NATIONAL HEALTH LABORATORY SERVICE
NATIONAL INSTITUTE FOR COMMUNICABLE DISEASES

COMMUNICABLE DISEASES SURVEILLANCE BULLETIN

VOLUME 10. NO 1 APRIL 2012

## FOREWORD

Communicable diseases do not rest, at least not for long. Their causative agents often break out unexpectedly, challenging efforts to control them. Whether virus, bacterium, parasite or vector, they have remarkable adaptive plasticity. In this issue we look at outbreaks of viral diseases mild and bitter, the increasing incidence of multi-drug resistant tuberculosis (MDR-TB) and a novel method for controlling insecticide resistant malaria vector mosquitoes in South Africa. In all cases the need for increased vigilance, improved surveillance, improved diagnostic procedures and improved control are highlighted, showing that the public health sector also cannot rest.

Basil Brooke, Editor

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# VIRAL HAEMORRHAGIC FEVER OUTBREAKS, SOUTH AFRICA, 2011

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**Introduction**

Outbreaks of viral haemorrhagic fevers (VHF) are characterised by case-mortality rates of up to 90%. They occur rarely but have the potential to cause sizeable epidemics, especially in the healthcare setting. Increased international travel also increases the possibility of VHFs spreading across borders. Viral haemorrhagic fevers known to occur in Africa include Ebola, Marburg, Crimean-Congo haemorrhagic fever (CCHF), Rift Valley fever (RVF), haemorrhagic fever with renal syndrome (caused by hantaviruses from the family *Bunyaviridae*), Lassa fever, Lujo haemorrhagic fever and related arenaviral infections. Only CCHF and RVF are endemic to South Africa, but occasionally cases of other VHFs are imported into South Africa through infected persons seeking treatment in local hospitals.[1] Here we report on the occurrence of laboratory-confirmed VHF cases in South Africa in 2011, with a focus on an RVF outbreak during the period 2008 to 2011.

**Crimean-Congo haemorrhagic fever**

South Africa is endemic for CCHF. Earlier serosurveillance studies of domestic cattle herds indicated a high prevalence of the disease, with positivity rates of up to 96%.[2] Despite intensive circulation of the CCHF virus in livestock and wild vertebrates human cases of the disease in South Africa remain a rare occurrence, and the prevalence of antibody in rural human populations is generally low (<1-2%).[3]

Since 1981, human cases have been confirmed almost annually

at the National Institute for Communicable Diseases (NICD). Following nearly 30 years of passive surveillance, a total of 187 cases have been laboratory-confirmed. Although cases have been recorded from all nine of South Africa's provinces, the semi-arid regions of the Northern Cape and the Free State are most afflicted. In the majority of cases infections were likely acquired from tick bites or the squashing of ticks whilst contact with infected blood or tissues was also an important source of exposure. There is a strong link with occupational exposure because the majority of patients were males (n=151, 83%) employed in the livestock industry (e.g. farmers, farm workers, slaughtermen).

In 2011, no CCHF cases were confirmed from a total of 36 suspected VHF cases that were investigated. Tick bite fever and meningococcaemia were the most common diagnoses in these patients. In total, five CCHF cases were laboratory confirmed in South Africa during 2010, compared to only three cases in 2009 and eleven cases in 2008 (table 1).

| | Province | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Year** | **GP** | **FS** | **NC** | **WC** | **EC** | **MP** | **LP** | **NW** | **KZN** | **Number of cases** | **Number of fatal cases** | **Number of recovered cases** |
| 2000 | 1 | 3 | 2 | 0 | 1 | 0 | 0 | 1 | 0 | 8 | 5 | 3 |
| 2001 | 0 | 1 | 2 | 1 | 0 | 0 | 0 | 1 | 0 | 5 | 2 | 3 |
| 2002 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 1 | 2 |
| 2003 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 2004 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 2 | 0 | 4 | 2 | 2 |
| 2005 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 |
| 2006 | 2 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 8 | 4 | 4 |
| 2007 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 2008 | 0 | 3 | 5 | 0 | 1 | 1 | 0 | 1 | 0 | 11 | 2 | 9 |
| 2009 | 0 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 3 | 1 | 2 |
| 2010 | 0 | 3 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 5 | 1 | 4 |
| 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

Table 1: Laboratory confirmed Crimean-Congo haemorrhagic fever cases by province by year. No cases were confirmed in 2011. (GP: Gauteng Province; FS: Free State; NC: Northern Cape; WC: Western Cape; EC: Eastern Cape; MP: Mpumalanga; LP: Limpopo; NW: North West Province; KZN: KwaZulu-Natal.

**Rift Valley fever**

Rift Valley fever (RVF) was first reported in South Africa when a large outbreak occurred during 1950 and 1951.[4,5] Three more outbreaks with confirmed human cases occurred in South Africa in 1953, between 1974 and 1976, and in 1999.[6,8] Small clustered outbreaks occurred in South Africa in 2008 and 2009, followed by a large, widespread outbreak during 2010 and 2011.[7] Between 2008 and 2011, more than 2 000 specimens from suspected RVF cases were submitted to the NICD-NHLS for laboratory confirmation.

In 2008, a total of 17 non-fatal human RVF cases were laboratory-confirmed from the Mpumalanga, Limpopo and Gauteng provinces where outbreaks in animals had occurred. Additional animal cases of RVF occurred in the North-West Province, but no human cases were confirmed from there. Affected persons included cattle farmers, farm workers as well as veterinary staff and students from the University of Pretoria Veterinary Faculty where autopsies on infected animals were carried out.[7] Confirmed cases were recorded in the first half of 2008 only (figure 1). One of the veterinary students developed a biphasic illness with late-onset encephalitis, a complication occasionally noted in RVF patients.

In 2009, outbreaks occurred in KwaZulu-Natal and the Northern Cape. Seven human cases in total were confirmed (figure 1). Five of these cases were infected before the onset of winter in KwaZulu-Natal. The remaining two cases from the Northern Cape were infected at the start of spring. No fatalities were recorded during

these outbreaks. In 2010, a total of 241 human cases were confirmed of which 25 proved fatal. The outbreaks were geographically linked with outbreaks in animals and occurred mostly cross the inland plateau of the country, specifically the Free State, Northern Cape, North-West, Eastern Cape and Western Cape provinces. The first animal RVF case was confirmed on 12th February 2010 in the eastern areas of the Free State. The first human case was confirmed by real-time RT-PCR and virus isolation on 17th February 2010 from the same area. The largest number of human cases and fatalities were subsequently reported from this province. The human RVF outbreak peaked in March 2010 when more than 100 cases were laboratory confirmed. Cases were continuously recognized up until September, with no cases recorded in October and November 2010. New cases were laboratory confirmed during the period of December 2010 to March 2011 (figure 1). A total of 13 902 animal cases were confirmed during the same period with 8 581 deaths. Animal cases consisted mostly of domestic livestock (predominantly sheep but also cattle and goats), although some game animals (buffalo, sable, nyala etc.) including exotics (alpaca, llama and Asian buffalo) were also infected (Data source: Directorate of Veterinary Services, Department of Agriculture, Forestry and Fisheries).

During the first half of 2011, RVF cases continued to be confirmed from the Free State, Northern Cape, Eastern Cape and Western Cape provinces, peaking again in March. The largest number of confirmed human cases was from the Eastern Cape. No human fatalities were recorded among the 37 confirmed cases during 2011 (figure 1). The outbreak among the human population seems to have ended in 2011, with the last case confirmed in May of that year. No new cases have been recorded in 2012 to date. In total, 302 human cases were confirmed from 2008 to 2011.

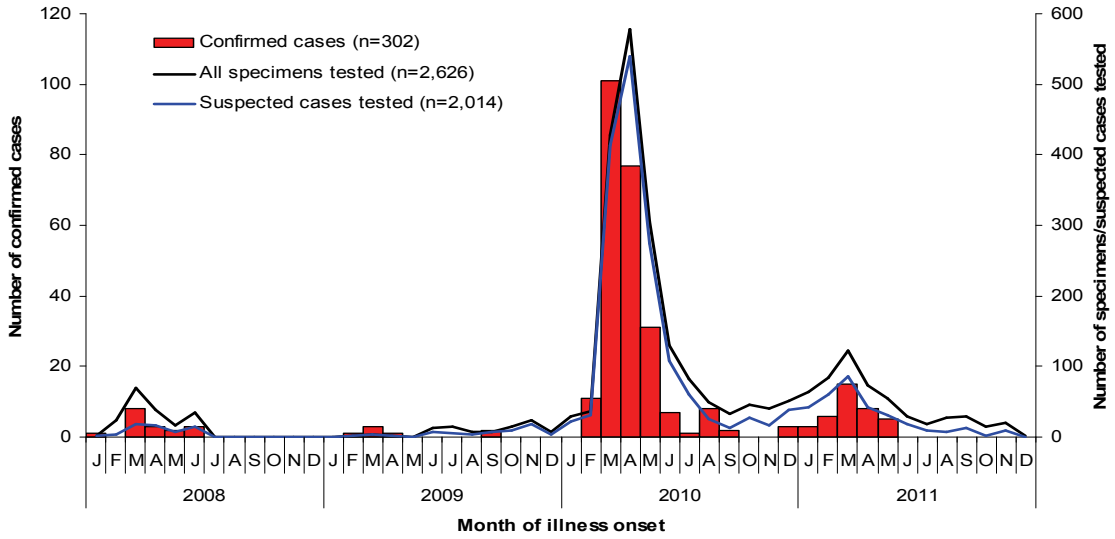


Figure 1: Epidemiological characteristics of the 2008 to 2011 Rift Valley fever outbreaks in humans, South Africa.

