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COMMUNICABLE DISEASES SURVEILLANCE BULLETIN

MARCH 2007

NICD NHLS

FOREWARD

This edition of the Communicable Diseases Surveillance Bulletin includes a summary of key surveillance findings for South Africa in 2006 from the National Institute for Communicable Diseases (NICD). Since 2005 the NICD has published a review of the key findings of surveillance activities for the previous year in the first edition of the Bulletin of that year.[1]

Many of the surveillance activities of the NICD focus on estimating the burden of laboratory-confirmed disease. Laboratory-confirmed cases are highly specific for the disease of interest but of necessity represent a minimum estimate of disease burden. Numbers presented may differ from other sources of surveillance data which may include clinical cases which have not been confirmed in the laboratory.

It is hoped that these data may be of use to interested parties both nationally and internationally and contribute to the goal of decreasing the burden of communicable diseases in South Africa.

References

1. Communicable Diseases Surveillance Bulletin. 2006 March; 4(1).http://www.wits.ac.za/NICD/pubs/survbull/2006/CommDisBullMarch06.pdf

Cheryl Cohen
Editor

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SUSPECTED MEASLES CASE-BASED SURVEILLANCE, SOUTH AFRICA, 2006

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The National Institute for Communicable Diseases (NICD) is accredited by WHO to perform measles and rubella IgM testing for national case-based surveillance and to provide genotypic analysis on positive cases. Blood and urine specimens from suspected measles cases nationally are submitted to NICD for confirmation. 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.

During 2006 the NICD tested 6620 blood specimens from cases of rash and fever for suspected measles case-based surveillance. The largest number, 1545 (23.3%), were from Gauteng, followed by 1219 (18.4%) from KwaZulu-Natal. Of these specimens 86 (1.3%) were positive for measles IgM antibodies, and 2948 (44.53%) for rubella IgM antibodies.

1. Measles

The 86 positive measles results were from 82 patients, the majority (29) of whom were from the North West Province (NWP), followed by 24 from Gauteng (Figure 1). Ages of patients with positive measles results ranged from 4 months to 41 years (median 5 years). 25 of the 29 cases reported from NWP were clustered in the Central District (Mafikeng area) and constituted an outbreak which lasted from 31 July to 13 November. A single strain of virus was

(Continued on page 2)

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identified (genotype D4). This same strain was circulating in Botswana, Zimbabwe and Zambia in 2005/6 and it is thus likely to have been introduced into South Africa from one of these countries.

Of the 82 measles IgM-positive cases, 61 specimens (serum or paired urine if available) were tested for the presence of measles virus genome. Only 15 (24.6%) were PCR-positive. Fourteen of these were cases from the North West Province measles outbreak described above. The other PCR-positive measles specimen was from an isolated case in the Gauteng at the end of August. The virus (genotype B2) was very closely related to the strain circulating in the Democratic Republic of Congo (DRC) and Angola.

Of the sera that had equivocal measles IgM serology (120), 62 were tested for measles virus genome. Only 3 were PCR-positive: 2, from Gauteng and Limpopo province, were identified as genotype B2 (identical to the strain circulating in the DRC and Angola). The other case, from Gauteng, was also shown to have an imported measles virus, genotype B3. This particular strain of virus is circulating widely in Africa (Nigeria, Burkina Faso, Cote d'Ivoire, Kenya, Uganda, Tanzania, Zambia) and was also introduced into Europe, Canada and the USA in 2006.

2. Rubella

There were 2930 patients with positive rubella IgM results in 2006. This is the highest annual number of rubella cases confirmed at NICD since the initiation of case-based surveillance in 1998 (figure 2). Patient ages ranged from 1 month to 56 years with a median of 7 years. 163 (5.19%) of the rubella IgM positive results were from women of childbearing age (12-55 years).

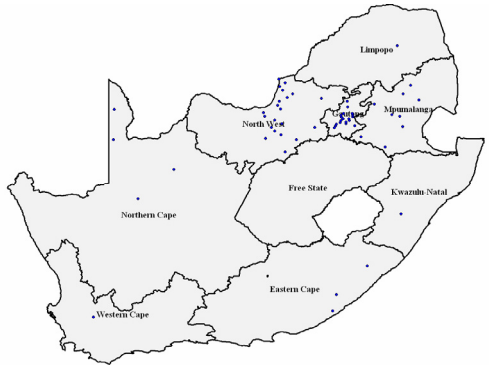

Figure 1: Geographic distribution of measles IgM positive cases confirmed at NICD, South Africa, 2006

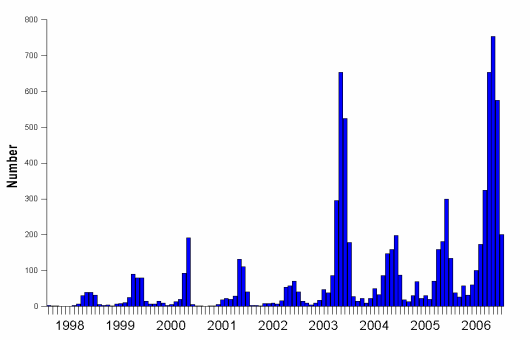

Figure 2: Number of rubella IgM positive cases confirmed at NICD by month, 1998-2006, South Africa

Acknowledgements: Special thanks to Elias Kekana, Cardia Esterhuyse, Nathi Ndlovu, Lynn Harvey and Theresa Mashaba for their hard work and dedication.

ACUTE FLACCID PARALYSIS (AFP) SURVEILLANCE, 2006

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1. National Polio Isolation Laboratory

The National Institute for Communicable Diseases (NICD) serves as national polio isolation laboratory for South Africa as well as Angola, Botswana, Lesotho, Mozambique, Namibia, and Swaziland. During the year 2263 stool specimens were received from patients with acute flaccid paralysis (AFP) from these seven countries. Of these 68 were from patients with onset of paralysis prior to 2006. Of the remainder 806 were from 401 South African cases, and 1457 from the six other countries served by the NICD.

South Africa (only patients from whom specimens were received included)

Case detection rate ranged from 1.48 to 9.33/100 000 population (mean 2.71) (figure 1). Of the 401 South African cases with onset of paralysis in 2006, one specimen only was received from 60 cases, and two or more specimens from 341. The date of onset of paralysis was known for 329 cases. Two specimens taken at least 24 hours apart and within 14 days of onset were received from 260/401 (64.84%) cases. Non-polio enteroviruses were isolated from 134 of the 806 specimens (non-polio isolation rate 16.63%, expected rate10%), and poliovirus, identified as Sabin type poliovirus from 8 specimens of five patients.

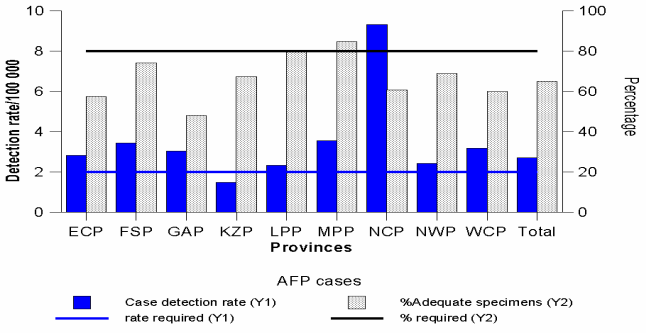

Figure 1: AFP case detection and stool adequacy rate, South Africa, 2006 (only patients from whom specimens were received included)

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Other Southern African countries

Of the 1457 specimens received from the six southern block countries served by the NICD, 32 were from patients with onset of paralysis prior to 2006. Two adequate stool specimens were received from 588 (79.46%) of the 740 patients with onset of paralysis in 2006 (range per country 66.67% to 93.81%). Non-polio enteroviruses were isolated from 151/2345 specimens with a non-polio enterovirus isolation rate of 6.44% (range per country 0 to 21.61%). Poliovirus was isolated from 78 specimens, 32 of which were identified as wild type polio 1, and the remainder as Sabin strains. The wild type isolates were from one patient in Angola, and 19 in Namibia.

2. Polio Molecular Unit

The Polio Molecular Unit of the NICD is a World Health Organisation Regional Reference Unit for polio. The Unit receives poliovirus isolates from South Africa and 35 other African countries. Isolates are characterized as vaccine or wild type using two intratypic differentiation methods, PCR and ELISA and are sequenced in order to answer epidemiological questions regarding the likely location of endemic poliovirus reservoirs and patterns of virus transmission.

Of the 731 wild-type polioviruses identified in 2006, 577 were from Nigeria, of these, 349 were polio type 1 and 228 were polio type 3. Other wild type polioviruses identified in 2006 were from Angola, Chad, Ethiopia Kenya, Namibia, Niger, Republic Democratic of Congo (DRC) and Somalia.

Wild polio virus type 1 (PV1) was highly endemic in northern Nigeria in 2006 with additional circulation occurring in the central provinces. Immunization coverage is much higher in the southern provinces of Nigeria. Nationwide Immunization Plus Days (IPDs) will be held in January 2007, using a combination of monovalent oral polio vaccine type 1 (mOPV) and trivalent oral polio vaccine (OPV).

2006 PV1 wild type isolates are distributed into three genotypes, South East Asian (SOAS), West African B (WEAF-B) and East African (EAAF). The WEAF-B genotype consists of viruses from Nigeria, Niger, and Chad. The EAAF genotype consists of the viruses from Ethiopia, Kenya and Somalia. SOAS genotype is an Indian genotype with strains from Angola, Namibia and DRC.

Following the SOAS outbreak in Angola in 2005, two countries were also affected by the same strain in Africa in 2006 namely: Namibia and Democratic Republic of Congo (DRC). The index case for Namibia was a 39 year old man from the Hardap region, south-east of the capital Windhoek, who had the onset of AFP on 8 May 2006. Nineteen cases of PV1 wild-type viruses and a contact were identified from Namibia all of which belong to the SOAS genotype (Figure 2). Six of the confirmed cases died. In addition to the case from the Hardap region, the wild-type confirmed cases were reported from two main regions, which are the most populated areas in the country: 1) Windhoek in the Khomas region and 2) a northern area bordering Angola with three adjacent regions namely Omusati, Oshana and Ohangwena (figure 3). The genetic analysis of the VP1 region showed that the virus was imported from Angola. The sequence of the index case showed a 97.46% identity to the case from Angola.

