

Communicable Diseases Communiqué

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Rift Valley fever update

An additional five laboratory-confirmed Rift Valley fever (RVF) cases have been identified since our last update. All of these cases are farm workers and laboratory confirmation was by serology. Four of the cases are from the Heidelberg area, Western Cape Province; this brings the total to seven cases in the area since the beginning of August. The most recent case reported onset of illness on 26 August 2010. The fifth additional case was reported from Victoria West, Northern Cape Province; date of symptom onset is currently unknown.

As of 17 September 2010, a cumulative total of 237 laboratory-confirmed human cases has been identified since the start of the epidemic in February 2010. Of the cases with known occupations, the majority (82%, 182/222) work within occupations where direct contact with animals frequently occurs. Furthermore, 94% (195/208) of the cases reported direct contact with RVF-infected ruminants prior to the onset of symptoms. Infrequent transmission by mosquito vectors and/or unpasteurised milk has also been observed. There remains much concern over a possible re-emergence of the outbreak in previously affected areas accompanying the seasonal increase in temperature and rainfall. It is therefore important that individuals involved in the livestock industry use appropriate personal protective equipment, especially when performing high-risk procedures,

which include: handling of animal tissue during slaughtering or butchering, assisting with animal births, conducting veterinary procedures and disposal of carcasses or fetuses. In addition, the unsafe consumption of fresh blood, raw milk or animal tissue in epizootic regions must be discouraged; all animal products (blood, meat and milk) should be thoroughly cooked before eating. Protection against mosquito bites through the use of insect repellents (containing 30-50% DEET), insecticide-treated bed nets, and wearing of light-coloured clothing are extra preventive measures.

Healthcare workers should continue to suspect RVF in patients meeting the case definition (see guidelines at www.nicd.ac.za/outbreaks/rvf/rvf_outbreak.htm) and submit specimens to the NICD for laboratory testing. Additionally, healthcare workers should bear in mind that certain RVF-complications often manifest a few weeks after the acute infection; meningoencephalitis may present up to 4 weeks later, and ocular complications (notably retinitis) may present weeks to months later. In such cases, the acute infection may have been extremely mild and not have been diagnosed initially.

Source: SA-FELTP, Special Pathogens and Outbreak Response Units, NICD; Departments of Health, and Agriculture, Forestry and Fisheries

Influenza

Viral Watch surveillance

The number of specimens collected from Viral Watch sites per week has started to decline, with only 75 and 54 specimens collected for the weeks ending 5 and 12 September, respectively. Over the past 26 years, the influenza season has lasted between 7 and 19 weeks (mean 10 weeks). This year, the season started in week 23 (week ending

13 June) and has lasted 14 weeks to date. By the week ending 12 September 2010, 1751 samples had been tested for influenza. Of these, 756 (43.2%) were positive for influenza. The majority, 410/756 (54.2%) were positive for influenza B, 208/756 (27.5%) were positive for influenza A H3N2 and 138/756 (18.3%) for influenza A H1N1 (2009). The

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peak detection rate for influenza was 64.6% at week 33 (week ending 20 August).

SARI surveillance

By the week ending 5 September 2010, 3114 samples had been tested for influenza and other respiratory viruses. Of these, 218/3114 (7%) were positive for influenza. The majority, 147/218(67%) were positive for influenza B, 63/218(29%) were positive for influenza A H3N2 and 8/218(4%) were

positive for influenza A H1N1 (2009). The peak detection rate for influenza in SARI patients was much lower than the detection rate from Viral Watch patients, with the peak detection rate of 29% at week 27 (week ending 9 July), followed by a smaller peak detection rate of 27% at week 31 (week ending 6 August).

