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NICD NHLS

FOREWORD

This edition of the Communicable Diseases Surveillance Bulletin contains a review of key findings from the main surveillance programmes of the National Institute for Communicable Diseases (NICD) for 2007. A similar review of data from the preceding year has been published in the first bulletin of the year since 2006. [1,2]

2008 has seen the establishment of an editorial board to assist with bulletin activities. The main role of the editorial board will be:

- To assist with decision making regarding bulletin format and content
- To ensure that the bulletin adequately represents the activities of the NICD
- To support and advocate for bulletin activities

It is hoped that the editorial board will assist in enabling the bulletin to achieve its publication objectives. These are to be a quarterly scientific publication for the regular dissemination of:

- surveillance findings of the NICD
- reviews and updates on local topics of public health importance
- guidelines developed by the NICD
- reports of outbreaks
- updates on research activities of the NICD

The surveillance data included in this edition contain relevant information on current trends in disease burden and resistance. These data should assist in guiding public health policy in South Africa.

Cheryl Cohen
Editor

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SUSPECTED MEASLES CASE-BASED SURVEILLANCE, SOUTH AFRICA, 2007

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The National Institute for Communicable Diseases (NICD) is accredited by the World Health Organization (WHO) to perform measles and rubella IgM testing for national case-based measles surveillance. Blood and urine specimens from suspected measles cases nationally are submitted to NICD for measles and rubella testing. Approximately 60% of suspected measles cases from Free State Province are tested at the NHLS Universitas Academic Laboratory at the University of the Free State (UFS). The numbers presented here represent specimens received by the NICD and UFS 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 (Dade-Behring, Marburg, Germany) diagnostic kits for the presence of anti-measles and anti-rubella immunoglobulin M (IgM). Amplification of ribonucleic acid (RNA) for genotyping was attempted on all 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).

During 2007 there were 3230 suspected cases of measles on whom blood specimens were submitted to the laboratory; of these 1938 had a urine specimen submitted. All provinces met the criteria for sufficient number of specimens collected, i.e. more than 2 suspected measles cases per 100,000/population with an overall figure of 6.8/100 000 population. However, 12 districts did not meet the criteria, i.e. one each in the Eastern Cape, Gauteng and Nothern Cape, 2 in the Free State, and 7 of the 11 districts in KwaZulu-Natal. The case detection rate in 2007 was lower than in 2006 (14.0/100 000), when a large outbreak of rubella occurred which may have contributed to increased reporting in that year.

1. Measles

There were 32 cases with positive measles IgM serology results in 2007 as compared to 81 cases in 2006 (Figure 1). Nine cases were from Gauteng, and 6 from the Eastern Cape (Table 1). Ages of patients with positive measles IgM results ranged from 10 months to 34 years (median 5 years). Of the 32 IgM positive cases 14 (44%) had a urine specimen available for PCR testing, and of these 6 were confirmed on PCR. Genotypic analysis was possible on 4 cases which belonged to clade B, genotype B3.1. One patient had a history of vaccination 6 days before the onset of rash and sequencing confirmed that the strain identified on PCR was vaccine virus. One additional case had a history of vaccination 2 weeks prior to the onset of rash but urine was not available for PCR. Ten of the measles IgM positive cases were also positive for rubella IgM.

Of 53 cases with equivocal measles IgM serology, 28 (53%) had urine specimens available for PCR testing. Two of these were identified as PCR positive for measles on urine specimens. The genotypes for these cases were also clade B, genotype B3.1.

Two cases from Mpumalanga were epidemiologically linked. Both cases were adults who resided in the same household. Preliminary investigation suggested that all other cases were sporadic importations; however detailed epidemiologic data were not available to the laboratory for the majority of cases.

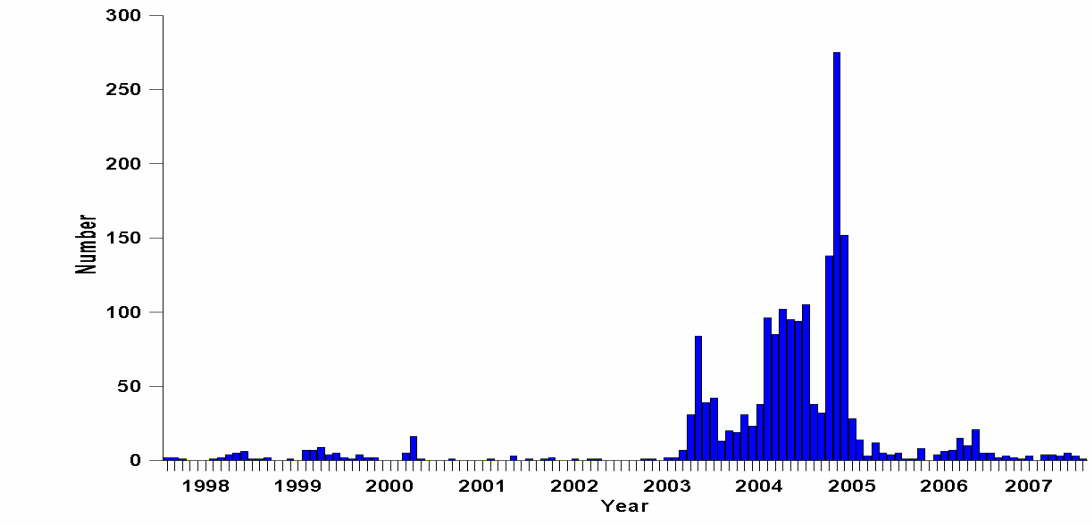

Figure 1: Number of measles IgM positive cases by year and month, South Africa, 1998-

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, 2006 and 2007.

Indicator	Year	Provinces									
		EC	FS	GA	KZ	LP	MP	NC	NW	WC	Total
Number of SMC	2006	579	106	1542	829	1209	841	372	754	415	6647
	2007	602	70	605	421	407	354	143	221	407	3230
SMC/100 000 population	2006	11.1	3.6	16.8	8.5	21.3	25.9	40.9	19.6	8.7	14.0
	2007	8.5	2.4	6.2	4.3	7.6	9.8	13.0	6.6	8.4	6.8
Measles IgM positive	2006	4	0	24	5	4	9	5	29	1	81
	2007	6	1	9	3	2	6	0	2	3	32
Rubella IgM positive	2006	290	62	612	460	631	282	156	367	120	2980
	2007	293	20	141	213	119	60	36	65	118	1064

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

2. Rubella

There were 1064 rubella IgM positive cases reported in 2007; the largest number were from the Eastern Cape. Patients' ages ranged from 11 months to 54 years with a median of 6 years. 56 (5.3%) of the rubella IgM positive results were from females aged ≥12 years.

The percentage of patients testing rubella IgM positive was highest (44.3%) in the 10-14 year age group followed by that of the 5 - 9 year age group (30.2%) (Figure 2).

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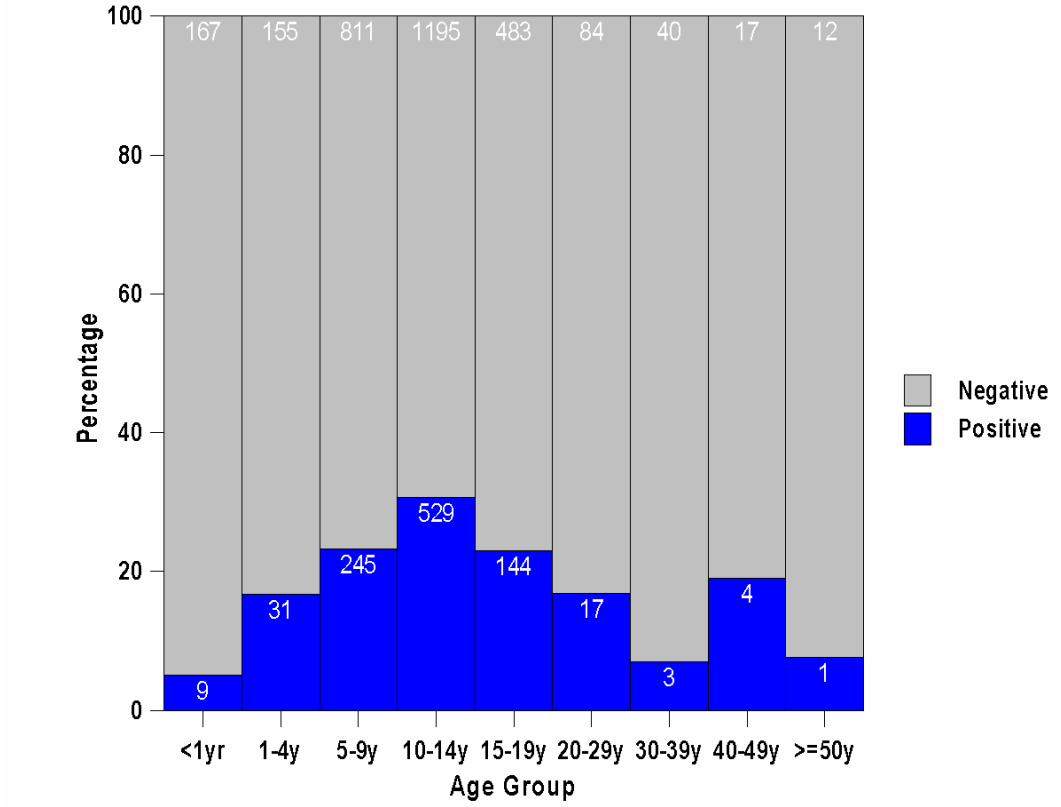

Figure 2: Percentage of specimens submitted for measles case-based surveillance testing rubella IgM positive per age group, South Africa, 2007 (inset numbers indicate the number of specimens testing positive and negative for rubella IgM respectively).

Discussion

The incidence of measles in South Africa has declined markedly since the widespread outbreak of 2003-2005. With this decrease in incidence, the laboratory diagnosis of suspected measles cases becomes more challenging as the predictive value of testing decreases. The three scenarios faced by the surveillance and the laboratory staff in the face of a positive measles IgM is whether the patient is a case of acute measles, or the test result is false positive, or the patient has been recently vaccinated against measles. Well-developed case definitions are useful in the main, but may not always have the required level of specificity. Of the 32 measles IgM positive cases it is likely that not all were true measles cases. The PCR-positive cases are likely sporadic and imported cases based on current measles virus genotype mapping in Africa. Genotyping provides information on whether there is endemic circulation or importation of measles. That no further cases were identified would indicate that coverage may be sufficiently high in these areas of sporadic cases to prevent transmission. Ten cases were also positive for rubella IgM and two cases had a history of recent vaccination (in one of whom vaccine virus was confirmed on PCR). Cross-reactivity with rubella, parvovirus or other viruses has been previously documented.[1] Positive measles serology following vaccination requires clearly defined criteria to exclude such cases. The difficulties in correctly interpreting the laboratory data are not insubstantial. For example, 30% of cases may be missed by serological testing in specimens collected less than 3 days of onset of rash.[1] Alternative strategies for measles diagnosis including rising IgG titres can be considered but require collection of a second specimen. Similarly, the use of alternative specimen collection, e.g. oral fluid or dried blood spots has advantages and disadvantages from both surveillance and laboratory perspectives. Thus, the identification of measles virus in urine using PCR provides a convenient tool for confirmation and genotyping. Almost half of all suspected measles cases did not have a urine specimen submitted for PCR testing, thus confirmatory PCR testing was not possible. In addition two PCR-positive cases were identified on urine specimens from cases who had equivocal measles IgM serology. Clinical data on suspected measles cases, particularly dates of measles vaccination and details of clinical symptoms with date of onset are essential for evaluation of cases. Unfortunately these data are not available for the majority of specimens. Submission of urine specimens with relevant clinical data is essential for all suspected measles cases. Nevertheless, the continued management of all positive IgM cases as true cases until proved otherwise must remain in place given the high infectiousness of measles.

Acknowledgements: we would like to thank Cardia Fourie, Teresa Mashaba, Lynn Harvey and Xolisa Stuurman for their assistance with laboratory processing of specimens.

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ACUTE FLACCID PARALYSIS (AFP) SURVEILLANCE, 2007

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Acute flaccid paralysis (AFP) surveillance is part of the World Health Organization (WHO) worldwide campaign to eradicate poliomyelitis. All cases of AFP, including Guillain-Barré syndrome, in children less than 15 years of age, or a patient of any age diagnosed as polio by a medical doctor must be regarded as possible polio cases until proven otherwise.

1. National Polio Isolation Laboratory

The NICD serves as the 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 year, 1756 stool specimens were received from patients with AFP from these seven countries. Of these 96 were from patients with onset of paralysis prior to 2007, or patients who were subsequently considered not to have AFP. Of the remainder, 550 specimens were from 266 South African cases, and 1107 from 577 cases from the six other countries served by the NICD (Figure 1).