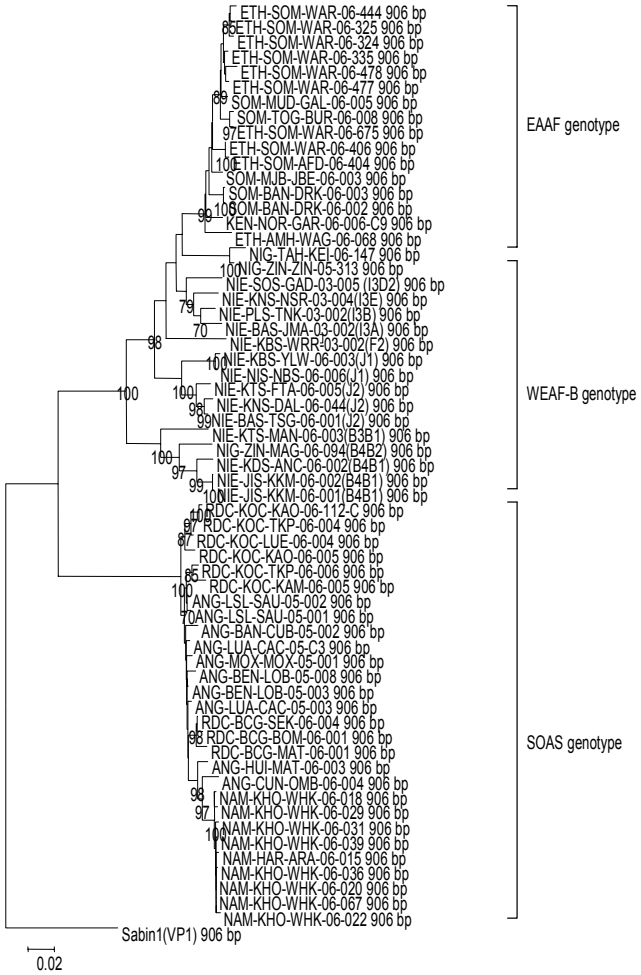

Figure 2: Neighbour-joining tree of the VP1 gene of WEAF-B wild PV1 representative of isolates of 2003-2006 from Africa. Bootstrap values of greater than 70% are shown at the branch nodes. Sabin type 1 was used as an out-group.

Wild PV1 was isolated from an Angolan case with onset of paralysis on 27 June 2006. In early January 2007 two stool specimens from a patient from Angola, with onset of paralysis on 14 November 2006, also yielded wild type polio 1 (figure 3).

WEAF-B wild PV3 is divided into four clusters A – D (Figure 4). Cluster A represents local circulation in Kebbi (KBS) Kano (KNS), Katsina (KTS), Jigawa (JIS), Zamfara (ZAS) and Kaduna (KDS) Northern provinces of Nigeria. Cluster B is not represented. Cluster D and cluster C are resolved into two countries, Nigeria and Niger. Two other cases of wild-type 3 were identified in Cameroon and Chad (data not shown).

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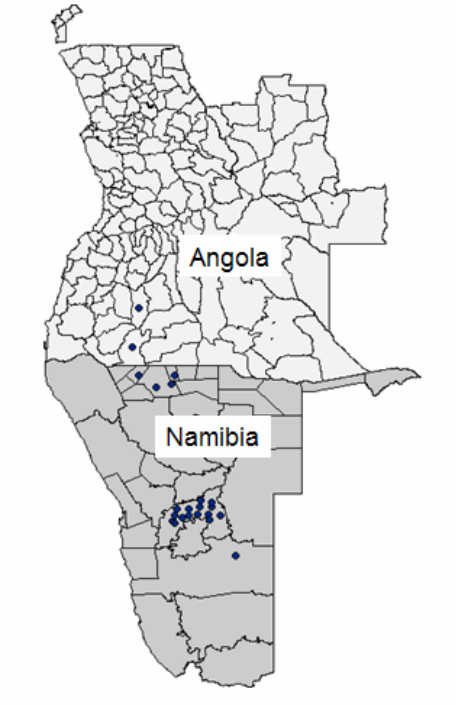

Figure 3: Distribution of laboratory-confirmed wild type polio cases by district, Angola and Namibia 2006

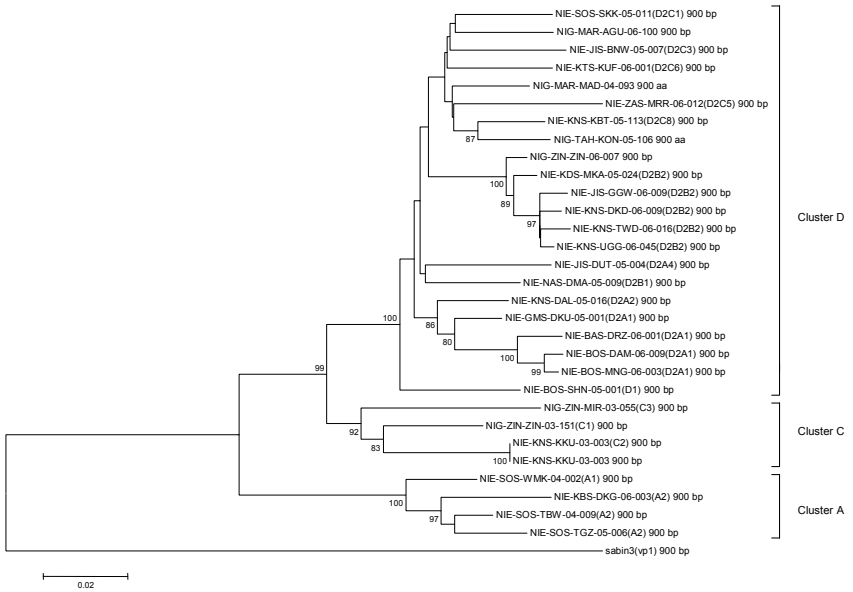

Figure 4: Neighbour-joining tree of the VP1 gene of WEAF-B wild PV3 representative of isolates of 2003-2006 from Africa. Bootstrap values of greater than 70% are shown at the branch nodes. Sabin type 3 was used as an out-group.

Acknowledgements: Special thanks to Olivia Lentsoane, Mashudu Rampilo, Mbavhalelo Denga, Peter Coetzee and Busisiwe Guliwe from molecular laboratories and Portia Ngcobondwana, Elliot Motaung, Doris Lebambo, Megan Vandecar, Cynthia Simelane and Abraham Sehata from the diagnostic laboratory.

RESPIRATORY VIRUS SURVEILLANCE, SOUTH AFRICA, 2006

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1. Viral Watch Surveillance Programme

During 2006 a total of 1449 specimens were received for detection of respiratory virus. Of these 1247 (86.1%) were received from the Viral Watch programme, started in 1984 and expanded substantially in 2005, designed to monitor timing of influenza activity and determine prevalent influenza strains. During 2006 the programme was rolled out in the Eastern Cape, Western Cape, and KwaZulu-Natal, adding a further 42 practitioners.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 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.

The first influenza isolate of the season was made from a specimen collected on 27 March, and the last from a specimen collected on 25 October (figure 1). A total of 554 influenza isolates were made, of which 540 (97.5%) were from the Viral Watch. The isolates were further identified as 496 influenza A, of which A H3N2 (A/Wisconsin/67/05-like) accounted for the majority, and 58 influenza B, mainly B/Malaysia/2506/04-like. A further 47 respiratory isolates were made during the year including 23 respiratory syncytial virus, 13 parainfluenza virus (3 type 1, 4 type 2, 5 type 3, 1 untyped), and 2 adenovirus.

2. Isolation, antigenic and genetic characterization

The influenza isolates were subtyped by the haemagglutination inhibition (HI) test using the kit supplied by the WHO Collaborating Centre (WHO CC) for Reference and Research on Influenza, Melbourne, and a proportion of them were characterised by sequencing the HA1 subunit of

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the haemagglutinin (HA) gene and performing phylogenetic analysis.

The majority of the influenza A isolates were identified as subtype H3N2 by HI. A further six were shown to be subtype H1N1 and the remaining 65 isolates could not be typed by HI, most likely due to low viral titres. Most of the influenza B isolates were B/Malaysia/2506/04 – like, belonging to the B/Victoria/2/87 lineage. Seven viruses belonging to the B/Yamagata/16/88 lineage (B/Shanghai/361/02-like viruses) were also identified.

Sequence analysis of the HA1 subunit revealed the H1N1 viruses isolated during the season showed very little genetic drift from the A/New Caledonia/20/99 vaccine strain. Amino acid changes were observed in the isolates sequenced at five residues. These South African isolates were very similar to viruses isolated in countries such as Egypt, Israel and Sweden.

The molecular characterisation of representative influenza H3N2 isolates revealed that the viruses circulating in South Africa exhibited extensive genetic drift relative to the A/California/7/04 vaccine strain. These changes mapped to antigenic sites A and B. The isolates shared a greater homology with the A/Wisconsin/67/05 strain with the characteristic S193F (antigenic site B) and D225N (receptor binding site) mutations seen in the A/Wisconsin/67/05-like viruses. All the isolates differed from the A/Wisconsin/67/05 strain at residues 122 (antigenic site A), 195 and 223 and sporadic mutations were seen at several other residues in antigenic site B.

Phylogenetic analysis of the HA1 subunit of representative South African 2006 influenza B viruses from both the B/Victoria/2/87 and B/Yamagata/16/88 genetic lineages showed that the 2006 B/Victoria/2/87-like isolates were closely homologous to the B/Malaysia/2506/04 vaccine strain and shared only one common amino acid change at position 109. In the B/Shanghai-like viruses, substitutions were seen at four common residues and sporadic changes were seen at several additional other residues.

The South African data was presented together with that obtained from viruses circulating mainly in Australia and New Zealand at the "WHO Consultation on the composition of the Influenza Vaccine for the Southern Hemisphere, 2007" in Geneva in September 2006. The decision was made to update the vaccine to contain the following strains:
A/New Caledonia/20/99 –like virus (H1N1)
A/Wisconsin/67/05 –like virus (H3N2)
B/Malaysia/2506/04-like virus.

3. Respiratory admissions data mining surveillance system

In 2006 a new surveillance system was established aiming to determine trends in hospital admissions for respiratory illness and examine the association between timing of admissions for respiratory and other diagnoses of interest and influenza isolations as determined through the viral watch surveillance system. The system utilizes data on numbers of respiratory and other admissions (classified according to the ICD-10 coding system) extracted from a national private hospital database. The timing of the peaks of admissions for pneumonia, acute upper respiratory infection, influenza and acute lower respiratory infection corresponded with peaks in influenza virus isolations in 2005 and 2006 (figure 2).