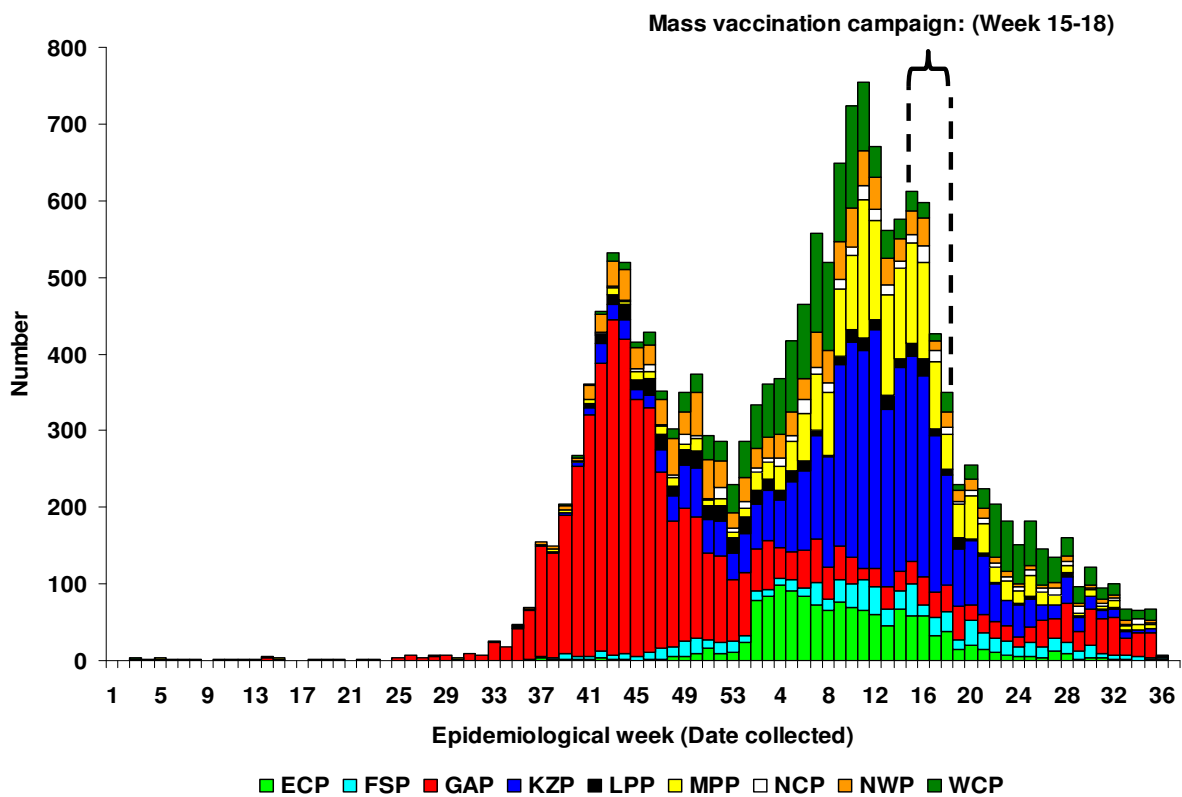
Source: Surveillance and Epidemiology Division, Respiratory Virus and Virus Diagnostic Units, NICD

Measles update

There have been 286 additional laboratory confirmed measles cases since the last published Communiqué, bringing the total to 17 640 cases from the beginning of 2009 to 7 September 2010. Cases have been reported from all nine provinces, with Gauteng (31%, 5 405/17 640), KwaZulu-Natal (24%, 4 168/17 640) and Western Cape (11%, 1 929/17 640) provinces accounting for the highest proportions of the total (Figure). Children aged < 1

year account for 35% (5 814/16 765) of cases, with 21% occurring in those aged 6 to 9 months. Although the measles outbreak is ongoing, there is a trend towards decreasing numbers of new cases reported each week.

