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COMMUNICABLE DISEASES SURVEILLANCE BULLETIN

MARCH 2010

NICD NHLS

FOREWORD

Given that the number of surveillance programmes has increased substantially at the NICD, it is no longer possible to include all the surveillance reports in one bulletin. For this reason it has been decided that, in contrast to previous years where most NICD surveillance reports were collated in the first bulletin of the year, we will now spread the surveillance reports throughout the year. In this March bulletin we present findings from several NICD surveillance programmes: measles, acute flaccid paralysis, respiratory viruses, viral haemorrhagic fevers, rabies and rotavirus. Results from additional programmes will be included in the May, August and November editions of the Bulletin.

2010 is an important year for South Africa and in the face of mass gatherings it is important to have robust surveillance programmes in place. Several of the diseases with reports presented in this edition of the bulletin have the potential for outbreaks, either related to mass events or seasonally coinciding with the 2010 FIFA world cup. In this context of heightened awareness the presence of baseline data from surveillance programmes is invaluable to assist in discerning between true outbreaks and seasonal increases, even given expected population increases during the event. It is also essential that surveillance programmes continue to function and are strengthened during 2010.

While South Africa remains polio free, surveillance data from the African region demonstrate that in some countries there has been an increase in wild-type polio in 2009 as compared to 2008. Due to high rates of travel within the region, South Africa remains vulnerable to the risk of polio importation. In addition, 2009 saw a widespread outbreak of measles affecting all provinces of South Africa which is still ongoing. If we wish to interrupt the transmission of measles and remain polio free it is critical that we achieve a high vaccine coverage (≥95% for measles and ≥90% for polio) in the national mass campaign planned for 2010. The first round will be from 12-23 April 2010 and will include measles vaccination for all children aged 6 months to 14 years (<15 years) as well as polio vaccine for children < 5 years. The second round will include a second dose of polio vaccine for children < 5 years as well as vitamin A and deworming for children aged 12 months to < 5 years. In addition, there will be an influenza vaccination campaign from March-May 2010 targeting designated high risk groups. Further details of the planned campaigns are available from http://www.doh.gov.za/docs/immunization/index.html. All health professionals are encouraged to support the campaign through advocacy and direct participation where possible.

Cheryl Cohen, Editor

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NATIONAL INSTITUTE FOR COMMUNICABLE DISEASES

WEB

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SUSPECTED MEASLES CASE-BASED SURVEILLANCE, SOUTH AFRICA, 2009

Epidemiology and Surveillance Unit, Specialised Molecular Diagnostics Unit, Viral Diagnostics General Isolation Unit and Serology Unit, National Institute for Communicable Diseases

The NICD is accredited by the World Health Organisation (WHO) to perform measles and rubella IgM testing for national case-based surveillance as part of the measles elimination strategy. The case definition for suspected measles is as follows: patients who present with fever ≥38ºC and rash, and at least one of the three C's (cough, coryza or conjunctivitis). Blood and urine specimens from suspected measles cases nationally are submitted to the NICD for confirmation. Approximately 60% of suspected measles cases from Free State Province are tested in that province. The numbers presented here represent specimens received by the NICD and may differ from those presented by the National Department of Health as they may receive information on cases where no specimens were taken.

All blood specimens were tested by Enzygnost (Siemens, Marburg, Germany) diagnostic kits for the presence of anti-measles and anti-rubella immunoglobin M (IgM). Amplification of ribonucleic acid (RNA) for genotyping was attempted in a sample of cases testing positive or equivocal for anti-measles IgM. For molecular analysis RNA was extracted directly from clinical specimens (urine if available, otherwise serum) and tested for the presence of Measles virus by reverse transcriptase polymerase chain reaction (RT-PCR).

By 31 December 2009, 15 291 specimens were collected from patients (n=15 059) who met the surveillance case definition. Gauteng province accounted for the highest proportions of specimens received (n=7 727, 50%). Data on type of specimen received was available for 13 661 patients. Of these patients, blood and urine specimens were received in 60% (n= 8 150) of the cases, blood only from 35% (n=4 821) and urine only from 0.2% (n=34)), and the rest were other specimen types.

Measles

In 2006, 2007 and 2008; 32, 81 and 40 confirmed measles cases were reported respectively, with an average of 51 cases per year. In 2009, a large measles outbreak was experienced. The outbreak started in the Tshwane district, Gauteng province in March 2009. The outbreak spread to other districts within Gauteng despite the mass measles vaccination campaign that took place in that Tshwane district from 24 August to 4 September. By 31 December 2009, a total of 5 857 confirmed measles cases were reported in all nine provinces.

Of the 5 857 cases with measles IgM positive results, the majority were from Gauteng (n=4 109, 70%), followed by North West province (n=455, 8%). Northern Cape reported the least number of confirmed measles cases (n=62, 1%) (Figure 1). Age and sex were known in 5 684 and 5 662 of the confirmed measles cases respectively. Age ranged from 0 months to 94 years with a median of five years. Age group 6-11 months were the most affected (n=1 406, 25%), followed by 1-4 years (n=925, 16%) (Figure 2). Of the 5 662 confirmed measles cases with known sex, 53% (n=3 020) were male and 47% (n=2 642) female.

Only a single genotype viz., B3.1 has been detected throughout the course of the epidemic. The identical virus was detected in specimens from Benin in early 2009. It is not possible to state that the virus was introduced directly from Benin given the viral surveillance in Africa is not optimal. The same viral genotype may thus be circulating in other countries in the region.

Rubella

A total of 2 975 rubella IgM positive cases were identified in 2009. There was an increase in the number of rubella IgM cases reported. Rubella cases increased from 1064 in 2008 to 2 975 in 2009. The highest proportion of cases (n=674, 23%) was observed in Gauteng province (Table1).

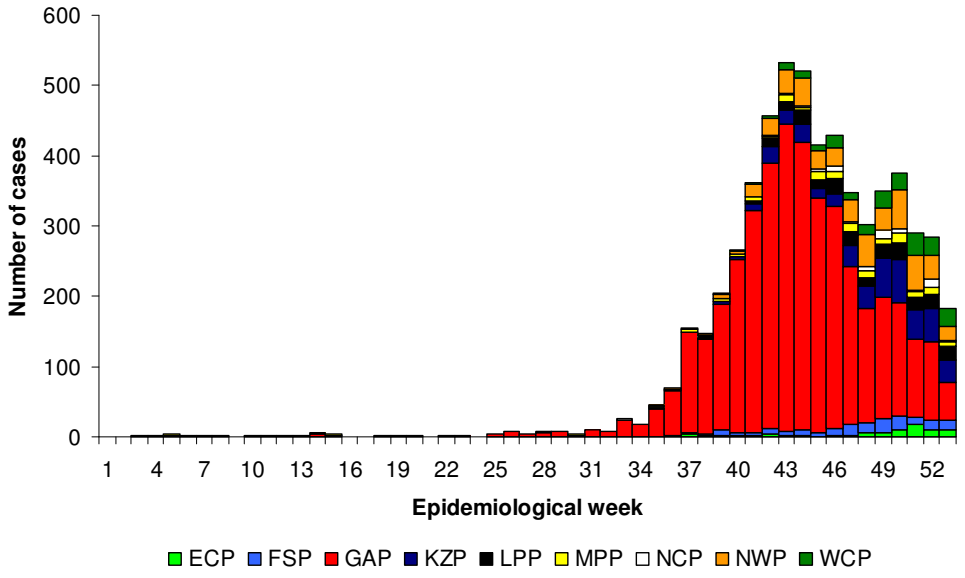

Figure 1: Measles IgM positive results per province: South Africa January-December 2009

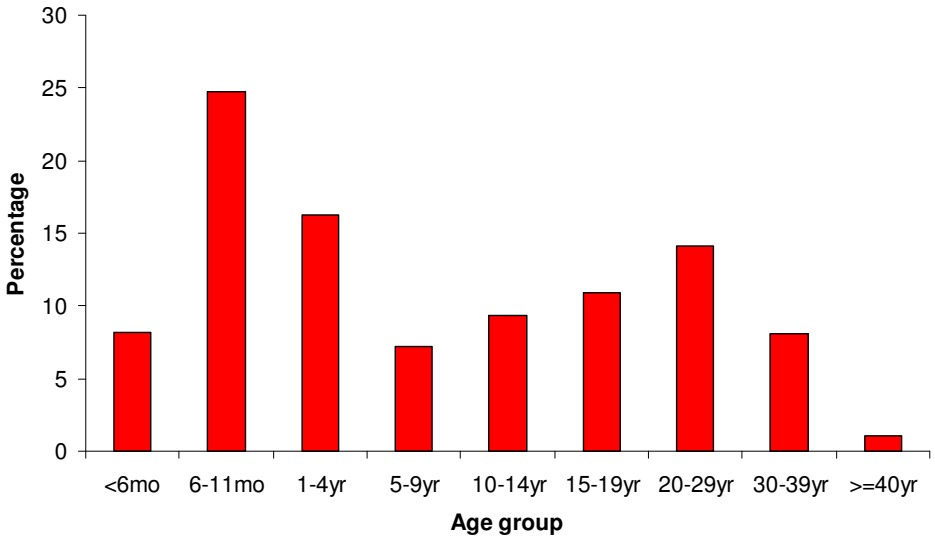

Figure 2: Age distribution of patients with measles, N=5684: South Africa January-December 2009

Table 1: Number and rate of suspected measles cases (SMC) with specimens submitted and measles and rubella IgM positive cases from suspected measles case-based surveillance, South Africa: 2009

		Provinces										
Indicator	**Year**	**ECP**	**FSP**	**GAP**	**KZP**	**LPP**	**MPP**	**NCP**	**NWP**	**WCP**	**Unknown province**	**TOTAL**
SMC	2009	667	353	7548	1498	1219	1031	350	1561	732	100	15 059
SMC/ 100 000 population	2009	10	12	722	15	23	29	31	46	14		31
Measles IgM positive	2009	80	164	4109	421	220	131	62	455	215		5 857
Rubella IgM positive	2009	273	73	674	501	443	355	151	340	133	32	2 975

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

Discussion

A widespread outbreak of measles occurred in 2009 and measles transmission is ongoing at the time of writing (February 2009). Although the highest incidence of disease was in the < 5 year age group, persons > 5 years of age accounted for 51% (n=2 887) of cases. The age group 6-11 months was the most affected, followed by the 1-4 year age group. This is unusual as the disease typically affects those < 5 years old. The fact that half the cases were > 5 years old indicates that an immunity gap exists in this age group.