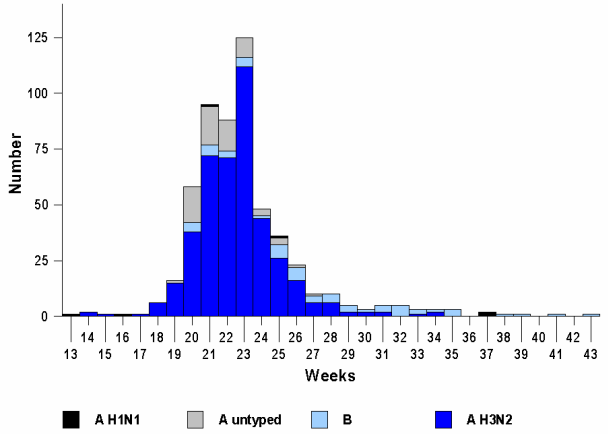

Figure 1: Number of influenza isolates by virus type and epidemiologic week 2006

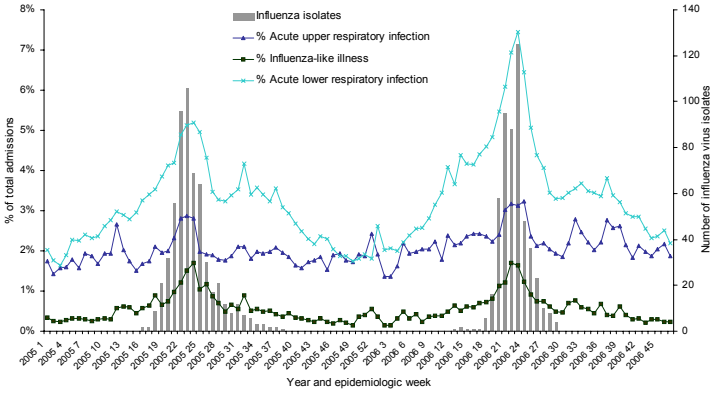

Figure 2: Percentage of admissions due to respiratory diagnoses of interest and influenza isolations by epidemiologic week, 2005-2006

Acknowledgements: Special thanks to Lynn Harvey, Teresa Mashaba, Megan Vandecar and Lorraine Cranston in the viral diagnostics team for their hard work and dedication, and to the Netcare Hospital Group.

VIRAL HAEMORRHAGIC FEVERS, SOUTH AFRICA, 2006

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Eight cases of Crimean-Congo haemorrhagic fever (CCHF) were confirmed in South Africa during 2006 (Table 1), most of them resulted from bites by infected ticks and 50% were fatal. Although there is no specific treatment for CCHF infections there is some evidence that Ribavirin can improve the prognosis if administered before day 5 after onset of illness.

A total of 186 cases of CCHF have been diagnosed in southern Africa from the time that the presence of the disease was first recognized in 1981 up until the end of 2006, including seventeen in Namibia, one in DRC, one in Tanzania, and 167 cases in South Africa. The largest group of cases, 85/186 (45,7%), arose from known tick bite or the squashing of ticks; 72/186 (38.7%), arose from known or potential contact with fresh blood or other tissues of livestock and/or ticks; 7/186 (3.8%) nosocomial infections arose from contact with blood or fomites of known CCHF patients, while in 21/186 (11.3%) 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 156/186 (83.9%) 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 southern Africa, but gradually declined to an overall rate of 19.9% (29/146) for a period of 1981-1998, most likely as a result of increased awareness leading to earlier recognition and institution of appropriate therapy. However, the case fatality rate drastically increased to 58.9% (23/39) for a period of 1999-2006 which suggests that there is a decline in awareness of the disease among clinicians, resulting in delayed recognition of cases.

One case, confirmed in December 2006 is worth mentioning with respect to the differential diagnosis of CCHF. The patient who lived in Prieska, (Northern Cape) clinically presented with fever, headache and myalgia two days after exposure to a *Hyalomma* tick. While the thrombocytopenia and raised hepatic transaminases were typical of CCHF, the white blood cell count and the peripheral blood smear was highly suggestive of leukemia. Indeed, the patient was subsequently confirmed as having a chronic myeloid leukemia (CML). The patient had previously been well and CML had not been suspected but testing for CCHF was done since the patient lived in CCHF endemic area, and developed a febrile illness 2 days after a tick bite. CCHF was confirmed by RT-PCR, serology and virus isolation. This is the first report of a patient with both CML and CCHF and highlights some of the difficulties as regards differential diagnosis of infectious diseases and clearly emphasis the need to exclude CCHF in any cases with a suggestive epidemiological and clinical history which is important for case management and protection of medical staff.

Table 1: Confirmed cases of Crimean-Congo haemorrhagic fever virus infection, South Africa, 2006.

Location of exposure	Month	Age/Sex	Virus Isolation*	PCR*	Antibody*	Died/ Survived	Source of infection
Bloemfontein, Free State	Jan	51 F	Pos.	Pos.	Neg.	Died	Tick bite
Upington, Northern Cape	Feb	61 M	Pos.	Not done	Pos.	Survived	Tick bite
Boshof, Northern Cape	Feb/March	50 M	Neg.	Pos.	Pos.	Survived	Livestock
Vereeniging, Gauteng	March	33 M	Pos.	Pos.	Neg.	Died	Tick bite
Vereeniging, Gauteng	April	25 M	Pos.	Pos.	Pos.	Died	Unknown
Hopetown, Free State	March	33 M	Pos.	Pos.	Pos.	Survived	Goats
Prieska, Northern Cape	Dec	35 M	Pos.	Pos.	Pos.	Died	Tick bite
Keimoes, Northern Cape	Dec	25 M	Pos.	Pos.	Pos.	Survived	Tick bite

*Pos. - Positive, Neg. - Negative

RABIES IN SOUTH AFRICA, 2006

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A total of 31 cases of human rabies were confirmed by the SPU during 2006 (Table 1). The number of cases confirmed was higher compared to those in 2005 (8 cases) which was due to rabies outbreak in Limpopo Province (LP). The majority of patients contracted rabies from contact with rabid dogs in LPP (21 cases), KwaZulu-Natal (KZ – 6 cases) or the Eastern Cape Province (EC – 3 cases). In one case rabies-related virus (Duvenhage virus - DUVV) was isolated from a 77-year old man who was scratched on the face by what appears to have been an insectivorous bat in February 2006 in North West Province (NW), about 80 km from the location where the first DUVV infection occurred 36 years earlier.

Table 1: Confirmed cases of human rabies, South Africa, 2006

Name	Age (yrs)/ Sex*	Location of exposure	Exposure (animal and date)	Date of onset	Date of ad-mission	Date of death	Final hospital
RK	7/f	Tshandama, LP	Dog Oct 05	2005/12/29	2006/01/02	2006/01/06	Donald Fraser
NW	10/f	Marindili, LP	Dog	2006/01/26	2006/01/30	2006/02/11	Polokwane
IN	9/m	Thavhani, LP	Dog Dec 05	2006/02/07	2006/02/07	2006/02/09	Tshilidzini
SM	5/f	Ha-Budeli, LP	Dog	2006/02/06	2006/02/12	2006/02/15	Tshilidzini
AS	7/m	Budeli Nwiini, LP	Dog Oct 05		2006/02/11	2006/02/17	Tshilidzini
NM	3/m	Scottburgh, KZ	Dog Jan 06			2006/03/03	GJ Crookes
NN	4/m	Mufulwi, LP	Dog Jan 06		2006/03/10	2006/03/13	Donald Fraser
NR	5	Vhutalu, LP	Dog Feb 06		2006/03/17	2006/03/18	Donald Fraser
AM	6/m	LP	Dog Feb 06	2006/03/07	2006/03/15	2006/04/01	Tshilidzini
AK	77/m	Sun City, NW	Bat Feb 06	2006/03/23	2006/03/25	2006/04/05	Durbanville Clinic
TM	11/m	Ha- Mavhunda, LP	Dog Sept 05		2006/03/31	2006/04/02	Tshilidzini
PN	9/m	Dzimauli, LP	Dog Mar 06	2006/04/04	2006/04/04	2006/04/12	Donald Fraser
TT	11/f	Pile Modale, LP	Dog Feb 06		2006/04/14	2006/04/15	Donald Fraser
AN	27/f	Giyani, LP	Dog Apr 06	2006/04/18	2006/04/21	2006/04/25	Tshilidzini
TB	12/f	Siloam Nzhelele, LP	Dog Dec 05	2006/04/30	2006/05/02	2006/05/09	Polokwane
MM	11/f	Tshino, LP	Dog Feb 06	2006/04/29	2006/05/02	2006/05/05	Tshilidzini
ZR	52/f	Hibberdene, KZ	Dogs Jan 06	2006/05/11	2006/05/11	2006/05/13	Port Shepstone
LS	18/f	Vhurivhuri, LP	Dog 06	2006/05/19	2006/05/19	2006/05/23	Donald Fraser
NRF		Umzimkulu, EC		2006/05/06			
KM	11/f	Tshilidzini, LP	Dog Mar 06	2006/07/02	2006/07/04	2007/07/08	Tshilidzini
CS		Gcilima, KZ	Dog June 06		2006/07/05	2006/07/07	Port Shepstone
LM	Ad/m	Idutywa, EC		2006/06/24	2006/07/05	2006/07/15	Cecilia Makiwane
MM	9/f	Mukovhawabale, LP	Dog May 06		2006/07/26	2006/08/01	Donald Fraser
MM	4/m	Maniini, LP	Dog Sept 06	2006/09/21	2006/09/21	2006/09/25	Tshilidzini
LM	8/m	Umtata, EC	Dog July 06		2007/09/26	2006/09/28	Nelson Mandela Academic
MK	11/m	Ha- Mutsha, LP	Dog Sept 06	2006/10/05	2006/10/08	2006/10/13	Tshilidzini
RB	3/f	Polokwane Hosp.,LP	Dog Aug 06	2006/10/09	2006/10/12	2006/10/17	Mankweng
LM	8/m	Paddock, KZ	Dog Sept 06		2006/10/28	2006/11/03	Inkosi Albert luthuli
PM	50/m	Hibiscus, KZ	Dog Oct 06		2006/11/29	2006/11/29	Port Shepstone
BS	70/f	Dweshula, KZ	Dog July 06		2006/12/22	2006/12/23	Port Shepstone
HN	32/m	Duthuni, LP	Dog Feb 06		2006/12/21	2007/01/03	Tshilidzini

*f – female, m- male, Ad - adult
Provinces of South Africa - EC: Eastern Cape, FS: Free State, GA: Gauteng, KZ: KwaZulu-Natal, LP: Limpopo, MP: Mpumalange, NC: Northern Cape, NW: North West, WC: Western Cape

SEXUALLY TRANSMITTED INFECTIONS SURVEILLANCE, SOUTH AFRICA, 2006

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During 2006, the Sexually Transmitted Infections (STI) Reference Centre undertook both clinical and microbiological STI surveillance within South Africa and, additionally, assisted other countries in the Southern African Region with their own STI surveillance projects.

1. National Clinical Surveillance for STIs

The STI Reference Centre continued to support the South African National Department of Health (NDoH) with the national STI clinical surveillance programme, funded through the NICD:CDC co-operative agreement. The final draft of the national clinical surveillance report (April 2004 – March 2005) was completed and sent to the NDoH in July 2006 for approval and further dissemination.

