey.

WHO, 2006, 'The Control of Neglected Zoonotic Diseases: A Route to Poverty Alleviation', in *Report of a Joint WHO/DFID-AHP Meeting with the Participation of FAO and OIE*, Geneva, 20–21 September, computer software, viewed 31 May 2011, from http://whqlibdoc.who.int/publications/2006/9789241594301_eng.pdf

WHO, 2008, 'Closing the gap in a generation: Health equity through action on the social determinants of health', *Final Report of the Commission on Social Determinants of Health*, WHO and Commission on Social Determinants of Health, Geneva.

WHO, 2010, '*World Health Statistics 2010',* computer software, viewed 23 June 2011, from http://www.who.int/whosis/whostat/EN_WHS10_Full.pdf

World Bank, 2001, *World Development Report 2000/01: Attacking poverty,* World Bank, Washington, DC.

World Bank, 2010, *People, pathogens and our planet, volume 1: Towards a one health approach for controlling zoonotic diseases, Report No. 50833-Glb*, viewed 17 June 2011, from http://siteresources.worldbank.org/INTARD/Resources/PPP_Web.pdf

Zinsstag, J., Meisser, A., Schelling, E., Bonfoh, B. & Tanner, M., 2011, 'From two Medicines to One Medicine to One Health and Beyond', 1st African One Health Conference, Johannesburg July, 14 2011, viewed 30 August 2011, from http://www.sacids.org/kms/resources/OneHealth_Johannesburg_Zinsstagetal_2011%20%282%29.pdf

Economic benefits or drivers of a 'One Health' approach: Why should anyone invest?

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How to cite this proceeding:
Rushton, J., Häsler, B., De Haan, N. & Rushton, R., 2012, 'Economic benefits or drivers of a "One Health" approach: Why should anyone invest?', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #461, 5 pages. http://dx.doi.org/10.4102/ojvr.v79i2.461

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

One Health concepts and ideas are some of the oldest in the health discipline, yet they have not become main stream. Recent discussions of the need for One Health approaches require some reflection on how to present a case for greater investments. The paper approaches this problem from the perspective of the control and management of resources for health in general. It poses the following questions, (1) where do we need extra resources for One Health, (2) where can we save resources through a One Health approach and (3) who has control of the resources that do exist for One Health? In answering these questions three broad areas are explored, (1) The management and resources allocated for diseases, (2) The isolation of parts of the society that require human and animal health services and (3) The use of resources and skills that are easily transferable between human and animal health.

The paper concludes that One Health approaches are applicable in many scenarios. However, the costs of getting people from different disciplines to work together in order to achieve a true One Health approach can be large. To generate tangible benefits requires careful management of specialist skills, knowledge and equipment, which can only be achieved by a greater openness of the human and animal health disciplines. Without this openness, policy makers will continue to doubt the real value of One Health. In summary the future success of One Health is about people working in the research, education and provision of health systems around the world embracing and managing change more effectively.

Introduction

Since 2008 a number meetings have taken place and documents produced (Food and Agriculture Organisation/World Animal Health Organisation/World Health Organisation 2008; Chatham House 2010; Canadian Public Health Agency 2009; World Bank 2010) that have raised the need for a more holistic approach to problems that affect the health of humans, animals and the general environment. Such an approach is not new, but the need for it has been given a much sharper focus with the increasing incidence of diseases that have the potential of creating large economic impacts, human deaths and losses of environmental diversity.

The articulation of the need to adopt a One Health approach was accepted at a meeting of governments (IMCAPI) in Hanoi in April 2010. This was followed by a meeting in Stone Mountain, Georgia, USA to discuss how One Health can be operationalised. One of the recommendations of that meeting was the need for a document that clearly presents an investment strategy for One Health. Whilst the authors acknowledge the ongoing work in this area, the general feeling is that the One Health approach is still some way from being main stream with human and animal health policy making. The questions that come to mind are:

- Why is One Health not main stream?
- If One Health is important, how can the case for a major paradigm shift be more persuasively presented?

The paper explores these questions from the perspective of resource allocation namely by looking at, (1) where extra resources are needed for One Health, (2) where resources can be saved with a One Health approach, and (3) who has control of the resources that do exist for One Health at the moment.

Background

The environments generating health problems are dynamic. The human population continues to grow with expansions greatest in the last fifty years occurring in the developing world (Figure 1).

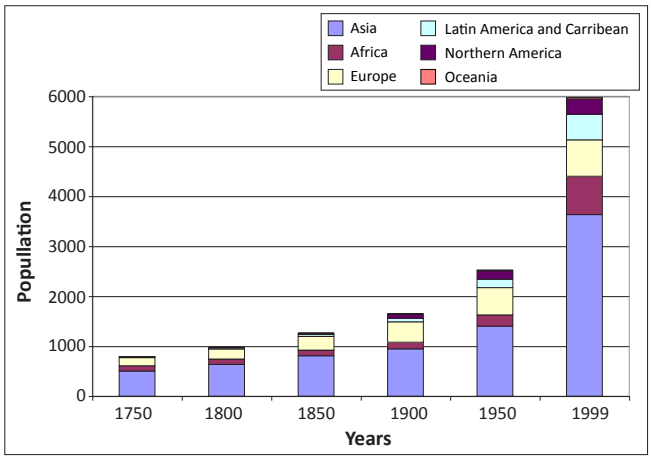
In addition to the growth in populations there are constant changes in the movement of people and settlement patterns. Some the most dramatic have been the movements of people from Central America and Mexico to North America in the last 15 years and in general the

movement of people to urban environments. To keep pace with these changes livestock systems and their associated value chains have evolved. Delgado *et al*. (1999) described this as a livestock revolution; in fact it was a second revolution, as the first occurred in the 1800s in Europe and the associated colonies (Rushton 2009). This first revolution was largely based on ruminant production, and the second and most recent revolution being based largely on intensive monogastric systems and to some extent from a growth in milk production. The world has seen greater specialisation and intensification of livestock systems leading to increased output per animal and per unit of labour. There has also been massive expansion of livestock populations particularly poultry and pigs and a concentration and clustering of livestock populations. In general there has been an increase in the sophistication and globalisation of livestock product value chains.

Originally these changes in the livestock sector were celebrated although there were early concerns of poorer livestock producers being left behind (De Haan *et al*. 2001; Heffernan 2002; Food and Agriculture Organisation 2005; Owen *et al*. 2005) and negative impacts on the environment (De Haan, Steinfeld & Blackburn 1997; Steinfeld *et al*. 2006).

What was not anticipated were the growing problems with the control of transboundary animal diseases and more specifically the resurgence of zoonotic diseases (Greger 2007). One of the issues that has been raised it is that as domestic livestock populations increase there has been greater contact with wildlife. Also as human populations have pushed into new areas there has been increased contact between human populations and wildlife. Therefore potentially two different sources of diseases either through direct contact with wild animals or through domestic species possible acting as liaison hosts. In addition to these contacts with wildlife the emerging food chains have generated greater levels of moral hazard (asymmetry of information) where people consuming livestock products are unlikely to know how animals are raised and fed, and how the product was handled and stored before it arrives on the plate. An extreme example of this comes from the UK where only 339000 people work in agricultural holdings (only 0.6% of the population) yet they produce food and therefore can affect the wellbeing of 60 million people (see Figure 2).

Responses to these existing and emerging challenges have been strong with greater control of many transboundary animal diseases, and success stores such as the global eradication of rinderpest and the regional eradication of diseases such as foot and mouth disease and classical swine fever. However, there have been major setbacks such as bovine spongiform encephalopathy, SARs and highly pathogenic avian influenza H5N1. The emergence of these problems and the apparent increase in the frequency at which such pathogens emerge (Woolhouse & Gowtage-Sequeria 2005; Woolhouse 2008; Jones *et al*. 2009) indicate a need to re-assess how the world deals with change and manages health. As the livestock sector and human society changes there is a need to monitor with different intensities and manage



Source: United Nations cited on http://www.statistics.gov.uk/StatBase/

FIGURE 1: World Population from 1850 to 1999.

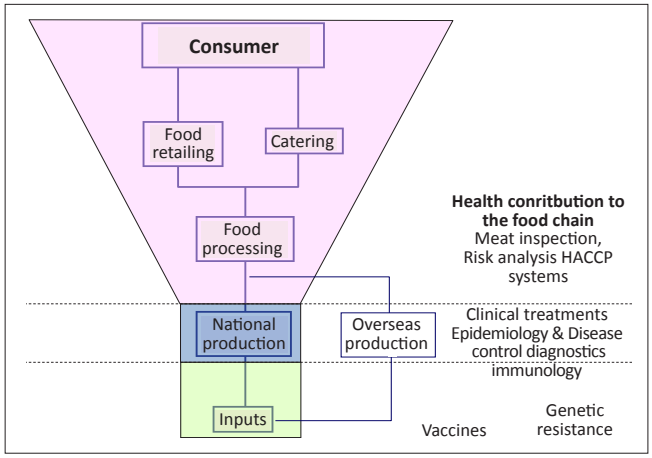


FIGURE 2: Health contributions to the food chain.

health risks in different ways. Where risks can be quantified there has been a tendency towards allocating resources for monitoring and management in a targeted manner. Yet the predictions of the emergence and re-emergence of disease problems have not been strong with obvious examples being BSE, H5N1, H1N1. There are two possibilities from this scenario: what we are observing are not predictable events they are random and cannot be identified through commonly used statistical methods or that our current risk models are inadequate at simulating reality. Either could be true, the more immediate challenge is to redirect resources so that problems as they emerge are addressed proportionately to the impact they cause and that response are not based on fears that are held. To achieve this there is a need for information on how resources are currently allocated and whether this use of use resources could be improved.

Economic logic for investment in One Health

Investments in One Health need to recognise two different aspects:

- Disease impact
  - Costs of disease in terms of losses in production of livestock
  - Costs of controlling the disease
  - Human health impacts and costs

- Avoidable losses – the costs of disease that can be avoided by implementing a disease control programme.

The disease impact gives some idea of the economic importance of a problem and whether there is a need to dedicate further resources in terms of education and research. The decision on surveillance and intervention needs to assess if their costs are less than the avoidable losses generated. The following sections will explore areas in One Health where this could be the case.

Looking at resources in the health system

The section is divided into three different areas, (1) resources dedicated to specific disease problems across humans and animals, (2) human populations that have poor access to resources for health and (3) resources can be moved between human and animal health issues easily.

Responses to specific diseases and health problems

Diseases can be relatively easily split into those that are problematic in humans, those that are problematic in animals and those can cause problems in both human and animal populations. The authors would suggest that the most appropriate approaches to diseases that cause problems in humans or animal populations need specialised approaches and in general this is how human health and animal health systems have evolved. However, the diseases that cause problems in both humans and animals – the zoonoses – require generalised approaches (Figure 3).

Some of the zoonotic diseases cause significant impacts in specific locations (Knobel *et al.* 2005 for rabies), and some that are classified as zoonotic cause huge disruptions (Otte *et al.* 2010 for HPAI H5N1). However, the zoonotic diseases as a whole do not tend to have a large enough impact in human and animal populations at the same time to have warranted the creation of generalised health service that approaches the problems from a combined human and animal population perspective (Maudlin, Eisler & Welburn 2009). For example, tuberculosis is one of world's major human disease problems, but the causative and self maintaining pathogen of this disease is not *Mycobacterium bovis*, the pathogen in cattle, it is *Mycobacterium tuberculosis*. Therefore whilst the disease complex as a whole could be considered zoonotic,

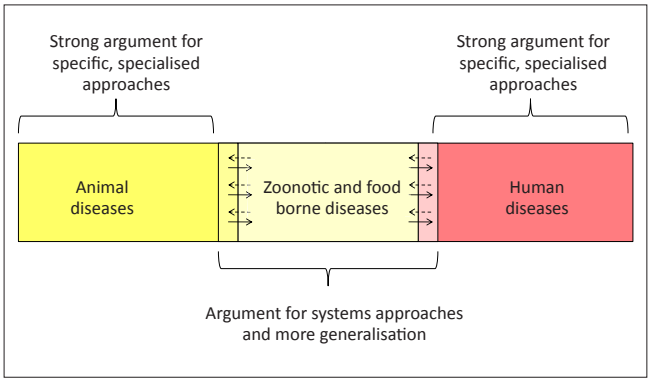


FIGURE 3: Disease groupings and the appropriate responses.

the pathogens tend to be species specific with some spillover into other species populations. Similarly, brucellosis pathogens are specific to livestock species with spillover into humans where the pathogen can remain but not spread between people. Probably the most challenging pathogens in terms of affecting both human and animal populations are the parasitic problems such as cysticercosis and cystic echinococcosis.

This is not to argue that zoonotic diseases are not important, but that as a group they do not seem to cause sufficient impact across both human and animal populations for societies to have a cadre of people who work across species or organisations that follow the disease across animal and human populations. The problem actually lies in where resources are spent on diseases that circulate in animals and cause spillover impacts in the human population. For example brucellosis causes a significant economic impact, but the costs of controlling the disease in animals are greater than the benefits generated in the animal population, it is only when benefits from the prevention of disease in humans is taken into account that the costs are exceeded (Roth *et al.* 2003). The implications for such disease problems is the need for One Health thinking at a much higher level of budgetary and resource allocation, so that control campaigns in animals are sufficiently well financed to lead to benefits in humans. This requires a proactive and preventative approach to disease management, a recognition that disease can be managed further upstream, which requires significant shifts in resource allocation. It does not necessarily mean closer working mechanisms in the field.

There are examples where there has been a need and a successful implementation of One Health in the eradication of disease with strong field level coordination such as the control of cystic echinococcosis and *Echinococcus* in places such as New Zealand and the ongoing attempts to remove cysticercosis from the northern area of Peru (Gonzalez 2011).

It is also argued that the non-communicable diseases is a problem that deserves more thought on how best to harness One Health approaches. Food chains process and refine food for both animals and humans and this has important implications on food intake nutritional health and resulting diseases. These aspects are rarely treated as One Health issues and are invariably observed and worried about rather than thinking of the underlying causes. They would require a more general rather than disease specific approach.

In common with all the disease groups there is the need for an understanding of the role of human behaviour in terms bringing host and pathogen together. There is also a need to understand and use how we react when a disease is present which could be in a positive manner in terms of controlling disease and also in negative manner leading to the maintenance of disease. The latter could be due to ignorance and/or economic gain. Finally human behaviour plays a role in consumption and therefore the emergence of non-communicable diseases.

To attract and retain resources that are applied to specialised activities in terms of dealing with diseases there would be a need for an agreement of the human and animal health leaders on who leads, who implements and who gets the resources. In some cases there will be strong arguments that integrated field level approaches are not necessary, but that these require One Health thinking at a budgetary allocation point. Other diseases do require much more integrated approaches such as specific parasitic diseases and the non-communicable diseases.

Where resources are scarce

Many people live in geographical isolation in areas where they are reliant on livestock. Making resources available in these areas for either human or animal health is difficult due to the limited availability of trained resources and the lack of demand for such services. The need for One Health approaches would make sense in terms of matching overall demand for animal and human health services and the potential to supply adequate services. Strong arguments for generalised services and these have been well documented (Schelling *et al*. 2007; Zinsstag *et al*. 2007).

Some lessons from these generalised approaches would benefit from examining the literature on the integrated rural development programmes (Roling & Wagemarkers 1998; Morton, Matthewmaan & Barton 1997; Van Veldhuizen *et al*. 1997). Indeed, One Health services probably need to incorporate aspects of animal production and genetics, water and sanitation and potentially plant health in such regions.

Where resources are underutilised

Many human health and animal health facilities are built that replicate capacity and in some cases have relatively low throughput. In addition there is human capacity building in data collection, storage and analysis skills in the two health areas. In the case of human capacity this can often be in too few numbers and/or with a low demand of their skills in their specific health field

Low throughput and low demand often leads to poor calibration of standards and variable output of results. Small numbers of trained people limit interchange and advancement in knowledge. There are strong arguments that certain aspects of human and veterinary diagnostics, data collection and analysis need to be combined to create synergies which will improve resource use.

Where resources, skills and institutions could provide a service to both human and animal health delivery and budget constraints limit how much redundancy can be allowed – laboratories are an obvious target, and more creative use could be made of epidemiology and socio-economics skills. Better linkages of human and animal health surveillance data may potentially be useful for emerging diseases.

Discussion and conclusions

The environment that leads to the emergence and re-emergence of health problems is dynamic and constantly changing. These changes have led to responses in terms of strengthening disease surveillance, internationally through WHO, OIE, FAO and nationally through multi and bi lateral programmes plus regional agreements. These have generated benefits in terms of:

- Improved understanding of health problem emergence and re-emergence in order to respond in a proportionate and timely manner.
- Generalised systems of health delivery where resources are scarce – very specific situations.
- Combined use of infrastructure and skillsets to improve the use of underutilised resources and create synergies.

The benefits are not constant as the environment is constantly changing. These changing benefits have changing costs that can only be estimated with better monitoring systems of:

- livestock systems
- value chains
- people working within and using these chains.

Yet we have weak systems to monitor the working and behaviour of livestock systems and their associated chains implying that One Health agenda should be expanded to include environmental concerns and human behaviour. Political reality of adopting a One Health agenda also requires thought and needs to be realistic with the development of evidence of added value from One Health approaches through systematic data collection and analysis. The current lack of evidence reflects a lack of funding, collaboration, management and support, and future work needs to pose the following questions:

- How do we improve the monitoring of facilitating environment so we can in real time:
  - estimate health problem impact with more accuracy
  - estimate the costs (direct costs and institutional costs) of monitoring and control
  - estimate benefits from mitigation activities.

We need to search for proportionate and rational responses that involve individuals, communities, Non-Governmental Organisations (NGOs), private and public sectors, and to recognise that no one mechanism will suit all situations – it requires a systems and people centred approach with strong technical leadership.

Acknowledgements

Madelon Meijer, Oxfam, The Netherlands; Malla Hovi, AHVLA, UK; Wantanee Kalpravidh, David Castellan – FAO, Bangkok; Nick Taylor - University of Reading, UK; Liz Redmond – FSA, UK; Katharina Stärk, Dirk Pfeiffer, Javier Guitian – RVC, UK; Mark Rweyemamu and the SACIDS team; Jeff Waage – Leverhulme Centre for Integrative Research on Agriculture and Health, LIDC, UK.

References

Canadian Public Health Agency, 2009, 'Report of the Expert Consultation on One Health', Winnapeg, March 2009, Canadian Government, Ottawa, Canada.

Chatham House, 2010, 'Shifting from Emergency Response to Prevention of Pandemic Disease Threats at Source', Chatham House, London, UK.

De Haan, C., Schillhorn van Veen, T., Brandenburg, B., Gauthier, J., Le Gall, F., Mearns, R. *et al*., 2001, 'Livestock development: Implications for rural poverty, the environment and global food security', The World Bank, Washington DC, USA.

De Haan, C., Steinfeld, H. & Blackburn, H., 1997, *Livestock and the Environment: Finding a Balance*, Wrenmedia, Suffolk, UK.

Delgado, C., Rosegrant, M., Steinfeld, H., Ehui, S. & Courbois, C., 1999, *Livestock to 2020. The Next Food Revolution*, Food, Agriculture and the Environment Discussion Paper 28. IFPRI, Washington DC, USA.

Food and Agriculture Organisation, 2005, 'Livestock Policies and Poverty Reduction in Africa, Asia and Latin America', Pro-Poor Livestock Policy Initiative Policy Brief, FAO, Rome Italy.

Food and Agriculture Organisation/World Animal Health Organisation/World Health Organisation, 2008, 'Contributing to One World, One Health - A Strategic Framework for Reducing Risks of Infectious Diseases at the Animal–Human–Ecosystems Interface', FAO, Rome, Italy.

Gonzalez, A., 2011, email, 29 November, agonzale@jhsph.edu.

Greger, M., 2007, 'The human/animal interface: Emergence and resurgence of zoonotic infectious diseases', *Reviews in Microbiology* 33, 243–299.

Heffernan, C., 2002, 'Livestock and the poor: issues in poverty focused development', in E. Owen, T. Smith, M.A. Steele, S. Anderson, A.J. Duncan, M. Herrero *et al*., (eds.), *Responding to the livestock revolution: The role of globalisation and implications for poverty alleviation* BSAS Publication 33, 229–245.

Jones, K.E., Patel, N.G., Levy, M.A., Storeygard, A., Balk, D., Gittlemman, J.L. *et al*., 2008, 'Global trends in emerging infectious diseases', *Nature (London)* 451, 990–993. http://dx.doi.org/10.1038/nature06536, PMid:18288193

Knobel, D.L., Cleaveland, S., Coleman, P.G., Fevre, E.M., Meltzer, M.I., Miranda, M.E.G. *et al*., 2005, 'Re-evaluating the burden of rabies in Africa and Asia', *Bulletin of the World Health Organization* 83, 360–368. PMid:15976877, PMCid:2626230

Maudlin, I., Eisler, M.C. & Welburn, S.C., 2009, 'Neglected and endemic zoonoses', *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences* 364, 2777–2787.

Morton, J., Matthewman, D. & Barton, D., 1997, *Livestock production extension: issues, case studies and policy options.* NRI Socio-economic Series 12. Chatham, UK: Natural Resources Institute.

Owen, E., Best, J., Devendra, C., Ku-Vera, J., Mtenga, L., Richards, W. *et al*., 2005, 'Introduction - the need to change the "mind-set"', in E. Owen, A. Kitalyi, N. Jayasuriya & T. Smith (eds.), *Livestock and Wealth Creation. Improving the husbandry of animals kept by resource-poor people in developing countries*, Nottingham University Press, Nottingham, UK, pp 1–11.

Otte, M.J., Hinrichs, J., Rushton, J., Roland-Holst, D. & Zilberman, D., 2008, 'Impacts of avian influenza virus on animal production in developing countries', *Perspectives in Agriculture, Veterinary Science, Nutrition and Natural Resources*, CABI, 3 No. 080.

Roling, N.G. & Wagemakers, M.A.E. (eds.), 1998, *Facilitating sustainable agriculture. Participatory learning and adaptive management in times of environmental uncertainty,* Cambridge University Press, Cambridge.

Roth, F., Zinsstag, J., Orkhon, D., Chimed-Ochir, G., Hutton, G., Cosivi, O. *et al*., 2003, 'Human health benefits from livestock vaccination for brucellosis: case study', *Bulletin of the World Health Organization* 81, 867–876.

Rushton, J., 2009, *The economics of animal health & production,* CAB International, Wallingford (UK) & Cambridge (USA).

Schelling, E., Bechir, M., Ahmed, M.A., Wyss, K., Randolph, T.F. & Zinsstag, J., 2007, 'Human and animal vaccination delivery to remote nomadic families, Chad.', *Emerging Infectious Diseases* 13, 373–379. http://dx.doi.org/10.3201/eid1303.060391, PMid:17552089, PMCid:2725911

Steinfeld, H., Gerber, P., Wassenaar, T., Castel, V., Rosales, M. & De Haan, C., 2006, *Livestock's Long Shadow Environmental Issues and Options*, FAO, Rome, Italy.

Van Veldhuizen, L., Waters-Bayer, A., Ramirez, R., Johnson, D. & Thompson, J., 1997, *Farmers' research in practice – lessons from the field*, Intermediate Technology Publications, London.

Woolhouse, M.E., 2008, 'Epidemiology: emerging diseases go global', *Nature (London)* 451, 898–899. http://dx.doi.org/10.1038/451898a, PMid:18288175

Woolhouse, M.E.J. & Gowtage-Sequeria, S., 2005, 'Host range and emerging and reemerging pathogens', *Emerging Infectious Diseases* 11, 1842–1847. http://dx.doi.org/10.3201/eid1112.050997, PMid:16485468, PMCid:3367654

World Bank, 2010, *People, Pathogens and Our Planet. Volume 1: Towards a One Health Approach for Controlling Zoonotic Diseases,* The World Bank, Washington, USA.

Zinsstag, J., Schelling, E., Roth, F., Bonfoh, B., Savigny, D.D. & Tanner, M., 2007, 'Human benefits of animal interventions for zoonosis control', *Emerging Infectious Diseases,* 13, 527–531. http://dx.doi.org/10.3201/eid1304.060381, PMid:17553265, PMCid:2725951

Foot-and-mouth disease virus serotypes detected in Tanzania from 2003 to 2010: Conjectured status and future prospects

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How to cite this proceeding:
Kasanga, C.J., Sallu, R., Kivaria, F., Mkama, M., Masambu, J., Yongolo, M. *et al.*, 'Foot-and-mouth disease virus serotypes detected in Tanzania from 2003 to 2010: Conjectured status and future prospects', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #462, 4 pages. http://dx.doi.org/10.4102/ojvr.v79i2.462

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

This study was conducted to investigate the presence of foot-and-mouth disease virus (FMDV) in different geographic locations of Tanzania. Epithelial tissues and fluids ($n = 364$) were collected from cattle exhibiting oral and foot vesicular lesions suggestive of FMD and submitted for routine FMD diagnosis. The analysis of these samples collected during the period of 2002 and 2010 was performed by serotype-specific antigen capture ELISA to determine the presence of FMDV. The results of this study indicated that 167 out of 364 (46.1%) of the samples contained FMDV antigen. Of the 167 positive samples, 37 (28.4%) were type O, 7 (4.1%) type A, 45 (21.9%) SAT 1 and 79 (45.6%) SAT 2. Two FMDV serotypes (O and SAT 2) were widely distributed throughout Tanzania whilst SAT 1 and A types were only found in the Eastern zone. Our findings suggest that serotypes A, O, SAT 1 and SAT 2 prevail in Tanzania and are associated with the recent FMD outbreaks. The lack of comprehensive animal movement records and inconsistent vaccination programmes make it difficult to determine the exact source of FMD outbreaks or to trace the transmission of the disease over time. Therefore, further collection and analysis of samples from domestic and wild animals are being undertaken to investigate the genetic and antigenic characteristics of the circulating strains, so that a rational method to control FMD in Tanzania and the neighbouring countries can be recommended.

Introduction

Foot-and-mouth disease virus (FMDV; family *Picornaviridae,* genus: *Aphthovirus*) exists as seven serotypes (O, A, C, Asia 1, Southern African Territories 1–3 [SAT 1–3]) and causes a highly contagious disease of ruminants and swine. Foot-and-mouth disease (FMD) is endemic in most of sub-Saharan Africa and is considered to be one of the most widely distributed transboundary animal diseases (TAD) in the world (OIE and FAO Reports 2003). In Tanzania, FMD is the most important viral TAD (Swai *et al.* 2009). FMDV in endemic settings across the world have been categorised into six pools; each comprising a different geographic location with different predominant serotypes. The FMDV pools include pool 1 in East Asia (O, A and Asia 1), pool 2 in Central Asia (O, A and Asia 1), pool 3 in Europe and South Asia (O, A and Asia 1), pool 4 in Southern, Eastern and Horn of Africa (A, O, SAT 1, 2 and 3), pool 5 in Western Africa (O, A, SAT 1 and 2), and pool 6 in Southern Africa (SAT 1, 2 and 3) (Paton, Sumption & Charleston 2009). Tanzania links East Africa and southern Africa in a region that overlaps between pools 4 and 6.

Since its first documentation in 1927 and first isolation of the virus in 1954, many FMD outbreaks have occurred across different areas of Tanzania. Unrestricted animal movements are an important mechanism by which FMD is spread within and across international borders (Di Nardo, Knowles & Paton 2011; Kivaria 2003). In order to limit the spread and economic impact of the disease, the control measures implemented during outbreaks in Tanzania typically consist of quarantine and restriction of animal movements particularly in areas with well-defined farming systems (Kivaria 2003). However, the presence of multiple FMD serotypes, and the occurrence of subclinical forms of the disease renders FMD control very difficult, particularly in pastoral agricultural systems where resources are limited. Establishing and quantifying the distribution of FMDV serotypes in different eco-climatic regions in the country will contribute to the understanding of FMD epidemiology, and provide knowledge to researchers, vaccine manufacturers and policy makers to more efficiently deploy resources to control FMD field outbreaks.

