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COMMUNICABLE DISEASES SURVEILLANCE BULLETIN

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FOREWORD

The May Bulletin of 2011 includes a report on a nosocomial outbreak as well as the GERMS-SA surveillance report.

We welcome the report of a nosocomial outbreak of multidrug-resistant *Pseudomonas aeruginosa* in a tertiary academic hospital. Many nosocomial outbreaks occur throughout South Africa each year but a minority undergo comprehensive investigation and even fewer are summarized for publication. This article highlights the challenges faced in many hospitals and emphasizes the importance of consistent maintenance of infection control procedures to prevent such outbreaks. In addition, the challenges in identification of such outbreaks are described. Outbreak descriptions of this nature lend support to the important role of strengthened surveillance for nosocomial pathogens.

The 2010 GERMS-SA surveillance report includes a summary of the main surveillance findings from national surveillance for the bacterial and fungal diseases under surveillance and includes findings from enhanced surveillance sites (ESS), in all 9 provinces, for the year 2010.

Two new pathogens (bacteraemic *Staphylococcus aureus* and *Klebsiella* species) were included under the GERMS-SA umbrella in July 2010. In the era of rapidly emerging antimicrobial resistance and frequent nosocomial outbreaks, sentinel laboratory-based surveillance for bacteraemic *S. aureus* and *K. pneumoniae* will allow GERMS-SA to detect emerging resistance, characterise nosocomial pathogens more carefully and document local epidemiology.

A major strength of a long-standing stable surveillance programme, such as GERMS-SA is the ability to document trends in disease incidence over time. In 2010, GERMS-SA has documented changes to the epidemiology of both cryptococcosis and IPD. The incidence of laboratory-confirmed cryptococcosis has decreased independent of changes to surveillance methods. It is likely that improved care, management and treatment of HIV-infected patients in South Africa has led to this decline. Ongoing surveillance will be able to document if this trend will continue. Unfortunately, the high in-hospital mortality has not changed; other public health measures such as screening to detect disease earlier may impact on outcomes. The decreased incidence of invasive pneumococcal disease amongst children less than 1 year is also likely a result of implementation of the 7-valent pneumococcal conjugate vaccine. These data provide important supporting evidence for the impact of major public health interventions such as vaccination and provision of care and treatment for HIV-infected persons.

Cheryl Cohen (Bulletin Editor)
Nelesh Govender and Vanessa Quan
(GERMS-SA Annual Report Editors)

CONTENTS

PRELIMINARY REPORT ON AN OUTBREAK OF MULTI-RESISTANT *PSEUDOMONAS AERUGINOSA* BLOODSTREAM INFECTION AMONG PATIENTS ADMITTED TO THE HAEMATOLOGY WARD OF A TERTIARY ACADEMIC HOSPITAL IN WESTERN CAPE, SOUTH AFRICA

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Background

An outbreak of Multi-resistant *Pseudomonas aeruginosa (*MRPA*)* infections was detected in a Clinical Haematology ICU (ward X) of a tertiary academic hospital after two patients died of MRPA bloodstream infection (BSI) in October 2010. Both patients had been hospitalized for prolonged periods of time and both were neutropenic. The initial response to the outbreak was a review of all practices in the ward, specifically maintenance of the environmental infection control system and adherence of basic infection control. In November another three patients experienced *P. aeruginosa* bloodstream infection, two of whom died. An outbreak investigation was then initiated which included a descriptive epidemiological investigation, collection and testing of environmental specimens, staff screening and a molecular epidemiology investigation with molecular typing of *P. aeruginosa* isolates. A moratorium on new admissions to the ward was put in place during the first week of December and the ward was thoroughly cleaned. After the ward was reopened another patient with MRPA-BSI died in January 2011. Subsequently MRPA has been isolated from non-sterile site specimens from two additional patients in February and March 2011. Laboratory surveillance of MRPA has been strengthened and the outbreak investigation is ongoing.

Description of Ward X

Ward X is a shared intensive care unit (ICU) with 12 isolation (private) rooms, of which 6 are used by a public sector hospital and 6 by a private hospital. Each room has an attached bathroom, as well as a nursing anteroom. Five of the rooms have a laminar flow system installed to provide protection against airborne pathogens. Access to the ward is controlled and strict infection control procedures are followed. Each hospital has its own nursing staff, as well as separate cleaning staff and cleaning equipment. Various facilities, including the nurses' station and sluice room, as well as staff facilities, such as tea room and change rooms, are shared by the two hospitals. Food is provided to all patients from the kitchen of the private hospital. Medical staff (doctors) attend patients from both hospitals.

The ward admits patients with malignant and non-malignant haematological conditions, most of whom have undergone bone marrow transplants (BMT) or are undergoing chemotherapy. These patients are usually highly immuno-compromised and are extremely susceptible to infections. Patients are confined to individual rooms, but do occasionally leave the ward, e.g. to visit the radiology department for investigations.

Epidemiological Findings

A search for additional MRPA isolates from blood specimens collected from ward X was conducted using the laboratory information system (DISALAB). An additional 4 patients were identified, thus yielding a total of 10 patients with MRPA bloodstream infection by the end of January 2011. There were no reported cases of MRPA infection in ward X prior to January 2010. The first two cases were detected in January 2010, 1 in May 2010, 1 in June, 2 in October 2010, 3 in November and 1 in January 2011 (Figure 1).

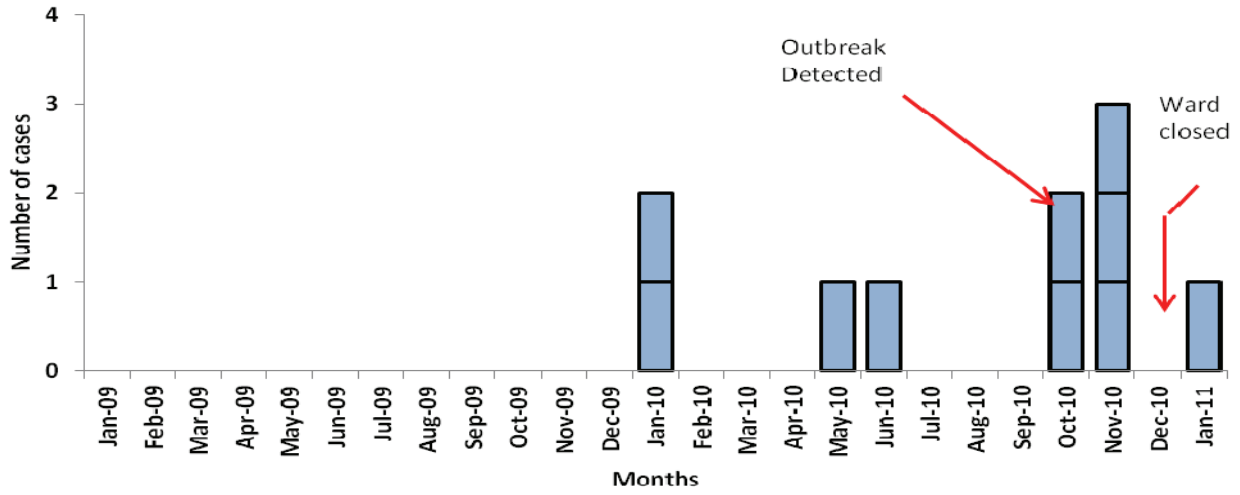

Figure 1: Distribution of multi-resistant *P. aeruginosa* bloodstream infection cases in the Clinical Haematology ward of a tertiary academic hospital, January 2009 – January 2011

One patient, case 9, was known to be colonized with MRPA four days prior to development of BSI: MRPA was isolated from a skin swab taken at the site of insertion of a vascular catheter. In another patient, case 10, gut colonization was detected concurrently with BSI. Apart from these 2 patients, there were no other patients in Ward X with MRPA colonization or infection detected at any other site, prior to February 2011.

The case fatality rate was 80% (n = 8). The ages of the cases ranged from 16 years to 60 years with the average of 36 years. The majority of cases were female (78%). Five of the cases were under the care of the private hospital and five under the public sector hospital. Six (67%) of the cases had a diagnosis of acute myeloid leukemia, the remaining three cases had aplastic anemia, B-cell lymphoma, and acute lymphoblastic leukemia respectively. (Table 1) Only very limited information obtained from the laboratory information system is included in this report, as the patient medical records have only recently been made available.

A search of the hospital information system (Clinicom) showed that there were 118 patients admitted to ward X between 1 December 2009 and 31 January 2011 with a total 4399 patient-days at risk in ward X. The 10 cases of MRPA BSI represent a cumulative incidence of 8.5% or an incidence density of 2 cases per 1000 patient-days. The average total length of hospitalization of the cases was 65 days, and ranged from 11 days to 126 days.

The time-line of hospitalization (Figure 2) of the cases shows that most of the cases had been admitted to Ward X concurrently with at least one other case. Case 1 and case 2 had admissions that overlapped in December 2009 and January 2010. Case 4 and case 8, who were detected in July and November respectively, had admissions that overlapped with each other and with that of case 2.

Table 1: Characteristics of *P. aeruginosa* bloodstream infection cases detected in the Haematology ward of a tertiary academic hospital from January 2010 to January 2011.

Patients	Hospital	Age	Sex	Diagnosis	Last white cell count before MRPA +ve specimen	Total length of hospitalization in (days) until death or discharge
Case 1	public	46	M	AML	0.03	11
Case 2	private	16	M	Aplastic Anemia	0.01	52
Case 3	public	28	F	B-cell Lymphoma	20.64	70
Case 4	private	58	F			74
Case 5	private	20	F	ALL	0.04	105
Case 6	public	37	F	AML	0.22	19
Case 7	private	48	F	AML	0.12	29
Case 8	private	46	F	AML	0.1	49
Case 9	public	60	F	AML	11.59	126
Case 10	public	20	F	AML	0.05	125

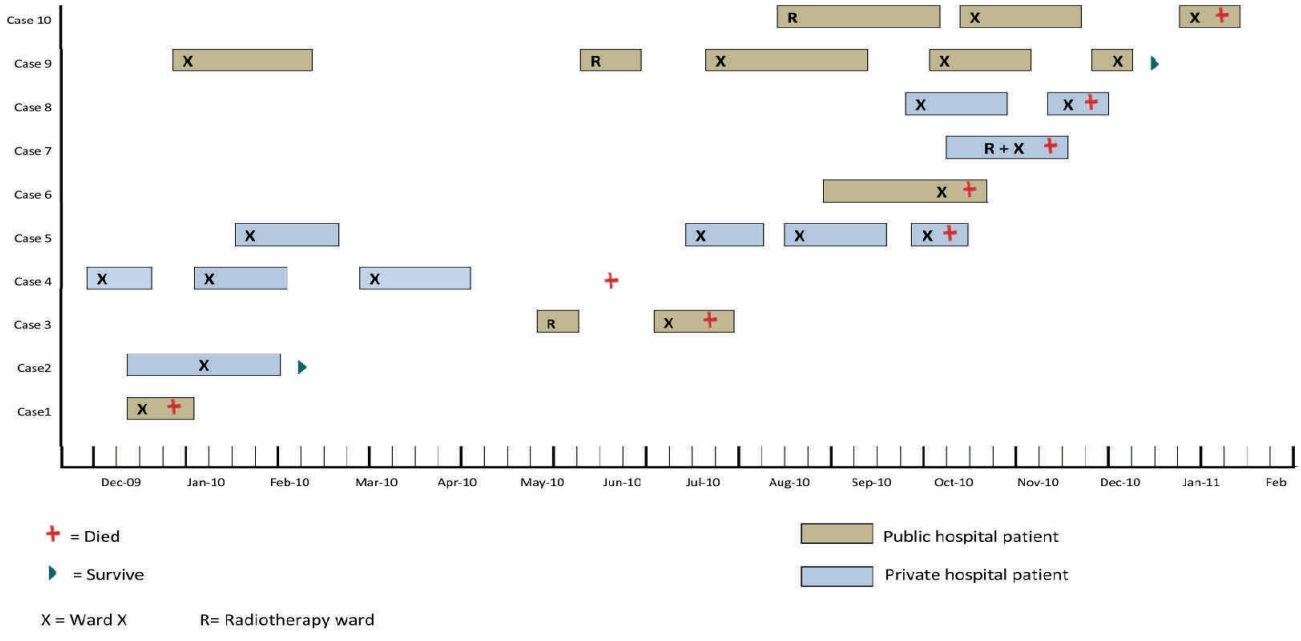

Figure 2: Timeline of hospitalization of multi-resistant *Pseudomonas aeruginosa* BSI cases.