2. Clinical Surveillance of STIs in Gauteng

The Gauteng Clinical STI Surveillance Programme is now in its 12th year and remains highly valued by the Provincial Department of Health in Gauteng, the STI Reference Centre's collaborative partner in this initiative. Throughout the year, data were received from 21 sentinel sites, and quarterly reports and an annual report for 2005 were produced. This programme remains the only clinical surveillance programme that produces timely and high quality data on STI syndrome caseload in South Africa and prides itself on rapid dissemination of information to Gauteng Province.

The trend in STI episodes presenting to the 21 sentinel primary healthcare facilities in Gauteng suggests a decline in STIs (Figure 1). Johannesburg and Ekhuruleni continued to see the largest numbers of STI patients (Figure 2). The relative prevalence of the main STI syndromes in women (Figure 3) and men (Figure 4) remained relatively constant over the six years from 2000 to 2005.

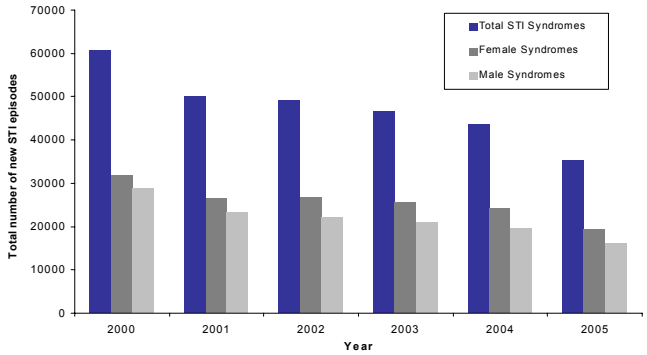

Figure 1. Gauteng Surveillance Programme: Total New STI Episodes in both males and females

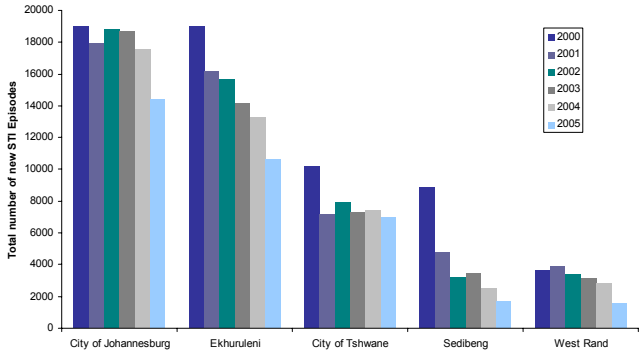

Figure 2. Gauteng Surveillance Programme: Total New STI Episodes by Year and Region

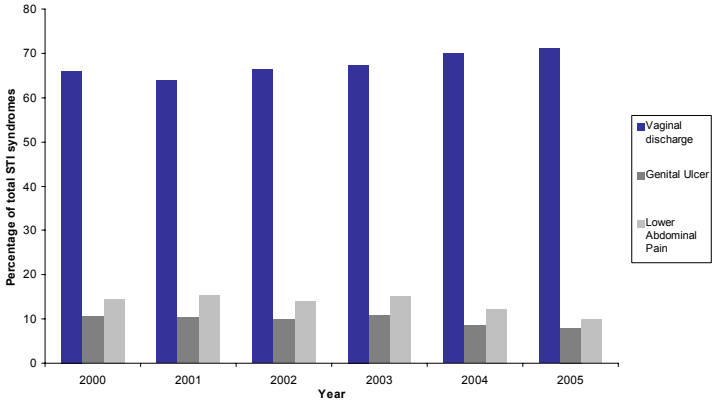

Figure 3. Gauteng Surveillance Programme: Leading STI syndromes in females

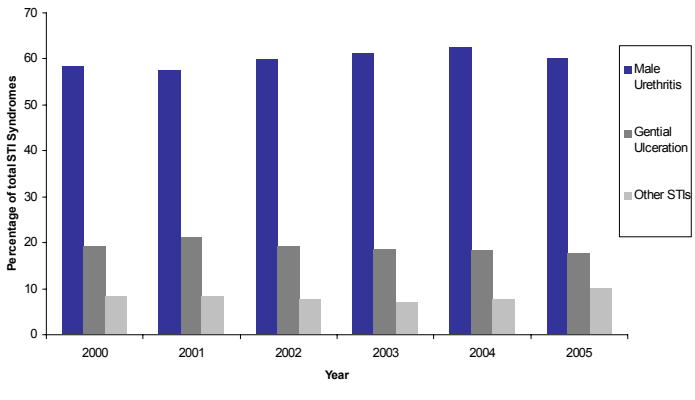

Figure 4. Gauteng Surveillance Programme: Leading STI syndromes in men

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3. Microbiological Surveillance for STIs

During 2006, the STI Reference Centre continued to co-ordinate the national microbiological surveillance of sexually transmitted infections in South Africa, again supported through PEPFAR funding via the NICD:CDC co-operative agreement. The programme aims to monitor the microbial aetiology of the main STI syndromes (male urethritis, vaginal discharge and genital ulcer disease) and their local or regional epidemiology. Antimicrobial susceptibility patterns of *Neisseria gonorrhoeae* are being investigated.

In the early part of 2006, microbiological STI surveillance was undertaken in the Northern Cape (Kimberley) by the STI Reference Centre. The CDC funds were also used to fund antimicrobial surveillance of gonococci in Kwa-Zulu Natal (Durban) and Mpumalanga with the support of the Microbiology Department, Nelson Mandela School of Medicine, University of KwaZulu Natal who undertook the testing of the specimens. In the last quarter of 2006, the STI Reference Centre employed two staff in the Western Cape and undertook both aetiological and antimicrobial resistance surveillance in Cape Town at Salt River Clinic. The work was undertaken in collaboration with the Microbiology Department of Tygerburg Hospital, University of Stellenbosch.

During 2006, the prevalence of ciprofloxacin resistance gonococci continued to increase (Figure 5) and a key meeting was held at the NDoH in October 2006 to further raise the issue of the failure of current first-line syndromic management protocols to cover gonococcal infections within the country. It is clear from current data that ciprofloxacin can no longer be relied upon to reliably cure gonorrhoea and its complications. The World Health Organisation advises that first-line therapy for gonococcal infection should be changed when clinical efficacy falls below 95%. The only alternative in South Africa at the present time is to use Ceftriaxone 250 mg i.m. as a single dose. The STI Reference Centre continues to argue the case for local manufacture or importation of Cefixime 400mg, which treats uncomplicated gonorrhoea as a single oral dose. Cefixime has many advantages to Ceftriaxone in terms of route of administration, nursing time and a lack of needlestick injury risk.

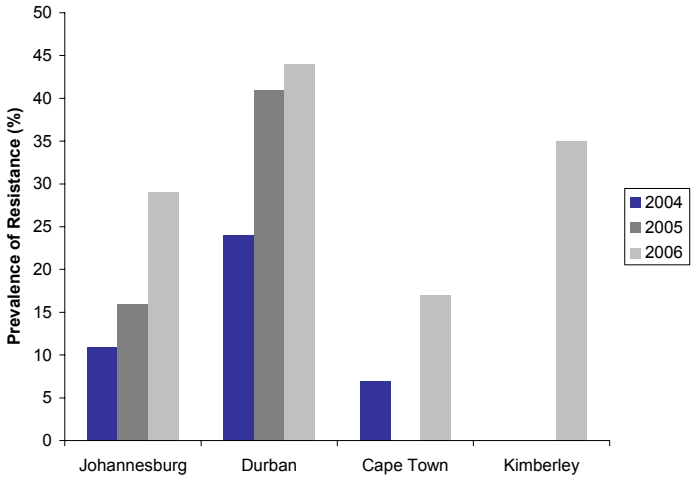

Figure 5: Ciprofloxacin resistance surveillance data for gonococci isolated from Johannesburg, Durban, Cape Town and Kimberley [2004 and 2005 Durban data were presented at the Sun City FIDSSA meeting, July 2005 and the 2006 Durban data were obtained with the financial support of PEPFAR funding administered through the NICD. The STI Reference Centre acknowledge the sharing of these data in NICD communiqués during 2006 by Professors Sturm and Moodley of the Microbiology Department, Nelson Mandela School of Medicine, University of KwaZulu Natal]

ANTHRAX IN SOUTH AFRICA, 2006

John Frean[1], Gill de Jong[2], Lucille Blumberg[2]
[1]Special Bacterial Pathogens Unit and [2]Epidemiology Division, NICD

A human case of probable cutaneous anthrax occurred in Kuboes, Namakwa District, in the Northern Cape Province in December, secondary to an outbreak in sheep and goats. About 20 human exposures to infected carcasses were reported. Near Ermelo, Mpumalanga Province, 70 people received antibiotic prophylaxis after exposure to 7 dead sheep on a farm in October. No human cases were reported. Animal anthrax is endemic in southern Africa and there have been recent outbreaks in wild and domestic animals in South Africa, Zimbabwe, Botswana and Namibia. The most common clinical presentation of anthrax is the cutaneous form, usually acquired via handling meat from dead livestock, accounting for more than 95% of cases. Cutaneous anthrax is usually a self-limited disease but antibiotics are used to prevent systemic invasion. Gastrointestinal anthrax is rare, even when infected meat is cooked and eaten; outside bioterrorism/biowarfare scenarios, inhalational anthrax is exclusively an industrial disease. Antibiotic prophylaxis for longer than 10 days is therefore not required under usual circumstances. Control of anthrax includes animal vaccination, proper disposal of carcasses and community education to discourage contact with animals that may have died of anthrax, particularly consumption of their meat.

GERMS-SA SURVEILLANCE REPORT, 2006

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 March 2006.

The pathogen-specific reports that follow provide results emanating from the surveillance programme for 2006. Incidence rates were calculated using mid-year population estimates for each year supplied by Statistics South Africa (Stats SA).

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1. GERMS-SA: A national South African surveillance network for bacterial and fungal diseases. Govender N, Quan V, Prentice E, von Gottberg A, Keddy K and McCarthy K for GERMS-SA. Communicable Diseases Surveillance Bulletin. May 2006. National Institute for Communicable Diseases, of the National Health Laboratory Service. Johannesburg. South Africa.

RESPIRATORY AND MENINGITIS PATHOGENS SURVEILLANCE, SOUTH AFRICA, 2006

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[1]Respiratory and Meningeal Pathogens Reference Unit, National Institute for Communicable Diseases

Meningococcal Disease in 2006

In 2006, 591 cases of meningococcal disease were reported to RMPRU. Rates of disease remained stable in Gauteng and Western Cape provinces, but Eastern Cape, Free State, Mpumalanga, Northern Cape and North West all reported more cases than the previous year (2005) (Table 1). In keeping with the seasonal pattern of disease, the number of cases reported increased during the winter and spring months (Figure 1).[1] Of all cases reported to us, cerebrospinal fluid (CSF) was the most common specimen yielding meningococci (Table 2).