Of the seven known FMDV serotypes, four (O, A, SAT 1 and SAT 2) have been previously identified and reported in Tanzania (Mlangwa 1983; Rweyemamu & Loretu 1972; Rweyemamu *et al*. 2008b; Swai, Mrosso & Masambu 2009; Vosloo *et al*. 2002). Despite the fact that FMD is endemic in Tanzania, only limited studies have been conducted to describe the spatial and temporal

distribution of FMD outbreaks (Picado *et al.* 2010) and FMDV serotypes in the country (Rweyemamu & Loretu 1972; Swai *et al.* 2009). Furthermore, no specific detailed studies have been undertaken to describe the molecular epidemiology of Tanzanian FMDV serotypes. These approaches can define the distribution patterns of viruses and factors determining endemicity of FMD in Tanzania, and data from such studies will provide important information that will underpin future efforts to control FMD. Therefore, the aim of this current study was to use routine laboratory diagnostic methods to establish the spatial and temporal distribution of FMDV serotypes in different regions of Tanzania. Sample collection targeted FMD outbreaks that occurred in the country between 2003 and 2010.

Materials and methods

Sample collection

All samples were collected from confirmed FMD outbreaks (based on clinical signs) in cattle in different regions of Tanzania. The samples were collected by staff from seven strategically placed veterinary investigation centres in the country. These samples were obtained from regions namely Arusha and Kilimanjaro (Northern zone), Mwanza and Kagera (Northern-Lake zone), Tabora, Rukwa and Kigoma (Western zone), Mtwara (Southern-coastal zone), Iringa and Mbeya (Southern zone), Dodoma and Singida (Central zone) and Dar-es-Salaam, Pwani and Morogoro (Eastern-Coastal zone). Epithelial tissues and fluid from oral and foot lesions were collected and immediately placed in virus transport media composed of equal amounts of sterile glycerol (50% v/v) and 0.04 M phosphate buffered saline at pH 7.2–pH 7.6, stored at +4 °C and transported to the Central Veterinary Laboratory (CVL), Dar-es-Salaam.

Laboratory analysis of samples

Vesicular fluids and tissue epithelia were analysed for FMDV antigens, and the viral antigen typed into different serotypes by the antigen detection ELISA (IAH, Pirbright) as previously described (Hamblin, Armstrong & Hedger 1984; Ferris & Dawson 1988).

Data analysis

The dependent variable tested was the seropositivity to FMDV antigen (positive or negative) and specific serotype, in this case types A, O, SAT 1 and SAT 2. The independent variables investigated were the geographic location of the origin of samples and/or FMD outbreaks.

Results

A total of 364 samples from different eco-climatic regions in Tanzania were examined for FMDV antigen. Of the tested samples, 167 (45.9%) were positive to at least one of the four serotypes of FMDV. Of the 167 positive samples, 37 (22.0%) were serotype O, 7 (4.2%) serotype A, 45 (27%) serotype SAT 1 and 78 (46.8%) serotype SAT 2 (Table 1). There were no samples that tested positive for more than one FMDV serotype. Serotype SAT 2 was identified throughout the Northern, Southern, Western, Eastern and Central zones of Tanzania (Figure 1). Serotypes O, SAT 1 and SAT 2 were identified at least from one geographic region every year from 2003 to 2010 (Table 1). Serotype A was exclusively found in the Eastern coastal zone (Figure 1), and was only detected more recently in 2009 (Table 1). Interestingly, serotype SAT 1 was consistently detected only in the Eastern coastal region throughout the eight years (2003–2010). Serotype O was mainly found in the Southern and Northern highlands as well as the Lake zones. Animals from which all the tested samples were obtained had no documented history or evidence of vaccination against FMDV.

Discussion

FMD is known to be endemic in Tanzania. These findings show that FMDV is widely distributed in many parts of the country with at least four serotypes being found. The four serotypes that were detected during the period of from 2003 to 2010 were O, A, SAT 1 and SAT 2. This is broadly

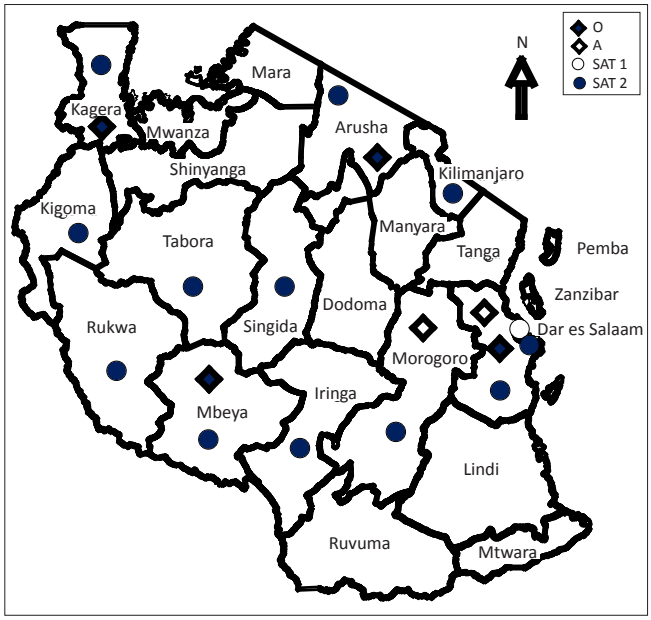

FIGURE 1: Geographic distribution of foot-and-mouth disease virus serotypes detected in Tanzania between 2003 and 2010.

TABLE 1: Summary of foot-and-mouth disease virus serotypes detected in Tanzania from 2003 to 2010.

Year	Samples tested	Samples positive†	Serotypes			
			O	A	SAT 1	SAT 2
2003	42	19	2	0	5	12
2004	39	19	4	0	6	9
2005	28	10	1	0	3	6
2006	57	27	10	0	4	13
2007	45	29	12	0	7	10
2008	35	15	4	0	4	7
2009	60	27	2	7	6	13
2010	58	21	2	0	10	9
Total	**364**	-	**37**	**7**	**45**	**78**

†, Samples that were positive to any of the four detected serotypes; A, O, SAT1 and SAT2.

in agreement with findings from previous studies, which reported the detection of these serotypes in this country from late 1950s (Rweyemamu & Loretu 1972; Vosloo *et al.* 2002). These observations suggest that serotypes O, A, SAT 1 and SAT 2 have been, and are also associated with the recent FMD outbreaks in different areas of Tanzania.

We have observed that the three serotypes (O, SAT 1 and SAT 2) were detected consistently every year from 2003 to 2010 (Table 1). Studies conducted by others have reported the existence of serotypes O and SAT 2 in Tanzania to be as far back as in 1950s with the detection of SAT 1 for the first time in 1971 (Rweyemamu & Loretu 1972, 1973). Together, these findings indicate that types O and SAT 2 are old in Tanzania and have consistently been observed in the country. These observations raise some questions such as, what factors could be involved in the successive existence of these serotypes? Are the viruses detected in 1950s genetically and antigenically related to the most recent strains? Are viruses from different geographic regions genetically related? All these call for a need of performing a detailed study on the molecular epidemiology of FMDV in Tanzania.

The current and previous studies indicate some enigma relating to the dynamics of Serotype A in Tanzania. During the period covered by this study (i.e. from 2003 to 2010) it was detected only in 2009 in the Eastern coastal zone. Rweyemamu and Loretu (1972) reported regular detection of this sero-type in the Northern, Northern-lake and Central zones of Tanzania for up to 1971. Swai *et al.* (2009), however, did not find serotype A in the samples collected between 1997 and 2004. The factors that may have led to the apparent disappearance of serotype A in Tanzania for possibly up to 38 years (from 1971 up to 2009) have not been elucidated. They could include high fragility of the virus and low number of samples that were brought for analysis. However, the reappearance of the type A viruses could possibly be a consequence of cross-border spread of the virus from the neighbouring countries, implying that unlike serotypes O, SAT 1 and SAT 2, serotype A might not be truly endemic in Tanzania. Similar observations have also been made in Iran that reported the emergence of serotype A sub-lineage 'A-Iran-05' (after long time of no periodic outbreaks of type A viruses), which has no closely related antecedents (Knowles *et al.* 2009). Together, these observations indicate the possibility of emergence of different serotype A strains in different geographic areas. Therefore further studies are required to determine the genetic characteristics of the old and new serotype A isolates detected in Tanzania. This will unravel the evolutionary characteristics of type A sub-lineages, which could be useful for the control of emerging type A variants in the region.

As the government of the United Republic of Tanzania is planning to adopt a strategic control programme for FMD through vaccination and controlled animal movements, a wider knowledge and understanding of FMDV dynamics and epidemiology should be taken into consideration. This will require the identification of high-risk 'hotspots' as well as potentially infected and FMD-free zones. This task will however need considerable political commitment and laboratory resources to define the spatial and temporal distribution of FMD outbreaks and serotypes in the country. The heterogeneity of FMDV serotypes observed in this study (Table 1) and their spatial occurrence (Figure 1) highlights the need for continuous surveillance of FMD so as to monitor the infection status and spread of FMDV serotypes in livestock and wildlife in Tanzania. As risk factors and transmission characteristics differ in each region (Swai *et al.* 2009; Picado *et al.* 2010), a regional FMD surveillance system involving Tanzania and the neighbouring countries will provide information on serotype spatiotemporal distribution for effective control of the disease.

Serotype SAT 2 was detected almost throughout the country for the whole period of 2003–2010 (Table 1), with the highest detection rate of 46.8%. Additional information from Veterinary Investigation Centres (VICs) in Tanzania (data not shown) indicates that SAT 2 strains were obtained from regions with relatively higher number of livestock, especially cattle. These findings suggest that SAT 2 could be incriminated as the cause of many FMD outbreaks in Tanzania. Efforts to isolate and characterise the viruses through genome sequencing are required so as to understand the genetic and antigenic features of field strains; information that is necessary for selection of appropriate vaccine candidate strains for use in Tanzania and neighbouring countries.

Serotype O seems to be widespread in the Northern, Southern, Western and Eastern zones, and not in the central parts of Tanzania (Figure 1). This observation agrees with the previous reports in Tanzania (Mlangwa 1983; MoWLD 2003). In addition, serotypes A and SAT 1 were also not detected from the central parts of Tanzania. However, it is important to recognise that the absence of serotypes O, A and SAT 1 from the central parts of Tanzania could be due to low number of samples submitted to CVL for diagnosis that may be related to under reporting of outbreaks, inappropriate cold chain facilities and logistics rather than the actual situation of disease occurrence.

The control of FMD, especially following outbreaks, requires timely identification and characterisation of circulating FMDV serotypes in a given geographic area. Tanzania is found in the Great Lakes (also known as the East African Community (EAC) or Southern-East Africa FMD epidemiological cluster characterised by a substantial diversity of circulating strains or topotypes (Rweyemamu *et al.* 2008a).

Five serotypes (A, O, C, SAT 1 and SAT 2) are known to be endemic in this cluster (Sahle et al. 2007; Vosloo *et al.* 2002), and a sixth serotype (SAT 3) has only been isolated from African buffaloes in Uganda in 1970 (Hedger, Forman & Woodford 1973). In the current study, we detected serotypes A, O, SAT 1 and SAT 2 in cattle from different geographic locations in Tanzania. So far serotypes C and SAT 3 have never been detected in Tanzania. Whether these serotypes, that have been reported to prevail in the EAC cluster, also exist in Tanzania remain unclear and need further systematic investigation.

The current spatiotemporal distribution of FMDV serotypes reported in this study indicates the absence of FMDV in

southeastern areas of Tanzania bordering Mozambique (Figure 1). This area has also been designated as low-density FMD area with no evidence of FMD outbreaks (Picado *et al.* 2010). These findings suggest that the southeastern corner, precisely the Mtwara corridor, could be considered as a potential FMD-free zone whereby strict surveillance and control programmes should be implemented for production of livestock meant for exportation.

In this study we have observed an inconsistency serotype distribution in Tanzania (Figure 1). The heterogenic distribution of the different serotype could be ascribed to several factors such as presence of diverse susceptible wildlife reservoirs, inadequate diagnostic capacities, diverse farming systems, socio-economic factors, uncontrolled animal movements, genetic and antigenic variation of the pathogen as well as lack of clear disease control policies (Rweyemamu 1984). However, the laboratory capacity in Tanzania and neighbouring countries has been considered to be inadequate in terms of facilities, equipment and diagnostic kits. Therefore, enhancement of laboratory capacity to undertake disease surveillance is of a paramount importance so as to keep pace with the accurate and timely identification of FMDV field strains, which is required for appropriate control strategies of FMD in the region.

We did not test any samples from wildlife in this study as all samples were obtained from FMD outbreaks in cattle. However, the epidemiological role of wildlife such as the African buffalo (*Syncerus caffer*) as carriers of particularly the SAT serotypes has been widely discussed (Hedger *et al.* 1973; Vosloo *et al.* 1996). A number of regions from which samples were derived are known to be potentially livestock-wildlife interface areas with predominantly high population of buffalos. Further studies are required to elucidate the epidemiological link of FMDV maintenance in wildlife, and transmission of the virus from livestock to wild animals or wild animals to livestock. Furthermore, the temporal and spatial dynamics of infection need to be conducted with the analysis of host animal distributions and contact opportunities, sero-surveys to estimate the level of infection and use of modern available techniques to track FMDV incursions into disease free areas.

Conclusion

We have detected the four FMDV serotypes O, A, SAT 1 and SAT 2, which are cocirculating in Tanzania. Sero-type A seems to occur in waves with periods (years) of apparent absence between epidemics. The presence of multiple serotypes and the complex epidemiology of FMD complicate the control of the disease through vaccination and establishment of FMD-free zones. Our findings also emphasize the importance of undertaking continuous surveillance to monitor the emergence and spread of FMDV strains. Therefore, further studies in both domestic and wild animals are required to investigate the genetic and antigenic characteristics of the circulating strains so that a rational control method for FMD in Tanzania and neighbouring countries can be recommended.

Acknowledgements

The authors wish to recognise the assistance provided by farmers and staff in Veterinary Investigation Centres for sample collection during FMD outbreaks. We appreciate the contribution of staff at the Central Veterinary Laboratory – Temeke during laboratory analysis of samples. We are grateful to the World Reference Laboratory (WRL), Pirbright, UK who provided ELISA kit for the study. The Ministry of Livestock Development and Fisheries, United Republic of Tanzania funded this work. The authors also acknowledge the Southern African Centre for Infectious Disease Surveillance for providing financial support (to CJK and RS) to present this paper at the first One Health conference in Africa, Johannesburg, South Africa, July 2011.

References

Di Nardo, A., Knowles, N.J. & Paton, D.J., 2011, 'Combining livestock trade patterns with phylogenetics to help understand the spread of foot and mouth disease in sub-Saharan Africa, the Middle East and Southeast Asia', *Revue Scientifique et Technique* (*OIE*) 30, 63–85.

Ferris, N.P. & Dawson, M., 1988, 'Routine application of enzyme-linked immunosorbent assay in comparison with complement fixation for the diagnosis of foot and mouth and swine vesicular diseases', *Veterinary Microbiology* 16, 201–209.

Hamblin, C., Armstrong, R.M. & Hedger, R.S., 1984, 'A rapid enzyme linked immunosorbent assay for the detection of foot and mouth disease viruses in epithelial tissues', *Veterinary Microbiology* 9, 435–443.

Hedger, R.S., Forman, A.J. & Woodford, M.H., 1973, 'FMDV in East African buffalo', *Bulletin of Epizootic Diseases in Africa* 21, 99–101.

Kivaria, F.M., 2003, 'Foot and mouth disease in Tanzania: an overview of its national status', *Veterinary Quarterly* 25, 72–78.

Knowles, N.J., Nazem Shirazi, M.H., Wadsworth, J., Swabey, K.G., Stirling, J.M., Statham, R.J. *et al*., 2009, 'Recent spread of new strain (A-Iran-05) of foot-and-mouth disease virus type A in the Middle East', Transboundary and Emerging Diseases 56, 157–169. Mlangwa, J.E.D., 1983, 'Foot-and-mouth disease (FMD) in Tanzania: some problems associated with its control', *Tanzania Veterinary Bulletin* 5, 49–59.

MoWLD., 2003, 'Foot-and-mouth disease increases livestock keeper's poverty: let us stamp it out', *Animal Health Newspaper* 4, 1–12.

Paton, D.J., Sumption, K.J. & Charleston, B., 2009, 'Options for control of foot-and-mouth disease: knowledge, capability and policy', *Philosophical Transactions of the Royal SocietyB* 364, 2657–2667. http://dx.doi.org/10.1098/rstb.2009.0100

Perry, B.D. & Rich, K.M., 2007, 'Poverty impacts of foot-and-mouth disease and the poverty reduction implications of its control', *Veterinary Record* 160, 238–241. http://dx.doi.org/10.1136/vr.160.7.238

Picado, A., Speybroeck, N., Kivaria, F., Mosha, R.M., Sumaye, R.D., Casal, J. & Berkvens, D., 2010, 'Foot-and-Mouth Disease in Tanzania from 2001 to 2006', *Transboundary and Emerging Diseases* 58, 44–52. http://dx.doi.org/10.1111/j.1865-1682.2010.01180.x

Rweyemamu, M.M. & Loretu, K., 1972, 'Observations on foot-and-mouth disease in Tanzania', *Bulletin of Epizootic Diseases of Africa* 22, 101–104.

Rweyemamu, M.M. & Loretu, K., 1973, 'Observations on the first epidemic of foot-and- mouth disease Type SAT 1 in Tanzania', *Bulletin of Epizootic Diseases of Africa* 21, 393–400.

Rweyemamu, M.M., 1984, 'Antigenic variation in foot-and-mouth disease: studies based on the virus neutralization reaction', *Journal of Biological Standardization* 12, 323–37. http://dx.doi.org/10.1016/S0092-1157(84)80013-X

Rweyemamu, M., Roeder, P., Mackay, D., Sumption, K., Brownlie, J., Leforban, Y. *et al*., 2008a, 'Epidemiological patterns of foot-and-mouth disease worldwide', *Transboundary and Emerging Diseases* 55, 57–72. http://dx.doi.org/10.1111/j.1865-1682.2007.01013.x

Rweyemamu, M., Roeder, P., MacKay, D., Sumption, K., Brownlie, J. & Leforban, Y., 2008b, 'Planning for the progressive control of foot-and-mouth disease worldwide', *Transboundary and Emerging Diseases* 55, 73–87. http://dx.doi.org/10.1111/j.1865-1682.2007.01016.x

Sahle, M., Dwarka, R.M., Venter, E.H. & Vosloo, W., 2007, 'Comparison of SAT-1 foot-and mouth disease virus isolates obtained from East Africa between 1971 and 2000 with viruses from the rest of sub-Saharan Africa', *Archives of Virology* 152, 797–804. http://dx.doi.org/10.1007/s00705-006-0893-x

Swai, E.S., Mrosso, A. & Masambu, J.I.G., 2009, 'Occurrence of foot-and-mouth disease serotypes in Tanzania: A retrospective study of tongue epithelial tissue samples', *Tanzania Veterinary Journal* 26, 7–12. http://dx.doi.org/10.4314/tvj.v26i1.49232

Vosloo, W., Bastos, A.D., Kirkbride, E., Esterhuysen, J.J., Janse van Rensburg D., Bengis., R.G. *et al*., 1996, 'Persistent infection of African buffalo (*Syncerus caffer*) with SAT-type foot-and-mouth disease viruses: rate of fixation of mutations, antigenic change and interspecies transmission', *Journal of General Virology* 77, 1457–1467. http://dx.doi.org/10.1099/0022-1317-77-7-1457

Vosloo, W., Bastos, A.D., Sangare, O., Hargreaves, S.K. & Thomson, G.R., 2002, 'Review of the status and control of foot-and-mouth disease in sub-Saharan Africa', *Revue Scientifique et Technique* (OIE) 21, 437–44.

Bovine tuberculosis at the human-livestock-wildlife interface: Is it a public health problem in Tanzania? A review

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Despite the apparent public health concern about Bovine tuberculosis (BTB) in Tanzania, little has been done regarding the zoonotic importance of the disease and raising awareness of the community to prevent the disease. Bovine tuberculosis is a potential zoonotic disease that can infect a variety of hosts, including humans. The presence of multiple hosts including wild animals, inefficient diagnostic techniques, absence of defined national controls and eradication programs could impede the control of bovine TB. In Tanzania, the diagnosis of *Mycobacterium bovis* in animals is mostly carried out by tuberculin skin testing, meat inspection in abattoirs and only rarely using bacteriological techniques. The estimated prevalence of BTB in animals in Tanzania varies and ranges across regions from 0.2% to 13.3%, which is likely to be an underestimate if not confirmed by bacteriology or molecular techniques. *Mycobacterium bovis* has been detected and isolated from different animal species and has been recovered in 10% of apparently healthy wildebeest that did not show lesions at post-mortem. The transmission of the disease from animals to humans can occur directly through the aerosol route and indirectly by consumption of raw milk. This poses an emerging disease threat in the current era of HIV confection in Tanzania and elsewhere. *Mycobacterium bovis* is one of the causative agents of human extra pulmonary tuberculosis. In Tanzania there was a significant increase (116.6%) of extrapulmonary cases reported between 1995 and 2009, suggesting the possibility of widespread *M. bovis* and *Mycobacterium tuberculosis* infection due to general rise of Human Immunodeficiency virus (HIV). This paper aims to review the potential health and economic impact of bovine tuberculosis and challenges to its control in order to safeguard human and animal population in Tanzania.

Introduction

Mycobacterium tuberculosis, Mycobacterium bovis, Mycobacterium bovis BCG, Mycobacterium canettii, Mycobacterium africanum, Mycobacterium pinnipedii, Mycobacterium microti, Mycobacterium caprae, the *dassie* and the *oryx* bacillus, and the recently discovered *Mycobacterium mungi* are closely related species that form the *M. tuberculosis* complex (MTBC). *Mycobacterium tuberculosis* and *M. bovis* are the most important species in the complex which commonly cause human and animal tuberculosis (TB), with concomitant negative consequences for human and animal health and economic costs.

The probability of *M. bovis* transmission is more likely to occur between animals, particularly those in close contact such as herd animals (Grange & Collins 1987). Humans can also be infected by *M. bovis* from contact with infected animals or animal products and the likelihood of infection and disease, as with human forms of TB are exacerbated by crowding and stress (Figueroa-Munoz & Ramon-Pardo 2008). The transmission of *M. bovis* between humans or from humans to animals is very rare. Although the occurrence of *M. bovis* in humans is relatively minor compared to the burden from *M. tuberculosis* as far as we know, there is concern that the HIV and AIDS pandemic may have magnified this risk.

Transmission of *M. bovis* at the livestock-wildlife or human-animal interface occurs essentially because of overlap in their territories (Aranaz *et al*. 2004). Encroachment of wildlife sanctuaries by humans in Tanzania has increased the likelihood of this interaction and infection. Specifically, boundary regions areas in protected areas are used increasingly for grazing of livestock and agriculture and this corresponds with areas where the remaining population of wildlife has been concentrated by this land-use pressure (Etter *et al*. 2006). Factors as source of infection and

How to cite this proceeding: Katale, B.Z., Mbugi, E.V., Kendal, S., Fyumagwa, R.D., Kibik, G.S., Godfrey-Faussett, P. *et al*., 2012, 'Bovine tuberculosis at the human-livestock-wildlife interface: Is it a public health problem in Tanzania? A review', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #463, 8 pages. http://dx.doi.org/10.4102/ojvr.v79i2.463

Note: Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

transmissions of *M. bovis* include persistence of *M. bovis* in infected animals after death and survival of *M. bovis* in the environment (Aranaz *et al*. 2004).

It is not only domestic animals that can experience pathology from *M. bovis*, but a wide range of wild animal species in Africa, including lion (*Panthera leo*), buffalo (*Syncerus caffer)*, wildebeest (*Connochaetes taurinus),* kudu (*Tragelaphus strepsiceros*), bushbuck (*Tragelaphus scriptus*), topi (*Damaliscus lunatus*) and a number of others (Cleveland *et al*. 2005). These animals can be a source of infection for livestock and humans (Aranaz *et al*. 2004) and in Tanzania, a classic example is the illegal hunting of resident and migratory herbivores in protected areas (Loibooki *et al*. 2002; Magige *et al*. 2008; Sinclair & Arcese 1995). The transmission of bovine tuberculosis from wildlife to humans in such cases is by direct contact between infected animals and hunters, either via aerosol contamination when the carcass is opened, through entry of organisms via cuts in the skin or through the alimentary system (Fanning & Edwards 1991; Georghiou *et al*. 1989; Robinson *et al*. 1988).

Tuberculosis in man is generally characterised by loss of weight, weakness, poor appetite, fever, a productive cough, and night sweats. *Mycobacterium tuberculosis* is the most common cause of human TB, however an unknown proportion of cases occur due to *M. bovis* (Acha & Szyfres 1987) at least partly because it is impossible to distinguish tuberculosis infection caused by *M. bovis* from *M. tuberculosis* from clinical signs alone. Bovine tuberculosis (BTB) is often subclinical; when present, clinical signs are not specifically distinctive and are characterised by weakness, anorexia, emaciation, dyspnoea, enlargement of lymph nodes, and cough, particularly with advanced tuberculosis (OIE 2009). Pathologically, BTB is characterised by the formation of granulomas (tubercles) that are usually yellowish and either caseous, caseo-calcareous or calcified and are sometimes encapsulated (De Lesle *et al*. 2002). However, in disseminated cases, multiple small granulomas may be found in numerous organs such as female genitalia (http://www.cfsph.iastate.edu). *Mycobacterium bovis* may present as extra pulmonary tuberculosis often as cervical lymphadenitis (Kleeberg *et al*. 198`4; Mfinanga *et al*. 2004)**.** Based on this premise**,** *M. bovis* may present either as pulmonary TB (47%) or extrapulmonary cases (53%), whereas *M. tuberculosis* presents as 82% pulmonary and 18% extrapulmonary cases (Owendidactic.org, n.d.). Currently, *M. bovis* accounts for only 1% of all human TB in developed countries as compared to 10% in the developing world (Etchechoury *et al*. 2010). According to a WHO report (2010), there was a significant increase (116.6%) of extrapulmonary cases of TB reported in Tanzania between 1995 and 2009, which is suggestive of an emerging *M. bovis* epidemic, however, information on the contributions of *M. bovis* infection to extra pulmonary cases is very limited and it is possible that these extra cases may also be HIV-related (Amanfu 2006). The survey conducted by National Tuberculosis and Leprosy Program, Tanzania (NTLP) in 2009 reported that 20.994 (37.2%) out of 64.417 TB patients in Tanzania were co-infected with HIV. According to the World Bank (2010) report on development indicators, the prevalence of HIV (% of population aged 15–49) in Tanzania is 5.6%.

Tanzania has the third largest domestic stock population in Africa (after Ethiopia and Sudan). According to the Ministry of Livestock Development and Fisheries (MLDF) national census conducted in 2007/2008, the total numbers were 18.5 million cattle, 13.1 million goats, 3.6 million sheep and 53 million poultry. The livestock production system is a pastoral and agro-pastoral system, where movement of animals searching for pasture and water is unrestricted. The presence of large numbers of livestock in traditional settings and where animals are kept in close contact with little veterinary service contributes to spread of disease. Previous studies conducted in pastoral communities in the Arusha region found that a history of TB in the family, drinking raw milk, eating raw animal products, poor ventilation and having poor knowledge concerning transmission of tuberculosis were risk factors for *M. bovis* infection and disease (Mfinanga *et al*. 2004).

