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1 ZOOBOTIC AND VECTOR-BORNE DISEASES

a Zika virus — a South African perspective

A 46-year-old industrial mechanic, resident in Cali, Colombia, arrived in Johannesburg on Wednesday 10 February. He became ill on Monday 15 February with anorexia, fever, and a fine, punctuate-like rash on his hands, thorax and neck. He had no joint or muscle pain, or conjunctivitis. He visited a private general practitioner who advised Zika virus testing. His illness was short-lived, and he felt better within 3 days. His blood specimen was positive by PCR for Zika virus at a private laboratory in Johannesburg. A second PCR test on the same specimen was conducted by the NICD, and confirmed the positive result. This is the first case of Zika virus infection imported into South Africa.

Zika virus (ZIKV) was recognized as a human pathogen for the first time in 1964 following an occupationally-acquired infection. The virus was isolated for the first time from a sentinel monkey in Uganda in 1947 and from *Aedes africanus* mosquitoes the following year. The virus remained confined to the equatorial belt of Africa and Asia, until 2007 when it caused an outbreak on Yap Island in the Pacific Ocean, followed by a rapid expansion of the virus' geographical range throughout other islands in the Pacific Ocean. In 2014 ZIKV reached South and Central America and spread explosively. As of February 2016, 23 countries in the Americas and Caribbean have reported active transmission of ZIKV. In Brazil alone estimation of ZIKV infections ranges from 500 000 to 1.5 million in 2015. Due the rise in cases of microcephaly and neurological disorders likely associated with ZIKV infections, the WHO recognized this situation as a public health emergency of international concern (PHEIC) on 1 February 2016. This declaration calls for a more co-ordinated effort to improve surveillance, mosquito control programs and fast-track the development of diagnostic assays, vaccines and antiviral therapeutics for ZIKV to investigate the causality of the observed disorders. As the number of cases increases, stronger scientific data will become available to better understand the pathogenesis of neonatal abnormalities. Recently the virus was detected in the amniotic fluid of two fetuses with microcephaly, and more convincingly in a separate study, the virus was detected in the brain of a fetus associated with severe brain injury and vertical transmission. A few cases of possible sexual transmission have also been observed, in addition to the isolation of ZIKV from infected patient semen. However, sexual transmission of ZIKV

remains a rare mode of the virus spread among humans.

Clinical diagnosis of ZIKV disease is complicated due to the non-specific clinical presentation and similarity with other arboviral infections especially dengue fever and chikungunya. About 80% of human infections are asymptomatic. Symptoms of ZIKV infection include fever (<38.5 °C), maculopapular rash, arthralgia (specifically involving the small joints of the hands and feet) and conjunctivitis. Laboratory diagnosis of acute ZIKV infection can be achieved through virus isolation in mice or tissue culture, and molecular testing by ZIKV specific RT-PCR. Due to the transient viremia caused by ZIKV infection, these assays are most useful up to day 5 post-disease onset. Serological testing is more complex due to the high level of cross-reactivity between flaviviruses. Diagnosis by serological testing is most accurate when testing paired serum samples collected at least 14 days apart. However, interpretation of serological result is complex and need to be addressed on a case-to-case basis. Infection with ZIKV is expected to induce lifelong immunity. The travel and clinical history of the patient must always be taken into account in aiding laboratory diagnosis. Cases submitted for laboratory investigation should have an epidemiological link to current ZIKV outbreaks. This implies a recent travel history to an affected country and/or sexual contact with a male who has travelled to one of the affected countries and has a history compatible with ZIKV disease. The required specimen type for ZIKV laboratory diagnosis is clotted blood or serum. There is no vaccine or treatment available for ZIKV.

The risk of ZIKV infection to the general South African population is low. The virus has not been reported in South Africa over decades. The highest health risk is to the unborn of pregnant women travelling to ZIKV affected areas, and possibly the unborn of pregnant woman that have sexual contact with male partners that are infected with ZIKV due to recent travel to ZIKV affected country. On the 8 of February 2016, the South African Department of Health issued a travel advisory recommending that pregnant women avoid travelling to affected areas or if they have to travel, that they should strictly follow steps to avoid mosquito bites. The advisory also states that the NICD offers testing to patients with compatible symptoms returning from affected areas. In

addition, steps are being taken to reduce the risk of translocation of possible infected mosquitoes from ZIKV affected countries. On the 5 of February 2016, NDOH Environmental and Port Health department issued an alert to intensify surveillance and screening for ZIKV in points of entry. This includes a number of steps: 1) continued mosquito monitoring of arriving aircrafts and increased mosquito surveillance to ensure that aircraft are sprayed with insecticide; 2) assessment of ship sanitation to a) identify possible vector breeding areas; b) ensure that steps have been taken to minimize insect breeding, and c) to inspect ships for

presence of mosquito vectors; 3) continued thermal scanning of all travellers at ports of entry; 4) referral of travellers at points of entry with compatible symptoms to a health care facility for further management; 5) increased monitoring of imported used tyre casings and 6) increased health education and awareness to travellers about ZIKV disease.

Source: Centre for Emerging and Zoonotic Diseases, and Division of Public Health, Surveillance and Response, NICD-NHLS; (januszp@nicd.ac.za)

b Crimean-Congo haemorrhagic fever

To date this year, there have been no reported cases, or suspected cases of Crimean-Congo haemorrhagic fever (CCHF). In 2015, a single case was diagnosed in Free State Province and the patient recovered uneventfully. In 2014 and 2013, there were six and five cases respectively.

CCHF is an emerging zoonotic disease transmitted by hard or ixodid ticks. The CCHF virus has been reported in more than 30 countries in Africa, the Middle East, Asia and south-eastern Europe. In 2011 the disease was reported for the first time in India. In Turkey and other eastern European countries up to thousands of cases of CCHF are reported annually. In South Africa, CCHF is a rare disease in humans with a total of 200 cases laboratory confirmed between 1981 and 2015. Fewer than ten cases are reported annually. CCHF has been reported from all nine provinces of South Africa, although most cases are reported from the Free State, Northern Cape and North West provinces.

The emergence of CCHF is linked to the expanding distribution of *Hyalomma* ticks, in particular *Hyalomma marginatum* (also known in South Africa as the 'bontpoot' tick). CCHF is typically reported in farmers, farm workers, slaughter house workers, veterinarians and others that may come into contact with these ticks. In South Africa, more than two-thirds of cases reported a tick bite or contact with ticks. In a few cases, contact with infected animal blood or tissues were implicated. Nosocomial transmission of CCHF has occurred on four occasions in South Africa since 1981.

The clinical presentation of CCHF varies greatly. The spectrum of disease ranges from sub-clinical or mild disease to fatal haemorrhagic manifestations. The incubation period varies based on the type of exposure. With tick bites, the incubation period is short (1-3 days), whilst contact with contaminated blood and tissues results in a longer incubation period of up to 7 days. The disease is generally characterized by an abrupt onset of fever with malaise, myalgia, abdominal pain, nausea, vomiting and diarrhea. Haemorrhage is not present in all cases and may manifest as a petechial rash, ecchymosis, epistaxis, melena, hematuria or intraabdominal bleeding. Routine blood screens will reveal invariably reveal thrombocytopenia and elevated liver transaminases whilst leukopenia and hyperbilirubinemia are also reported. Confirmatory laboratory testing is available from the National Institute for Communicable Diseases. The Institute offers an array of laboratory tests including RT-PCR, indirect immunofluorescence assay, IgG and IgM ELISA and virus isolation. The Institute operates the only biosafety level 4 laboratory where safe and secure handling and storage of the haemorrhagic fever viruses can be executed.

There are no vaccines or specific treatments for CCHF virus infection. The effectiveness of ribavirin is contentious at the present moment. Management is supportive. Infection control precautions are essential to avoid nosocomial transmission.

Source: Centre for Emerging and Zoonotic Diseases, NICD-NHLS (januszp@nicd.ac.za)

c Rabies update and review of human cases, 1983-2015

Update

No human rabies cases have been reported to date this year in South Africa. Rabies was confirmed in a domestic unvaccinated cat on a plot bordering on the Roodeplaat Dam and Nature Reserve in the City of Tshwane, about 30 km from the centre of the city. The cat appeared ill, and was euthanized. Rabies was confirmed by PCR. Sequencing of the virus is currently underway to identify whether the strain is a canid or viverid biotype. There were no confirmed human exposures prior to capture. After capture at Onderstepoort Veterinary Institute, a nurse, nine students and four animal technicians were exposed and received post-exposure prophylaxis. After the diagnosis was made, a ring vaccination campaign was conducted in the area, and approximately 350 pets were vaccinated.