The burden of serogroup W135 disease in Gauteng Province stabilised in 2006, with total rates of disease similar to those of last year (approximately 4/100,000), and most of that disease being due to W135 (257/314, 82%) (Table 3). Cases of W135 disease were reported from all provinces. The preponderance of serogroup B disease in Western Cape Province was still noted: 26/49 (53%) 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 26/197 (13%). This rate was similar compared to last year (42/216, 19%; p=0.09).

Only 18/467 (4%) 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 1: Number of cases and incidence rates of meningococcal disease as reported to RMPRU by province, South Africa, 2005 and 2006

Province	2005		2006	
	n	Cases/ 100,000	n	Cases/ 100,000
Eastern Cape	10	0.14	22	0.31
Free State	25	0.85	45	1.52
Gauteng	359	3.98	360	3.91
KwaZulu-Natal	25	0.26	20	0.21
Limpopo	12	0.21	8	0.14
Mpumalanga	21	0.65	27	0.83
Northern Cape	7	0.78	14	1.54
North West	15	0.39	26	0.68
Western Cape	70	1.51	69	1.45
South Africa	544	1.16	591	1.25

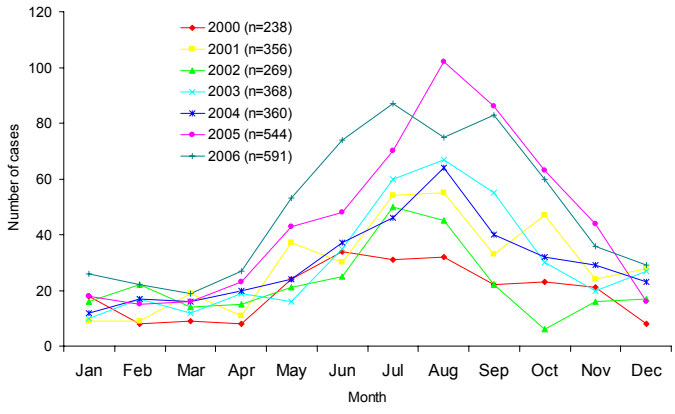

Figure 1: Number of cases of meningococcal disease in South Africa as reported to RMPRU by month and year (2000-2006)

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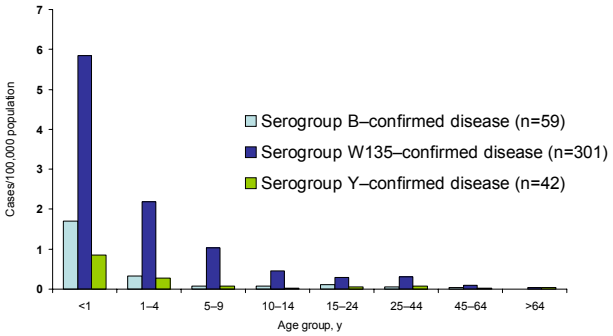

Figure 2: Reported age-specific incidence rates for confirmed serogroups B, W135 and Y, South Africa, 2006 (of 591 cases reported, 556 had known age, and 474 had viable isolates available for serogrouping)

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

Site of specimen	n	%
CSF	436	74
Blood	152	26
Other	3	0.5
Total	591	

Table 3: Number of cases of meningococcal disease reported to RMPRU by serogroup and province (n=591, 474 (80%) with isolates for further testing), South Africa, 2006

Province	No isolate available	Serogroup A	B	C	W135	X	Y	Non-groupable	Total
Eastern Cape	2	0	7	3	5	0	5	0	22
Free State	8	0	5	3	16	1	12	0	45
Gauteng	46	3	20	17	257	0	17	0	360
KwaZulu-Natal	13	0	0	2	3	0	2	0	20
Limpopo	5	0	0	1	2	0	0	0	8
Mpumalanga	6	1	3	1	15	0	1	0	27
Northern Cape	6	0	1	1	4	0	2	0	14
North West	11	0	1	3	10	0	1	0	26
Western Cape	20	0	26	10	7	0	5	1	69
Total	117	4	63	41	319	1	45	1	591

Haemophilus influenzae disease in 2006

The total number of cases of *Haemophilus influenzae* invasive disease reported in 2006 to our unit was 300. Of these 207 (69%) had viable isolates for further testing and 71/207 (34%) were confirmed as serotype b (Table 4). Serotype b isolates were more likely to be isolated from CSF than non-typeable *H. influenzae* (34/71 vs. 7/102, p<0.001) (Table 5).

Table 4: Number of cases of *Haemophilus influenzae* disease reported to RMPRU by serotype and province (n=300, 207 (69%) with isolates for further testing), South Africa, 2006

Province	No isolate available	Serotype a	b	c	d	e	f	Non-typeable	Total
Eastern Cape	3	0	2	0	0	0	1	2	8
Free State	5	0	4	0	0	0	1	7	17
Gauteng	39	9	30	0	2	1	11	61	153
KwaZulu-Natal	25	1	16	0	0	0	3	11	56
Limpopo	0	0	1	0	0	0	0	0	1
Mpumalanga	1	0	2	0	0	0	0	1	4
Northern Cape	3	1	4	0	0	0	0	0	8
North West	2	0	0	0	0	0	0	1	3
Western Cape	15	1	12	1	0	0	2	19	50
Total	93	12	71	1	2	1	18	102	300

Table 5: Number and percentage of cases of *Haemophilus influenzae* disease as reported to RMPRU by specimen type, South Africa, 2006

Site of specimen	Serotype b		Serotypes a, c, d, e, f		Non-typeable		No isolate available	
	n	%	n	%	n	%	n	%
CSF	34	48	11	32	7	7	17	18
Blood	36	51	23	68	90	88	63	68
Other	1	1	0	0	5	5	13	14
Total	71		34		102		93	

(Continued on page 12)

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.[2] In 2006, a total of 48 cases of Hib were reported in children <5 years (Figure 3). Non-typeable strains were the most common *H. influenzae* causing disease in infants (Figure 4). The apparent increase in Hib in 2003 is probably related to improvements in surveillance (Figure 5).[2] Since 2003 rates of Hib disease as recorded by our surveillance network in infants <1 year of age have stabilised, and although there seems to be an increase in 2006, this is not significant (p=0.3, chi-squared test for trend, 2003 to 2006).

Seventeen percent of serotype b strains were resistant to ampicillin (all producing beta lactamase), 12 of 71 isolates tested, while 13% (13/102) of non-typeable strains were resistant (p=0.4).

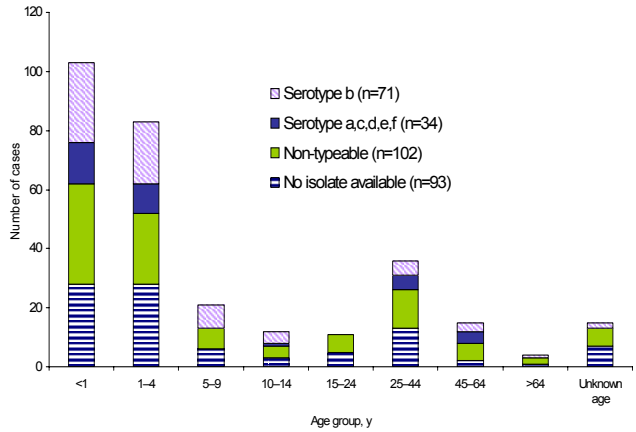

Figure 3: Number of cases of *Haemophilus influenzae* reported to RMPRU by serotype and age group, South Africa, 2006 (of 300 cases reported, 285 had known age, and 207 had viable isolates available for serotyping)

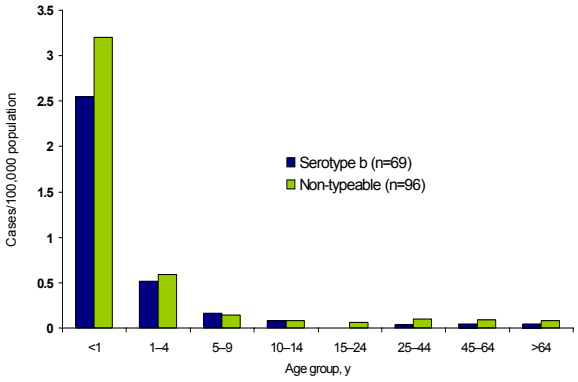

Figure 4: Reported age-specific incidence rates of serotype b and non-typeable *Haemophilus influenzae* disease, South Africa, 2006 (of 300 cases reported, 285 had known age, and 207 had viable isolates available for serotyping)

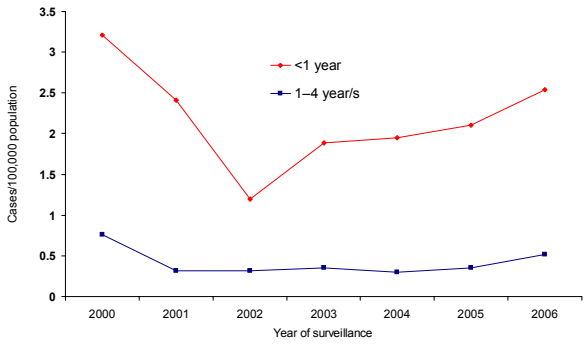

Figure 5: Incidence rates of *Haemophilus influenzae* serotype b disease in children <5 years, South Africa, 2000-2006

Invasive pneumococcal disease in 2006

The same trends of reported invasive pneumococcal disease were documented in 2006, with disease rates by province varying widely (Table 6). The age group at highest risk of disease in South Africa was infants <1 year of age (Figure 6). The majority of episodes reported to us were diagnosed from positive blood culture specimens (Table 7).