South Africa

A further 11 specimens were received in the first two weeks of January 2008 from patients with onset of paralysis in 2007. Case detection rate by province ranged from 1.1 to 4.0, with the overall rate being 1.8 cases per 100 000 population. The percentage of adequate stool specimens per province ranged from 66.8% to 91.3%, with an overall rate of 78.6% (Figure 2). Non-polio enteroviruses were isolated from 73 specimens, non-enteroviruses from a further 26 specimens and Sabin types 1, 2 and 3 from 23 specimens from 14 patients.

Other southern African countries

The majority of specimens received were from Angola i.e. 586 specimens from 293 patients. Wild type polio 1 was isolated from 8 patients from Angola with onset of paralysis between 25 April and 8 July 2007. With the exception of one patient from Benguella, all were from 4 districts in Luanda province. Non-polio enterovirus was isolated from 165 specimens, and non-enterovirus from a further 3. Poliovirus identified as Sabin virus was isolated from the specimens of 16 patients from Angola and 2 from Namibia.

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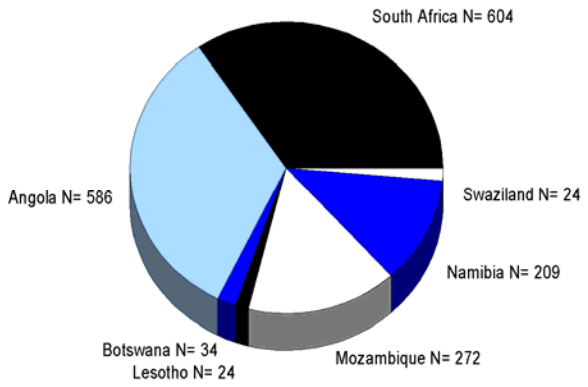

Figure 1: Number of stool specimens from AFP cases received for virus isolation by country, southern Africa, 2007.

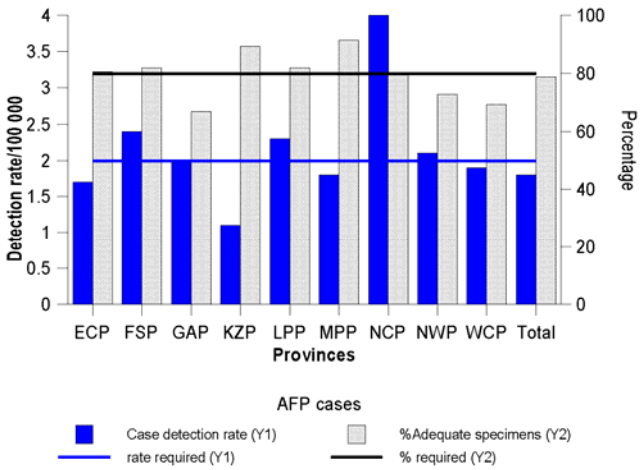

Figure 2: AFP case detection and stool adequacy rate, South Africa, 2007 (only patients from whom stool specimens were received included).

2. Polio Molecular Unit

During 2007, the unit received 1245 poliovirus isolates which were characterized as vaccine or wild type using two intratypic differentiation methods, PCR and ELISA. These isolates were sent to the NICD from National and Regional laboratories throughout Africa namely, Angola, Benin, Burkina Faso, Burundi, Cameroon, Chad, Cote d'Ivoire, Congo, Eritrea, Ethiopia, Kenya, Madagascar, Malawi, Mozambique, Namibia, Nigeria, Niger, Democratic Republic of Congo (DRC), Rwanda, South Africa, Somalia, Sudan, Uganda, Zambia and Zimbabwe. Original specimens from AFP cases were received from several southern African countries and any polio isolates were treated as above. The total number of wild poliovirus cases with onset of paralysis in 2007 was 366. Nigeria still remains the highly endemic country in Africa with a total of 286 wild type polioviruses (data not shown).

Poliovirus type 1 wild type isolates are distributed into three genotypes, India (SOAS), West African B (WEAF-B) and East African (EAAF) (Figure 3). The WEAF-B genotype consists of viruses from Nigeria, Niger, Chad, Benin, and Cameroon. The EAAF genotype consists of the viruses from Ethiopia and Somalia while SOAS viruses are from Angola and DRC.

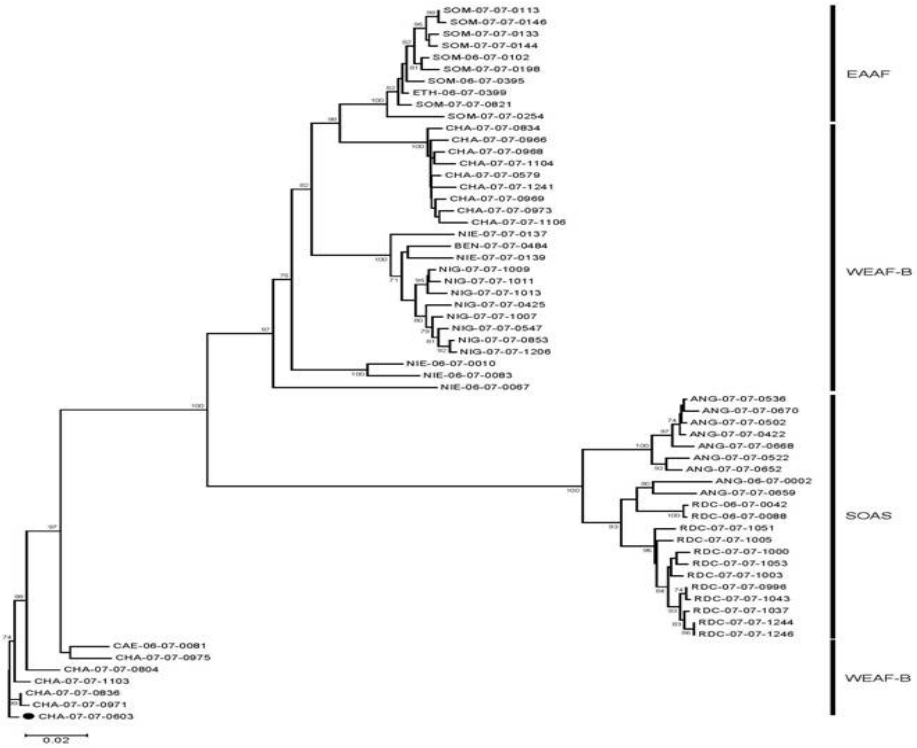

Figure 3: Neighbour-joining phylogenetic tree of wild-type 1 poliovirus (VP1 gene nucleotide sequences 906 nucleotides (nt). Numbers at branches nodes refer to the number of bootstrap repetitions (of 1000) at which the distal sequences grouped together. Closed circle represents an outgroup.

Of the identified PV1 wild-types, 44 were from DRC. Other wild type polioviruses identified in 2007 were from Angola, Benin, Cameroon, Chad, Ethiopia, Niger, Nigeria, and Somalia. In 2007, Chad was re-infected by two polio wild-type viruses. One of the cases of wild-type 1 was near the border with Cameroon. The second case was close to the border with Darfur, Sudan, in the same area from which a polio importation into Durfur occurred in 2004. The sequence of the index case showed a 98.68% identity to the case from Nigeria.

WEAF-B wild PV3 is divided into clusters and sub-clusters and cluster D still remains the most active cluster (Figure 4). The wild-type 3 case identified in Chad was from N'Djamena region and the first wild-type 3 since November 2006. The Chad virus has moved into the south of the country in the area that had not been covered by Supplemental Immunization Activities (SIAs) putting the neighboring countries like Central African Republic (CAR) at risk. The National Immunization Days (NIDs), covering 95% of the target population of the country, will be held on 23-26 February 2008, using monovalent oral polio vaccine type 1(mOPV1). No Vaccine Derived Polioviruses (VDPVs) were identified in 2007.

Acknowledgements: Special thanks to P Ngcobondwana, N Radebe, H du Plessis, D Lebambo, E Motaung, M Vandecar, C Simelane, A Sehata, P Coetzee, O Lentsoane and B Guliwe for assistance with laboratory diagnostics and molecular analysis.

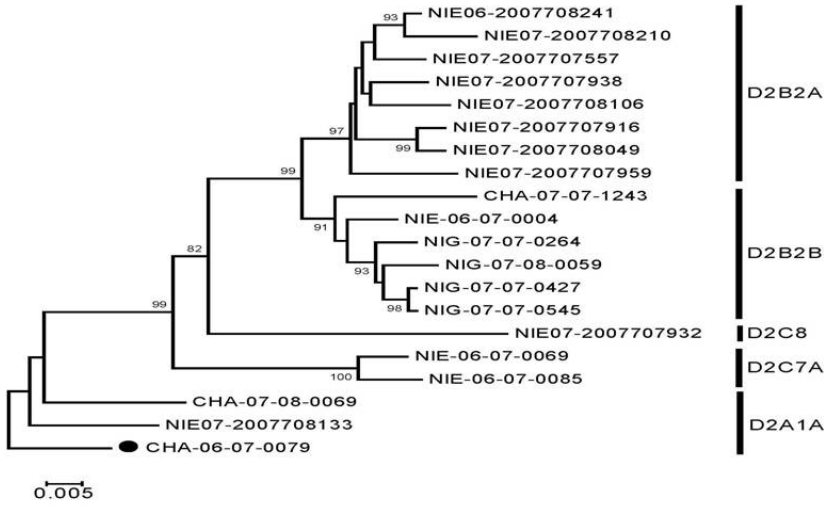

Figure 4: Neighbour-joining phylogenetic tree of wild-type 3 poliovirus VP1 gene nucleotide sequences 900 nucleotides (nt). Numbers at branches nodes refer to the number of bootstrap repetitions (of 1000) at which the distal sequences grouped together. Closed circle represents an outgroup.

RESPIRATORY VIRUS SURVEILLANCE, SOUTH AFRICA, 2007

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1. "Viral Watch" surveillance system

During 2007 a total of 1624 specimens were received for detection of respiratory viruses. Of these 1439 (88.6%) were received from the Viral Watch programme, started in 1984 and expanded substantially in 2005. This programme was specifically designed to monitor the timing of influenza activity in the community, and detect the type of influenza strains prevalent. During 2007, sites were added in the Free State, Mpumalanga and Limpopo, bringing the total number of practitioners reporting to 141. Throat swabs are submitted from these centres throughout the year from patients with respiratory tract infections of recent onset i.e. within 48 - 72 hours, and without obvious bacterial cause, and transported to the laboratory in viral transport medium for isolation of virus. Specimens from Gauteng, Free State, Mpumalanga, Limpopo and the Eastern Cape are submitted directly to NICD, whereas specimens from the Western Cape and KwaZulu-Natal are tested at the respective laboratories and positive specimens sent to NICD for confirmation, serotyping and sequencing.

A total of 533 influenza isolates were made, of which 511 (95.9%) were from the Viral Watch. The isolates were further identified as 390 influenza A, of which A H3N2 accounted for 215 (80.8%), and 143 influenza B, 76 (53%) of which were identified as B/Malaysia/2506/04-like (Figure 1).

The 2007 season started later than the previous 2 years. After some sporadic isolates early in the year, the number of isolates started to increase substantially from the week starting 25 June, although the first isolate of the season was made from a specimen collected on 12 April. The season peaked in the week starting 30 July, and the

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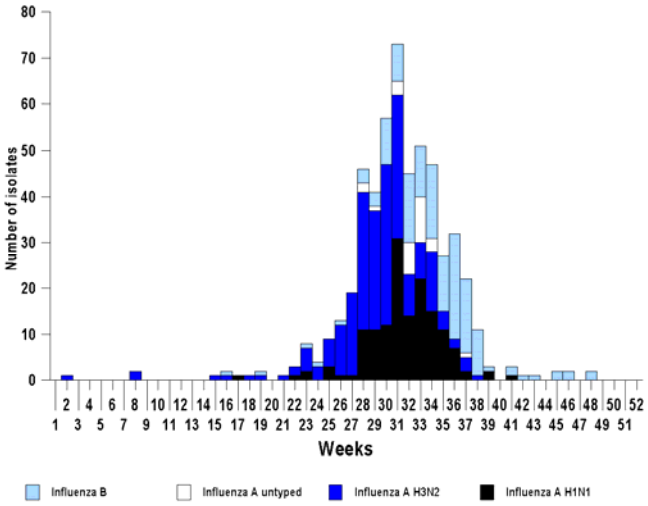

Figure 1: Number of influenza virus isolates by virus type and epidemiologic week, South Africa, 2007.

number of isolates started decreasing from the week starting 10 September, with the last isolate of the year being made from a specimen collected on 29 November.