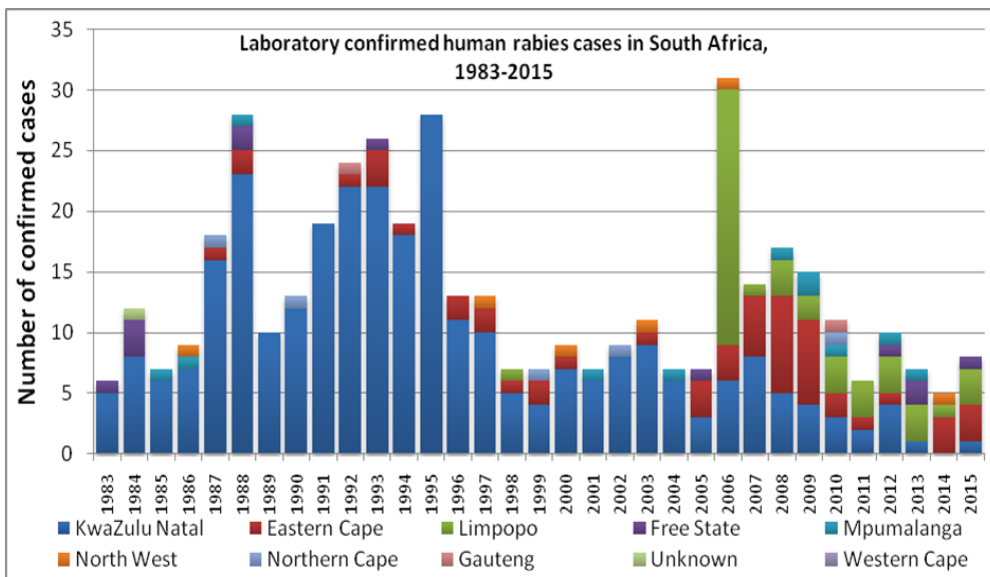
Sporadic cases of viverid rabies are seen from time to time in mongeese on the outskirts of Gauteng so it is advisable that all domestic cats and dogs in be vaccinated against rabies at least twice in the first year and then every three years as per the regulations. When persons are bitten by animals, a risk assessment for rabies must be carried out in all areas in South Africa, including urban and peri urban areas.

Human Rabies cases, 1983-2015

More than 80% of human rabies cases confirmed in South Africa are linked to exposures involving domestic dogs, although rabies is also reported in other species. Historically KwaZulu-Natal Province was most affected, but in the last ten years dog rabies has emerged in several additional locations in South Africa (Figure 1). Currently dog rabies cases are reported from the Mpumalanga, Limpopo,

Eastern Cape, North West, Free State and KwaZulu-Natal provinces. In the past ten years an average of 13 human cases has been reported (range 5-31) per year. In 2015, eight confirmed human rabies cases were diagnosed from the following provinces: KwaZulu-Natal (n=1); Limpopo (n=3); Eastern Cape (n=3) and Free State (n=1). Three probable and one suspected human rabies cases were identified.

Rabies is an invariably fatal disease, but it is completely preventable with the use of rabies post-exposure prophylaxis (PEP) in patients exposed to potential (or confirmed) rabid animals. Exposures are categorized based on the risk of the exposure as category 1, 2 or 3. Category 1 exposures present cases where the risk of exposure to rabies virus is negligible and no response is required. An example of a category 1 exposure is the petting of a potentially (or confirmed) rabid animal. Category 2 exposures present intermediate risk for rabies transmission and include superficial exposures such as grazes or surface scratches that did not draw any blood. Category 2 exposures require a regimen of four intramuscular doses of rabies vaccine administered in the deltoid muscle. Category 3 exposures include any exposure that results in a wound however minimal, that draws blood: A break in the skin barrier may allow the penetration of rabies virus-laden saliva from the animal that was involved in the exposure. Category 3 exposures require copious wound washing, followed by provision of rabies immunoglobulin (at 20 IU/kg, in deltoid muscle not receiving the rabies vaccine) and four doses of rabies vaccine administered. An updated rabies PEP guidance poster is available from the NICD website for further information (www.nicd.ac.za).



Source: Centre for Emerging and Zoonotic Diseases, NICD-NHLS; Onderstepoort Veterinary Institute, Gauteng Department of Agriculture and Rural Development; (januszp@nicd.ac.za)

Figure 1. Laboratory-confirmed human rabies in South Africa, 1983-2015

2 SEASONAL DISEASES

a An entomological survey of an odyssean malaria outbreak investigation in West Rand District

On the 22 January 2016, a case of malaria was reported from Leratong Hospital, West Rand District in Mogale City, Gauteng Province. The patient was a 34-year-old female, who was born in Standerton, Mpumalanga Province, and had lived in Simunye settlement (near Westonaria) since 2005. She was an underground worker for a gold mine, and while residing at the mine residence temporarily in late December and early January, suddenly fell sick. There was no history of recent travel to a malaria endemic area, use of needles or blood transfusion. She experienced the onset of fever, severe headache, fatigue and body cramps on the 4th January 2016. She visited the mining health facility, and a nurse treated her and sent home. However her condition worsened compelling her to visit the same facility again on 16 January, and she received treatment for 'flu. On the 20 January upon her visiting the facility for the third time, a malaria rapid test was performed. She was immediately treated with Coartem but her health continued to deteriorate to the extent of being unconscious. On the 21 January while unconscious she was taken to Leratong Hospital. The presence of malaria parasites were noted during a routine haematology differential smear on the 22 January. *Plasmodium falciparum* was confirmed on blood smear, with 3.1 % parasitaemia. She was admitted to ICU, responded positively to treatment and has since recovered and been discharged.

The patient's residence in Simunye and the mine where she worked are flanked by, or close to busy roads: N12, R559, R93 and R28. The mine residence accommodates 214 employees plus 60 undocumented migrants, comprising individuals from all the Provinces in South Africa and migrant workers from the neighbouring countries such as Mozambique and Zimbabwe. An investigation of the

property revealed several potential breeding and resting sites: blocked storm water canal in front of the room, leaking pipes, borrow pits along the main pipe line, and open drains with stagnant or very slow moving water. Upon investigation, the bodies of water, open drains and borrow pits revealed the presence of adult mosquito and larvae (Figure 2). All adult mosquitoes and larvae collected were identified as *Culex* species, which are not vectors of malaria; thus they could not be implicated in disease transmission. Entomological investigation was also conducted at the patient's residence in Simunye as she had spent some nights and weekends there, especially when she was not on a night shift.

Conclusion

The entomological survey concludes that even though there is the possibility for malaria vectors to breed near the mine residence, it is unlikely that the current case was due to a locally-breeding population as no evidence of anopheline adult and larvae were found. It is therefore concluded that this case of malaria is classified as odyssean. The infective mosquito could have been introduced by road transport or by an individual inadvertently carrying it in a bag or suitcase.

Recommendation

It is crucial that the community be educated about malaria and the transmission of the disease. Speaking to the hostel managers and the patient it was apparent that there is a general lack of knowledge of malaria epidemiology. The potential breeding and resting sites on the property could be minimised by urgent maintenance of the infrastructure e.g. fixing the leaks and replacing the metal drain covers/lids with plastic ones. The use of mosquito coils and other available repellents by residents should be encouraged.



Source: Centre for Opportunistic, Tropical & Hospital Infections, NICD-NHLS; West Rand District and Gauteng Provincial Departments of Health. (johnf@nicd.ac.za)

Figure 2. Stagnant water around the mine residence - potential mosquito breeding and resting sites

b Suspected enterovirus meningitis/encephalitis outbreak in City of Tshwane, Gauteng Province: final report

Last year in the November and December communiques we reported on a possible enterovirus outbreak in the Tshwane District. In total 42 cases were reported; 40 cases were children under the age of 10 years and 2 cases were adults. The median age of all cases was 4 years (range 1 month to 42 years). All samples from suspected cases were processed at private diagnostic laboratories - 31 samples tested positive for enterovirus and 11 samples were negative; NICD requested all samples for further testing and genotyping.