The diagnosis of TB in cattle in Tanzania is done by tuberculin skin testing (TST), meat inspection in abattoirs and rarely by bacteriological or molecular techniques The commonly used diagnostic test for human TB in primary health centres and hospitals is microscopic examination and culture, and speciation are not done routinely (Mfinanga *et al*. 2004). Although meat is recommended to be abbatoir-inspected before entering markets, proper meat inspection is not effectively carried out due to the inadequacy of the veterinary service sector as a result of the withdrawal of public veterinary services. In addition, the recommended test and slaughter policy, a disease control program based on slaughter of positive reactors animals, is not properly implemented (see Figure 1) despite our knowledge that this policy has successfully reduced the prevalence of bovine tuberculosis (Michel *et al*. 2009). It is only Algeria, Burkina Faso, Cameroon, Morocco, Namibia and South Africa out of 48 countries in Africa that apply a test-and slaughter policy as a control measure and consider bovine tuberculosis as a notifiable disease (Cosivi 1998). The lack of public finances are obstacles in the control of bovine tuberculosis in many countries. In Tanzania, lack of clear policies on how bovine tuberculosis can be controlled and the failure of health authorities to recognise *M. bovis* as cause of tuberculosis hinder the control of the disease (Kazwala *et al*. 2006).

Few studies have confirmed *M. bovis* in humans in Tanzania (Kazwala *et al*. 2001; Mfinanga *et al*. 2004), however, there is a body of evidence for *M. bovis* infection in man and a description of the relationships between *M. bovis* isolates found in humans and cattle. In their study, Kazwala *et al*. (2001) found that *M. bovis* isolates from man had a 70% – 80% genetic relatedness to those found in cattle, arguably suggesting an infection and evolutionary relationship between them. *Mycobacterium bovis* infection in man has also been reported in other countries in Africa including Nigeria, Zaire and Egypt (Cosivi 1998; Idrisu *et al*. 1977; Idigbe *et al*. 1986; Nafeh *et al*. 1992).

This paper aims to review the current situation of *M. bovis* infection in animals and discuss the zoonotic importance of *M. bovis* in Tanzania. The paper also highlights the burden of tuberculosis, risk factors for infection, communities' knowledge on prevention of the disease and challenges to its control in order to safeguard the human and animal population in Tanzania.

Distribution of bovine tuberculosis in animals in Tanzania

Mycobacterium bovis was demonstrated in Tanzania for the first time in 1952 (Markham 1952). Thereafter, it has been isolated from livestock, wildlife and humans. The prevalence of *M. bovis* in cattle varies between districts, with more infection in older cattle than yearlings and calves (Kazwala *et al*. 2001). Variation in *M. bovis* infection in different geographical areas of the country suggests that there are *M. bovis* infection foci (or hotspots). Shirima *et al*. (2003) suggested that many factors could contribute to *M. bovis* foci, including the presence of mycobacteria in the environment, management practices where animals are extensively grazed and overcrowded at watering points and auction markets. According to Cleveland *et al*. (2007), flooding has also been suggested as a propagating factor of *M. bovis* in the environment.

Reports from several studies that have been conducted in various districts have reported *M. bovis* infection in livestock as well as wildlife. The prevalence of presumed *M. bovis* infection determined by using a single intradermal comparative tuberculin test (SICTT) was 1.7% (n = 181) and 0.4% (n = 259) in Kibaha and Morogoro respectively (Mdegela *et al*. 2004) (Table 1). Durnez *et al*. (2009) reported a prevalence of 2.4% (n = 728) *M. bovis* infection in cattle from 49 herds belonging to extensive and intensive management systems. The prevalence of *M. bovis* infection reported by this study was in same range as demonstrated previously in the same region by Shirima *et al*. (2003). According to Kazwala *et al*. (1996), the highest prevalence of 13.3% *M. bovis* infection was reported in the Southern Highlands and larger herds of cattle had a higher rate of bovine tuberculosis. The prevalence of *M. bovis* infection in cattle in other parts of the country are as follows: Shinyanga, Mwanza, Bukoba: 0.2% in intensively managed farms (Jiwa *et al*. 1997), Rift valley districts (Babati, Hanang, Mbulu and Karatu) 0.93% (Kazwala *et al*. 2001), Manyara region 0.9% (Cleveland *et al*. 2007).

The prevalence of *M. bovis* in cattle has been reported to be higher in intensive systems than in pastoral production systems (Shirima *et al*. 2003). However, in contrast, Durnez *et al*. (2009) reported a higher prevalence of bovine tuberculosis in the extensive than in an intensive system. Husbandry practices in the country could contribute to the difference in prevalence of *M. bovis* infection in extensive and intensive systems. Free movement of animals, overcrowding in communal grazing areas and watering points might contribute to its spread.

A study by Kazwala and colleagues (2006) reported the similarity of *M. bovis* isolates from different geographical locations, which was attributed to migration of cattle as well as sale to local communities**.** Uncontrolled movement of cattle together with a decline in service of public sector in the provision of veterinary services impeded disease control programs in Tanzania. The withdrawal of a public veterinary service forced livestock keepers to take the responsibility of treating their own livestock in order to fill this vacuum (ftp.fao.org/docrep/fao).

In Tanzania, *M. bovis* infections have been confirmed in a number of wildlife species including buffalo (*Syncerus caffer*), African civet (*Civettictis civetta*, n = 1), lion (*Panthera leo*), wildebeest (*Connochaetes taurinus*), topi (*Damaliscus lunatus*) and lesser kudu (*Tragelaphus imberbis*) (Table 2). Of particular note, *M. bovis* has been recovered from apparently healthy wildebeest that did not show lesions at post-mortem (Cleveland *et al*. 2005). These reports are not comprehensive surveys and little information is available on the disease status in wildlife. Analysis of serum samples by using Enzyme Immunoassay (EIA) detected *M. bovis* antibodies in 4% of Serengeti lions, 6% (n = 17) buffalo (*Syncerus caffer*) in Tarangire and 2% (n = 41) wildebeest in the Serengeti (Cleveland *et al*. 2005). It is important to note that the WHO recently issued a statement on the unreliability of serology to diagnose TB and these results are thus at best likely to be a significant underestimate and quantitatively inaccurate.

Countrywide survey of *Mycobacterium bovis* infection in humans

Tuberculosis accounts for approximately 6% of all deaths and 8% of all diseases in humans (NTLP 2007). However, the contribution of *M. bovis* to human tuberculosis in Tanzania is unknown, owing to the absence of efforts in most laboratories in hospitals and health centres to differentiate between the species of the *M. tuberculosis* complex. Despite the lack of data, according to Kazwala *et al*. (2001), *M. bovis* infection is considered as a pathogen of concern to people living in rural areas. In many developed countries, human TB caused by *M. bovis* accounts for around 1% of all TB cases, and sporadic cases occur either in elderly people by reactivation of ancient infections or in immigrants from countries where bovine TB has not been eradicated (De la Rua-Domenech 2006; Etchechoury *et al*. 2010). In the developing world the contribution of *M. bovis* to human tuberculosis is higher and account for an estimated 10% of all TB cases (Cousins *et al*. 1999; Etchochoury *et al*. 2010). Shitaye *et al*. (2007) reported that *M. bovis* infection in man depends on the prevalence of the disease in cattle, socioeconomic conditions, consumer habits, food hygiene practices and medical prophylaxis measures.

Human tuberculosis due to *M. bovis* is mostly the result of transmission from cattle to man and in many cases results into extrapulmonary manifestation (Cosivi 1998; Daborn *et al*., 1997; Kazwala *et al*. 2001; Mfinanga *et al*. 2004; Amanfu *et al*. 2006; Munyeme & Munang'andu 2011). It

has been suggested that *M. bovis* infection in man increases proportionately to the total number of TB cases and that HIV is a major factor for development of active TB disease (Cosivi 1998). In developing countries *M. bovis* infection in humans is also increasing due to the lack of control and diagnostic measures, and pasteurisation of milk (Etter *et al*. 2006). Thus there is every reason to be seriously concerned that the HIV pandemic will result in an increase of human tuberculosis due to *M. bovis*, and a greater degree of transmission of infection to other humans and to animals could well occur. Information on cross transmission of *M. bovis* infection between livestock, wildlife and man in Tanzania is limited. This situation is similar to that of other developing countries where *M. bovis* infection in man is almost certainly underreported (Cosivi *et al*. 1998; Munyeme & Munang'andu 2011) due to the lack of diagnostic facilities to distinguish tuberculosis caused by *M. bovis* and *M. tuberculosis*.

In Africa, consumption of raw fresh milk and improperly cooked or raw meat, and the attitude of some communities which regard bushmeat (poached or hunted wildlife) as a cheap source of protein, represents one of the major risk factors for humans with respect to infection with *M. bovis* (Aranaz *et al*. 2004; Etter *et al*. 2006). Kazwala *et al* (1998) found that out of 805 milk samples that were collected, 31 (3.9%) were positive for mycobacteria. In this study, atypical mycobacteria represented with 87% of the positive samples, however, 6.5% contained *M. bovis*. Whilst these samples represent a minority of the positives, the results show that raw milk is a threat to public health. Moreover, Durnez *et al*. (2009) reported a high prevalence of *M. bovis* and recovered atypical mycobacteria isolates from milk samples in and around Morogoro, Tanzania, and concluded that the populace, especially cattle owners in an extensive system, should be educated concerning bovine tuberculosis. The high level presence of atypical mycobacteria in milk also poses a danger to immunocompromised individuals, especially HIV and AIDS patients.

Mfinanga *et al*. (2004), in their study in the Arusha region, northern Tanzania, investigated 457 biopsy specimens, of which 65 (14.2%) were positive on culture for mycobacteria. In this study, the proportion of atypical mycobacteria was 31 (47.7%) compared to 7 (10.8%) *M. bovis*, and 27 (41.5%) *M. tuberculosis*. They concluded that atypical mycobacteria were more common than *M. tuberculosis* and therefore HIV and raw milk are major risk factors identified for *M. bovis* and non-tuberculous mycobacterial adenitis.

The finding that atypical mycobacteria are common was confirmed in a study by Durnez *et al*. (2011), in Morogoro, Tanzania, where 7.3% of 645 terrestrial small mammals sampled in cattle farms were positive for atypical mycobacteria. A high proportion of the atypical mycobacteria were recovered in insectivores as opposed to rodents. Insectivores feed on insects that spend most of their time in the ground. The recovery of atypical mycobacterium from this source is not surprising perhaps, since mycobacteria are well known environmental and soil dwelling microbes. What is important in this work is that Durnez *et al*. (2011) established a direct correlation between the proportion of atypical mycobacterium in reacting and non-reacting tuberculin farms, complicating the interpretation of tuberculin skin testing (TST) results.

Mycobacterium bovis is one of the well-known causative agents of human extra-pulmonary tuberculosis. This situation prevails in Tanzania, where an early study (Daborn *et al*. 1997), showed that seven out of nineteen lymph node biopsies from suspected extra-pulmonary tuberculosis patients were infected with *M. tuberculosis* and four with *M. bovis*. In most developing countries, the extent of human tuberculosis due to *M. bovis* and the frequency of *M. bovis* extra-pulmonary tuberculosis is not known (Chen *et al*. 2009; Cosivi 1998). However, between 1995 and 2009, the number of reported extra-pulmonary TB cases increased from 6195 to 13 417 in Tanzania (WHO 2010, see Table 3), which is in all likelihood an underestimate (Kazwala *et al*. 2001). The available literature shows that *M. bovis* infections are correlated with people who keep large numbers of cattle (Kazwala 1996) and most cases of extra-pulmonary TB were found in regions with a high proportion of cattle to humans (Kazwala *et al*. 1993). A WHO (2006) zoonotic survey reported the following extr-apulmonary cases in several regions with a high population of cattle in Tanzania; Arusha (30%), Mbeya (28.1%), Iringa (27.3%), Shinyanga (19.8%), Mara (19.7%), Dodoma (19.4%) and Mwanza region (10.8%). A study conducted by Mfinanga *et al*. (2004) found that a disproportionately high number of mycobacterial adenitis was found in subsistence farmers and livestock keepers in Arusha and Mbeya region which is suggestive of cross transmission of *M. bovis*.

A high proportion of cattle are kept in traditional settings in rural areas where knowledge regarding *M. bovis* infection is generally minimal (Mfinanga *et al*. 2004). According to Pušić *et al*. (2008), the persistence of bovine tuberculosis is mostly linked to the traditional extensive breeding system and free-ranging cattle.

Mycobacterium bovis is resistant to pyrazinamide, one of the four first line TB antibiotics and prognosis is often poor (WHO 2010). Given this scenario, it is not surprising that multidrug-resistant strains (MDR) of *M. bovis* have been detected in the USA (Bouvet *et al*. 1993) and Spain (Guerrero *et al*. 1997; Rivero *et al*. 2001).

A study conducted in Tanzania reported genetic relatedness of *M. bovis* isolates in man to those found in cattle (Kazwala *et al*. 2006). These authors found one strain of *M. bovis* from a human patient in Arusha region that had the same genotype as *M. bovis* from cattle within the same geographical area.

Risk factors for *Mycobacterium bovis* infection and disease in humans

The risk factors for bovine tuberculosis are similar in different geographical areas. According to a study conducted in pastoral communities in the northern part of Tanzania, the risk factors for bovine tuberculosis in man were found to

be traditional practices such as sleeping in the same house as animals, lack of knowledge regarding the disease and its risks, HIV and AIDS, raw milk consumption and poor ventilation of houses (Mfinanga *et al*. 2004). In sub-Saharan Africa, active competition between large-scale commercial food enterprises and smaller, less regulated farmers who frequently ignore safety standards for hygiene and product quality, increases the risk of zoonotic tuberculosis (Etter *et al*. 2006).

Consumption of raw fresh milk is also a risk factor for bovine tuberculosis. Mdegella *et al* (2004) and colleagues in their study in Morogoro and Kibaha districts concluded that despite the low prevalence of tuberculosis in milk in the study herds, milk consumers are at high risk of being infected with the disease and insisted that farmers should be educated about the risk of bovine tuberculosis and associated health risks. Mfinanga *et al*. (2003) in their study on the role of livestock keeping in tuberculosis trends in pastoral communities in Babati, Hanang, Mbulu and Karatu districts in the Arusha region, found that all ethnic groups possessed habits and beliefs that increased the risk of being infected with both bovine and human tuberculosis.

In their study, Mdegella *et al*. (2004) found that 14% of milk samples (n = 109) were positive for atypical mycobacteria. In addition, Mfinanga *et al*. (2004) found that several activities, including handling animals and animal products, specifically milking, herding cattle and goats, hunting, slaughtering, handling skins and hides, moving cow dung and plastering walls with dung or mud, might increase risk of zoonotic tuberculosis.

Role of husbandry practice in transmission of bovine tuberculosis

Husbandry practices in Tanzania are divided into three categories, namely, extensive, intensive and semi-intensive systems. The extensive system is traditional and the most popular husbandry practice, and is the main source of milk and meat for Tanzania but receives very little attention from veterinary services. The extensive farming system is practised mostly in rural areas where animals share grazing land and watering points. Most of the cattle kept in Tanzania are Zebu (*Bos indicus)* which are relatively resistant to diseases (Frankel & Soule 1981; Wambura *et al*. 1998). The intensive systems are usually dairy and pig farms which are located in peri-urban areas and are intended for milk and pork production. In these systems, animals are frequently kept indoors and fed complete rations, but in some cases they are grazed outside to supplement feeding.

A single comparative intradermal tuberculin test (SCITT) survey conducted in different farming systems in the eastern zone of Tanzania found that bovine tuberculosis occurred both in intensive and pastoral farming systems, with significantly higher prevalence in the intensive system than in pastoral systems (Shirima *et al*. 2003). This could be attributed to husbandry practices in especially dairy cattle that are confined indoors, where close contact between animals and lack of ventilation increase chances of disease transmission. However, this is contrary to results presented by Durnez *et al*. (2009) in a more recent study, who showed that *M. bovis* infection in extensive farming systems was higher than intensive systems. The contradiction of infection rate in different farming systems could be explained by considering the management practices of each farming practice, or that systems have changed over time. In the extensive system, free movement of cattle which share grazing land and watering points facilitate disease transmission. Poor animal housing and drainage systems designs in intensive farm systems and water supply are key elements that could play a big role in diseases transmission (Pool 1945).

Challenges for control of bovine tuberculosis in cattle in Tanzania

In Tanzania, *M. bovis* is considered as a neglected disease and it has not been assigned as a notifiable disease (Kazwala *et al*. 2006). When it comes to disease control, most resources are directed to notifiable diseases such as contagious bovine pleuropneumonia, African swine fever, rinderpest, contagious caprine pleuropneumonia and Rift Valley fever.

Extensive husbandry practices are widespread and cattle move from one place to another searching for grazing and watering points. This situation is exacerbated during drought, when nomadic tribes move and establish temporary settlements in areas where grazing land and water are available. Bovine tuberculosis could be eradicated at the national level if attention is given at policy level. According to Collins (2006), the success of a national eradication programme, include a clear identification of the goals, of the policies that guide actions and of the sequences of actions that are required within the programme to accomplish these goals. Eradication is possible if movement of cattle is controlled, if there is compulsory testing of all cattle within specified intervals, if positive reactors are removed (slaughtered in a controlled manner), if compensation is provided to farmers for all positive reactors, if compulsory identification is done, and if there is establishment and maintenance of disease free areas, and sufficient funds and manpower to fulfil the task are provided (http://www.pathobiologics.org/ivphc/ref/MCCRINDLE_SHANGAI_2006.pdf). However, this has been impossible for most developing countries because of cost implications. In The Netherlands and Australia, eradication of bovine tuberculosis was successfully achieved due to the practical involvement of farmers as stakeholders (Collins 2006). However, the success of bovine tuberculosis eradication programmes in developed countries was achieved at a time when herds were smaller, and the intensity and demands of production were lower (Collins 2006).

The presence of maintenance hosts in wildlife populations also impede bovine tuberculosis eradication programs (Etter *et al*. 2006). White *et al*. (2008) reported that the presence of multiple hosts for bovine tuberculosis complicate control measures not only because of resistance variation between

the different host species but also because of ecological and behavioural differences. The African buffalo is a known maintenance host of bovine tuberculosis. Aerosol transmission of *M. bovis* within buffalo herds is favoured by their social behaviour (Michel *et al*. 2006) and can be transferred to domestic cattle by intermingling. Globally, the presence of wildlife maintenance hosts threatens *M. bovis* eradication programs (Etter *et al*. 2006). In Tanzania, the National policy on control of wildlife diseases in protected areas is to leave nature to take its own course. There are very few circumstances where treatment or intervention occurs or is allowed. Wildlife immunisation is not allowed in National Parks, Game Reserves and Game Controlled Areas. In circumstances where bovine tuberculosis control in wildlife is not practised and communities around protected area conduct illegal bush hunting in wildlife areas, the risk for cross-transmission of diseases to livestock and humans remain very high.

According to Cross and Gertz (2006) vaccination could potentially control bovine tuberculosis, but combining vaccination and culling of infected animals is a more attractive management option. This is perhaps of little importance for TB control at this stage, since there is no evidence that vaccination against TB would be successful, even if attempted. Furthermore, vaccination of cattle against bovine tuberculosis or improvement in tuberculosis testing procedures will have no effect on wildlife tuberculosis prevalence (Kao *et al*. 1997). In Britain, culling of a maintenance host of bovine tuberculosis, the badgers (*Meles meles*), increased the prevalence of bovine tuberculosis in the cattle population because of ecological and social disturbances of the badger populations (White *et al*. 2008). Thus, the reduction of transmission risk between species is not a simple matter.

However, a reduction of or minimising contact between wildlife and livestock could serve as a priority for future management of the disease in Tanzania. Collins and Grange (1983) reported that 'it is axiomatic that no control measures against transmissible diseases can be totally effective unless all reservoirs of the causative agent can be eliminated'. As in other parts of the world, the challenge facing the control of tuberculosis is the lack of an effective vaccine. The current TB vaccine, *M. bovis* Bacille Calmette-Guérin (BCG) provides little or no protection against pulmonary tuberculosis in cattle and man (Hogartha *et al*. 2005). Nevertheless, bovine tuberculosis could be controlled if there are sound control measures such as regular skin testing and removal of reactors, meat inspection in abattoirs, restriction of cattle movements (Pušić *et al*. 2008).

Conclusion

There is a remarkable paucity of information available on the zoonotic importance of bovine tuberculosis in humans, particularly in developing countries such as Tanzania. The lack of diagnostic facilities to distinguish between *M. bovis* and *M. tuberculosis* is a challenge. Moreover, high proportions of atypical mycobacteria in clinical specimens indicate a widespread environmental occurrence of these organisms, which further complicates accurate diagnosis. Lack of clear policies and implementation regarding control of bovine tuberculosis in cattle impedes control of the disease. Widespread evidence of *M. bovis* infection in animals and humans should be an alarm sign for medical and veterinary health professionals and government bodies. This illustrates the importance of the 'One Health Concept' that can bring together medical and veterinary practitioners as an important tool to fight diseases of public health and economic importance.

Acknowledgement

The authors would like to thank the Southern African Centre for Infectious Diseases Surveillance (SACIDS) funded by the Welcome Trust for support. Prof Mark Rweyemamu, The Director SACIDS is acknowledged for his ideas and encouragement in writing of the manuscript. The SACIDS Collaborative institutions in the United Kingdom (Royal Veterinary College and London School of Hygiene and Tropical Medicine) through London International Development Centre (LIDC) are also acknowledged for their support during a stay in the UK.

Competing interest

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

Authors' contributions

B.Z.K. (Muhimbili University of Health and Allied Sciences) reviewed the literature, drafted and wrote the manuscript. E.V.M. (Muhimbili University of Health and Allied Sciences) contributed to the drafting and critical review of the manuscript. S.K. (Royal Veterinary College) made conceptual contribution and critical review of the manuscript. R.D.F. (Tanzania Wildlife Research Institute) made conceptual contribution and significant review of the manuscript. G.S.K. (Kilimanjaro Clinical Research Institute) assisted in critical review of the manuscript and made contribution on preparation of the manuscript. P.G.F. (London School of Hygiene and Tropical Medicine) made conceptual contribution and critical review of the manuscript. J.D.K. (Tanzania Wildlife Research Institute) contributed to the drafting, conceptualization and critical review of the manuscript. P.v.H. (University of Stellenbosch) made conceptual contribution and critical review of the manuscript. M.I.M. (Muhimbili University of Health and Allied Sciences) made conceptual contribution and critical review of the manuscript. All authors have read and approved the final manuscript.

References

Acha, P.N. & Szyfres, B., 1987, *Zoonotic tuberculosis, in Zoonoses and communicable diseases common to man and animals*, 2nd edn., Pan American Health Organisation/World Health Organisation, Scientific publication No 503, Washington.

Amanfu, W., 2006, 'The situation of tuberculosis and tuberculosis control in animals of economic interest', *Tuberculosis* 86, 330–335. http://dx.doi.org/10.1016/j.tube.2006.01.007

Aranaz, A., Juan, L., Montero, N., Sanchez, C., Galka, M., Delso, C. *et al*., 2004, 'Bovine tuberculosis (*Mycobacterium bovis*) in wildlife in Spain', *Journal of Clinical Microbiology* 42, 2602–2608. http://dx.doi.org/10.1128/JCM.42.6.2602-2608.2004

Bouvet, E., Casalino, E., Mendoza-Sassi, G., Lariven S., Vallée E., Pernet M. *et al*., 1993, 'Nosocomial outbreak of multidrug-resistant *Mycobacterium bovis* among HIV-infected patients: A case control study', *AIDS* 7, 1453–1460. http://dx.doi.org/10.1097/00002030-199311000-00008

Chen, Y., Chao, Y., Deng, Q., Liu, T., Xiang, J., Chen, J. *et al*., 2009, 'Potential challenges to the stop TB plan for humans in China; Cattle maintain *M. bovis* and *M. tuberculosis*', *Tuberculosis* 89, 95–100. http://dx.doi.org/10.1097/00002030-199311000-00008

Cleaveland, S., Mlengeya, T., Kazwala, R.R., Michel, A., Kaare, M.T., Jones, S.L. *et al*., 2005, 'Tuberculosis in Tanzanian wildlife', *Journal of Wildlife Diseases* 41, 446–453.

Cleaveland, S., Shaw, D.J., Mfinanga, S.G., Shirima, G., Kazwala, R.R., Eblate E. *et al*., 2007, '*Mycobacterium bovis* in rural Tanzania, risk factors for infection in human and cattle populations', *Tuberculosis* 87, 30–43. http://dx.doi.org/10.1016/j.tube.2006.03.001

Collins, C.H. & Grange, J.M., 1983, 'The bovine tubercle bacillus', *Journal of Applied Bacteriology* 55, 13–29. http://dx.doi.org/10.1111/j.1365-2672.1983.tb02643.x

Collins, J.D., 2006, 'Tuberculosis in cattle: Strategic planning for the future', *Veterinary Microbiology* 112, 369–381. http://dx.doi.org/10.1016/j.vetmic.2005.11.041

Cosivi, O., Grange, J.M, Daborn, C.J., Rviglione, M.M., Fujikura, T., Cousins, D. *et al*., 1998, 'Zoonotic tuberculosis due to *Mycobacterium bovis* in developing countries', *Emerging Infectious Diseases* 4, 59–70. http://dx.doi.org/10.3201/eid0401.980108

Cousins, D.V., Williams, S.N. & Dawson, D.J., 1999, 'Tuberculosis due to Mycobacterium bovis in the Australian population, DNA typing of isolates, 1970–1994', *The International Journal of Tuberculosis and Lung Disease* 3**,** 722–731.

Cross, P.C. & Getz, W.M., 2006, 'Assessing vaccination as a control strategy in an ongoing epidemic', 'Bovine tuberculosis in African buffalo', *Ecological Modelling* 96, 494–504. http://dx.doi.org/10.1016/j.ecolmodel.2006.02.009

Daborn, C.J., Grange, J.M. & Kazwala, R.R., 1996, 'The bovine tuberculosis cycle–An African perspective', *Society for Applied Bacteriology symposium series* 25, 27s–32s. http://dx.doi.org/10.1111/j.1365-2672.1996.tb04595.x

De la Rua-Domenech, R., 2006, 'Human *Mycobacterium bovis* infection in the United Kingdom: Incidence, risks, control measures and review of the zoonotic aspects of bovine tuberculosis', *Tuberculosis* 86, 77–109. http://dx.doi.org/10.1016/j.tube.2005.05.002

De Lesle, G.W., Bengis, R.G., Schmitt, S.M. & O' Brien, D.J., 2002, 'Tuberculosis in free-ranging wildlife: detection, diagnosis and management', *Revue Scientifique et Technique* (OIE) 21, 317–334.