Case 8 had multiple episodes of admission that overlapped with those of most other cases. Case 5, 6, 7, 8 and 9 had been in ward X concurrently in October 2010. Cases 3, 6, 8 and 9 had previously been admitted in the radiotherapy ward at the public sector hospital. There is no evidence of admission to other wards for the remainder of the patients.

Staff Screening

A total of 51 Ward X staff members were requested to submit stool specimens to test for gut colonization by *P. aeruginosa*. This included 9 doctors, 9 public hospital nursing staff, 2 porters, 16 private hospital nursing staff and 8 private hospital kitchen staff. A search of the laboratory information system showed that the compliance rate for submission of stool specimens was 54.9% (n = 28). Twenty six specimens were tested and two were rejected because the specimens were unlabelled. *P. aeruginosa* was not detected in any specimen, although three specimens contained probable nosocomial pathogens; two ESBL-producing *Klebsiella pneumoniae*, and one multi-resistant *Acinetobacter baumannii*.

Staff were also asked to report any other possible personal sources of MRPA colonization, for example, chronic otitis externa or any infection around the finger nails.

Environmental screening

A total of 48 environmental screening specimens were collected from the following sites:

- Swabs from taps in all 12 isolation rooms
- Samples of tap water from all 12 isolation rooms
- Swabs from Puritan bottles (used by individual patients for respiratory therapy) in laminar flow rooms 1,2 and 3
- Cleaning equipment, including buckets, mops and cloths used from both hospitals

A single isolate of multi-resistant *P. aeruginosa* was isolated from a mop.

In addition, samples of food and swabs from the food kitchen were tested at a private laboratory and no isolates of multi-resistant *P. aeruginosa* reported.

Molecular typing

In order to determine the relatedness of the *P. aeruginosa* isolates, the isolates were subjected to molecular typing by pulsed-field gel electrophoresis (PFGE) following digestion with the restriction enzyme *SpeI*. PFGE is considered the "gold standard" typing tool for the sub typing of bacterial

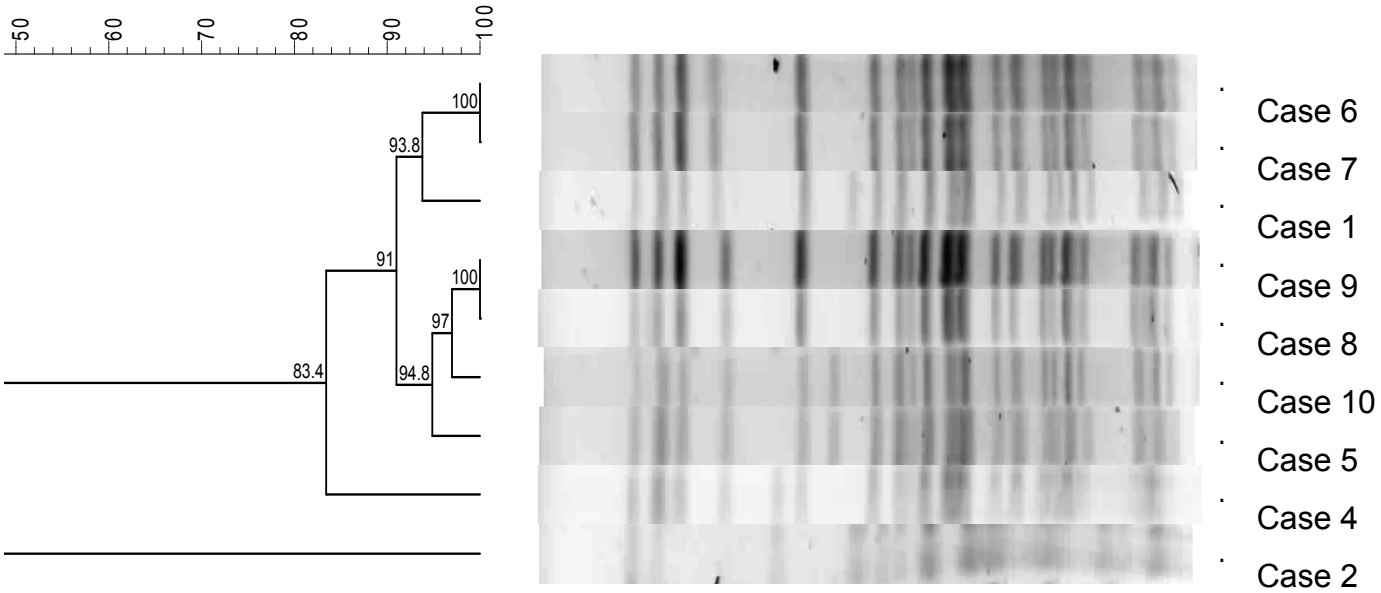

Figure 3: Dendrogram and PFGE photograph of *P. aeruginosa* isolates from outbreak cases.

isolates in a local outbreak setting. Details of the PFGE method are provided in Appendix 1.

PFGE analysis (Figure 3) showed that 8/9 strains are closely related, whereas one isolate, Case 2, is unrelated. Cases 6 and 7 show 100% PFGE profile homology, as do Cases 8 and 9. There is clearly a distinct cluster with 91% homology involving Cases 1, 5, 6, 7, 8, 9, and 10. Case 4 is similar, but slightly different to these strains with 83% homology. *P. aeruginosa* isolates with greater than 87% homology are considered to be identical.

The isolate from case 3 was not included in this figure, but had previously been shown to be closely related to the major Ward X outbreak strain. The environmental strain isolated from a mop was shown to be unrelated to the outbreak strain.

Discussion

The investigation was carried out with the objectives being to describe the outbreak, to determine common patient characteristics and to identify the possible source of the outbreak and the route of transmission with the ultimate goal being to halt the spread of *P. aeruginosa* in ward X. The results obtained from molecular typing show a very close relationship between the cases detected between October 2010 and January 2011. The strain was most likely introduced to the unit by in January 2010 by Case 1 and subsequently spread to the other patients.

However, the route of transmission remains unknown. Direct patient-to-patient transmission of MRPA in the unit can be ruled out since patients are kept in private rooms with very little or no contact. Since medical staff are a common link between patients in the two sections of the unit, spread from a colonized staff member is a possible source of transmission. Thus far in the investigation staff screening has been inconclusive. Since just over 50% of staff members complied with screening it is unknown whether any of the remaining staff were colonized by *P. aeruginosa*. Participation of doctors in the screening process was particularly low.

Patients from both hospitals are also exposed to items from the communal areas of the unit. While environmental sampling did not reveal any contamination with MRPA, this does completely exclude the environment as a potential source of infection. It is possible that the strain may be associated with particular isolation rooms in the unit that were/are colonized with the MRPA strain. The unit, however, does not keep record of which rooms were occupied by particular patients and therefore it was not possible to verify this. It is also possible that patients may have been exposed to MRPA while in another ward in the hospital, but current data from the hospital information system does not support this hypothesis.

Irrespective of the original source of the MRPA outbreak strain or whether it was subsequently transmitted from human or environmental locations, the most alarming feature of this outbreak is that the ongoing spread of the organism implies a breach in the infection control procedures in a unit that is specifically designed for maximal protection of vulnerable patients. Multi-resistant pathogens such as MRPA may be sporadically introduced into units such as this, but should be contained by the standard infection control precautions in place. The timing of any such failures of infection control precautions cannot be determined with any certainty, as there may be considerable delay between colonization with a particular pathogen and the onset of infection. Although the haematology unit had a policy of regular screening of patients for gut colonization with resistant pathogens, this had not been implemented consistently in the weeks and months preceding the outbreak. Hence it is not possible to determine whether a significant number of patients had gut colonization with MRPA prior to the outbreak. Colonisation or infection with MRPA was not detected in any Ward X patient, apart from Cases 9 and 10, as mentioned previously.

Detection of this outbreak was facilitated by the severity of the outcome, as the immunocompromised patients succumbed very rapidly to BSI with such an aggressive pathogen. Even so, the failure to recognize the outbreak for the first 9 months highlights the need to strengthen laboratory surveillance of infections among highly susceptible patients. There is also a need to determine baseline data for nosocomial pathogens among high risk groups, which will serve as a basis for early detection of outbreaks.

Further Actions

A retrospective case-control investigation for risk factors associated with *P. aeruginosa* bloodstream infection among patients admitted to ward X between December 2009 and January 2011 is currently under way. The *P. aeruginosa* bacteraemia cases will be compared to patients with BSIs due to Gram-negative bacilli other than *P. aeruginosa* (infected controls) and to patients not infected with *P. aeruginosa* or other Gram-negative bacilli (non-infected controls). Antecedent variables (factors) that will be assessed include length of hospitalization in X, demographics (age and sex), immunity status (white cell count), prior antibiotic use (30 days before infection), devices (IV lines, Nasogastric tubes, urinary catheters, and mechanical ventilation), diagnosis and co morbidities. Mortality within 7 days will also be compared between cases and infected controls.

Appendix 1

Isolates were inoculated on to boiled blood agar plates and incubated aerobically overnight at 37°C. For the preparation of cell suspensions a loopful of bacteria was collected from the agar surface and suspended in 1ml of cell suspension buffer (CSB; 100mM Tris [pH8.0], 100mM EDTA [pH8.0]**)**. The cell concentrations were adjusted to between 0.700 and 0.799 absorbance at 600nm using a spectrophotometer (Biomate).

A total of 10 isolates were prepared at one time and these were kept at room temperature before mixing with low-melting point agarose (SeaPlaque® Agarose, Lonza Rocklands). The low melting point agarose was prepared by adding 2% (w/v) low-melting point agarose to TE buffer (10mM Tris, 1mM EDTA pH 8.0) and completely melting the agarose in a microwave oven before allowing it to cool in a 50°C water bath. Once cooled, sodium-dodecyl sulfate (SDS) was added to the melted agarose to a final concentration of 1%. Aliquots of 400ul of the prepared cell suspensions were added to 400ul of the low-melting point agarose containing SDS and mixed gently by pipetting 2-3 times. 400ul of this mixture was dispensed into reusable plug moulds (Amersham Biosciences). The plug moulds were placed at 4°C and allowed to solidify for 10 minutes.

A total of 1ml of cell lysis buffer one (CLS buffer-1; 50mM Tris, 50mM EDTA, Lysozyme [25mg/ml] and proteinase K [1.5mg/ml] was prepared and the plugs were removed from the mould and added to the CLS buffer 1. Cell lysis was performed at 37°C in a shaking water bath for 60 minutes. The plugs were then transferred to another tube containing 1ml CLS buffer-2 (CLS buffer 2, 0.5M EDTA [pH 8.0], 1% sarcosyl and proteinase K [400ug/ml] and incubated at 56°C with shaking for an hour.

The agarose plugs were carefully removed from the lysis solutions and rinsed 3 times in ultra-pure water (reagent grade type 1) and then 3 times with TE buffer (10mM Tris, 1mM EDTA pH 8.0). Each of the washing steps was performed for 20 minutes at 50°C with agitation. After the last step plugs are stored in TE buffer at 4°C until further usage.

Restriction enzyme digestion of the intact genomic DNA in the agarose plug was carried out using a quarter of a sample plug, cut with a scalpel and placed in a micro centrifuge tube. The micro centrifuge tube contained 20ul of the restriction buffer (1X) with bovine serum albumin (100ug/ml) and 1U of *Spe*I (Fermentas) enzyme added to a final volume of 200ul for each plug. Plugs were then incubated at 37°C for 1 hour.