Of the 42 identified cases, NICD received 18 cerebrospinal fluid specimens and 7 stool specimens from the private diagnostic laboratories. Of the eight samples that could be genotyped, three tested negative for enteroviruses, five were echovirus 6, three were coxsackievirus A9, one was coxsackievirus B2, one was coxsackievirus B4, one was coxsackievirus B5 and one was echovirus 11. The other ten samples could not be genotyped due to low viral load in the samples. Telephonic interviews were conducted with parents of children admitted to hospital and for whom contact information was available. Although there was a suggestion of institutional (crèche/school) and household (sibling) clusters, due to the unavailability of specimens, these clusters could not be confirmed by molecular methods. No additional common risk factors were identified between these children.

Due to the diverse genotypes identified and the fact that we could not type half of the received specimens, no clear conclusions could be made

regarding this cluster of cases, nor whether it was an outbreak or a normal seasonal increase of cases. Historical data from South Africa shows that enteroviruses do cause seasonal increases during summer months (1, 2). More data are needed and NICD has therefore started routine meningitis surveillance at sentinel sites.

Human-to-human transmission of enteroviruses is usually via the faecal-to-oral route and, to prevent spread, improvements in hygiene and sanitation are advised. Children who are ill should stay at home to avoid infecting others. No public health action is required for individual cases and people who are close contacts of viral meningitis patients do not need prophylactic antibiotic treatment.

References

1. Keen GA, McIntyre JP. Summer aseptic meningitis in Cape Town, 1981-1986. *South African Medical Journal* 1986;69(8):473-4.
2. McIntyre JP, Keen GA. Laboratory surveillance of viral meningitis by examination of cerebrospinal fluid in Cape Town, 1981-9. *Epidemiology and Infection*. 1993;111(2):357-71.

Sources: Centre for Respiratory Diseases and Meningitis, NICD-NHLS (cherylc@nicd.ac.za)

c A conjunctivitis outbreak at a primary school in the City of Johannesburg, Gauteng Province

On 3 February 2016, a staff member at a school in the City of Johannesburg, Gauteng Province, alerted the NICD Outbreak Response Unit (ORU) of a suspected outbreak of 'pinkeye' (conjunctivitis) at the school. The school campus comprises a pre-school, primary school (grades R-7) and a high school (grades 8-12). The pupils affected were both boys and girls from the primary school.

According to the informant, the number of pupils affected rose from 4 cases on Monday 01 February 2016 to 31 cases on Thursday 04 February 2016. The cases were pupils from grade R to grade 7 (age range 5 – 13 years) and 3 teachers from those grades. The first 4 cases reported having painful

eyes to the school officials after attending their Monday morning assembly meeting.

During this meeting, a science experiment demonstration was performed by an independent science educational company. According to the company's representative, only water and dry ice were used in the illustration. No chemicals had been used.

The NICD ORU together with the Provincial Communicable Disease Control Coordinator (CDCC) and the district ophthalmologist visited the school on Thursday 04 February 2016. A case investigation form (CIF) for conjunctivitis was developed and a line list of the affected pupils was obtained from the

school. Verbal consent was obtained from the parents/guardians of the affected pupils. The CIFs were completed telephonically by contacting the parents/guardians.

Of the 31 cases, 21 were present on the day of the site visit. Ten were at home. Eye swabs were taken in 21 cases and ten (48%) CIFs were completed. Eleven parents/guardians were unavailable to complete the CIFs. Of the ten completed CIFs, all complained of an itchy tearing eye. The symptoms were mostly bilateral. 40% (4/10) had photophobia and 60% (6/10) complained of a burning sensation in their eyes. No cases had a discharge, nor complained of redness of the eyes. No cases reported systemic symptoms or lymphadenopathy. In the 21 cases seen at the school, the disease process was mild with no complications. Amongst the ten cases interviewed, none had contact with any person outside the school who had similar symptoms 14 days prior to the onset of illness. No environmental risk factors such as fumigation of the school or chlorination of the swimming pool were reported.

All 21 specimens tested negative for adenovirus, herpes viruses, enterovirus, *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Neisseria meningitidis*. Specimens were not tested for *Staphylococcus aureus*.

In outbreak settings, the commonest viral causes of infective conjunctivitis are adenoviruses, herpes simplex viruses and picornaviruses such as enteroviruses. *Staphylococcus aureus*, *Haemophilus influenzae* and *Streptococcus pneumoniae* are some of the common causes of bacterial conjunctivitis. In some cases, *Neisseria meningitidis* has been implicated. Person-to-person transmission of infective causes of conjunctivitis perpetuate localised outbreaks. Prevention is through attention to handwashing and preventing sharing of towels and hand cloths. Allergic or irritant conjunctivitis may also be responsible for outbreaks.

In this outbreak, the causative agent is unknown. The 21 cases from whom samples were obtained were well and not symptomatic at the time of taking the eye swabs. The ten symptomatic children were at home and therefore not accessed. Also, no nasopharyngeal swabs were taken to look for carrier status of common pathogens. Health promotion messages describing infection control measures were distributed at the school to prevent further spread of infection.

Source: Division of Public Health, Surveillance and Response (outbreak@nicd.ac.za)

3 RESPIRATORY DISEASES

a Three travel-associated Legionnaires' disease (TALD) cases associated with a hotel in Cape Town

Legionnaires' disease (LD) is a notifiable condition in South Africa. It is caused by infection by Gram-negative bacteria in the *Legionella* genus. *Legionella* spp. are ubiquitous and are found in the environment and natural water sources. They thrive at environmental temperatures between 20-50°C. Legionnaires' disease is predominantly associated with water systems in the built environment. These include hot and cold piped water systems in large buildings, cooling towers, decorative fountains and respiratory therapy equipment. Colonisation of these water systems by *Legionella* species results in the creation of aerosolised droplets containing *Legionella* bacteria. Legionnaires' disease is acquired when aerosols are inhaled. The incubation period normally ranges from 2 to 10 days. Infection with the bacterium *Legionella* commonly presents as severe pneumonia, often requiring hospitalization. Human-to-human transmission is

exceptional, and until recently, had never been described (see References)

Many reported Legionnaires' disease outbreaks have been associated with hotel stay (travel-associated Legionnaires' disease (TALD)) or hospital admission (health-care associated Legionnaires' disease), due to colonisation of the complex water systems of large buildings. Persons admitted to hospitals are particularly vulnerable if appropriate preventive measures are not maintained in hospital water systems, on account of their risk factors for acquisition of legionellosis. Risk factors include age >50 years, current or past smoking, diabetes, chronic lung disease, systemic malignancy and renal or hepatic failure.

Data on the prevalence and epidemiology of Legionnaires' disease in South Africa are limited.

Sporadic disease has been reported in a recent study of syndromic pneumonia surveillance at two sentinel sites in South Africa from June 2012 through September 2014, in which *Legionella* spp. were detected in 21 (1.2%) of 1805 cases (see References). This study reported that community-acquired LD in South Africa occurs predominantly in chronically ill adults with HIV and/or TB infection, and the majority of cases are not diagnosed and are sub-optimally treated.

In December and early January 2016, the NICD and NHLS Infection Control Laboratory received a notification from the European Legionnaires' disease Surveillance Network (ELDSNet) that 3 Dutch travellers who had stayed at a Cape Town hotel in March 2014 (1 case) and December 2015 (2 cases) had subsequently developed LD. The hotel was notified, and a risk assessment audit of the facility was conducted by the NHLS Infection Control laboratory and the NICD in January 2016. The hotel was found to have a *Legionella* control programme in place, with no prior samples testing positive for *Legionella*. The hotel was found to not have any high-risk features such as decorative water features, sauna, jacuzzi or gym. Water samples were taken from hot and cold water tanks and the rooms where patients had stayed, as well as a randomly-selected room on each floor of the 13-storey building. A total of 43 water samples was collected. Emergency remedial action was requested including increasing the hot water temperature, hyper-chlorination of the water system, closing guest rooms where cases had stayed, as well as the distribution of a letter and information sheet to all guests who stayed in the hotel within a 2-week window period before the date of the report.

Legionella pneumophila serogroup 1 was detected in both hot water tanks, one cold water tank and from water obtained from the room where one of the cases had stayed. A follow-up audit was carried out in February to verify completion of corrective actions and to re-sample. The NHLS and NICD are in on-going communication with the management of the hotel regarding *Legionella* remediation and control.