Durnez, L., Eddyani, M., Mgode, G.F., Katakweba, A., Katholi, R.I., Kazwala R.R. *et al*., 2008, 'First detection of mycobacteria in African rodents and insectivores, using stratified pool screening', *Applied and Environmental Microbiology* 74, 768–773. http://dx.doi.org/10.1128/AEM.01193-07

Durnez, L., Katakweba, A., Sadiki, H., Katholi, C.R., Kazwala, R.R., Machang'u, R.R. *et al*., 2011, 'Mycobacteria in terrestrial small mammals on cattle farms in Tanzania', *Veterinary Medicine International.* http://dx.doi.org/10.4061/2011/495074

Durnez, L., Sadiki, H., Katakweba, A., Machang'u, R.R., Kazwala, R.R., Leirs, H. *et al*., 2009, 'The prevalence of *Mycobacterium bovis*-infection and typical mycobacterioses in cattle in and around Morogoro, Tanzania', *Tropical Animal Health and Production* 41, 1653–1659. http://dx.doi.org/10.1007/s11250-009-9361-4

Etchechoury, I., Echeverria Valencia, G., Morcillo, N., Sequeira, M.D., Imperiale, B., López, M. *et al*., 2010, 'Molecular typing of *Mycobacterium bovis* Isolates in Argentina: First description of a person-to-person transmission case', *Zoonoses Public Health* 57, 375–381. http://dx.doi.org/10.1111/j.1863-2378.2009.01233.x

Etter, E., Donald, P., Jori, F., Caron, A., Goutard, F. & Roger, F., 2006, 'Risk analysis and bovine tuberculosis, a re-emerging zoonosis', *Annals of the New York Acadamy of Sciences* 1081, 61–73. http://dx.doi.org/10.1196/annals.1373.006

Fanning, A. & Edwards, S., 1991, '*Mycobacterium bovis* infection in human beings in contact with elk (*Cervus elaphus*) in Alberta, Canada', *Lancet* 338, 1253–1255. http://dx.doi.org/10.1016/0140-6736(91)92113-G

Figueroa-Munoz, J. & Ramon-Pardo, P., 2008, 'Tuberculosis control in vulnerable groups', *Bulletin of the World Health Organization*, Part issues 86, 657–736.

Frankel, O.H. & Soule, E.M., 1981, 'Conservation and Evolution', Cambridge University Press, Cambridge, USA.

Georghiou, P., Patel, A.M. & Konstantinos, A., 1989, '*Mycobacterium bovis* as an occupational hazard in abattoir workers', *Australian and New Zealand Journal of Medicine* 19, 409–410. http://dx.doi.org/10.1111/j.1445-5994.1989.tb00289.x

Grange, J.M. & Collins, C.H., 1987, 'Bovine tubercle bacilli and disease in animals and man', *Epidemiology and Infection* 99, 221–234. http://dx.doi.org/10.1017/S0950268800067686

Guerrero, A., Cobo, J., Fortún, J., Navas, E., Quereda, C., Assensio, A. *et al*., 1997, 'Nosocomial transmission of *Mycobacterium bovis* resistant to 11 drugs in people with advanced HIV-1 infection', *Lancet* 350, 1738–1742. http://dx.doi.org/10.1016/S0140-6736(97)07567-3

Hogarth, P.J., Logan, K.E., Vordermeier, H.M., Singh, M., Hewinson, R.G. & Chambers, M.A., 2005, 'Protective immunity against *Mycobacterium bovis* induced by vaccination with Rv3109c–a member of the *esat*-6 gene family', *Vaccine* 23, 2557–2564. http://dx.doi.org/10.1016/j.vaccine.2004.11.030

Idigbe, E.O., Anyiwo, C.E. & Onwujekwe, D.I., 1986, 'Human pulmonary infections with bovine and atypical mycobacteria in Lagos, Nigeria', *Journal of Tropical Medicine and Hygiene* 89, 143–8.

Idrisu, A. & Schnurrenberger, P., 1997, 'Public health significance of bovine tuberculosis in four northern states of Nigeria, a mycobacteriologic study', *Nigerian Medical Journal* 7, 384–7.

Jiwa, S.H.F., Kazwala, R.R., Aboud, A.A.O. & Kalaye, W.J., 1997, 'Bovine tuberculosis in the Lake Victoria zone of Tanzania and its possible consequences for human health in the HIV/AIDS era', *Veterinary Research Communication* 21, 533–539. http://dx.doi.org/10.1023/A:1005900929479

Kao, R.R., Robberts, M.G. & Ryan, T.J., 1997, 'A model of bovine tuberculosis control in domesticated cattle herds', *Proceedings. Biological Sciences/ The Royal Society* 264, 1069–1076.

Kazwala, R.R, Daborn, C., Sharp, J.M., Kambarage, D.M., Jiwa, S.F. & Mbembeti, N.A., 2001, 'Isolation of *Mycobacterium bovis* from human cases of cervical adenitis in Tanzania, a cause for concern?', *International Journal of Tuberculosis and Lung Diseases* 5, 87–91.

Kazwala, R.R., 1996, 'Molecular epidemiology of bovine tuberculosis in Tanzania', PhD thesis, Edinburgh, University of Edinburgh.

Kazwala, R.R., Catley, A.P., Daborn, C.J. & Kambarage, D.M., 1993, 'The zoonotic implications of bovine tuberculosis in Tanzania', in P.M. Msolla & S.I.K. Kimera (eds.), Proceedings of the 10th Tanzania Veterinary Association scientific conference. Morogoro, Tanzania: *Tanzania Veterinary Association* 1993, 67–81.

Kazwala, R.R., Daborn, C.J., Kusiluka, L.J.M., Jiwa, S.F.H., Sharp, J.M. & Kambarage, D.M., 1998, 'Isolation of Mycobacterium species from raw milk of pastoral cattle of the Southern Highlands of Tanzania', *Tropical Animal Health and Production* 30, 233–239. http://dx.doi.org/10.1023/A:1005075112393

Kazwala, R.R., Daborn, C.J., Kusiluka, L.J.M., Jiwa, S.F.H., Sharp, J.M. & Kambarage, D.M., 1998, 'Isolation of Mycobacterium species from raw milk of pastoral cattle of the Southern Highlands of Tanzania', *Tropical Animal Health and Production* 30, 233–239. http://dx.doi.org/10.1023/A:1005075112393

Kazwala, R.R., Kambarage, D.M., Daborn, C.J, Nyange, J., Jiwa, S.F.H. & Sharp, J.M., 2001, 'Risk factors associated with the occurrence of bovine tuberculosis in cattle in the Southern Highlands of Tanzania', *Veterinary Research Communications* 25, 609–614. http://dx.doi.org/10.1023/A:1012757011524

Kazwala, R.R., Kusiluka, L.J.M., Sinclair, K., Sharp, J.M. & Daborn, C.J., 2006, 'The molecular epidemiology of *Mycobacterium bovis* infections in Tanzania', *Veterinary Microbiology* 112, 201–210. http://dx.doi.org/10.1016/j.vetmic.2005.11.026

Kleeberg, H.H., 1984, 'Human tuberculosis of bovine origin in relation to public health', *Revue Scientifique et Technique* (OIE) 3, 11–32.

Loibooki, M., Hofer, H., Campbell, K.L.I. & East, M.L., 2002, 'Bushmeat hunting by communities adjacent to the Serengeti National Park, Tanzania, the importance of livestock ownership and alternative sources of protein and income', *Environmental Conservation* 29, 391–398. http://dx.doi.org/10.1017/S0376892902000279

Magige, F.J., Holmern, T., Stokke, S., Mlingwa, C. & Røskaft, E., 2008, 'Does illegal hunting affect density and behaviour of African grassland birds? A case study on ostrich (*Struthio camelus*)', *Biodiversity and Conservation* 18 , 1361–1373. http://dx.doi.org/10.1007/s10531-008-9481-6

Markham, A.E.G., 1952, 'Bovine tuberculosis in the Southern Highlands Province of Tanganyika', PhD thesis, University of London, UK.

Mdegela, R.H., Kusiluka, L.J., Kapaga, A.M., Karimuribo, E.D., Turuka, F.M., Bundala, A. *et al*., 2004, 'Prevalence and determinants of mastitis and milk-borne zoonoses in smallholder dairy farming sector in Kibaha and Morogoro districts in Eastern Tanzania', *Journal of Veterinary Medicine* B, *Infectious Diseases and Veterinary Public Health* 51, 123–128. http://dx.doi.org/10.1111/j.1439-0450.2004.00735.x

Mfinanga, G.S., Morkve, O., Kazwala, R.R., Cleaveland, S., Sharp, J.M., Shirima, G.M. *et al*., 2003, 'Tribal differences in perception of tuberculosis, a possible role in tuberculosis control in Arusha, Tanzania', *The International Journal of Tuberculosis and Lung Disease* 7, 933–941.

Mfinanga, S.G.M., Morkve, O., Kazwala, R.R., Cleaveland, S., Sharp, M.J., Kunda, J. *et al*., 2004, 'Mycobacterial adenitis, role of *Mycobacterium bovis,* non tuberculous mycobacteria, HIV infection, and risk factors in Arusha, Tanzania', *East African Medical Journal* 81, 171–178. http://dx.doi.org/10.4314/eamj.v81i4.9150

Michel, A.L., Bengis, R.G., Keet, D.F., Hofmeyr, M., De Klerk, L-M., Cross, P.C. *et al*., 2006, 'Wildlife tuberculosis in South African conservation areas, implications and challenges', *Veterinary Microbiology* 112**,** 91–100. http://dx.doi.org/10.1016/j.vetmic.2005.11.035

Michel, A.L., Coetzee, M.L., Keet, D.F., Maré, L., Warren, R., Cooper, D. *et al*., 2009, 'Molecular epidemiology of *Mycobacterium bovis* isolates from free ranging wildlife in South African game reserves', *Veterinary Microbiology* 133, 335–343. http://dx.doi.org/10.1016/j.vetmic.2008.07.023

Ministry of Livestock Development and Fisheries (MLDF) (National livestock census - sample statistics 2007 – 2008, Agricultural policy, Dar es Salaam, Tanzania.

Munyeme, M. & Munang'andu, H.M.A., 2011, 'Review of bovine tuberculosis in the Kafue basin ecosystem', *Veterinary Medicine International*.

Nafeh, M.A., Medhat, A., Abdul-Hameed, A-G., Ahmad, Y.A., Rashwan, N.M. & Strickland, G.T., 1992, 'Tuberculous peritonitis in Egypt, the value of laparoscopy in diagnosis', *The American Journal of Tropical Medicine and Hygiene* 47, 470–477.

National Tuberculosis and Leprosy Programme Tanzania (NTLP), 2009, Annual report 1995, Ministry of Health, Dar es Salaam, Tanzania.

National Tuberculosis and Leprosy Programme Tanzania (NTLP), 2007, Annual report 1995, Ministry of Health, Dar es Salaam, Tanzania.

OIE Terrestrial Manual, 2009, Bovine Tuberculosis, World assembly of delegates of the OIE in May 2009, Chapter 2.4.7.

Pool, W.A., 1945, 'The Eradication of Tuberculosis in Cattle, Section of comparative medicine, Disease in relation to animal husbandry', *Proceedings of the Royal Society of Medicine* 39, 77–82.

Pušić, A., Savić-Jevdenić, S., Lalošević, D., Bugarski, D., Prodanov, J. & Grgić, Z., 2008, 'An epidemiological evaluation of bovine tuberculosis in South Backa region of Vojvodina Province', *Lucrări Stiinlifice Medicină Veterinară*, vol. xli, Timisoara.

Rivero, A., Márquez, M., Santos, J., Pinedo, A., Sánchez, M.A., Esteve, A., Samper, S. & Martin, C., 2001, 'High rate of tuberculosis reinfection during a nosocomial outbreak of multidrug-resistant tuberculosis caused by *Mycobacterium bovis* strain B', *Clinical Infectious Disease* 32, 159–161. http://dx.doi.org/10.1086/317547

Robinson, P., Morris, D. & Antic, R., 1988, *Mycobacterium bovis* as an occupational hazard in abattoir workers', *Australian and New Zealand Journal of Medicine* 18, 701–703. http://dx.doi.org/10.1111/j.1445-5994.1988.tb00156.x

Shirima, G.M., Kazwala, R.R. & Kambarage, D.M., 2003, 'Prevalence of bovine tuberculosis in cattle in different farming system in Tanzania', *Preventive Veterinary Medicine* 57, 167–172. http://dx.doi.org/10.1016/S0167-5877(02)00214-3

Shitaye, J.E., Tsegaye, W. & Pavlik, I., 2007, 'Bovine tuberculosis infection in animal and human populations in Ethiopia: A review', *Veterinary Medicine* 52, 317–332.

Sinclair, A.R.C. & Arcese, P., 1995, Serengeti II, 'Dynamics, management, and conservation of an ecosystem', The University of Chicago Press, LTD., London.

Wambura, P.N., Gwakisa, P.S., Silayo, R.S. & Rugaimukamu, E.A., 1998, 'Breed-associated resistance to tick infestation in *Bos indicus* and their crosses with *Bos Taurus*', *Veterinary Parasitology* 77, 63–70. http://dx.doi.org/10.1016/S0304-4017(97)00229-X

White, P.C.L., Bohm, M., Marion, G. & Hutchings, M.R., 2008, 'Control of bovine tuberculosis in British livestock – There is no "silver bullet", *Trends in microbiology* 16, 420–427. http://dx.doi.org/10.1016/j.tim.2008.06.005

World Bank Report, 2010, Development Indicators, viewed on 15 July 2011, from http://data.worldbank.org/news/world-development-indicators-2010-released

World Health Organization (WHO), 2006, 'The Control of Neglected Zoonotic Diseases, A route to Poverty alleviation', Report of a Joint WHO/DFID-AHP Meeting with the participation of FAO and OIE, Geneva, 20 and 21 September 2005, from http://www.int/zoonoses/Report_Sept06.pdf.

World Health Organization (WHO), 2010, Global tuberculosis control, Global, region and country-specific for key indicators, viewed 25 June 2011, from www.who.int/tb/data

Identification of the plague reservoir in an endemic area of Zambia

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How to cite this abstract:
Hang'ombe, B.M., Nakamura, I., Kaile, D., Mweene, A.S., Samui, K.L., Kilonzo, B.S. *et al.*, 2012, 'Identification of the plague reservoir in an endemic area of Zambia', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #464, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.464

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Yersinia pestis, the bacterial agent of plague, is primarily a parasite of wild rodents that persists in permanent, discrete enzootic foci throughout the world. The disease is transmitted in humans by bites from fleas of wildlife rodent species. Therefore surveillance is the ultimate public health solution through plague detection in domestic dogs, other carnivores and wild rodents. The investigations of die-offs amongst plague-susceptible colonial rodents are also significant to determine the presence of *Y. pestis* in a susceptible population.

This study details the identification of the plague reservoir in a suspected endemic area of Zambia. The study was undertaken through rodent investigation for the presence of *Y. pestis*. A total of 105 rodents were sampled routinely and during a suspected plague period. On dissection 4 (3.81%, 95% CI: 1.23–10.0) rodents sampled during an outbreak showed signs of spleen enlargement. The blood, liver, lymph nodes and spleen of each rodent were subjected to culture on 6% sheep blood agar and MaCconkey agar. Colonies obtained were identified as *Y. pestis* by colony morphologic features, biochemical profiles, mouse inoculation assay and polymerase chain reaction (PCR). The PCR primers used targeted the *Y. pestis* plasminogen activator gene, chromosomal ferric iron uptake regulation gene and the outer membrane protein B gene.

The isolates were also subjected to antibiotic sensitivity tests using the disk diffusion method on Mueller-Hinton agar with sensitivity being observed with ampicillin, amoxicillin, chloramphenicol, gentamycin, streptomycin, tetracycline and trimethoprim-sulfamethoxazole. The findings, identifies a natural reservoir of *Y. pestis* in Zambia providing the public health officials with a definite host for the control strategy.

Filoviral haemorrhagic fevers: A threat to Zambia?

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How to cite this abstract:
Changula, K. & Mweene, A.S., 2012, 'Filoviral haemorrhagic fevers: A threat to Zambia?', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #465, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.465

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Filoviral haemorrhagic fevers (FVHF) are caused by agents belonging to Filoviridae family, Ebola and Marburg viruses. They are amongst the most lethal pathogens known to infect humans. Incidence of FVHF outbreaks are increasing, with affected number of patients on the rise. Whilst there has been no report yet of FVHF in Zambia, its proximity to Angola and Democratic Republic of Congo, which have recorded major outbreaks, as well as the open borders, increased trade and annual migration of bats between these countries, puts Zambia at present and increased risk. Previous studies have indicated bats as potential reservoir hosts for filoviruses. An increasing population with an increasing demand for resources has forced incursion into previously uninhabited land, potentially bringing them into contact with unknown pathogens, reservoir hosts and/or amplifying hosts. The recent discovery of a novel arenavirus, Lujo, highlights the potential that every region, including Zambia, has for being the epicentre or primary focus for emerging and re-emerging infections. It is therefore imperative that surveillance for potential emerging infections, such as viral haemorrhagic fevers be instituted. In order to accomplish this surveillance, rapid detection, identification and monitoring of agents in patients and potential reservoirs is needed. International co-operation is the strategy of choice for the surveillance and fight against emerging infections. Due to the extensive area in which filoviral infections can occur, a regional approach to surveillance activities is required, with regional referral centres. There is a need to adopt shared policies for the prevention and control of infectious diseases. There is also need for optimisation of currently available tests and development of new diagnostic tests, in order to have robust, highly sensitive and specific diagnostic tests that can be used even where there are inadequate laboratories and diagnostic services.

Rift Valley fever: Real or perceived threat for Zambia?

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How to cite this abstract:
Dautu, G., Sindato, C., Mweene, A.S., Samui, K.L., Roy, P., Noad, R. *et al.*, 2012, 'Rift Valley fever: Real or perceived threat for Zambia?', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #466, 6 pages. http://dx.doi.org/10.4102/ojvr.v79i2.466

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Rift Valley fever (RVF) in Zambia was first reported in 1974 during an epizootic of cattle and sheep that occurred in parts of Central, Southern and Copperbelt Provinces. In 1990, the disease was documented in nine districts of the provinces of Zambia. In the last two decades, there have been no reports of RVF. This long period without reported clinical disease raises questions as to whether RVF is a current or just a perceived threat. To address this question, World Organisation for Animal Health (OIE) disease occurrence data on RVF for the period 2005–2010 in the Southern Africa Development Community (SADC) was analysed. From the analysis, it was evident that most countries that share a common border with Zambia had reported at least one occurrence of the disease during the period under review. Due to the absence of natural physical barriers between Zambia and most of her neighbours, informal livestock trade and movements is a ubiquitous reality. Analysis of the rainfall patterns also showed that Zambia received rains sufficient to support a mosquito population large enough for high risk of RVF transmission. The evidence of disease occurrence in nearby countries coupled with animal movement, and environmental risk suggests that RVF is a serious threat to Zambia. In conclusion, the current occurrence of RVF in Zambia is unclear, but there are sufficient indications that the magnitude of the circulating infection is such that capacity building in disease surveillance and courses on recognition of the disease for field staff is recommended. Given the zoonotic potential of RVF, these measures are also a prerequisite for accurate assessment of the disease burden in humans.

Introduction

Rift Valley fever (RVF) is an economically important, emerging arthropod-borne viral disease of both livestock and man. The disease was first identified in 1931 following sudden death of lambs and ewes on a single farm along the shores of Lake Naivasha in the greater Rift Valley of Kenya (Daubney *et al*. 1931, reviewed in Pepin *et al*. 2010). The importance of the disease lies in its public health impact and the economic losses resulting from the cessation of trade in livestock and livestock related products. This has been shown by the prolonged import bans from countries in the Horn of Africa where RVF has been registered, causing great hardship to the livestock trade based communities.

In Zambia, RVF was first reported in 1974 during an epizootic of cattle and sheep that occurred in Chisamba (Central Province) and Mazabuka (Southern Province) districts and some parts of Copperbelt Province (Hussuein *et al*. 1987). Human death due to RVF disease in Chisamba was also previously reported (Watts *et al*. 1984). Several other epizootics have been reported in the same areas (Department of Veterinary and Tsetse Control Services annual reports 1975–1989). For a long time the disease was known to be confined to the same outbreak areas. However, a sero-epidemiological study carried out between January 1990 and March 1991 showed that the disease could have a country wide distribution (Samui *et al*. 1997).

Although RVF is considered endemic in Zambia, the clinical disease has not been reported in the last two decades. This long period without reported cases raises questions as to whether RVF is a current, or just a perceived threat. This article reviews some of the reasons as to why RVF has not been reported in Zambia in the recent past through focusing on the aetiology, epidemiology and risk factors associated with the disease. Furthermore, the OIE disease occurrence data on RVF for the period 2005–2010 in the Southern Africa Development Community (SADC) will be analysed.

Aetiology

Rift Valley fever is an arthropod-borne viral disease caused by a Rift Valley fever virus (RVFV) of the family *Bunyaviridae* and genus *Phlebovirus*. The RVFV genome is made up of three segments

namely L, M and S which are packaged together in the virions in the form of ribonucleoprotein (RNP). The L and M segments present in the virus particle are of negative polarity. The L segment encodes a single protein which is the viral RNA-dependent RNA polymerase (Muller *et al*. 1994; Pepin *et al*. 2010), and for the precursor to the glycoproteins. The M segment encodes four proteins, NSm1, NSm2 and two glycoproteins, Gc and Gn (Collett *et al*. 1985; Collett *et al*. 1986; Schmaljohn & Hooper 2001). The S segment utilises an ambisense strategy to code for two proteins, the nucleoprotein N, in the negative polarity, and a non-structural protein, NSs, in the positive polarity (Giorgi *et al*. 1991). The virus is resistant to heat and could stay active for four months at 21 °C and 3 hours at 56 °C (Flick *et al*. 2005). However, it can be inactivated by strong calcium or sodium hypochlorite, especially when treated for three hours at 56 °C (Sossah 2009).

There is only one serotype of RVFV known to date (Martin *et al*. 2008). In Zambia, there is no evidence regarding the physical isolation of RVF virus from the field. However, there is enough serological evidence to suggest the presence of the virus (Davies *et al*. 1992). Clinical manifestations of the disease in ruminant livestock, especially sheep and cattle, are characterised by high mortality (100% in neonatal animals and 10% – 20% amongst adult animals) and high abortion rates particularly in infected pregnant animals (Coetzer 1977, 1982; Swanepoel 1994). In humans the disease is self-limiting, although complications of hemorrhagic fever, retinitis, blindness, and encephalitis may occur in 1% – 2% of affected individuals with a case fatality of approximately 10% – 20% (Madani *et al*. 2003).

Epidemiology

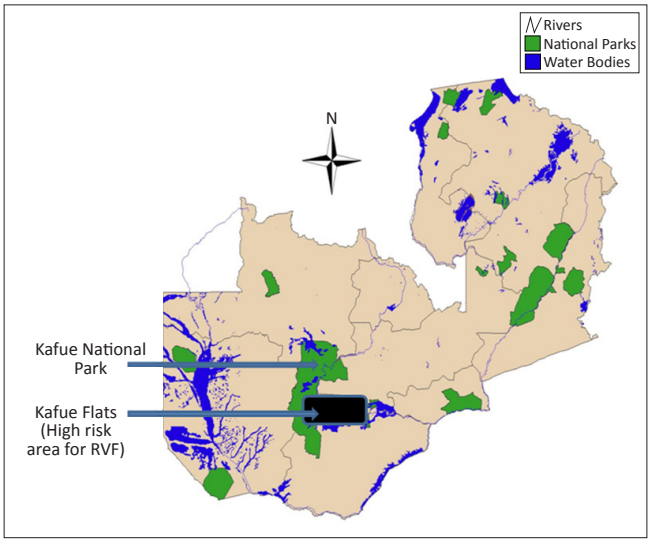
Rift Valley fever disease is an important endemic problem in sub-Sahara Africa which includes Zambia. The disease outbreaks in Africa occur at irregular intervals of 5–15 years in the savannah grasslands and 25–35 years in the semi-arid regions. Rift Valley fever virus has demonstrated a real capacity to emerge in virgin areas as shown by the outbreaks in Egypt (1977, north of the Sahara desert), Madagascar (1979), Saudi Arabia and Yemen (2000), outside the continent of Africa (Centre for Disease Control and Prevention 2000; Morvan *et al*. 1992; Shoemaker *et al*. 2002).

Rift Valley fever virus has two transmission cycles, namely the enzootic and epizootic cycles. Enzootic cycle occurs during periods of normal amounts of rainfall. In the enzootic cycle, RVF virus is maintained by low-level activity within the mosquito vector population involving transovarial transmission with occasional infection and amplification of virus in wildlife such as African buffaloes (*Syncerus caffer*) or susceptible livestock. Epizootic or epidemic cycles occur following extended periods of exceptionally plentiful rainfall and subsequent flooding of dambos, which results in the emergence of abundant numbers of floodwater *Aedes* mosquitoes. These transovarially infected mosquitoes feed on susceptible livestock (e.g. sheep and cattle) that rapidly develop high-titer viremias and signs of clinical disease. The infected livestock in turn infect secondary bridge mosquito vectors such as the *Culex* or *Anopheles* spp. (Coetzer 1977, 1982; Turell *et al*. 1984) and thereafter, human infections develop as a result of bites from infected mosquitoes (*Aedes, Culex* or *Anopheles* spp.), exposure to infectious aerosols, handling of aborted fetal materials, or percutaneous injury during slaughtering or necropsy of viremic animals (Meegan 1981; Van Velden *et al*. 1977). It is unclear whether humans have any important biological role as amplification hosts in the RVF virus epizootic or epidemic life cycle.

The past distribution of RVF outbreak in Zambia is well documented (Department of Veterinary and Tsetse Control Services annual reports, 1975–1989; Davies *et al*. 1992; Hussein *et al*. 1987; Samui *et al*. 1997). The high risk areas have been identified where several RFV epizootics had occurred. These areas include Ndola in the Copperbelt Province, Chisamba in the Central Province, and Lusaka and Mazabuka in Southern Province (Hussein *et al*. 1987). Rift Valley fever clinical signs were limited to susceptible *Bos taurus* cattle and imported sheep. However, a RVF sero-epidemiological study carried out in 5 traditionally managed herds that graze in the Kafue flats (flood plain grasslands [Figure 1]) showed that RVF was not only a threat to the commercial exotic breeds but also to the indigenous local breeds. For instance, a study carried out by Ghirotti *et al*. (1991) in the Kafue flats showed that 14% of the indigenous cattle tested seroconverted to RVF. The 14% RVF sero-prevalence rate was attributed to high concentration of wild and domestic ruminants grazing together in the flood plains during the dry season. It is worthwhile to mention here that no studies have been done to determine the role of wildlife in the maintenance of RVFV in Zambia. Furthermore, the sero-epidemiological study carried out between January 1990 and March 1991 in at least one district of each of the nine provinces showed the existence of RVF in the respective districts studied (Samui *et al*. 1997). The high positive rates were also observed in areas where cattle grazed in dambos or flood plains (Table 1 and Figure 2). The results of this study suggest that RVF was not only endemic in the commercial farms of Chisamba, Lusaka and Mazabuka but could be endemic throughout most of the cattle producing parts of the country. The implication of these results are that the traditional farmers who graze their cattle in the flood plains or dambos together with all those involved in livestock production are particularly at risk of contracting RVF if it is still circulating at high prevalence in cattle, sheep and goats, and if the local environment is favourable for transmission of the virus.

In Zambia the disease has not been reported for the last two decades. This period without detected disease does not necessary mean that RVF is not a threat to Zambia. This is so because from past RVF research, a low level of RVF virus transmission has been detected in livestock and humans during inter-epizootic periods (IEP). For example, a study carried out in animals born before the 1997–1998 and after the 2006–2007 outbreaks in Kenya showed a low IgG prevalence

against RVF, indicating that virus transmission continued in Kenya during an IEP (Rostal *et al.* 2010). Similarly another study carried out in Senegal during an IEP in sheep and goats indicated a 2.9% seroprevalence (Chevalier *et al*. 2003). In Zambia, a study carried out during 1982–1986 on a sentinel herd using indigenous breeds at Lutale in Mumbwa showed a low level of seasonal RVF virus activity of 3% – 8% (Davies *et al*. 1992). The studies carried out in Zambia and other parts of Africa clearly support the existence of low degree of RVFV transmission during the IEP and that this low level of seasonal virus activity could generate epizootics as witnessed by the 1985–1986 epizootics in Zambia (Hussein *et al*. 1987). More interestingly, evidence of interepidemic human transmission of RVFV has been reported. In Kenya, research done on children born after the documented RVF outbreak of 1997–1998 showed that low-level interepidemic transmission to humans continued to occur (LaBeaud *et al*. 2008). Although there are no studies done on interepidemic human transmission of RVFV in Zambia, the results of the previous studies done in animals and humans during IEP clearly shows that RVF is a serious threat to Zambia.