A 1% (w/v) pulsed-field certified agarose (Bio-Rad Laboratories) gel was prepared in 150ml of 0.5X TBE buffer (0.045M Tris-borate, 0.001M EDTA). After melting the agarose was allowed to cool at 60°C before pouring and then left to set for 1 hour. Following restriction enzyme digestion, the plugs were loaded into the gel and the entire gel was placed in the electrophoresis chamber and immersed in 0.5X TBE buffer. PFGE was performed using CHEF-DR II system (Amersham Biosciences) at 14°C with 6V/cm^3. The initial and final switch times were 5s and 30s, respectively for 20 hours. The gel was visualised by staining with ethidium bromide (10mg/ml) in 400ml ultra-pure sterile water for 15 minutes and then de-staining in Ultra pure sterile water for 2 hours. The restriction profiles were visualised using UV transillumination and the image captured using Chemi genius Bio imaging system (Syngene). The DNA profiles were analyzed by GelCompar software (version 4.6 Applied Maths, Belgium). DNA profiles obtained were normalized to the reference strains, PAO1 and Lambda markers (New England Biolabs). A 1% band tolerance was used for the comparison of profiles and cluster analysis was done using the un-weighted pair group method and arithmetic average (UPGMA).

THE GROUP FOR ENTERIC, RESPIRATORY & MENINGEAL DISEASE SURVEILLANCE IN SOUTH AFRICA (GERMS-SA)

Introduction

As in previous years, the 2010 GERMS-SA Annual Report includes a summary of key data from national surveillance, including clinical data from enhanced surveillance sites (ESS) for the year. The surveillance methodology did not change in 2010 and audit cases were not detected from NHLS laboratories in KwaZulu-Natal. Two new pathogens (bacteraemic *Staphylococcus aureus* and *Klebsiella* species) were included under the GERMS-SA umbrella in July 2010 and the interim reports are included. The new, scaled-up HIV/AIDS prevention and treatment plan was launched in April 2010 with the objective to reduce the rate of infection by 50% by 2011 and to provide antiretroviral (ARV) treatment to 80% of those who need to be on treatment.[1] GERMS-SA, as a mature surveillance system, is well positioned to monitor the impact of national interventions such as vaccines and the Comprehensive Care, Management and Treatment Programme for HIV/AIDS.

The methods utilised by the GERMS-SA surveillance programme have been previously described in detail.[2] Further details of GERMS-SA methods are available in the GERMS-SA annual report (access at www.nicd.ac.za). Incidence rates were calculated using mid-year population estimates for 2009 and 2010 from Statistics South Africa (Table 1).[3] Incidence rates in the HIV-infected and AIDS populations were calculated for 2009 and 2010, using estimated population denominators from the Actuarial Society of South Africa (ASSA) 2003 model (Table 1), assuming that the HIV/AIDS prevalence amongst cases with known status was similar to those with unknown status.[4] All reported incidence rates are expressed as cases per 100 000 population, unless otherwise stated.

Table 1: Population denominators used to calculate incidence rates, 2009 and 2010.

Province	General population*		HIV-infected population**		AIDS population **	
	2009	2010	2009	2010	2009	2010
Eastern Cape	6 648 601	6 743 823	757 818	785 217	79 705	84 991
Free State	2 902 518	2 824 570	395 344	396 068	50 111	51 196
Gauteng	10 531 308	11 192 029	1 454 006	1 455 350	166 078	171 132
KwaZulu-Natal	10 449 141	10 645 508	1 567 048	1 572 457	206 294	209 638
Limpopo	5 227 503	5 439 552	451 553	468 659	47 390	50 275
Mpumalanga	3 606 572	3 617 513	459 051	462 687	59 336	60 107
Northern Cape	1 147 137	1 103 918	69 595	71 434	7 458	8 093
North West	3 450 517	3 200 649	501 066	504 224	62 634	64 916
Western Cape	5 356 844	5 223 908	309 102	318 115	28 391	31 338
South Africa	**49 320 141**	**49 991 470**	**5 64 583**	**6 034 211**	**707 397**	**731 686**

Data source: *Statistics South Africa; **Actuarial Society of South Africa (ASSA)

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ENHANCED SURVEILLANCE SITE (ESS) PROJECT

National Microbiology Surveillance Unit, National Institute for Communicable Diseases

In 2010, of 18 385 surveillance patients detected by GERMS-SA, 4 307 (23%) were diagnosed at enhanced surveillance sites. Of case patients with recorded HIV status, 82% (2 578/3 140) were HIV-infected (Table 2). The proportion of case patients with confirmed HIV infection varied by surveillance disease: unsurprisingly, a very high proportion of patients with AIDS-defining infections like cryptococcosis (99%) and PCP (83%) were HIV-infected; HIV infection amongst patients with invasive pneumococcal disease and non-typoidal salmonellosis, for which HIV is a known risk factor, were both 74%, and less than one third (29%) of patients with invasive meningococcal disease were HIV-infected.

Table 2: Number and percentage* of patients, diagnosed with laboratory-confirmed disease at GERMS-SA enhanced surveillance sites, with confirmed HIV-1 infection**, South Africa, 2010, n=4307.

Pathogen	Case patients, n	Case patients with completed case report forms, n (%)	Case patients with known HIV status, n (%)	Case patients with confirmed HIV infection, n (%)
Cryptococcus species	1761	1468 (83)	1373 (94)	1359 (99)
Pneumocystis jirovecii	120	90 (75)	84 (93)	70 (83)
Neisseria meningitidis	173	158 (91)	132 (84)	38 (29)
Streptococcus pneumoniae	1723	1454 (84)	1183 (81)	871 (74)
Haemophilus influenzae	192	150 (78)	129 (86)	66 (51)
Salmonella species†	318	267 (84)	224(84)	166 (74)
Shigella species†	20	16 (80)	15 (94)	8 (53)
Total	**4307**	**3603 (84)**	**3140 (87)**	**2578 (82)**

*The percentage (in brackets) in each cell was calculated using the numerator from that cell and the corresponding denominator from the cell to the left; **HIV infection was confirmed by an age-appropriate, laboratory test and recorded by surveillance officers at enhanced surveillance sites; †Invasive.

SALMONELLA ENTERICA SEROTYPE TYPHI AND *S. ENTERICA* SEROTYPES PARATYPHI A, PARATYPHI B AND PARATYPHI C

Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Results

Salmonella Typhi isolation by month shows the effect of a foodborne outbreak in Pretoria (Tshwane) in May and June (Figure 1).[1] *Salmonella* Typhi isolates from both invasive and non-invasive sites are reported in Table 3. A single isolate of *Salmonella* Paratyphi B L (+) tartrate (+) (*Salmonella* Paratyphi B var. Java) was received from a stool specimen of a 37 year-old male in Gauteng. A second isolate of *Salmonella* Paratyphi B L (-) tartrate (-) was received from a 10 month-old infant in KwaZulu Natal (Figure 1). No isolates of *Salmonella* Paratyphi A or of *Salmonella* Paratyphi C were received. The number of isolates within each age group is reported in Table 4, indicating that most isolates are from patients in the 5 year – 34 year age group, although infection is seen in both older and younger age groups. The occurrence of the typhoid fever outbreak in May and June contributed to the extended age range in comparison with past years.[1] No isolates of *Salmonella* Typhi received in 2010 were resistant to ciprofloxacin, the treatment of choice, but resistance to nalidixic acid remains cause for concern (Table 5). The *Salmonella* Paratyphi B isolates were fully susceptible to all antimicrobials tested.

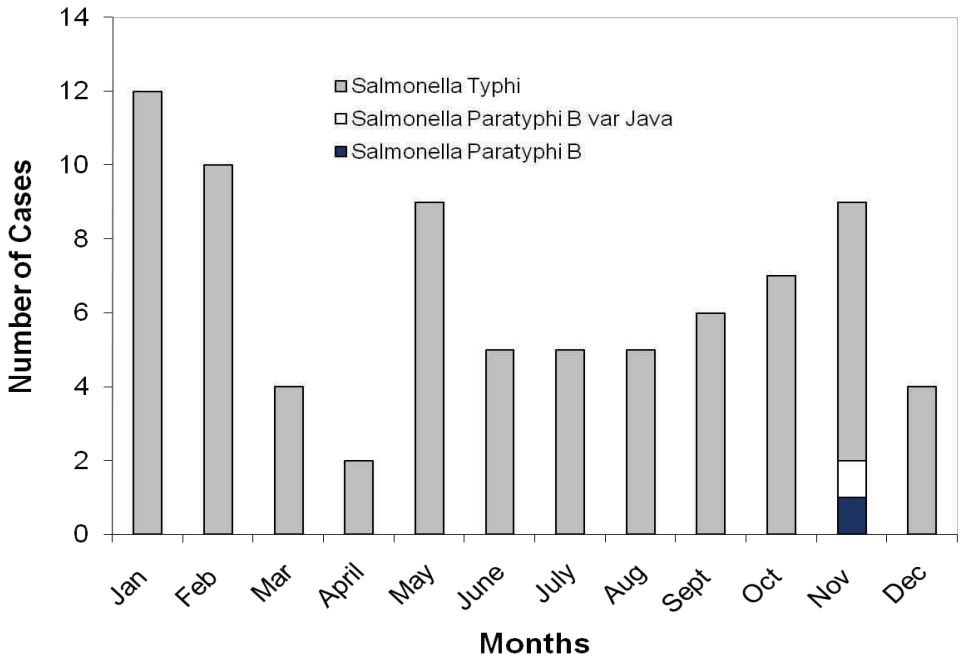

Figure 1: Number of non-invasive and invasive cases of Salmonella typhi and Paratyphi B, reported to GERMS-SA, by month of specimen collection, South Africa 2010, (n = 76) including audit reports.

Table 3: Number of invasive and non-invasive *Salmonella* Typhi cases reported to GERMS-SA, South Africa, 2010, n=76.

Province	Non-invasive *Salmonella* Typhi	Invasive *Salmonella* Typhi
Eastern Cape	2	9
Free State	0	2
Gauteng	4	25
KwaZulu-Natal	2	7
Limpopo	1	0
Mpumalanga	2	9
Northern Cape	0	0
North West	0	0
Western Cape	7	6
South Africa	**18**	**58**

Table 4: Number of *Salmonella* Typhi isolates reported to GERMS-SA by age category, South Africa, 2010, n=76.

Age category (years)	*Salmonella* Typhi isolates
0 - 4	10
5 – 14	21
15 – 24	14
25 – 34	16
35 - 44	6
45 - 54	5
55 - 64	1
≥ 65	1
Unknown	2
Total	**76**

Table 5: Antimicrobial susceptibility test results for all *Salmonella* Typhi isolates received by GERMS-SA, South Africa, 2010, n=74 (excluding audit reports, missing isolates, mixed and contaminated cultures).

Antimicrobial agent	Susceptible (%)	Intermediate (%)	Resistant (%)
Ampicillin	66 (89)	1 (1.4)	7 (10)
Trimethoprim	64 (87)	0 (0)	10 (14)
Sulphamethoxazole	46 (62)	0 (0)	28 (38)
Chloramphenicol	63 (85)	1 (1.4)	10 (14)
Nalidixic acid	63 (85)	0 (0)	11 (15)
Ciprofloxacin	74 (100)	0 (0)	0 (0)
Tetracycline	71 (96)	0 (0)	3 (4)
Streptomycin	67 (91)	0 (0)	7 (10)
Imipenem	74 (100)	0 (0)	0 (0)
Ceftriaxone	74 (100)	0 (0)	0 (0)

Discussion

Salmonella Typhi isolates from both invasive and non-invasive sites are included in these analyses, as both add to burden of infection in South Africa and thus represent a public health risk, although data may not reflect actual burden of disease. This is compounded by the challenges of alternative diagnostic methods for typhoid fever, including both clinical and serological. The number of reported *Salmonella* Typhi isolates was regarded as a substantial underestimate and thus incidence rates were not calculated. These thus exclude those patients in whom a serological or clinical diagnosis was made without culture. Certain antimicrobials were tested for epi-demiological purposes only, and should not be used for treatment of typhoid fever. 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.[2] Response to ciprofloxacin may be poor in the presence of nalidixic acid resistance. The ciprofloxacin E-test is recommended to guide antimicrobial management in such cases.[2] Ceftriaxone would be regarded as the alternative therapy of choice in these cases, as well as those typhoid fever cases where the organism is fully resistant to ciprofloxacin.