Legionnaires' disease can be radiologically and clinically indistinguishable from other aetiological

causes of pneumonia. Penicillins and other β -lactam antibiotics which are the first line of treatment advised for community acquired pneumonia, are inactive against *Legionella* spp. Therefore a high index of suspicion is required, and diagnostic tests for LD should be specifically asked for, especially in the following clinical scenarios: 1) patients with severe pneumonia, in particular those requiring intensive care; 2) immunocompromised individuals with pneumonia; 3) patients with pneumonia in the setting of a legionellosis outbreak; 4) patients who have travelled away from their home within two weeks before the onset of illness, and 5) patients suspected of health-care associated pneumonia.

Available test methods include urinary antigen test (UAT), and culture and real-time PCR on lower respiratory tract specimens such as sputum. For every probable or confirmed case of Legionnaires' disease, a case investigation (form available on the NICD website www.nicd.ac.za) should be conducted to identify potential sources of exposures. The local health department, as well as NICD, should be notified as soon as possible in order to formulate an appropriate public health response, and prevent further cases. For further information please refer to the NICD guidelines for Legionnaires' disease, or contact the NICD (outbreak@nicd.ac.za) or Dr Nicole Wolter nicolew@nicd.ac.za.

References

Correia AM, Ferreira JS, Borges V, Nunes A, Gomes B, Capucho R, *et al.* Probable person-to-person transmission of Legionnaires' Disease. *N Engl J Med.* 2016 Feb 4;374(5):497-8.

Wolter N, Carrim M, Cohen C, Tempia S, Walaza S, Sahr P, *et al.* Legionnaire's disease in South Africa, 2012-2014. *Emerg Infect Dis.* 2016 Jan;22(1):131-3.

Source: Centre for Respiratory Diseases and Meningitis, and Division of Public Health, Surveillance and Response, NICD-NHLS; Infection Control Laboratory, Parktown, Johannesburg, NHLS. (annev@nicd.ac.za, nicolew@nicd.ac.za)

b Influenza - guidance for the upcoming season

The influenza season in South Africa occurs in the winter months, on average beginning in the first week of June. However, there is variation – in past years, the season has started as early as the last week of April or as late as the first week of July. Data from the NICD influenza surveillance programmes (the 'Viral Watch', influenza-like illness (ILI) surveillance at primary healthcare clinics and pneumonia surveillance for severe disease in hospitalised patients) show that during 2015, the predominant circulating influenza subtype was influenza A(H1N1)pdm09, followed by influenza A (H3N2), and influenza B. The season started in week 16 (ending 19 April), peaked in week 23 (ending 7 June) and ended in week 37 (ending 13 September). Vaccination is the most effective strategy to prevent influenza. It is essential that health care practitioners familiarize themselves with influenza vaccination guidelines in preparation for the coming season.

Recommended influenza vaccine formulation for 2016

Influenza vaccine composition is updated frequently because circulating influenza viruses continuously evolve. The following strains have been recommended by the World Health Organization (WHO) for the trivalent inactivated influenza vaccine (IIV) 2016 southern hemisphere influenza season:

- an A/California/7/2009 (H1N1)pdm09-like virus
- an A/Hong Kong/4801/2014 (H3N2)-like virus
- a B Brisbane/60/2008-like virus.

These recommendations include a change in the A (H3N2) and B strains when compared with the composition of the trivalent IIV used for the southern hemisphere during the 2015 season.

Timing of influenza vaccination

Vaccination should be administered each year before the influenza season, i.e. from March (or as soon as the vaccine becomes available) to June. Protective antibodies develop by two weeks post-immunization. Vaccination may not be helpful if administered late in the season, although it is still advised for persons at risk of severe influenza.

Healthcare workers are encouraged to discuss influenza vaccination with their patients, especially amongst those who are at increased risk for severe influenza-associated complications.

Groups recommended for influenza vaccination

The following groups of persons are advised to receive influenza vaccine:

- Pregnant women irrespective of stage of pregnancy, or postpartum
- Persons with underlying medical conditions such as chronic pulmonary (including tuberculosis) and cardiac diseases, chronic renal diseases, diabetes mellitus and similar metabolic disorders, individuals who are immunosuppressed and individuals who are morbidly obese (body mass index ≥ 40 kg/m²)
- Persons living with HIV infection
- Healthcare workers
- Residents of chronic care and rehabilitation facilities, including homes for the elderly.
- Persons aged >65 years
- Children aged 6 months - 59 months
- Persons aged 6 months - ≤ 18 years on long-term aspirin therapy
- Adults and children who are family contacts of individuals at high risk of severe influenza
- Any persons wishing to minimise the risk of influenza acquisition, especially in workplace settings where large-scale absenteeism could cause significant economic losses.

Detailed recommendations on target groups, dosages and contraindications for the 2016 influenza season will be published in the March issue of the South African Medical Journal.

Source: Centre for Respiratory Diseases and Meningitis, NICD-NHLS (cherylc@nicd.ac.za).

4 SEXUALLY TRANSMITTED INFECTIONS

a Sentinel surveillance of sexually transmitted infection (STI) syndrome aetiologies among patients attending a public healthcare facility in Johannesburg: report on the findings from 2015 and comparison with 2014 data

Introduction

The syndromic approach to the management of sexually transmitted infections (STIs) in primary healthcare centres (PHCs) is based on the identification of a group of symptoms and easily recognizable signs associated with a number of well-defined aetiologies. Periodic aetiological surveillance of STI syndromes is critical in validating the existing treatment algorithms.

The NICD conducted a survey among patients presenting to a community-based primary healthcare facility in Johannesburg. The main objective was to determine the microbial aetiologies of the three major STI syndromes, namely male urethritis syndrome (MUS), vaginal discharge syndrome (VDS) and genital ulcer syndrome (GUS) in adult (>18 years) patients. Secondary objectives were to determine (a) the prevalence of HIV co-infection in patients presenting with STI syndromes; and (b) the antimicrobial susceptibility to extended-spectrum cephalosporins (ESCs) of *Neisseria gonorrhoeae* isolates from MUS patients.

Swabs were used for the sampling of genital discharge (vaginal, endocervical, urethral) and genital ulcers in consenting adult patients. Swab-extracted DNA was tested by multiplex real-time PCR assays for STI pathogens. Minimum inhibitory concentrations (MICs) to ceftriaxone and cefixime in *N. gonorrhoeae* isolates were determined using the E-test® method. Vaginal smears from VDS patients were examined microscopically for the presence of bacterial vaginosis (BV) and candidiasis. Giemsa-stained ulcer impression smears were screened for *Klebsiella granulomatis*. Serum specimens were tested for HIV. A total of 200 MUS, 200 VDS and 100 GUS in 2014 and 150-200 MUS, 100 VDS, and 100 GUS cases in 2015 were required to satisfy sampling requirements.

Results

In 2015, a total of 379 STI patients presenting to the PHC was tested: 169 MUS, 107 VDS and 103 GUS. Among MUS cases, *Neisseria gonorrhoeae* remained the most common aetiological agent detected (152/169, 89.9%) followed by *Chlamydia*

trachomatis (31/169, 18.3%). There was a statistically significant increase in the prevalence of *N. gonorrhoeae* and a relative decrease in the prevalence of *Chlamydia trachomatis* compared to 2014 (Table 1). Most infections (134/169, 79.3%) had a single aetiology.

Among VDS patients, BV was the commonest cause (68/107, 63.6%), followed by *Chlamydia trachomatis* (28/107, 26.2%) and *Trichomonas vaginalis* (27/107, 25.2%), respectively (table 1). Of the 68 patients with BV, 32 (47%) were co-infected with one or more STI pathogens. A relative increase in the prevalence of *Trichomonas vaginalis* in 2015 was not statistically significant ($p = 0.07$). There were no significant differences in aetiological findings between 2014 and 2015.

The prevalence of GUD pathogens was as follows: HSV (57/103, 55.3%) and *Treponema pallidum* (4/103, 3.9%). No cases of lymphogranuloma venereum (LGV), chancroid and donovanosis were detected for both years (Table 2). Only one patient had mixed ulcer aetiology (HSV and *Treponema pallidum*) detected by PCR. An ulcer-derived pathogen was not identified in 42/103 (41%) GUS cases.