RVF, Rift Valley fever.

FIGURE 1: Map of Zambia showing the location of the Kafue Flats.

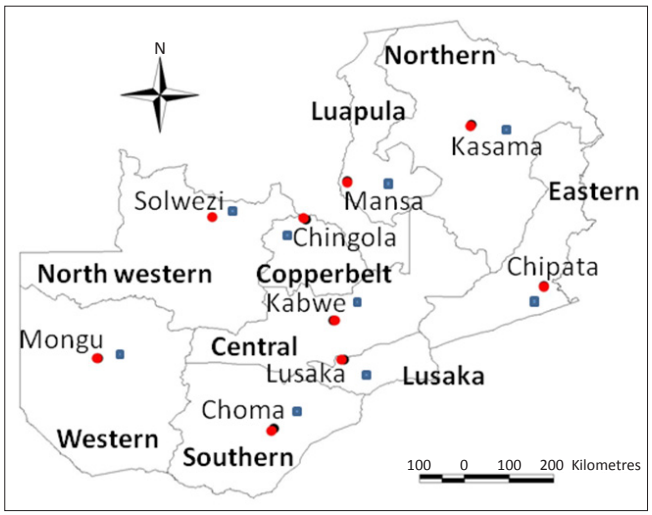


FIGURE 2: Map of Zambia showing sampling location denoted with square dots.

Although a low level virus activity has been demonstrated during IEP in studies carried out in Kenya and Zambia (Davies *et al*. 1992; Rostal *et al.* 2010), no RVF associated abortion or death was observed. This implies that the infected livestock developed no clinical signs or developed mild febrile illness with no obvious clinical disease. The lack of specific RVF signs during IEP implies that the presence of RVF could only be detected through specific, well-focused, active surveillance. Therefore countries like Zambia with limited resources to carry out this type of surveillance during IEP could have problems in detecting the threat of RVF early and subsequently fail to report the disease. Analysis of the OIE disease occurrence data on RVF for the period 2005–2010 in the SADC region showed that most countries that share a common border with Zambia had reported at least one occurrence of the disease during the period under review. Since conditions which predispose to RVF activities tend to occur on a regional level (Davies *et al.* 1992), the failure to detect the disease could be linked to the weak national surveillance system.

Inability of the field veterinary staff to recognise the clinical, pathological and epidemiological features of the disease is yet another challenge as far as reporting of RVF occurrence is concerned. For example, when confronted with a disease that involves abortion during IEP, RVF is not included on

TABLE 1: Distribution of Rift Valley fever amongst cattle in Zambia.

District	Results of a seroepidemiological study in nine districts					
	Number of herds tested	Number positive	% herds positive	Number of cattle tested	Number of cattle positive	% cattle positive
Kasama	1	1	100	30	1	3.3
Mansa (d)	1	1	100	198	25	12.1
Chipata	3	2	66.7	162	2	1.2
Chingola	6	3	50	202	11	5.4
Solwezi (d)	2	2	100	181	25	13.8
Kabwe (d)	6	6	100	215	24	11.2
Lusaka (d/fp)	1	1	100	15	3	20
Mongu (fp)	6	6	100	206	47	22.8
Choma (d)	6	5	83.3	212	10	4.7
Total	**32**	**27**	**88.9 av**	**1421**	**147**	**10.5 av**

Source: Adopted from Samui, K.L., Inoue, S., Mweene, A.S., Nambota, A.M., Mlangwa, J.E., Chilonda, P. *et al.*, 1997, 'Distribution of Rift Valley fever among cattle in Zambia', *Japanese Journal of Medical Science & Biology* 50, 73–77. PMid:9559442
av, average; d, dambos; fp, flood plain.

the list of differential diagnosis. It is worthwhile to mention here that in Zimbabwe, RVF-associated abortions were found in cattle over a period of 7 inter-epizootic years (1971–1977) and the temporal pattern suggested a possible annual emergence of infected mosquitoes (Swanepoel 1981). This report shows clearly why it important to include RVF in the list of differential diagnosis especially when specimens are collected from cattle that have aborted. However, the diagnosis of RVF during IEP is further undermined by a shortage of RVF reagents which comes as a result of lack of planning or funding. It is worth mentioning here that during IEP, awareness and preparedness tend to decrease drastically as limited resources required for surveillance activities are redirected to other areas.

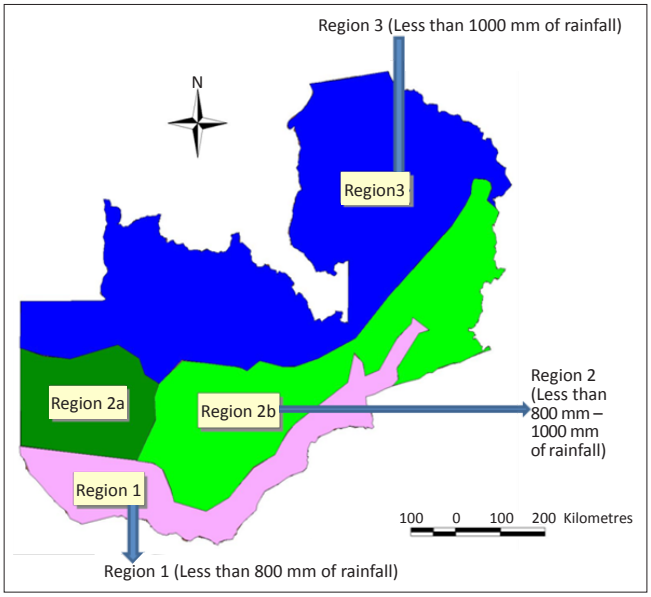
Risk factors associated with Rift Valley fever

There are several factors associated with the occurrence of RVF. These includes climatic conditions (rainfall, temperature, cloud cover), geographical features (dambos, flood plains), vegetation cover, livestock trade (both local and international) and human activities (such as building of dams, irrigation schemes).

Rainfall is one of the determinants of RFV outbreaks and this has been analysed in relation to the RVF epizootics in Kenya (Anyamba *et al.* 2009, 2010; Davies *et al*. 1985; Richards *et al.* 2010). Zambia receives a good amount of rainfall annually and the rainfall pattern is divided into three agro-ecological zones namely region I, II and III (Figure 3). Region I, the driest, is most prone to drought and receives less than 800 mm of rain annually. This region includes the Zambezi and Luangwa valleys. Region II covers the central part of Zambia extending from the east through to the west. It receives rainfall of between 800 mm and 1000 mm. Region III covers the northern part of the country and receives more than 1000 mm of rainfall in a season. Region II and III are more prone to flooding and have high incidences of malaria due to high vector activities. Therefore, the amount of rain tend to increase towards the north and decrease towards the south. The rainfall is considered to influence the onset of disease by producing a rising water table, to the point where seasonal flooding occurs, particularly in certain geomorphic formations known as 'dambos'.

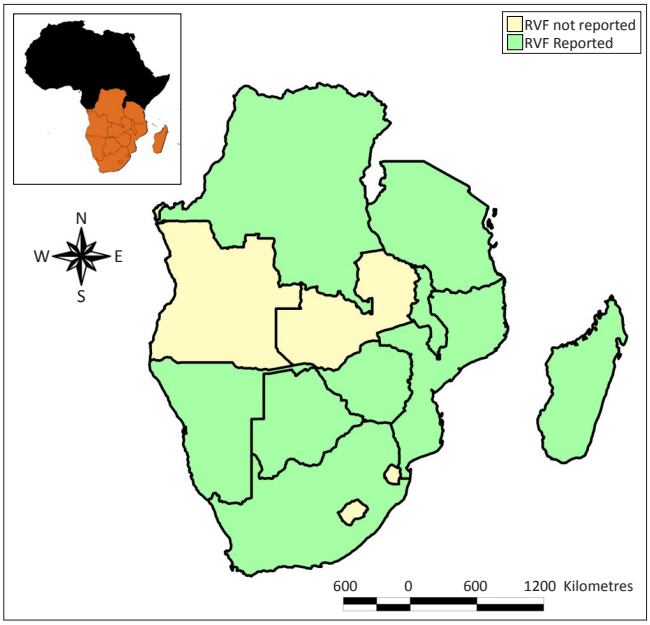
Flooding of the dambos results in the emergence of abundant numbers of floodwater *Aedes* sp., in particular *Aedes mcintoshi* (Linthicum *et al*. 1984). These transovarially infected mosquitoes are responsible for initiating epizootics of RVF, which then recruit other vectors for its propagation (Linthicum *et al*. 1985). It should be noted that the flooded dambos are the most favoured breeding sites for a variety of mosquito species that are capable of transmitting RVF (Davies & Highton 1980). Above all the humid conditions and cloud cover that exist during prolonged rainy periods allow a greater proportion of the adult *Aedes* population to survive through more feeding-oviposition cycles than in the hot, dry conditions usually prevailing in these areas (Davies *et al*. 1985)

Vegetation changes, due to a change in climatic conditions, has an effect on mosquito habitats. For instance, in the rainy season the proliferation of vegetation and increase in vegetation biomass favours the increase in population of mosquito species that are capable of transmitting diseases to livestock and humans. The dry season does not favour vegetation proliferation and hence there are fewer mosquito-borne diseases. In Zambia, a sentinel herd study was carried out in 1982–1986 to determine whether annual RVF virus activity occurred and was associated with seasonal rains. The results showed that a low level RVF virus activity of



Region I, cover the Zambezi and Luangwa valleys (less than 800 mm); Region II, Covers the central, western and eastern parts of the Country (800 mm – 1000 mm); Region III, covers the northern parts of the country (above 1000 mm).

FIGURE 3: Map of Zambia showing the three agro-ecological zones.



Countries which reported Rift Valley fever are indicated green and those that have not reported are indicated yellow.
RVF, Rift Valley fever.

FIGURE 4: Map showing the occurrence of Rift Valley fever in the Southern Africa Development Community region in the period 2005–2010.

3% – 8% did occur in each of the years. However, in 1985–1986, more than 20% of the animals seroconverted and this greater activity was associated with vegetation changes (Davies *et al*. 1992). The vegetation change was detected by remote sensing satellite imagery.

Livestock trade has previously been associated with the introduction of RVF in news areas (Centre for Disease Control and Prevention 2000; Madani *et al*. 2003). There is a lot of livestock trade between Zambia and her neighbours, which means that the introduction and spread of new diseases from neighbouring countries is high. OIE disease occurrence data on RVF for the period 2005–2010 in the SADC region showed that most countries that share a common border with Zambia had reported at least one occurrence of the disease during the period under review (Figure 4 OIE, World Animal Health Information Database [WAHID] Interface). Due to the absence of natural physical barriers between Zambia and most of her neighbours and given that the conditions which predispose to RVF activity do occur on a regional level, there is a high probability that RVFV could be circulating in Zambia.

Conclusion

This review demonstrates that RVFV is a threat to Zambia as the environmental risk factors conducive for its propagation are widely distributed in most livestock producing areas.

Despite the threat posed by RVF in Zambia, little research has so far been done. Most studies documented so far were limited to the high risk areas and only conducted during RVF outbreaks. Little is known about RVF virus activities during IEP both in the high risk and low risk areas. Currently, there is no information regarding the different types of RVF virus strains found in Zambia. Nothing is known of their virulence, pathogenicity or distribution in the different ecological zones of the country. The current prevailing hypothesis is that RVF virus is maintained in the eggs of *Aedes* mosquitoes which are seasonal floodwater breeders (Davies *et al*. 1985). However, different *Aedes* spp. have been implicated in the transmission of RVFV in different regions of Africa. For example, *Ae. ochraceus, Ae. vexans arabiensis* and *Ae. dalzieli* are known vectors of RVFV in West Africa (Fontenille *et al. 1998*) where as *Ae. mcintoshi/ circumluteolus* are known vector of RVFV in East Africa (Huang 1985). In Zambia, the potential mosquito vector species that might be involved in the enzootic or epizootic cycles has never been documented. Baseline data regarding their distribution and ecology is missing. The role of these mosquitoes in the maintanance of RVFV is not well understood. Human infection through direct contact with aborted foetuses, meat and other animal byproducts during RVF outbreaks (LaBeaud *et al*. 2008), the specifics of what types of animal exposure are most risky, have not yet been elucidated. Although human death due RVF was reported in the endemic areas of Zambia (Watts *et al*. 1984), no studies have been done to determine whether RVF transmission to human occurs during IEP. Lastly, the current RVF early warning system needs to be improved by including spatial and population parameters so as to achieve higher precision and confidence.

Therefore, in order to control RVF in the endemic and non-endemic areas of Zambia, future research should aim at addressing the above mentioned gaps. The data generated from such research will help veterinary, health policy makers, planners and other stakeholders in prioritising, designing and implementing cost effective and sustainable RVF control programs.

Acknowledgements

This work has been supported by Wellcome Trust Grant WT087546MA to the Southern African Centre for Infectious Disease Surveillance (www.sacids.org) and its collaborating UK partner institutions, namely the London School of Hygiene and Tropical Medicine (http://www.lshtm.ac.uk), the Royal Veterinary College (http://www.rvc.ac.uk) and the London International Development Centre (http://www.lidc.org.uk).

Competing interests

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

References

Anyamba, A., Linthicum, K.J., Small, J., Britch, S.C., Pak, E., De La Rocque, S. *et al*., n.d., 'Prediction, assessment of the Rift Valley fever activity in East and Southern Africa 2006-2008 and possible vector control strategies', *American Journal Tropical Medicine and Hygiene* 83 (2 suppl.), 43–51.

Anyamba, A., Chretien, J.P., Small, J., Tucker, C.J., Formenty, P.B., Richardson, J.H. *et al*., 2009, 'Prediction of a Rift Valley fever outbreak', *Proceedings of the National Academy of Science U S A* 106, 955–959. http://dx.doi.org/10.1073/pnas.0806490106, PMid:19144928

Centers for Disease Control and Prevention, 2007, 'Rift Valley fever Outbreak – Kenya, November 2006 – January 2007', *MMWR Morbidity and Mortality Weekly Report* 56, 73–76. PMid:17268404

Centers for Disease Control and Prevention, 2000, 'Outbreak of Rift valley fever – Saudi Arabia, August – October, 2000', *MMWR Morbidity and Mortality Weekly Report* 49, 905–908. PMid:11043643

Centers for Disease Control and Prevention, 2000, 'Outbreak of Rift valley fever – Yemen, August – October 2000', *MMWR Morbidity and Mortality Weekly Report* 49, 1065–1066. PMid:11186611

Coetzer, J.A., 1982, 'The pathology of Rift Valley fever. II. Lesions occurring in field cases in adult cattle, calves and aborted foetuses', *Onderstepoort Journal of Veterinary Research* 49, 11–17. PMid:7122060

Coetzer, J.A., 1977, 'The pathology of Rift Valley fever. I. Lesions occurring in natural cases in new-born lambs', *Onderstepoort Journal of Veterinary Research* 44, 205–211. PMid:613292

Collett, M.S., 1986, 'Messenger RNA of the M segment RNA of Rift Valley fever virus', *Virology* 151, 151–156. http://dx.doi.org/10.1016/0042-6822(86)90114-5

Collett, M.S., Purchio, A.F., Keegan, K., Frazier, S., Hays, W. & Anderson, D.K., 1985, 'Complete nucleotide sequence of the M RNA segment of Rift Valley fever virus', *Virology* 144, 228–245. http://dx.doi.org/10.1016/0042-6822(85)90320-4

Daubney, R., Hudson, J.R. & Garnham, P.C., 1931, 'Enzootic hepatitis or Rift Valley fever. An undescribed virus disease of sheep, cattle and man from East Africa', *Journal of Pathology and Bacteriology* 34, 545–579. http://dx.doi.org/10.1002/path.1700340418

Davies, F.G., Kilelu, E., Linthicum, K.J. & Pegram, R.G., 1992, 'Patterns of Rift Valley Fever activities in Zambia', *Epidemiology and Infection* 108, 185–191. http://dx.doi.org/10.1017/S0950268800049633

Davies, F.G., Linthicum, K.J. & James, A.D., 1985, 'Rainfall and epizootic Rift Valley fever', *Bull World Health Organ* 63, 941–943. PMid:3879206

Davies, F.G. & Highton, R.B., 1980, 'Possible vectors for Rift Valley fever in Kenya', *Transactions of the Royal Society of Tropical Medicine and Hygiene* 74, 815–825. http://dx.doi.org/10.1016/0035-9203(80)90213-8

Department of Veterinary and Tsetse Control Services, 1975–1989, Annual reports of the Department of Veterinary and Tsetse Control Services, Government printers, Lusaka, Zambia.

Fontenille, D., Traore-Lamizana, M., Diallo, M., Thonnon, J., Digoutte, J.P. & Zeller, H.G., 1998, 'New vectors of Rift Valley Fever in West Africa', *Emerging Infectious Diseases* 4(2), 289–293. http://dx.doi.org/10.3201/eid0402.980218, PMid:9621201

Ghirotti, M., Semproni, G., De Meneghi, D., Mungaba, F.N., Nannini, D., Calzetta, G. *et al*., 1991, 'Sero-prevalences of selected cattle diseases in the Kafue flats of Zambia', *Veterinary Research Communication* 15, 25–36. http://dx.doi.org/10.1007/BF00497787, PMid:1646515

Giorgi, C., Accardi, L., Nicoletti, L., Gro, M.C., Takehara, K. & Hilditch, C., 1991, 'Sequences and coding strategies of the S RNAs of Toscana and Rift Valley fever viruses compared to those of Punta Toro, Sicilian sandfly fever, and Uukuniemi viruses', *Virology* 180, 738–753. http://dx.doi.org/10.1016/0042-6822(91)90087-R

Huang, Y.-M., 1985, 'A new African species of *Aedes*', *Mosq Syt* 17, 108–117.

Hussein, N.A., Chizyuka, R.Z., Kasiazek, T.G., Scott, R. & Boulos, B.A.M., 1987, 'Epizootic of Rift Valley fever in Zambia, 1985', *Veterinary Record* 121, 111. http://dx.doi.org/10.1136/vr.121.5.111, PMid:2889285

LaBeaud, A.D., Muchiri, E.M., Ndzovu, M., Mwanje, M.T., Muiruri, S. & Peters, C.J., 2008, 'Interepidemic Rift Valley fever virus seropositivity, northeastern Kenya', *Emerging Infectious Diseases* 14, 1240–1246. http://dx.doi.org/10.3201/eid1408.080082, PMid:18680647

Linthicum, K.J., Davies, F.G. & Kairo, D.A., 1985, 'Rift Valley fever virus (family Bunyaviridae, genus Phlebovirus) isolations from Diptera collected during inter-epizootic period in Kenya', *Journal Hygiene (London)* 95, 197–209. http://dx.doi.org/10.1017/S0022172400062434

Linthicum, K.J., Davies, F.G., Bailey, C.L. & Kairo, A., 1984, 'Mosquito species encountered in a flooded grassland dambo in Kenya', *Mosquito News* 44, 228–234.

Madani, T.A., Al-Mazrou, Y.Y. & Al-jeffri, M.H., 2003, 'Rift Valley fever epidemic in Saudi Arabia: epidemiological, clinical, and laboratory characteristics', *Clinical Infectious Disiease* 37, 1084–1092. http://dx.doi.org/10.1086/378747, PMid:14523773

Martin, V., Cheavalier, V., Ceccato, P., Anyamba, A., De Simone, L. & Lubroth, J., 2008, 'The Impact of climate change on the epidemiology and control of Rift Valley fever', *Scientific and Technical Review (OIE)* 27, 413–426. PMid:18819669

Meegan, J.M., 1981, 'Rift Valley fever in Egypt: An overview of the epizootics in 1977 and 1978', *Contribution to Epidemiology and Biostatistics* 3, 100–113.

Morvan, J., Rollin, P.E., Laventure, S., Rakotoarivony, I. & Roux, J., 1992, 'Rift Valley fever epizootic in the central highlands of Madagascar', *Research Virology* 143, 407–415. http://dx.doi.org/10.1016/S0923-2516(06)80134-2

Muller, R., Poch, O., Delarue, M., Bishop, D.H.L. & Bouloy, M., 1994, 'Rift Valley fever virus L segment: Correction of the sequence and possible functional role of newly identified regions conserved in RNA-dependent polymerases', *Journal of Gen Virology* 75, 1345–1352. http://dx.doi.org/10.1099/0022-1317-75-6-1345, PMid:7515937

OIE, n.d., '*World Animal Health Information Database (WAHID) Interface'*, viewed n.d., from http://web.oie.int/wahis/public.php?page=home

Pepin, M., Bouloy, M., Bird, H.B., Kemp, A. & Paweska, J., 2010, 'Rift Valley fever virus (*Bunyaviridae: Phlebovirus*): An update on pathogenesis, molecular epidemiology, vectors, diagnosis and prevention', *Veterinary Research* 41, 61. http://dx.doi.org/10.1051/vetres/2010033, PMid:21188836

Rich, K. & Wanyioke, F., 2010, 'An assessment of the regional and national socio-economic impacts of the 2007 Rift Valley fever outbreak in Kenya', *American Journal of Tropical Medicine and Hygiene* 83, 52–57. http://dx.doi.org/10.4269/ajtmh.2010.09-0291, PMid:20682906

Richards, E.E., Masuoka, P., Brett-Major, D., Smith, M., Klein, T.A., Kim, H.C. *et al*., 2010, 'The relationship between mosquito abundance and rice field density in the Republic of Korea', *International Journal of Health Geographic* 9, 32. http://dx.doi.org/10.1186/1476-072X-9-32, PMid:20573242

Samui, K.L., Inoue, S., Mweene, A.S., Nambota, A.M., Mlangwa, J.E., Chilonda, P. *et al*., 1997, 'Distribution of Rift Valley fever among cattle in Zambia', *Japanese Journal of Medical Science & Biology* 50, 73–77. PMid:9559442

Schmaljohn, C. & Hooper, J.W., 2001, '*Bunyaviridae*: The virus and their replication', in D.M. Knipe, P.M. Howley, D.E. Griffin *et al*. (eds.), *Fields Virology,* 4th edn., pp. 1581–1602, Lippincott, Williams & Wilkins, Philadelphia, PA.

Shoemaker, T., Boulianne, C., Vincent, M.J., Pezzanite, L., Al-Qahtani, M.M., Al-Mazrou, Y. *et al*., 2002, 'Genetic analysis of viruses associated with emergence of Rift Valley fever in Saudi Arabia and Yemen, 2000–2001', *Emerging Infectious Diseases* 8, 1415–1420. PMid:12498657

Sossah, C., 2009, 'OIE regional seminar on RE-emergence of RVF in southern Africa', How to better predict and respond, Sossah, South Africa.

Swanepoel, R.C.J., 1994, 'Rift Valley fever', in J.A.W. Coetzer, G.R. Thompson, R.D. Tustin (eds.), *Infectious diseases of Livestock with special reference to southern Africa*, pp. 688–717, Oxford University Press, Cape Town.

Turell, M.J., Bailey, C.L. & Rossi, C.A., 1984, 'Increased mosquito feeding on Rift Valley fever virus-infected lambs', *American Journal of Tropical Medicine and Hygiene* 33, 1232–1238. PMid:6150656

Van Velden, D.J., Meyer, J.D., Olivier, J., Gear, J.H. & McIntosh, B., 1977, 'Rift Valley fever affecting humans in South Africa a clinicopathological study', *South African Medical Journal* 51, 867–871. PMid:561445

Watts, T.E., Tembo, G. & Ngandu, N.H., 1984, 'Rift Valley fever in Chisamba, Zambia', *Medical Journal of Zambia* 18, 10–11.

The epidemiology and socio-economic impact of Rift Valley fever epidemics in Tanzania: A review

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How to cite this abstract:
Sindato, C., Karimuribo, E. & Mboera, E.G., 2012, 'The epidemiology and socio-economic impact of Rift Valley fever epidemics in Tanzania: A review', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #467, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.467

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

A review was conducted to provide comprehensive update on Rift Valley fever (RVF) in Tanzania, with particular attention devoted to trend of occurrence, epidemiological factors, socio-economic impact and measures which were applied to its control. Information presented in this paper was obtained through extensive literature review. Rift Valley fever was documented for the first time in Tanzania in 1977. This was followed by epidemics in 1997 and 2007. Contrary to the latest epidemic in 2007 sporadic cases of RVF during the previous epidemics were confined to mainly livestock and mostly affecting northern parts of Tanzania. The latest disease epidemic expanded to cover wider areas (mostly northern and central zones) of the country involving both human and domestic ruminants. During the latest disease outbreak 52.4% (n = 21) of regions in Tanzania mainland were affected and majority (72.7, n = 11) of the regions had concurrent infections in human and animals.

Phylogenetic comparison of nucleotide and amimo acid sequences revealed different virus strains between Kenya and Tanzania.

Epidemiological factors that were considered responsible for the previous RVF epidemics in Tanzania included farming systems, climatic factors, vector activities and presence of large population of ruminant species, animal movements and food consumption habits. Majority of the RVF positive cases in the latest epidemic were livestock under pastoral and agro-pastoral farming systems.

The disease caused serious effects on rural people's food security and household nutrition and on direct and indirect losses to livestock producers in the country. Psycho-social distress that communities went through was enormous, which involved the thinking about the loss of their family members and/or relatives, their livestock and crop production. Socially, the status of most livestock producers was eroded in their communities.

Cessation of lucrative trade in ruminants resulted in serious economic losses to the populations who were totally dependent upon this income. Livestock internal market flows drastically dropped by 37% during latest epidemic. Rift Valley fever epidemics had dramatic impact of RVF outbreak on the international animal trade in which there was a 54% decline in exports equivalent to loss of $352 750.00. The estimate of loss as a result of deaths for cattle was $4 243 250.00 whereas that of goats and sheep was $2 202 467.00.

Steps taken to combat epidemics included restriction of animal movements, ban of the slaughter of cattle and vaccination of livestock and health education.

From past epidemics we have learnt that each subsequent outbreak had expanded to cover wider areas of the country. The disease had dramatic socio-economic impacts both at community and nation at large. The main challenges related to the control of RVF outbreaks included lack of preparedness plan for RVF, poor coordination and information transmission, limited facilities and manpower for RVF outbreak intervention. Control of the 2007 RVF epidemic was largely the result of animal and human health agencies working in an integrated manner.

Epidemiological aspects of bovine trypanosomosis in an endemic focus of eastern Zambia: The role of trypanosome strain variability in disease pattern

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How to cite this abstract:
Masumu, J., Tshilenge, G. & Mbao, V., 2012, 'Epidemiological aspects of bovine trypanosomosis in an endemic focus of eastern Zambia: The role of trypanosome strain variability in disease pattern', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #468, 5 pages. http://dx.doi.org/10.4102/ojvr.v79i2.468

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Bovine trypanosomosis displays various epidemiological aspects in various areas. In some instances the disease has a high prevalence in animals with high impact on production whereas in other cases the disease has a low impact on production despite a high level of infection in animals. In addition epidemiological changes are frequently observed in various areas and are related to many factors including the vectors, the host, the parasites, the environment as well as the livestock management. However the implication of these factors in these changes is not fully elucidated. In eastern Zambia, factors predicting the establishment of severe infection in cattle are all present. However trypanosomosis occurring in cattle in this area has a low impact on livestock production. Several studies on the characterisation of trypanosome strains circulating in domestic and wild animals have been conducted in order to clarify the epidemiology of this disease in this area. These studies aimed at evaluating genetic and biological characteristics of these strains including their virulence profiles, their transmissibility by tsetse flies, their resistance to drugs and interference between different strains. In this review these findings are analysed in order to elucidate the implication of trypanosome strain variability in the distribution and the expression of this disease in the study area. The evolutionary trends of the situation occurring in this study area are also explained. Use of these findings is the context of disease control in the study area is further discussed.