For the period 2006-2008, numbers of measles IgM positive cases have remained at relatively low. The current widespread measles outbreak indicates that even in a setting of low reported cases, communities still remain vulnerable to measles importation. It is always necessary to maintain immunity among the population even in the absence of a circulating virus. In 2008, South Africa reported vaccine coverage of 85%, which is less than the target of 90% that forms part of the measles elimination strategy. Efforts to maintain the vaccine coverage at 95% as recommended by the World Health Organisation (WHO) should be emphasized.

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General Isolation Unit, NICD Receiving Laboratory and administrative staff

ACUTE FLACCID PARALYSIS SURVEILLANCE, 2009

Enterovirus Isolation Unit, Epidemiology and Surveillance Unit, Polio Molecular Unit, National Institute for Communicable Diseases

The Enterovirus Isolation Unit serves as national isolation laboratory for South Africa as well as six other Southern African countries i.e. Angola, Botswana, Lesotho, Mozambique, Namibia, and Swaziland.

During the course of the year 2156 stool specimens were received from patients with AFP from these countries (Figure 1). Of these 110 were from patients with onset of paralysis prior to 2009. Of the remainder, 807 specimens were collected from 413 South African cases, and 1237 specimens were collected from the six other countries listed above. In early January 2010 a further 17 specimens were received from 8 South African cases with onset of paralysis in 2009, bringing the total number of cases in 2009 to 421.

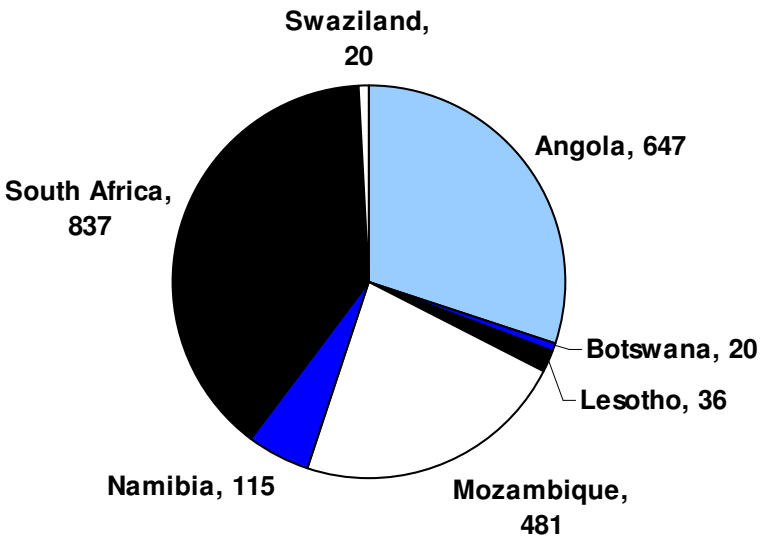

Figure 1: Number of stool specimens from AFP cases received for virus isolation by country

1: South African cases

Of the 421 South African cases with onset of paralysis in 2009, one specimen only was received from 65 cases, and two or more specimens from 356. The date of onset of paralysis was known for 358 (85%) cases. Two specimens taken at least 24 hours apart and within 14 days of onset were received from 283/421 (67%) cases (range per province 57% to 83%) (Figure 2). Non-polio enteroviruses were isolated from 84, and non-enteroviruses from 17 of the 837 specimens (non-polio isolation rate 12%) and poliovirus, identified as Sabin type poliovirus, was identified from four specimens taken from three patients.

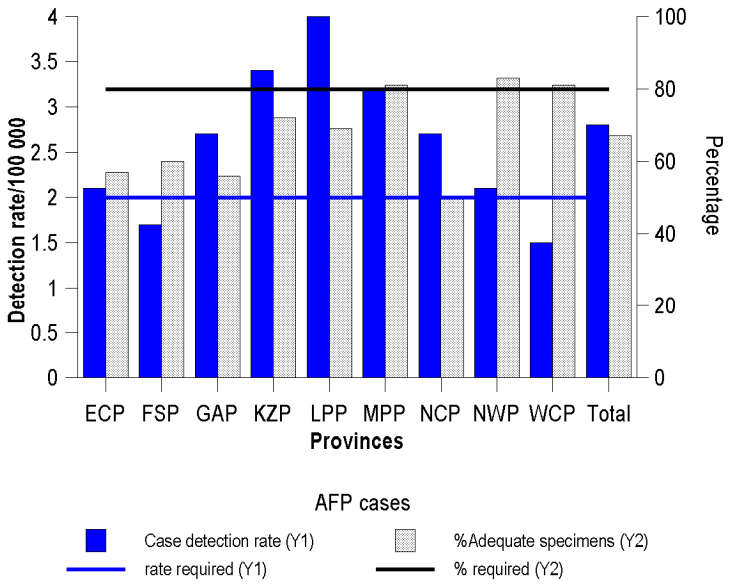

Figure 2: AFP case detection and stool adequacy rate, South Africa, 2009 (only patients from whom stool specimens were received included)

2: Other Southern African countries

Of the 1319 specimens received from the other six southern block countries served by the NICD, 74 were from patients with onset of paralysis prior to 2009. Two adequate stool specimens were received from 570 (91%) of the 624 patients with onset of paralysis in 2009 (range per country 80% to 100%). Non-polio enteroviruses were isolated from 148/1319 specimens giving a non-polio enterovirus isolation rate of 12% (range per country 0 to 14%). Poliovirus was isolated from 109 specimens, 48 of which were identified as wild type polio 1, and the remainder as Sabin strains. The wild type isolates were from 30 patients in Angola with dates of onset ranging from 26 December 2008 to 15 September 2009.

Molecular Characterisation of Polioviruses

The molecular Polio unit serves as a Regional Reference Laboratory with the capacity of a Specialized Reference Laboratory. The unit serves as the only sequencing laboratory for polioviruses in Africa; and contributes to the training of the African laboratory Network through World Health Organization (WHO) workshop programmes.

Workload dramatically increased in 2009. The laboratory received 2963 isolates by December 2009 compared to 1515 isolates received in 2008. These were characterized as vaccine or wild type using two intratypic differentiation methods: PCR and ELISA. These isolates were sent by the African laboratories that are part of the Polio Laboratory Network. The isolates were tested by RT-PCR, ELISA,

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Real-time RT-PCR and Sequencing. The high volume increase was due to an outbreak experienced in West and Central Africa. In addition, all known Sabin strains were undergoing Vaccine Derived Polioviruses (VDPVs) screening using Real-Time PCR.

Twenty VDPVs were identified in 5 countries namely Ethiopia, Guinea, DRC, Somalia and Malawi. Four cases representing VDPV type 2 were identified in the Democratic Republic of Congo, three with date of onset of paralysis in 2009 and one in 2008; this is a continuation of a VDPV outbreak in the DRC since 2005. In Somalia, two 2009 cases and one contact were identified as VDPV type 2. In Ethiopia, two VDPV type 2 cases and 1 VDPV type 1 were reported. A single Malawi case was a VDPV type 1 and a Guinea case was a VDPV type 2.

In total, 906 isolates were sequenced of which 785 were identified as wild types and twenty as VDPVs. One hundred and one were identified as Sabin strains due to less than 10 nucleotide difference from the parental Sabin strain.

Two wild type PV1 genotypes are still circulating in Africa, WEAF-B and SOAS genotypes (Figure 3). SOAS genotype originated in India and was introduced to Africa in 2005, affecting Angola, DRC, Namibia, Burundi and Central African Republic. In 2009, only Burundi and Angola were affected by SOAS genotype.. Western African B (WEAF-B) genotype has affected mostly West African countries with a high outbreak experience in Guinea. This genotype has subsequently spread to East Africa, affecting Kenya.

Few wild type poliovirus 3 cases were identified in 2009 and only 5 countries were affected by both WEAF-B and SOAS genotypes (Figure 4). The SOAS genotype was only detected in 3 cases, all from DRC. Cameroon, Central African Republic, Chad and Niger were affected by the WEAF-B genotype.