HIV seroprevalence rates were as follows: approximately 30% in MUS, 40% in VDS and 55% among GUS cases. HIV co-infection rates in 2015 were not significantly different from those in 2014. All gonococcal isolates from male urethral discharge specimens demonstrated low ES-cephalosporin MICs that were within the susceptible range.

Conclusions:

N. gonorrhoeae remains the commonest cause of MUS, and decreased susceptibility to ESCs was not detected in any isolate. Ongoing monitoring of antimicrobial susceptibilities in high-risk populations is essential. Herpes simplex virus remains the commonest detectable cause of genital ulceration, validating the continued use of acyclovir in syndromic management. The cause of ulceration in 41% of patients without a diagnosis requires further research. Bacterial vaginosis is the predominant cause of an abnormal vaginal discharge among female patients; however, a significant proportion was co-

infected with one or more STI pathogens. The HIV seroprevalence among STI patients is high, underlining the importance of linkage to universal HIV testing and treatment for these patients in primary healthcare settings.

Source: Centre for HIV and STI, NICD-NHLS (adrianp@nicd.ac.za; ranminik@nicd.ac.za).

Table 1. The prevalence (%) of STI pathogens in patients presenting with genital discharge in Johannesburg during 2014 and 2015

Pathogen	MUS			VDS		
	2014 (n=208)	2015 (n=169)	*p value	2014 (n=200)	2015 (n=107)	*p value
<i>Neisseria gonorrhoeae</i>	168 (80.5)	152(89.9)	0.01	35(17.4)	23(21.5)	0.44
<i>Chlamydia trachomatis</i>	61(29.3)	31(18.3)	0.01	41(20.5)	28(26.2)	0.28
<i>Trichomonas vaginalis</i>	6 (3.0)	3(1.8)	0.74	32(16.0)	27(25.2)	0.07
<i>Mycoplasma genitalium</i>	12(6.0)	1(0.5)	0.00	24(12.0)	13(12.2)	1.00
Bacterial vaginosis				109(54.2)	68(63.6)	0.21
Candidiasis				43(21.4)	26(24.3)	0.78

*p values reflect significance of difference between 2014 and 2015 data based on Fischer's exact tests

Table 2. The prevalence (%) of STI pathogens in patients presenting with genital ulcer syndrome in Johannesburg

Pathogen	2014 (n=84)	2015 (n=103)	*p value
<i>Herpes simplex virus</i>	53 (63.1)	57 (55.3)	0.50
<i>Treponema pallidum</i>	6 (7.1)	4 (3.9)	1.00
<i>Haemophilus ducreyi</i>	0 (0.0)	0 (0.0)	-
<i>Chlamydia trachomatis L1-L3</i>	0 (0.0)	0 (0.0)	-
<i>Klebsiella granulomatis</i>	0 (0.0)	0 (0.0)	-
No pathogens detected	25 (29.8)	42 (40.8)	0.49

*p values reflect significance of difference between 2014 and 2015 data based on Fischer's exact tests

5 ENTERIC DISEASES

a *Salmonella* Typhi cases in South Africa, 2016

A case series of 19 cases of typhoid fever was reported in the Communiqué, January 2016. As of 12 February 2016, a total of 30 typhoid fever case-patients had been reported in five provinces across the South Africa (Figure 3). Diagnosis was based on the isolation of *Salmonella* Typhi in blood culture (93%, n=28) and stool specimens (7%, n=2).

The ages of these 30 case-patients range from 9 months to 52 years with a median of 11 years (IQR 7 – 30 years). A single case-patient’s age is unknown. Five (5/30; 17%) case-patients are children <5-years-old while nine are adults 20-45 years of age. Females account for 60% (n=18) of cases. Two deaths (8%) have been reported. All case patients were admitted to hospital. The average length of stay was 7.4 days. Amongst the 30 case-patients, 3 epidemiological clusters were identified through collation of data obtained from the case investigation forms as follows:

- 1) Two case patients (1 from KwaZulu-Natal Province, 1 from Western Cape Province) were cousins and had travelled together to India in the month prior to becoming ill.
- 2) Two siblings were initially admitted with clinical typhoid fever. Their care-giver was found to have a positive stool culture for typhoid fever, though she was asymptomatic at the time.
- 3) A further two siblings were identified as case-patients. Investigation of the index sibling revealed that his younger brother had a positive stool culture and was asymptomatic at the time. However, the second sibling became symptomatic and presented at hospital with a pyrexial illness and was found to have a positive blood culture for *Salmonella* Typhi before his stool culture isolates were confirmed.

Amongst the 25 case-patients in whom travel history is known, 13 (13/25; 52%) reported a history of travel outside their hometown/city within 1 month before the onset of illness.

PFGE typing (Figure 4) of 15 isolates identified 9 profiles of which 7 were unique. Eight and 3 isolates respectively shared identical profiles. The eight identical strains shared a common profile with the Zimbabwean 2012 outbreak strain. Of these eight strains, 3 had a travel history (2 to Zimbabwe and 1 to Eastern Cape Province) while 4 had no travel history and the travel history for one isolate was unknown. The three isolates with the second identical PFGE profile were obtained from two patients who were cousins and who had travelled to India over December before becoming ill. Isolates from the other two epidemiological clusters described above are not represented in Figure 4.

The concern by the health care workers, the media, the public and government structures over typhoid fever cases in South Africa and particularly in Gauteng has facilitated an in depth investigation into the epidemiology of typhoid fever in South Africa over January and February of 2016. These investigations have highlighted the need for continuing thorough case management and contact tracing in order to contain further person-to-person spread and prevent contamination of water sources, ongoing surveillance for typhoid fever, the use of more discriminatory molecular epidemiological tools to identify recent transmission events. The absence of typhoid fever cases outside of urban settings requires further investigation.

Source: Division of Public Health, Surveillance and Response, Centre for Enteric Diseases, NICD-NHLS (outbreak@nicd.ac.za)

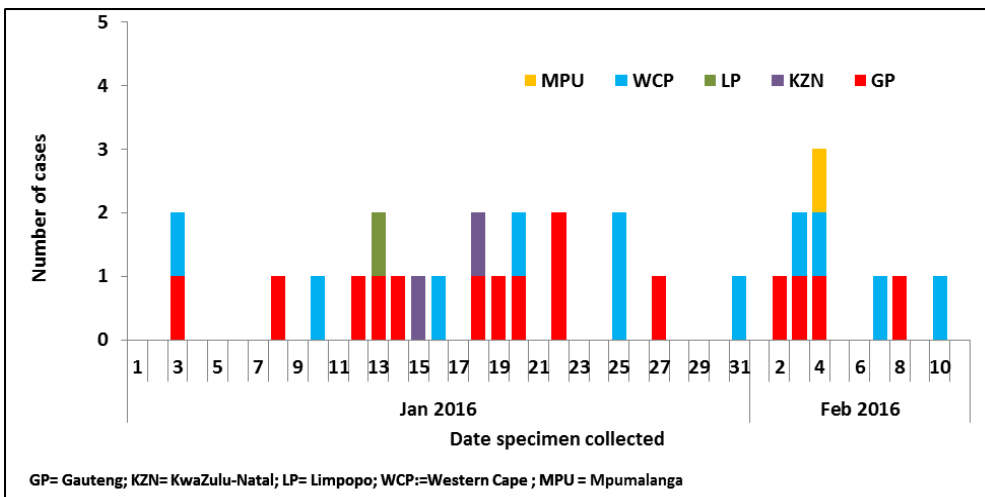


Figure 3. Number of Laboratory-confirmed typhoid fever cases identified in five South African Provinces, 1 January to 12 February 2016