Bovine trypanosomosis in eastern Zambia: Background

Bovine trypanosomosis is endemic in eastern province of Zambia. Previous epidemiological studies using parasitological, serological or molecular techniques showed that the prevalence of the disease varies in different districts (Machila, Sinyangwe & Mubanga 2001; Sinyangwe, Delespaux & Brandt 2004; Simukoko, Marcotty & Phiri 2007). The same variation is observed within a particular district where a high prevalence of the disease is observed in some villages compared to other. In some cases no infection has been detected in cattle kept in some villages. In 1996, trypanosome infections were found in cattle in only 74% of sampling sites with a prevalence of infection varying between villages from 0% to 64% (Sinyangwe *et al.* 2004). Such variations are related to many factors including the habitat fragmentation of tsetse flies that affects tsetse density in various areas (Ducheyne, Mweempwa & De Pus 2009). For example the decrease prevalence of the infection in cattle from 1998 to 2003 in Petauke where tsetse habitat has widely been fragmented supports this observation.

The temporal trends of the infection in cattle revealed no (Sinyangwe *et al.* 2004) or little (Simukoko *et al.* 2007) variations between seasons although higher prevalence seems to be observed during the rainy season (Simukoko *et al.* 2007) when tsetse flies are more abundant (Van den Bossche & De Deken 2002). Such variations influence drug management and, although farmers treat their animals throughout the year, most drugs are selectively administered during this period (Van den Bossche, Doran & Connor 2000).

In this area, the same selection approach is used by farmers in administrating trypanocides drugs in animals. In most cases oxen and cows are more treated (Van den Bossche *et al.* 2000) since these animals categories, especially oxen are more infected with trypanosomes (Simukoko *et al.* 2007). Although this selective treatment resulted in the reduction (1.5 treatments per year) of drugs administered to animals (Van den Bossche *et al.* 2000) it could not limit the development and spread of drug resistance in the area. From 1996 (Sinyangwe *et al.* 2004) to 2003 (Delespaux, Dinka & Masumu 2008), drug resistance to diminazene aceturate increased fivefold although drug use didn't change during this period of time (Van den Bossche *et al.* 2000). Despite a high proportion of resistant strains in the area farmers continue using trypanocides which effect needs to be evaluated.

Trypanosoma congolense remains the most prevalent trypanosome species affecting cattle in this area with more than 95% of the infections (Sinyangwe *et al.* 2004; Simukoko *et al.* 2007). In eastern province only the Savannah type has been previously characterised in livestock (Masumu, Marcotty & Geysen 2006a). This type was previously shown to be highly virulent in cattle (Bengaly, Sidibe & Ganaba 2002). Furthermore it is efficiently transmitted by *Glossina morsitans morsitans*, the main vector of trypanosomes in this area. Bloodmeal analyses revealed that cattle provide 75% of meals to *G. m. morsitans* in this area (Van den Bossche & Staak 1997) and thus are highly exposed to infections with these highly virulent *T. congolense* Savannah strains circulating in this area. However the cattle breed (Angoni) reared on the plateau of eastern province are not trypanotolerant. Taken together, these factors, including a high level of resistance to trypanocides in trypanosome strains circulating in cattle and the restricted number of treatment per year, favour for the development of an epidemic situation in cattle on the plateau of eastern province. However economical surveys revealed that the disease has a low impact on livestock production (Doran 2000).

The isolation and characterisation of trypanosome isolates from cattle in this endemic area revealed sound features that explain the epidemiology of bovine trypanosomosis in this particular area. In this review trypanosome characteristics observed in this area will be analysed for their implication in the epidemiology of bovine trypanosomosis on the plateau of eastern province in Zambia.

Biological characteristics of trypanosome strains circulating in eastern province

Trypanosomes can only influence the distribution and expression of the disease in various areas and different hosts only if they are genetically and phenotypically different. In order to test this, *T. congolense* circulating in the domestic and wild animals in eastern province were characterised at genetic level (Masumu, Geysen & Vansnick 2006b; Masumu, Geysen & Van den Bossche 2009a) as well as for biological characters including the virulence profile (Masumu *et al.* 2006a; Van den Bossche, Chitanga & Masumu 2011), the transmissibility by tsetse flies (Masumu, Marcotty & Ndeledje 2006c; Masumu, Akoda & Van den Bossche 2010) and the possible existence of cross-protection between different strains (Masumu *et al.*, 2009). It appears that many strains of *T. congolense* circulate in the eastern province (Masumu *et al.* 2006b; 2009a). Very few isolates co-circulate in different areas indicating a more restricted distribution. In trypanosomes circulating in domestic animals most of the strains were of low virulence (Masumu *et al.* 2006a). Only 20% of them were highly virulent. On the other hand most of the trypanosomes circulating in wild animals were highly virulent (Van den Bossche *et al.* 2011) highlighting the trypanotolerant trait of these animals. The presence of high prevalence of virulent strains in wild animals is also a clear indication of these animals being reservoirs of these strains in eastern province. In domestic cycle where trypanosomes circulate only in domestic animals, geographical distribution of these strains indicated that high virulent trypanosome strains are present in limited villages (4 out of 11) and different virulent profiles were observed in various areas suggesting an uneven distribution of those virulent trypanosome strains (Masumu *et al.* 2006a). Whether the circulation of low virulent trypanosome strains in domestic animals could protect them from the adverse affect of the high virulent strains was also assessed (Masumu *et al.* 2009b). Indeed there is a clear indication of the existence of cross-protection between trypanosomes of high and low virulence but the degree of protection varies in different combinations of strains. This finding suggests that the effect of virulent trypanosome strains may vary depending on the combination of the high and low virulent strains in a particular area. Only in areas where most virulent strains do not cross-react with the majority of low virulent strains will the disease have high impact on animal health and thus livestock production. In areas where high level of cross-protection is observed between strains of high and low virulence the effects of such virulent strains will be minimal. The epidemiological importance of the virulent strains was further stress by the fact they were efficiently transmitted by tsetse flies compared to the strains of low virulence (Masumu *et al.* 2006c).

Involvement of the trypanosome strain variability in the geographic distribution of bovine trypanosomosis in eastern province

The geographical distribution of trypanosomosis depend on multiple factors including the abundance and infection rate of tsetse flies, the livestock management, the level of drug resistance and drug use in a particular area and the degree of livestock to resist to tsetse fly bites as well as to trypanosome strains circulating in the area. In the context of eastern province, significant variations are observed in the distribution of tsetse in various areas. In most cases high prevalence of the disease is observed in areas highly infested by tsetse flies. Within the same geographic area such variations are related to the abundance of tsetse flies throughout the year. In eastern province it appears that tsetse flies are more abundant during the rainy season (Van den Bossche & De Deken, 2002) explaining the high prevalence of the disease previously observed during this period (Simukoko *et al.* 2007). Such variations may also be explained by the variations observed in the strains of *T. congolense* circulating in these areas with a high prevalence of the disease likely being observed in areas where most trypanosome strains are efficiently transmitted by tsetse flies. Although highly virulent strains are efficiently transmitted that low virulent ones, further analyses revealed that during the chronic phase of infection the transmission of low virulent strains increases (Masumu *et al.* 2010). This indicates that high and low virulent strains are all transmitted by tsetse flies in this area since most low virulent strains induced chronic infections in cattle.

The distribution of the disease can also be influenced by the level of drug resistance to trypanocides in strains circulating in various areas. It can be assumed that high prevalence of infection will be observed in areas where the level of drug resistance is also high. This can partially be explained by the persistence of infection after treatment with trypanocides in animals infected with resistant strains. In addition although there is not evident correlation between resistance and transmission to tsetse flies (personal observation) the level of drug resistance in individual trypanosome strains may influence the infection rate in individual tsetse flies in case of persistence of drugs in tsetse after feeding on treated animals prior infection through an infected bloodmeal. In eastern province high prevalence was observed in areas where the level of drug resistance was also high (Sinyangwe *et al.* 2004).

Involvement of the trypanosome strain variability in the expression of bovine trypanosomosis in eastern province

The expression of trypanosomosis in various areas varies considerably (Van den Bossche 2001). In most cases severe infections are observed when animals are kept near game areas whereas a rather mild infection occurs in areas where wild animals are rather absent. In eastern province bovine trypanosomosis is rather mild on the plateau but when livestock are kept near the Luangwa valley where wild animals are abundant they suffer from severe infections. This was previously suggested to result from the presence of heterologous challenge in wild areas as a consequence of the circulation of diverse strains in these areas (Van den Bossche 2001). Our findings revealed that even in areas where wild animals are absent several trypanosome strains circulate in domestic animals suggesting that the number of trypanosome strains is not important for the expression of the disease in a given area (Masumu *et al.* 2009a). Instead from the findings on virulence and cross-protection obtained in eastern province it appears that the expression of the disease in various areas is largely dependent on the virulence of trypanosome strains circulating in each particular area with severe disease being observed in areas where virulent strains are more prevalent. Our findings on high prevalence of virulent trypanosome strains in the wild environment compared to the plateau where wild animals are absent and where most strains are of low virulence support this observation (Van den Bossche 2001). However as indicated previously high level of interference between high and low virulent strains may attenuate the effects of the disease in animals previously infected with low virulent strains even after challenge with virulent strains (Masumu, Marcotty & Geerts 2009b). The low impact the disease has on the plateau of eastern province may be explained by the circulation in animals of a high prevalence of low virulent trypanosome strains and the existence of a high level of cross-protection between the few virulent strains and the high prevalent low virulent strains. On the other hand the high prevalence of virulent strains in the wild environment explains the severity of the disease observed in these areas.

Trypanosome strain variability and the development of drug resistance in eastern province

The rapid development of drug resistance in eastern province arise many questions. On the plateau of eastern province a fivefold increase of drug resistance was observed from 1996 (Sinyangwe *et al.* 2004) to 2003 (Delespaux *et al.* 2008). In the absence of factors underlying the development of drug resistance in the area (Van den Bossche *et al.* 2000), such increase may be explained by the circulation of resistant strains between different areas. However our findings on genetic characterisation on the plateau indicate that the circulation of trypanosome strains is very limited (Masumu *et al.* 2009a). Even in geographically close villages similar strains were rarely observed. Indeed the circulation of trypanosome strains in various areas can be facilitated by the movement of the infected host or infected vectors. In this area livestock movement is very restricted. In addition tsetse flies do not move freely in various areas due to the fragmentation of their habitats. Variations in the distribution of tsetse flies are more linked to the distribution of cattle (Van den Bossche & Staak 1997). These two factors are in favour of a more restriction of trypanosome strains circulating in various areas. Such restriction cannot facilitate the circulation of resistant trypanosome strains. Instead the development of drug resistance seems to be induced locally. Although genetic exchange was only reported in *Trypanosoma brucei* (Jenni, Marti & Schweizer 1986) not *T. congolense*, it may be assumed that the spread of drug resistance in eastern province is related to genetic exchange (Delespaux, Dinka & Masumu 2008) between resistant and susceptible trypanosome strains. Indeed resistant and susceptible trypanosome strains do circulate in animals belonging to the same village (Masumu *et al.* 2006a). Since two different strains can infect an individual animal, and tsetse flies can be infected more than once, the presence of two different strains in the same fly will favour the exchange of genetic material. Further studies are needed to clarify the role of genetic exchange in the spread of drug resistance.

From the past to the future

The high prevalence of low virulent trypanosome strains and the presence of high level of interference between strains of high and low virulence in eastern province, an area where tsetse flies take their blood meal principally from a susceptible animal i.e. cattle (Van den Bossche & Staak 1997), is a perfect illustration of an endemic situation where the vector (tsetse flies), the parasite (trypanosomes) and the host (cattle) live in harmony. The current situation results from the fragmentation of tsetse habitat and the elimination of wild animals. Indeed many decades from now wild animals were prevalent in this area. Consequently the virulence profile of trypanosome strains was similar to that occurring in Luangwa valley where the trypanotolerant trait of wild animals selects against trypanosomes of low virulence. However the elimination of wild animals on this plateau

led to a situation where tsetse depends on livestock for their survival (Van den Bossche & Staak 1997) and trypanosome circulate only in susceptible animals. Since the presence of virulent strains induces severe infections in these susceptible animals, the later are either treated or die from their infection. This results in the elimination of these virulent strains and the subsequently high prevalence of strains that affect animal health to a lesser degree. Only virulent strains that can interfere with strains of low virulence can persist in the susceptible host animal thus explaining the persistence of 20% of the virulent strains in this area (Masumu *et al.* 2006a).

The situation prevailing on the plateau of eastern province indicates clearly that bovine trypanosomosis is endemic in this area. Although the prevalence of the disease varies largely in different areas (Machila *et al.* 2001; Sinyangwe *et al.* 2004; Simukoko *et al.* 2007), further changes are still to occur in the future in various areas. For example, Petauke district that had a high prevalence of the disease and a high prevalence of drug resistance in 1990s (Sinyangwe *et al.* 2004) is no longer the major focus of bovine trypanosomosis in eastern province. The high level of fragmentation resulting from the increased surface of cultivation of crops induced a spectacular reduction of tsetse fly abundance and subsequently a low prevalence in animals (Simukoko *et al.* 2007). On the other hand Katete district remains an area of concern since the level of infection in animals is still high (Simukoko *et al.* 2007) and more important the increase of drug resistance is alarming (Delespaux *et al.* 2008).

It's obvious that this situation is far from being stable in the near future. Indeed the presence of a wild environment in Luangwa valley where about 14% of wild animals are infected with trypanosomes (Anderson, Mubanga & Fevre 2011) constitutes a serious threat to livestock production in eastern province. Although habitat fragmentation renders difficult to tsetse flies to move towards districts like Katete, Petauke, other district like Msoro that is located near the game is constantly at high risk of trypanosomosis. In addition this district may constitute a buffer zone between the high risk area of the valley and the plateau of eastern province. In this district the high challenge of tsetse flies and the tranfer of highly virulent trypanosome strains from the wild environment will result in a high infection rate in livestock including pigs and small ruminants that are rather resistant to trypanosomes. This was demonstrated in a survey where all livestock species (cattle, pigs, sheep and goats) kept in Msoro district were highly infected (> 10%) with trypanosomes contrarily to the situation occurring on the plateau where most infections are restricted to cattle (Simukoko *et al.* 2007). Indeed in the absence of wild animals, the presence of such infections in livestock other than cattle is epidemiologically very important. First, in such areas of high risk of trypanosomosis farmers prefer exploiting small ruminants and pigs for their relative tolerance to *T. congolense*. Their relative number will exceed that of cattle. Further since these animals are trypanotolerant and rarely treated with trypanocides they will maintain trypanosomial infection for a long period of time and thus increase the risk of tsetse flies being infected with trypanosomes even virulent to cattle. Such risk will increase the severity of the disease in cattle in those particular areas explaining the habitual low production observed in cattle in such areas. Second this encroachment of people and livestock to the game area constitutes a new epidemiological situation where virulent trypanosome strains circulating in wild animals (Van den Bossche *et al.* 2011) will gradually be introduced into the livestock management area and finally reach the plateau of eastern Zambia. Whilst such movement of virulent trypanosome strains from the wild to the interface will be favoured by tsetse flies movement, the transfer of these strains from the interface to the plateau of eastern province will probably be occasioned by livestock movement. Since cattle movement is very restricted small ruminants and pigs will be the major factor for the circulation of trypanosomes between areas and the introduction of virulent trypanosome strains in various areas including the plateau where the situation is currently more endemic and rather stable. This will be facilitated for example by the commercial and social exchanges between families in the area that increases the movement of animals from one area to another. When infected with trypanosomes, pigs and small ruminants will favour the circulation of these virulent strains even in areas further away from the game areas. It would be advisable to conduct a follow up of trypanosome strains circulating on the plateau e.i. in Katete district where trypanosomes are more resistant but rather low virulent and at the interface e.i. in Msoro district where a high prevalence of virulent strains may be present whilst being less or no resistant to trypanocides.

Trypanosome-related factors and disease control strategies

Several control strategies have been used in eastern province for bovine trypanosomosis. The extent of drug resistance to either diminazene aceturate or isometamidium chloride indicates clearly that caution should be made in using these drugs to control trypansomosis in cattle. It is not well known how cattle withstand the infection in this particular area where drug use is minimal (Van den Bossche *et al.* 2000) and a high proportion of trypanosome strains are resistant to either or both drugs (Delespaux *et al.* 2008; Sinyangwe *et al.* 2004). Amongst the reasons is the circulation of a high prevalence of low virulent strains in livestock. Indeed infections induced by these strains have minimal effect on livestock production. In most cases low virulent strain can affect reproductive parameters but do not necessarily cause animal death. Like in theileriosis the presence of such infection can be beneficial in protecting animals against adverse effects of virulent strains.

In this area, farmers use production-oriented strategy where only sick animals are treated. Although this strategy could not reduce the development of drug resistance it reduces animal mortality (Van den Bossche *et al.* 2000). Our findings and the presence of high prevalence of resistant strains are in

favour of such control strategy. Not only treating sick animals also is economically acceptable by resource-limited farmers, it favours the establishment of premonition state in animals that are subsequently protected against adverse effects of virulent strains. It's possible that this strategy will remain the strategic option used by farmers in the future although it does not boost the reproduction of infected animals (Van den Bossche *et al*. 2000). Such control strategy cannot be applied in areas displaying a different epidemiological situation like in Msoro or the game areas of Luangwa valley where the trypanosome profiles are rather threatening for livestock.

Conclusion

Many factors can influence the epidemiology of bovine trypanosomosis in various areas. Very few studies have so far been deeply conducted for the understanding of the involvement of the parasite in the epidemiology of this disease. Indeed this can only be possible if sound information is yielded at genetic and biological levels. This implies the development of appropriate tools for genotypic and phenotypic characterisation. In the situation prevailing on the plateau of eastern province of Zambia where wild animals are not present and tsetse flies depends largely on a susceptible host, the outcome of the disease on livestock health and production was found to be correlated with the profiles of trypanosome strains circulating in animals. Disease control through parasite management leading to the establishment of premonition state in animals is thus encouraged and further supports the control strategy so far adopted by farmers. Similar features can likely be observed in areas displaying a similar epidemiological situation although cautious is needed to generalise these findings. Finally the approach adopted here to clarify the epidemiology of a particular disease in a specific epidemiological condition can be applied to any disease provided appropriate tools are available for such studies.

Acknowledgements

Competing Interest

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

References

Anderson, N.E., Mubanga, J., Fevre, E.M., Picozzi, K., Eisler, M.C., Thomas, R. *et al*., 2011, 'Characterisation of the wildlife reservoir community for human and animal trypanosomosis in the Luangwa Valley, Zambia', *PLoS Negl Trop Dis* 5, e1211. http://dx.doi.org/10.1371/journal.pntd.0001211

Bengaly, Z., Sidibe, I., Ganaba, R., Desquesnes, M., Boly, H. & Sawadogo, L., 2002, 'Comparative pathogenicity of three genetically distinct types of *Trypanosoma congolense* in cattle: Clinical observations and haematological changes', *Veterinary Parasitology* 108, 1–19. http://dx.doi.org/10.1016/S0304-4017(02)00164-4

Delespaux, V., Dinka, H., Masumu, J., Van den Bossche, P. & Geerts, S., 2008, 'Five-fold increase in *Trypanosoma congolense* isolates resistant to diminazene aceturate over a seven-year period in Eastern Zambia', *Drug Resistance Updates* 11, 205–209. http://dx.doi.org/10.1016/j.drup.2008.10.002

Doran, M., 2000, 'Socio-economics of trypanosomosis', RTTCP, publishing, *Bovine Trypanosomosis in Southern Africa, Harare* 3, 156.

Ducheyne, E., Mweempwa, C., De Pus, C., Vernieuwe, H., De Deken, R., Hendrickx, G. *et al*., 2009, 'The impact of habitat fragmentation on tsetse abundance on the plateau of eastern Zambia', *Preventive Veterinary Medecine* 91, 11–18. http://dx.doi.org/10.1016/j.prevetmed.2009.05.009

Jenni, L., Marti, S., Schweizer, J., Betschart, B., Lepage, R.W.F., Wells, J.M. *et al*., 1986, 'Hybrid formation between African trypanosomes during cyclical transmission', *Nature* 322, 173–175. http://dx.doi.org/10.1038/322173a0

Machila, N., Sinyangwe, L., Mubanga, J., Hopkins, J.S., Robinson, T. & Eisler, M.C., 2001, 'Antibody-ELISA seroprevalence of bovine trypanosomosis in the Eastern Province of Zambia', *Preventive Veterinary Medicine* 49, 249–257. http://dx.doi.org/10.1016/S0167-5877(01)00178-7

Masumu, J., Akoda, G. & Van den Bossche, P., 2010, 'Transmissibility, by *Glossina morsitans morsitans*, of *Trypanosoma congolense* strains during the acute and chronic phases of infection', *Acta Tropica* 113, 195–198. http://dx.doi.org/10.1016/j.actatropica.2009.09.002

Masumu, J., Geysen, D. & Van den Bossche, P., 2009a, 'Endemic type of animal trypanosomosis is not associated with lower genotypic variability of *Trypanosoma congolense* isolates circulating in livestock', *Research in Veterinary Science* 87, 265–269. http://dx.doi.org/10.1016/j.rvsc.2009.03.003

Masumu, J., Marcotty, T., Geerts, S., Vercruysse, J. & Van den Bossche, P., 2009b, 'Cross-protection between *Trypanosoma congolense* strains of low and high virulence', *Veterinary Parasitology* 163, 127–131. http://dx.doi.org/10.1016/j.vetpar.2009.04.006

Masumu, J., Marcotty, T., Geysen, D., Geerts, S., Vercruysse, J., Dorny, P., et al., 2006a, 'Comparison of the virulence of *Trypanosoma congolense* strains isolated from cattle in a trypanosomosis endemic area of eastern Zambia', *International Journal for Parasitology* 36, 497–501. http://dx.doi.org/10.1016/j.ijpara.2006.01.003

Masumu, J., Geysen, D., Vansnick, E., Geerts, S. & Van den Bossche, P., 2006b, 'A modified AFLP for *Trypanosoma congolense* isolate characterization', *Journal of Biotechnology* 125, 22–26. http://dx.doi.org/10.1016/j.jbiotec.2006.01.016

Masumu, J., Marcotty, T., Ndeledje, N., Kubi, C., Geerts, S., Vercruysse, J., Dorny, P. & Van den Bossche, P., 2006c, 'Comparison of the transmissibility of *Trypanosoma congolense* strains, isolated in a trypanosomosis endemic area of eastern Zambia, by *Glossina morsitans morsitans*', *Parasitology* 133, 331–334. http://dx.doi.org/10.1017/S0031182006000369

Simukoko, H., Marcotty, T., Phiri, I., Geysen, D., Vercruysse, J. & Van den Bossche, P., 2007, 'The comparative role of cattle, goats and pigs in the epidemiology of livestock trypanosomosis on the plateau of Eastern Zambia', *Veterinary Parasitology* 147, 231–238. http://dx.doi.org/10.1016/j.vetpar.2007.04.005

Sinyangwe, L., Delespaux, V., Brandt, J., Geerts, S., Mubanga, J., Machila, N. *et al*., 2004, 'Trypanocidal drug resistance in Eastern province of Zambia', *Veterinary Parasitology* 119, 125–135. http://dx.doi.org/10.1016/j.vetpar.2003.11.007

Van den Bossche, P., Chitanga, S., Masumu, J., Marcotty, T. & Delespaux, V., 2011, 'Virulence in *Trypanosoma congolense* Savannah subgroup. A comparison between strains and transmission cycles', *Parasite Immunology* 33, 456–60. http://dx.doi.org/10.1111/j.1365-3024.2010.01277.x

Van den Bossche, P. & De Deken, R., 2002, 'Seasonal variations in the distribution and abundance of the tsetse fly, *Glossina morsitans morsitans* in eastern Zambia', *Medical and Veterinary Entomology* 16, 170–176. http://dx.doi.org/10.1046/j.1365-2915.2002.00360.x

Van den Bossche, P., 2001, 'Some general aspects of the distribution and epidemiology of bovine trypanosomosis in southern Africa', *International Journal for Parasitology* 31, 592–598. http://dx.doi.org/10.1016/S0020-7519(01)00146-1

Van den Bossche, P., Doran, M. & Connor, R.J., 2000, 'An analysis of trypanocidal drug use in the Eastern Province of Zambia', *Acta Tropica* 75, 247–258. http://dx.doi.org/10.1016/S0001-706X(00)00059-0

Van den Bossche, P. & Staak, C., 1997, 'The importance of cattle as a food source for *Glossina morsitans morsitans* Westwood (Diptera: Glossinidae) in Katete District, Eastern Province, Zambia', *Acta Tropica* 65, 105–109. http://dx.doi.org/10.1016/S0001-706X(97)00658-X

Human cystic echinococcosis in South Africa

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How to cite this abstract:
Mogoye, B., Menezes, C.N., Grobusch, M.P., Wahlers, K. & Frean, J., 2012, 'Human cystic chinococcosis in South Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #469, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.469

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Cystic echinococcosis (CE) is caused by the tapeworm, *Echinococcus granulosus*. The tapeworms resides in the small intestines of canids and the lifecycle involves both intermediate and definitive hosts. Humans are accidental intermediate hosts. Cystic echinococcosis is an economically important infection constituting a threat to public health, and is considered an emerging disease around the world. There are at least 10 *Echinococcus* strain types (G1 – G10), each exhibiting diversity of morphology, development and host range. The epidemiology of CE is poorly understood in South Africa. A retrospective data analysis of the National Health Laboratory Service (NHLS) laboratory information system on echinococcosis serology, microscopy and histopathology results in eight provinces (excluding KwaZula-Natal) showed an overall positivity rate in submitted diagnostic samples of 17.0% (1056/6211), with the Eastern Cape (30.4%), North West (19.0%) and Northern Cape (18.0%) provinces showing highest rates. The data showed considerable variability between provinces. The review also showed that most proven cases were negative on serology, implying that the actual number of patients could be underestimated. To our knowledge, no data exist about the prevalent strains of *E. granulosus* and this prospective study will attempt to fill that gap. The aim is to genotype strains causing the disease in South Africa. Two different polymerase chain reaction (PCR) methods will be used to respectively target the 12S rRNA and *nad* 1 genes. To date, three samples have been genotyped as G1, G5 and G6; suggesting diversity of strains prevalent in the country, but more data is needed for a clearer picture.