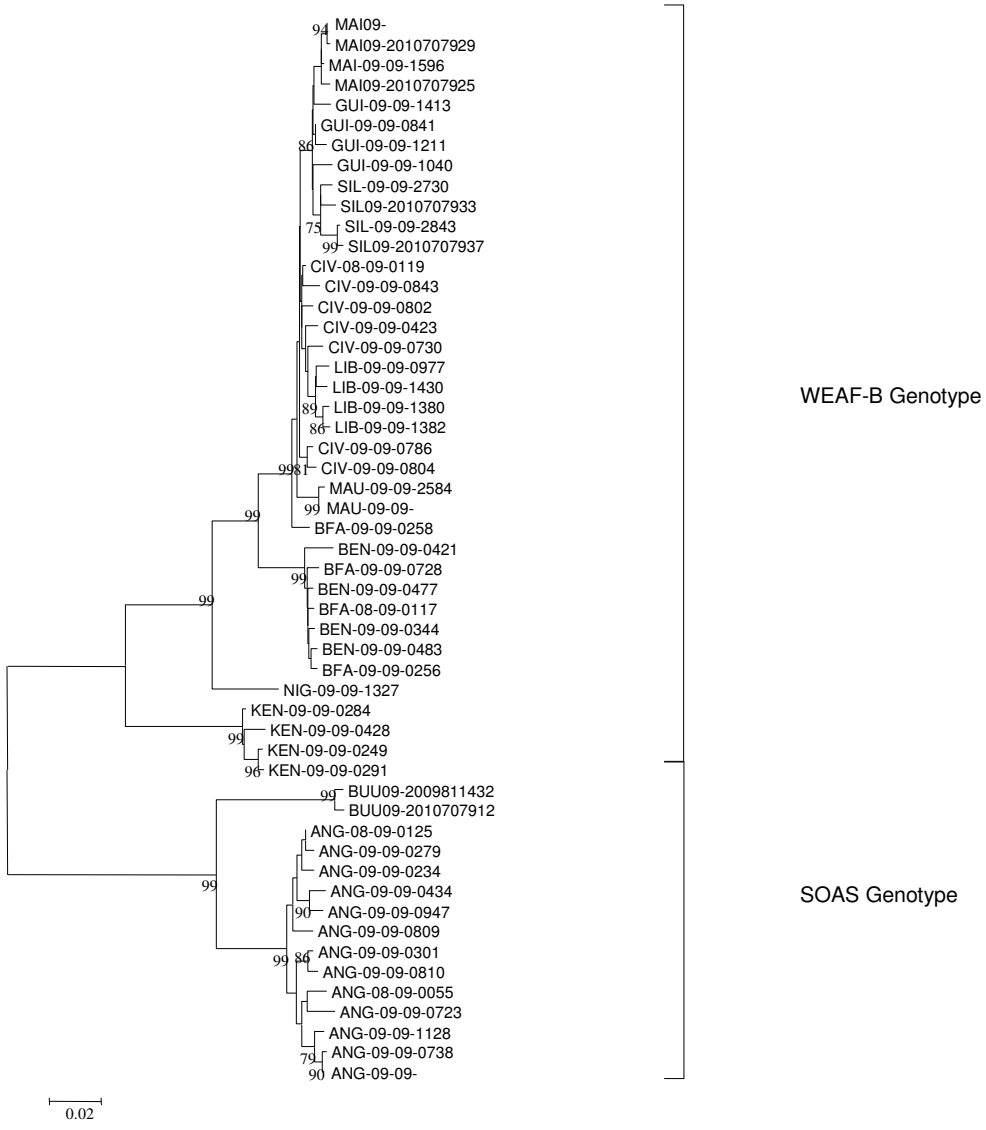

Figure 3: Selected wild type poliovirus 1 circulating in Africa in 2009. Only bootstrap values of more than 70 were shown.

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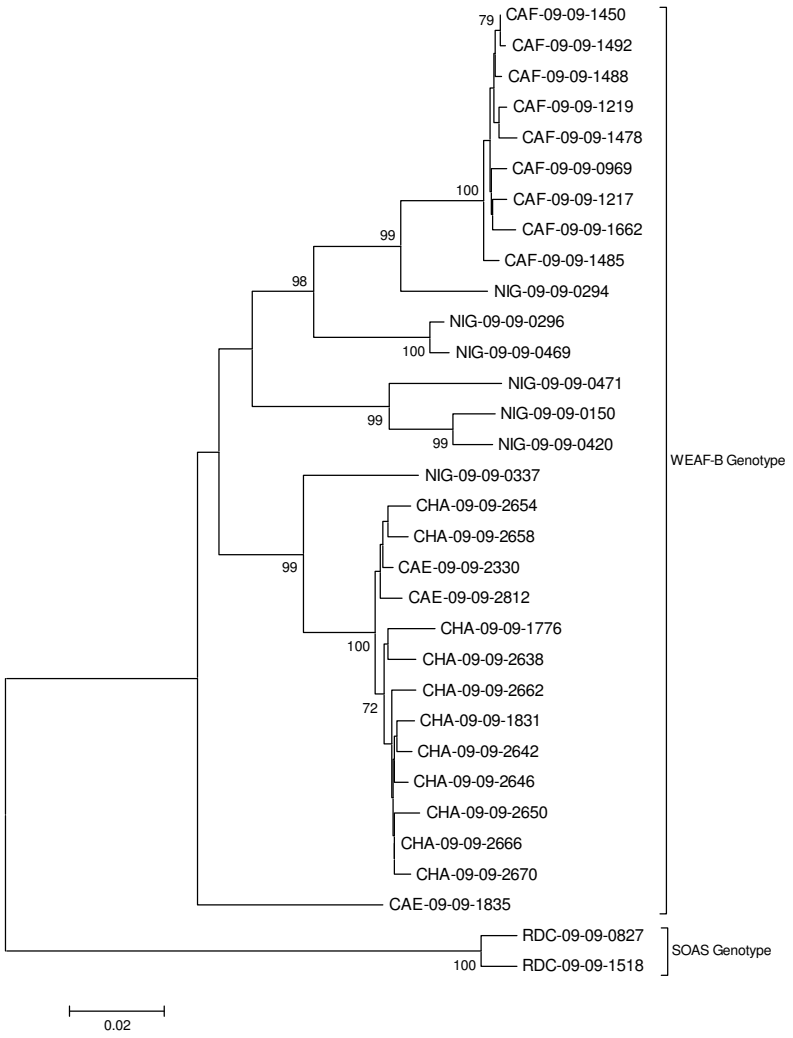

Figure 4: Selected wild type poliovirus 3 circulating in Africa in 2009. Only bootstrap values of more than 70 were shown.

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RESPIRATORY VIRUS SURVEILLANCE, SOUTH AFRICA 2009

Epidemiology and Surveillance Unit, Respiratory and Meningeal Pathogens Reference Unit, Respiratory Virus Unit, Viral isolation Unit, National Institute for Communicable Diseases

Overview of programmes

The NICD houses the National Influenza centre, a regional World Health Organization Reference laboratory for Influenza and is responsible for national influenza surveillance programmes and characterization of strains as part of the annual global vaccine update by the WHO. There are a number of influenza surveillance programmes, each focusing on different aspects. These include:

1. The viral watch surveillance programme
2. The severe acute respiratory infections (SARI) programme
3. The respiratory morbidity data mining surveillance system
4. Influenza-associated mortality surveillance programme

This bulletin will provide preliminary surveillance findings for the year 2010 the first 3 programmes. In addition we include a brief description of findings from enhanced surveillance related to the pandemic influenza A H1N1 outbreak and details of molecular characterization of influenza viruses for 2009.

1. The viral watch surveillance programme

The Viral Watch sentinel surveillance programme, started in 1984, was specifically designed to monitor influenza

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activity in the community. Since 2008 there have been sites in all nine provinces. In early 2009 a further 24 sites were added in Limpopo and the Western Cape bringing the total number of sites to 243, of these sites, 165 submit specimens directly to the NICD. Sites in the Western Cape submit specimens to NHLS Tygerberg Hospital laboratory and sites in KwaZulu-Natal submit specimens to the department of Virology at Inkosi Albert Luthuli Central Hospital/University of KwaZulu-Natal. In July, in response to the emergence of pandemic influenza A H1N1, Enhanced Viral Watch centres at 12 public hospitals in 8 provinces were enrolled to detect influenza strains in hospitalized patients. In week 32 sites were requested to limit the number of specimens submitted to the laboratory due to the challenges of processing large numbers of diagnostic and surveillance specimens.

During 2009, 3354 specimens were received at the NICD for detection of respiratory viruses from Viral Watch centres, and 123 from Enhanced Viral Watch centres. In addition another 328 positive specimens were received from centres in the Western Cape and KwaZulu-Natal for confirmation, serotyping and sequencing. In week 32 practitioners were asked to limit the number of specimens submitted to no more than 5 per week.

Two distinct peaks of influenza virus circulation were observed with predominantly influenza A H3N2 circulating from week 20 through week 27 and influenza A H1N1 circulating from week 28 through week 42. Between weeks 10 (week starting 2 March) and week 42 (week starting 12 October), seasonal influenza was detected in 1054 specimens. These were identified as 59 influenza A untyped, 4 A H1N1, 866 A H3N2, and 125 influenza B virus. The detection rate increased to >10% in the week starting 11 May (week 20), and peaked at 78% in week 24 (Figure 1a). The first pandemic influenza A H1N1 virus in a Viral Watch specimen was detected in a specimen collected on 22 June (week 26). A total of 743 specimens testing positive for pandemic influenza A H1N1 were identified from Viral Watch and Enhanced Viral Watch specimens.

The age distribution of patients with pandemic H1N1 differed from that of patients with seasonal A H3N2 in 2009, as well as of those with seasonal A H1N1 during 2008. However, the age distribution of those with influenza B in 2009 was similar to those with pandemic influenza A H1N1 (Figure 2).

For analysis of seasonal distribution provinces were grouped into 3 climatic regions. These were the Central Plateau which contained the Free State, Gauteng, Northern Cape and North West provinces; the North Eastern Sub-Tropical Region which contained KwaZulu-Natal, Mpumalanga and Limpopo; and the Southern Coastal Belt which contained the Eastern and Western Cape provinces. Seasonal influenza peaked in week 22 in the Southern Coastal Belt, followed by week 24 for both the Central and North East sub-tropical Regions. Pandemic H1N1 isolates peaked in week 32 for both the Central Region and Southern Coastal Belt, and in week 34 in the North Eastern Sub-Tropical Region (Figure 3).