A further 60 respiratory isolates made during the year including 37 respiratory syncytial virus, 7 parainfluenza virus (4 type 1, 1 type 2, 3 type 3, 1 untyped), and 9 adenovirus.

2. Characterisation of 2007 influenza isolates

A number of the influenza A and B isolates were characterised by partial sequencing the HA1 subunit of the haemagglutinin (HA) gene and performing phylogenetic analysis to determine genetic drift from the vaccine strains. Sequence analysis of the HA1 subunit revealed the H1N1 viruses isolated during the season showed major genetic drift from A/New Caledonia/20/99, the southern hemisphere vaccine strain for 2007. The majority of the isolates were related to the A/Solomon Islands/3/06 northern hemisphere 2007/8 vaccine strain.

The H3N2 isolates had drifted substantially from the A/Wisconsin/67/05 vaccine strain with amino acid changes mapping to antigenic sites A, B and D. Many of the isolates analysed were related to the B/Brisbane/10/07 reference strain.

Phylogenetic analysis of representative South African 2007 influenza B viruses from the two B genetic lineages showed that the B/Malaysia/2506/04-like isolates were related to the vaccine strain while those from the B/Yamagata/16/88 lineage had evolved substantially with many amino acid substitutions compared to the earlier isolates from this lineage.

The drift observed in the South African viruses from the respective vaccine strains was confirmed by the WHO Collaborating Centres for Reference and Research on Influenza (Melbourne and London). This trend was reported for many other countries in the southern hemisphere (WHO Consultation on the Composition of the Influenza Vaccine for the Southern Hemisphere, 2008, Geneva, September 2007). It was thus recommended that the H1N1, H3N2 and influenza B strains should all be updated for the 2008 southern hemisphere influenza vaccine, to provide a better match to the circulating viruses.

3. Respiratory consultations data mining surveillance system

During 2007, there were 1039256 consultations reported to the NICD through the respiratory morbidity data mining surveillance system. Of these 4635 (0.45%) were coded as due to influenza (ICD codes J10-11), 21578 (2.08%) were coded as pneumonia (ICD codes J12-18) and 14475 (1.39%) were coded as chronic respiratory disease (ICD codes J41-47). The timing of the peak in respiratory consultations was similar to the timing of the peak in influenza virus isolations (Figure 2).

Acknowledgements: Special thanks to Xolisa Stuurman, Lynn Harvey, Nathi Ndlovu and Teresa Mashaba in viral diagnostics and to the Netcare Hospital Group.

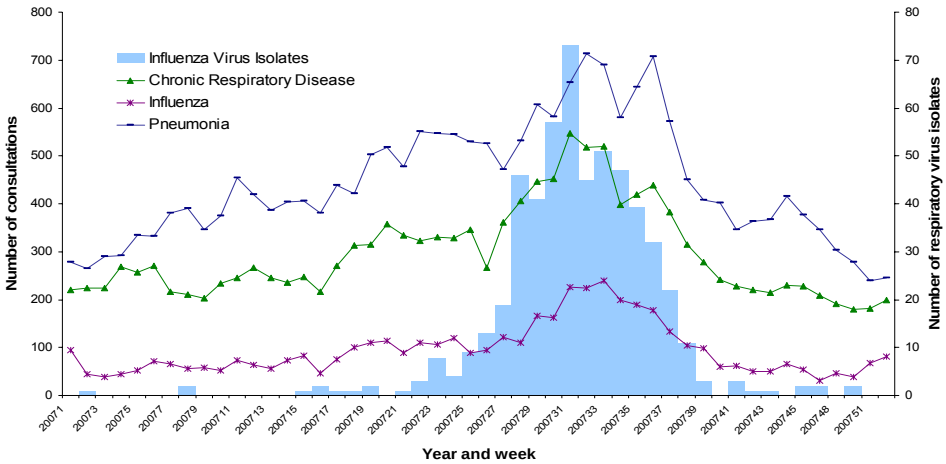

Figure 2: Number of consultations due to respiratory diagnoses of interest and influenza virus isolations by epidemiologic week, South Africa, 2007.

VIRAL HAEMORRHAGIC FEVERS, SOUTH AFRICA, 2007

Janusz Paweska[1], Jacqueline Weyer[1], Pat Leman[1], Robert Swanepoel[1], Lucille Blumberg[2]
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Of 48 suspected viral haemorrhagic fevers in South Africa in 2007, only one case of Crimean-Congo haemorrhagic fever (CCHF) was laboratory confirmed. There is a growing awareness of the disease and the possibility of the diagnosis of CCHF in clinically compatible cases as evidenced by many requests for differential diagnosis. The other cases included probable or confirmed cases of tick-bite fever, leptospirosis, herpes hepatitis, meningococcal disease, HIV infection, and leukaemias. The case of CCHF confirmed in 2007 worked on a sheep farm near Prieska in the Northern Cape Province prior to becoming ill. This is an area known to be endemic for CCHF in South Africa. The patient was admitted to the isolation ward in Kimberley Hospital where he recovered fully following supportive and antiviral treatment with ribavirin. Although there is no specific treatment for CCHF, there is some evidence that ribavirin can improve the prognosis if administered before day 5 after onset of illness.

A total of 187 cases of CCHF have been confirmed in southern Africa from the time that the presence of the disease was first recognized in 1981 up until the end of 2007, including seventeen in Namibia, one in DRC, one in Tanzania, and 168 cases in South Africa. The largest group of cases, 85/187 (45,5%), arose from known tick bites or the squashing of ticks; 72/187 (38.5%), arose from known or potential contact with fresh blood or other tissues of livestock and/or ticks; 7/187 (3.7%) nosocomial infections arose from contact with blood or fomites of known CCHF patients. In 23/187 (12.3%) cases there was no direct evidence of contact with livestock or ticks, but the patients lived in or visited a rural environment where such contact was possible. Most patients were employed in the livestock industry, and males constitute 157/187 (84%) of all cases of the disease diagnosed to date. The case fatality rate fluctuated around 30% in the first few years when CCHF was initially recognized in South Africa, but gradually declined to an overall rate of 19.9% (29/146) for the period of 1981-1998, most likely as a result of increased awareness leading to earlier recognition and institution of appropriate supportive therapy. Unfortunately, the case fatality rate drastically increased to 57.5% (23/40) for a period of 1999-2007 which might suggest that there is a decline in awareness of the disease among clinicians, resulting in delayed recognition of cases.

In February 2007, the unit confirmed an imported case of Lassa fever in a Nigerian public health physician. The patient was medically evacuated from Nigeria to South Africa and admitted to a hospital in Pretoria. The patient was involved in an immunization campaign in Nigeria, a country endemic for Lassa fever, before becoming ill. The Unit also consulted with the local health care workers with regards to the management of the patient and monitoring of contacts for possible secondary cases. This is the first known imported and managed case of Lassa fever in South Africa.

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RABIES IN SOUTH AFRICA, 2007

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Compared to 31 laboratory-confirmed human rabies cases in South Africa in 2006, 14 cases were confirmed in 2007. The majority of cases confirmed in 2007 resulted from dog exposure, but one followed mongoose exposure. Efforts to control rabies in the Limpopo Province, where in 2006 rabies suddenly re-emerged, resulted in significant reduction of reported cases in this region - there was only one case reported from the Limpopo Province in 2007, compared to 22 cases confirmed in 2006. The remaining cases were from KwaZulu Natal (n = 8) and Eastern Cape (n = 5) (Table 1). One case each from Botswana, Zambia and Swaziland and 2 cases from Namibia were confirmed in 2007 (Table 2).

Since 1983 when the Unit became involved in the diagnosis of human rabies in southern Africa, 468 human rabies cases have been confirmed. The vast majority of cases involved exposures to rabid dogs, but 5 of the confirmed cases were associated with mongoose exposures. In Southern Africa, an enzootic cycle of rabies in herpestid species (i.e. mongoose) is well known. Rabies virus is associated in this type of host only locally and on the island of Puerto Rico. Although reportedly up to 40% of confirmed veterinary cases of rabies are attributed to this biotype in South Africa, mongoose rabies specifically circulates in herpestids on the more sparsely human inhabited central plateau of South Africa, thus partially accounting for the few cases diagnosed. Of the 5 confirmed cases attributed to mongoose exposures, two cases involved patients with a history of rabies post-exposure prophylaxis. Both these cases had severe

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wounds to highly innervated areas such as the hands, which have often been associated with short incubation periods, suggesting that the virus enters nerves before vaccine-induced immunity can protect against disease.

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Table 1: Laboratory-confirmed cases of human rabies, South Africa, 2007.

Identifier	Age (years)	Sex	Place of exposure		Animal exposed to	Date of exposure (month and year)	Date of onset	Date of admission	Date of death	Hospital of death
			Town or district	Province						
SD	54	f	Empangeni	KZ	Cat	January 2007	-	2007/01/26	2007/01/27	Empangeni Garden Clinic
BM	Child	-	Bizana	EC	Dog	December 2006	-	2007/02/07	2007/02/12	Port Shepstone
TN	9	m	Pietermaritzburg	KZ	Dog	December 2006	2007/02/19	2007/02/22	2007/02/28	Greys
GM	8	m	Hibberdene	KZ	Dog	February 2007	2007/02/19	2007/02/24	2007/02/24	Port Shepstone
NM	-	-	Kokstad	KZ	Dog	February 2008	-	2007/03/27	-	Greys
AG	14	m	Umtata	EC	Dog	December 2006	-	2007/04/04	2007/04/04	Nelson Mandela Academic
TM	7	f	Vhembe	LP	-	-	2007/05/17	2007/05/29	2007/05/31	Died at home
MG	20	m	-	KZ	Dog	May 2007	-	2007/07/08	2007/07/08	Greys
JM	9	m	Umtata	EC	-	-	2007/08/13	2007/08/15	2007/08/17	Nelson Mandela Academic
SD	13	-	Umhlatuze	KZ	Dog	September 2007	2007/08/21	2007/09/21	2007/09/22	Ngwelezane
MZF	42	m	Umtata	EC	Dog	-	-	-	2007/10/17	Nelson Mandela Academic
DR 121/07	35	m	Nongoma	KZ	Dog	August 2007	-	-	2007/10/24	-
PM 1035/07	-	-	Pinetown	KZ	-	-	-	-	-	
SZ	8	m	Midlands	KZ	Dog	September 2007	-	2007/12/21	2007/12/27	Midlands

Key to abbreviations used in table: - Data not available, f - female, m - male, KZ - KwaZulu-Natal Province. EC - Eastern Cape Province, LP - Limpopo Province

Table 2: Laboratory-confirmed cases of human rabies, countries neighbouring South Africa, 2007.

Identifier	Age (years)	Sex	Place of exposure		Animal exposed to	Date of exposure (month and year)	Date of onset	Date of admission	Date of death	Hospital of death
			Town	Country						
TdJ	41	f	Lusaka	Zambia	Dog	March 2007	2007/05/17	2007/05/20	2007/06/04	Morningside Clinic
MD	4	f		Swaziland	Dog	May 2007	-	-	2007/06/04	Mbabane
KM	13	m	Gabarone	Botswana	Mongoose	July 2007	2007/08/11	2007/08/13	-	Princes Marina
PM 749/07	30	-	Oshakati	Namibia	-	-	-	-	2007/10/16	Oshakati
TT	-	m	Oshakati	Namibia	-	-	-	-	2007/12/04	Oshakati

Key to abbreviations used in table: - Data not available, f - female, m - male

SEXUALLY TRANSMITTED INFECTIONS SURVEILLANCE, SOUTH AFRICA, 2007

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Sexually transmitted infections (STI) microbiological surveillance was undertaken in the Western Cape (Cape Town) and in Gauteng (Johannesburg) between November 2006 and April 2007. This work was performed in collaboration with the NHLS Microbiology Department of Tygerberg Hospital, University of Stellenbosch. The aim of the surveillance was to determine a) the aetiology of the male urethritis syndrome (MUS), vaginal discharge syndrome (VDS) and genital ulcer syndrome (GUS), b) the prevalence of HIV co-infection in patients with these syndromes, and c) the antimicrobial susceptibility of *Neisseria gonorrhoeae* isolates to ciprofloxacin.

1. Aetiological Findings

In the Cape Town survey, which took place in November 2006 and again in January to February 2007, a total of 399 consecutive STI patients were recruited (94 VDS, 290 MUS, 15 GUS). Between January and April 2007, 506 consecutive STI patients were also recruited in Johannesburg (218 MUS, 210 VDS, 78 GUS). Among men presenting with MUS, only those with visible urethral discharge were asked to participate in the survey; men with dysuria alone were not enrolled.