Source: Divisions of Epidemiology and Virology, NICD



Province abbreviations: ECP=Eastern Cape; FSP=Free State; GAP=Gauteng; KZP=KwaZulu-Natal; LPP=Limpopo; MPP=Mpumalanga; NCP=Northern Cape; NWP=North West; WCP=Western Cape

Figure: Measles IgM positive results per province: South Africa, January 2009 to 7 September 2010

Meningococcal disease

Sporadic cases of meningococcal disease continue to be reported across the country. The numbers of cases are expected to increase during June and July, and to peak during the months of August to October. Laboratory-based reporting has inherent delays, so not all clinical cases may be reflected in our reports for this month.

By the end of epidemiological week 35 (week ending 3 September), a total of 251 laboratory-confirmed cases was reported to the Respiratory and Meningeal Pathogens Reference Unit (RMPRU), NICD (Table). These cases showed diversity in serogroups, which is in keeping with sporadic endemic disease in the country. Serogroup data are available for 172/251 (68%) of cases. Serogroups B and W135 have been identified most commonly this

year (61/172, 35% serogroup B and 75/172, 44% serogroup W135). Other serogroups included: A (1%, 2/172), C (7%, 12/172) and Y (14%, 21/172).

The winter and spring seasons are when numbers of meningococcal disease cases typically increase. As such, there should be a high index of suspicion for meningococcal disease, which may present with nonspecific early signs and symptoms. Disease typically has a rapid progression and should be managed as a medical emergency in order to reduce morbidity and mortality.

Source: Respiratory and Meningeal Pathogens Reference Unit, NICD

Table: Number of laboratory-confirmed meningococcal disease cases reported by epidemiological week 35, 2009 and 2010, by province

Province	2009	2010
Eastern Cape	21	16
Free State	10	14
Gauteng	145	127
KwaZulu-Natal	22	13
Limpopo	2	6
Mpumalanga	34	14
Northern Cape	6	16
North West	14	8
Western Cape	51	37
South Africa	305	251

Viral haemorrhagic fevers

There have been no new laboratory-confirmed cases of viral haemorrhagic fevers since the last published Communiqué.

A total of 3 Crimean-Congo haemorrhagic fever (CCHF) cases has been confirmed for South Africa for 2010 to date. The cases originated from Free

State (n=1) and the Northern Cape (n=2) provinces. In addition, two cases have been reported from Namibia for 2010 to date.

Source: Special Pathogens Unit, NICD

Rabies update

Several cases of rabies have been confirmed in domestic dogs in the greater Johannesburg area in the past few weeks. The affected areas include Sophia Town, Randfontein, Kibler Park and Roodepoort. These animals were pets and the source of exposure of these animals is unclear. The isolates were characterized and shown to be of the canid biotype originating from KwaZulu-Natal Province. Cases of rabid dogs were also confirmed in May 2010 in Witpoortjie (Roodepoort), and in Linden in 2009.

To date, a total of 9 human rabies cases has been laboratory confirmed for South Africa for 2010 to date. These cases originated from Mpumalanga (n=1), KwaZulu-Natal (n=3), Eastern Cape (n=2) and Limpopo (n=3) provinces. Each death is a public health failure and could have been prevented by timely and appropriate PEP. It is likely that the actual number of rabies-related deaths is significantly higher than this, since rabies is not always considered in the differential diagnosis of patients with fatal encephalitis.

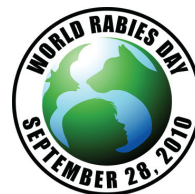
September 28 is World Rabies Day, a global initiative to raise awareness of this often neglected disease (visit www.worldrabiesday.org for more information). Despite being virtually preventable in modern times, the age-old scourge of rabies still accounts for an estimated 55 000 human deaths per year globally. The overwhelming brunt of disease is borne in the developing countries of Asia and Africa, with an estimated 31 000 and 24 000 cases respectively per year (Source: World Health Organization). These figures, however, almost certainly underestimate the true burden of disease, as it is believed that rabies is grossly underreported in developing countries. In a study on human dog-bite incidence in Tanzania, the extrapolated number of human rabies cases exceeded the reported number of cases 100-fold.¹