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Report compiled by Karen Keddy

NON-TYPHOIDAL *SALMONELLA ENTERICA* (NTS)

Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Results

Invasive diseases does not appear to have a seasonal prevalence, but increased number of non-invasive disease due to NTS in the earlier months of the year may be a surveillance artefact, due to increased surveillance for foodborne disease prior to FIFA 2010 World Cup (Figure 2). The number of cases of invasive and non-invasive disease, by province, reported to GERMS-SA, is stated in Table 6. The number of cases of invasive and non-invasive disease, by age group, is shown in Table 7, but incidence rates have only been calculated for invasive NTS, due to differences in stool-taking practices in adult and paediatric medical care. Most invasive isolates were identified from blood cultures, although isolates were frequently identified from both blood culture and another site, including stool and other normally-sterile sites (Table 8). Multi-drug resistance remains a challenge, including resistance to first -line antimicrobial agents and the quinolones (Table 9). Of the NTS isolates tested, 153/1976 (7.7%) were extended-spectrum beta-lactamase (ESBL) producers (Table 9). Multi-drug resistant serotypes included primarily *Salmonella* Typhimurium and *Salmonella* Isangi (Table 10).

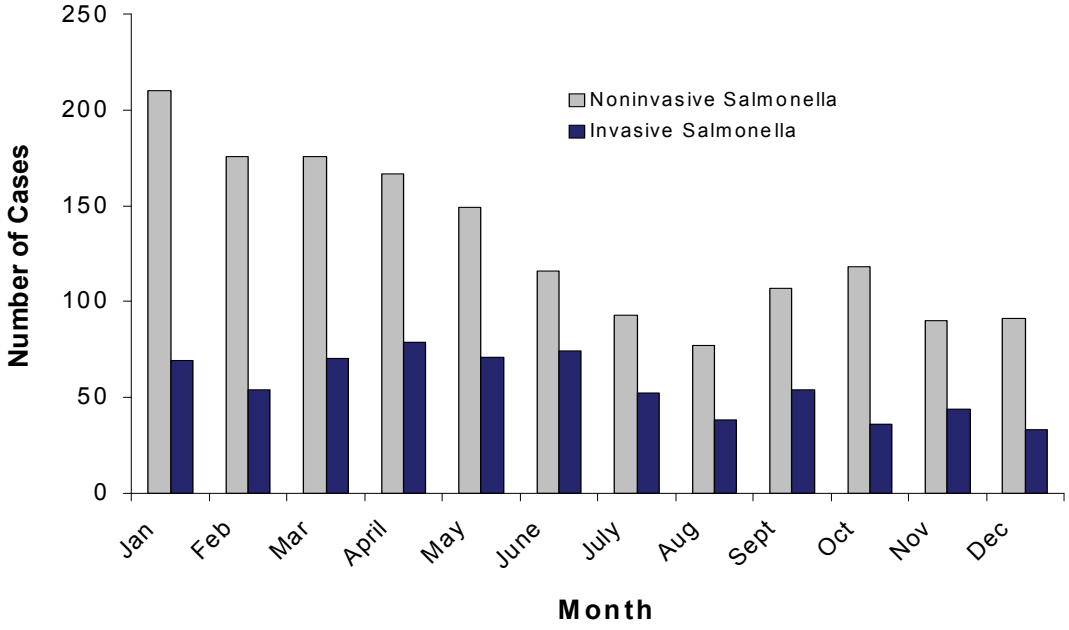

Figure 2: Number of non-invasive and invasive, non-typhoidal *Salmonella* cases, reported to GERMS-SA, by month of specimen collection, South Africa, 2010, n=2244 (including audit reports).

Table 6: Number* of invasive and non-invasive non-typhoidal *Salmonella* cases reported to GERMS-SA, by province, South Africa, 2010, n=2244 (including audit reports).

Province	Non-invasive, non-typhoidal *Salmonella* isolates	Invasive, non-typhoidal *Salmonella* isolates
Eastern Cape	211	55
Free State	54	22
Gauteng	706	381
KwaZulu-Natal	206	79
Limpopo	30	12
Mpumalanga	112	18
Northern Cape	15	15
North West	46	17
Western Cape	190	75
South Africa	**1570**	**674**

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

Discussion

Non-typhoidal salmonellosis may be a food-borne disease, for which data are poorly captured in South Africa, and where the patients normally present with gastroenteritis, or may be an AIDS-defining illness, in which case the organism frequently becomes invasive. No marked seasonal prevalence was noted in 2010 for invasive or non-invasive isolates. *Salmonella* Infantis appears to be gaining importance as a common serotype in South Africa. Certain antimicrobial agents were tested for epidemiological reasons only, and should not be used for treatment. Antimicrobial resistance remains a cause for concern.

Table 7: Number of cases and incidence rates for invasive* and non-invasive non-typhoidal *Salmonella* reported to GERMS-SA by age category, South Africa, 2010, n=2244 (including audit reports).

Age Category (years)	Cases		
	Non-invasive	Invasive	Incidence rate for invasive disease**
0 - 4	613	184	3.6
5 - 14	167	35	0.3
15 - 24	95	42	0.4
25 - 34	196	140	1.7
35 - 44	160	131	2.2
45 - 54	117	66	1.6
55 - 64	78	34	1.1
≥ 65	75	22	0.9
Unknown	69	20	-
Total	**1570**	**674**	**1.3**

*Incidence rates for non-invasive non-typhoidal *Salmonella* were not calculated because specimens may not have been submitted for culture from all patients, with gastroenteritis due to non-typhoidal *Salmonella,* in clinical practice; **Incidence rates are expressed as cases per 100 000 population.

Table 8: Number of non-typhoidal *Salmonella* cases reported to GERMS-SA by primary anatomical site of isolation*, South Africa, 2010, n=2244 (including audit reports).

Specimen	n	%
CSF	24	1
Blood culture	598	27
Stool	1329	59
Other	293	13
Total	**2244**	**100**

*Many cases had multiple isolates of the same serotype, including those with isolates from an invasive site of origin and a second isolate from stool, or isolates from two different normally-sterile sites.

Table 9: Antimicrobial susceptibility test results for all non-typhoidal *Salmonella* isolates received by GERMS-SA, South Africa, 2010, n=1976 (excluding audit reports, missing isolates, mixed and contaminated cultures).

Antimicrobial agent	Susceptible (%)		Intermediate (%)		Resistant (%)	
Ampicillin	1630	(83)	3	(0.2)	343	(17)
Trimethoprim	1647	(83)	0	(0.0)	329	(17)
Sulphamethoxazole	990	(50)	0	(0.0)	986	(50)
Chloramphenicol	1666	(84)	17	(0.9)	293	(15)
Nalidixic acid	1768	(90)	0	(0.0)	208	(11)
Ciprofloxacin	1965	(99)	3	(0.2)	8	(0.4)
Tetracycline	1494	(76)	31	(1.6)	451	(23)
Streptomycin	1597	(81)	0	(0.0)	379	(19)
Imipenem	1976	(100)	0	(0.0)	0	(0)
Ceftriaxone	1823	(92)	0	(0.0)	153	(8)

Table 10: Commonest invasive and non-invasive non-typhoidal *Salmonella* serotypes reported to GERMS-SA by province, South Africa, 2010, n=1384 (excluding audit reports).

Province	Serotype				
	Enteritidis	Heidelberg	Infantis	Isangi	Typhimurium
Eastern Cape	32	3	1	32	85
Free State	15	0	3	0	32
Gauteng	337	15	27	18	295
KwaZulu-Natal	79	5	4	33	72
Limpopo	7	1	0	7	7
Mpumalanga	21	14	5	0	32
Northern Cape	4	0	0	1	14
North West	14	1	0	1	14
Western Cape	49	6	4	2	92
South Africa	**558**	**45**	**44**	**94**	**643**

Report compiled by Karen Keddy

SHIGELLA SPECIES

Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Results

Higher isolation rates in January through to May are potentially a surveillance artefact, due to heightened awareness of food and waterborne disease prior to the FIFA 2010 World Cup and increased testing of symptomatic patients. Slightly increased numbers from January to March in 2010 do however suggest seasonality (Figure 3). Although the primary burden of disease due to *Shigella* is non-invasive dysentery or diarrhoea, invasive disease remains an important cause of morbidity in South Africa (Table 11). The predominant burden of disease, including both invasive and non-invasive shigellosis, is in the under-five-year age group (Table 12). Quinolone resistance remains low, but fluoroquinolone resistance appears to be emerging (Table 13). Nine of 1588 (0.6%) isolates tested were ESBL-producers. Predominant serotypes confirm that *S. flexneri* 2a remains the commonest cause of shigellosis in South Africa. *S. dysenteriae* type 1 was not isolated in 2010 (Table 14).

Discussion

Shigella infection is largely due to water-borne outbreaks in South Africa, although person-to-person transmission may play a role. Certain antimicrobials were tested for surveillance purposes only, and should not be used for treatment. Resistance to the third generation cephalosporins and fluoroquinolones remains low, but should continue to be monitored.

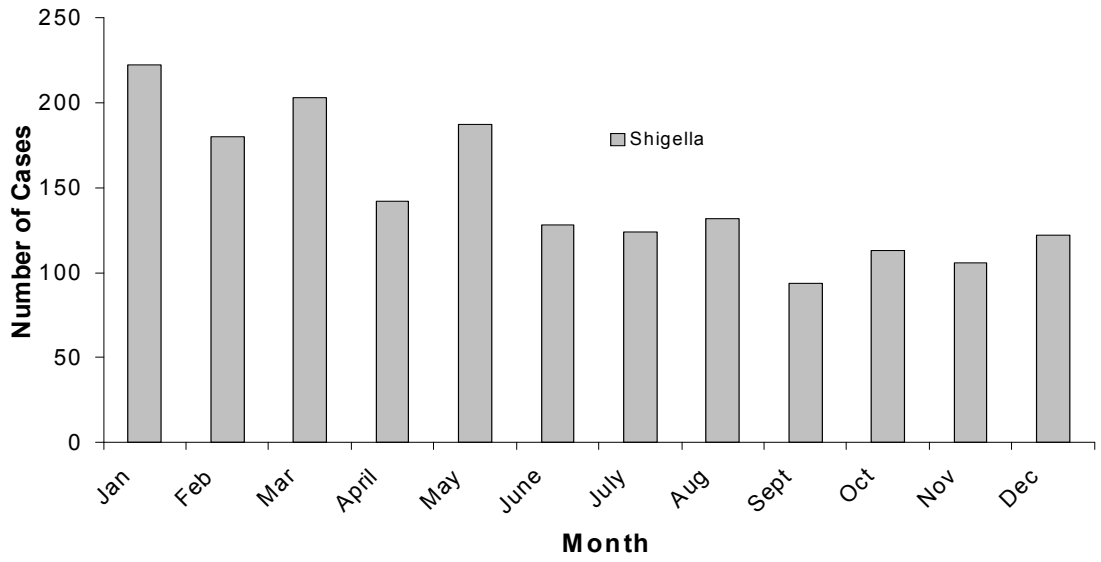

Figure 3: Number of non-invasive and invasive *Shigella* isolates, reported to GERMS-SA, by month of specimen collection, South Africa, 2010, n=1753 (including audit reports).

Table 11: Number of invasive and non-invasive *Shigella* isolates reported to GERMS-SA by province, South Africa, 2010, n=1753 (including audit reports).

Province	Non-invasive *Shigella*	Invasive *Shigella*
Eastern Cape	264	6
Free State	53	0
Gauteng	692	19
KwaZulu-Natal	133	9
Limpopo	17	1
Mpumalanga	50	2
Northern Cape	35	1
North West	36	1
Western Cape	424	10
South Africa	**1704**	**49**

Table 12: Number of cases* and incidence rates for *Shigella* (invasive and non-invasive)** reported to GERMS-SA by age category, South Africa, 2010, n=1753.

	Cases		
Age Category (years)	Non-invasive	Invasive	Incidence rate for invasive disease**
0 - 4	820	16	0.3
5 - 14	247	5	0.1
15 - 24	81	1	0.01
25 - 34	180	5	0.1
35 - 44	115	10	0.2
45 - 54	77	5	0.1
55 - 64	60	0	0.0
≥ 65	54	2	0.1
Unknown	70	5	-
Total	**1704**	**49**	**0.1**

*Cases may be under-reported due to local clinical practices: no mixed infections were identified.
**Incidence rates are expressed as cases per 100,000 population.