The number of animal cases, human cases and deaths from the provinces were directly proportional, suggesting that most humans were infected through direct contact with infected animals. Indeed, 254 (89%) of the confirmed cases reported a history of contact with animal tissues or bodily fluids. A total of 242 (83%) cases reported working in occupations with likely exposure to animals and their products. All fatal cases occurred in 2010 (case fatality ratio 10%), whilst the overall case fatality ratio from 2008 to 2011 was 8%.

The 2008 and 2009 outbreaks were caused by viruses from lineage C of the RVFV phylogenetic tree, whereas the 2010 and 2011 outbreaks were caused by viruses closely related to a Namibian isolate from 2004 on lineage H.[9] One isolate from the 2010 outbreak was genetically distinct from other isolates of the same outbreak, and was closely related to the Smithburn neurotropic vaccine strain. The patient, a veterinarian from whom this isolate was obtained, experienced a needle stick injury while vaccinating livestock that were likely already infected by wild-type circulating virus. This isolate is therefore likely a reassortant of the wild-type and the vaccine RVFV strains.[9]

Laboratory confirmation of RVF cases requires the use of laboratory tests targeting different analytes (antibody, antigen, and nu-

cleic acid). The Centre for Emerging and Zoonotic Diseases (CEZD - formerly the Special Pathogens Unit), NICD-NHLS, uses a spectrum of laboratory tests which were developed and validated in-house over several years.[10,11] Isolation of live virus from clinical specimens requires the use of suckling mice, which is the most sensitive method available for the isolation of arthropod borne viruses. Less sensitive tissue culture can also be used as an alternative because of the degree of viraemia of RVFV in the host. The isolation of live virus is the gold standard for diagnosis of a current infection. Nucleic acid techniques used by CEZD during the outbreaks include a hydrolysis probe-based real-time reverse transcription polymerase chain reaction (real-time RT-PCR) and a loop-mediated isothermal amplification (LAMP). Detection of viral nucleic acid is indicative of a current infection, but does not necessarily indicate the presence of the replicating virus. Nucleic acid detection methods are very sensitive, but their disadvantages include possible cross-contamination (i.e. false positives) and reaction inhibition due to inhibitory factors in clinical material (i.e. false negatives). Detection of antibodies specifically against RVF virus can be used to indicate current/recent (IgM) or past infections (IgG). The number of days between onset of symptoms and the taking of samples post infection from the patient is a determining factor in which test(s) would yield positive results. This is a consequence of the short viraemia during RVFV infection and the transient nature of the IgM response. Because the time of infection in the field cannot specifically be determined, and because of inherent variation in the responses of individuals to infection, it is best to conduct a battery of tests detecting different analytes for RVF diagnosis. Ideally clinicians should also submit paired serum samples, taken during the acute and convalescent phases to show a four-fold increase in IgG titres for RVF confirmation. Table 2 shows the number of human cases confirmed by different laboratory techniques during the 2010 and 2011 outbreak. The largest proportion of cases was confirmed by both PCR and virus isolation, followed by serology (IgM). Small numbers of cases were confirmed by other methods or combinations, indicating the importance of testing multiple analytes.

| Assay | PCR only | PCR + isolation | PCR + IgM | PCR + Isolation + IgM | Isolation + IgM | IgM only | Isolation only |
|---|---|---|---|---|---|---|---|
| Number of confirmed cases | 15 | 129 | 4 | 16 | 3 | 106 | 5 |

Table 2: Laboratory confirmation of human Rift Valley Fever cases by method, 2010 to 2011.

As a consequence of increased public awareness during the RVF outbreak, large numbers of specimens were submitted for laboratory analysis. Only a small percentage of these proved to be RVF-positive, but testing of these specimens for other arthropod-borne viral infections as a part of differential diagnosis revealed, as expected, exposures to other endemic arboviruses. For example, in 2010 a total of 193 patients were found to have IgM antibodies against West Nile virus, and 207 patients had IgM antibodies against Sindbis virus. These data suggest that some patients were infected with arboviruses transmitted to them during periods of increased mosquito activity.

**Imported VHF cases**

No additional cases of viral haemorrhagic fever were confirmed during 2011. Since RVF and CCHF viruses are the only VHF agents endemic to South Africa, other cases would most likely have been imported from neighbouring or other African countries. For example, until recently, Lassa virus was the only known arenavirus to cause VHF in humans in Africa. The importation of a previously unrecognized arenavirus to South Africa following evacuation of a critically ill patient from Zambia in September 2008 resulted in a dramatic VHF nosocomial outbreak in Johannesburg with a case fatality rate of 80%. International collaboration during this outbreak allowed for rapid identification of the novel virus (provisionally named Lujo virus), thus reassuring the public and public health authorities that medical research communities have powerful tools to rapidly detect and respond to the challenges of unknown pathogens. Nevertheless, the history of the outbreak shows that cross-border transfers of patients can lead to the unintentional spread of dangerous pathogens with dramatic public health consequences.[12]

**Conclusion**

RVF and CCHF have caused widespread disease in South Africa. Furthermore, there remains an ongoing risk of importation of VHFs with a high potential for subsequent local spread. It is therefore particularly important that healthcare professionals throughout South Africa maintain a high index of suspicion for suspected VHF

cases, which often do not present with bleeding. Rapid implementation of infection control measures, including full barrier precautions, is life saving. It is vital that a thorough history is obtained from all patients, which should include accurate information concerning town and country of origin, as well as details of occupation and activities in that country. This information, together with a detailed clinical history, will inform subsequent laboratory testing. All suspected VHF cases require an immediate telephonic notification to the local Department of Health, and should be reported to the NICD-NHLS doctor on call (Tel. +27-82-883-9920) prior to sending specimens.

**Acknowledgements**

The authors would like to thank the technical staff of the Centre for Emerging and Zoonotic Diseases, Special Viral Pathogens Reference Laboratory for their contributions.

**References**

1. Archer AN, Blumberg LH, Jansen van Vuren P, Kemp A, Leman PA et al. Viral haemorrhagic fever outbreaks in South Africa, 2007-2009. *Communicable Disease Surveillance Bulletin*, National Institute for Communicable Diseases 2009; 15-16.
2. Swanepoel R, Burt FJ. Crimean-Congo haemorrhagic fever. In: *Infectious Diseases of Livestock*. 2nd Edition. Coetzer JAW, Tustin RC (eds). Oxford University Press 2004; 1077-1086.
3. Swanepoel R, Paweska JT. Crimean-Congo haemorrhagic fever. *Oxford Textbook of Zoonoses Zoonoses: Biology, Clinical Practice, and Public Health Control*, 2ed edition (eds. Palmer SR, Soulsby L, Torgerson PR and Brown DWG), Oxford University Press 2011; 287-293.
4. Mundel B, Gear J. Rift Valley fever; 1. The occurrence of human cases in Johannesburg. *S Afr Med J* 1951; 25: 797-800.
5. Joubert JD, Ferguson AL, Gear J. Rift Valley fever in South Africa: 2. The occurrence of human cases in the Orange Free State, the north-western Cape Province, the western and southern Transvaal. A. Epidemiological and clinical findings. *S Afr Med J* 1951; 25: 890-891.
6. Van Velden DJJ, Meyer JD, Olivier J, Gear JHS, McIntosh B. Rift Valley fever affecting humans in South Africa. *S Afr Med J* 1976; 51: 867-871.
7. Archer BN, Weyer J, Paweska J, Nkosi D, Leman P, Tint KS, et al. Outbreak of Rift Valley fever affecting veterinarians and farmers in South Africa, 2008. *S Afr Med J* 2011; 101: 263-6.
8. Anonymous. 1999 – Rift Valley fever in South Africa. WHO Global Alert and Response, 10 February 1999. http://www.who.int/csr/don/1999_02_10/en/index.html (Accessed 16/04/2012).
9. Grobbelaar AA, Weyer J, Leman PA, Kemp A, Paweska JT, Swanepoel R. Molecular epidemiology of Rift Valley fever virus. *Emerg Infect Dis* 2011; 17: 2270-2276.
10. Le Roux CA, Kubo T, Grobbelaar AA, Jansen van Vuren P, Weyer J, Nel LH, Swanepoel R, Morita K, Paweska JT. Development and evaluation of a real-time reverse transcription-loop-mediated isothermal amplification assay for rapid detection of Rift Valley fever virus in clinical specimens. *J Clin Microbiol* 2009; 47: 645-651.
11. Paweska JT, Burt FJ, Swanepoel R. Validation of IgG-sandwich and IgM-capture ELISA for the detection of antibody to Rift Valley fever virus in humans. *J Virol Methods* 2005; 124: 173-181.
12. Paweska JT, Sewlall NH, Ksiazek TG, Blumberg LH, Hale MJ, Lipkin IW, Weyer J, Nichol ST, Rollin PE, McMullan LK, Paddock CD, Briese T, Mnyaluza J, Dinh T-H, Mukonka V, Ching P, Duse A, Richards G, de Jong G, Cohen C, Ikalafeng B, Mugero C, Asamugha C, Malotle MM, Nteo DM, Misiani E, Swanepoel R, Zaki SR and Members of the Outbreak Control and Investigation Teams. Nosocomial outbreak of novel arenavirus infection, southern Africa, *Emerg Infect Dis* 2009; 15: 1598-1602.