The alarming occurrence of such large numbers of cases may be attributed to lack of control of the dis-

ease in animals in the face of many competing priorities, as well as issues related to rabies post-exposure prophylaxis (PEP), including: lack of awareness of the need for and protocols of PEP, and the availability and accessibility of modern biologicals for rabies PEP. In South Africa between 5 and 31 human cases have been laboratory-confirmed annually since 1983. The majority of animal and human cases are reported from the coastal provinces of KwaZulu-Natal and the Eastern Cape. Nevertheless, all provinces in South Africa are considered endemic for rabies, since cases have been reported in domestic and/or wild animals in all provinces (refer to NICD-NHLS Communiqué Volume 9, No. 8 Additional Issue for recent outbreak information). From a public health perspective the domestic dog remains the most important vector of rabies to the human population, with very few cases linked to exposure to other domestic animals (such as cats) or wildlife. It is crucial to consider rabies PEP for all animal-bite victims. By evaluating the particulars of each case, i.e. the nature of the exposure, the health of the animal involved etc. the healthcare worker should determine the need for rabies PEP (refer to PEP guideline included).

References:

1. Cleaveland S, Fevre EM, Kaare M, Coleman PG. Estimating human rabies mortality in the United Republic of Tanzania from dog bite injuries. Bull World Health Organ 2002; 80: 304-310



Source: Special Pathogens and Outbreak Response Units, NICD; Rabies Laboratory, Ondestepoort Veterinary Institute

Fatal Epstein-Barr virus

Epstein-Barr virus (EBV) was identified as the likely cause of illness in two young adult male siblings who died in a Johannesburg hospital in September. The

onset of illness was 10 days apart. Both brothers presented as acute febrile illness with pharyngitis

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and tender submental lymphadenopathy. No pharyngeal membrane was noted. The course of illness in both patients was similar and progressed over a week with the development of a generalized morbilliform rash, hepatosplenomegaly, hepatic failure, metabolic acidosis, renal failure, DIC with bleeding, ARDS and CNS pathology (convulsions in one patient, blurred vision in the other). Key laboratory features included the presence of atypical lymphocytes suggestive of EBV with a monocytosis and thrombocytopenia, and marked transaminitis and conjugated hyperbilirubinaemia. EBV was suspected on the basis of these findings, but initial test results showing a low EBV viral load with inconclusive EBV serology, the unusually severe clinical course, and a history of travel to game farms and potential exposure to animals there, raised concerns about the possibility of alternate diagnoses including the haemorrhagic fever viruses. This prompted a formal outbreak intervention with contact tracing and monitoring and the introduction of infection prevention and control procedures as for VHF. Extensive tests for the agents of VHF on serum

and tissue samples were negative by PCR and serology. A liver biopsy showed submassive necrosis and will be further reviewed. Hyper-reactive serum with positive antibody results for a number of infectious agents, notably leptospirosis, confounded the investigations. Repeat EBV viral load testing confirmed the presence of very high numbers of EBV copies. One of the patients had undergone an autologous bone marrow transplant more than 14 years previously for non-Hodgkins lymphoma, but had been in remission for 10 years and was not on any immunosuppressives. Both brothers had otherwise been well with no other evidence of recurrent or other severe infections. Further testing will be done to identify genetic markers associated with severe EBV infections. Rare X-linked lymphoproliferative disorders have been described in association with severe and fulminant EBV infection.

Source: Outbreak Response, Special Pathogens, Special Bacterial Pathogens, FELTP and EM Units, NICD; Ampath and Lancet laboratories; Anatomical Pathology Unit, NHLS and University of the Witwatersrand; Specialist Physicians.