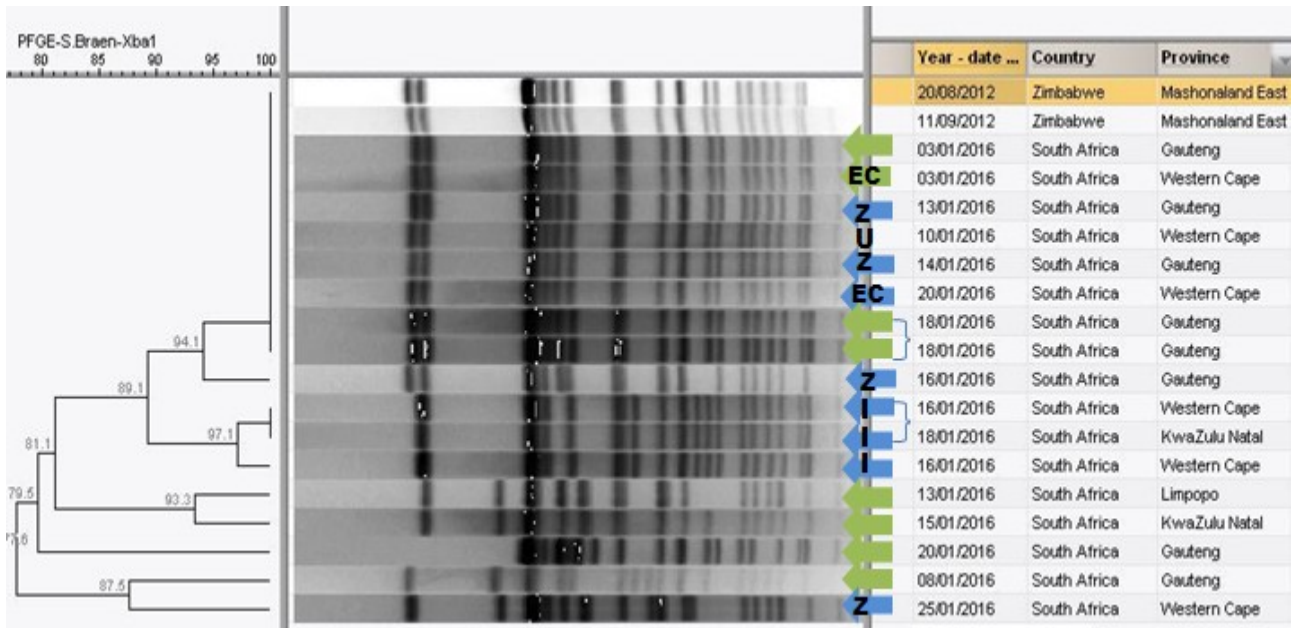


Figure 4. Pulsed-field gel electrophoresis patterns showing genetic relatedness of 15 typhoid fever isolates obtained in January 2016. Blue arrows=positive travel history within 30 days preceding isolation of the organism (destination as follows: Z=Zimbabwe, I=India). Green arrows=no positive travel history (travel history of relatives within preceding 30 days: EC=Eastern Cape)

b Botulism — a public health emergency

Case presentation

A 42-year-old male presented to a local hospital on the 8 January 2016 with sudden onset of slurred speech and difficulty in swallowing. On examination he was noted to have bilateral cranial nerve weakness, but no sensory loss or lower limb weakness. He was afebrile, fully conscious and cooperative. The following day his condition deteriorated rapidly with bilateral arm weakness; later that afternoon he developed respiratory failure requiring intubation and ventilation. He subsequently had a cardiac arrest and demised.

Due to the nature of his presentation the clinician considered botulism as part of the differential diagnosis. The patient had reported abdominal cramps and diarrhoea a week prior to presentation which resolved after treatment with buscopan. A detailed food history was not available. No other persons were affected. Serum was sent to NICD Special Bacterial Pathogens Unit for botulism toxin testing. The mouse bioassay, which is the reference method for detection of botulinum neurotoxin (1) was negative after 21 days.

Discussion

Foodborne botulism is the most common presentation of botulism. Growth of the organism

and production of botulinum toxin in foods only occurs under particular conditions, namely anaerobic, low-salt, low-acid conditions. Proper refrigeration at temperatures below 3°C retards the growth of *Clostridium botulinum*. The organism is susceptible to high salt, high oxygen, and low pH levels. The spores are heat-tolerant and will survive boiling water for an extended period of time (2), but the botulinum toxin is denatured and thus deactivated by thorough cooking at temperatures greater than 80°C. *C. botulinum* produces 7 immunologically distinct toxins, which are designated by the letters A–G. Several related clostridial species (e.g., *Clostridium baratii* and *Clostridium butyricum*) also produce botulinum toxins.

In South Africa there is little public awareness and medical knowledge about botulism (3). The diagnosis of botulism can be made rapidly if the presenting symptoms are associated with ingestion of a unique food item. A detailed history of food is essential. A single case of foodborne botulism represents a public health emergency that may warrant a thorough outbreak investigation because the contaminated food may still be available to cause illness in others. Therefore, it is critical for clinicians who suspect botulism to discuss the case

immediately with local department of health communicable disease outbreak response teams and the clinical microbiologist. Investigation of a suspected case of botulism includes an immediate search for other possible cases and identification of suspected food exposures, as well as confirmation of the diagnosis. If a number of people are affected, a rapid and detailed epidemiological investigation is warranted to assure the source is identified and controlled. Diagnostic testing of both case specimens (serum, vomit, stomach contents, stool) and foods should be performed.

References

1. Botulism. NICD Communiqué. 2015:14 (3)
2. Schantz EJ, Johnson EA. Properties and use of botulinum toxin and other microbial neurotoxins in medicine. *Microbiol Rev.* 1992 Mar;56(1):80-99.
3. Freaun J, Arntzen L, van den Heever J, Perovic O. Fatal type A botulism in South Africa, 2002. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* 2004;98(5):290-5.

Source: Groote Schuur Microbiology laboratory NHLS; Centre for Emerging and Zoonotic Diseases, NICD-NHLS; Western Cape Provincial Government, Department of Health. (colleen.bamford@nhls.ac.za)

6 INTERNATIONAL OUTBREAKS OF IMPORTANCE TO SOUTH AFRICAN TRAVELLERS AND HEALTHCARE WORKERS

a Ebola virus disease (EVD) outbreak: situation update

In Sierra Leone, after the re-emergence of EVD cases on 14 January 2016, no new laboratory-confirmed EVD cases have been reported since 21 January 2016. Associated contacts of the two recent cases reported in Sierra Leone in January 2016 after the country was declared Ebola free on 7 November 2015 have completed their 21-day monitoring period. However 48 remain untraced, of whom 18 are considered high risk. Efforts to trace the contacts are ongoing and will continue for at least a further 21 days from 3 February 2016. Guinea and Liberia were declared free of Ebola transmission on 29 December 2015 and 14 January 2016 respectively and have since entered a 90-day period of heightened surveillance. As at 14 February 2016, a cumulative total of 28 603 cases (laboratory-confirmed, probable and suspected) including 11 301 deaths with a case fatality rate of 40% has been reported in Guinea, Liberia and Sierra Leone. A summary of case numbers and deaths reported is shown in Table 3.

The EVD outbreak in West Africa had a devastating impact in the three countries (Guinea, Liberia and Sierra Leone) that experienced widespread and intense transmission. As a result, these countries still need to recover and have to build health systems that can be able to prevent, detect and respond to outbreaks. The World Health Organization (WHO) continues to support and strengthen surveillance activities in these countries. They are currently assisting the Ebola-affected countries and other countries in the region with assessing, restructuring and strengthening their integrated disease surveillance, preparedness and response systems. More details are available at

<http://www.who.int/features/2016/rebuilding-health-systems/en/#> and <http://www.who.int/csr/disease/ebola/health-systems-recovery/surveillance/en/#>

Situation in South Africa

As at 10 February 2016 there have been no EVD cases in South Africa associated with the current outbreaks in West Africa. In addition, there are no suspected cases of EVD in South Africa at present. The risk of Ebola being introduced into South Africa still remains low. However a high index of suspicion is necessary given on-going EVD transmission in West Africa.

Testing for viral haemorrhagic fever viruses (including Ebola virus) in South Africa is only available at the NICD. EVD testing is neither warranted nor useful for persons that are not suffering from a clinical illness compatible with EVD, even in the event of compatible travel histories. The tests cannot be used to determine if the patient has been exposed to the virus and may develop the disease later. Requests for testing (with a detailed clinical, travel and exposure history) should be directed to the NICD Hotline at 082 883 9920 (a 24-hour service, for healthcare professionals only).