Resource mapping and emergency preparedness to infectious diseases in human and animal populations in Kibaha and Ngorongoro districts, Tanzania

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How to cite this abstract:
Karimuribo, E.D., Jones, B., Matee, M.I., Kambarage, D.M., Mounier-Jack, S. & Rweyemamu, M.M., 2012, 'Resource mapping and emergency preparedness to infectious diseases in human and animal populations in Kibaha and Ngorongoro districts, Tanzania', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #470, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.470

A rapid situation analysis was conducted in Kibaha and Ngorongoro districts in Tanzania to map resources as well as analysing emergency preparedness to infectious diseases in animal (domestic and wild) and human populations. Kibaha was chosen as a district close to a commercial city (Dar es Salaam) while Ngorongoro represented a remote, border district with high interactions between humans, domestic and wild animals. In this study, data on resources and personnel as well as emergency preparedness were collected from all wards (n = 22), human health facilities (n = 40) and livestock facilities in the two districts using interview checklists and questionnaires. Descriptive statistics for resources were calculated and mapped by district. Kibaha district had a higher human population density, more health workers, better equipped health facilities and better communication and transport systems. On the other hand, Ngorongoro had a higher population of livestock and more animal health facilities but a poorer ratio of animal health workers to livestock. The average ratio of health personnel to population in catchment areas of the health facilities was 1:147 (range of 1:17–1:1200). The ratio of personnel to human population was significantly higher in Kibaha (1:95) than in Ngorongoro (1:203) district ($p = 0 < 0.001$). Considering the limited resources available to both human and animal health sectors and their different strengths and weaknesses there are opportunities for greater collaboration and resource-sharing between human and animal health for improved surveillance and emergency-preparedness.

Note: Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

First International One Health congress

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How to cite this abstract:
Jeggo, M.H., 2012, 'First International One Health congress', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #471, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.471

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

More than 650 people from around 60 countries attended the 1st International One Health Conference, held in Melbourne from 14 to 16 February 2011. Scientists, clinicians, government and community members from a range of disciplines came together to discuss the benefits of working together to promote a One Health approach to human, animal and environmental health. One Health embraces systems thinking and recognising the interdependence of people, animals and environment. The conference was hosted by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) and was supported by international agencies, the Australian and Canadian governments, and industry.

The Organising Committee recognised from the outset, the need to provide a forum not just for scientific presentation, but for open discussion and dialogue around the policy and political issues, as well as the science that drives the One Health agenda. The Committee was also cognizant of the need to embrace a definition of One Health that includes food security and food safety and included the social and economic pressures that shapes this area. The meeting was therefore organised under four themes with plenary sessions followed by breakout parallel sessions for each of these. The themes covered Disease Emergence, Environmental Drivers, Trade, Food Security and Food Safety, and Science Policy and Political Action. The plenary session commenced with one or two keynote presentations by world leaders on the topic being covered, followed by panel discussions involving six to eight experts and involving all participants at the congress. Each of the panel members spoke briefly on the topic covered by the keynote speaker and were asked to be as provocative as possible. The discussions that followed allowed debate and discussion on the keynote presentations and the panel members comments. This was followed by six to eight parallel breakout sessions involving in depth papers on the session's topic. Throughout the conference at various times, sponsored sessions dealt with particular areas of science or policy providing a further framework not only to learn current science but for debate and discussion. A full copy of all abstracts is available on the web at http://www.springerlink.com.

In concluding the Congress recognised the interdependence of, and seeks to improve human, animal and environmental health; recognised that communication, collaboration and trust between human and animal health practitioners is at the heart of the One Health concept; agreed that a broad vision that includes other disciplines such as economics and social behaviour is essential to success. The Congress stressed the need to promote the 'do-able' such as improving surveillance and response for emerging infectious diseases whilst developing the broader approach. It identified a need to emphasise community participation and development of community capacity, and especially, an open transparent dialogue with both a 'ground up' and 'top down' approach that would lead to an improved understanding of our ecosystems, including molecular ecobiology, are an essential part of One Health.

Foot-and-mouth disease control in Zambia: A review of the current situation

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How to cite this abstract:
Sinkala, Y., Pfeiffer, D., Kasanga, C., Muma, J.B., Simuunza, M. & Mweene, A., 2012, 'Foot-and-mouth disease control in Zambia: A review of the current situation', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #472, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.472

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Zambia has been experiencing low livestock productivity as well as trade restrictions owing to the occurrence of foot-and-mouth disease (FMD) and contagious bovine pleura pneumonia (CBPP). Foot-and-mouth disease was first recorded in Zambia in 1933 in the Western Province and since then the country has experienced repeated outbreaks. Bearing in mind the pressure that may be existing on the many risk factors for FMD including climate change, there is need to review our knowledge on FMD control.

We present the spatial distribution of the FMD outbreaks that have been recorded in Zambia in the last twenty years, and the effect of the vaccinations and movement control that have been applied. We propose further strain characterisation of previous FMD outbreaks, including full sequence of VP1 gene and the 5′UTR site. The data will be geo-coded and populated with risk factor attributes. We also present preliminary findings of the buffalo and cattle probang sampling that was conducted in Lochnivar and Kafue National Park. We further probang sampled 25 buffalo at each interface area in Sioma Ngwezi, Lukusuzi and Lower Zambezi national parks. Villages in close proximity to the buffalo populations as well as those not in close proximity will be multistage cluster sampled for comparison. The data will be geo-coded and populated with risk factor and foot-and-mouth disease virus (FMDV) characterisation attributes. Data collected using a pre-tested structured questionnaire will be geo-coded and populated with identified risk factors and stored in a database and will be spatially modelled to determine their effect on FMD occurrence and control measures. New outbreaks of FMD that may occur will be investigated to find out if there are new strains involved, species affected and predisposing risk factors.

The authors conclude that impacts of FMD on livelihoods if appropriate control measures are not put in place are far more devastating especially at community level. Presented with the current poverty levels failure to institute result oriented control measures will exacerbate the already life-threatening situation.

Genomic sequence of infectious bursal disease virus from Zambia suggests evidence for genome re-assortment in nature

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Kasanga, C.J., Yamaguchi, T., Munang'andu, H.M., Wambura, P.N., Ohya, K. & Fukushi, H., 2012, 'Genomic sequence of infectious bursal disease virus from Zambia suggests evidence for genome re-assortment in nature', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #473, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.473

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Infectious bursal disease virus (IBDV) is a bi-segmented RNA virus, which belongs to the genus *Avibirnavirus* of the family *Birnaviridae*. Two serotypes, 1 and 2, exist in IBDV. The serotype 1 IBDVs are the causative agents of infectious bursal disease (IBD) in chickens worldwide and lead to immunosuppression in young birds. Genome re-assortment has been speculated to occur and contribute to the emergence of new IBDV strains. However, evidence was lacking until recently when two re-assortant viruses were detected in China. In this study, we determined the complete nucleotide sequence of an IBDV, designated KZC-104, from a confirmed natural IBD outbreak in Lusaka, Zambia in 2004. The genome consisted of 3074 and 2651 nucleotides in the coding regions of segments A and B, respectively. Alignment of both nucleotide and deduced amino acid sequences, and phylogenetic analysis revealed that the genome segment A of KZC-104 was derived from a very virulent strain, whereas its segment B was derived from a classical attenuated strain. On BLAST search, the full-length segments A and B sequences showed 98% closest nucleotide homology to the very virulent strain D6948 and 99.8% closest nucleotide homology to the classical attenuated strain D78, respectively. This is a unique IBDV reassortant strain, which has emerged in nature involving segment B of a live attenuated vaccine. This observation provides direct evidence for the involvement of vaccine strains in the emergence of reassortant IBDV in the field. Taken together, these findings suggest an additional risk of using live IBDV vaccines, which may act as genetic donors for genome re-assortment. Further studies are required to investigate the epidemiology and biological characteristics of reassortant strains so that the appropriate and safe IBDV vaccines can be recommended.

Infectious diseases of economic importance: Molecular biological characteristics of foot-and-mouth disease viruses collected in Tanzania from 1967 to 2009

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How to cite this abstract:
Kasanga, C.J., Sallu, R., Mpelumbe-Ngeleja, C.A.R., Wadsworth, J., Ferris, N.P., Hutchings, G.H. *et al.*, 'Infectious diseases of economic importance: Molecular biological characteristics of foot-and-mouth disease viruses collected in Tanzania from 1967 to 2009', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #474, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.474

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Foot-and-mouth disease (FMD) is endemic in Tanzania. Since the first reports in 1954, FMD has caused significant economic losses in the country due to mortality and morbidity of livestock and costs associated with controlling the disease. The aim of this study was to review the serotype and genetic relationships of the FMD virus (FMDV) recovered from outbreaks in Tanzania, and compare them with viruses detected from elsewhere in the sub-Saharan region. At the World Reference Laboratory for foot-and-mouth disease (WRLFMD), a total of 106 FMD viruses have been isolated from samples collected between 1967 and 2009 from northern, southern, eastern and central parts of Tanzania. The presence of FMDV was determined by laboratory methods such as VI, CF, antigen ELISA and RT-PCR. Phylogenies of VP1 sequences were determined by the Neighbour-joining method. Foot-and-mouth disease virus SAT1 was the most frequent serotype (46.2%; n = 49) isolated in Tanzania followed by O (26.4%; n = 27), A (14.1%; n = 15) and SAT 2 (11.3%; n = 13). Genotyping showed that type O viruses fell into either the EAST AFRICA 1 (EA-1) or EA-2 topotypes, type A's into the AFRICA topotype (genotype I), type SAT 1's into topotype I and type SAT 2's into topotype IV. This study reveals that serotypes A, O, SAT1 and SAT2 cause FMD outbreaks in Tanzania. Recent samples from outbreaks in 2008, 2009 and 2010 have been typed as serotypes A, O, SAT1 and SAT2. Phylogenetic analysis of FMDV isolates from Tanzania showed that they are genetically related to lineages and topotypes from West and East Africa. In Tanzania, lack of comprehensive animal movement records and inconsistent vaccination programs make it difficult to determine the exact source of FMD outbreaks or to trace the transmission of the disease over time. Therefore, further collection and analysis of samples from domestic and wild animals, together with improved local epidemiological investigation of FMD outbreaks is required to elucidate the complex epidemiology of FMD in the sub-Saharan region.

Development of a curriculum for training in One Health analytical epidemiology at the University of Zambia

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How to cite this course:
Muma, J.B., Simuunza, M., Mwachalimba, K., Munyeme, M., Namangala, B., Hankanga, C. *et al*., 2012, 'Development of a curriculum for training in O ne Health analytical epidemiology at the University of Zambia', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #475, 4 pages. http://dx.doi.org/10.4102/ojvr.v79i2.475

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Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Recently, the world has witnessed emergence of novel diseases such as avian influenza, HIV and AIDS, West Nile Virus and Ebola. The evolution of these pathogens has been facilitated mainly by a constantly evolving animal-human interface. Whilst infectious disease control was previously conceptualised as either public health or animal health related issues, the distinction between disciplinary foci have been blurred by multiple causal factors that clearly traverse traditional disciplinary divides. These multiple evolutionary pressures have included changes in land use, ecosystems, human-livestock-wildlife interactions and antibiotic use, representing novel routes for pathogen emergence. With the growing realisation that pathogens do not respect traditional epistemological divides, the 'One Health' initiative has emerged to advocate for closer collaboration across the health disciplines and has provided a new agenda for health education.

Against this background, the One Health Analytical Epidemiology course was developed under the auspices of the Southern African Centre for Infectious Diseases Surveillance by staff from the University of Zambia with collaborators from the London School of Hygiene and Tropical Medicine and the Royal Veterinary College in London. The course is aimed at equipping scientists with multidisciplinary skill sets to match the contemporary challenges of human, animal and zoonotic disease prevention and control. Epidemiology is an important discipline for both public and animal health. Therefore, this two-year programme has been developed to generate a cadre of epidemiologists with a broad understanding of disease control and prevention and will be able to conceptualise and design holistic programs for informing health and disease control policy decisions.

Introduction

In recent years, there has been increased discourse in the global health community on the subject of One Health (Meisser, Schelling & Zinsstag 2011; Zinsstag *et al*. 2011). One Health is a systems approach to health in social-ecological systems incorporating the health of humans, animals and the environment. This has been prompted by the unprecedented emergency of novel zoonotic pathogens such as severe acute respiratory syndrome (SARS), avian influenza, West Nile virus, Rift Valley fever and Ebola, amongst others. Furthermore, it has been recognised by the World Health Organisation (WHO) that some of the classical zoonoses such as rabies, cysticercosis, trypanosomosis and bovine tuberculosis have not been given the appropriate attention (Briggs & Hanlon 2007; Cleaveland, Haydon & Taylor 2007; Fooks 2005; WHO 2005). Taylor, Latham and Woolhouse (2001) observed that out of 1415 human pathogens, 61% are zoonotic. The increased risks of pathogen transmission from animals to humans have been attributed to a number of anthropogenic factors such as complex patterns of global change; the inextricable interconnection of humans through trade and travel; increased intensification of animal husbandry; the development of peri-urban systems for livestock production, increased domestication of wildlife in game ranches and encroachment of people and their livestock into wildlife areas (Zinsstag *et al*. 2011). The steady growth in the world population has resulted in limited space for human habitation, leading to continuous human encroachment on wildlife sanctuaries and this has amplified the risk of occurrence of new emerging diseases (Marcotty *et al*. 2009). A joint WHO, Food and Agriculture Organisation (FAO) and World Organisation for Animal Health (OIE) report on emerging zoonoses (2004), argued that zoonotic diseases are on the increase owing to shortfalls in public health infrastructure and policy, a paucity of public health scientific studies to answer questions and build expertise, and a lack of integrated human and animal health surveillance (WHO 2005).

The avian influenza pandemic demonstrated that no single profession can fight these disease threats single-handedly. Therefore, all players from human, veterinary and environmental health are indispensible in addressing the threats to health confronting our world. All these and

many other factors have drawn professions from different disciplines to engage in joint efforts in finding solutions to the threat that infectious diseases pose to human and animal survival (Powdrill, Nipp & Rinderknecht 2010). Dedomon (2010) noted that, One Health is a paradigm that serves the needs of all and encourages the spirit of collaboration and scientific inquiry for the common good. Not only has the need for interdisciplinary participation been acknowledged in projects involving control of zoonoses (Marcotty *et al*. 2009; Roth *et al*. 2003; Zinsstag *et al*. 2007) but there has been a new awakening to change the way health professions are educated (Marcotty *et al*. 2009; Zinsstag *et al*. 2011). Consequently, we have seen increased advocacy for joint training in public health which has resulted in more learning institutions taking up the challenge of breaking traditional educational boundaries in health training. A good example of integrated training where the One Health concept has be embraced principally and structurally is the College of Veterinary Medicine, Nursing and Allied Health (CVMNAH) at Tuskegee University in the United States of America (Habtemariam 2011). The College is composed of the School of Veterinary Medicine (Animal Health) and the School of Nursing and Allied Health (Human Health) and is said:

> to be the only one of its kind in the United States where Animal Health (Veterinary Medicine) and Human Health (Nursing & Allied Health) are merged under one College within the framework of One Health-One Medicine. (Habtemariam 2011)

Further, in 2003, Kansas State University started to offer a new Master's program in One Health Public Health, with collaboration from Agriculture, Arts and Sciences, Human, Ecology and Veterinary Medicine (Michael 2010). More recently, Sokoine University of Agriculture introduced a course in One Health Molecular Biology aimed at increasing collaboration in diagnosis of infectious diseases, between the medical and veterinary sectors across sub-Saharan Africa and it envision to improvement of the quality of laboratory diagnosis and the standardisation of diagnostic methods across human and veterinary diagnostic laboratories, which is essential in facilitating international trade as advocated for by the Sanitary and Phytosanitary (SPS) Agreement under the World Trade Organisation (WTO). This paper discusses the development of the One Health Analytical Epidemiology at the University of Zambia, whose aim is to equip scientists with skills in analytical epidemiology for the efficient prevention and control of human, animal and zoonotic diseases.

Development of the course curriculum

The curriculum was developed by staff from the University of Zambia's Schools of Medicine and Veterinary Medicine. After the initial document was developed by the local staff, consultations were held with collaborators from the London School of Hygiene and Tropical Medicine (LSHTM), the Royal Veterinary College (RVC) in London. Further consultations were held with staff from the Sokoine University of Agriculture in Tanzania.

Curriculum development started with the formation of a joint working group comprising representatives from the Medical and Veterinary Schools of the University of Zambia (UNZA). The working group drew on expertise from epidemiology, public health and demography. The first committee meeting was held in September 2009 and involved brain storming on the *modus operadi* regarding the process of curriculum development. A work plan was formulated, where targets were set and responsibilities assigned to each member of the committee, which included the review of reference materials collected from collaborating institutions. Subsequently, key courses were identified and a draft course outline proposed (Table 1). Each member of the working group was assigned to develop a structure for the course they were comfortable with. After a series of meetings, a zero-draft was developed and circulated amongst members of the group for review and comments. Finally, a four day workshop was organised for in-depth review of the zero draft. The output of the workshop was a working document that was ready for external review.

External consultations were in two forms. Firstly, there was soliciting for course materials of related programmes from collaborating institutions and personal discussions with the expert in the identified fields. Materials were collected from the LSHTM and the RVC in London, and Muhimbili University of Health and Allied Sciences (MUHAS) in Tanzania. The Curriculum for MSc in Public Health currently running at the School of Medicine at UNZA was also included in the reference material. Secondly, a visit was made to the UK in April 2011, with the main purpose of presenting the working document to collaborators in the North and receiving comments. A series of meetings were held of small groups of specialists in epidemiology and economics at both LSHTM and the RVC. The visits presented us with an opportunity to discuss the curriculum with

TABLE 1: Programme structure for the One Health Analytical Epidemiology and distribution of courses by semester.

Semester	Course name	Description	Hours
Semester 1	Research Methodology and Computer Applications	Core course	117
	Principles of Epidemiology and Biostatistics	Core course	132
	Disease Surveillance and Risk Analysis	Core course	120
	Emerging and Re-emerging Disease	Core course	117
Semester 2	Infectious Disease Modelling and Geographical Information System	Core course	123
	Advanced Statistical Methods in Epidemiology	Core course	117
	One Health Medicine and Globalisation	Core course	91
	Health Economics, Policy, Monitoring and Evaluation	Elective course	120
	Molecular Epidemiology and Bioinformatics Environmental Epidemiology	Elective course	91

leading experts and improve curriculum design. During this final visit, the outline of the draft curriculum and its contents were thoroughly revised and modified. Two of the proposed courses were re-conceptualised and renamed and another two were merged in order to conform to the One Health concept. The visit also presented an opportunity to learn and share knowledge on the techniques of developing the curriculum and somehow strengthened the interactions with various professionals of different expertise in the field of medicine and veterinary medicine.

Upon returning from the UK, a visit was made to the Sokoine University of Agriculture in Tanzania where further consultations with regional expertise were held. After this external review, the curriculum was finalised and presented to the Joint School of Veterinary Medicine and School of Medicine Board of Studies and then subsequently to the University of Zambia Senate for final approval in June 2011.

Structure of the drafted curriculum

The output of the discussions, workshops and inter-institutional consultations resulted into the Curriculum for the Master of Science in One Heath Analytical Epidemiology that will be offered by the University of Zambia, starting from September 2011. The programme is structured such that by the end of the training, students of MSc One Health Analytical Epidemiology should be able to do the following:

- demonstrate knowledge of the concept of 'One Health' and its application in the development of health policy and the control and prevention of infectious diseases
- demonstrate knowledge on how interactions between human and animal populations and environmental changes can lead to emergency and re-emerging of infectious diseases
- plan, undertake and analyse data from a research project concerning human, animal and zoonotic diseases and be able to monitor and evaluate activities for policy and programme development
- apply a scientific style of writing in the presentation of research
- apply economic and socio-economic concepts and methods in the design, implementation and evaluation of health delivery services
- determine the factors affecting the spread of disease through human and animal populations and be able to prevent or control such spread.

The courses that the students will have to undertake in order for the above stated learning outcomes to be achieved are shown in Table 1. It is hoped that at the end of their training students will have acquired skills in analytical epidemiology for efficient prevention and control of human, animal and zoonotic diseases.

The programme shall comprise two parts, a taught component executed by coursework in the first year (Part I) and a research project that will culminate in the submission of a dissertation in the second year (Part II). The two parts shall be undertaken consecutively. The first part (Part I) of the programme shall be undertaken over a period of two semesters (Semesters 1 and 2) of fulltime study and shall comprise:

- course work as required by the School Board of Graduates Studies (http://www.unza.zm)
- lectures, practicals and tutorials as well as participation in field trips and seminars
- continuous assessment which will include written tests, assignments, laboratory or field reports and seminar presentations
- a written examination at the end of each semester
- submission of a research proposal for part II of the programme for approval.

The second part (Part II) of the program shall be undertaken over a period of two semesters (Semesters 3 and 4) of full-time study and shall comprise supervised research work culminating in the submission of a dissertation. The topic (research proposal) of the dissertation shall be guided by the One Health theme and shall be approved by the School Postgraduate Committee in the last seven weeks of the second semester of Part I. For successful implementation of the course, no student will be permitted to proceed to Part II of the degree program unless he or she has satisfied the requirement of Part I.

Teaching strategies

Course delivery will mainly be done by experts from the Schools of Medicine and Veterinary at the University of Zambia. Experts from other southern African academic and research institutions within the Southern African Centre for Infectious Disease Surveillance (SACIDS) consortium as well as staff from collaborating research institutions in industrialised countries, especially the RVC and LSHTM will also participate in course delivery. This will create a blend of professional knowledge and skills in epidemiology and One Health that will provide a unique learning environment.

Career opportunities for graduates

Students graduating with an MSc in One Health Analytical Epidemiology would be professionals with international competence to work in the local and regional markets in training and research institutions, government public health system and international bodies tasked with management of human and animal health such as WHO, OIE, FAO et cetera.

Admission criteria

All applications will be considered through the Directorate of Research and Graduate Studies of the University of Zambia. The following shall be eligible to apply for the Masters degree in Analytical Epidemiology at the University of Zambia:

- Graduates from any recognised University in the field of Biological Sciences, Medicine, Veterinary Medicine/ Science and any other related field of study.
- The minimum requirement for medical and veterinary candidates is a first degree with at least B grade for Epidemiology, Statistics or Community Medicine. Applicants must also have obtained a minimum grade of B in A level mathematics.
- Medical and veterinary graduates with at least one year of related medical or veterinary experience will have an added advantage.

- Graduates from Biological Sciences must have at least a Merit grade and those with at least two years of post-qualifying experience will have an added advantage.
- Candidates wishing to pursue any specific course(s) under this programme may be considered, and will be awarded a certificate upon successful completion. Such students shall not be eligible for the award of the MSc. in One Health Analytical Epidemiology.
- In general, rules and regulations prescribed by the Directorate of Research and Graduate Studies at the University of Zambia shall apply (http://www.unza.zm).

We have developed a Master of Science programme in One Health Analytical Epidemiology which is aimed at furthering the holistic approach in combating the threat of infectious diseases using the combined resources of human and veterinary professionals and allied sciences. This has come out of the recognition that the control of diseases in the Southern African Development Community (SADC) region requires acquisition and strengthening of human knowledge and skills by training professionals with competence in One Health and epidemiology. Thus, graduates of this training programme will not only be epidemiologists, but will also have an understanding of the concept of One Health.

This curriculum is tailored towards epidemiological understanding of disease dynamics and thus does not claim to offer all that is needed to be learnt on the subject of 'One Health'. However, it is acknowledged that epidemiology is a key discipline in effective management of human, animal and environmental health. In order to make the programme more applicable, other vital subjects such as global health and economics have been fused in so as to supplement what is ordinarily known as classical epidemiology. Collectively, these tools are meant to help in understanding the close association that exists between human and animal health as evidenced in the recent occurrence of the H1N1 Avian Influenza pandemic. The programme will therefore emphasise the need for inter-sectoral collaboration in disease surveillance, epidemic disease preparedness and response as well as development of enabling policy platforms, across the human, animal and eco-health sectors. It has become apparent that the responsibility to ensure public health, especially as it pertains to the risk of emerging and re-emerging zoonoses, is a shared responsibility of professionals in the human and veterinary medicine and other allied sciences such as ecology (Michael 2010).

Marcotty *et al.* (2009) strongly encouraged collaboration between the veterinary and the medical sectors, in the diagnosis, monitoring and control of zoonotic brucellosis and tuberculosis. These authors further, added that useful collaborations could be nurtured at undergraduate- and postgraduate-training levels, through, for example, common public-health modules or courses. This programme has been designed to train a new generation of world class scientists who will serve to address the heavy burden and threat of emerging and re-emerging infectious diseases in Africa that greatly hamper public health and animal health and thus socio-economic development of developing countries (WHO 2005). The successful implementation of the programme strongly depends on support from other SACIDS consortium universities in southern Africa as well as the LSHTM, RVC and other internationally reputable institutions.

Conclusion

It is hoped that the training programme will create increased collaboration between professions in medical, veterinary and allied sciences and create a platform for sharing their knowledge and resources to better promote human, animal and environmental health. The medical profession would, benefit greatly from an improved knowledge and understanding of the epidemiology of zoonotic diseases whilst the veterinary sector would, have a better appreciation of what is expected from them, in terms of controlling the zoonoses in order to assure public health.

Acknowledgement

The Wellcome Trust is thanked for funding the development of the curriculum through SACIDS. The curriculum development was part of wider SACIDS Consortium projects under the framework of the One Heath Concept. We are further grateful for the support we received from the London International Development Centre, London School of Hygiene and Tropical Medicine, the Royal Veterinary College, Sokoine School of Agriculture, Muhimbili University of Health and Allied Sciences and the University of Zambia. We would also like to thank Professor Kenny Samui for his contribution to the successful development of this curriculum.

References

Briggs, D. & Hanlon, C.A., 2007, 'World Rabies Day: focusing attention on a neglected disease', *Veterinary Record* 161, 288–289. http://dx.doi.org/10.1136/vr.161.9.288, PMid:17766802

Cleaveland, S., Haydon, D.T. & Taylor, L., 2007, 'Overviews of pathogen emergence: which pathogens emerge, when and why?', *Current Topics in Microbiology and Immunology* 315, 85–111. http://dx.doi.org/10.1007/978-3-540-70962-6_5

Cleaveland, S., Laurenson, M.K. & Taylor, L.H., 2001, 'Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence', *Philosophical Transactions of the Royal Society B: Biological Sciences* 356, 991–999. http://dx.doi.org/10.1098/rstb.2001.0889, PMid:11516377

Dedomon, E.R., 2010, 'The One Health Initiative through a 50-Year Lens', *One Health News Letter* Fall, 3–5.

Fooks, A.R., 2005, 'Rabies remains a "neglected disease"', *EuroSurveillance* 10, 211–212. PMid:16972345

Habtemariam, 2011, *The College of Veterinary Medicine, Nursing and Allied Health*, viewed 31 August 2011 from, http://www.tuskegee.edu/academics/colleges/cvmnah/office_of_the_dean_-_cvmnah.aspx

Marcotty, T., Matthys, F., Godfroid, J., Rigouts, L., Ameni, G., Gey van Pittius, N. *et al.*, 2009, 'Zoonotic tuberculosis and brucellosis in Africa: Neglected zoonoses or minor public-health issues? The outcomes of a multi-disciplinary workshop', *Annals of Tropical Medicine and Parasitology* 103, 401–411. http://dx.doi.org/10.1179/136485909X451771, PMid:19583911

Meisser, A., Schelling, E. & Zinsstag, J., 2011, 'One health in Switzerland: a visionary concept at a crossroads?', *Swiss Medical Weekly* 141, 13201.

Michael, B., 2010, 'An innovative approach to graduate Public Health education', *One Health Newsletter* Fall, 1–2.

Powdrill, T.F., Nipp, T.L. & Rinderknecht, J.L., 2010, 'One Health approach to influenza: Assessment of critical issues and options', *Emerging Infectious Diseases* 16, e1. http://dx.doi.org/10.3201/eid1608.100673, PMid:20678310

Roth, F., Zinsstag, J., Orkhon, D., Chimed-Ochir, G., Hutton, G., Cosivi, O. *et al.*, 2003, 'Human health benefits from livestock vaccination for brucellosis: case study', *Bulletin of World Health Organisation* 81, 867–876. PMid:14997239

Taylor, L.H., Latham, S.M. & Woolhouse, M.E., 2001, 'Risk factors for human disease emergence', *Philosophical Transactions of the Royal Society B: Biological Sciences* 356, 983–989. http://dx.doi.org/10.1098/rstb.2001.0888, PMid:11516376

WHO, 2005, *The control of neglected zoonotic diseases: A route to poverty alleviation,* Geneva, Switzerland.