# ARBOVIRUS SURVEILLANCE: A SHORT REPORT ON TWO HUMAN CASES OF WESSELSBRON DISEASE IN SOUTH AFRICA

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**Introduction**

Wesselsbron virus (WESSV), an arthropod-borne flavivirus, causes abortions in sheep and goats, mortality of newborn lambs and goat kids, as well as neuropathy and mortality in horses.[1,2] Wesselsbron virus has been identified in a wide range of vertebrate hosts across Africa. These include cattle, goats, sheep, gerbils, wild ruminants, camels, pigs, donkeys and horses from localities in Angola, Botswana, Cameroon, Central African Republic, Cóte d'Ivoire, Kenya, Madagascar, Mozambique, Namibia, Nigeria, Senegal, South Africa, Uganda, and Zimbabwe. This virus has also been isolated in a limited number of mosquito species including *Aedes caballus, A. circumluteolus, A. juppi, A. mcintoshi* and *Mansonia uniformis*.[3] When rainfall patterns result in the inundation of grasslands and the formation of extensive ephemeral pans, many of the ''floodwater'' mosquitoes breed prolifically. Among these are *A. circumluteolus* and *A. mcintoshi* which are known vectors of arboviruses such as Rift Valley fever virus (*Bunyaviridae*), Middelburg virus (*Togaviridae*) and Wesselsbron virus (*Flaviviridae*).[4] It is then not surprising that, historically, reported cases of WESS disease have coincided with those of Rift

Valley fever (RVF), as both causative agents are transmitted by the same species and share the same biological niche.

**This study**

Climatic conditions in many parts of South Africa precipitated an extensive RVF outbreak beginning in 2008 and peaking in April 2010. By September 2010, a reported total of 13 902 animal cases with 8 581 deaths, and 241 laboratory confirmed human cases with 25 deaths were recorded.[5] Specimns from suspected human RVF cases that were submitted to the Centre for Emerging and Zoonotic Diseases for laboratory confirmation yielded two cases of WESS disease. Typically, suspected arbovirus cases are routinely screened for South Africa's most common arboviruses. Laboratory confirmation relies on the submission of blood specimens (preferably acute and convalescent phase sera) which is then subject to a battery of tests including virus isolation in cell culture and in suckling mice, immunofluorescent- and haemagglutination-inhibition assays, ELISAs, RT-PCR, viral genome sequencing, and electron microscopy, as each case requires. In the two WESSV cases in question, live virus was isolated from serum specimens from suspected RVF cases, by inoculation into suckling mice (animal ethics clearance number 107/06). These isolates where characterized by electron microscopy, PCR and molecular sequence analysis. Detailed methodologies are described elsewhere.[6] Case details for the laboratory-confirmed WESSV cases were obtained telephonically (ethics protocol reference number M060449, Human Research Ethics Committee, University of the Witwatersrand).

Wesselsbron virus is morphologically typical of members of the *Flaviviridae*, in being 45-55nm in diameter with a tightly-fitting envelope (figure 1). RT-PCR and partial sequencing analysis of the NS5 gene of the isolates confirmed the diagnosis of WESSV infection (results not shown).

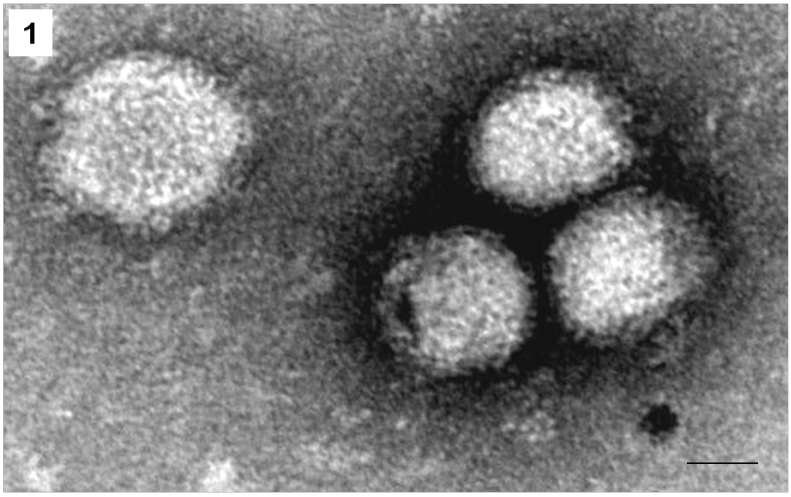


Figure 1: Transmission electron micrograph of Wesselsbron virus particles from cultured isolate, negatively-stained with 0.5% uranyl acetate and viewed at 80kV on an FEI BioTwin Spirit TEM. Scale bar = 20nm.

Both of the laboratory-confirmed WESSV cases were male sheep farmers, one from the North West Province and the other from the Northern Cape. In both cases symptoms included an acute, febrile illness of 6 to 10 days, with headache, myalgia and arthralgia. The flu-like illness was cleared with symptomatic treatment and no sequelae were experienced.

**Discussion**

The true burden of WESS disease is not understood. Infections are almost certainly under-reported and under-detected due to the mildness of human disease associated with WESSV, as well as a lack of surveillance in animal populations. It is noteworthy that although WESS disease cases are considered mild and self-resolving, the two cases reported here were severe enough to warrant medical consultation.

Since 1955, when the virus was first described[7], and excluding the 2 cases reported here, only 29 acute cases have been confirmed (table 1). All of these involved laboratory workers or field workers on arbovirus research fieldtrips. Infections were either acquired from exposure to live virus preparations in the laboratory, from exposure to infected animals or from mosquitoes – so the risk factors are similar to those for RVFV.

| Locality | Case history | Reference |
|---|---|---|
| Wesselsbron, Free State, South Africa | Five laboratory workers from veterinary laboratory with exposure to 1955 WESS outbreak isolates reported illness. Single isolation and four seroconversions were noted. | Weiss et al, 1956 |
| Lake Simbu, Tongaland (now Kosi Bay, KwaZulu Natal Province, South Africa) | 35 year old male mosquito collector during field expedition in 1955. Virus isolated (strain H177) from blood. | Smithburn et al, 1957 |
| Lake Simbu, Tongaland (now Kosi Bay, KwaZulu Natal Province, South Africa) | Field worker (no details provided) in 1955. No virus isolated, but seroconversion indicated. | Smithburn et al, 1957 |
| Middelburg, Northern Cape Province, South Africa | Two field workers in 1957. Both were bitten by mosquitoes but one also performed necropsies on sheep. Virus isolated from one patient and only seroconversion recorded in the other. | Heymann et al, 1958 |
| Entebbe, Uganda | Laboratory acquired infection in 1959. Infected through splash in the eye. | Weinbren, 1959 |
| Dakar, Senegal | Laboratory acquired infection in 1965 | Swanepoel, 1989 |
| Ndumu, KwaZulu Natal, South Africa | Field worker with exposure to mosquitoes in 1966 | Swanepoel, 1989 |
| New Haven, United States of America | Laboratory-acquired infection in 1969. Possible aerosol transmission noted. Live virus isolated from throat swab. | Justines and Shope, 1969 |
| Ibadan, Nigeria | Laboratory-acquired infection in 1972. Patient was exposed to mosquito suspensions prepared for virus isolation and challenge virus used for neutralization assays. | Tomori et al, 1981 |
| Pienaars River, Limpopo, South Africa | Laboratory field worker exposed to mosquitoes in 1972. | McIntosh, 1980 |
| Bloemfontein, Free State, South Africa | Circumstances of infection not clear - 1974. | McIntosh, 1980 |
| Bangui, Central African Republic | Laboratory acquired infection in 1974. | Swanepoel, 1989 |
| Johannesburg, Gauteng, South Africa | Circumstances of infection not clear - 1976. | McIntosh, 1980 |
| Bangui, Central African Republic | 9 cases including one laboratory-acquired infection from 1981-1983. | Swanpoel, 1989 |
| Dakar, Senegal | Circumstances of infection not clear - 1983. | Swanepoel, 1989 |
| Bultfontein, Free State, South Africa | Field scientist infected while collecting mosquitoes in April 1996. | Jupp and Kemp, 1998 |

Table 1: Summary of documented human Wesselsbron disease cases prior to this study.