Source: Division of Public Health Surveillance and Response, NICD-NHLS; (outbreak@nicd.ac.za)

Table 3. Number of Ebola virus disease cases and deaths in Guinea, Liberia and Sierra Leone (as at 14 February 2016)

Country	Total cases (laboratory-confirmed, probable and suspected)	Total deaths
Guinea	3 804	2 536
Liberia	10 675	4 809
Sierra Leone (as at 7 November 2015)	14 122	3 955
Sierra Leone (from 14 January 2016)	2	1
Total	28 603	11 301

Source: World Health Organization: Ebola outbreak - Ebola situation report of 17 February 2016 (www.who.int)

b The current Lassa fever outbreak in Nigeria

The Lassa fever outbreak in West Africa continues to gather momentum. ProMED reported on 13 February 2016 that Nigeria has recorded 176 cases with 108 deaths - a case fatality rate of 61 percent. Of the total, 78 cases have been confirmed, amongst which 49 have died (63%). As of 13 February, 20 states are currently following up contacts, or have suspected or probable cases with laboratory results pending or laboratory confirmed cases. The states with the highest number of deaths include Niger, Taraba, Bauchi, Kano, Edo and Ondo. Cases have been detected in Abuja and Lagos.

Lassa fever is caused by an arenavirus, Lassa fever virus (LFV), and was first identified in humans after the death of two American missionary nurses in the town of Lassa in northern Nigeria in 1969. The main reservoir of LFV is the common African multimammate rat *Mastomys natalensis*. These rats associate closely with humans and are commonly found in and around rural households and food storage throughout the entire continent. The virus is shed in urine and faeces of infected rats for possibly the whole lifetime of the animal (2-3 years). Transmission to humans occurs easily by inhalation or ingestion of dried excreta (e.g. while sweeping), touching of infected excretions, urine or contaminated water, floors or other surfaces with hands, especially when having cuts or sores or with mouth, eyes or nose. Secondary person-to-person infection in the household and community and both hospital and laboratory transmission can also occur

via contact of infected blood, saliva or urine (3-9 weeks)

The risk of Lassa fever correlates strongly with areas having 1 200-1 500mm rain per year, and seasonality. It is postulated that environmental conditions that favour the survival of the virus outside the vertebrate host promote acquisition of infection by rodents (in the moist, rainy season), and by humans (viral aerosol stability during the dry season). Outbreaks in humans peak from November to March each year. Vegetation and temperature are less important ecological factors determining areas of risk. In geographical and political terms, areas of risk in West Africa are shown in Figure 6 and include Guinea (Kindia, Faranah and Nzerekore), Liberia (Lofa, Bong and Nimba), Nigeria (southern two-thirds of the country) and Sierra Leone (Kenema and Kailahun).

Lassa fever does not occur in South Africa, although the vertebrate host has a widespread distribution across Africa. It is postulated that several other arenaviruses that are not pathogenic in humans exploit the environmental niche in African multimammate rats elsewhere across the continent, thus preventing the spread of the disease. However, several cases of Lassa fever have been imported to South Africa in returning travellers. A public health physician involved in an immunisation campaign in Nigeria in 2007 became ill and was evacuated to Johannesburg. A diagnosis of Lassa was confirmed. A South African working in

Sierra Leone died after contracting the virus in Makeni, northern Sierra Leone in 2010.

The incubation period of Lassa fever is 6-21 days. Onset is gradual with high fever, rigors, shivering, malaise, headache, myalgia and sore throat. Cough, chest pain, and cramping abdominal pains with nausea, vomiting, and diarrhea may also occur. Most deaths are the result of multi organ failure, usually 2 weeks after the onset of illness. Deafness

is a common complication in one-third of infections in mild as well in severe cases and presents in various degrees and is often permanent. The differential diagnosis of Lassa fever includes malaria, typhoid, Ebola infections.

Source: Division of Public Health, Surveillance and Response, NICD-NHLS (outbreak@nicd.ac.za)

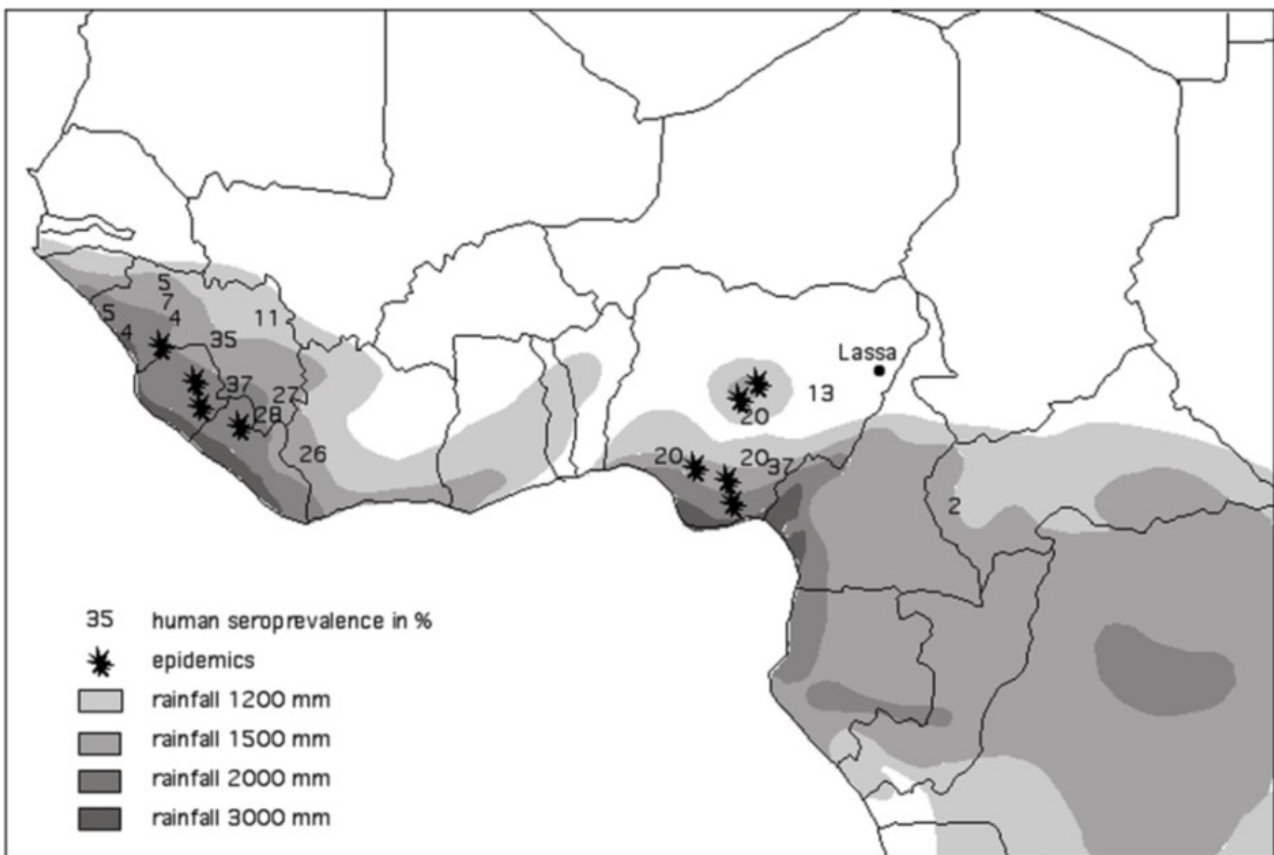


Figure 5. West and Central Africa mean annual rainfall (1951-1989), Lassa fever nosocomial outbreaks (stars) and human Lassa seroprevalence (numbers in %). From: Fichet-Calvet E, Rogers DJ. Risk maps of Lassa fever in West Africa. PLoS Neg Trop Dis. 2009. 3 (3):3388.

7 SURVEILLANCE FOR ANTIMICROBIAL RESISTANCE

a Update on carbapenemase-producing Enterobacteriaceae

CPE have become a threat to healthcare and patient safety worldwide by compromising empiric antibiotic therapeutic choices and increasing morbidity, hospital costs and the risk of death. CPE surveillance is required to determine the extent of the problem as a first step in order to restrain the emergence and spread of CPE. The Johannesburg Antimicrobial Resistance Laboratory and Culture Collection (AMRL-CC) of the Centre for Opportunistic, Tropical and Hospital Infections (COTHI) at the NICD test referred isolates of suspected carbapenemase-producing Enterobacteriaceae (CPE) for the presence of selected carbapenemase genes. In January 2016, a total of 81 Enterobacteriaceae isolates was received. Seventy-seven isolates were screened, 61 of which expressed carbapenemases (Table 4 and Table 5). The majority of these CPE isolates were *Klebsiella pneumoniae* (43) followed by *Enterobacter cloacae* (7).