Table 13: Antimicrobial susceptibility test results for *Shigella* isolates received by GERMS-SA, South Africa, 2010, n=1588 (excluding audit reports, missing isolates, mixed and contaminated cultures).

Antimicrobial agent	Susceptible (%)	Intermediate (%)	Resistant (%)
Ampicillin	870 (55)	1 (0.1)	717 (45)
Trimethoprim	132 (8)	0 (0.0)	1456 (92)
Sulphamethoxazole	271 (17)	0 (0.0)	1317 (83)
Chloramphenicol	1090 (69)	27 (1.7)	471 (30)
Nalidixic acid	1571 (99)	0 (0.0)	17 (1)
Ciprofloxacin	1584 (100)	0 (0.0)	4 (0.3)
Tetracycline	619 (39)	40 (2.5)	929 (59)
Streptomycin	622 (39)	0 (0.0)	966 (61)
Imipenem	100 (0)	0 (0.0)	0 (0)
Ceftriaxone	1582 (100)	0 (0.0)	6 (0.4)

Table 14: Commonest* invasive and non-invasive *Shigella* serotypes reported to GERMS-SA by province, South Africa, 2010, n=1218 (excluding audit reports, missing isolates, mixed and contaminated cultures).

Province	*S. dysenteriae* type 1	*S. flexneri* type 2a	*S. flexneri* type 3a	*S. flexneri* type 6	*S. sonnei* phase I/II
Eastern Cape	0	93	42	13	45
Free State	0	8	4	5	15
Gauteng	0	154	60	68	237
KwaZulu-Natal	0	56	11	8	34
Limpopo	0	3	1	1	1
Mpumalanga	0	4	4	11	8
Northern Cape	0	7	2	5	2
North West	0	4	1	2	5
Western Cape	0	179	58	15	52
South Africa	**0**	**508**	**183**	**128**	**399**

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

Report compiled by Karen Keddy

DIARRHOEAGENIC *ESCHERICHIA COLI* (DEC)

Enteric Diseases Reference Unit, National Institute for Communicable Diseases

Results

An increased number of cases in the first half of the year is potentially a surveillance artefact, as discussed above (Figure 4). Enteropathogenic *E. coli* (EPEC) remains the commonest cause of diarrhoea, due to this pathogen, identified in South Africa (Table 15). The predominance of cases in younger children under five years of age may reflect, in part, specimen-taking practices, as well as the burden of diarrhoeal disease in this age group (Table 16). Three patients had mixed infections with three different DEC pathotypes and 23 patients had mixed infections with two different DEC pathotypes. Six isolates of *E. coli* O157 were received, two of these were enterohaemorrhagic *E. coli* (EHEC), and four were enteropathogenic *E. coli* (EPEC). A range of serotypes were associated with Shiga-toxigenic *E. coli* (STEC) and EHEC, including O157 (two isolates), O26 (two isolates), O111, O117, O115 and O5. The commonest serotypes associated with EPEC included O55, O111, O119, O127, O145 and O109. Diverse serotypes were also noted for other enterovirulent *E. coli* isolates. Identification of both EHEC and STEC was incidental.[1]

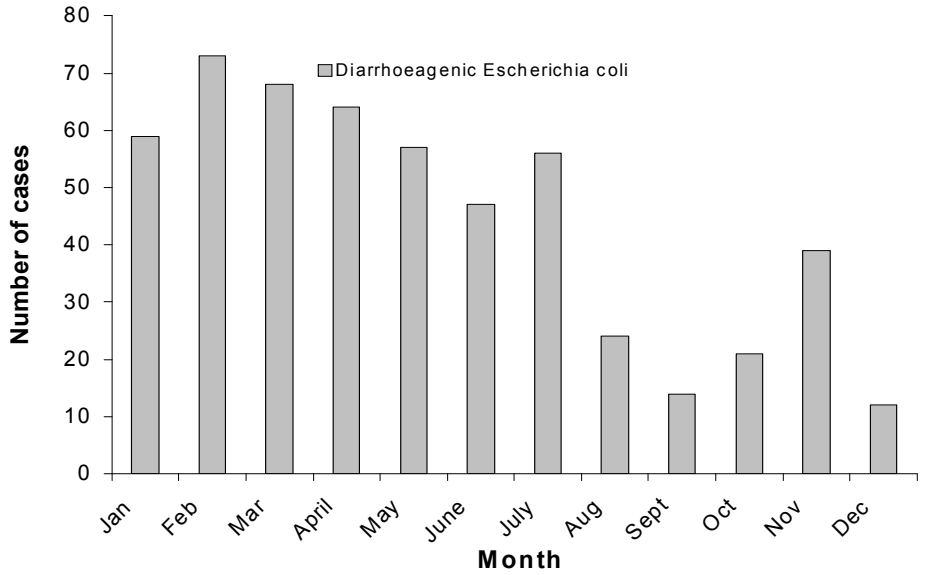

Figure 4: Number of diarrhoeagenic *Escherichia coli* isolates, reported to GERMS-SA, by month of specimen collection, South Africa, 2010, n=534.

Table 15: Number of diarrhoeagenic *Escherichia coli* isolates reported to GERMS-SA by province, South Africa, 2010, n=534.

Province	DAEC	EAggEC	STEC/ EHEC	EIEC	EPEC	ETEC
Eastern Cape	5	18	0	0	37	1
Free State	1	0	0	0	1	0
Gauteng	29	17	7	4	277	5
Kwazulu-Natal	1	0	1	0	4	0
Limpopo	1	1	0	0	2	0
Mpumalanga	50	20	0	3	23	7
Northern Cape	0	2	0	0	4	0
North West	0	0	0	0	8	0
Western Cape	3	0	0	0	2	0
South Africa	**90**	**58**	**8**	**7**	**358**	**13**

DAEC: diffusely-adherent *E. coli*; EAggEC: enteroaggregative *E. coli*; STEC/EHEC: Shiga-toxigenic *E. coli* or enterohaemorrhagic *E. coli*; EIEC: enteroinvasive *E. coli*; EPEC: enteropathogenic *E. coli*; ETEC: enterotoxigenic *E. coli.*

Table 16: Number of diarrhoeagenic *E. coli* isolates reported to GERMS-SA by age category, South Africa, 2010, n=534.

Age category (years)	DAEC	EAggEC	EHEC/ STEC	EIEC	EPEC	ETEC
0 - 4	52	45	7	3	344	9
5 - 14	5	2	0	0	3	1
15 - 24	2	3	0	0	0	0
25 - 34	11	1	0	2	4	2
35 - 44	10	2	1	1	2	0
45 - 54	3	1	0	1	1	0
55 - 64	2	1	0	0	0	1
≥ 65	3	1	0	0	0	0
Unknown	2	2	0	0	4	0
Total	**90**	**58**	**8**	**7**	**358**	**13**

DAEC: diffusely-adherent *E. coli*; EAggEC: enteroaggregative *E. coli*; STEC/EHEC: Shiga-toxigenic *E. coli* or enterohaemorrhagic *E. coli*; EIEC: enteroinvasive *E. coli*; EPEC: enteropathogenic *E. coli*; ETEC: enterotoxigenic *E. coli.*

Discussion

Incidence rates were not calculated as numbers were not viewed as being fully representative. Actual burden of disease due to diarrhoeagenic *E. coli* is probably greatly underestimated in South Africa, as management is primarily syndromic and centres on rehydration. As a result, clinicians are unlikely to prioritise stool-taking in uncomplicated cases of diarrhoea. Disease in the past appears to have been primarily associated with water-borne outbreaks, due to high level of faecal contamination in water sources, and this trend appears to be continuing. 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. Seasonality graphs may be affected by current specimen-taking and laboratory diagnostic practices may not be optimal to accurately reflect burden of illness in South Africa of disease due to diarrhoeagenic *E. coli*.

Reference

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Report compiled by Karen Keddy

VIBRIO CHOLERAE O1

Enteric Diseases Reference Unit, National Institute for Communicable Diseases

A single case of cholera due to *Vibrio cholerae* O1 Ogawa was reported in 2010 in South Africa. The organism was isolated from the stool of a 37 year-old woman, who presented with profuse watery diarrhoea on returning from a trip to India in June.[1] Molecular epidemiological techniques using pulsed field gel electrophoresis (PFGE) confirmed that the isolate was closely related to known Indian strains of *Vibrio cholerae* O1.

Reference

1. NICD. Imported cholera. NICD Communique. http://www.nicd.ac.za/assets/files/NICDCommJune10Vol09_06.pdf Accessed 25 March 2011

Report compiled by Karen Keddy

CRYPTOCOCCUS SPECIES

Mycology Reference Unit, National Institute for Communicable Diseases

Results

During 2010, 7371 case patients, with laboratory-confirmed, incident cryptococcal episodes, were reported. The overall incidence for the general South African population decreased in 2010 (Table 17). Similarly, incidence amongst HIV-infected individuals (140/100 000 in 2009 and 122/100 000 in 2010) and people sick with AIDS (12/1000 in 2009 and 10/1000 in 2010) decreased. Incidence decreased in all provinces except the Western Cape where the incidence remained stable (Table 17). The peak incidence of cryptococcosis was recorded amongst patients aged 35-39 years (Figure 5). Two hundred and twelve children, younger than 15 years, had laboratory-confirmed cryptococcosis; 48/212 (23%) were younger than 1 year-old. Where gender was known (7258/7371, 98%), 53% patients were female. Most patients (6623/7371; 90%) were diagnosed with meningitis (laboratory tests on cerebrospinal fluid positive for *Cryptococcus* species), and 649/7371 (9%) were diagnosed with fungaemia (Table 18). Ninety two patients were diagnosed by culture of urine, sputum, pleural fluid and other specimen types. At enhanced surveillance sites, 1761 patients were diagnosed with cryptococcosis, with viable isolates received from 1296/1761 (73%) patients. Isolates were typed from 1296 cases; 1240 (96%) were identified as *Cryptococcus neoformans* and 51 (4%) were identified as *Cryptococcus gattii.* Of note, both *C. gattii* and *C. neoformans* were isolated from 4 patients. *C. gattii* cases were diagnosed in 8 provinces: Gauteng (n=24), Mpumalanga (n=11), Limpopo (n=5), KwaZulu-Natal (n=5), North West (n=4), Western Cape (n=3), Northern Cape (n=3) and Free State (n=1). The in-hospital case-fatality ratio for patients at enhanced surveillance sites did not significantly change between 2009 and 2010 (591/1812 (33%) vs. 503/1459 (34%)); p=0.2).

Discussion

In 2010, almost 1000 fewer incident cases were detected by GERMS-SA, compared with 2009. The overall incidence also decreased. This may indicate that the National HIV/AIDS Comprehensive Care, Management and Treatment (CCMT) Programme has made an impact. Most patients continued to be diagnosed with meningitis.

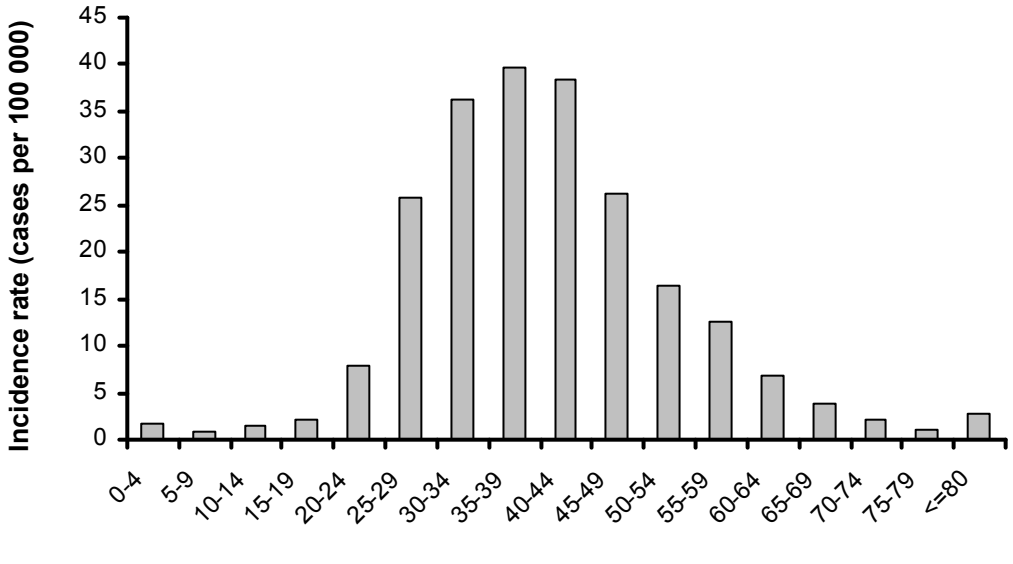


Figure 5: Age-specific incidence rates for laboratory-confirmed, cryptococcal cases, reported to GERMS-SA, South Africa, 2010, n=7371.