Pathogens were detected by multiplex polymerase chain reaction (M-PCR) on swabs collected from VDS, MUS and GUS cases. Smears from VD cases were examined for the presence of bacterial vaginosis (BV) and *Candida* by microscopy. *Neisseria gonorrhoeae* was the most common aetiological agent for MUS (85%, Cape Town; 71%, Johannesburg) (Table 1). *Chlamydia trachomatis* was the second most frequent cause of urethral discharge (13%, Cape Town; 24%, Johannesburg). Trichomoniasis was detected more frequently among men with MUS in Johannesburg than in Cape Town (13% vs. 3%, p = 0.006). No pathogen was detected among 8% (24/290) of MUS cases in Cape Town and 15% (32/217) of MUS cases in Johannesburg.

Trichomoniasis was the most frequent STI accounting for VDS presentations (19%, Cape Town; 34%, Johannesburg) (Table 1). Bacterial vaginosis (BV), which is not an STI, was the most common cause of VDS in women (Table 2). Candidiasis, a potential marker for immuno-suppression due to HIV, was more frequently detected in Johannesburg compared to Cape Town (26% vs. 20%, p = 0.021). No aetiological cause was found for 27% (25/94) and 11% (22/206) of VDS cases in Cape Town and Johannesburg respectively.

Fewer patients were recruited with GUS compared to MUS and VDS during the surveillance exercise (Table 3). Genital herpes accounted for the majority of ulcers where an infectious aetiology was established (5/7, 71%, Cape Town; 40/46 87%, Johannesburg). Syphilis was the second most frequent cause of genital ulceration; single cases of chancroid and lymphogranuloma venereum were detected in Johannesburg and no cases of donovanosis were detected in either city.

Table 1: The prevalence of the sexually transmitted infections pathogens among patients with male urethritis Syndrome (MUS) and vaginal discharge syndrome (VDS) in Cape Town and Johannesburg.

Pathogen	Cape Town		Johannesburg	
	MUS (n = 290)	VDS (n = 94)	MUS (n = 217)	VDS (n = 206)
Neisseria gonorrhoeae	247 (85%)	15 (16%)	154 (71%)	27 (13%)
Chlamydia trachomatis	39 (13%)	12 (13%)	53 (24%)	32 (16%)
Trichomonas vaginalis	10 (3%)	18 (19%)	28 (13%)	70 (34%)

Table 2: Prevalence of bacterial vaginosis and candidiasis in patients with vaginal discharge syndrome (VDS) in Cape Town and Johannesburg.

Diagnosis	Cape Town	Johannesburg
	VDS (n = 90)	VDS (n = 207)
Bacterial vaginosis	41 (46%)	74 (36%)
Candidiasis	18 (20%)	53 (26%)

Table 3: Aetiology of genital ulcer syndrome in Cape Town and Johannesburg.

City	Herpes simplex virus	*Treponema pallidum*	*Haemophi-lus ducreyi*	*Chlamydia trachomatis L1-L3*	*Klebsiella granulomatis*
Cape Town (n = 15)	5 (33%)	2 (13%)	0 (0%)	0 (0%)	0 (0%)
Johannesburg (n = 76)	40 (53%)	5 (7%)	1 (1%)	1 (1%)	0 (0%)

Table 4: HIV seroprevalence for patients with male urethritis syndrome (MUS), vaginal discharge syndrome (VDS) and genital ulcer syndrome (GUS) in Cape Town and Johannesburg.

City	MUS	VDS	GUS
Cape Town	69/281 (25%)	40/93 (43%)	4/14 (29%)
Johannesburg	81/211 (39%)	104/199 (52%)	57/76 (75%)

Discussion

These data confirm that STI patients are an important group to target for HIV prevention initatives. The relatively high HIV prevalences recorded among the surveyed patients in both cities emphasize the importance of actively offering HIV voluntary counseling and testing (VCT), as well as discussing the benefits of antiretroviral therapy and knowledge of HIV status, during clinical consultations.

MUS is once again confirmed as the best candidate for longitudinal clinical surveillance of STI trends. It is for this reasons that 'new cases of MUS' is one of the required elements in the National Department of Health's minimum District Health Information System (DHIS) data set. In contrast, VDS is an inadequate syndrome to use as a marker of true STI prevalence since many cases are not, in fact, due to STIs. *Neisseria gonorrhoeae* and *Chlamydial trachomatis* were both confirmed as important pathogens, particularly in women where these diseases have serious complications.

Genital herpes is now the major cause of GUS. Consideration is currently being given to revising GUS flow charts to improve management of genital herpes, especially in terms of those co-infected with HIV who may develop chronic herpetic ulceration. For genital ulceration, it is important to provide high quality counselling and health education around genital herpes, which may be a recurrent and psychologically disturbing condition. Syphilis remains an infrequent but important cause of genital ulceration.

2. Antimicrobial Susceptibility Findings

In the survey reported here, 50/245 (20%) Cape Town gonococcal isolates and 47/149 (32%) Johannesburg isolates were resistant to ciprofloxacin. HIV serostatus was significantly associated with the isolation of quinolone (ciprofloxacin) resistant gonococci (QRNG) ($p = 0.034$). The prevalence of QRNG among MUS patients suggests that ciprofloxacin is no longer reliable as an effective therapeutic agent to treat gonorrhoea.

Given the facts that a) gonorrhoea is the most frequent cause of MUS (Table 1), b) MUS is the most common STI presentation in men, and c) the high prevalence of HIV co-infection among MUS patients (Table 4), it is important for ciprofloxacin to be replaced with either cefixime or ceftriaxone as first-line anti-gonococcal therapy – the revised Essential Drugs Programme Primary Care guidelines will address this issue.

Acknowledgements: Thanks for the successful completion of this surveillance go to: a) the Western Cape team: Professor Elizabeth Wasserman, Ms. Nina du Plessis, Ms. Marie Slabbert, Sr. Anita van Zyl, b) the Gauteng team: Sr. Martha Sello, Mr. Alex Vezi, Mr. Sipho Mbabela, Sr. Dudu Ntuli, Mr. Obed Mohlomonyane, c) Dr. Simba and staff at Alexandra Health Centre, d) Sr. Amila Latif and team at Salt River Clinic, e) Ms. G. Sifanelo at the Cape Town Health Department, f) Laboratory Staff at the STI Reference Centre, and f) PEPFAR and CDC-South Africa (funding through the NICD:CDC Co-operative Agreement).

ANTHRAX, PLAGUE AND BOTULISM IN SOUTH AFRICA, 2007

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Incidence of these diseases is low in humans in South Africa, but the results of laboratory investigations of suspected cases are reported here, because of their potential public health impact. Most suspected cases of anthrax were from the Northern Cape Province. All laboratory investigations for these 3 pathogens were negative in 2006 and 2007 (Table). Surveillance for plague is mainly in the form of rodent sampling in a few sentinel sites, most intensively in the Coega development zone, Eastern Cape Province (all results negative in 2006 and 2007; data not shown).

Table: Numbers of suspected human cases with specimens submitted for testing at NICD 2006-2007.

	2006	2007
Anthrax	11*	11
Botulism	3	3
Plague	1	1

*In addition, 78 persons exposed in 'white powder' incidents had nasal swabs cultured

GERMS-SA SURVEILLANCE REPORT, SOUTH AFRICA, 2007

Introduction

GERMS-SA is a national laboratory-based surveillance programme for bacterial and fungal diseases. The programme, coordinated by the National Institute for Communicable Diseases, is a collaborative effort between participating South African clinical microbiology laboratories, academic and public health partners.[1] Additional data can be found in the GERMS-SA Annual Report (access at www.nicd.ac.za) to be published in April 2008).

The pathogen-specific reports that follow provide results emanating from the surveillance programme for 2007. Incidence rates were calculated using mid-year population estimates for each year, provided by Statistics South Africa, unless otherwise specified. A surveillance audit was performed for NHLS laboratories in 8 provinces (excluding KwaZulu-Natal) between January and December 2007 using the NHLS Corporate Data Warehouse (CDW); the purpose of this audit was to detect laboratory-confirmed cases not reported to the surveillance programme. The audit included all pathogens, except *Pneumocystis jirovecii* and diarrhoeagenic *E. coli*. Audit cases are included in case counts for 2007; more than 2,500 additional cases were detected by the surveillance audit. It is important to keep this in mind when interpreting 2006-2007 trend data.

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NEISSERIA MENINGITIDIS

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In 2007, 455 cases of meningococcal disease were reported to RMPRU, and an additional 47 cases were identified on audit: a total of 502 cases of laboratory-confirmed meningococcal disease were identified by the surveillance system during the year. Rates of disease (Table 1) did not change substantially from 2006; the most marked change was a reduction in disease in Gauteng Province. In keeping with the seasonal pattern of disease, the number of cases reported increased during the winter and spring months (Figure 1)[1;2]. Of all cases reported to RMPRU, cerebrospinal fluid (CSF) was the most common specimen yielding meningococci (Table 2).

Table 1: Number of cases and incidence rates of meningococcal disease as reported to RMPRU by province, South Africa, 2006 and 2007.

Province	2006		2007	
	n	Cases/100,000	n	Cases/100,000
Eastern Cape	32	0.45	18	0.25
Free State	46	1.55	39	1.31
Gauteng	364	3.95	254	2.71
KwaZulu-Natal	20	0.21	33	0.34
Limpopo	8	0.14	9	0.16
Mpumalanga	26	0.80	26	0.79
Northern Cape	14	1.54	9	0.98
North West	25	0.65	37	0.95
Western Cape	69	1.45	77	1.59
South Africa	**604**	**1.27**	**502**	**1.05**

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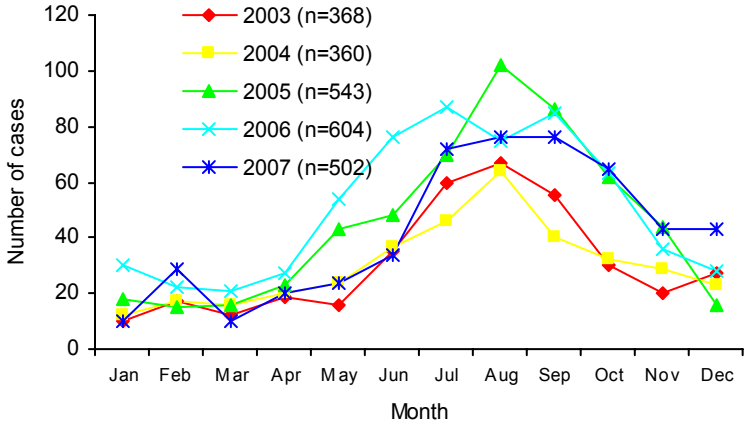

Figure 1: Number of cases of laboratory-confirmed meningococcal disease in South Africa as reported to RMPRU by month and year (2003-2007).

Table 2: Number and percentage of cases of meningococcal disease as reported to RMPRU by specimen type, South Africa, 2006 and 2007.

Site of specimen	2006		2007	
	n	%	n	%
CSF	448	74	384	76
Blood	152	25	116	23
Other	4	1	2	0.4
Total	**604**		**502**	

Cases of W135 disease were reported from all provinces. In Gauteng Province, the burden of serogroup W135 disease decreased in 2007, with incidence of disease estimated at 3/100,000 population, and most of that disease being due to W135 (143/194, 74%) (Table 3). The preponderance of serogroup B disease in Western Cape Province was still noted: 25/51 (49%) of all cases serogrouped.

Burden of disease was greatest in children less than five years of age. Age and serogroup -specific incidence rates show that infants were at greatest risk of disease for all serogroups (Figure 2).

Preliminary analysis of case fatality rates, as calculated in enhanced surveillance sites where in-hospital outcome is specifically looked for, was 35/183 (19%). This rate was similar compared to 2006 (30/224, 13%; p=0.12).

Only 6/277 (2%) isolates had penicillin MICs > 0.06µg/ml, and would be considered non-susceptible. The clinical relevance of increasing MICs is unclear, and penicillin is, at present, still being recommended as the drug of choice for therapy for confirmed meningococcal disease.

Table 3: Number of cases of meningococcal disease reported to RMPRU by serogroup and province (n=502, 353 (70%) with specimens or viable isolates available for serogrouping), South Africa, 2007.