2. The severe acute respiratory infections (SARI) programme

The Severe Acute Respiratory Infection surveillance (SARI) was initiated in February 2009. The aim of the surveillance was to describe the trends in numbers of patients admitted with SARI at sentinel hospitals and to determine the relative contribution of influenza and other respiratory viruses to SARI presentation in a setting with high HIV prevalence.

The first site, Chris Hani Baragwanath Hospital (CHBH, Gauteng province), started enrolling patients on the 9th of February 2009, followed by Mapulaneng and Matikwana Hospitals (Agincourt, Mpumalanga) in April 2009 and the last site, Edendale Hospital (Kwazulu-Natal) on the 2nd of September 2009. All adult and paediatric patients admitted to sentinel hospitals who met the case definition for SARI were prospectively enrolled into the surveillance programme. Detailed demographic and clinical data, in-hospital management, laboratory results and outcome data

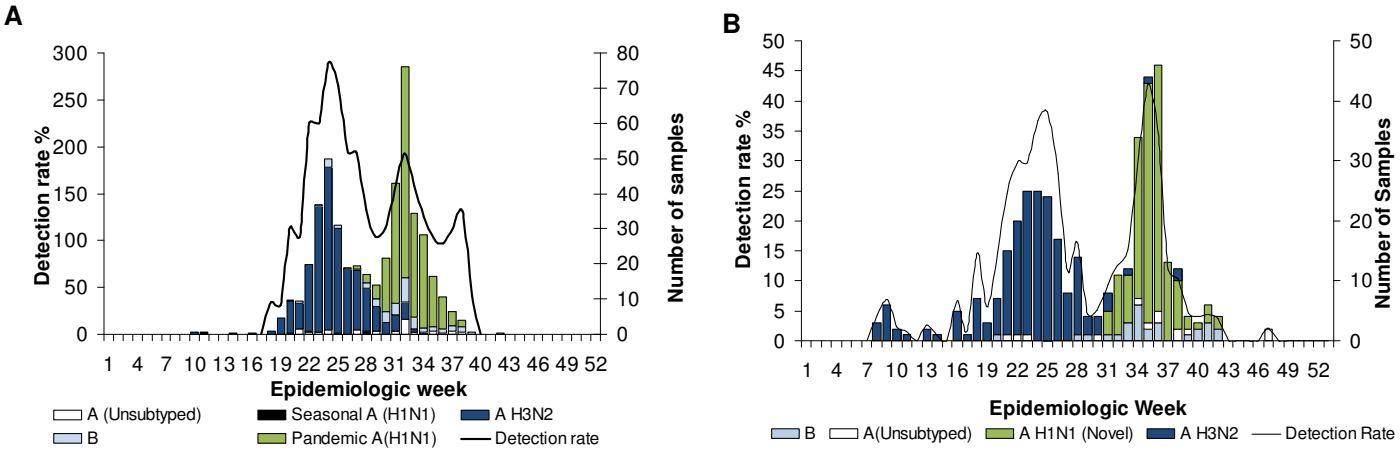

Figure 1: Number of influenza virus isolates by virus type and epidemiologic week, South Africa, 2009
A -Viral Watch surveillance programme, B—SARI surveillance programme

(Continued on page 8)

were collected. Respiratory samples (nasopharyngeal aspirates for children less than 5 years or throat and nasopharyngeal swabs for patients 5 years of age or older) and blood samples were collected from enrolled patients. Respiratory samples were tested for influenza, adenovirus, respiratory syncytial virus (RSV), human metapneumovirus, enterovirus, rhinovirus and para-influenza virus type1, 2 and 3 using multiplex realtime polymerase chain reaction (PCR). Samples positive for influenza were sub typed using a one step quantitative reverse transcriptase PCR real time essay. Quantitative real-time PCR (lytA) was used to detect pneumococcus DNA from blood specimens.

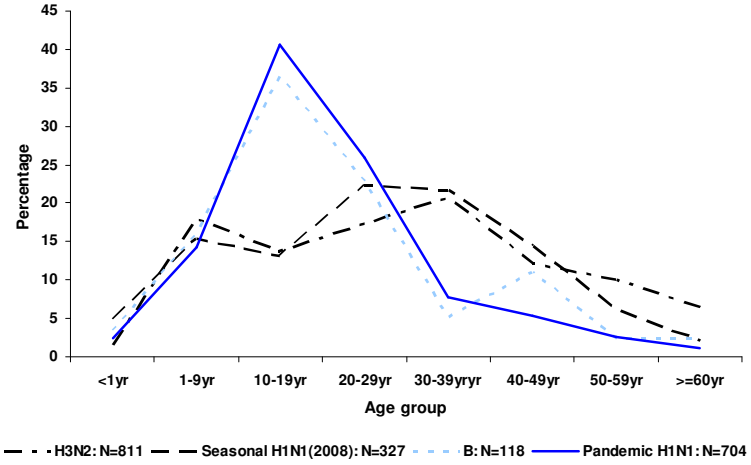

Figure 2: Percentage of patients testing positive for influenza by age group and virus type. Viral Watch programme, South Africa, 2009.

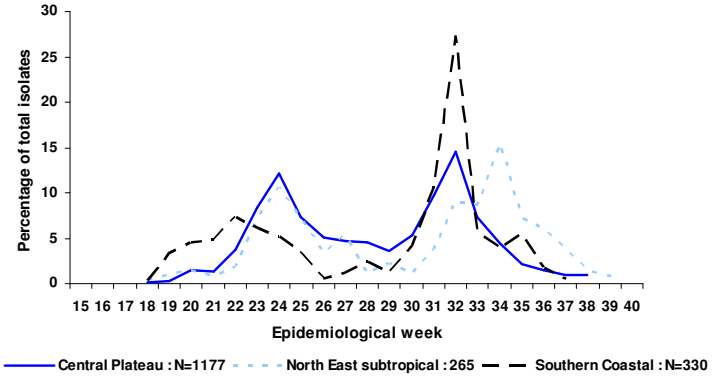

Figure 3: Influenza virus detection rate by epidemiological week for 3 climatic zones, Viral Watch programme, South Africa, 2009.

Between 9th February 2009 (week 7) and 3rd January 2010 (week 53), 3693 patients were enrolled into the SARI surveillance programme. The majority (2963/3693, 80%) of enrolled patients were from CHBH. Sixty percent (3141/3592) of the patients were under 5 years of age and 51% (1833/3599) were female. Of the 3693 patients enrolled, influenza results were available for 3642 (99%) patients. Of these, 391 (11%) samples were positive for influenza on multiplex PCR, 159 (41%) of positive samples were identified as pandemic Influenza A H1N1, 194(49%) as A H3N2, 27(7%) as influenza B and 13 (3%) as A (unsubtyped). Two samples were co-infected with pandemic influenza A H1N1 and influenza B. Similar to the viral watch, two peaks of influenza were observed in 2009, although the timing of the second peak was later in the SARI programme. The first peak was dominated by influenza A H3N2 and the second by pandemic influenza A H1N1 (Figure 1b). Detection rate for seasonal influenza A H3N2 was high in young children, peaking in the 2-4 year age group. Detection rate for pandemic influenza was highest in 15 to 24 year old age group (Figure 4).

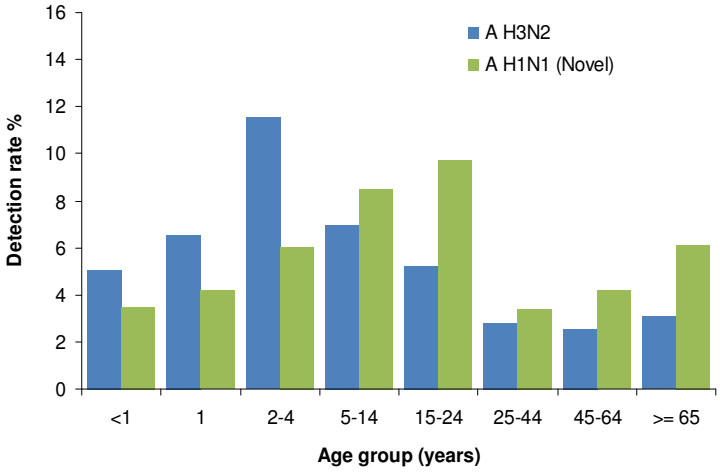

Figure 4: Influenza detection rate by age group and virus subtype amongst SARI patients, 2009

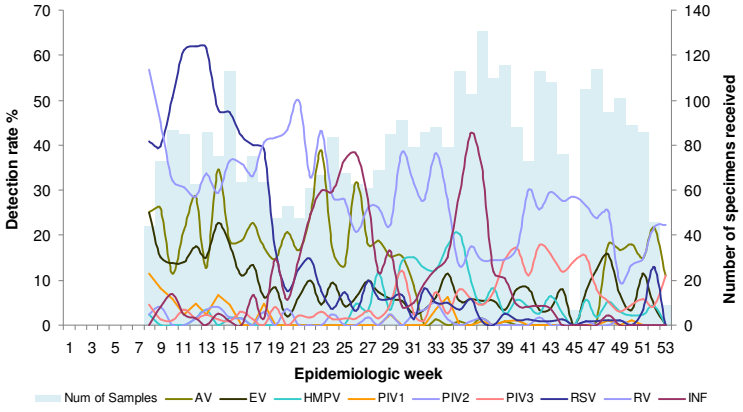

Figure 5: Number of samples and detection rate for respiratory viruses by epidemiologic week, SARI, 2009
AV—Adenovirus, EV-Enterovirus, HMPV—human metapneumovirus, PIV 1, 2, 3—Parainfluenza virus type 1, 2 and 3, RSV—Respiratory syncytial virus, INF—Influenza virus

Respiratory syncytial virus (RSV) was isolated in 14% (520/3647), adenovirus in 12 % (423/3647), rhinovirus 27% (993/3647), parainfluenza 1 in 1% (51/3647), parainfluenza 2 in 1% (40/3647), parainfluenza 3 in 6%

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(214 /3647), human metapneumovirus in 5% (177/3647) and enterovirus in 9% (316/3647) of SARI cases. The respiratory syncytial virus (RSV) season preceded the influenza season (Figure 5). Testing for pneumococcus began in week 20 and of 2023 patients with specimens tested for the presence of pneumococcal DNA 140 (7%) tested positive. An increase in pneumococcal detection rate was observed during the period when influenza was circulating (Figure 6).