Table 17: Number of cases and incidence of cryptococcal disease reported to GERMS-SA by province, South Africa, 2009 and 2010, n=15701.

	2009*		2010*	
Province	**n**	**Incidence****	**n**	**Incidence****
Eastern Cape	1393	21	1336	20
Free State	483	17	460	16
Gauteng	2125	20	2117	19
KwaZulu-Natal	1455	14	1053	10
Limpopo	682	13	552	10
Mpumalanga	836	23	734	20
Northern Cape	82	7	65	6
North West	738	21	555	17
Western Cape	536	10	499	10
South Africa	**8330**	**17**	**7371**	**15**

*A similar surveillance audit was performed for NHLS laboratories in 8 provinces (excluding KwaZulu-Natal) in 2009 and 2010, detecting additional microscopy (India ink), cryptococcal antigen and culture-confirmed cases; **Incidence rates were calculated based on population denominators provided by Statistics South Africa, and are expressed as cases per 100,000 population.

Table 18: Number and percentage of cases of cryptococcal disease reported to GERMS-SA by specimen type, South Africa, 2009 and 2010, n=15701.

Site of specimen	**2009**		**2010**	
	n	**%**	**n**	**%**
CSF	7676	92	6623	90
Blood	579	7	649	9
Other	75	1	92	1
Unknown	0	0	7	<1
	8330		**7371**	

The demographic profile of patients with cryptococcosis mirrored the profile of HIV-infected patients in South Africa. Although very few children were diagnosed with cryptococcosis, more than a quarter of paediatric cases were diagnosed amongst infants <1 year-old. In 2010, a low proportion of patients were infected with *C. gattii; C. gattii* cases were diagnosed across the country. The in-hospital mortality of patients with cryptococcosis remained high, and is probably due to patients entering the health care system with advanced cryptococcal disease.

Report compiled by Nelesh Govender

PNEUMOCYSTIS JIROVECII

Parasitology Reference Unit, National Institute for Communicable Diseases

Results

In 2010, 298 cases of *P. jirovecii* pneumonia (PCP) were reported (Table 19), with 307 specimens available for analysis. Numbers of *P. jirovecii*-positive specimens peaked in children less than one year of age and in the 20 to 59 year age group (Figure 6). Of cases with known gender, 60% (178/298) were female. Of all reported case patients, 120 (40%) were diagnosed at enhanced surveillance sites and had clinical data available. During admission, 84% (75/89) of patients who tested for HIV, were HIV-positive. Where outcome was known, in-hospital mortality rate was 33% (30/91). In 17% (16/93) of patients this was their second or later hospitalization for PCP. Of patients who recovered, 95% (57/60) were discharged with a lower respiratory tract infection as the final diagnosis. Most of the patients had concurrent infections, of which clinically-diagnosed candidiasis (30/85) and tuberculosis

(23/85) were the most common. Restriction fragment length polymorphism (RFLP) analysis was performed on 141 of the 307 specimens received for 2010 (Figure 7) to determine prevalent mutations in the DHPS gene. The most frequent observed mutations were the wild type + M1 mix (47/141), followed by wild type + M3 or wild type + M1 + M2 mix (25/141) and wild type (23/141).

Discussion

According to published data, *Pneumocystis* pneumonia (PCP) is the opportunistic infection that patients most often present with when HIV infection is diagnosed for the first time.[1] The number of cases reported here does not approximate the true burden of disease in South Africa, and for this reason PCP surveillance through GERMS-SA ended on 31 December 2010. Analysis of the data and specimens collected are ongoing. Currently, the Parasitology Reference Unit is proposing to add PCP as an aetiological agent to the current severe acute respiratory infections (SARI) surveillance study, a prospective, hospital -based sentinel surveillance initiated in 2009. In this surveillance system, persons hospitalised with acute respiratory illness, who meet inclusion criteria have clinical data and specimens obtained for aetiology testing.

In the beginning of 2010 we introduced a restriction fragment length polymorphism (RFLP) test to determine the extent of the two main dihydropteroate synthase (DHPS) gene mutations [M1 at codon 55 and M2 at codon 57 (M3 mutation is a combination of these two)] circulating in the population under surveillance. It is suggested that these mutations are linked to sulfa-drug resistance, and are more likely to occur in patients who have previously been exposed to sulfa drugs.[2] Antimicrobial drug resistance has emerged as a possible contributor to failure of patients to respond to PCP therapy, although results correlating resistance markers with clinical outcome have been conflicting.[3] We have found a high number of DHPS mutations in specimens processed so far, indicating that mutations are a common occurrence in the surveillance population.[4] The relationship between these mutations and treatment failure and patient outcome still needs to be investigated.

Table 19: Number of *Pneumocystis jirovecii* pneumonia (PCP) cases reported to GERMS-SA by province, South Africa, 2009-2010, n=669.

Province	2009	2010
Eastern Cape	37	22
Free State	19	10
Gauteng	141	160
KwaZulu-Natal	19	9
Limpopo	0	0
Mpumalanga	6	3
Northern Cape	0	1
North West	44	20
Western Cape	105	73
South Africa	**371**	**298**

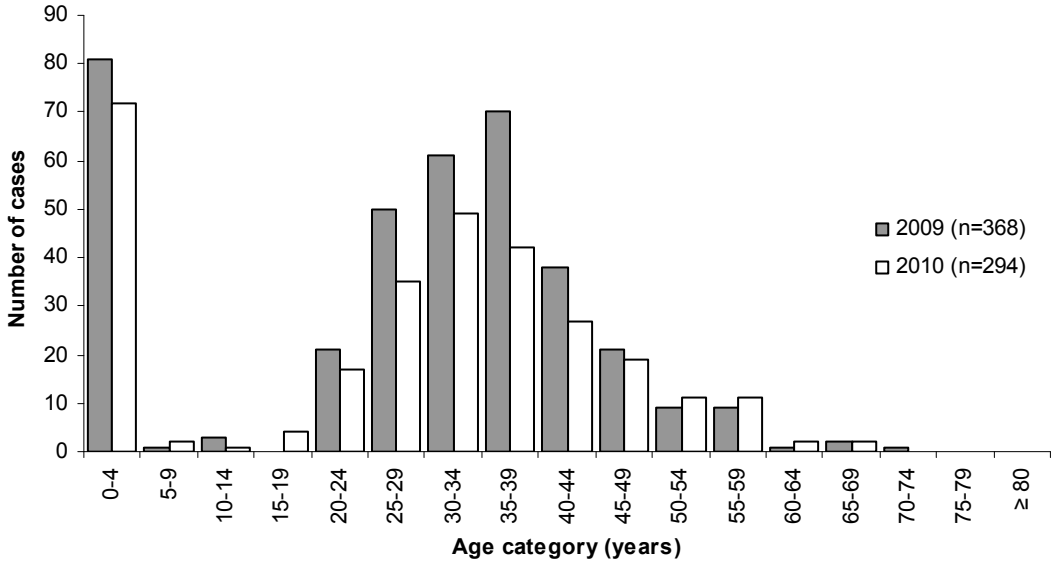
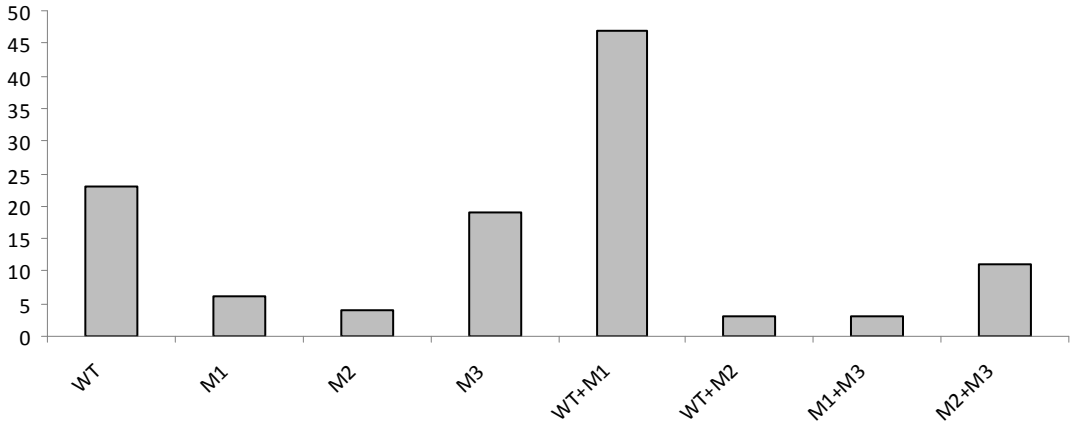


Figure 6: Number of laboratory-confirmed, *Pneumocystis jirovecii* pneumonia (PCP) cases reported to GERMS-SA, by age category, South Africa, 2009-2010, n=677.



WT: wild type genotype; M1: mutation at codon 55; M2: mutation at codon 57; M3: double mutation at codons 55 and 57; WT+M1, WT+M2, WT+M3, WT+M1+M2, M1+M3, M2+M3: genotype mixes

Figure 7: *Pneumocystis jirovecii* DHPS genotypes identified in specimens sent to Parasitology, NICD through the GERMS-SA network, 2010 (n=141)

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3. Crothers K, Beard CB, Turner J, Groner G, Fox M, Morris A et al. Severity and outcome of HIV-associated *Pneumocystis* pneumonia containing *Pneumocystis jirovecii* dihydropteroate synthase gene mutations. AIDS 2005 May; 19(8): 801-5.
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Report compiled by (in alphabetical order): Desiree du Plessis, John Frean and Bhavani Poonsamy

NEISSERIA MENINGITIDIS

Respiratory & Meningeal Pathogens Reference Unit, National Institute for Communicable Diseases

Results

In 2010, 366 cases of meningococcal disease were reported, and an additional 38 cases were identified on audit: a total of 404 cases of laboratory-confirmed meningococcal disease was identified by the surveillance system during the year (Table 20). The number of cases reported increased during the winter and spring months (Figure 8). Of all cases reported, cerebrospinal fluid (CSF) was the most common specimen yielding meningococci (Table 21), and the number of cases diagnosed on blood culture remained similar in 2010 compared to 2009 (p=0.1). Cases of W135 disease were reported from all provinces, and this serogroup was the most predominant in South Africa (159/334, 48%) (Table 22), but the proportion decreased from 2009 (235/397, 59%; p=0.002). Minor year-on-year fluctuations of disease by province were noted, for example there was a more than 50% reduction of disease incidence in Mpumulanga and a doubling of disease incidence in the Northern Cape. However, for both these provinces, this represented a small number of cases. In Gauteng, the incidence of meningococcal disease was estimated at 1.67 cases per 100 000 population, and most of that disease was due to serogroup W135 (92/161, 57%). The preponderance of serogroup B disease in Western Cape was still noted: 33/61 (54%) of all isolates serogrouped. Risk of disease was greatest amongst children less than five years of age. Age and serogroup-specific incidence rates show that infants were at greatest risk of disease for the three most common serogroups (Figure 9). Preliminary analysis of case-fatality ratios, as calculated at enhanced surveillance sites where in-hospital outcome is specifically looked for, was 27/158 (17%) in 2010, compared to 24/157 (15%) in 2009 (p=0.7). Of the viable isolates tested for antimicrobial resistance, 4/229 (2%) isolates had penicillin minimum inhibitory concentrations (MICs) >0.06µg/ml, and would be considered intermediately resistant.