West African trypanosomiasis

A 40-year-old immigrant from Democratic Republic of Congo (DRC) presented to a Cape Town hospital following a seizure. He had flown to South Africa one year previously from his home in Kibibi village, Bandudu (DRC) and had been well until four months prior to admission. His friend had noted a reduction in his cognitive abilities, more recent somnolence, with fever and shivering for the week prior to admission. On admission to hospital, slowed mentation, cervical lymphadenopathy and a temperature of 38.4°C was noted. A lumbar puncture showed 49 lymphocytes/mm³, total protein of 0.77g/L and normal glucose level. Gram stain, cryptococcal latex antigen test and TB microscopy were negative. Chest x-ray showed bilateral hilar adenopathy, but no pulmonary infiltrate. Syphilis serology was negative, as was a rapid test for HIV (subsequently confirmed by fourth generation ELISA). He was started on empiric therapy for TB meningitis with Rifafour® and prednisone.

Having failed to respond to therapy, he was transferred to Groote Schuur Hospital one month after

starting TB treatment. On presentation, he was somnolent, had markedly slowed speech, and was disorientated to time, place and person. He had a Parkinsonian tremor in his upper limbs, but no other focal neurological deficit. Posterior cervical triangle lymphadenopathy was present and there was no change in the chest x-ray appearance. Repeat lumbar puncture showed 20 lymphocytes/mm³, 14 polymorphs/mm³, protein 0.57g/L and a normal glucose level.

In view of his country of origin and the constellation of symptoms (increased somnolence, decreased mentation, basal ganglia involvement, and posterior cervical adenopathy -Winterbottom's sign) a clinical diagnosis of stage II West African trypanosomiasis (WAT) was made. This was confirmed on examination of a blood buffy coat sample which showed a single trypomastigote. Neither lymph node aspirate nor CSF showed evidence of trypomastigotes. Morula cells (cells of Mott), were abundant in the CSF, which had an elevated IgM level. The card

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agglutination test for trypanosomiasis (CATT) confirmed disease caused by *Trypanosoma brucei gambiense*. Eflornithine was procured through the World Health Organization programme for Neglected Tropical Diseases (Geneva) and he was treated with 100mg/kg i.v. 6 hourly for 14 days. He made an excellent clinical recovery, being fully conversant, mobile and without tremor on discharge.

Two weeks later, a 24-year-old man from Fasaka village near Kikwit in Kwilu Province (DRC) was referred to hospital from a local psychiatric unit. He had immigrated in 2007, travelling overland through Zambia and Zimbabwe. He became unwell in December 2009, with increasing somnolence, reversal of sleep patterns and aggression at night, inability to study, and progressive difficulty pronouncing words. He had lost <10% of his body weight over a 5 month period and had a history of dagga (cannabis) abuse until 3 months prior to admission. He was difficult to rouse from sleep, showed signs of weight loss, posterior cervical lymphadenopathy, cogwheel rigidity of the upper limbs and slight splenomegaly. A screen for substance abuse, including amphetamines, cannabinoids and opiates was negative. Lumbar puncture revealed 555 lymphocytes/mm³, glucose 0.3mmol/L and protein 1.63g/L. Multiple morula cells were evident as was a single trypomastigote in a concentrated specimen of 10ml CSF. Buffy coat and lymph node aspirate were negative for trypomastigotes. CATT results are awaited and treatment is being initiated.

Unlike East African trypanosomiasis caused by *T. b. rhodesiense*, WAT is generally a more chronic, indolent infection, which may present months to years

after being bitten by an infected tsetse fly. Stage I (haemolymphatic) disease occurs when parasites are disseminated, and presents weeks to months later with fever which can be recurrent. A pruritic irregular circinate rash often occurs; lymphadenopathy develops later, including the characteristic Winterbottom's sign. Hepatosplenomegaly is variably present.

Stage II disease (meningoencephalitic) marks the invasion of trypomastigotes into the CNS. Irritability, personality changes and inability to concentrate are often early signs, followed by the characteristic development of reversal of sleep patterns and progressive somnolence. Extrapyramidal signs often occur, with a range of manifestations including choreiform movements, tremors, fasciculations, ataxia and Parkinsonism.