Province	Serogroup								
	No isolate available	A	B	C	W135	Y	Z	Non-groupable	Total
Eastern Cape	8	0	2	2	3	3	0	0	18
Free State	14	0	5	3	12	4	1	0	39
Gauteng	60	5	30	4	143	12	0	0	254
KwaZulu-Natal	15	0	4	4	10	0	0	0	33
Limpopo	2	0	0	0	6	1	0	0	9
Mpumalanga	13	0	1	0	9	3	0	0	26
Northern Cape	2	0	2	3	2	0	0	0	9
North West	9	0	4	1	21	2	0	0	37
Western Cape	26	0	25	7	15	4	0	0	77
South Africa	**149**	**5**	**73**	**24**	**221**	**29**	**1**	**0**	**502**

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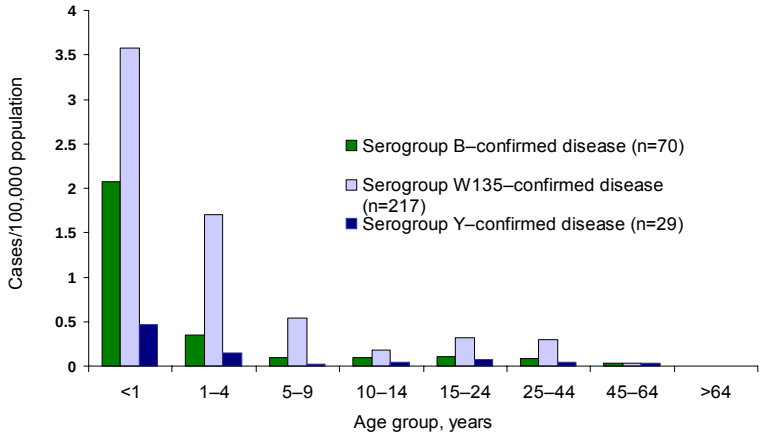

Figure 2: Reported age-specific incidence rates for confirmed serogroups B, W135 and Y, South Africa, 2007 (of 502 cases reported, 485 had known age, and 353 had specimens or viable isolates available for serogrouping).

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HAEMOPHILUS INFLUENZAE

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The number of cases of *Haemophilus influenzae* invasive disease reported in 2007 to RMPRU was 332, while an additional 88 cases were identified during the national audit (total number of cases available for analysis was 420). Of these, 226 (54%) had isolates or specimens available for serotyping, and 84/226 (37%) were confirmed as serotype b (Table 1). Serotype b isolates were more likely to be isolated from CSF than non-typeable *H. influenzae* (40/84 [48%] vs. 7/94 [7%], p<0.001) (Table 2).

Since the introduction of the *H. influenzae* serotype b (Hib) conjugate vaccine into the Expanded Programme on Immunisation (EPI) for South Africa in 1999, there has been a reduction in cases reported due to this serotype.[1] In 2007, a total of 54 cases of Hib were reported in children <5 years (Figure 1); an additional four cases were identified on polymerase chain reaction (PCR) testing of transport specimens. Serotype b has again become the more important *H. influenzae* causing disease in infants (Figure 2). Since 2003, rates of Hib disease as recorded by our surveillance network in infants <1 year of age have increased, and there seems to be a continued increase in 2007, although not reaching significance (p=0.094, chi-square test for trend, 2003 to 2007) (Figure 3). This increase in cases of Hib disease in children <1 year needs to be monitored carefully and further analysis of these cases will follow.

Twenty-six percent of serotype b strains were resistant to ampicillin (all producing beta-lactamase), 20 of 77 isolates tested, while 14% (13/94) of non-typeable strains were resistant (p=0.045).

Table 1: Number of cases of *Haemophilus influenzae* disease reported to RMPRU by serotype and province (n=420, 226 (54%) with specimens or viable isolates available for serotyping), South Africa, 2007.

Province	Serotype								
	No isolate available	a	b	c	d	e	f	Non-typeable	Total
Eastern Cape	21	0	3	0	0	0	2	2	28
Free State	12	0	6	0	1	1	2	1	23
Gauteng	83	5	34	1	1	2	19	57	202
KwaZulu-Natal	25	0	17	1	0	1	2	20	66
Limpopo	6	0	0	0	0	0	0	0	6
Mpumalanga	10	0	4	0	0	1	2	5	22
Northern Cape	2	0	1	0	0	0	0	0	3
North West	2	0	2	0	0	0	0	0	4
Western Cape	33	4	17	0	1	1	1	9	66
South Africa	**194**	**9**	**84**	**2**	**3**	**6**	**28**	**94**	**420**

(Continued on page 15)

Table 2: Number and percentage of cases of *Haemophilus influenzae* disease as reported to RMPRU by specimen type, South Africa, 2007

Site of specimen	No isolate available		Serotype b		Serotypes a, c, d, e, f		Non-typeable	
	n	%	n	%	n	%	n	%
CSF	45	23	40	48	16	33	7	7
Blood	123	63	41	49	31	65	76	81
Other	26	13	3	4	1	2	11	12
Total	**194**		**84**		**48**		**94**	

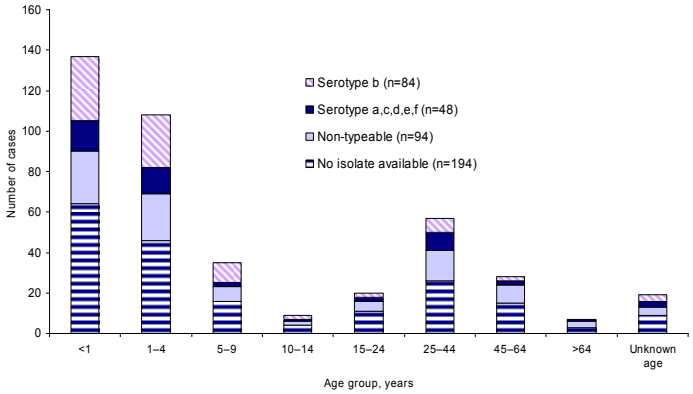
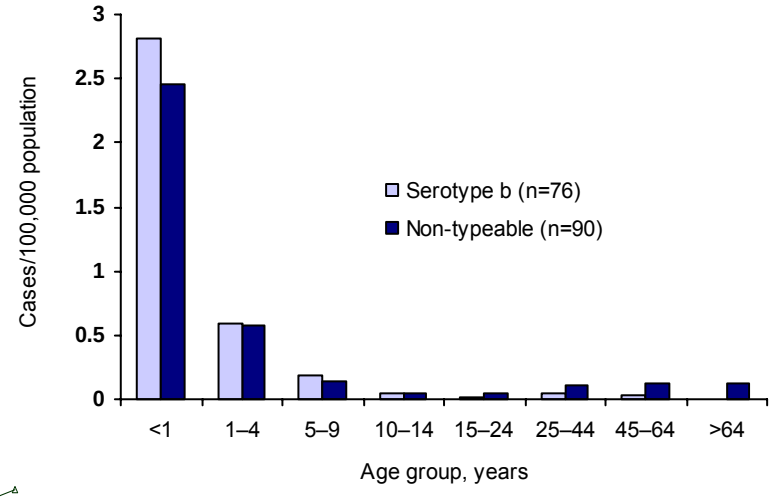


Figure 1: Number of cases of *Haemophilus influenzae* reported to RMPRU by serotype and age group, South Africa, 2007 (of 420 cases reported, 401 had known age, and 226 had specimens or viable isolates available for serotyping).

Figure 2: Reported age-specific incidence rates of serotype b and non-typeable *Haemophilus influenzae* disease, South Africa, 2007 (of 420 cases reported, 401 had known age, and 215 had viable isolates available for serotyping).



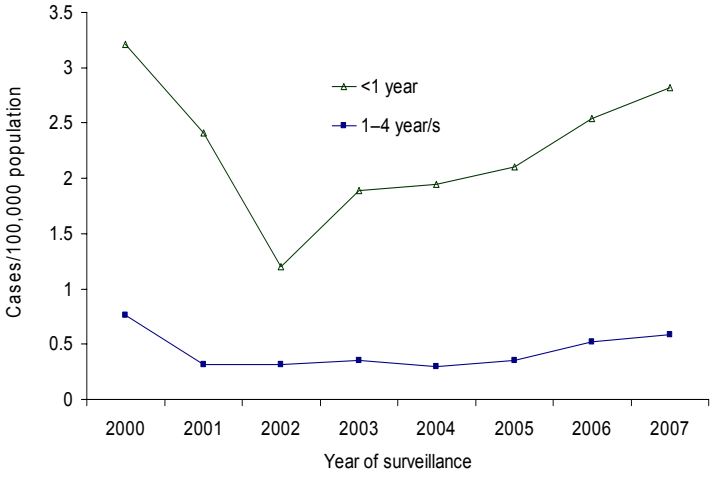


Figure 3: Incidence rates of *Haemophilus influenzae* serotype b disease in children <5 years, South Africa, 2000-2007 (excluding cases identified using polymerase chain reaction (PCR) on specimens – only done in 2007).

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STREPTOCOCCUS PNEUMONIAE

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The same trends of reported invasive pneumococcal disease as reported in previous years were documented in 2007, with disease rates by province varying widely (Table 1). The age group at highest risk of disease in South Africa was infants <1 year of age (Figure 1). The majority of episodes reported to RMPRU were diagnosed from positive blood culture specimens (Table 2).

Penicillin non-susceptible isolates have increased from 2006 (1429/3327, 43% in 2007 compared to 1107/3423, 32% in 2006, p<0.0001), and this ranged from 30% to 54% in different provinces (Table 3). Non-susceptible isolates were common in children less than 5 years of age (Figure 2). The increased resistance occurred in all age groups (data not shown), and on preliminary analysis may be related to an increase of serotype 19A and 6A strains that are resistant to penicillin and trimethoprim-sulfamethozaxole. This increase in non-susceptible strains will be investigated further.

PREVENAR® (7-valent conjugate pneumococcal vaccine) was launched in South Africa in the private sector in 2005 by Wyeth South Africa (Pty) Ltd, and is at present the only available vaccine for the prevention of pneumococcal disease in children <2 years. The proportion of disease in 2007 in children <5 years due to the seven serotypes in the vaccine (4, 6B, 9V, 14, 18C, 19F and 23F), and serotype 6A (ongoing evidence for cross-protection within this serogroup,[1] in South Africa is more than 70% according to our data (Table 4).This supports advocacy from clinicians and parents for vaccine price reduction and the possible inclusion of this vaccine in the EPI in the future.

Table 1: Number of cases and incidence rates of invasive pneumococcal disease reported to RMPRU by province, South Africa, 2006 and 2007.

Province	2006		2007	
	n	Cases/100 000	n	Cases/100 000
Eastern Cape	289	4.09	354	5.00
Free State	227	7.66	313	10.54
Gauteng	2107	22.89	2259	24.14
KwaZulu-Natal	462	4.75	537	5.47
Limpopo	102	1.80	148	2.59
Mpumalanga	209	6.44	277	8.47
Northern Cape	37	4.07	57	6.23
North West	139	3.61	221	5.70
Western Cape	488	10.27	567	11.69
South Africa	**4060**	**8.57**	**4733**	**9.89**

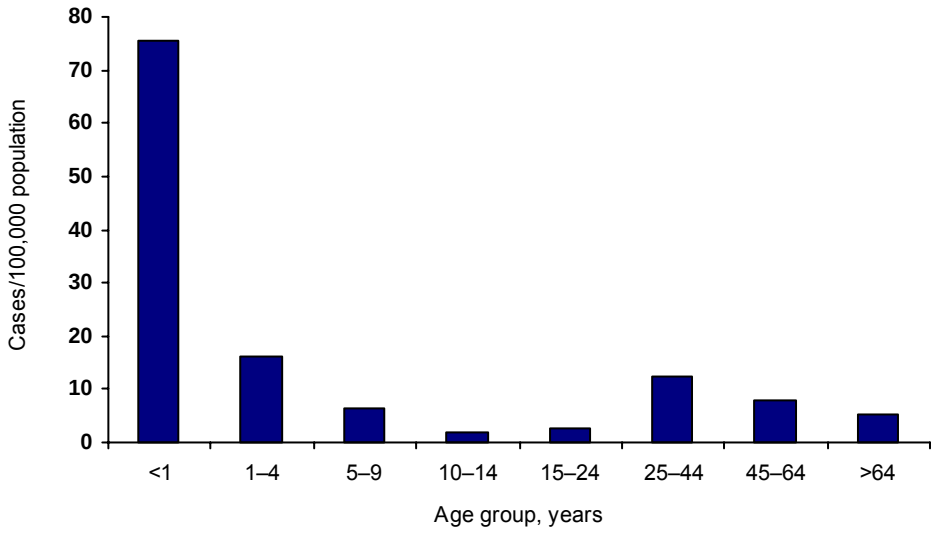

Figure 1: Reported age-specific incidence rates for invasive pneumococcal disease, South Africa, 2007 (4733 cases reported, age known in 4493).