Table 20: Number of cases and incidence rates of meningococcal disease reported to GERMS-SA by province, South Africa, 2009 and 2010, n=866 (including audit cases).

Province	2009		2010	
	n	Incidence rate*	n	Incidence rate*
Eastern Cape	36	0.5	31	0.5
Free State	18	0.6	26	0.9
Gauteng	203	1.9	187	1.7
KwaZulu-Natal	32	0.3	22	0.2
Limpopo	3	0.1	13	0.2
Mpumalanga	67	1.9	28	0.8
Northern Cape	9	0.8	20	1.8
North West	19	0.6	11	0.3
Western Cape	75	1.4	66	1.3
South Africa	**462**	**0.9**	**404**	**0.8**

*Incidence rates were calculated based on population denominators provided by Statistics South Africa, and are expressed as cases per 100 000 population.

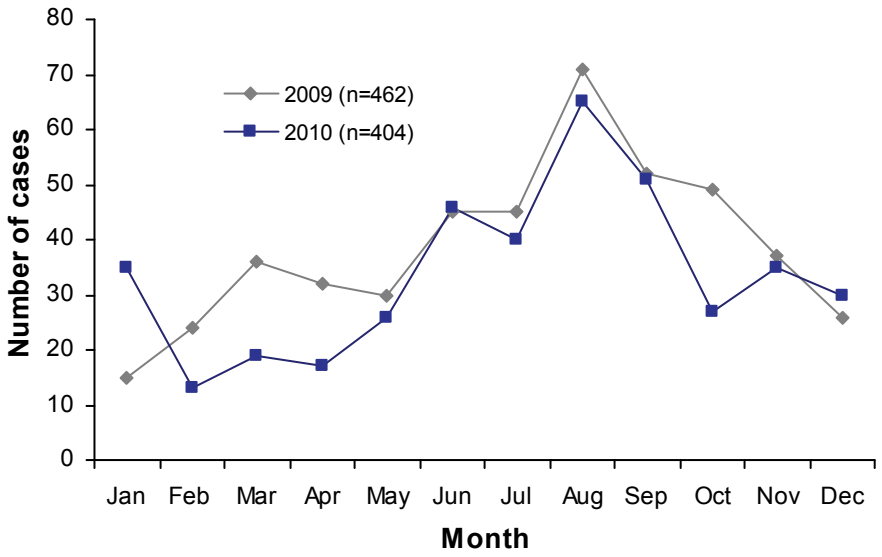

Figure 8: Number of laboratory-confirmed, invasive, meningococcal cases, reported to GERMS-SA, by month and year, South Africa, 2009-2010, n=866.

Table 21: Number and percentage of cases of meningococcal disease reported to GERMS-SA by specimen type, South Africa, 2009 and 2010, n=866.

Site of specimen	2009		2010	
	n	%	n	%
CSF	336	73	312	77
Blood	124	27	91	23
Other	2	0.4	1	0.2
	462		**404**	

Table 22: Number of cases of invasive meningococcal disease reported to GERMS-SA by serogroup and province, South Africa, 2010, n=404*.

Province	Serogroup							
	Serogroup not available	A	B	C	W135	X	Y	Total
Eastern Cape	5	0	7	2	15	0	2	31
Free State	10	0	9	1	4	0	2	26
Gauteng	26	3	38	10	92	2	16	187
KwaZulu-Natal	2	0	4	4	8	0	4	22
Limpopo	6	0	1	0	5	0	1	13
Mpumalanga	7	0	3	0	16	0	2	28
Northern Cape	6	0	2	2	3	0	7	20
North West	3	0	3	2	2	0	1	11
Western Cape	5	0	33	4	14	0	10	66
South Africa	70	3	100	25	159	2	45	404

*334 (83%) with specimens or viable isolates available for serogrouping

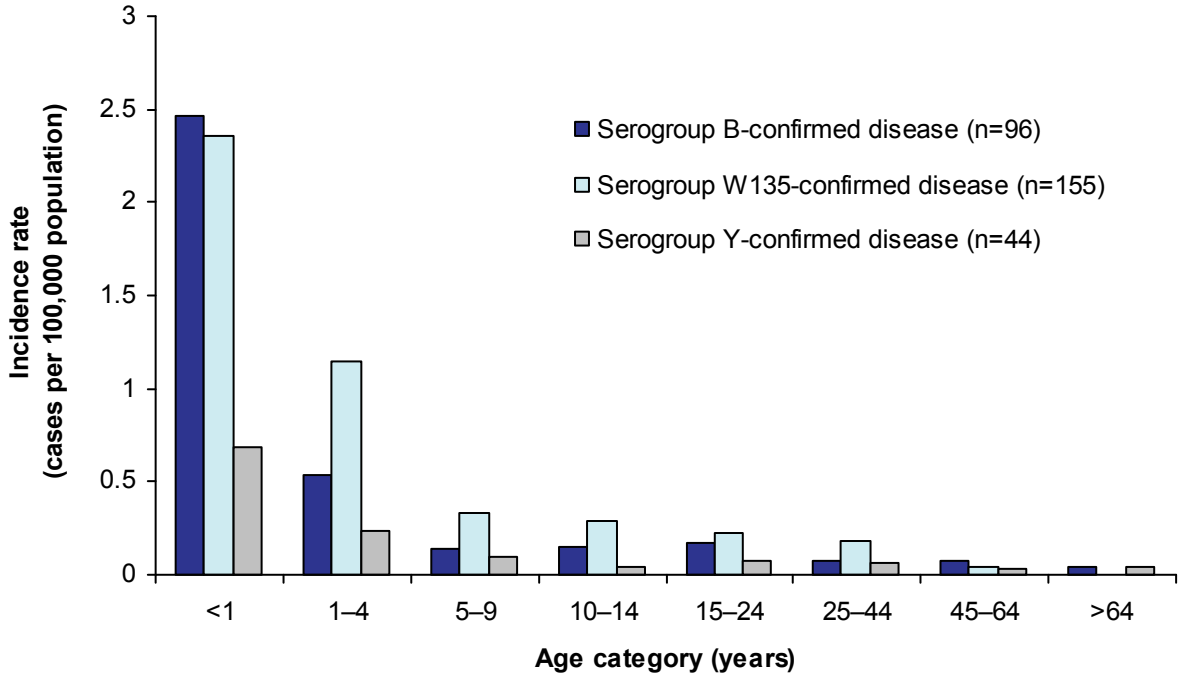

Figure 9: Age-specific incidence rates for laboratory-confirmed, invasive, meningococcal cases, by serogroup, South

Discussion

Overall incidence of disease did not change from 2009 and serogroup W135 disease decreased but remained the predominant serogroup. Changes in meningococcal disease incidence in provinces may reflect improved laboratory confirmation of disease and better reporting to the surveillance network, or may reflect a true increase in incidence. Case-fatality ratios have remained similar compared to 2009. The prevalence of intermediate resistance to penicillin remained low in 2010. The clinical relevance of increased MICs is unclear, and penicillin is, at present, still being recommended as the drug of choice for therapy for confirmed meningococcal disease.

Report compiled (in alphabetical order) by Linda de Gouveia and Anne von Gottberg

HAEMOPHILUS INFLUENZAE

Respiratory & Meningeal Pathogens Reference Unit, National Institute for Communicable Diseases

Results

The number of cases of *Haemophilus influenzae* invasive disease reported in 2010 was 313, while an additional 91 cases were identified during the national audit (total number of cases available for analysis was 404). Of these, 294 (73%) had isolates or specimens available for serotyping, and 123/294 (42%) were confirmed as serotype b (Table 23). Serotype b isolates were more likely to be isolated from CSF than non-typeable *H. influenzae* (81/123, 66% vs. 12/125, 10%, p<0.001) (Table 24). In 2010, a total of 82 cases of *H. influenzae* serotype b (Hib) were reported amongst children <5 years (Figure 10). Serotype b was the more common *H. influenzae* causing disease amongst infants (Figure 11). Rates of Hib disease as recorded by our surveillance network amongst infants <1 year of age were similar in 2010 as compared to 2009 (p=0.8) (Figure 12). Twenty percent of serotype b strains were non-susceptible to ampicillin (MIC>1mg/L, all producing beta lactamase), 17 of 85 isolates tested, while 12% (10/85) of non-typeable strains were non-susceptible (p=0.1).

Table 23: Number of cases of invasive *Haemophilus influenzae* disease reported to GERMS-SA by serotype and province, South Africa, 2010, n=404*.

Province	Serotype								
	Serotype not available	a	b	c	d	e	f	Non-typeable	Total
Eastern Cape	30	0	10	0	0	0	0	4	44
Free State	9	1	10	0	1	0	1	3	25
Gauteng	33	4	44	1	2	3	9	71	167
KwaZulu-Natal	2	0	18	0	1	1	1	8	31
Limpopo	2	1	4	0	0	0	0	3	10
Mpumalanga	5	1	9	0	0	0	1	0	16
Northern Cape	1	0	7	0	0	1	0	2	11
North West	3	0	3	0	0	0	1	1	8
Western Cape	25	8	18	0	1	1	6	33	92
South Africa	**110**	**15**	**123**	**1**	**5**	**6**	**19**	**125**	**404**

*294 (73%) with specimens or viable isolates available for serotyping.

Table 24: Number and percentage of cases of invasive *Haemophilus influenzae* disease reported to GERMS-SA by specimen type, South Africa, 2010, n=404.

Site of specimen	No serotype available		Serotype b		Serotypes a, c, d, e, f		Non-typeable	
	n	%	n	%	n	%	n	%
CSF	20	18	81	66	13	28	12	10
Blood	46	42	40	33	31	67	96	77
Other	44	40	2	2	2	4	17	14
Total	**110**		**123**		**46**		**125**	

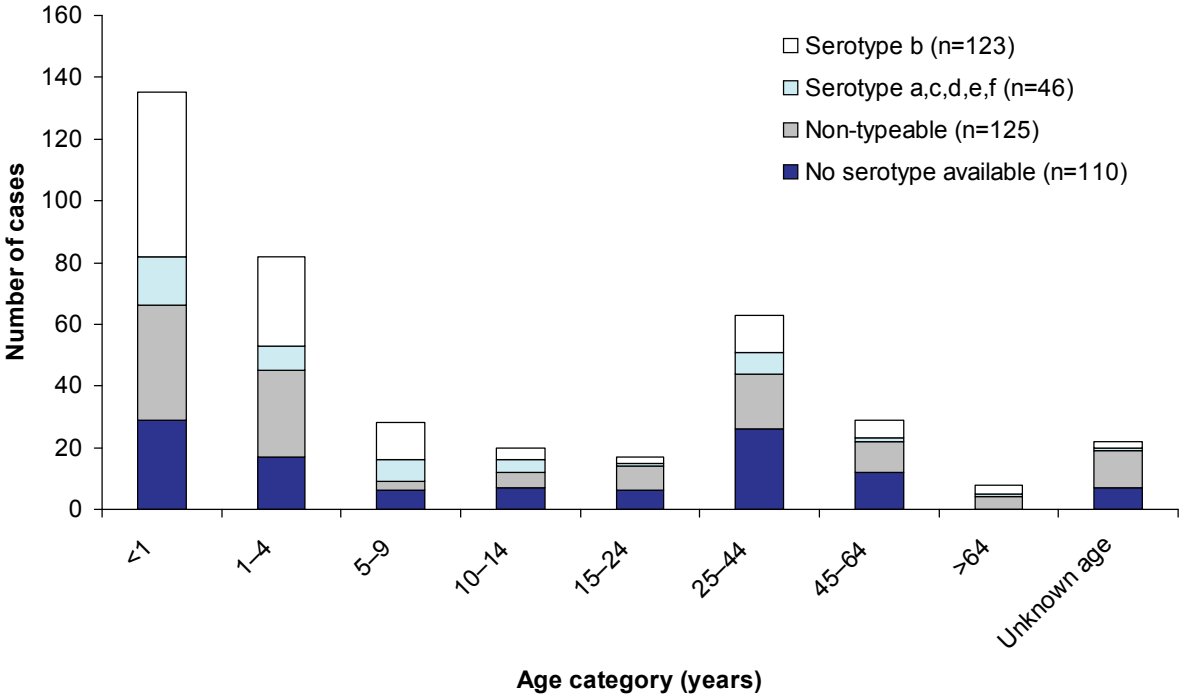

Figure 10: Number of laboratory-confirmed, invasive, *Haemophilus influenzae* cases, reported to GERMS-SA, by serotype and age group, South Africa, 2010, n=404 (age unknown for n=22; specimens or viable isolates unavailable for serotyping for n=110).