Overall, penicillin non-susceptible isolates have not increased from 2005 (1106/3422, 32% in 2006 compared to 1131/3656, 31% in 2005, p=0.2), and this ranges from 23% to 39% in different provinces (Table 8). Non-

Table 6: Number of cases and incidence rates of invasive pneumococcal disease as reported to RMPRU by province, South Africa, 2005 and 2006

Province	2005		2006	
	n	Cases/100 000	n	Cases/100 000
Eastern Cape	218	3.10	187	2.65
Free State	214	7.25	228	7.70
Gauteng	2260	25.06	2070	22.49
KwaZulu-Natal	465	4.82	462	4.75
Limpopo	73	1.30	102	1.80
Mpumalanga	229	7.11	209	6.44
Northern Cape	32	3.55	37	4.07
North West	114	2.98	139	3.61
Western Cape	502	10.80	488	10.27
South Africa	4107	8.76	3922	8.28

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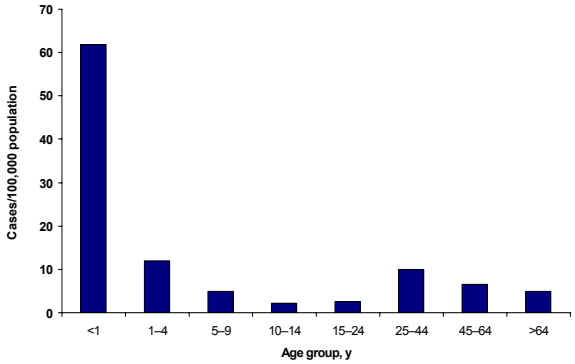

Figure 6: Reported age-specific incidence rates for invasive pneumococcal disease, South Africa, 2006 (3922 cases reported, age known in 3649)

Table 7: Number and percentage of cases of invasive pneumococcal disease as reported to RMPRU by specimen type, South Africa, 2006

Site of specimen	n	%
CSF	1300	33
Blood	2404	61
Other	218	6
Total	3922	

Table 8: Proportion of penicillin non-susceptible isolates from IPD cases reported to RMPRU in 2006 by province, South Africa

Province	Susceptible		Intermediately resistant		Resistant		No isolate available
	n	%	n	%	n	%	n
Eastern Cape	106	67	53	33	0	0.0	28
Free State	161	76	50	24	0	0.0	17
Gauteng	1158	66	583	33	2	0.1	327
KwaZulu-Natal	264	63	154	37	2	0.5	42
Limpopo	63	70	27	30	0	0.0	12
Mpumalanga	111	60	72	39	1	0.5	25
Northern Cape	25	74	9	26	0	0.0	3
North West	96	77	29	23	0	0.0	14
Western Cape	332	73	121	27	3	0.7	32
South Africa	2316	68	1098	32	8	0.2	500

susceptible isolates were common in children less than 1 year (283/600, 47%), and proportions were similar to those in 2005 (287/645, 44%), p=0.3 (Figure 7).

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 vaccine for the prevention of pneumococcal disease in children. The proportion of disease in 2006 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[3], in South Africa is more than 70% according to our data (Table 9).This supports advocacy from clinicians and parents for the vaccine price to be reduced and the possible inclusion of this vaccine in the EPI in the future.

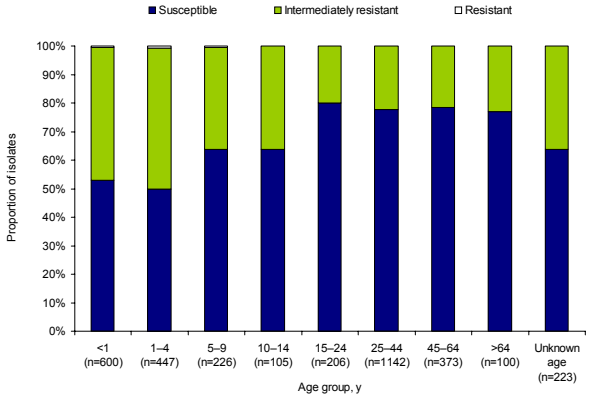

Figure 7: Number of cases of IPD reported to RMPRU in 2006 by age group and susceptibility to penicillin (3922 cases reported, 3422 with viable isolates)

Table 9: Proportion of cases reported in 2006 in children less than 5 years of age caused by the serotypes contained in the 7-valent vaccine, South Africa

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	27	5	43	74
Free State	42	9	76	67
Gauteng	327	54	546	70
KwaZulu-Natal	85	16	137	74
Limpopo	10	1	18	61
Mpumalanga	32	7	49	80
Northern Cape	8		11	73
North West	12	6	21	86
Western Cape	80	23	143	72
South Africa	623	121	1044	71

(Continued on page 14)

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CRYPTOCOCCAL SURVEILLANCE, SOUTH AFRICA, 2006

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

A total of 6372 incident cases of cryptococcosis were reported during 2006. Four hundred and thirty seven recurrent episodes were recorded (patient discharged and readmitted or if admission data were not available, repeat isolate >=30 days after the first isolate). In total, 5917 isolates were received by the MRU of which 5555 (94%) were viable. *Cryptococcus gattii* was detected in 136 of 4929 culture positive incident cases (2.7%).

The overall incidence rate in the South African general population was 13/100,000. Using projected/estimated denominators from the Medical Research Council report on the Demographic Impact of AIDS in South Africa,[1] the incidence of cryptococcosis amongst all HIV-infected individuals was 113/100,000 cases, and amongst people sick with AIDS was 10/1000 AIDS cases.

The provincial incidence rates for 2005 and 2006 reveal an increase in incidence rates in every province (Table 1). There is a trend to higher incidence rates within urban centres of South Africa (Figure 1). *C. gattii* was identified predominantly from cases presenting in the northern parts of South Africa (Figure 2). The highest incidence of cryptococcosis was in the 35-39 years age group (Figure 3); where gender was known (6205/6372, 97%), 55% of cases occurred in females. In children under 12 years of age, 93 cases were identified.

Table 1: Number of cases and incidence rates of *Cryptococcus* spp. as reported to MRU by province, South Africa, 2005 and 2006

Province	2005		2006	
	n	Cases/100 000	n	Cases/100 000
Eastern Cape*	447	7	1230	17
Free State	227	9	300	10
Gauteng	1571	16	1947	21
KwaZulu-Natal	882	9	1393	14
Limpopo	123	2	221	4
Mpumalanga	348	11	453	14
Northern Cape	50	1	64	7
North West	206	6	391	10
Western Cape	332	7	373	8
South Africa	4186	9	6372	13

*A complete surveillance audit was performed for the Eastern Cape in 2006; 616/1230 (50%) incident cases were detected by audit

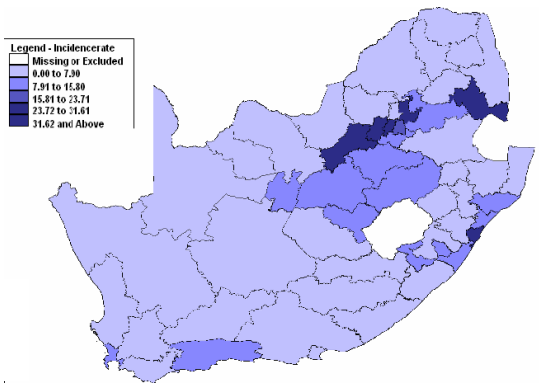

Figure 1. Chloropleth distribution map of incidence of cryptococcosis by health district in South Africa, 2006 (based on preliminary data excluding Eastern Cape audit cases)

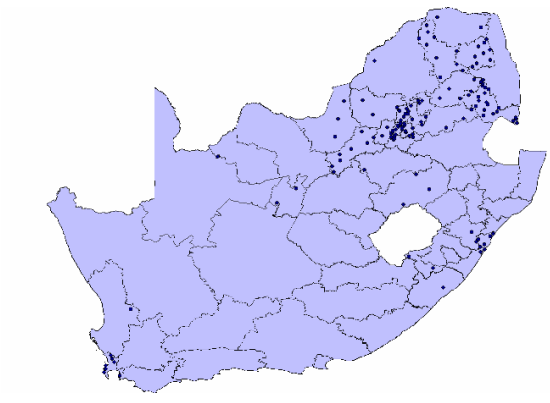

Figure 2. Cases of *Cryptococcus gattii* by health district of South Africa, 2006 (based on preliminary data)

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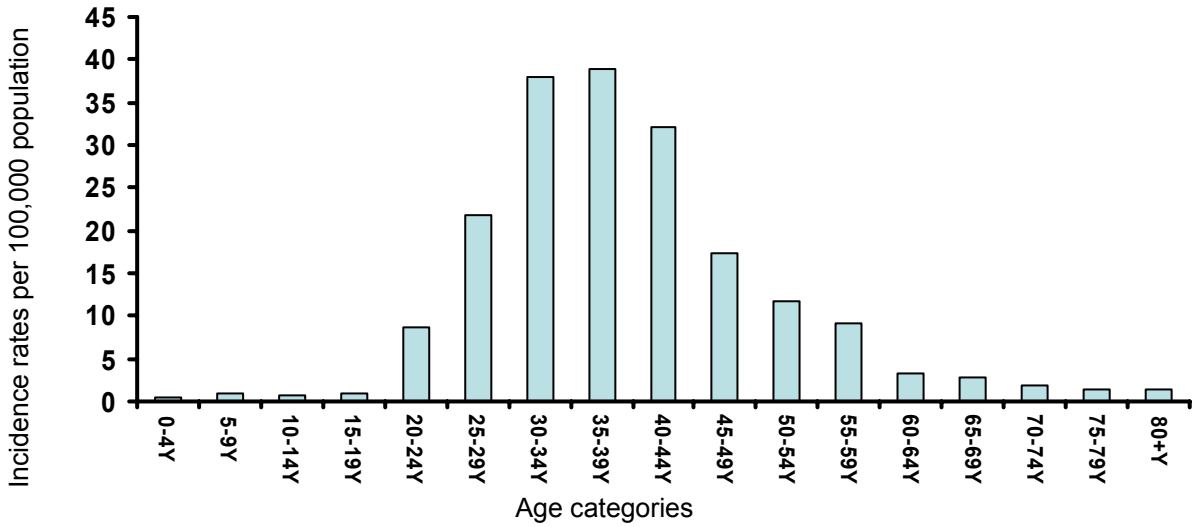

Figure 3. Age-related incidence of cryptococcosis in the general population, South Africa, 2006 (n = 6372, ages unknown in 10% [657/6372] cases).

Most incident cases (92%) were diagnosed with meningitis (tests on CSF positive for *Cryptococcus* spp.), and 4.4% with fungaemia (Table 2). The remainder of cases (17) originated from positive cultures of the pleural fluid and other sites.

Table 2: Number and percentage of cases of cryptococcal disease as reported to MRU by specimen type, South Africa, 2006

Site of specimen	n	%
CSF	5883	92
Blood	282	4.4
Other	17	0.3
Unknown	190	3
Total	6372	

Of 1486 incident cases presenting to enhanced surveillance sites and with completed clinical case report forms at the time of analysis, 507 cases (34%) died in hospital.

Interpretation of findings

Incidence rates of cryptococcosis amongst the general population in every province of South Africa were higher than 2005 rates. This appears not to be an artifact of reporting as preliminary analysis reveals that the increase in numbers is occurring at hospitals that were included in 2005 data. Given evidence from a population-based surveillance study conducted in Gauteng 2002-4 that shows that incidence of cryptococcosis may be a surrogate marker for AIDS prevalence,[2] it is reasonable to infer that the numbers of AIDS cases in South Africa have increased since 2005. Mortality rates amongst cryptococcosis patients admitted to enhanced surveillance sites is exceedingly high.