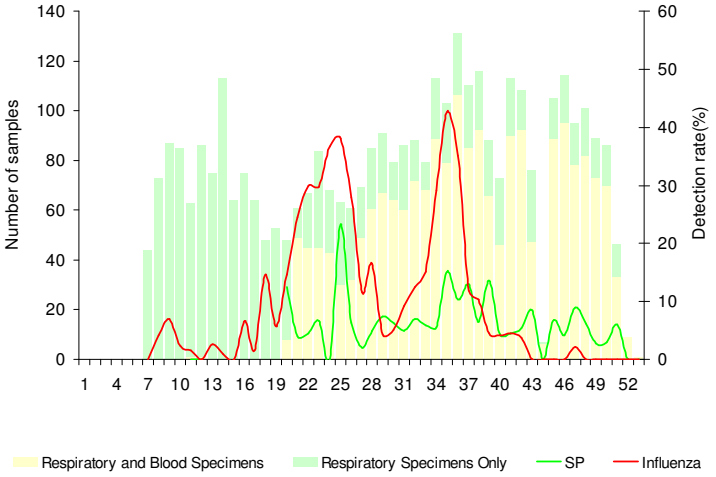

Figure 6: Number of samples and detection rate for influenza and *Streptococcus pneumoniae* by epidemiologic week, SARI 2009.

3. Respiratory morbidity data mining surveillance system

During 2009 there were 1 132 331 consultations reported to the NICD through the respiratory morbidity mining surveillance system. Of these 3.4% (38044) were due to pneumonia or influenza (P&I) (ICD codes J10-18). Two peaks in P&I consultations were observed corresponding to the timing of the two peaks in influenza virus isolations (Figure 7). The second peak corresponding to the period of circulation of pandemic influenza A H1N1 was substantially higher than that associated with seasonal influenza. This may be partly related to an increased awareness of influenza, related to the pandemic.

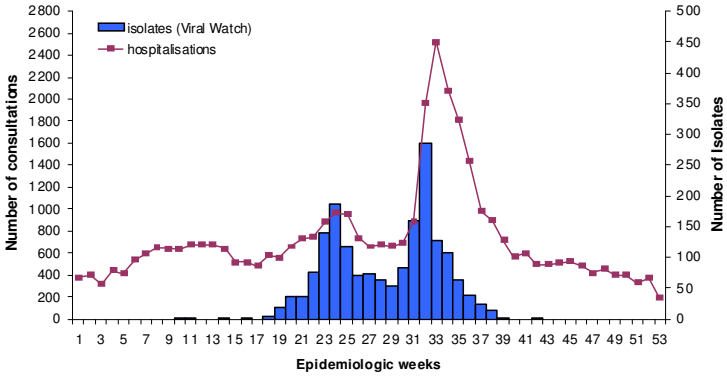

Figure 7: Number of private hospital consultations* with a discharge diagnosis of pneumonia and influenza (P&I) and viral isolates**, South Africa, 2009.

*Consultations data from weekly reports of consultations at the Netcare hospital group. Discharge diagnosis is according to ICD coding by clinicians and does not represent laboratory confirmation of aetiology.

**Viral isolation data from the Viral Watch sentinel surveillance programme.

4. Enhanced surveillance related to the pandemic influenza A H1N1 outbreak

In addition to the circulation of the typical seasonal strains of influenza virus in 2009, South Africa experienced importation and extensive local transmission of the pandemic influenza A(H1N1) 2009 strain. Details on the epidemiology and factors associated with deaths have previously been discussed elsewhere (1). Furthermore, the NICD publishes regular situation reports on the website and within the monthly Communicable Diseases Communiqué (both available via www.nicd.ac.za). A brief summary of findings is provided here.

Cases: The NICD established surveillance for centralised reporting of laboratory-confirmed cases of pandemic influenza diagnosed by private and public laboratories throughout South Africa. From 28 April 2009 (when local diagnostic capacity established) to 31 December 2009, 12,636 laboratory-confirmed cases were reported. Following importation of the virus we observed rapid local transmission, with the epidemic peaking in August 2009 at >2,000 new cases per week, followed by a rapid decline in the frequency of new cases reported. From October 2009 to date, sporadic cases continue to be detected; however, these have all been associated with international travel. Children aged 5-19 years (53% of total cases) account were the most affected (median age 16 years).

Deaths: During the same period, 93 pandemic influenza A (H1N1) related deaths have been confirmed in South Africa. Preliminary analysis of data gathered on further investigation of deceased patients show a high prevalence of HIV-infection (19/38 tested, 50%) and pregnancy (26/91 with known status, 28%), suggesting that these comorbidities are possible risk factors for fatal infection.

5. Molecular characterisation of influenza viruses

As part of the routine diagnosis and surveillance programmes described above the respiratory virus unit which houses the National Influenza Centre received 9797 specimens in total for 2009, of which 9357 were appropriate for testing for Influenza. Specimens were received through provision of routine diagnostic services from doctors across the country (2395 specimens received) and the remainder from the influenza surveillance programmes. In total 1096 cases were subtyped as H3N2, 1206 as pandemic H1N1 and 6 as seasonal H1N1. Influenza B was identified in 112 cases. The unit has also tested Influenza A specimens from various Southern African countries including Namibia, Angola, Botswana, Zimbabwe, Swaziland, Lesotho, Mozambique, Malawi, Zambia, Seychelles and Madagascar and identified the first novel H1N1 cases for these countries and provided training to them to establish their own testing capabilities.

Resistance testing

The neuraminidase proteins of 105 pandemic H1N1 specimens have been sequenced and investigated for Oseltamivir resistance and pathogenic markers. None of the specimens had the H275Y mutation on the NA gene

(Continued on page 10)

that is associated with resistance. Investigation of the HA D222G and PB2 E627K pathogenic markers identified in patients that died in the Northern hemisphere using sequence analysis indicated the absence of these mutations in SARI patients in South Africa. These mutations do not seem to be specifically associated with influenza disease severity in SA.

Molecular epidemiology of the H1N1 outbreak

Molecular epidemiological investigations of the South African H1N1 pandemic Influenza strains are underway. A selection of specimens from patients infected during the 2009 pandemic have been sequenced and compared with isolates from the rest of the world using Bayesian phylogenetic analysis. This included patients identified at the early stages of the outbreak, consisting mostly of travellers or their direct contacts (imported strains) in June and July and patients identified after community transmission had been established (August) through to the end of the outbreak (October). The presence of 3 separate clusters could be demonstrated in the country based on the HA genes (Figure 8). Limited drift were identified over time away from the initial strain that was isolated when it first appeared in June in South Africa. Strains similar to the original introductory strains circulated for several months, although recent strains differ from original importations which may suggest positive selection and drift. The early strains that were isolated were submitted to the WHO for comparison to the vaccine strain and differed by 0.8% while by September strains had 1.3% amino acid differences to the vaccine strain.

Molecular epidemiology of recent importations will determine if further drift away from vaccine occurred when the next wave hits. For the moment WHO Collaborating centers indicated that the vaccine strain A/ California/7/2009 is antigenically similar to circulating strains. Further investigations of cases of severe disease are continuing.