(Continued on page 17)

Table 2: Number and percentage of cases of invasive pneumococcal disease reported to RMPRU by specimen type, South Africa, 2007.

Site of specimen	n	%
CSF	1723	36
Blood	2587	55
Other	423	9
Total	**4733**	

Table 3: Number and percentage of penicillin non-susceptible isolates from invasive pneumococcal disease cases reported to RMPRU by province, South Africa, 2007.

Province	No isolate available	Susceptible		Intermediately resistant		Resistant	
	n	n	%	n	%	n	%
Eastern Cape	135	126	58	93	42		0.0
Free State	84	134	59	95	41		0.0
Gauteng	682	918	58	649	41	10	0.6
KwaZulu-Natal	115	194	46	228	54		0.0
Limpopo	59	62	70	27	30		0.0
Mpumalanga	99	105	59	72	40	1	0.6
Northern Cape	16	23	56	18	44		0.0
North West	78	94	66	48	34	1	0.7
Western Cape	138	242	56	185	43	2	0.5
South Africa	1406	1898	57	1415	43	14	0.4

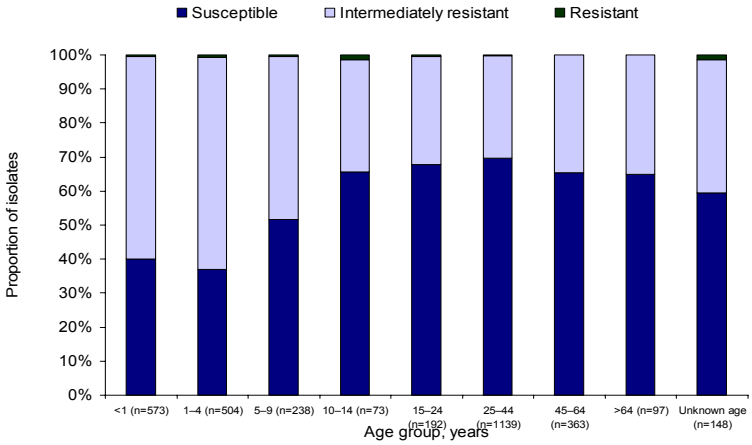

Figure 2: Number of cases of IPD reported to RMPRU in 2007 by age group and susceptibility to penicillin 4733 cases reported, 3327 with viable isolates).

Table 4: Number and percentage of cases reported in children less than 5 years of age caused by the serotypes contained in the 7-valent vaccine, South Africa, 2007.

Province	7-valent serotypes (4, 6B, 9V, 14, 18C, 19F and 23F)	Serotype 6A	Total isolates available for serotyping	% of IPD due to 7-valent serotypes including 6A
Eastern Cape	41	13	75	72
Free State	51	5	74	76
Gauteng	253	62	457	69
KwaZulu-Natal	104	24	171	75
Limpopo	17	2	22	86
Mpumalanga	37	2	51	76
Northern Cape	9	0	14	64
North West	24	2	35	74
Western Cape	94	27	178	68
South Africa	**630**	**137**	**1077**	

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CRYPTOCOCCUS spp.

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Results

During 2007, 6309 incident cases of cryptococcosis were reported. In total, 5762 isolates were received by MRU, of which 5327 (93%) were viable. Of 5277 isolates which were typed, 5169 (98%) were identified as *Cryptococcus neoformans*; the remaining 108 were identified as *Cryptococcus gattii*. Eighty-six cases of *C. gattii* infection were identified; most cases were diagnosed in the north-eastern regions of the country (Figure 1).

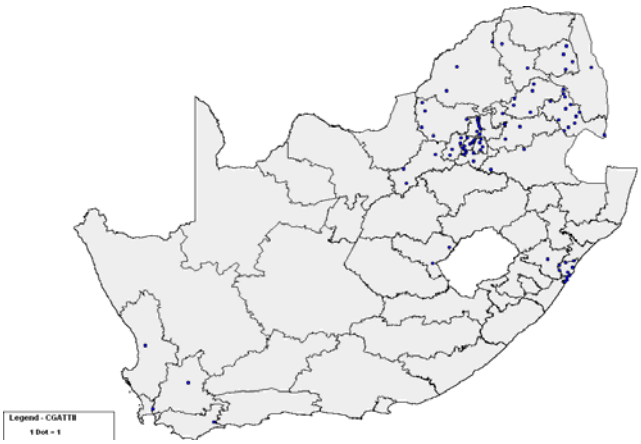

Figure 1: Cases of *Cryptococcus gattii* (n=86) by health district of South Africa, 2007.

The overall incidence rate in the South African general population was 13/100,000, unchanged from 2006. The incidence of cryptococcosis amongst HIV-infected individuals was 114/100,000 cases, and amongst people sick with AIDS was 10/1000 AIDS cases using projected denominators from the Medical Research Council Report on the Demographic impact of AIDS in South Africa.[1]

The overall trends in provincial incidence rates between 2006 and 2007 are difficult to interpret, given that surveillance audits may have influenced case-counting (Table 1). Most cases of laboratory-confirmed cryptococcosis continue to be diagnosed in urbanised or mining districts (Figure 2).

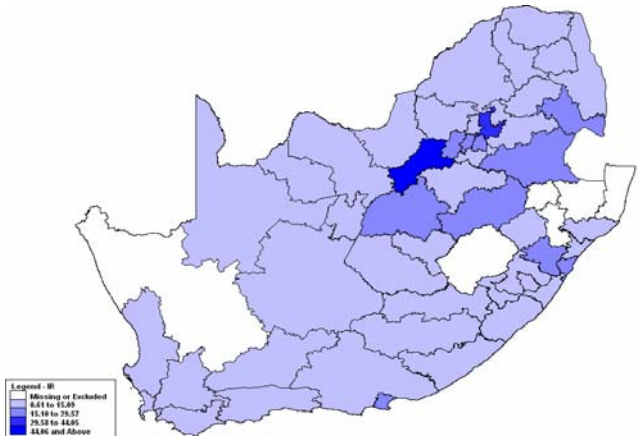

Figure 2: Chloropleth distribution map of incidence of cryptococcosis by health district in South Africa, 2007.

The highest incidence of cryptococcosis was in the 30-34 year age group (Figure 3). Where gender was known (6180/6309, 98%), 54% of cases occurred in females. In children under 15 years of age, 116 cases were identified.

Most incident cases (93%) were diagnosed with meningitis (laboratory tests on cerebrospinal fluid positive for

Table 1: Number of cases and incidence rates of *Cryptococcus* spp. as reported to MRU by province, South Africa, 2006 and 2007.

Province	2006*		2007§	
	n	Cases/100 000	n	Cases/100 000
Eastern Cape	1357	19	709	10
Free State	287	10	506	17
Gauteng	1856	20	1919	20
KwaZulu-Natal	1348	14	1265	13
Limpopo	215	4	333	6
Mpumalanga	439	14	550	17
Northern Cape	63	7	48	5
North West	378	10	567	15
Western Cape	353	7	412	8
South Africa	**6296**	**13**	**6309**	**13**

*In 2006, a surveillance audit was performed for NHLS laboratories in the Eastern Cape; 953 additional microscopy (India ink) or culture-confirmed incident cases were detected by audit.
§In 2007, a surveillance audit was performed for NHLS laboratories in 8 provinces (excluding KwaZulu-Natal); 936 additional culture-confirmed incident cases were detected on audit.

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Cryptococcus species), and 3.8% with fungaemia (Table 2). The remainder of cases (n=23) were diagnosed by culture of urine, sputum, pleural fluid and other specimen types.

Table 2: Number and percentage of cases of cryptococcal disease as reported to MRU by specimen type, South Africa, 2007.

Site of specimen	n	%
CSF	5863	93
Blood	244	3.8
Other	23	0.4
Unknown	179	2.8
Total	**6309**	

Of 1456 incident cases presenting to enhanced surveillance sites (tertiary or regional hospitals) and with completed clinical case report forms at the time of analysis, 459 cases (32%) died during the first hospital admission.

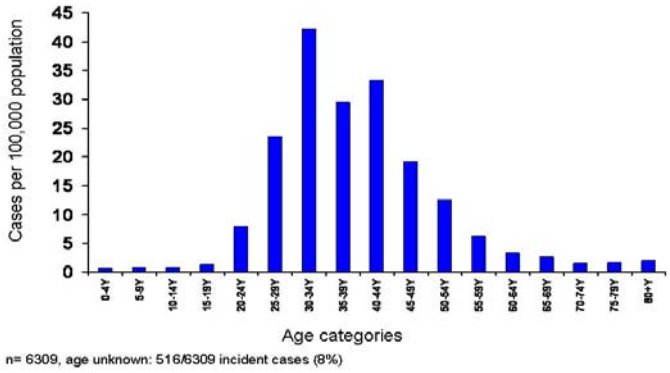

Figure 3: Age-related incidence of cryptococcosis in the general population, South Africa, 2007 (n = 6309, ages unknown in 8% (516/6309 cases).

Discussion

Overall, the number of laboratory-confirmed cases of cryptococcosis detected by GERMS-SA has not changed between 2006 and 2007. Most patients continue to be diagnosed with meningitis in urbanised centres, and this probably reflects clinician specimen-taking practices. The demographic profile of cases with cryptococcosis continues to mirror the profile of HIV-infected patients in South Africa. Paediatric cryptococcosis remains a relatively uncommon clinical entity, though it must be considered as part of a differential diagnosis. The in-hospital mortality of patients with cryptococcosis remains unacceptably high. It is hoped that the recent publication of Southern African guidelines for the clinical management of HIV-infected patients with cryptococcosis will have an impact on patient survival.[2]

Provincial trends have been influenced by the differing nature of surveillance audits performed during two consecutive years. In 2006, a surveillance audit was only performed for NHLS laboratories in the Eastern Cape; 953 additional microscopy (India ink) or culture-confirmed incident cases were detected by audit. In 2007, a surveillance audit was performed for NHLS laboratories in 8 provinces (excluding KwaZulu-Natal); 936 additional culture-confirmed incident cases were detected on audit. In the Eastern Cape, the smaller detected case number in 2007 may be explained by the incomplete surveillance audit performed in that year, compared with the more comprehensive audit undertaken in 2006. The increase in detected cases in Free State, Limpopo, Mpumalanga and North West may be explained by more active case-finding (driven by audits) in 2007. Despite active case-finding in 2007, stable case numbers in Gauteng, Western Cape and Northern Cape suggest that access to highly active antiretroviral treatment (HAART) through the National HIV/AIDS Comprehensive Care, Management and Treatment (CCMT) Programme may have made an impact. No audits were performed in KwaZulu-Natal in either year; stable case numbers in 2007 may also suggest the impact of access to HAART. A comprehensive surveillance audit to detect cases diagnosed by India ink or cryptococcal antigen testing at NHLS laboratories in 8 provinces will be completed for 2007; this should help to clarify the above findings. These preliminary findings will require further careful investigation.

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PNEUMOCYSTIS JIROVECII PNEUMONIA

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[1]Parasitology Reference Unit, National Institute for Communicable Diseases

In 2007, 286 cases of *Pneumocystis* pneumonia (PCP) were reported to the Parasitology Reference Unit through the GERMS-SA surveillance system (Table 1).

Table 1: Number of *Pneumocystis* pneumonia cases reported by province, 2006-2007

Province	2006	2007
Eastern Cape	25	30
Free State	6	16
Gauteng	177	144
KwaZulu Natal	7	20
Limpopo	0	0
Mpumalanga	17	12
Northern Cape	0	0
North West	0	13
Western Cape	72	51
South Africa	284	286

The number of cases reported was about the same compared to 2006. These numbers do not reflect the true burden of PCP in the country, as PCP is not fully reported, for several reasons. Few laboratories test for *Pneumocystis* pneumonia; bronchoscopy is expensive, and induced sputum requires specialized equipment and trained personnel to obtain adequate samples.[1]

Numbers of PCP isolates peak in children less than one year of age and in the 21 to 60 year age group (Figure 1). Of cases with known gender 59% (144/244) were female. Studies have shown that heterosexual women and women of unknown risk appear more likely than their male counterparts to contract PCP.[2]

The discharge diagnosis for PCP at enhanced sites was mostly unknown (204/252). Forty five of the patients have been discharged with a lower respiratory tract infection (LRTI), 2 with bactaeremia and one with meningitis.