The final phase is marked by progressive neurological deterioration, eventual coma and death (which may be due to bacterial super-infection or aspiration pneumonia). A thorough travel history is vital in such cases, and as South Africa has a large immigrant population from many countries that are endemic for WAT, an increased index of suspicion of WAT is required in any patient presenting with psychiatric symptoms, abnormal sleep patterns or somnolence, or meningoencephalitis who hails from a WAT-endemic region. CSF findings of stage II WAT may mimic tuberculosis or viral meningoencephalitis, and since TB is prevalent in South Africa, there is a high probability that cases of WAT may be misdiagnosed as TB meningitis.

Source: Division of Infectious Diseases and HIV Medicine, Department of Medicine, Groote Schuur Hospital, Cape Town; Outbreak Response Unit, NICD.

Salmonella Heidelberg foodborne illness outbreak

On Tuesday 17 August, the Department of Health was informed of a possible foodborne illness outbreak amongst persons attending a funeral at Tjakastad, Albert Luthuli Sub-district, Mpumalanga Province. The funeral took place on Friday 13 August; later the same day, 4 people presented to Tjakastad Community Healthcare Centre (CHC) complaining of diarrhoea, abdominal cramps and vomiting. A further 13 people presented to the CHC over the weekend (of which one required referral for hospital admission), and another 17 people were

attended to at Embhuleni Hospital. A total of 12 patients required admission to hospital, one of whom (a 71-year-old female) died shortly after admission. Rectal swab specimens were collected from 10 patients and submitted to Embhuleni Hospital NHLS laboratory for testing.

The meal served at the funeral included cabbage, pumpkin, porridge, chicken stew, mashed potatoes, cake and mageu. The Environmental Health

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Practitioner submitted food samples to the NHLS Infection Control Services Laboratory in Johannesburg. Non-typhoidal *Salmonella* spp was isolated from 7 of the 10 rectal swab specimens, as well as 5 food samples (cabbage, raw and mashed potatoes, cake and mageu). The Enteric Diseases Reference Unit at the NICD further characterised the isolates; all were identified as *Salmonella* Heidelberg, having identical PFGE patterns. Additional foodborne pathogens were detected in two food samples: toxin-producing *Bacillus cereus* and enterotoxin C-producing *Staphylococcus aureus* from the chicken stew, and toxin-producing *B. cereus* from the cake. salmonellosis is a major cause of bacterial enteric illness in both animals and humans, and the association of the disease in farm animals with cases and outbreaks of human infection is gaining increasing recognition worldwide. *S. Heidelberg* has a predilection for infecting poultry, and numerous outbreaks related to contaminated poultry meat products and eggs have been reported in the literature. Although it is one of the top five serotypes isolated from human Salmonellosis in the US and Canada, it is an uncommon human-related serotype in South Africa. A recently published study reported that *S. Heidelberg* is the 6th most common serotype isolated from food animals in South Africa, poultry accounting for 99% of isolates.¹ It is likely

that contaminated chicken meat or eggs were used in the funeral meal, and that cross-contamination during/after food preparation by foodhandler/s resulted in the numerous food types harbouring *Salmonella*. Health education regarding food safety is critical to preventing foodborne illness outbreaks, and the WHO's 'Five keys to safer food' pamphlet² promotes practices that are easily implemented in most settings, namely: keep clean, separate raw and cooked, cook thoroughly, keep food at safe temperatures, and use safe water and raw materials. All healthcare practitioners should have knowledge of and promote food safety whenever the opportunity arises.

References:

1. Kidanemariam A, Engelbrecht M, Picard J. Retrospective study on the incidence of *Salmonella* isolations in animals in South Africa, 1996 to 2006. *J S Afr Vet Assoc* 2010 Mar; 81(1):37-44.
2. http://www.who.int/foodsafety/publications/consumer/en/5keys_en.pdf

Source: Department of Health, Mpumalanga; NHLS Infection Control Services Laboratory; Outbreak Response and Enteric Diseases Reference Units, NICD.