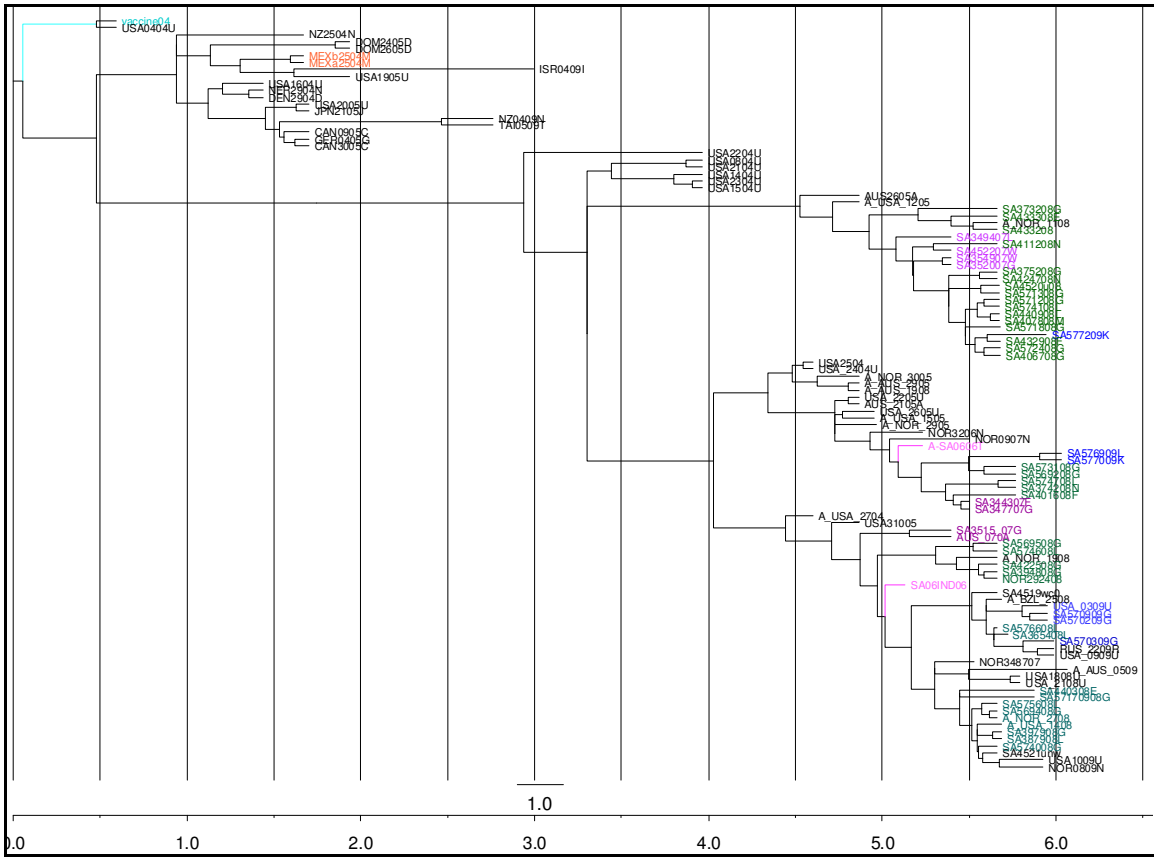

Figure 8: Bayesian evolutionary analysis over time of pandemic Influenza A H1N1 strains identified in South Africa in 2009 compared to isolates from the rest of the world. The vaccine strain is shown in turquoise on the left, the original Mexican isolates in red, the first South African isolates identified in June in pink, South African isolates from July in purple, from August in green and from September in blue.

Report compiled by (alphabetical order): Brett Archer, Lucille Blumberg, Amelia Buys, Cheryl Cohen, Jo McAnerney, Jocelyn Moyes, Dhamari Naidoo, Marthi Nieuwoudt, Adrian Puren, Juno Thomas, Anne von Gottberg, Marietjie Venter, Sibongile Walaza, Nicole Wolter

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(Continued on page 11)

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Viral watch programme: participating "Viral watch docs"
Respiratory morbidity data mining surveillance system: Netcare hospital group
Patients who kindly agreed to participate in surveillance.

ROTAVIRUS SURVEILLANCE IN SOUTH AFRICA, 2009

Enteric Diseases Reference Unit, Epidemiology and Surveillance Unit, Parasitology Reference Unit and Viral Gastroenteritis Unit, National Institute for Communicable Diseases

Rotavirus surveillance programme

Diarrhoeal disease accounts for 1.8 million deaths in children each year[1]. Globally, rotavirus is reported to be the leading aetiologic cause of diarrhoea, with the highest burden of illness in children younger than 5 years of age and resulting in over half a million deaths annually[2, 3]. In South Africa, 17% of deaths amongst children less than 4 years of age are estimated to be caused by diarrhoea. The mortality and morbidity burden attributable to rotavirus disease globally has been well established. Improvements in sanitation and the availability of clean water have not decreased the rate of rotavirus diarrhoea in developed countries, focusing the need to develop vaccine`s as the first strategy of prevention[4]. The current World Health Organization (WHO) recommendations for rotavirus vaccines are to include these vaccines in all national immunization programs and the use is strongly recommended in countries where diarrhoeal deaths account for ≥10% mortality in children less than five years of age[5].

In 2009, active surveillance for rotavirus infection was implemented at 5 sentinel hospitals in 4 Provinces in South Africa (Gauteng, Mpumalanga, Northwest and Kwazulu-Natal). The programme aims to estimate the number of hospitalisations due to severe diarrhoea and laboratory-confirmed rotavirus infection in HIV-infected and uninfected children. Additional programme objectives include determining the prevalent rotavirus strains in different geographical areas of South Africa and monitoring trends in rotavirus disease following the introduction of the Rotarix® vaccine into the expanded programme on immunization in August 2009.

Stool specimens for diagnosis of rotavirus and other diarrhoeal causative agents, are collected from children < 5 years of age with diarrhoea (defined as 3 or more loose stools in 24 hours) of < 7 days duration. Testing for rotavirus is performed at the Viral Gastroenteritis Unit, NICD and Diarrhoeal Pathogens Research Unit, University of Limpopo Medunsa campus using, the ProSpecT Rotavirus ELISA kit (Oxoid, UK) and the GastroVir strip (Coris Bioconcept, Belgium), respectively. The Rotavirus screening results were confirmed by RT-PCR based on standardized methods described in the WHO Manual of Rotavirus Detection and Characterization Methods[6]. Test results of the screening were either, positive, negative or equivocal. In the case of an equivocal result, confirmatory testing was performed using RT-PCR. RT-PCR genotyping was performed utilizing standardised methods described in the WHO Manual of Rotavirus Detection and Characterization Methods[6]. The VP7-specific primers used included G1, G2, G4, G3, G8, G9, G10 and G12. The VP4 -specific primers utilized included P[4], P[6], P[8], P[9], P [10], P[11] and P[14].

In addition to rotavirus testing, stool swabs in Cary Blair media were collected at the respective sites and sent to the Enteric Disease Reference Unit (EDRU) at NICD for further bacterial investigation. Any raw clinical material left after rotavirus testing was sent to the Parasitology Reference Unit (PRU) at NICD for detection of parasitic causes of diarrhoea.

Case investigations forms are completed by surveillance officers at the sites, providing information on the demographics, clinical signs and symptoms and the outcome of each child enrolled into the programme.

Data collection started first at Dr. George Mukhari Hospital (Northwest) in the first week of April (05/04/2009), at Chris Hani Baragwanath (Gauteng) in the fourth week of April (23/04/2009), and in Agincourt (Mpumalanga) hospitals in May (06/05/2009 for Mapulaneng and 20/05/2009 for Matikwana). Surveillance at Edendale hospital in Kwazulu-

(Continued on page 12)

Natal was initiated in 2010. We present a preliminary report of the findings of the first year of surveillance.

A total of 962 cases of diarrhoea were reported to the Rotavirus surveillance programme in 2009. Results of rotavirus testing are currently available for 830 patients, of whom 398 (48%) tested positive for rotavirus (Table 1). Rotavirus circulation occurred throughout the surveillance period but 2 seasonal peaks were observed: the first from week 17-25 and the 2nd lower peak from week 29-38 (Figure 1). The highest number of patients and the highest rotavirus detection rate was observed in children aged < 1 year (Figure 2).

(Continued on page 13)

Table 1: Numbers of specimens, rota cases and detection rate per hospital, South Africa, 2009

	Date of initiation	Number of specimens tested	Number positive	Detection rate
Dr. George Mukhari Hospital	05/04/2009	420	182	43
Chris Hani Baragwanath Hospital	23/04/2009	221	125	57
Mapulaneng Hospital	06/05/2009	86	47	55
Matikwana Hospital	20/05/2009	103	44	43
All hospitals		830	398	48

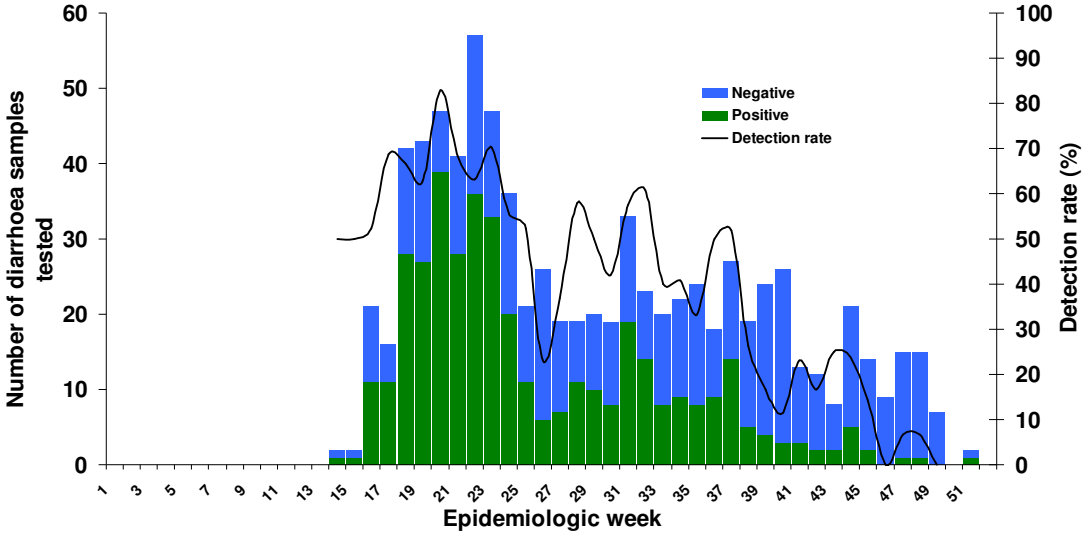
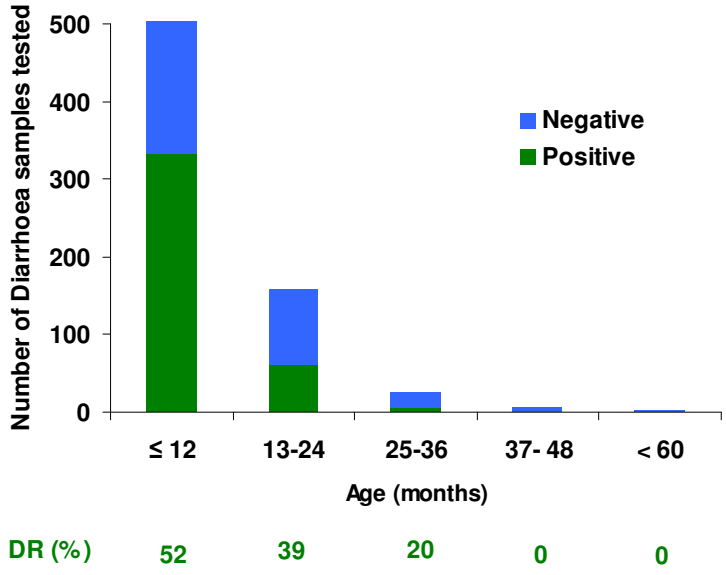


Figure 1: Epidemic curve of specimens tested and rotavirus detection rate for all surveillance sites, South Africa, 2009



(DR=Detection Rate of Rotavirus cases on total number of diarrhoea samples tested)

Figure 2: Age distribution of diarrhoea and rotavirus cases tested, South Africa, 2009

A total of 469 samples were available for analysis of the presence of bacterial pathogens and 114 (24%) tested positive. The most common pathogen isolated was diarrheagenic *E.coli* (n=93, 82%). A further 376 samples had sufficient clinical material to test for presence of parasites and 22(6%) were positive. The most common parasite isolated was *Cryptosporidium* spp (n=18, 82%).