Both WESSV cases described here involved sheep farmers. Although arboviral diseases, including WESS disease, are typically not associated with significant human mortality, the associated morbidity may be substantial. In addition, anthropogenic influences such as global climate change, which alter vector habitats, are thought to be the primary driving factor behind the emergence of arboviral diseases globally. It is therefore important that arboviral surveillance is maintained and improved. A revealing example is the reporting of severe encephalitis in two immunocompromised adults in Europe, caused by Usutu virus disease.[8] Usutu virus is another African mosquito-borne *Flavivirus*, but with predominantly avian hosts. Migratory birds and global warming have combined to permit the establishment of this virus as a resident pathogen in the northern hemisphere, where it is causing disease in humans and extensive mortality in bird populations.[9]

**Acknowledgements**

The authors would like to thank the technical staff of the Centre for Emerging and Zoonotic Diseases, Special Viral Pathogens Reference Laboratory for their contributions.

**References**

1. Swanepoel R, Coetzer JAW. Wesselsbron disease. In: Coetzer JAW, Tustin RC, eds. *Infectious diseases of livestock*. Second edition. South Africa, Oxford University Press, 2004; 987-994.
2. Human S, Gerdes T, Williams J, Leman P, Paweska J, Steyl J, Stroebel J, Venter M. Molecular epidemiology and characterisation of Wesselsbron virus and co-circulating alphaviruses in sentinel animals in South Africa. *International Symposia on veterinary Epidemiology and Economics (ISVEE) proceedings, ISVEE* 2009; 12: 253.
3. Swanepoel R. Wesselsbron virus disease. In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Boca Raton, CRC Press Inc, 1989; 32-57.
4. Jupp PG. *Mosquitoes of Southern Africa*. Ekogilde Publishers, South Africa, 1996.
5. Special Pathogens Unit. National Institute for Communicable Diseases Annual Review 2010-2011; 33-37.
6. Weyer J, Thomas J, Leman PA, Grobbelaar AA, Kemp A, Paweska JT. Human cases of Wesselsbron disease, South Africa 2010-2011; (in prep).
7. Weiss KE, Haig DA, Alexander RA. Wesselsbron virus – a virus not previously described associated with abortion in domestic animals. *Onderstepoort Journal of Veterinary Research* 1956; 27(2): 183-195.
8. Nikolay B, Diallo M, Boye CS. Usutu virus in Africa. *Vector Borne Zoonotic Dis.* 2011; 11: 1317-1323.
9. Vazquez A, Jimenez-Clavero M, Franco L, Donoso-Mantke O, Sambri V, Niedrig M, Zeller H, Tenorio A. *Usutu virus: potential risk of human disease in Europe*. Euro Surveill. 2011; 16(31). pii:19935.

# EMERGENCE OF RABIES IN SOUTH AFRICA, 2005-2011

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**Introduction**

Rabies is a zoonotic disease caused by infection with the bullet-shaped lyssaviruses. Globally, the "classic" rabies virus (formerly referred to as genotype one lyssavirus) cause the vast majority of animal and human rabies cases. The domestic dog is the principle vector of rabies virus to humans in those developing countries where rabies occurs. Although accurate statistics are not available, a study by the World Health Organization conservatively estimated that 50 – 70 000 human cases occur per annum.[1] Rabies is considered the deadliest viral infection known, with a mortality rate of 100%.

In South Africa, rabies has been reported since the 1800s. Incidental introductions during the colonial period were recorded, but do not appear to have produced sustained transmission cycles. Rabies in herpestid species, especially the yellow mongoose, was probably introduced into South Africa roughly 200 hundred years ago.[2] Canine rabies has been introduced on multiple occasions, probably most significantly during the late 1970s in KwaZulu-Natal.[3] Human cases have been confirmed annually at the National Institute for Communicable Diseases since 1983.[4] In 2011, six cases of human rabies were laboratory confirmed. These originated from Limpopo (n=3), KwaZulu-Natal (n=2) and the Eastern Cape (n=1).

**Outbreak of rabies in Limpopo Province**

In 2005 a marked increase in confirmed rabies cases in domestic dogs was recorded from Limpopo, mostly from the northern Vhembe district. The cases increased from less than ten confirmed cases per annum during the period 1994-2004, to 35 cases in 2005 and 100 cases in 2006.[5] A concomitant rise in the number of cases in black backed jackals and cattle was also noted during

this period. A total of 22 human cases were confirmed in 2006.[5] Increased travel between Zimbabwe and South Africa and a deterioration of veterinary resources in Limpopo are implicated as contributory factors to the cause of the outbreak. Molecular sequencing of rabies virus isolates collected during the outbreak indicates that the virus was likely introduced into Limpopo from Zimbabwe.[5]

Initial control efforts in Limpopo did reduce the number of detected cases, but single cases are still diagnosed every year. Since 2006, a further 11 cases have been laboratory confirmed, all acquired in the Vhembe district.

**Spread of rabies in Mpumalanga Province**

The Mpumalanga Province borders on Mozambique and Swaziland, both of which are reportedly severely afflicted by rabies. Rabies has therefore commonly been associated with the eastern and southern borders (most significantly the Nkomazi district) of Mpumalanga, but was under control in the rest of the province. In 2008, a rise in the number of confirmed rabies cases was recorded in the interior, spreading northwards. Molecular sequencing of rabies virus isolates from the outbreak supported the hypothesis of a spread of the disease from the Nkomazi district to the rest of the province[6]. Since 2008, four human cases of rabies have been laboratory confirmed and a further three suspected cases noted. Previous human cases from Mpumalanga were diagnosed in 1985 and 1986.

**Outbreak of rabies in Gauteng Province, 2010-2011**

From January 2010 to June 2011, 49 positive rabies cases were confirmed in dogs mainly from the south-western parts of Johannesburg (n=49) and from Pretoria (n=2), both regions previously considered to be rabies-free. The peak of the outbreak was recorded in October 2010, with 15 confirmed animal (primarily dogs) cases. One human case was confirmed in a 26-month old male that was scratched by a pet puppy one month prior to onset of the illness. Molecular sequencing of the G-L intergenic region of rabies virus isolates obtained from dogs and from the 26-month old male indicated an introduction of rabies from KwaZulu-Natal.[7] This outbreak marks the first local spread of rabies in domestic dogs and the first locally acquired human rabies case in Johannesburg.

**Conclusion**

Rabies has emerged (or re-emerged) at several localities in South Africa where it was previously under control. These outbreaks highlight the importance of continued vigilance for rabies in South Africa and should encourage sustained efforts at control. Human rabies cases invariably follow outbreaks of the disease in domestic dogs. Therefore, control of the disease in dogs, through thorough and strategic vaccination programs, is the mainstay of rabies prevention in humans.

**Acknowledgements**

The authors would like to thank the technical staff of the Centre for Emerging and Zoonotic Diseases, Special Viral Pathogens Reference Laboratory for their contributions.

**References**

1. Knobel DL, Cleaveland S, Coleman PG, Fevre EM, Meltzer MI, Miranda EG, Shaw A, Zinsstag J, Meslin F. Re-evaluating the burden of rabies in Africa and Asia. *Bull World Health Organ* 2005; 83: 1-13.
2. Van Zyl N, Markotter W, Nel LH. Evolutionary history of African mongoose rabies. *Virus Res* 2010; 150: 93-102.
3. Swanepoel R. Rabies. In: Coetzer JAW, Tustin RC. (eds.), *Infectious diseases of livestock: with special reference to Southern Africa*. Oxford University Press, Cape Town. 2004: 1121-1184.
4. Weyer J, Szmyd-Potapczuk AV, Blumberg LH, Leman PA, Markotter W, Swanepoel R, Paweska JT, Nel LH. Epidemiology of human rabies in South Africa, 1983 – 2007. *Virus Res* 2011; 155: 283-290.
5. Cohen C, Sartorius B, Sabeta C, Zulu Gugulethu, Paweska J et al. Epidemiology and molecular virus characterization of reemerging rabies, South Africa. *Emerg Infect Dis* 2007; 13 (12): 1879-1886.
6. Mkhize GC, Ngoepe EC, Du Plessis BJA, Reininghaus B, Sabeta CT. Re-emergence of dog rabies in Mpumalanga Province, South Africa. *Vector borne Zoonotic Dis* 2010; 10: 921-926.
7. Sabeta C, Geertsema P, Mohale D, Miyen J, Weyer J et al. Emergence of rabies in Gauteng Province, South Africa. (in prep).