It is important to note that these figures do not represent the current burden of CPEs in South Africa. Given that CPE infections are currently not reportable or notifiable in South Africa, there is no platform for appropriate surveillance reports and consequently no locally representative data is available. This is of major concern as meaningful data can inform public health policy and highlight priorities for action. Controlling the spread and limiting the impact of CPEs in South Africa will require intensive efforts in both the public and private healthcare sectors going forward. NHLS and private laboratories are encouraged to submit suspected CPE isolates based on antimicrobial susceptibility testing (AST) criteria to AMRL-CC, NICD/NHLS. Please telephone (011) 555 0342/44 or email olgap@nicd.ac.za; for queries or further information.

Source: Centre for Opportunistic, Tropical, and Hospital Infections, NICD-NHLS; (olgap@nicd.za.za)

Table 4. Enterobacteriaceae by CPE enzyme type, AMRL-CC, COTHI, NICD, January–December 2015 and January 2016

Organism	NDM		OXA-48 & Variants	
	Jan-Dec-15	Jan 2016	Jan-Dec-15	Jan 2016
<i>Enterobacter cloacae</i>	23	4	15	3
<i>Escherichia coli</i>	14	-	41	3
<i>Klebsiella pneumoniae</i>	295	19	161	24
<i>Providencia rettgeri</i>	23	3	-	-
<i>Serratia marcescens</i>	47	3	6	2
Grand total	402	29	223	32

NDM: New Delhi metallo-beta-lactamase; **OXA:** oxacillinase.

Table 5: Enterobacteriaceae isolates by specimen type and province, AMRL-CC, CO THI, NICD, January–December 2015 and January 2016

Organism	EC	FS	GA	KZ	WC	Total Jan 2016	Total Jan-Dec 2015
<i>Enterobacter cloacae</i>	4	-	6	2	4	16	111
Non-sterile	2	-	1	1	-	4	19
Sterile	2	-	4	-	4	10	75
Unknown	-	-	1	1	-	2	15
Not stated	-	-	-	-	-	-	2
<i>Escherichia coli</i>	1	-	1	-	1	3	66
Non-sterile	1	-	1	-	-	2	9
Sterile	-	-	-	-	1	1	50
Unknown	-	-	-	-	-	-	7
Not stated	-	-	-	-	-	-	-
<i>Klebsiella pneumoniae</i>	5	1	25	12	6	49	543
Non-sterile	2	-	4	1	1	8	81
Sterile	3	-	20	6	5	34	333
Unknown	-	1	1	5	-	7	123
Not stated	-	-	-	-	-	-	6
<i>Providencia rettgeri</i>	1	-	2	-	-	3	24
Non-sterile	-	-	-	-	-	-	1
Sterile	1	-	2	-	-	3	14
Unknown	-	-	-	-	-	-	9
Not stated	-	-	-	-	-	-	-
<i>Serratia marcescens</i>	1	-	-	4	-	5	55
Non-sterile	-	-	-	1	-	1	-
Sterile	1	-	-	-	-	1	12
Unknown	-	-	-	3	-	3	42
Not stated	-	-	-	-	-	-	1
Total	12	1	34	18	11	76	799

8 TOXIN INGESTION

a Cases of suspected scombroid poisoning following fish ingestion in Gauteng Province

A number of cases of possible scombroid fish poisoning have been identified by private physicians in Johannesburg: A 37-year-old male presented with nausea and vomiting, abdominal pain, hot flushes, tingling of the lips, severe malaise and hypotension (blood pressure 80/60 mmHg) following ingestion of fish at a well-known fish restaurant chain. A 52-year-old male presented with severe vertigo, nausea, vomiting and hot flushes after a fish meal, also eaten at a well-known fish restaurant chain. Similar symptoms (flushing, abdominal discomfort, nausea and vomiting, numbness of the lips) were reported by a family on the West Rand, who ate fish obtained from a fishery.

Scombroid fish poisoning occurs after eating fish that contains high levels of histamine because it has been poorly preserved. Certain fish in the Scombridae family – including mackerels, tuna, bonitos, sardine, anchovy and herring, contain large amounts of the amino acid histidine. At

temperatures above 16°C, histidine is converted to histamine by contaminating bacteria. When ingested, the histamine provokes symptoms identical to those of an allergic reaction – flushing of the face and upper body, severe headache, palpitations, itching, abdominal cramps and diarrhoea. In severe cases, hypotension, arrhythmia and respiratory compromise may occur and require hospital admission and supportive care. Treatment is with anti-histamines. Symptoms are usually self-limiting and last for 10-14 hours. Fish contaminated with histamine has a peppery, sharp, or salty taste, and a bubbly feel, though it may also taste normal. Histamine is temperature stable, so cooking, smoking, canning or freezing will not destroy histamine, nor render fish safe to eat. Prevention of scombroid is through appropriate preservation of fish at temperatures <3.3°C.

Source: Division of Public Health, Surveillance and Response, NICD-NHLS (outbreak@nicd.ac.za)

9 BEYOND OUR BORDERS

The 'Beyond our Borders' column focuses on selected and current international diseases that may affect South Africans travelling abroad. Numbers correspond to Figure 6 on page 20.

1. Pope Francis visiting Mexico

Pope Francis will be visiting Mexico from 12-18 February 2016 and the assembling of large crowds at many occasions is expected. Mass gatherings such as these pose significant health and safety risks for attendees. Transmission of communicable diseases is likely and accidents leading physical injury are a persistent risk. Mexico is currently experiencing a Zika virus outbreak amidst the larger on-going outbreak in Central and South America. The potential for further spreading of the Zika virus is present. The USA CDC has issued special recommendations for pregnant women traveling to Mexico in addition to specific safety recommendations pertaining to mass gatherings. (<http://wwwnc.cdc.gov/travel/page/travel-to-mass-gatherings>)

2. Yellow Fever in Angola

A yellow fever outbreak is currently ongoing in Angola. The latest cases have occurred in Huila province, close to the Namibian border where 8 deaths have been reported amongst 41 suspected cases. As of 12th February, there have been over 250 suspected cases and 51 registered deaths. Viana municipality in Luanda Province is the most affected, with 92 cases and 29 deaths, followed by Huila Province. Travellers to Angola need a yellow fever vaccine at least 10 days prior to their trip, including official certification. General mosquito bite prevention steps are also advised. Persons travelling to South Africa from Angola require a valid yellow fever vaccination certificate

3. Northern hemisphere seasonal influenza

The Global Influenza Programme monitors influenza activity worldwide and publishes an update every two weeks. They reported an increase in levels of influenza activity in the temperate zones of the northern hemisphere on 08 February 2016. Influenza A(H1N1)pdm09 is the most detected virus.

Measures to help prevent influenza virus infections during travel include avoiding close contact with sick people, washing hands often with soap and water, using an alcohol-based hand sanitizer, avoiding touching one's eyes, nose, and mouth. See article on page 9 regarding preparation for the southern hemisphere influenza season.

4. Human infection with avian influenza A (H7N9) virus – China

From 21 December 2015 to 25 January 2016, 28 laboratory-confirmed cases of human infection with avian influenza A (H7N9) virus, including five deaths, were reported from China. Cases originated from 6 provinces and no clusters were found. The majority (25 cases, 89%) reported exposure to live poultry or live poultry markets.

WHO advises that travellers to countries with known outbreaks of avian influenza should avoid

poultry farms, contact with animals in live bird markets, entering areas where poultry may be slaughtered, and contact with any surfaces that appear to be contaminated with animal or poultry faeces. Travellers should practise regular hand washing with soap and water and should follow good food safety practices at all times.

5. Ebola - Liberia, Sierra Leone, Guinea

See article on page 14.

6. Zika virus –Thailand

Several cases of Zika virus infection have been reported in Thailand. Women who are pregnant or planning a pregnancy and their partners who had hoped to visit Thailand may wish to reconsider their travel plans. Zika virus is a mild infection and asymptomatic in over 75% of persons. Acquisition of Zika can be prevented through prevention of mosquito bites. See article on page 2.

7. Zika virus in South America and Caribbean

See article on page 2.

Source: Division of Public Health Surveillance and Response, NICD-NHLS (outbreak@nicd.ac.za)



Figure 6. Current outbreaks that may have implications for travellers. Number correspond to text above. The red dot is the approximate location of the outbreak or event