Molecular characterisation of rotavirus strains identified through the rotavirus surveillance programme, 2009

Rotaviruses are classified according to epitopes on the smooth outer capsid protein (encoded by the VP7 gene) and short spike protein (encoded by the VP4 gene), specifying G and P types, respectively. These proteins are the major antigens inducing neutralizing immune responses during rotavirus infections. Although 12 different G genotypes and 15 P genotypes[7], have been detected in humans, serotypes G1P[8], G2P[4], G3P[8], G4P[8] and G9P[8] are thought to be an important cause of diarrhoea in infants and young children worldwide[7].

Overall, genotype G1P[8] predominated and were the most frequently detected strain in all the surveillance sites, especially Matikwane where G1P[8] was responsible for 70% of cases (Table 2). The globally common G1P[8], G2P[4], G3P[8], G4P[8] and G9P[8] strains were responsible for 55% cases while genotypes with unusual G and P combinations were evident in 39% cases. The majority of the unusual genotypes included G2P[6], G12P [8], G12P[4] and G1P[6] strains.

In addition to G1P[8] at CHBH, G2P[6] strains were detected in 19% of cases, G2P[4] in 11%, G12P[8] in 9% and G12P[4] in 7% of cases (Table 2). At Mapulaneng, other strains detected included G12P[8] in 14% of cases, G2P[4] and G9P[8] in 12% of cases and G2P[6] in 9% of cases. Rotavirus strains circulating at the Matikwane Hospital differed from those seen in the two other sites as almost 80% of strains were from the globally common serotypes and only 15% strains revealed unusual genotypes.

The typing data from the three sites represents baseline genotyping results prior to the monovalent G1P[8] rotavirus vaccine introduction. While the vaccine has been available on the private market in South Africa since July 2006, it was only introduced into the national extended program of immunization (EPI) in August 2009. Limited genotyping data is available from CHBH, generated during the rotavirus efficacy trial conducted between 2005 and 2007[8]. During the trial, serotype G1P[8] was detected in 57% cases with G8P[4], G9P[8] and G12P[6] strains also circulating. Previous data from Mpumalanga (n=57) dating back to 1998/1999, revealed G1P[8] strains in 74% of

Table 2: Summary of rotavirus genotypes circulating amongst hospitalised children (< 5 years) with diarrhoea at Chris Hani Baragwanath (CHBH), Mapulaneng and Matikwane Hospitals between April and December 2009, South Africa

	CHBH		Mapulaneng		Matikwane		Total	
Genotype	**n**	**%**	**n**	**%**	**n**	**%**	n	%
Usual Genotypes								
G1P[8]	60	34	14	33	32	70	106	40
G2P[4]	19	11	5	12	4	9	28	11
G4P[8]	3	2	0	0	0	0	3	1
G9P[8]	4	2	5	12	1	2	10	4
Total	86	49	24	56	37	80	147	55
Unusual Genotypes								
G1P[6]	9	5	2	5	1	2	12	5
G1P[4]	3	2	1	2	1	2	5	2
G2P[6]	33	19	4	9	1	2	38	14
G2P[8]	5	3	0	0	2	4	7	3
G3P[4]	1	>1	0	0	0	0	1	>1
G9P[6]	1	>1	0	0	0	0	1	>1
G12P[4]	12	7	2	5	1	2	15	6
G12P[6]	2	1	0	0	0	0	2	>1
G12P[8]	15	9	6	14	1	2	22	8
Total	81	46	15	35	7	15	103	39
Mixed and Non-typeables								
Mixed	2	1	2	5	0	0	4	2
G1P?	1	>1	0	0	0	0	1	>1
G2P?	2	1	0	0	0	0	2	>1
G9P?	0	0	1	2	0	0	1	>1
G?P[8]	0	0	1	2	1	2	2	>1
ND	4	2	0	0	1	2	5	2
Total	9	5	4	9	2	4	15	6
Grand Total	176	66	43	16	46	17	265	

(Continued on page 13)

cases and G2P[4] in 5% of cases[9]. The data from the Mapulaneng and Matikwane Hospitals represents the first genotyping data from the Mpumalanga area in a decade.

The genotyping data revealed that the majority of children (63%) infected with rotavirus in these three sites should be directly protected by administration of the monovalent G1P[8] vaccine. The recently published vaccine efficacy study from Africa seems to indicate that while variation in type-specific vaccine efficacy is evident, it is unlikely that differences in strain diversity contributed to lower vaccine efficacy seen in Malawi site[8].

Unusual rotavirus strains were detected in 39% cases (range 16%-46%), which is similar to the 22 to 33% range seen by Seheri and colleagues (*in press*)[10] during the burden of rotavirus disease study conducted at the Dr George Mukhari Hospital, Ga-Rankuwa between 2003 and 2005. However, these results are contrary to recent genotyping results from Europe where unusual or reassortant strains and potential zoonotic strains are only responsible for 2.5% cases[11].

Continuous monitoring of the circulating rotavirus strains will be required to assess the impact of vaccination with a monovalent vaccine on rotavirus serotype epidemiology and the emergence of any strains in response to the vaccine pressure.

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Patients who kindly agreed to participate in surveillance.

VIRAL HAEMORRHAGIC FEVER OUTBREAKS IN SOUTH AFRICA, 2007-2009

Outbreak Response Unit and Special Pathogens Unit, National Institute for Communicable Diseases

Introduction

The viral haemorrhagic fevers are characterized by high case mortality rates of up to 90% and although considered rare they have the potential to cause explosive outbreaks, especially in the nosocomial settings. The African viral haemorrhagic fevers include Ebola, Marburg, Crimean-Congo haemorrhagic fever (CCHF), Rift Valley fever (RVF), hantavirus infection with renal syndrome, Lassa fever and related arenaviral infections. Of these, CCHF and RVF are endemic to South Africa. Here we report on the occurrence of laboratory confirmed cases of VHF in South Africa for the period 2007 to 2009.

Congo-Crimean Haemorrhagic Fever (CCHF)

One, eleven and three cases of CCHF were confirmed respectively during 2007 to 2009 from South Africa. These cases were reported from the Northern Cape, Free State, Western Cape, Eastern Cape, North West and Mpumalanga provinces. Tick bite exposures were reported in 60 % of these cases (n=9). In two of the cases (13.3%) exposure to possibly infected sheep tissues and blood were reported, and the source was unknown in the remainder of the cases. During 2008 tick bite associated cases were reported during the winter months of June and July when tick activity is considered to be low. Cases of CCHF have been confirmed in South Africa since 1981 from all the provinces, except the Limpopo province.

Rift Valley fever (RVF)

Small, focal outbreaks of RVF have been reported in South Africa since 2008. In 2008 RVF outbreaks were reported from Mpumalanga, Limpopo, Gauteng and the North West provinces, and in 2009 in KwaZulu-Natal and the Northern Cape. More recently, in 2010, the outbreak extended even further to the Free State province (not reported here). The northern Eastern Cape also reported outbreaks of disease in animals but no suspected human cases were investigated from this area. A total of 25 confirmed human cases have been associated with these outbreaks, 18 in 2008 and 7 in 2009. All of these cases were linked to occupational exposures and included veterinarians, veterinary students, farmers and farm workers and also a staff member from a veterinary clinical research farm. All of these cases have reportedly recuperated without sequelae.

Prior to these focal outbreaks, RVF was confirmed nine years ago, in 1999, in aborted buffalo in the Kruger National Park. No human cases were confirmed during this outbreak. The last reported cases of RVF in humans in South African prior to the 2008 outbreak were more than 30 years ago during the 1970s[1]. Molecular investigations of isolates of the 2008 and 2009 South Africa outbreaks indicated the close relation of the outbreak strains with isolates from the 2006-2007 East African outbreaks.

Arenavirus infections

A) Imported case of Lassa fever

In February 2007 a 46-year old public health physician from Nigeria was evacuated to South Africa for medical treatment. When the patient arrived in South Africa VHF was suspected and the Special Pathogens Unit (SPU) of the NICD-NHLS confirmed Lassa fever by reverse-transcription PCR and serology. The patient was isolated and contacts monitored. The patient passed away 5 days after admission to the South African hospital.

Lassa fever is common in West African countries but has not been described elsewhere. Seroprevalence in certain West African populations have been recorded as high as 55 %. Only an estimated 15 % of infected patients develop clinical illness with a range from mild, febrile manifestation to fatal multi-organ failure. This case was the first reported importation of Lassa fever in South Africa.