# NEWLY DIAGNOSED MULTI-DRUG RESISTANT TUBERCULOSIS IN GAUTENG, SOUTH AFRICA, 2004 TO 2010

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**Background**

Multi-drug resistant tuberculosis (MDR-TB), defined as resistance to isoniazid and rifampicin, is a significant global health problem.[1] South Africa is among the 27 high MDR-TB burden countries (i.e. countries estimated to have had at least 4000 MDR-TB cases arising annually and/or at least 10% of newly registered TB cases with MDR-TB) that collectively account for about 85% of the world's cases of MDR-TB.[2] It has the largest burden of MDR-TB in Africa, with about 9,070 laboratory-confirmed diagnoses in 2009.[3] The escalation of TB cases is largely driven by co-infection with human immunodeficiency virus (HIV), resulting in an ever increasing pool of TB patients from which MDR-TB cases arise. In Gauteng, the smallest and most densely populated province in South Africa[4], the prevalence of MDR-TB between 2001 and 2002 was 1.4% among patients with newly diagnosed and culture-positive TB, and 5.5% among patients previously treated for TB.[5] The number of MDR-TB cases has increased substantially since the last survey, and is worsened by the HIV epidemic.[6] The aim of the study is to describe the epidemiology of MDR-TB in Gauteng province, based on available surveillance data captured in the Corporate Data Warehouse (CDW) of the National Health Laboratory Service (NHLS) from 2004 to 2010.

**Methods**

A descriptive analysis of laboratory-based surveillance data, obtained from the Centre for Tuberculosis of the National Institute for Communicable Diseases (NICD), was performed. All new MDR-TB patients diagnosed at NHLS laboratories in Gauteng between 2004 and 2010, who attended government health facilities, were included in the study. For MDR-TB detection, the conventional drug susceptibility testing (DST) method used was the Mycobacteria Growth Indicator Tube (MGIT) system, BD BACTEC™ MGIT™ 960 (Becton Dickinson, Sparks, Md, USA), while with the introduction of the rapid molecular line probe assay (LPA), some MDR-TB cases were diagnosed by the GenoType® MTBDR*plus* (Hain Life science, GMBH, Nehren, Germany) LPA. Data analysis was performed using Excel, EpiInfo and Stata statistical software.

**Results**

A total of 6437 records of newly diagnosed MDR-TB patients during the period 2004 to 2010 were analyzed. The main patient demographics are given in table 1.

The number of MDR-TB cases increased from 591 in 2004 to 1100 in 2010, with steep increases in 2007 and 2009, as shown in figure 1.

Over the seven-year period of the analysis, the majority of the cases by far occurred in the 25-44 years age group, followed by those within the 45-64 years group. A substantial number of MDR-TB cases were diagnosed in the less than 5 age group during this period (figure 2).

Figure 3 shows the distribution of cases by gender, with greater numbers of males being newly diagnosed with MDR-TB up to 2009.

Geographically, there were more cases in the urban and industrialized districts (City of Johannesburg, City of Tshwane and Ekurhuleni Metro), as shown in figure 4.

Table 1: Gender and age range of newly diagnosed MDR-TB patients, 2004 to 2010.

| Variable | Number | % |
|---|---|---|
| Gender* | | |
| Male | 3419 | 55 |
| Female | 2787 | 45 |
| Age (years)* | | |
| Range | 1-88 | |
| Mean (SD) | 35.9 (±11.4) | |

**Note: missing values for sex = 231; age = 32*

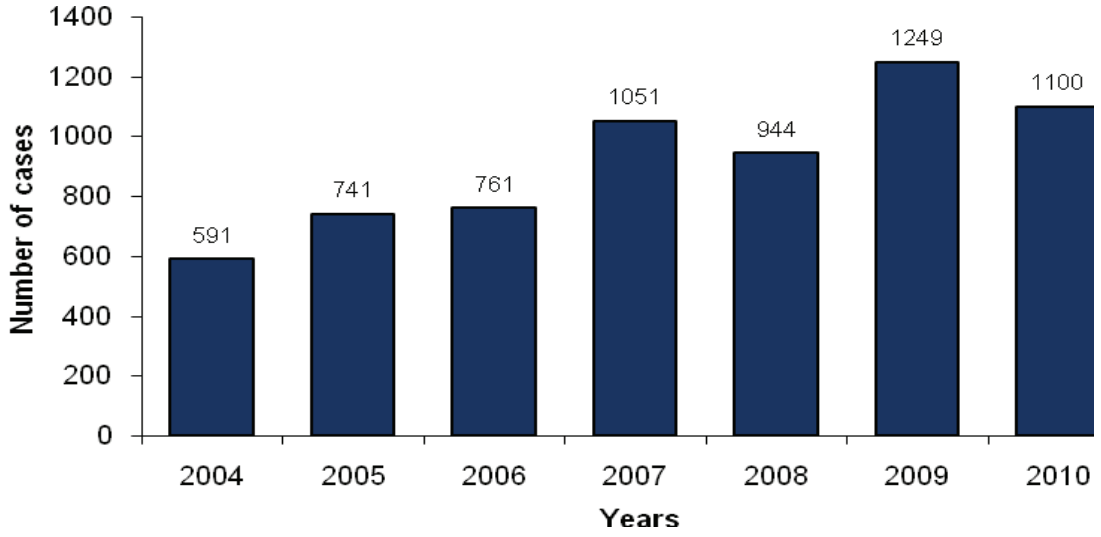


Figure 1: Newly diagnosed MDR-TB cases, Gauteng.

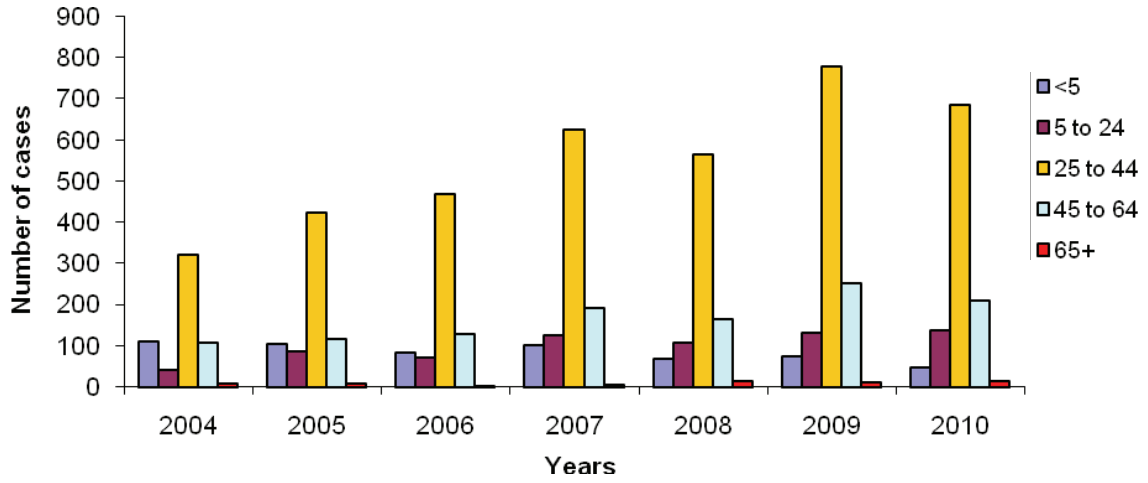


Figure 2: Age-group distribution of newly diagnosed MDR-TB cases, Gauteng.

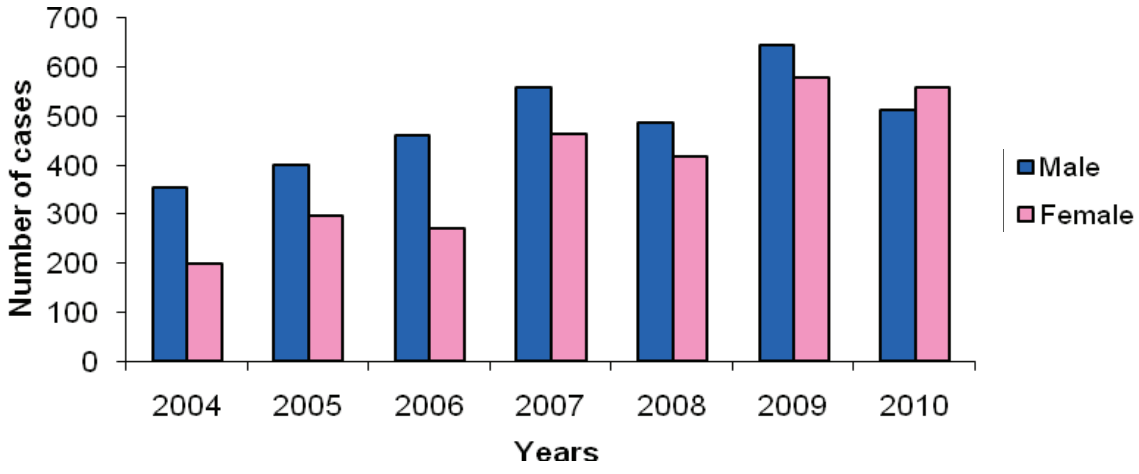


Figure 3: Gender distribution of newly diagnosed MDR-TB cases, Gauteng.