References

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PNEUMOCYSTIS JIROVECII PNEUMONIA (PCP) SURVEILLANCE, SOUTH AFRICA, 2006

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Sentinel-site surveillance started in May 2006; laboratories (including the Parasitology Reference Unit (PRU), NICD) that offer PCP diagnostic tests were requested to supply clinical isolates. Cases diagnosed at PRU from 1 January 2006 have been retrospectively included in this report. Table 1 shows laboratory-confirmed (IFA and/or PCR) cases of PCP accumulated for the period January–December, 2006. These data show an incomplete picture of the burden of PCP, for a number of reasons:

- laboratory diagnosis of PCP is restricted to relatively few large, mainly tertiary hospital, laboratories;

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- optimal respiratory sampling (e.g. bronchoalvelar lavage or saline-induced sputum) is seldom readily available, and therefore sensitivity of detection is often compromised;

Despite these limitations, the access to isolates from diverse areas of the country is useful for examining genetic diversity of strains and for monitoring molecular markers that may be relevant to cotrimoxazole resistance.

Table 1: Number of *Pneumocystis* pneumonia (PCP) cases reported to PRU by province, South Africa, 2006*

Province	2006
Eastern Cape	25
Free State	6
Gauteng	177
KwaZulu Natal	7
Limpopo	0
Mpumalanga	17
Northern Cape	0
North West	0
Western Cape	52
South Africa	284

*1 January – 31 December 2006

ENTERIC DISEASES SURVEILLANCE, SOUTH AFRICA, 2006

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Non-typhoidal *Salmonella* (NTS)

Table 1: Number* of invasive and non-invasive non-typhoidal *Salmonella* isolates (n = 1874) reported to EDRU by province, South Africa, 2006

Province	Invasive non-typhoidal *Salmonella*	Non-invasive non-typhoidal *Salmonella*
Eastern Cape	91	118
Free State	24	36
Gauteng	568	200
KwaZulu-Natal	132	196
Limpopo	7	34
Mpumalanga	43	85
Northern Cape	0	15
North West	16	58
Western Cape	97	154
South Africa	978	896

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

Greater numbers of cases in children less than one year (table 2) may reflect clinical practice, as well as burden of HIV. Increased numbers in the 15 to 64 year age group probably reflect the burden of HIV in South Africa.

Table 2: Case numbers and incidence rates (cases per 100 000 population) for invasive* non-typhoidal *Salmonella* reported to EDRU by age category, 2006

Age Category (years)	Cases	
	Number	Incidence rate
<1	173	16.3
1 - 5	98	1.9
6 - 14	34	0.4
15 - 64	513	1.7
>64	14	0.6
Total	832	1.8

*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.

Table 3: Number of non-typhoidal *Salmonella* isolates reported to EDRU by anatomical site of isolation*, 2006

Specimen	n	%
CSF	26	1.5
Blood culture	831	47.5
Stool	761	43.5
Other	133	7.6
Total	1751	

*Note that many cases had multiple isolates, including those with isolates from an invasive site and a second isolate from stool. Duplicate isolates are not reflected in the table.

Table 4: Results of antimicrobial susceptibility testing for all non-typhoidal *Salmonella* isolates (n = 1751) received by EDRU, 2006

Antimicrobial tested	Susceptible (%)	Intermediately resistant (%)	Resistant (%)
Ampicillin	45.6	0.1	54.4
Cotrimoxazole	49.3	0.0	50.7
Chloramphenicol	61.4	0.7	37.9
Nalidixic acid	62.3	0.0	37.7
Ciprofloxacin	99.5	0.1	0.5
Tetracycline	56.4	4.6	39.1
Kanamycin	68.5	12.2	19.3
Streptomycin	54.8	0.0	45.2
Imipenem	99.7	0.1	0.2
Ceftriaxone	73.8	0.1	26.2

Certain antimicrobial agents are tested for epidemiological reasons only and should not be used for treatment. Of those NTS isolates tested, 461 (26.3%) 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 376 (21%) NTS isolates. Pentavalent resistance (resistance to five or more antimicrobials) was observed in 876 (50%) isolates. Multi-drug resistant serotypes included *Salmonella* Typhimurium, *Salmonella* Isangi, *Salmonella* Muenchen and a newly recognised multi-drug resistant isolate, *Salmonella* Eppendorf (Table 5).

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Table 5: Commonest invasive and non-invasive non-typhoidal *Salmonella* serotypes (n = 1474) reported to EDRU by province, 2006

	Dublin	Enteritidis	Isangi	Typhimurium	Virchow
Eastern Cape	2	8	73	56	1
Free State	3	5	0	42	0
Gauteng	18	72	56	508	2
KwaZulu-Natal	5	14	94	134	3
Limpopo	0	4	4	12	0
Mpumalanga	9	6	4	53	23
Northern Cape	0	2	0	8	0
North West	0	3	8	56	0
Western Cape	7	10	38	130	1
South Africa	44	124	277	999	30

Typically, *Salmonella* Typhimurium and *Salmonella* Enteritidis are the commonest NTS isolates internationally. Large numbers of *Salmonella* Isangi reflect ongoing nosocomial transmission.[2] The number of *Salmonella* Virchow isolates was unusually high compared with previous years; this was associated with a food-borne outbreak of salmonellosis in Mpumalanga.

A lack of seasonality in NTS isolates reflects the nosocomial nature of many of the cases, as well as the burden of disease associated with HIV (Figure 1).

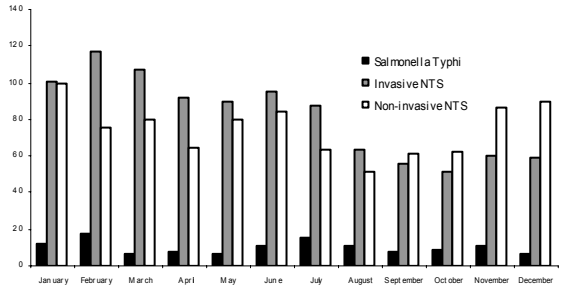


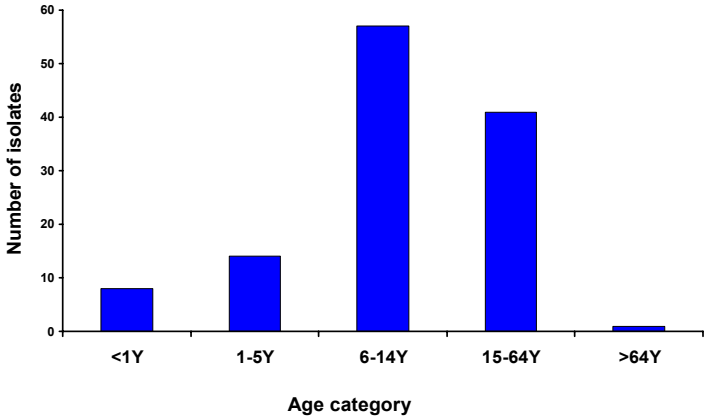
Figure 1. Numbers of NTS and *Salmonella* Typhi isolates received by EDRU in 2006 by month of isolation.

Salmonella Typhi

Table 1: Number of invasive and non-invasive *Salmonella* Typhi isolates (n = 124) reported to EDRU by province, South Africa, 2006

Province	Invasive *Salmonella* Typhi	Non-invasive *Salmonella* Typhi
Eastern Cape	44	7
Free State	1	0
Gauteng	12	4
KwaZulu-Natal	14	1
Limpopo	3	3
Mpumalanga	9	5
Northern Cape	0	0
North West	0	0
Western Cape	20	1
South Africa	103	21

Non-invasive isolates from stool or rectal swabs may reflect screening for the carrier state or follow-up of typhoid fever patients after treatment. Serological methods of diagnosis, e.g. Widal test and modifications of the Widal test using a rapid slide agglutination test are still used for diagnosis (not reflected in this report). The total number of reported isolates may thus not reflect actual numbers of cases in South Africa for the year; national incidence rates have not been calculated. Culture is the preferred method of diagnosis as it provides important information on antimicrobial resistance. No isolates were received from the Northern Cape or North West provinces. One *Salmonella* Paratyphi A and one *Salmonella* Paratyphi B isolate was received from Gauteng and the Free State respectively. Higher isolate numbers from KwaZulu Natal and Eastern Cape may reflect endemicity in these provinces.



*Age category unknown for 3 isolates

Figure 1. Number of *Salmonella* Typhi isolates reported to EDRU (n = 124) by age category*, 2006

The disease typically peaks between 6 and 14 years of age (Figure 1). The number of isolates from younger age groups, particularly in infants under one year of age, is of concern. The *Salmonella* Paratyphi A isolate was obtained from a 59 day old infant and the *Salmonella* Paratyphi B isolate was obtained from a 46 year old adult male.

No significant monthly variation in number of isolates was noted, indicating that there were no major outbreaks detected (See Non-typhoidal *Salmonella*, Figure 1).

Certain antimicrobials are tested for epidemiological purposes only and should not be used for treatment of typhoid fever. All isolates received in 2006 were susceptible to ciprofloxacin (Table 2), 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

(Continued on page 18)

resistance. Response to ciprofloxacin may be poor in the presence of nalidixic acid resistance.[1] Both *Salmonella* Paratyphi isolates were fully susceptible to all antimicrobial agents tested.

Table 2: Results of antimicrobial susceptibility testing for all *Salmonella* Typhi isolates (n = 124) received by EDRU, 2006

Antimicrobial tested	Susceptible (%)	Resistant (%)
Ampicillin	60.5	39.5
Cotrimoxazole	64.5	35.5
Chloramphenicol	93.5	5.6
Nalidixic acid	96.8	3.2
Ciprofloxacin	100.0	0.0
Tetracycline	58.1	41.9
Kanamycin	100.0	0.0
Streptomycin	62.9	37.1
Imipenem	100.0	0.0
Ceftriaxone	100.0	0.0

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Shigella

Table 1: Number of invasive and non-invasive *Shigella* isolates (n = 1113) reported to EDRU by province, South Africa, 2006

Province	Invasive Shigella	Non-invasive Shigella
Eastern Cape	1	120
Free State	4	48
Gauteng	22	206
KwaZulu-Natal	14	182
Limpopo	0	20
Mpumalanga	1	38
Northern Cape	0	32
North West	0	17
Western Cape	13	395
South Africa	55	1058

A higher number of non-invasive isolates submitted from the Western Cape may be due to local clinical practice (more stool specimens are submitted for diagnosis) as there was no predominance of any serotype for a given month or metropolitan area (full data not shown).

Table 2: Case numbers* and incidence rates (cases per 100 000 population) for *Shigella* (invasive and non-invasive) reported to EDRU by age category, 2006

Age Category (years)	Cases	
	Number	Incidence rate
<1	127	12.0
1 - 5	422	8.4
6 - 14	95	1.1
15 - 64	358	1.2
>64	41.0	1.7
Total	1043	2.2

*Cases may be underreported due to local clinical practices.