B) Nosocomial outbreak of novel arenavirus infection

A nosocomial outbreak caused by a previously unknown arenavirus was reported in Johannesburg during September and October 2008[2]. The index case was evacuated to South Africa for medical treatment from Lusaka, Zambia. The patient had a 10 day history of illness that was at the time clinically diagnosed as septicaemia, possibly typhoid or tick bite fever. The patient passed away after being admitted to the Johannesburg hospital for two days without a confirmed diagnosis. Three subsequent secondary cases were: a paramedic that was involved in the medical evacuation, a nurse taking care of the index patient, and a cleaner who cleaned the ward where this patient was hospitalised. One tertiary case, and the only survivor of the outbreak, involved a nurse that had contact with the paramedic. Intensive laboratory testing was done by SPU, NICD-NHLS, the Centers for Disease Control and Prevention (Atlanta, USA) and the University of Columbia (New York, USA). Within two weeks after the receipt of the first clinical specimen laboratory findings, including full genome sequencing indicated that the causative agent was an Old World arenavirus but not Lassa fever virus, which was provisionally named Lujo virus[2,3].

Concluding remarks

Apart from endemic VHFs, the first importations of highly pathogenic arenaviruses to South Africa occurred in 2007-2009. VHFs should be considered as a differential diagnosis in febrile patients with travel history but also when a risk assessment indicates probable occupational exposure to VHF agents.

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(Continued on page 16)

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HUMAN RABIES IN SOUTH AFRICA, 2009

Outbreak Response Unit and Special Pathogens Unit, National Institute for Communicable Diseases

A total of 15 human rabies cases were confirmed in South Africa during 2009 compared to 17 in 2008 and 14 in 2007. Rabies cases were reported from the Eastern Cape (n=7); KwaZulu Natal (n=4); Limpopo (n=2) and Mpumalanga (n=2) provinces. Seven of the cases were linked to dog exposures but a source of exposure was not known or reported for the remaining cases. In addition, six cases were confirmed from Namibia compared to four cases during 2008. Since 2005, more than ten cases have been consistently reported annually. This is in contrast to an average of less than ten cases confirmed during the 90's through 2004. The rise in the number of human rabies cases has been partially attributed to the re-emergence of canine rabies in the Limpopo province[1]. Following confirmation of 22 human cases in 2006 in the Limpopo province[1], the outbreak appears to be waning in the province with 1-3 cases being confirmed for 2007-2009. Unfortunately, an alarming rise in the number of dog rabies has been reported in the Mpumalanga province since 2008, particularly from districts were it has been effectively controlled before[2]. Although single confirmed human cases have been reported from the province, a number of suspected cases have also been investigated. Common problems with laboratory confirmation of human rabies cases in South Africa is the decline of consent for the collection of post mortem specimens and/or submission of inadequate specimens for laboratory testing. Likewise there have also been an increasing number of canine rabies cases from the Free State since 2000[3]. Only five cases of human rabies have been confirmed in the Free State since 1983, with one case since 2000. An increasing number of cases has also been confirmed from the Eastern Cape in the past 5 years.

The difficulty in clinical recognition of cases and inadequate awareness amongst medical health practitioners and the public are the major reasons for rabies remaining a largely neglected and underreported disease in most developing countries. Analysis of alternative sources of data such as dog bite registers could be an important contribution toward estimating and defining the true public health burden of rabies in countries were surveillance and clinical confirmation are low.

Report compiled by (alphabetical order): Lucille Blumberg, Patricia Leman, Janusz Paweska, Jacqueline Weyer

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Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 31 December 2008/2009*

Disease/Organism	Cumulative to 31 December, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Anthrax	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	0	0	0	0	0
Botulism	2009	0	0	0	0	0	0	0	0	0	0
	2008	0	0	0	0	0	0	0	0	0	0
Cryptococcus spp.	2009	1,353	541	2,146	1,433	452	806	62	782	620	8,195
	2009	1,458	513	2,395	1,626	695	863	783	100	635	9,068
Haemophilus influenzae, invasive disease, all serotypes	2008	32	24	170	43	4	21	4	6	88	392
	2009	39	22	159	43	4	27	8	11	75	388
Haemophilus influenzae, invasive disease, < 5 years											
Serotype b	2008	5	7	21	9	2	3	2	2	9	60
	2009	8	8	14	14	1	2	3	2	20	72
Serotypes a,c,d,e,f	2008	2	2	18	0	0	1	0	0	7	30
	2009	0	1	19	2	0	3	0	1	6	32
Non-typeable (unencapsulated)	2008	3	3	15	3	0	1	0	0	8	33
	2009	1	1	31	12	1	2	1	0	11	60
No isolate available for serotyping	2008	11	0	47	10	1	8	0	2	16	95
	2009	8	4	25	4	1	6	2	4	1	55
Measles	2008	7	1	12	5	1	3	2	5	4	40
	2009	80	164	4,109	421	220	131	62	455	215	5,857
Neisseria meningitidis, invasive disease	2008	29	21	224	34	5	36	8	15	88	460
	2009	36	18	202	32	3	67	9	19	75	461
Novel Influenza A virus infections***	2008	0	0	0	0	0	0	0	0	0	0
	2009	682	314	5,580	2,258	545	500	134	465	2115	12,635
Plague	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	0	0	0	0	0
Rabies	2008	8	0	0	5	3	1	0	0	0	17
	2009	7	0	0	4	2	2	0	0	0	15
**Rubella	2008	488	18	301	612	197	303	34	171	38	2,162
	2009	273	73	674	501	443	355	151	340	133	2,975
Salmonella spp. (not typhi), invasive disease	2008	105	30	491	112	14	46	20	28	88	934
	2009	39	27	312	92	5	32	12	24	71	614
Salmonella spp. (not typhi), isolate from non-sterile site	2008	229	61	505	192	59	121	22	53	177	1,419
	2009	156	38	726	156	5	118	18	52	215	1,484
Salmonella typhi	2008	10	1	22	11	2	26	0	0	10	82
	2009	12	3	26	4	0	8	1	1	11	66
Shigella dysenteriae 1	2008	0	0	0	0	0	0	0	0	1	1
	2009	0	0	1	0	0	1	0	0	0	2
Shigella spp. (Non Sd1)	2008	187	85	510	142	33	96	28	23	409	1,513
	2009	250	73	606	155	8	71	16	19	414	1,612
Streptococcus pneumoniae, invasive disease, all ages	2008	356	320	2,356	573	112	257	84	193	586	4,837
	2009	362	309	2,254	528	111	302	88	175	639	4,768
Streptococcus pneumoniae, invasive disease, < 5 years	2008	98	109	664	204	24	76	34	43	212	1,464
	2009	97	68	624	163	18	88	46	27	202	1,333
Vibrio cholerae O1	2008	0	0	2	0	0	32	0	0	0	34
	2009	2	0	47	0	618	310	0	28	4	1,009
Viral Haemorrhagic Fever (VHF)											
Crimean Congo Haemorrhagic Fever (CCHF)	2008	1	3	0	0	0	1	5	1	0	11
	2009	0	1	0	0	0	0	1	0	1	3
Other VHF (not CCHF)****	2008	0	0	7	0	10	4	0	0	0	21
	2009	0	0	0	5	0	0	2	0	0	7

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. For 2009 the total figure of 2975 includes 32 cases from unknown provinces.

*** Confirmed cases from NHLS and private laboratories nationally, province unknown for 42 cases

**** All Rift Valley fever except for 3 novel arenavirus (LuJo) during 2008

Table 2: Provisional laboratory indicators for NHLS and NICD, South Africa, corresponding periods 1 January - 31 December 2008/2009*

Programme and Indicator	Cumulative to 30 December, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Acute Flaccid Paralysis Surveillance											
Cases < 15 years of age from whom specimens received	2008	62	20	56	70	49	38	6	16	32	349
	2009	51	15	70	112	80	41	8	23	21	421
Laboratory Programme for the Comprehensive Care, Treatment and Management Programme for HIV and AIDS											
CD4 count tests											
Total CD4 count tests submitted	2008	181,394	288,330	532,480	632,903	195,272	114,213	169,153	199,338	45,285	2,358,368
	2009	220,278	353,434	643,375	837,362	233,081	131,300	223,035	259,028	52,466	2,953,359
Tests with CD4 count < 200/µl	2008	52,823	109,235	200,970	219,941	65,896	39,650	59,317	74,006	13,757	835,595
	2009	58,151	111,065	214,457	248,709	70,862	40,084	69,868	79,866	16,963	910,025
Viral load tests											
Total viral load tests submitted	2008	62,099	121,955	244,194	278,760	81,198	49,601	73,395	69,161	19,463	999,826
	2009	87,589	142,006	298,645	344,257	96,780	48,078	91,391	89,280	22,266	1,220,292
Tests with undetectable viral load	2008	49,851	60,730	145,664	159,613	50,997	29,578	41,995	38,357	10,690	587,475
	2009	71,785	87,365	206,394	245,034	68,029	35,734	58,268	60,159	14,225	846,993
Diagnostic HIV-1 PCR tests											
Total diagnostic HIV-1 PCR tests submitted	2008	17,076	24,821	48,838	60,526	14,403	9,410	13,873	11,921	3,123	203,991
	2009	17,220	28,384	54,437	74,438	17,802	10,754	17,852	20,205	3,593	244,685
Diagnostic HIV-1 PCR tests positive for HIV	2008	1,543	3,246	7,015	8,953	2,414	1,712	2,385	2,366	486	30,120
	2009	1,367	2,805	5,892	7,190	2,103	1,287	2,268	2,504	420	25,836

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.

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

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