Before the widespread use of prophylaxis, it was estimated that 80% of people with AIDS would eventually develop PCP.[3] Trimethoprim-sulfamethoxazole (TMP-SMX, cotrimoxazole) is used for treatment and prophylaxis. Prophylaxis has reduced the incidence of PCP, but has resulted in a significant correlation between the use of sulfa-drugs and point mutations in the dihydropteroate synthase (DHPS) gene.[4] US studies have shown that patients with DHPS mutations are three times more likely to die compared to those with wild-type DHPS genes.[4] Access to isolates from different areas in the country is useful for monitoring this aspect of cotrimoxazole resistance and the genetic diversity of strains.

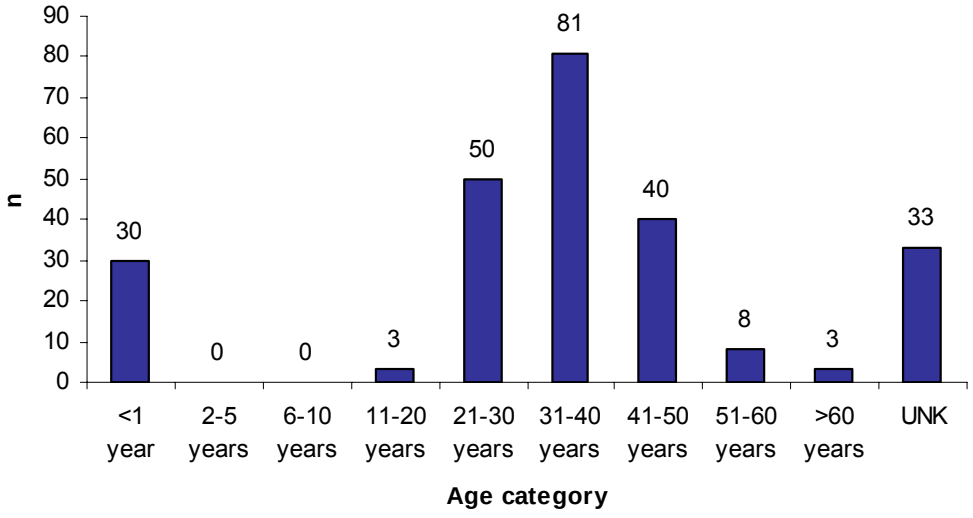

Figure 1: Number of *Pneumocystis jirovecii* positive cases by age group, South Africa,

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SALMONELLA ENTERICA SEROTYPE TYPHI

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[1]Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Table 1: Number of invasive and non-invasive *Salmonella* Typhi isolates (n =71) reported to EDRU by province, South Africa, 2007.

Province	Invasive *Salmonella* Typhi	Non-invasive *Salmonella* Typhi
Eastern Cape	10	2
Free State	0	1
Gauteng	20	1
KwaZulu Natal	9	1
Limpopo	4	2
Mpumalanga	6	6
Northern Cape	0	0
North West	2	0
Western Cape	7	0
South Africa	58	13

Table 2: Number of *Salmonella* Typhi isolates reported to EDRU (n =71) by age category, 2007.

Age category (years)	Number of isolates
<1	4
1 - 5	7
6 - 14	18
15 - 64	37
>64	0
Unknown	5
South Africa	71

Two isolates of *Salmonella* Paratyphi A from the Western Cape and two of *Salmonella* Paratyphi C were received, from the Western Cape and Gauteng respectively. One adult female with *Salmonella* Paratyphi A was of Indian origin and the isolate may have been imported. No travel history was elicited from the reaming three adults.

Numbers of *Salmonella* Typhi isolates were regarded as too low to calculate incidence rates. These results are for culture confirmed cases and thus exclude those patients in whom a serological diagnosis was made without culture.

Salmonella Typhi isolation by month shows seasonality, with increased numbers in January and February and October through December. No major outbreaks were detected in 2007.

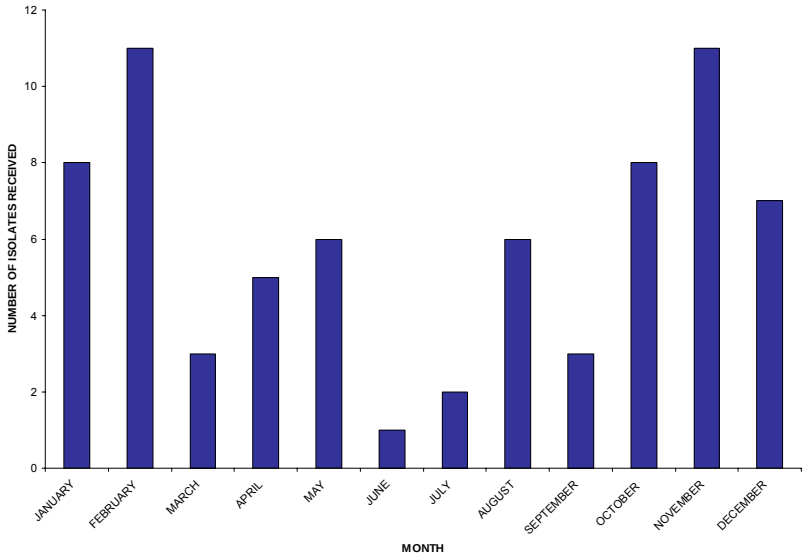

Figure 1: Number of *Salmonella* Typhi isolates reported to EDRU by month of isolation.

Certain antimicrobials are tested for epidemiological purposes only and should not be used for treatment of typhoid fever. All *Salmonella* Typhi isolates received in 2007 were susceptible to ciprofloxacin (Table 3), the treatment of choice, although the occurrence of nalidixic acid resistance is cause for concern. Nalidixic acid resistance may be used as a marker for quinolone resistance; it is indicative of the potential for an organism to develop fluoroquinolone resistance. Response to ciprofloxacin may be poor in the presence of nalidixic acid resistance.[1] The *Salmonella* Paratyphi A isolates were both resistant to nalidixic acid, but susceptible to ampicillin,

(Continued on page 22)

cotrimoxazole and chloramphenicol. One isolate of *Salmonella* Paratyphi C was fully susceptible to all antimicrobials tested, but the other was resistant to nalidixic acid.

Table 3: Results of antimicrobial susceptibility testing for all *Salmonella* Typhi isolates (n = 70) received by EDRU, 2007.

Antimicrobial tested	Susceptible (%)	Intermediate (%)	Resistant (%)
Ampicillin	91.4	0.0	6.6
Cotrimoxazole	91.4	0.0	6.6
Chloramphenicol	97.1	0.0	2.1
Nalidixic acid	92.9	0.0	5.4
Ciprofloxacin	100.0	0.0	0.0
Tetracycline	88.6	1.1	7.9
Kanamycin	100.0	0.0	0.0
Streptomycin	92.9	0.0	5.4
Imipenem	100.0	0.0	0.0
Ceftriaxone	100.0	0.0	0.0

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1. Crump JA, Barrett TJ, Nelson JT, Angulo FJ. Reevaluating fluoroquinolone breakpoints for *Salmonella enterica* serotype Typhi and for non-Typhi salmonellae. Clin Infect Dis 2003 July 1;37(1):75-81.

NON TYPHOIDAL *SALMONELLA ENTERICA* (NTS)

Karen Keddy[1] for GERMS-SA
[1]Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Table 1: Number* of invasive and non-invasive non-typhoidal *Salmonella* isolates reported to EDRU by province, South Africa, 2007 (n = 2082).

Province	Invasive non-typhoidal *Salmonella*	Non-invasive non-typhoidal *Salmonella*
Eastern Cape	87	197
Free State	48	46
Gauteng	478	357
KwaZulu Natal	114	110
Limpopo	28	56
Mpumalanga	37	139
Northern Cape	13	23
North West	43	58
Western Cape	90	158
South Africa	938	1144

*Incidence rates have not been calculated as there may be regional differences in specimen collection practices.

Table 2. Case numbers and incidence rates for invasive* non-typhoidal *Salmonella* reported to EDRU by age category, 2007 (n = 2085).

Age Category (years)	Cases		
	Non-invasive	Invasive	Incidence rate/100000 (invasive disease)
<1	261	203	19.11
1 - 5	194	97	1.92
6 - 14	74	54	0.60
15 - 64	486	516	1.71
>64	46	21	0.85
Unknown	86	47	-
Total	1147	938	1.96

*Incidence rates for non-invasive non-typhoidal *Salmonella* have not been calculated because not all cases of gastroenteritis due to non-typhoidal *Salmonella* may be cultured in clinical practice.

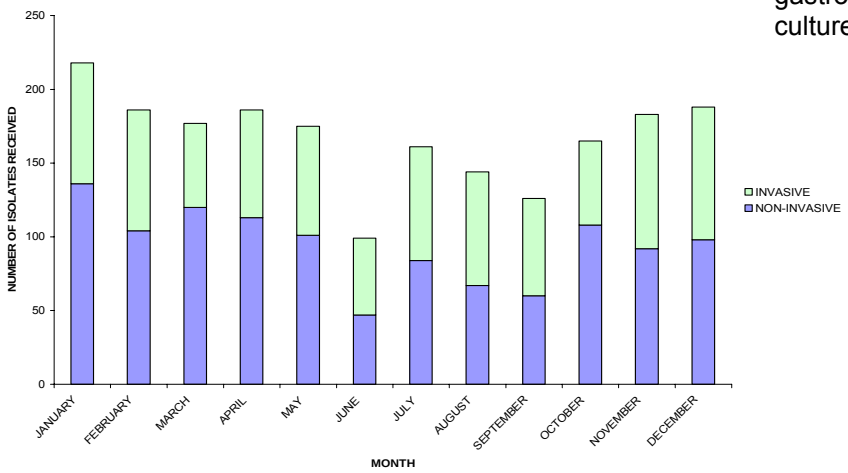

Figure 1. Number of non-invasive and invasive non-typhoidal *Salmonella* isolates reported to EDRU by month of isolation, 2007 including those identified on audit.

(Continued on page 23)

A lack of clear seasonality reflects the nosocomial nature of many of the invasive cases, as well as the burden of disease associated with HIV infection. Non-invasive disease appears seasonal.

Certain antimicrobial agents are tested for epidemiological reasons only and should not be used for treatment. Of those NTS isolates tested, 266 (17.7%) were noted to be extended spectrum beta-lactamase (ESBL) producers. Nalidixic acid resistance is a cause for concern because it is a marker of increasing resistance to the quinolones and is associated with poor response to fluoroquinolone treatment in clinical cases.[1] Nalidixic acid resistance, in combination with ESBL production, was identified in 209 (13.9%) NTS isolates. Pentavalent resistance (resistance to five or more antimicrobials) was observed in 513 (33.8%) isolates. Multi-drug resistant serotypes included primarily *Salmonella* Typhimurium and *Salmonella* Isangi (Table 5).

Table 3: Number of non-typhoidal *Salmonella* isolates reported to EDRU by anatomical site of isolation*, 2007.

Specimen	n	%
CSF	24	1.15
Blood culture	826	39.62
Stool	858	41.15
Other	377	18.08
Total	2085	100

*Note that many cases had multiple isolates, including those with isolates from an invasive site and a second isolate from stool.

Table 4. Results of antimicrobial susceptibility testing for all non-typhoidal *Salmonella* isolates (n = 2185) received by EDRU, 2007.

Antimicrobial tested	Susceptible (%)	Intermediately resistant (%)	Resistant (%)
Ampicillin	62.6	0.1	37.3
Cotrimoxazole	63.4	0.0	36.6
Chloramphenicol	66.7	1.5	31.8
Nalidixic acid	71.6	0.0	28.4
Ciprofloxacin	99.7	0.2	0.1
Tetracycline	54.7	10.9	34.4
Kanamycin	85.5	6.9	7.7
Streptomycin	66.2	0.0	33.8
Imipenem	100.0	0.0	0.0
Ceftriaxone	82.3	0.0	17.7

Table 5. Commonest invasive and non-invasive non-typhoidal *Salmonella* serotypes (n = 1193) serotyped by EDRU by province, 2007.

	Enteritidis	Isangi	Newport	Typhimurium	Virchow
Eastern Cape	10	51	1	80	1
Free State	16	3	1	41	0
Gauteng	97	42	9	358	6
Kwazulu-Natal	26	50	1	81	1
Limpopo	9	4	0	21	1
Mpumalanga	7	6	0	50	2
Northern Cape	0	0	0	8	0
North West	9	0	0	26	21
Western Cape	205	15	35	75	4
South Africa	199	171	47	740	36

Reference

1. Crump JA, Barrett TJ, Nelson JT, Angulo FJ. Reevaluating fluoroquinolone breakpoints for *Salmonella enterica* serotype Typhi and for non-Typhi salmonellae. Clin Infect Dis 2003 July 1;37(1):75-81.