It is evident that the predominant burden of disease is in the under five-year age group.

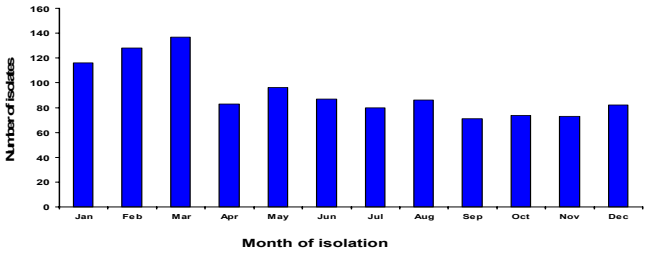

Figure 1. Number of non-invasive and invasive *Shigella* isolates reported to EDRU by month of isolation, 2006

Higher isolation rates between January and March in 2006 suggest seasonality.

The majority of isolates submitted were from stool (n = 1045), but 59 isolates were identified from blood cultures and other sterile sites. Nine isolates originated from other non-sterile sites.

Table 3: Results of antimicrobial susceptibility testing for all *Shigella* isolates (n = 1113) received by EDRU, 2006

Antimicrobial tested	Susceptible (%)	Intermediately resistant (%)	Resistant (%)
Ampicillin	48.7	0.3	51.0
Cotrimoxazole	17.6	0.0	82.4
Chloramphenicol	60.6	1.2	38.2
Nalidixic acid	98.6	0.1	1.3
Ciprofloxacin	99.8	0.1	0.1
Tetracycline	46.4	0.8	52.8
Kanamycin	99.5	0.1	0.4
Streptomycin	40.9	0.0	59.1
Imipenem	100.0	0.0	0.0
Ceftriaxone	99.5	0.0	0.5

Four of the isolates tested were found to produce extended spectrum beta-lactamases (ESBL). Quinolone resistance remains low. Certain antimicrobials were tested for surveillance purposes only and should not be used for treatment.

(Continued on page 19)

Table 4: Commonest* invasive and non-invasive *Shigella* serotypes (n = 814) reported to EDRU by province, 2006

	S. dysenteriae type 1	*S. flexneri* type 1b	*S. flexneri* type 2a	*S. flexneri* type 6	*S. sonnei* phase II
Eastern Cape	0	33	43	4	14
Free State	0	7	21	3	11
Gauteng	0	34	89	20	29
KwaZulu-Natal	1	46	48	19	27
Limpopo	0	4	5	2	2
Mpumalanga	0	9	10	8	0
Northern Cape	0	10	8	1	4
North West	0	5	4	0	1
Western Cape	1	108	104	30	49
South Africa	2	256	332	87	137

*Including *Shigella dysenteriae* type 1

A known outbreak of *Shigella sonnei* phase II in the Northern Cape is represented by only four submitted isolates; this is an under-representation of the actual number of cases. The predominance of *Shigella flexneri* 2a is typical of developing countries. *Shigella sonnei* is isolated more frequently in the developed world and is represented by a single serotype that can undergo phase variation.

Diarrhoeagenic *Escherichia coli* (DEC)

Table 1: Number* of diarrhoeagenic *Escheria coli* isolates (n = 130) reported to EDRU by province, South Africa, 2006

	EAggEC	EHEC	EIEC	EPEC	ETEC	STEC
Eastern Cape	7	0	0	35	3	0
Free State	0	0	0	2	1	0
Gauteng	6	1	1	26	1	1
KwaZulu-Natal	2	0	0	0	0	0
Limpopo	0	0	0	3	0	0
Mpumalanga	5	0	0	5	1	0
Northern Cape	0	0	0	0	0	0
North West	9	0	0	15	3	0
Western Cape	1	0	0	1	0	1
South Africa	30	1	1	87	9	2

EAggEC, enteroaggregative *E. coli*; EHEC, entero-haemorrhagic *E. coli*; EIEC, enteroinvasive *E. coli*; EPEC, enteropathogenic *E. coli*; ETEC, enterotoxigenic *E. coli*; STEC, Shiga-toxigenic *E. coli* (refer to methods section for an explanation of molecular pathotype determination)
*Incidence rates have not been calculated as numbers are not viewed as being fully representative.

Current clinical microbiology laboratory standard operating procedures are selective for detection of enteropathogenic *E. coli* (EPEC). The single enterohaemorrhagic *E. coli* (EHEC) isolate received from Gauteng (serotype O111) and the two Shiga-toxigenic *E. coli* (STEC) isolates received from Western Cape and Gauteng (both serotype O117) require specific note. No further history was available for the child with EHEC. Both children with STEC presented with dysentery; the identified genotypic pattern (*stx*1 positive, *eae* negative) in combination with serotype O117 has not been associated with haemolytic uraemic syndrome.[1] There was no known epidemiological linkage between these cases, but a high degree of clonality has been recognised in these isolates previously using molecular techniques. The preferential use of MacConkey agar with sorbitol for identifying *E. coli* O157 may result in EHEC infection due to other serotypes not being diagnosed.

Table 2: Number of diarrhoeagenic *E. coli* isolates (n = 130) reported to EDRU by age category, 2006

Age category (years)	EAggEC	EHEC	EIEC	EPEC	ETEC	STEC
<1	15	0	0	55	5	2
1 - 5	12	1	0	24	2	0
6 – 14	0	0	0	0	0	0
15 - 65	2	0	0	2	0	0
>65	0	0	0	1	0	0
Age unknown	1	0	1	5	2	0
Total	30	1	1	87	9	2

The predominance of isolates in children < 1 year may reflect culturing practices; infants are more likely to have stools taken for culture due to the devastating effects of diarrhoea at this age.

The commonest enteropathogenic E. coli serotypes reported in 2006 were serotype 0119 (n = 25), 055 (n = 18, 0111 (n = 10), 0142 (n = 8) and 0127 (n = 7).

The occurrence of serotype O55 is of interest as it has previously been shown that enterohaemorrhagic *E. coli* O157 evolved from this serotype.[2] Common enteroaggregative *E. coli* serotypes identified included O128ABC (n = 4), O127 (n = 3), O125 ABC (n = 2) and O147 (n = 2). No more than two isolates of any particular serotype of enterotoxigenic *E. coli* were received; serotypes included O11, O110, O115, O128 and variants, and O55, which is traditionally associated with EPEC. The single isolate of enteroinvasive *E. coli* (EIEC) received was serotyped as O28A.

References

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Vibrio cholerae

No *Vibrio cholerae* isolates from cases in South Africa were received by EDRU in 2006.

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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 2005/2006

	Disease/ Organism	Case Definition	Subgroup	Cumulative to 31 Dec, 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 Programme		2005	38	21	38	53	37	17	6	29	24	263
				2006	48	20	55	36	44	35	7	24	29	298
	Measles	Measles IgM positive cases from suspected measles cases, all ages		2005	478	1	44	74	2	5	0	1	16	621
				2006	3	0	19	2	1	9	4	21	1	60
	Rubella	Rubella IgM positive cases from suspected measles cases , all ages		2005	162	28	277	166	63	125	77	79	35	1012
				2006	159	37	409	194	336	137	69	139	88	1568
	VHF	Laboratory-confirmed cases of CCHF (unless otherwise stated), all ages		2005	0	0	0	0	0	0	0	0	1	1
				2006	0	2	2	0	0	0	4	0	0	8
	Rabies	Laboratory-confirmed human cases, all ages		2005	4	1	0	2	0	0	0	0	0	7
				2006	3	0	0	6	21	0	0	1	0	31
BACTERIAL AND FUNGAL DISEASES	*Haemophilus influenzae*	Invasive disease, all ages	All serotypes	2005	12	21	141	29	1	9	2	1	44	260
				2006	8	17	153	56	1	4	8	3	50	300
		Invasive disease, < 5 years	Serotype b	2005	4	3	18	4	0	0	0	1	6	36
				2006	2	4	23	8	1	1	1	0	8	48
			Serotypes a,c,d,e,f	2005	1	2	9	2	0	1	0	0	5	20
				2006	1	1	16	2	0	0	1	0	3	24
			Non-typeable (unencapsulated)	2005	2	4	40	4	0	2	0	0	5	57
				2006	1	5	40	4	0	1	0	0	7	58
			No isolate available for serotyping	2005	2	1	17	3	0	1	0	0	10	34
				2006	1	3	24	15	0	1	1	1	10	56
	Neisseria meningitidis	Invasive disease, all ages		2005	10	25	359	25	12	21	7	15	70	544
				2006	22	45	360	20	8	27	14	25	69	590
	Streptococcus pneumoniae	Invasive disease, all ages	Total cases	2005	218	214	2260	465	73	229	32	114	502	4107
				2006	187	228	2070	461	102	209	37	139	486	3919
			Penicillin non-susceptible isolates	2005	52	55	651	140	16	56	8	23	130	1131
				2006	53	50	585	156	27	73	9	29	124	1106
			No isolate available for susceptibility testing	2005	25	10	281	34	12	21	3	10	55	451
				2006	28	17	327	41	12	25	3	14	30	497
		Invasive disease, < 5 years		2005	75	74	646	176	19	64	5	23	203	1285
				2006	50	83	587	151	21	57	11	28	153	1141
	Salmonella spp. (not typhi)	Invasive disease, all ages		2005	38	26	523	104	9	36	0	4	68	808
				2006	47	23	556	118	4	34	0	16	77	875
		Confirmed cases, isolate from a non-sterile site, all ages		2005	143	31	206	178	29	70	7	43	173	880
				2006	112	36	197	197	31	80	15	58	153	879
	Salmonella typhi	Confirmed cases, isolate from any specimen, all ages		2005	31	0	32	12	8	91	0	0	10	184
				2006	51	1	16	15	6	14	0	0	21	124
	Shigella dysenteriae 1	Confirmed cases, isolate from any specimen		2005	0	0	0	0	0	0	0	0	5	5
				2006	0	0	0	1	0	0	0	0	1	2
	Shigella spp. (Non Sd1)	Confirmed cases, isolate from any specimen, all ages	All serotypes	2005	145	36	264	178	19	55	8	15	317	1037
				2006	121	52	228	195	20	39	32	17	407	1111
	Vibrio cholerae O1	Confirmed cases, isolate from any specimen, all ages	All serotypes	2005	0	0	0	0	0	0	0	0	0	0
				2006	0	0	0	0	0	0	0	0	0	0
	Cryptococcus (*Cryptococcus spp.*)	Invasive disease, all ages	Total cases (incl. *C neoformans*)	2005	447	227	1571	882	123	348	50	206	332	4186
				2006	1230	300	1947	1393	221	453	64	391	373	6372
			C. gattii	2005	4	2	31	17	20	16	0	9	9	108
				2006	3	4	43	9	25	28	3	13	8	136

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