Beyond Our Borders: infectious disease risks for travellers

The "Beyond Our Borders" column focuses on selected and current international diseases that may affect South Africans travelling abroad.

Disease & Countries	Comments	Advice to travellers
Anthrax: Bangladesh	As of 13 September 2010, at least 495 people have contracted anthrax since the outbreak began in mid-August 2010. Cases have originated from 8 Upazila Parishad (sub-districts), where infected livestock (most frequently cattle) have been identified. A number of reports document that meat from deceased or dying cattle is shared among village families, accounting for the large scale and rapid spread of human infections.	Anthrax is transmitted from animals to humans by ingestion, inhalation or handling of infected animal products. Travellers are advised to avoid contact with animals or animal products within high-risk areas. Vaccines are not available to the general public.

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Disease & Countries	Comments	Advice to travellers
<u>Hand-foot-and-mouth disease (HFMD):</u> Asia	Since the beginning of 2010, an increasing number of HFMD cases have been reported throughout Asia. As of 13 May 2010, 77 000 cases and 40 deaths were reported in China alone.	There is no vaccine available to prevent HFMD, and management of disease focuses on the treatment of symptoms (esp. fever). Travellers to countries currently experiencing outbreaks are advised to wash hands often with soap and water, especially before eating, after coughing or sneezing and after going to the bathroom. Consider packing and regularly using an alcohol-based hand gel (minimum 60% alcohol). Avoid sharing eating utensils/cups.
<u>Poliomyelitis:</u> Angola and Democratic Republic of Congo	Angola and DR Congo are currently experiencing outbreaks of wild poliovirus type 1. These outbreaks have been ongoing since April 2007; however, recent reports indicate the outbreaks have spread to re-infect areas previously classified as polio-free. The WHO has warned that there is currently a high risk of international spread from these countries, given the limited impact of control measures to date and historical cross-border spread of communicable diseases from these two countries.	Travellers who have previously received three or more doses of OPV or IPV should be offered a booster dose of polio vaccine before departure. Non-immunised individuals require a complete course of vaccine. It is also important to note that vaccination does not guarantee the travellers' safety. Travellers are additionally advised to follow safe food and water practices, and practice good hand hygiene to prevent infection.
<u>Influenza:</u> Southern Hemisphere and southern Asia	As of 10 September 2010, the WHO Global Influenza Programme reported influenza activity to be most intense in the temperate areas of the Southern Hemisphere and southern Asia. India is currently experiencing a countrywide outbreak of H1N1 (2009). Chile has reported an unusually later peak to their influenza season. Australia and New Zealand are reporting stabilising or decreasing numbers in new infections. The Central African Republic reported their first ever detection of H1N1 (2009).	Travellers are advised to avoid close contact with people suffering from acute respiratory infections and, where possible, crowded enclosed spaces. Frequent hand-washing, especially after direct contact with ill persons or their environment may reduce the risk of infection. Ill persons are encouraged to practice good cough etiquette (maintain distance, cover coughs and sneezes with disposable tissues or clothing, wash hands).

References: ProMED-Mail (www.promedmail.org), World Health Organization (www.who.int), Centers for Disease Control and Prevention (www.cdc.gov), Europe Media Monitor (<http://medusa.jrc.it/medisys/helsinkiedition/en/home.html>); last accessed 2010/09/20.

Source: Travel Health and Outbreak Response Units, NICD.

This communiqué is published by the National Institute for Communicable Diseases (NICD), a division of the National Health Laboratory Service (NHLS), on a monthly basis for the purpose of providing up-to-date information on communicable diseases in South Africa. Much of the information is therefore preliminary and should not be cited or utilised for publication. Questions and comments may be addressed to: The Outbreak Response Unit: outbreak@nicd.ac.za; Private Bag X4, Sandringham, 2131, South Africa