Zinsstag, J., Schelling, E., Roth, F., Bonfoh, B., De Savigny, D. & Tanner, M., 2007, 'Human benefits of animal interventions for zoonosis control', *Emerging Infectious Diseases* 13, 527–531. http://dx.doi.org/10.3201/eid1304.060381, PMid:17553265

Zinsstag, J., Schelling, E., Waltner-Toews, D. & Tanner, M., 2011, 'From "One Medicine" to "One Health" and systemic approaches to health and well-being', *Preventive Veterinary Medicine* 101, 148–156. http://dx.doi.org/10.1016/j.prevetmed.2010.07.003, PMid:20832879

MSc: In One Health Molecular Biology - P. Wambura

One Africa One Health www.sacids.org

MSc In One Health Molecular Biology

Sokoine University of Agriculture

The MSc in One Health Molecular Biology course is offered at the Faculty of Veterinary Medicine of Sokoine University of Agriculture.

The course focuses on practical skills and a thorough understanding of how to apply molecular biology to One Health – an emerging discipline which promotes collaboration between the veterinary and medical professions.

Upon completion, researchers will be highly-skilled professionals able to:

- demonstrate knowledge and understanding of molecular biological techniques and their applications in the detection, identification and monitoring of infectious diseases of humans and animals
- demonstrate specialist knowledge for understanding how prophylactic or curative treatments or therapies for diseases may be developed through molecular research of host and pathogen genomes
- devise molecular biological approaches to find newer ways that will improve the health of humans, animals and our environment
- join international PhD programmes or secure employment in academic and research institutions

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Course Structure

Year 1

- Research methods
- Statistics and data management
- Advanced molecular biology
- Immunology of infectious diseases
- One Health medicine
- Comparative genomics and bioinformatics
- Techniques in molecular and cellular biology
- Pathogen evolution and emerging infectious diseases

During the second semester students will receive mentoring to develop a research proposal, which must be presented to an academic panel for approval. The research proposals will be related, as far as possible, to One Health themes identified in the main SACIDS framework, and will address:

- Pathogen biology
- Host pathogen interactions
- Detection, identification and surveillance of disease
- Biodiversity or environmental determinants of One Health

Year 2

During the second year, the learning process will be pro-active and will include a literature review, intensive laboratory work, statistical analysis and writing a dissertation

Partnerships

The curriculum has been developed in partnership with the UK's London School of Hygiene and Tropical Medicine, the Royal Veterinary College and others, which offer world-class postgraduate programmes in health and related disciplines.

Course modules are taught by academicians from SUA and Muhimbili University for Health and Allied Sciences (MUHAS) supported by internationally recognised staff from SACIDS Partner institutions and others.

Experienced scientists from SACIDS institutions will combine strong capacity in molecular biology and One Health to provide a unique blend of professional knowledge and skills for the graduates of this innovative MSc programme.

While the SACIDS financial support targets students from the SADC region, students from other parts of Africa or elsewhere who are able to meet their own cost are also eligible.

Acknowledgement

The One Health Molecular Biology MSc course is supported by SACIDS through a grant from the Wellcome Trust under its African Institutions Initiative (www.wellcome.ac.uk)

Source: Author's original material as presented at the conference

How to cite this course: Wambura, P., 'MSc: In One Health Molecular Biology', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #476, 1 pages. http://dx.doi.org/10.4102/ojvr.v79i2.476
Note: Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

MSc One Health Analytical Epidemiology - M.C. Simuunza

MSc One Health Analytical Epidemiology

COURSE DESCRIPTION

This exciting new course focuses on provision of knowledge in analytical epidemiology and on how to apply this knowledge in the control of diseases in both human and animal populations (One Health Concept). The course has been designed to train a new generation of human capital which will provide the necessary leadership in solving various health challenges using epidemiological tools to improve the health and welfare of our communities.

WHO SHOULD APPLY?

Applicants for the course should have a degree in medicine, veterinary medicine, or a basic degree in biological sciences. Graduates in health related field of study such as statistics, demography, food science and public health may apply provided they have interest in health and have experience of having worked in an institution dealing with health issues. **There are limited scholarships available for this course provided by the Wellcome Trust through SACIDS.**

THE LEARNING ENVIRONMENT

The MSc in One Health Analytical Epidemiology will be offered by the School of Veterinary Medicine in collaboration with the School of Medicine at the University of Zambia. All the students will be registered by the School of Veterinary Medicine. The School of Veterinary Medicine was established in 1984 and has some of the best learning facilities in Zambia. The School has long standing experience in conducting postgraduate training and research in diseases of veterinary and public health importance.
The School is located within the University of Zambia main campus.

Course delivery will mainly be done by experts from the Schools of Veterinary Medicine and Medicine of the University of Zambia. Experts from other Southern African academic and research institutions within the SACIDS consortium as well as staff from collaborating research institutions in industrialized countries, especially the Royal Veterinary College (RVC) and the London School of Hygiene and Tropical Medicine (LSHTM) will also participate in course delivery. This will create a blend of professional knowledge and skills in epidemiology and One Health that will provide a unique learning environment.

THE SOUTHERN AFRICAN CENTRE FOR INFECTIOUS DISEASE SURVEILLANCE

SACIDS is a One Health consortium of southern African academic and research institutions involved with infectious diseases of humans and animals in the Democratic Republic of Congo (DRC), Mozambique, South Africa, Zambia and Tanzania. Staff from these institutions are working in smart partnership with colleagues from LSHTM and RVC – two University of London Colleges which participate in the interdisciplinary London International Development Centre (LIDC). SACIDS also collaborates with the International Livestock Research Institute (ILRI) and continues to forge linkages with other institutions from industrialised countries. The SACIDS headquarters is located at the Sokoine University of Agriculture.

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Source: Author's original material as presented at the conference

Course continues on the next page →

How to cite this course: Simuunza, M.C., 'MSc One Health Analytical Epidemiology', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #477, 2 pages. http://dx.doi.org/10.4102/ojvr.v79i2.477

Note: Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

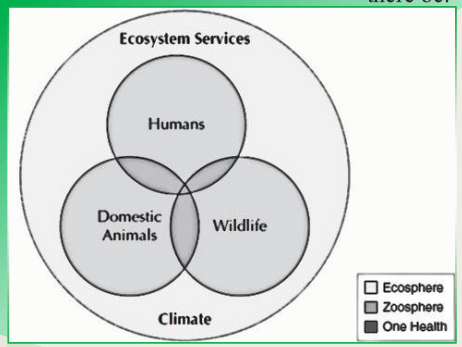
Source: Authors original material as presented at the conference

One Health Analytical Epidemiology

This course aims to equip scientists with skills in analytical epidemiology for the efficient prevention and control of human, animal and zoonotic diseases. One health is a scientific concept whose mission is to foster closer professional interaction, collaboration and educational opportunities across the veterinary, medical and allied sciences for the purpose of improving human and animal health.

"...between animal and human medicine there are no dividing lines--nor should there be."

Rudolf Virchow (1821-1902).



Course outcomes

Upon completion of the MSc in One Health Analytical epidemiology, graduates will be able to:

- Demonstrate knowledge of the concept of "One Health" and its application in the development of health policy and the control and prevention of infectious diseases.
- Demonstrate knowledge on how interactions between human and animal populations and environmental changes can lead to emergency and re-emerging of infectious diseases.
- Plan, undertake and analyze data from a research project concerning human, animal and zoonotic diseases and be able to monitor and evaluate activities for policy and programme development.
- Apply a scientific style of writing in the presentation of research.
- Apply economic and socio-economic concepts and methods in the design, implementation and evaluation of health delivery services.
- Determine the factors affecting the occurrence and spread of disease through human and animal populations and be able to prevent or control such spread.

Course Structure

The course is a two- year full-time MSc programme of the University of Zambia calendar of semesters and academic years comprising of a taught component in the first year and a research project culminating into the submission of dissertation in the second year.

Year One

Students will undertake the following core courses:

- Research Methodology and Computer Applications
- Principles of Epidemiology and Biostatistics
- Disease Surveillance and Risk Analysis
- Emerging and Re-emerging Diseases
- Infectious Disease Modeling and Geographical Information System
- Advanced Statistical Methods in Epidemiology
- One Health Medicine and Globalization

In addition students will be able to choose one elective course from the following:

- Health Economics, Policy, Monitoring and Evaluation
- Molecular Epidemiology and Bioinformatics
- Environmental Epidemiology

Year Two

Year two shall comprise supervised research work culminating into submission of a dissertation and shall be undertaken over a period of two semesters of full-time study. The topic (research proposal) of the dissertation shall be guided by the One Health Theme and shall be approved by the School Postgraduate Committee in the last seven weeks of the second semester of part I.

Acknowledgements
The One Health Analytical Epidemiology MSc programme is supported by SACIDS through a grant from the Wellcome Trust (www.wellcome.ac.uk) under the African Institution Initiative.

Leptospirosis in South Africa

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How to cite this poster:
Saif, A., Frean, J., Rossouw, J. & Trataris, A.N., 2012, 'Leptospirosis in South Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #478, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.478

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Leptospirosis is a common zoonosis worldwide. It has a ubiquitous distribution and causes a wide spectrum of disease. Leptospirosis therefore has a broad reservoir host range, and many infected species of animals excrete leptospires in their urine, which leads to contamination of soil and water. Typical descriptions of the disease include a biphasic (anicteric form) and fulminant disease in the icterohaemorrhagic form. Only a few local case reports of human leptospirosis have been published, the most recent one being in 1974.

A rodent-related zoonosis study (RatZooMan) was conducted from 2003 until 2006 in three provinces (Limpopo, KwaZulu-Natal and the Eastern Cape). Of the people sampled in Cato Crest (Durban, KwaZulu-Natal Province), 43/217 (19.8%) were seropositive for leptospirosis. Of the clinical samples sent to the Special Bacterial Pathogens Reference Unit from all over the country for testing in 2009, 16/176 (9%) were IgM positive; in 2010 and January 2011 to May 2011, 14/215 (6.5%) and 12/96 (12.5%), respectively, were IgM positive.

The apparent incidence of leptospirosis in the South African population is moderately high based on the detected positives in suspected cases; it is thought that the circulating infection rate may be even higher when looking at the RatZooMan results. This may be due to underreporting and undiagnosed cases. Communities in informal settlements in urban areas are especially at risk as infected rodent populations are a continuous source of transmission.

Bartonella henselae and *Bartonella quintana* seroprevalence in HIV-positive, HIV-negative and clinically healthy volunteers in Gauteng, South Africa

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How to cite this poster:
Trataris, A.N., Arntzen, L., Rossouw, J., Frean, J. & Karstaedt, A., 2012, '*Bartonella henselae* and *Bartonella quintana* seroprevalence in HIV-positive, HIV-negative and clinically healthy volunteers in Gauteng, South Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #479, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.479

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Bartonella is a genus of opportunistic, Gram-negative bacilli transmitted from animals to human hosts. Bartonellae are newly emerging pathogens that can cause a variety of clinical manifestations in both immunocompromised and healthy persons.

The aims were to determine the IgG and IgM seroprevalences of *Bartonella henselae* and *Bartonella quintana* in immunocompromised and immunocompetent individuals using an immunofluorescence assay (IFA).

A total of 382 HIV-positive outpatients of the Chris Hani Baragwanth HIV-clinic, 382 retrospective residual samples from HIV-negative antenatal patients, and 42 clinically healthy volunteers were tested using a commercially available IFA kit to determine the prevalence of IgG and IgM antibodies to *B. henselae* and *B. quintana*.

The IgM and IgG seroprevalences for the HIV-positive patients were 14% (53/382) and 32% (121/382), respectively, compared to 18% for both IgM (62/342) and IgG (63/342) in the HIV-negative antenatal patients. Similarly, the prevalence for IgM was 17% (7/42) and IgG was 19% (8/42) for the clinically healthy volunteers.

HIV-positivity appears to be a significant risk factor for *Bartonella* infection, compared with healthy subjects. Although IFAs have a high sensitivity for *Bartonella* antibody detection, they have various limitations including cross-reactivity with other closely-related human pathogens.

Towards One Health Knowledge Networks: A Southern African Centre of Infectious Disease Surveillance case study

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How to cite this poster:
Beda, E., 2012, 'Towards One Health Knowledge Networks: A Southern African Centre of Infectious Disease Surveillance case study', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #480, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.480

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

The dynamic nature of new information and/or knowledge is a big challenge for information systems. Early knowledge management systems focused entirely on technologies for storing, searching and retrieving data; these systems have proved a failure. Juirsica and Mylopoulos[1] suggested that in order to build effective technologies for knowledge management, we need to further our understanding of how individuals, groups and organisations use knowledge.

As the focus on knowledge management for organisations and consortia alike is moving towards a keen appreciation of how deeply knowledge is embedded in people's experiences, there is a general realisation that knowledge cannot be stored or captured digitally. This puts more emphasis in creating enabling environments for interactions that stimulate knowledge sharing.

Our work aims at developing an un-obtrusive intelligent system that glues together effective contemporary and traditional technologies to aid these interactions and manage the information captured. In addition this system will include tools to aid propagating a repository of scientific information relevant to surveillance of infectious diseases to complement knowledge shared and/or acts as a point of reference.

This work is ongoing and based on experiences in developing a knowledge network management system for the Southern African Centre of Infectious Disease Surveillance (SACIDS), A One Health consortium of southern African academic and research institutions involved with infectious diseases of humans and animals in partnership with world-renowned centres of research in industrialised countries.

1.Using Ontologies for knowledge management: An information systems perspective, Igor Jurisica, John Mylopoulos, Eric Yu, University of Toronto, Toronto, Ontario, Canada.

Phytochemical isolation of compounds from *Sceletium tortuosum* and activity testing against *Plasmodium falciparum*

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Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Malaria is a major health care problem in tropical regions due to the increasing resistance of *Plasmodium falciparum* against widely available antimalarial drugs. Traditional societies relied on medicinal plants to treat parasitic infections. As a result, drugs like quinine and artemisinin were isolated from herbs and barks (Varughese *et al*. 2010). *Sceletium tortuosum* has been used as medicine for social and spiritual purposes by San hunter gatherers and Khoi pastoralists. *Sceletium tortuosum* is rich in alkaloids, one of the important classes of natural product producing treatment for parasitic infections (Kayser *et al*. 2002).

Laboratory preparation of extracts of fresh *S. tortuosum* plant material was conducted mimicking traditional methods of preparation using organic solvents. Mesembrine was isolated from a methanol extract using conventional column chromatography. Sixteen extracts and mesembrine were evaluated for antiplasmodium activity using a plasmodium lactate dehydrogenase culture sensitivity assay with chloroquine as reference drug.

Of the sixteen extracts, four showed activity against *P. falciparum* with IC_{50} ranging between 1.47 µg/mL and 7.32 µg/mL. Extracts prepared from stored material at -20 °C showed no antiplasmodium activity. The four originally active extracts were re-screened six months later, but the antimalarial activity could not be reproduced. To determine discrepancy in biological results, chemical profiling of the extracts was done using high performance liquid chromatography technique. Differences were observed in the profiles of the active extracts when compared to those of stored plant material.

The instability of plant constituents observed could be a result of plant storage suggesting that the plant is best used when fresh.

Impact of HIV and AIDS on food security in Rufiji District, Tanzania

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How to cite this poster:
Kayunze, K.A., 2012, 'Impact of HIV and AIDS on food security in Rufiji District, Tanzania', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #482, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.482

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Relatively high prevalence of HIV and AIDS and food insecurity in Rufiji District whilst the linkage between the two problems was not known was the basis of this study. Data were collected amongst 225 households between November 2005 and October 2006 through participatory rural appraisal (PRA), household income and expenditure survey (HIES) and structured interviews. Binary logistic regression was used for analysis in which case the dependent variable was food security in terms of food insecure (0) and food secure (1) based on kilocalories consumed per adult equivalent per day. The independent variables included having been affected by HIV and AIDS in terms of not affected (0) and affected (1). The results reveal that the odds for households affected by HIV and AIDS to be food secure were 0.705 times as high as the odds for households not affected by HIV and AIDS to be food secure. This means that households affected by HIV and AIDS were less likely to be food secure as opposed to those not affected by HIV and AIDS. The B statistic for having been affected by HIV and AIDS was negative (B = -0.350) meaning that being affected by HIV and AIDS had negative impact on food security. However, the Wald statistic which shows the magnitude of impact was small (0.251) and not significant (p = 0.617). This shows that HIV and AIDS had little impact on food security. Based on these findings, it is concluded that although being affected by HIV and AIDS has negative impact on food security, it does not automatically make households food insecure, especially in a short run, and that some non-HIV and AIDS factors like high dependency ratio and low ability to buy food have bigger negative impact than that of HIV and AIDS on food security. On the basis of the conclusion, it is recommended that efforts to improve food security amongst households affected by HIV and AIDS should consider both HIV and AIDS and non-HIV and AIDS factors.

Immunogeno: Protective mechanism for Rift Valley fever in the Democratic Republic of Congo

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How to cite this poster:
Tshilenge, G., 2012, 'Immunogeno: Protective mechanism for Rift Valley fever in the Democratic Republic of Congo', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #483, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.483

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Rift Valley fever (RVF) is an acute, fever causing viral disease that affects domestic animals and humans.

In Democratic Republic of Congo (DRC), this pathology is not well documented. No epidemic of the RVF has not been reported but sera samples collected in six provinces surveyed from 2005 to 2006 revealed 14% of apparent prevalence and, high apparent prevalence (20%) of antibodies against RVF virus was reported in Katanga Province during the same survey; this serological evidence was associated with abortions cases in Cattle (Mulumba *et al*. 2009). Livestock immunisation is important for control of Rift Valley fever virus (RVFV) epidemics; however immunisation of susceptible domestic animals in endemic countries does not protect animals against the clinical disease but prevents the propagation of virus to human population through reduction of the amplification degree in host animals. The humoral immunity is sufficient for protection for animals as well as for humans. The infection caused by RVFV leads to neutralisation of the immunity of the animal (Barnard 1979).

Various immunological studies have been made on the characterisation of immune response during RVFV epidemics but, until now several studies have been concentrated on the response of the innate immune particularly based on signal interferon system than the response of the adaptive immune and cell mediated humoral immune. The available information on the immune response related to RVFV does not seem to provide enough information on various mechanisms of the response immune system.

The aim of the study is based on mechanism of immune response system including protective effect of immunisation against RVFV. In addition, epidemiological and molecular studies will be assessed. As a matter of fact, following studies will be conducted:

- evaluation of the immunological protection against Rift Valley fever in vaccinated and non-vaccinated cattle using IgG and IgM ELISAs in Katanga Province
- assessment of cellular response to Rift Valley fever disease in vaccinated and naturally infected cattle
- molecular characterisation of RVFV strains circulating in vaccinated and non vaccinated cattle
- assessment of protective effect related to vaccinal strains in cattle, using a longitudinal survey.

The studies will be carried out Northern Katanga Province within two areas, one with historyof circulation of RVFV and other without history RVFV circulation.

Whole blood, spleen, liver, lymph node will be collected as target tissues from cattle carcasses. In addition, goats and sheeps samples will be collected alongside from each area in order to clarify the disease situation. Serological tests based on the detection of Ig M and Ig G will be used. DIVA tests, LAMP, and IHC techniques will be used. Within previously vaccinated areas in the above mentioned areas and those that are not vaccinated, the collected samples will be analysed using RT-PCR/RT-LAMP.

In vitro experimental studies systems will be carried out using animal PMBCs that will be infected with wild type of RVF virus as well as with vaccinal strains, such as clones 13 and MP12 to characterise various cell types such as CD4 T cells, CD8 T cells, B-cells, NK cells and, macrophages will be studied with regard to activation and apoptosis signals on various post – infection days, using flow cytometry. A pool of animals will be vaccinated with the Clone 13 and another with the MP12 to determine the traceability. The monitoring of the immune response will be done through the measurement of immunoglobulin G (Ig G) and immunoglobulin M (Ig M). RT-PCR, spectrophotometer or Facs methods will be used for the dosage of cells T CD4 + and Cell T CD8+.

Investigation of water sources as reservoirs of *Vibrio cholerae* in Bepanda, Douala and determination of physico-chemical factors maintaining its endemicity

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How to cite this poster:
Tatah, A.J.-F.K., Pulcherie, K.M.C., Mande, N.L. & Akum, N.H., 2012, 'Investigation of water sources as reservoirs of *Vibrio cholerae* in Bepanda, Douala and determination of physico-chemical factors maintaining its endemicity', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #484, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.484

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Cholera remains a significant cause of mortality in developing countries. Outbreaks of the disease are associated with poverty, lack of potable water and poor sanitation. The survival and persistence of *Vibrio cholerae* in water has been shown to depend on physico-chemical factors. We studied water sources in Bepanda, an overcrowded neighbourhood in Douala, Cameroon, with limited access to portable water and very poor sanitary conditions as reservoirs of *V. cholerae*.

We analysed 318 samples from various sources (well, tap, stream) from February to July 2009 using standard microbiological techniques and characterised isolates serologically using the polyvalent O1/O139 antisera. Susceptibility to antibiotics previously used for cholera treatment in Douala was studied using the disk diffusion method. Physico-chemical factors (temperature, pH and salinity) that could maintain the endemicity of the organism were analysed using standard methods. Eighty-seven (27.4%) samples were contaminated, with high isolation rates being obtained from streams (52.4%) and wells (29.8%). The number of isolates was significantly higher ($P < 0.05$) in the rainy season (35.5%). We detected 23 (24%) O1 serogroup isolates in streams and wells, whilst 64 (66.6%) were non-O1/non-O139. Temperature and salinity correlated positively with the occurrence of the organisms. All isolates were susceptible to fluoroquinolones but high resistance rates to trimethoprim or sulfamethozaxole and tetracycline were observed.

Vibrio cholerae is endemic in Bepanda with O1 and non-O1/non-O139 serogroups co-existing in the streams and wells hence the possibility of future outbreaks of cholera if sanitation and drinking water quality are not improved. Temperature and salinity are amongst the factors maintaining the endemicity of the organism.

Cysticercosis in the Democratic Republic of Congo

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How to cite this poster:
Dorny, P., Kabwe, C., Kirezi, K., Lukanu, K., Lutumba, P., Maketa, V. *et al.*, 2012, 'Cysticercosis in the Democratic Republic of Congo', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #485, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.485

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Cysticercosis, caused by *Taenia solium* eggs, is a zoonotic disease whose consequences can be severe especially in the cerebral localisation (neuorcysticercosis). Indeed, neurocysticercosis is the first cause of epilepsy amongst the infectious etiology group. Following the increase of epilepsy cases in Kinshasa and Bas-Congo, it was important to assess the fraction attributable to neurocysticercosis especially as data on cysticercosis in Democratic Republic Of Congo (DRC) dating from 1970.

A joint study between veterinary and human doctors was conducted in the provinces of Bas-Congo and Kinshasa between 2008 and 2010. Blood samples were collected from the general population, patients with epilepsy and pigs. These samples were analysed using ELISA antigen in the laboratory of the Institute of Tropical Medicine in Antwerp. Patients positive to ELISA antigen took the CT scan exam for the confirmation of neurocysticercosis. In the province of Kinshasa, of 530 epileptic patients, 6.3% were identified as neurocysticercosis cases. Out of a total of 498 pigs, 38.9% were positive for cysticercosis. In the province of Bas-Congo, of 943 inhabitants from Malanga village, 21.6% were positive with predominance in males (26.4% versus 17.5%). A total of 145 pigs from 5 villages were examined and 41.2% found positive.

We can conclude that cysticercosis in the DRC has been neglected for a long time and cysticercosis could be a real major public health problem. Prospective studies addressing the consequences of cysticercosis in communities are needed in order to prevent epilepsy due to neurocysticercosis.

Unexpectedly low seroprevalence of toxoplasmosis in South Africa

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How to cite this poster:
Kistiah, K., Frean, J., Winiecka-Krusnell, J. & Barragan, A., 2012, 'Unexpectedly low seroprevalence of toxoplasmosis in South Africa', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #486, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.486

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Toxoplasmosis is an infection of warm-blooded vertebrates caused by the obligate intracellular protozoan parasite, *Toxoplasma gondii*. It is one of the most common parasitic diseases of humans, infecting approximately one-third of the world's population. In persons with advanced HIV, toxoplasmosis represents a major opportunistic infection of the central nervous system. Approximately two-thirds of all people living with HIV live in sub-Saharan Africa. In areas such as this, toxoplasmosis could theoretically pose a huge threat. There is little known about *T. gondii* prevalence in humans in Africa. Geographically, prevalences vary widely on this continent, as observed in other parts of the world. There is limited historical information about the disease in South Africa. More knowledge is needed at a regional level about the risk of toxoplasmosis, diagnostic issues, and measures to reduce the risk to susceptible persons. The seroprevalence of *T. gondii* in selected populations, namely HIV-positive and HIV-negative individuals, and a more general sample biased towards pregnant women, was therefore investigated and found to be 9.8% (37/376), 12.8% (48/376) and 6.4% (32/497) respectively. Compared with historical data from South Africa, the prevalence has decreased substantially; however, the incidence of clinical disease is unknown, despite the very high burden of HIV and AIDS cases (5.9 million and 0.7 million, respectively in 2009). This study provided information relating to the diagnosis and current seroprevalence of *T. gondii* in South Africa. Many questions still remain to be answered however, to fully understand the impact of this parasite on the country's population.

Co-infections of malaria and soil-transmitted helminths in localities with different levels of urbanisation in the Mount Cameroon region

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How to cite this poster:
Lum, E., Kimbi, H.K., Mbuh, J., Ndamukong-Nyanga, J., Njunda, A.L. & Lello, J., 2012, 'Co-infections of malaria and soil-transmitted helminths in localities with different levels of urbanization in the Mount Cameroon region', *Onderstepoort Journal of Veterinary Research* 79(2), Art. #487, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.487

Note:
Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011.

Malaria co-exists with intestinal helminths and they have different effects on infected individuals. A total of 235 and 208 children from Ekona and Great Soppo respectively of both sexes aged 4–14 years were enrolled into a cross-sectional study.

Capillary blood was collected for detection and determination of malaria parasitaemia as well as PCV. Stool samples were collected for quantitative determination of helminth ova by Kato-Katz technique.

The prevalence of malaria and helminths was higher in Ekona than Great Soppo. In Great Soppo, *Trichuris* was the most prevalent helminth than Great Soppo and an association was found between these co-infections. More children were co-infected in Ekona and co-infecting species were *Ascaris* and *Plasmodium falciparum*.

The prevalence of malaria and intestinal helminths as well as co-infection was lower in Great Soppo than in Ekona, probably due to increased urbanization in Great Soppo than Ekona.

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The work in the papers and abstracts was supported by Wellcome Trust Grant WT087546MA to the Southern African Centre for Infectious Disease Surveillance (http://www.sacids.org) and its collaborating UK partner institutions, namely the London School of Hygiene and Tropical Medicine (http://www.lshtm.ac.uk), the Royal Veterinary College (http://www.rvc.ac.uk) and the London International Development Centre (http://www.lidc.org.uk).

The work presented by Esron Karimuribo *et al*. was supported by Rockefeller Foundation.

How to cite this item: Proceedings of the Conference of the Southern African Centre for Infectious Disease Surveillance 'One Health' held at the National Institute for Communicable Diseases, Johannesburg, July 2011, *Onderstepoort Journal of Veterinary Research* 79(2), Art. #488, 1 page. http://dx.doi.org/10.4102/ojvr.v79i2.488