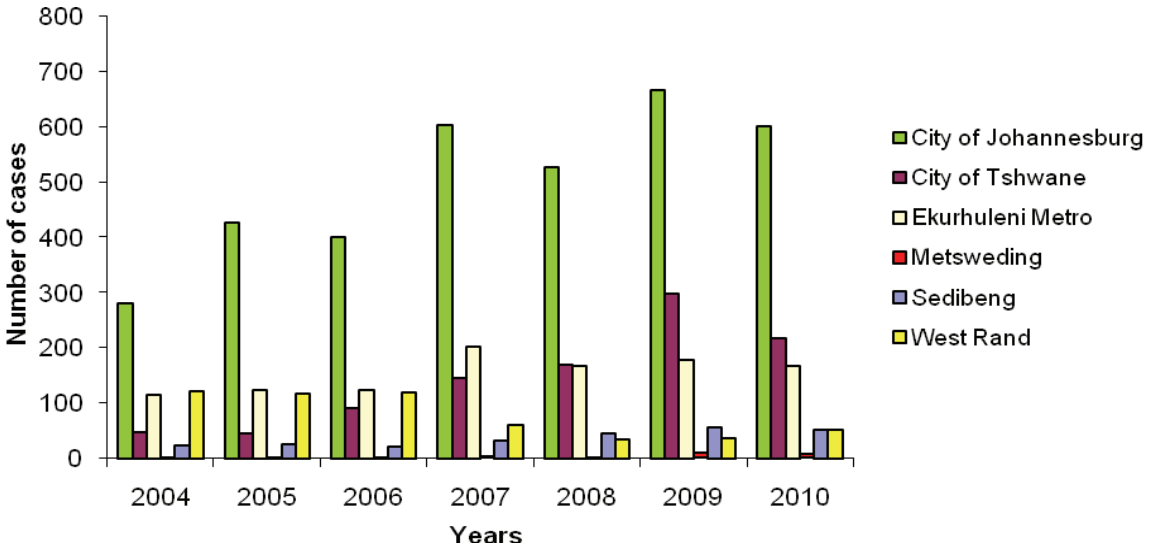


Figure 4: Newly diagnosed MDR-TB cases by health district, Gauteng.

**Discussion**

Tuberculosis has socio-economic consequences as reinforced by this study which shows that the economically active 25-44 years age group was the most affected by MDR-TB. Furthermore, the moderate predominance of affected males, who are most likely breadwinners, will have exerted an adverse effect on the socio-economic wellbeing of families.

The prevalence and incidence rates of TB, at all ages, are higher in males than in females, except in childhood, when they are higher in females. Differences in risk of exposure to infection, health seeking behaviour, economic consequences and stigma are some of the associated risk factors.[7]

In South Africa, there has been a steady increase in the number of MDR-TB cases since 2004, possibly due to increased case detection.[8] Between 2001 and 2002, the national prevalence of MDR-TB was 1.6% among newly diagnosed TB patients and 6.6% among those with previously treated TB.[9] The 2002-2007 drug resistance survey of new TB cases and previously treated cases showed slight increases of MDR-TB to 1.8% and 6.7% respectively.[10]

The densely populated City of Johannesburg, home to MDR–TB patients (Sizwe hospital), recorded the largest number of MDR-TB cases in Gauteng. The occurrence of extensively drug-resistant tuberculosis (XDR-TB) in KwaZulu-Natal very likely induced increased laboratory testing in 2007. Similarly, the roll-out of the LPA molecular diagnostic method for DST may also have led to an increase in newly diagnosed MDR-TB cases in 2009.

**Conclusion**

Case detection of MDR-TB patients in Gauteng is increasing. Because of the danger of drug resistance amplification due to inappropriate treatment of MDR-TB cases with first-line drugs, and the risk of transmission resulting in primary MDR-TB cases, a study on MDR-TB treatment uptake following diagnosis is being undertaken in the province.

**Limitations**

The laboratory-diagnosed surveillance data do not contain clinical details of patients, including co-morbidity related to HIV status. Also, rates were not calculated due to mobility of the patient population and unreliable denominator information on population statistics.

**References**

1. World Health Organization. Multidrug and Extensively Drug-resistant TB (M/XDR-TB): 2010 Global Report on Surveillance and Response. World Health Organization 2010; WHO/HTM/TB/2010.3.
2. World Health Organization. Global tuberculosis control 2010. World Health Organization 2010; (WHO/HTM/TB/2010.7).
3. Ndjeka N. Policy framework for decentralization of MDR-TB services. Department of Health, South Africa, 2011. Available: http://www.auruminstitute.org/conferences/documents/Tembisa_TB_Symposium-Norbert_Ndjeka.pdf
4. Statistics South Africa. Census 2001: Census in brief. 2nd ed. Statistics South Africa, Pretoria 2003.

5. Weyer K, Lancaster J, Brand J, Van der Walt M, Levin J. Survey of tuberculosis drug resistance in South Africa: 2001–2002. Medical Research Council of South Africa, 2004.
6. Andrews JR, Shah NS, Gandhi N, Moll A, Friedland G. Multidrug-Resistant and Extensively Drug-Resistant Tuberculosis: Implications for the HIV Epidemic and Antiretroviral Therapy Rollout in South Africa. *J Infect Dis* 2007; 196: S482–490.
7. Gender and Health. Gender and tuberculosis. Department of gender and women health, World Health Organization. Geneva, Switzerland, 2002.
8. Department of Health. Multi-drug resistant tuberculosis: A policy framework on decentralized and deinstituitionalized management for South Africa. Pretoria, South Africa. 2011.
9. Weyer K, Lancaster J, Brand J, Van der Walt M, J L. Survey of tuberculosis drug resistance in South Africa: 2001–2002: Medical Research Council of South Africa 2004.
10. World Health Organization. Anti-tuberculosis drug resistance in the world: the WHO/IUATLD Global Project on Anti- Tuberculosis Drug Resistance Surveillance. Fourth global report. Geneva: World Health Organization 2008; WHO/HTM/TB/2008.394.

# NOVEL MALARIA CONTROL: CAN TRADITIONAL CLAY POTS BE USED TO DELIVER ENTOMOPATHOGENIC FUNGI TO MALARIA VECTORS IN NORTHERN KWAZULU-NATAL?

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**Background**

Malaria vector control relies principally on the application of chemical insecticides.[1] These are applied by indoor house spraying of residual formulations, as larvicides and as fabric treatments, particularly bed-nets. Although effective, these control options are frequently hampered by the development of insecticide resistance in target vector populations.[2]

There is a renewed enthusiasm for malaria control in Sub-Saharan Africa with a view to sequential elimination and eradication.[3] Boosting control effectiveness toward elimination requires a broader strategy that allows for the development and incorporation of new control tools to complement existing ones. The entomopathogenic fungi *Beauveria bassiana* and *Metarhizium anisopliae* are candidate biocontrol agents that have been shown to be highly effective against anopheline mosquitoes including malaria vector species.[4-8]

*Metarhizium anisopliae* and *B. bassiana* are pathogenic to a wide range of insect species.[4] Upon contact, their conidia are able to penetrate the cuticle and invade the internal cavity and organs, usually causing death within ten days of infection.[9] As disease vector control agents, their effectiveness depends on spore viability and persistence, spore formulation, mode of spore delivery, and ambient environmental conditions.[10-13]

African water storage pots (ceramic) have previously been shown to be an effective method of delivering oil formulated fungus spores to malaria vector species because they are attractive sites for resting mosquitoes under laboratory[6] and field conditions.[14] The aim of this study was to test the possibility of attracting anophelines, including malaria vector species, to ceramic pots in a field setting in the Mamfene region of northern Kwazulu-Natal, an area of seasonal malaria transmission. Malaria control at Mamfene is based on seasonal indoor spraying of houses with residual insecticides.