VIBRIO CHOLERAE

Karen Keddy[1] for GERMS-SA
[1]Enteric Diseases Reference Unit, National Institute for Communicable Diseases

No *Vibrio cholerae* O1 isolates from cases in South Africa were received by EDRU in 2007. No imported cases were identified.

SHIGELLA

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[1]Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Table 1: Number of invasive and non-invasive *Shigella* isolates (n =1477) reported to EDRU by province, South Africa, 2007.

Province	Invasive *Shigella*	Non-invasive *Shigella*
Eastern Cape	10	196
Free State	4	81
Gauteng	27	432
KwaZulu Natal	7	123
Limpopo	2	25
Mpumalanga	7	126
Northern Cape	0	52
North West	0	41
Western Cape	9	349
South Africa	66	1425

The predominant burden of disease is in the under five-year age group (Table 2).

Table 2. Case numbers* and incidence rates for *Shigella* (invasive and non-invasive) reported to EDRU by age category, 2007.

Age Category (years)	Cases	
	Number	Incidence rate/100000 population
<1	164	15.44
1 - 5	587	11.63
6 - 14	149	1.65
15 - 64	466	1.54
>64	50	2.03
Unknown	75	-
Total	1491	3.12

*Cases may be underreported due to local clinical practices.

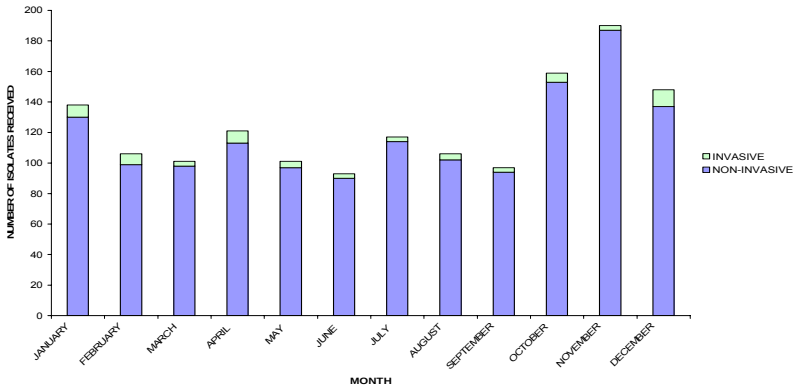

Figure 1. Number of non-invasive and invasive *Shigella* isolates reported to EDRU by month of isolation, 2007, including audit specimens.

(Continued on page 25)

Higher isolation rates in January and from October to December in 2007 suggest seasonality. During this period, a number of water-borne outbreaks were detected, with numerous enteric pathogens implicated.[1] This also resulted in increased numbers of *Shigella* isolates seen during this time period.

Quinolone resistance remains low. Nine of 1227 isolates tested were ESBL-producers. Certain antimicrobials were tested for surveillance purposes only and should not be used for treatment.

Table 3. Results of antimicrobial susceptibility testing for all *Shigell*a isolates (n =1227) received by EDRU, 2007.

Antimicrobial tested	Susceptible (%)	Intermediately resistant (%)	Resistant (%)
Ampicillin	50.7	0.1	49.3
Cotrimoxazole	16.9	0.0	83.1
Chloramphenicol	66.1	0.2	33.8
Nalidixic acid	99.0	0.0	1.0
Ciprofloxacin	100.0	0.0	0.0
Tetracycline	50.9	1.1	47.9
Kanamycin	99.4	0.2	0.4
Streptomycin	43.7	0.0	56.3
Imipenem	100.0	0.0	0.0
Ceftriaxone	99.3	0.0	0.7

Table 4. Commonest* invasive and non-invasive *Shigella* serotypes (n = 1229) reported to EDRU by province, 2007.

	S. dysenteriae type 1	*S. flexneri* type 1b	*S. flexneri* type 2a	*S. flexneri* type 6	*S. sonnei* phase II
Eastern Cape	0	33	62	10	7
Free State	1	14	19	8	0
Gauteng	0	41	113	44	17
Kwazulu-Natal	0	33	34	5	1
Limpopo	0	2	3	4	3
Mpumalanga	0	17	19	13	1
Northern Cape	0	9	22	5	0
North West	0	3	5	5	0
Western Cape	0	67	109	33	6
South Africa	1	219	386	127	35

*Including *Shigella dysenteriae* type 1. Although these isolates are rare the potential for future epidemics remains while these strains are in circulation.

Reference

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DIARRHOEAGENIC *ESCHERICHIA COLI* (DEC)

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[1]Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Table 1. Number of diarrhoeagenic *Escherichia coli* isolates (n = 275) reported to EDRU by province, South Africa, 2007, representing 259 infectious episodes, including those patients which had more than one pathotype (see below). No audits were conducted.

	DAEC	EAggEC	EHEC/ STEC	EIEC	EPEC	ETEC
Eastern Cape	5	9	0	0	18	6
Free State	0	0	0	0	3	0
Gauteng	1	13	0	0	44	2
Kwazulu-Natal	0	0	0	0	0	0
Limpopo	0	0	0	0	2	0
Mpumalanga	53	26	2	11	17	20
Northern Cape	1	4	1	0	4	4
North West	7	4	0	2	0	1
Western Cape	4	1	6	0	4	0
South Africa	71	57	9	13	92	33

DAEC, Diffusely adherent *E. coli*; EAggEC, enteroaggregative *E. coli*; EHEC, STEC, Shiga-toxigenic *E. coli* enterohaemorrhagic *E. coli*; EIEC, enteroinvasive *E. coli*; EPEC, enteropathogenic *E. coli*; ETEC, enterotoxigenic *E. coli*.

Incidence rates have not been calculated as numbers are not viewed as being fully representative. EHEC O26, which has been associated with outbreaks in the past, was identified during the Delmas diarrhoeal outbreak in November 2007.[1-3] Serotypes associated with EPEC included O111, O119, O127 and O55. STEC serotypes were more diverse, but included three STEC O107 from Western Cape; diverse serotypes were also noted for other enterovirulent *E. coli* isolates. Identification of both EHEC and STEC was incidental.[4] The occurrence of serotype O55 is of interest as it has previously been shown that enterohaemorrhagic *E. coli* O157 evolved from this serotype .[5]

The predominance of isolates received in children under the age of one year may reflect culturing practices; infants are more likely to have stools taken for culture due to the devastating effects of diarrhoea in children of this age. Numbers of isolates through the year were greatly affected by the occurrence of diarrhoeal outbreaks,[6] during which a number of patients were identified as having more than one pathogen, including a number of pathotypes of diarrhoeagenic *E. coli*. This is particularly notable in adults in whom typically "paediatric" stains of *E. coli* were identified. Increased numbers of both DAEC and EAggEC were identified during the diarrhoeal outbreaks.

Table 2. Number of diarrhoeagenic *E. coli* isolates (n = 275) reported to EDRU by age category, 2007.

Age category (years)	DAEC	EAggEC	EHEC/ STEC	EIEC	EPEC	ETEC
<1	12	22	0	0	47	8
1 - 5	15	14	1	3	24	12
6 – 14	7	2	0	3	3	2
15 - 65	34	14	2	6	10	8
>65	1	1	0	0	0	0
Unknown	2	4	6	1	8	3
Total	71	57	9	13	92	33

(Continued on page 27)

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ERRATUM

Table: Provisional listing: Number of laboratory-confirmed cases in South Africa of diseases under surveillance reported to the NICD, corresponding periods 1 January - 31 December 2005/2006. Acute Flaccid Paralysis, Measles, Rubella (March 2007;5:1:20). The numbers published in the table for 2006 were incorrect and reflected the period 1 January-30 September 2006 instead of 1 January – 31 December 2006. The correct figures for the period 1 January – 31 December 2006 are published in this edition of the Communicable Diseases Surveillance Bulletin, Table: Provisional listing: Number of laboratory-confirmed cases in South Africa of diseases under surveillance reported to the NICD, corresponding periods 1 January - 31 December 2006/2007 Acute Flaccid Paralysis, Measles, Rubella on page 28.

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Provisional listing: number of laboratory-confirmed cases in South Africa of diseases under surveillance reported to the NICD, corresponding periods 1 January - 31 December 2006/2007

	Disease/ Organism	Case Definition	Subgroup	Cumulative to 31 December, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
VIRAL DISEASES	Acute Flaccid Paralysis	Cases < 15 years of age from whom specimens have been received as part of the Polio Eradication Pro-gramme		2006	68	30	76	51	48	44	30	29	38	414
				2007	41	22	51	37	44	23	10	22	26	276
	Measles	Measles IgM positive cases from suspected measles cases, all ages		2006	4	0	24	5	5	9	5	30	1	83
				2007	6	1	11	3	2	7	0	2	2	34
	Rubella	Rubella IgM positive cases from suspected measles cases , all ages		2006	294	62	616	462	638	285	157	370	122	3006
				2007	295	19	145	240	120	62	36	66	118	1101
	VHF	Laboratory-confirmed cases of CCHF (unless otherwise stated), all ages		2006	0	2	2	0	0	0	4	0	0	8
				2007	0	0	0	0	0	0	1	0	0	1
	Rabies	Laboratory-confirmed human cases, all ages		2006	3	0	0	6	21	0	0	1	0	31
				2007	4	0	0	9	1	0	0	0	0	14
BACTERIAL AND FUNGAL DISEASES	*Haemophilus influenzae*	Invasive disease, all ages	All serotypes	2006	19	17	155	59	1	4	7	4	51	317
				2007	28	23	202	66	6	22	3	4	66	420
		Invasive disease, < 5 years	Serotype b	2006	2	4	22	9	1	1	1	1	8	49
				2007	1	2	25	11	0	2	1	2	14	58
			Serotypes a,c,d,e,f	2006	1	1	16	2	0	0	1	0	3	24
				2007	1	1	18	1	0	0	0	0	6	27
			Non-typeable (unencapsulated)	2006	1	5	40	4	0	1	0	0	7	58
				2007	0	1	34	8	0	2	0	0	4	49
			No isolate available for serotyping	2006	8	3	26	18	0	1	0	1	11	68
				2007	12	7	50	14	2	6	2	0	17	110
	Neisseria meningitidis	Invasive disease, all ages		2006	32	46	356	20	8	26	14	33	69	604
				2007	18	39	254	33	9	26	9	37	77	502
	Streptococcus pneumoniae	Invasive disease, all ages	Total cases	2006	289	227	2094	464	102	209	37	151	487	4060
				2007	354	313	2259	537	148	277	57	221	567	4733
			Penicillin non-susceptible isolates	2006	53	50	584	156	27	73	9	32	124	1108
				2007	93	95	659	228	27	73	18	48	187	1428
			No isolate available for susceptibility testing	2006	130	16	363	44	12	25	3	15	29	637
				2007	135	84	682	115	59	99	16	79	138	1407
		Invasive disease, < 5 years		2006	75	83	606	158	22	58	11	32	153	1198
				2007	125	107	620	201	39	75	20	51	214	1452
	Salmonella spp. (not typhi)	Invasive disease, all ages		2006	47	23	556	118	4	34	0	16	77	875
				2007	77	48	458	105	24	31	13	41	83	880
		Confirmed cases, isolate from a non-sterile site, all ages		2006	112	36	197	197	31	80	15	58	153	879
				2007	195	45	356	109	54	133	23	58	158	1131
	Salmonella typhi	Confirmed cases, isolate from any specimen, all ages		2006	51	1	16	15	6	14	0	0	21	124
				2007	12	1	21	10	6	12	0	2	7	71
	Shigella dysenteriae 1	Confirmed cases, isolate from any specimen		2006	0	0	0	1	0	0	0	0	1	2
				2007	0	1	0	0	0	0	0	0	0	1
	Shigella spp. (Non Sd1)	Confirmed cases, isolate from any specimen, all ages	All serotypes	2006	121	52	228	195	20	39	32	17	407	1111
				2007	206	84	458	130	27	133	52	41	358	1489
	Vibrio cholerae O1	Confirmed cases, isolate from any specimen, all ages	All serotypes	2006	0	0	0	0	0	0	0	0	0	0
				2007	0	0	0	0	0	0	0	0	0	0
	Cryptococcus (*Cryptococcus spp.*)	Invasive disease, all ages	Total cases (incl. *C neoformans*)	2006	1357	287	1856	1348	215	439	63	378	353	6296
				2007	709	506	1919	1265	333	550	48	567	412	6309
			C. gattii	2006	3	4	42	9	25	27	3	14	8	135
				2007	0	2	28	11	14	19	0	8	4	86

Abbreviations: VHF - Viral Haemorrhagic Fever; CCHF - Crimean-Congo Haemorrhagic Fever

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

0 = no cases reported