**Methods**

Traditional clay pots were procured from a potter in Mamfene. The pots were made using locally sourced clay and were moulded and fired using traditional techniques. Six pots were deployed to three randomly selected households in Mamfene sector 9. In each household, one pot was placed indoors in close proximity to a bed where one of the householders slept, and one pot was placed outdoors in a well shaded area near each house. The pots were deployed during the rainy summer season from October 2009 through to March 2010. Approximately 250 ml water was placed in each pot during collection periods to create a cool, humid atmosphere.

Mosquitoes were collected early each morning (between 07h00 and 10h00) from each pot over a five day period each month for the duration of the study. Anophelines found resting in the clay pots were sorted into species using morphological keys[15] and PCR based methods.[16,17]

**Results**

Five anopheline species were collected from inside the clay pots. These were *An. arabiensis*, *An. merus*, *An. quadriannulatus*, *An. parensis* and *An. vaneedeni*. Of these, *An. arabiensis* and *An. merus* are implicated as vectors of malaria[15,18] although none from these collections were found positive for *Plasmodium falciparum* sporozoites based on an enzyme linked immunosorbent assay (ELISA)[19]. A summary of the collections by household, pot location (indoor or outdoor), species and gender is given in table 1. Of the 107 specimens collected, *An. arabiensis* accounted for the bulk (57.9%) followed by *An. parensis* (23.4%) and *An. merus* (14%). Only single specimens of *An. quadriannulatus* and *An. vaneedeni* were found, and three specimens could not be identified to species. *Anopheles arabiensis* were generally found resting in the indoor and outdoor pots, whilst *An. parensis* and *An. merus* were almost exclusively indoor and outdoor resting respectively. There was significant variation between households in terms of the species diversity and numbers collected, with household 118 proving the most productive and household 100 the least productive. In general, the numbers of anophelines collected in the clay pots were comparable with previous collection records using exit window traps in the same area.[20-22]

| Household no. | *Anopheles* spp. | Outdoor | | Indoor | |
|---|---|---|---|---|---|
| | | ♂ | ♀ | ♂ | ♀ |
| **7** | arabiensis | 4 | 9 | 4 | 10 |
| | merus | 11 | 4 | - | - |
| | parensis | - | 1 | - | - |
| **118** | arabiensis | 4 | 6 | 1 | 19 |
| | quadriannulatus | - | - | - | 1 |
| | parensis | - | - | 3 | 16 |
| | vaneedeni | - | - | - | 1 |
| **100** | arabiensis | 3 | 2 | - | - |
| | parensis | - | - | 1 | 4 |

Table 1: Occurrence and numbers of anopheline mosquitoes found resting in clay pots placed either indoors or outdoors by household, species and gender in the Mamfene region of northern Kwazulu/Natal.

**Discussion**

The relatively low number of mosquitoes found resting in the clay pots at Mamfene during the collection period is a consequence of the effectiveness of the insecticide based malaria vector control programme implemented there by the Kwazulu-Natal Department of Health. Yet even under field conditions where vector control measures are already in place, clay pots proved to be suitable resting sites for anophelines, including malaria vector species, that had escaped intoxication from insecticide deposits on dwelling walls.

The suitability of the clay pots as resting sites in Mamfene was most apparent for *An. arabiensis*, which were found resting in pots placed indoors and outdoors. This species has been shown to be less susceptible to vector control based on indoor spraying of residual insecticide because of its variable host-seeking and resting behaviour, e.g. feeding on cattle or humans and resting indoors or outdoors.[15] Fungus treated clay pots therefore offer a means of exploiting this behavioural plasticity by placing pots indoors and outdoors, enabling the delivery of infective spores regardless of resting preference of target mosquitoes. The deployment of fungus treated clay pots in this way also offers a means of targeting insecticide resistant mosquitoes. This is an important consideration in the Mamfene region where the *An. arabiensis* population has been shown to carry resistance to pyrethroids and DDT.[21,22] Entomopathogenic fungi are promising agents for managing insecticide resistance because they are equally virulent against insecticide susceptible and resistant mosquitoes [8] and fungal infection carries the added benefit of attenuating the expression of insecticide resistance in malaria vector species. [23] We conclude that clay pots can be used to deliver infective fungal spores to malaria vector mosquitoes under field conditions in northern Kwazulu-Natal.

**Acknowledgements**

Malaria Control Programme, Kwazulu-Natal Department of Health.

**References**


1. Rosendaal J. Vector control methods for use by individuals and communities. World Health Organization. Geneva, 1997, pp 412.
2. Ranson H, N'Guessan R, Lines J, Moiroux N, Nkuni Z, Corbel V. Pyrethroid resistance in African anopheline mosquitoes: what are the implications for malaria control? *Trends Parasitol* 2011; 27: 91-8.
3. Kitua A, Ogundahunsi O, Lines J, Mgone C. Conquering malaria: enhancing the impact of effective interventions towards elimination in the diverse and changing epidemiology. *J Glob Infect Dis* 2011; 3: 161-165.
4. Scholte E-J, Knols BGJ, Samson RA, Takken W. Entomopathogenic fungi for mosquito control: A review. *J Insect Sci* 2004; 4:19.
5. Blanford S, Chan BHK, Jenkins N, Sim D, Turner RJ, Read AF, Thomas MB. Fungal pathogen reduces potential for malaria transmission. *Science* 2005; 308: 1638-1641.
6. Farenhorst M, Farina D, Scholte E-J, Takken W, Hunt RH, Coetzee M, Knols BGJ. African water storage pots for the delivery of the entomopathogenic fungus *Metarhizium anisopliae* to the malaria vectors *Anopheles gambiae s.s.* and *Anopheles funestus*. *Am J Trop Med Hyg* 2008; 78: 910-916.
7. Mnyone LL, Koenraadt CJ, Lyimo IN, Mpingwa MW, Takken W, Russell TL. Anopheline and culicine mosquitoes are not repelled by surfaces treated with the entomopathogenic fungi *Metarhizium anisopliae* and *Beauveria bassiana*. *Parasit Vectors* 2010; 3: 80.
8. Kikankie C, Brooke BD, Knols BGJ, Koekemoer LL, Farenhorst M, Hunt RH, Thomas MB, Coetzee M. The infectivity of the entomopathogenic fungus *Beauveria bassiana* to insecticide-resistant and susceptible *Anopheles arabiensis* mosquitoes at two different temperatures. *Malar J* 2010; 9: 71.
9. Roy HE, Steinkraus DC, Eilenberg J, Hajek AE, Pell JK. Bizarre interactions and endgames: Entomopathogenic fungi and their arthropod hosts. *Annu Rev Entomol* 2006; 51: 331–357.
10. Scholte E-J, Njiru BN, Smallegange RC, Takken W, Knols BGJ. Infection of malaria (*Anopheles gambiae* s.s.) and filariasis (*Culex quinquefasciatus*) vectors with the entomopathogenic fungus *Metarhizium anisopliae*. *Malar J* 2003; 2: 29.
11. Scholte E-J, Takken W, Knols BGJ. Infection of adult *Aedes aegypti* and *Ae. albopictus* mosquitoes with the entomopathogenic fungus *Metarhizium anisopliae*. *Acta Trop* 2007; 102: 151–158.
12. Thomas MB, Read AF. Can fungal biopesticides control malaria? *Nat Rev Microbiol* 2007; 5: 377-383.
13. Mnyone LL, Kirby MJ, Lwetoijera DW, Mpinqwa MW, Knols BGJ, Takken W, Russell TL. Infection of the malaria mosquito, *Anopheles gambiae*, with two species of entomopathogenic fungi: effects of concentration, co-formulation, exposure time and persistence. *Malar J* 2009; 8: 309.
14. Odiere M, Bayoh MN, Gimnig J, Vulule J, Irungu L, Walker E. Sampling outdoor, resting *Anopheles gambiae* and other mosquitoes (Diptera: Culicidae) in western Kenya with clay pots. *J Med Entomol* 2007; 44: 14-22.
15. Gillies MT, Coetzee M. A supplement to the *Anophelinae* of Africa south of the Sahara. 2nd Ed. *Publications of the South African Institute of Medical Research No.55,* 1987.
16. Scott JA, Brogdon WG, Collins FH. Identification of single species of the *Anopheles gambiae* complex by polymerase chain reaction. *Am J Trop Med Hyg* 1993, 49; 520-529.
17. Koekemoer LL, Kamau L, Hunt RH, Coetzee M. A cocktail polymerase chain reaction assay to identify members of the *Anopheles funestus* (Diptera: Culicidae) group. *Am J Trop Med Hyg* 2002; 66: 804-811.
18. Sinka ME, Bangs MJ, Manguin S, Coetzee M, Mbogo CM, Hemingway J, Patil AP, Temperley WH, Gething PW, Kabaria CW, Okara RM, Van Boeckel T, Godfray HC, Harbach RE, Hay SI. The dominant *Anopheles* vectors of human malaria in Africa, Europe and the Middle East: occurrence data, distribution maps and bionomic précis. *Parasit Vectors* 2010; 3: 117.
19. Wirtz RA, Zavala F, Charoenvit Y, Campbell GH, et al. Comparative testing of *Plasmodium falciparum* sporozoite monoclonal antibodies for ELISA development. *Bull WHO* 1987; 65: 39-45.
20. Mouatcho JC, Hargreaves K, Koekemoer LL, Brooke BD, Oliver S, Hunt RH, Coetzee M. Indoor collections of the *Anopheles funestus* group (Diptera: Culicidae) in sprayed houses in northern Kwazulu-Natal, South Africa. *Malar J* 2007; 6: 30.
21. Hargreaves K, Hunt RH, Brooke BD, Mthembu J, Weeto MM, Awolola TS, Coetzee M. *Anopheles arabiensis* and *An. quadriannulatus* resistance to DDT in South Africa. *Med Vet Entomol* 2003; 17: 417-422.
22. Mouatcho JC, Munhenga G, Hargreaves K, Brooke BD, Coetzee M, Koekemoer LL. Pyrethroid resistance in a major African malaria vector *Anopheles arabiensis* from Mamfene, northern Kwazulu/Natal, South Africa. *S Afr J Sci* 2009; 105: 127-131.
23. Farenhorst M, Mouatcho JC, Kikankie CK, Brooke BD, Hunt RH, Thomas MB, Koekemoer LL, Knols BGJ, Coetzee M. Fungal infection counters insecticide resistance in African malaria mosquitoes. *PNAS* 2009; 106: 17443-17447.

Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 31 December 2010/2011*

| Disease/Organism | Cumulative to 31 December, year | EC | FS | GA | KZ | LP | MP | NC | NW | WC | South Africa |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Anthrax | 2010 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Botulism | 2010 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Cryptococcus spp.* | 2010 | 1330 | 457 | 2099 | 962 | 568 | 703 | 63 | 532 | 490 | 7204 |
| | 2011 | 1236 | 357 | 1938 | 1037 | 417 | 597 | 66 | 453 | 498 | 6599 |
| *Haemophilus influenzae*, invasive disease, all serotypes | 2010 | 45 | 25 | 167 | 21 | 10 | 16 | 11 | 8 | 92 | 395 |
| | 2011 | 34 | 25 | 154 | 38 | 4 | 21 | 12 | 8 | 95 | 391 |
| *Haemophilus influenzae*, invasive disease, < 5 years | | | | | | | | | | | |
| Serotype b | 2010 | 6 | 8 | 27 | 8 | 3 | 8 | 5 | 2 | 15 | 82 |
| | 2011 | 8 | 5 | 20 | 12 | 1 | 4 | 7 | 3 | 17 | 77 |
| Serotypes a,c,d,e,f | 2010 | 0 | 2 | 9 | 0 | 1 | 2 | 0 | 0 | 10 | 24 |
| | 2011 | 1 | 1 | 12 | 3 | 0 | 0 | 0 | 0 | 5 | 24 |
| Non-typeable (unencapsulated) | 2010 | 1 | 1 | 46 | 4 | 0 | 0 | 1 | 1 | 11 | 65 |
| | 2011 | 2 | 2 | 31 | 6 | 0 | 1 | 0 | 0 | 14 | 56 |
| No isolate available for serotyping | 2010 | 13 | 4 | 17 | 0 | 2 | 4 | 0 | 1 | 6 | 47 |
| | 2011 | 6 | 4 | 19 | 1 | 3 | 6 | 1 | 3 | 2 | 47 |
| Measles | 2010 | 1309 | 674 | 1617 | 3837 | 290 | 1844 | 374 | 758 | 1796 | 12499 |
| | 2011 | 4 | 2 | 36 | 23 | 1 | 2 | 8 | 8 | 8 | 92 |
| *Neisseria meningitidis*, invasive disease | 2010 | 31 | 26 | 187 | 22 | 13 | 28 | 20 | 11 | 67 | 405 |
| | 2011 | 50 | 26 | 134 | 26 | 8 | 18 | 6 | 5 | 52 | 325 |
| Novel Influenza A virus infections | 2010 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Plague | 2010 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rabies | 2010 | 2 | 0 | 1 | 3 | 3 | 1 | 1 | 0 | 0 | 11 |
| | 2011 | 2 | 0 | 0 | 1 | 3 | 0 | 0 | 0 | 0 | 6 |
| **Rubella | 2010 | 436 | 116 | 389 | 366 | 122 | 188 | 70 | 287 | 361 | 2335 |
| | 2011 | 518 | 58 | 750 | 407 | 474 | 429 | 83 | 358 | 191 | 3268 |
| *Salmonella* spp. (not typhi), invasive disease | 2010 | 35 | 19 | 312 | 69 | 12 | 16 | 13 | 9 | 64 | 549 |
| | 2011 | 25 | 22 | 259 | 105 | 4 | 35 | 6 | 9 | 58 | 523 |
| *Salmonella* spp. (not typhi), isolate from non-sterile site | 2010 | 163 | 42 | 634 | 175 | 14 | 90 | 8 | 30 | 146 | 1302 |
| | 2011 | 140 | 21 | 503 | 234 | 11 | 62 | 18 | 18 | 240 | 1247 |
| *Salmonella typhi* | 2010 | 11 | 2 | 29 | 9 | 1 | 10 | 0 | 0 | 13 | 75 |
| | 2011 | 10 | 2 | 20 | 12 | 1 | 10 | 0 | 1 | 16 | 72 |
| *Shigella dysenteriae 1* | 2010 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Shigella* spp. (Non Sd1) | 2010 | 282 | 57 | 720 | 144 | 18 | 56 | 36 | 45 | 445 | 1803 |
| | 2011 | 215 | 40 | 598 | 164 | 8 | 30 | 35 | 12 | 451 | 1553 |
| *Streptococcus pneumoniae*, invasive disease, all ages | 2010 | 388 | 318 | 1847 | 426 | 110 | 240 | 106 | 183 | 587 | 4205 |
| | 2011 | 341 | 230 | 1612 | 360 | 56 | 192 | 68 | 187 | 563 | 3609 |
| *Streptococcus pneumoniae*, invasive disease, < 5 years | 2010 | 73 | 51 | 408 | 111 | 16 | 42 | 38 | 33 | 135 | 907 |
| | 2011 | 52 | 50 | 329 | 72 | 9 | 42 | 22 | 32 | 107 | 715 |
| *Vibrio cholerae* O1 | 2010 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| | 2011 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| Viral Haemorrhagic Fever (VHF) | | | | | | | | | | | |
| Crimean Congo Haemorrhagic Fever (CCHF) | 2010 | 0 | 3 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 5 |
| | 2011 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| ***Other VHF (not CCHF) | 2010 | 17 | 124 | 0 | 0 | 0 | 0 | 81 | 9 | 11 | 242 |
| | 2011 | 17 | 3 | 0 | 0 | 0 | 0 | 3 | 0 | 14 | 37 |

**Footnotes**

*Numbers are for cases of all ages unless otherwise specified. Data presented are provisional cases reported to date and are updated from figures reported in previous bulletins.

**Rubella cases are diagnosed from specimens submitted for suspected measles cases

***All cases for 2010 and 2011 were confirmed as Rift Valley Fever

Provinces of South Africa: EC – Eastern Cape, FS – Free State, GA – Gauteng, KZ – KwaZulu-Natal, LP – Limpopo, MP – Mpumalanga, NC – Northern Cape, NW – North West, WC – Western Cape

U = unavailable, 0 = no cases reported

Table 2: Provisional laboratory indicators for NHLS and NICD, South Africa, corresponding periods 1 January - 31 December 2010/2011*

| Programme and Indicator | Cumulative to 31 December 2011 | EC | FS | GA | KZ | LP | MP | NC | NW | WC | South Africa |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Acute Flaccid Paralysis Surveillance** | | | | | | | | | | | |
| Cases < 15 years of age from whom specimens received | 2010 | 51 | 19 | 67 | 76 | 45 | 33 | 2 | 19 | 23 | 335 |
| | 2011 | 70 | 32 | 78 | 91 | 75 | 39 | 7 | 19 | 21 | 432 |

Footnotes

*Numbers are for all ages unless otherwise specified. Data presented are provisional numbers reported to date and are updated from figures reported in previous bulletins.

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The Communicable Diseases Surveillance Bulletin is published by the National Institute for Communicable Diseases (NICD) of the National Health Laboratory Services (NHLS), Private Bag X4, Sandringham, 2131, Johannesburg, South Africa.

Suggested citation: [Authors' names or National Institute for Communicable Diseases (if no author)]. [Article title]. Communicable Diseases Surveillance Bulletin 2012; 10(1): [page numbers]. Available from http://www.nicd.ac.za/ pubs/ survbull/2011/CommDisBull April 2012.pdf



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