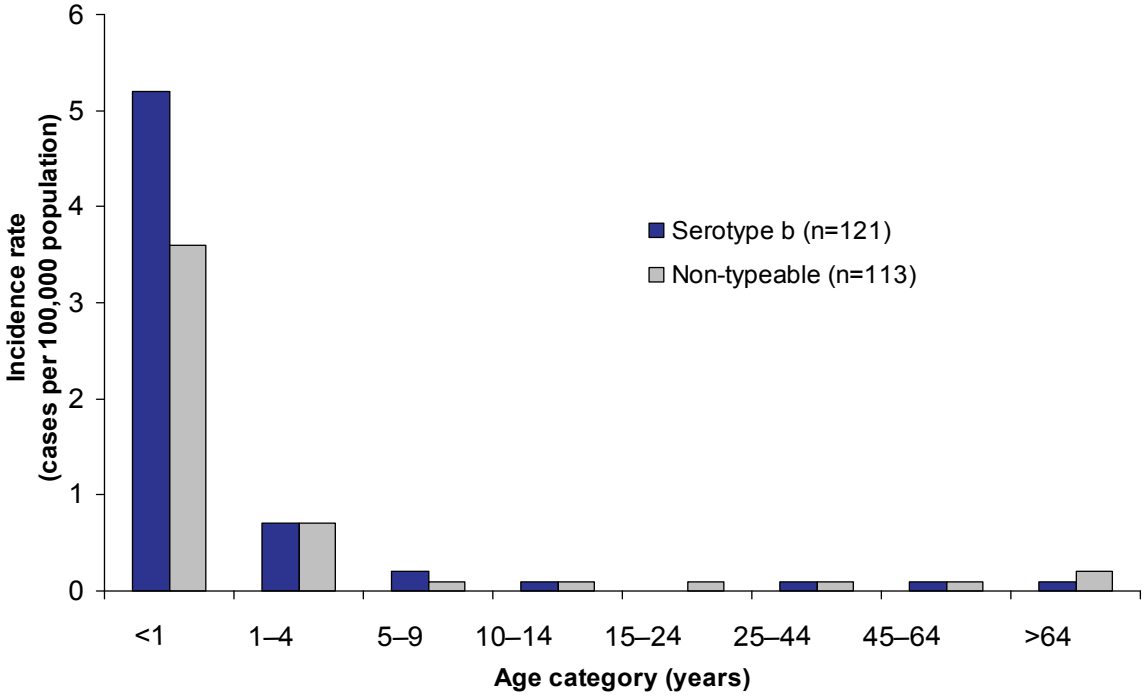

Figure 11: Age-specific incidence rates for laboratory-confirmed, invasive *Haemophilus influenzae* disease, reported to GERMS-SA, by serotype, South Africa, 2010, n=404 (age unknown for n=22; viable isolates unavailable for serotyping for n=110).

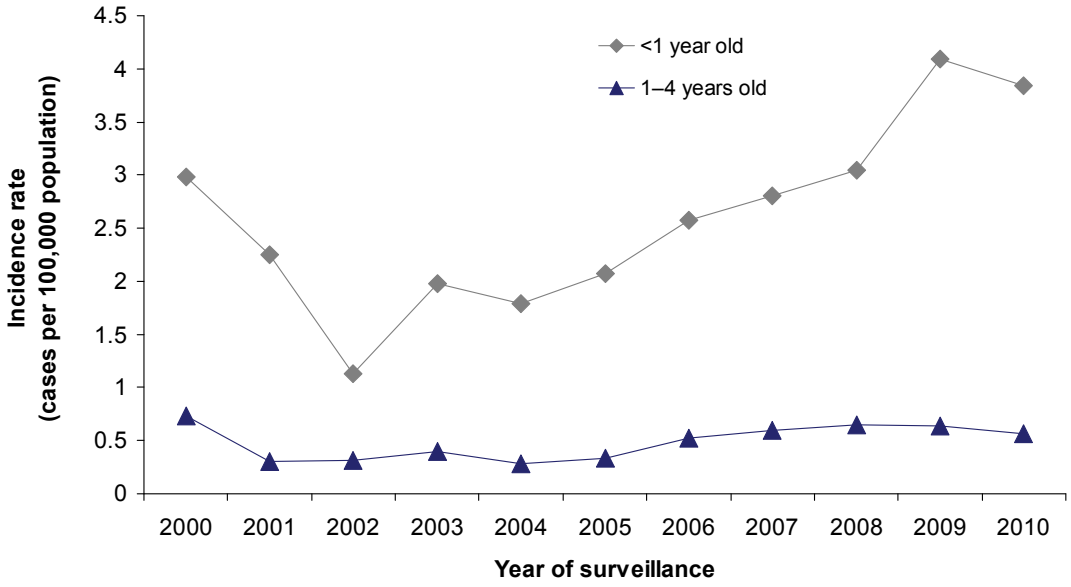

Figure 12: Incidence rates of laboratory-confirmed, *Haemophilus influenzae* serotype b disease, reported to GERMS-SA, in children <5 years old, South Africa, 2000-2010 (excluding cases identified using polymerase chain reaction (PCR) on specimens which was only done 2007-2010).

Discussion

Since the introduction of the 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. Population-based studies in South Africa before the introduction of the conjugate Hib vaccine had demonstrated annual rates of invasive Hib disease of 170 per 100 000 infants below one year of age and any increases noted recently were small in comparison to the substantial decline in disease subsequent to the introduction of the vaccine.[1,2] Recognising that our surveillance system underestimates disease, reported cases of Hib disease amongst children <1 year are being monitored carefully. In April 2009, the updated infant vaccination programme in South Africa introduced a booster dose of conjugate Hib vaccine given at 18 months as part of a combination vaccine (Pentaxim: diphtheria-tetanus-acellular pertussis-inactivated poliovirus-*Haemophilus influenzae* type-b conjugate). It is hoped that this booster will improve long-term protection against disease and impact on ongoing Hib transmission in the community. However it is too early to comment on the stabilisation of rates of Hib in children <1 year comparing 2010 to 2009, and we urge clinical and laboratory staff to continue reporting all cases of *H. influenzae.*

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Report compiled (in alphabetical order) by Linda de Gouveia and Anne von Gottberg

STREPTOCOCCUS PNEUMONIAE

Respiratory & Meningeal Pathogens Reference Unit, National Institute for Communicable Diseases

Results

Incidence of reported invasive pneumococcal disease (IPD) varied widely by province (Table 25). The age group at highest risk of disease in South Africa was infants <1 year of age, and there was an ongoing significant reduction in disease comparing 2010 to 2009, p<0.001 (Figure 13). The majority of episodes reported to GERMS-SA were diagnosed from positive blood culture specimens (Table 26). Penicillin non-susceptible isolates (MIC>0.06mg/L), have remained stable (1478/3389, 44% in 2009 compared to 1204/2857, 42% in 2010, p=0.2). Prevalence of non-susceptible strains ranged from 29% to 52% in different provinces (Table 27). Penicillin non-susceptible isolates were common amongst children less than 5 years of age (Figure 14). Ceftriaxone non-susceptibility was detected amongst 8% (225/2855) of all IPD cases, and in 7% (73/1094) of isolates detected from CSF specimens. Prevenar (7-valent conjugate pneumococcal vaccine, PCV7) was introduced into the Expanded Programme on Immunisations (EPI) in South Africa from 1 April 2009. The number of cases amongst children less than 5 years of age due to common serotypes in 2009 (including the seven serotypes in PCV7: 4, 6B, 9V, 14, 18C, 19F and 23F) are compared with 2009 in Figure 15. The percentage of disease in 2010 amongst children <5 years due to PCV7 and newer valency vaccine formulations are shown in Table 28.

Table 25: Number of cases and incidence rates of invasive pneumococcal disease reported to GERMS-SA by province, South Africa, 2009 and 2010, n=8975.

Province	2009		2010	
	n	Incidence rate*	n	Incidence rate*
Eastern Cape	362	5.4	388	5.8
Free State	308	10.6	318	11.3
Gauteng	2256	21.4	1847	16.5
KwaZulu-Natal	529	5.1	426	4.0
Limpopo	111	2.1	109	2.0
Mpumalanga	301	8.4	241	6.7
Northern Cape	88	7.7	105	9.5
North West	175	5.1	183	5.7
Western Cape	639	11.9	589	11.3
South Africa	**4769**	**9.7**	**4206**	**8.4**

*Incidence rates were calculated based on population denominators provided by Statistics South Africa, and are expressed as cases per 100,000 population.

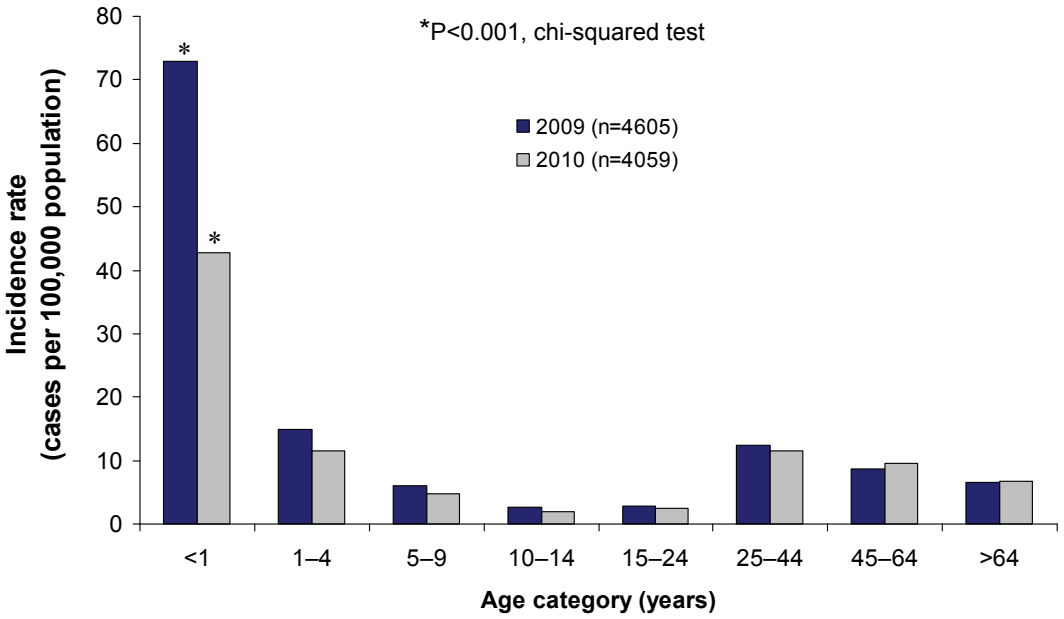

Figure 13: Age-specific incidence rates for laboratory-confirmed, invasive pneumococcal disease, reported to GERMS-SA, South Africa, 2009 and 2010 (2008: n=4769; age unknown for n=164; 2009: n=4206; age unknown for n=147).

Table 26: Number and percentage of cases of invasive pneumococcal disease reported to GERMS-SA by specimen type, South Africa, 2009 and 2010, n=8975.

Site of specimen	2009		2010	
	n	%	n	%
CSF	1800	38	1709	41
Blood	2517	53	2025	48
Other	452	9	472	11
	4769		**4206**	

Table 27: Number and percentage of penicillin non-susceptible isolates from invasive pneumococcal disease cases reported to GERMS-SA by province, South Africa, 2010, n=4206.

Province	Isolate not available	Susceptible*		Intermediate*		Resistant*	
	n	n	%	n	%	n	%
Eastern Cape	164	128	57	84	38	12	5
Free State	107	131	62	72	34	8	4
Gauteng	616	715	58	404	33	112	9
KwaZulu-Natal	68	200	56	133	37	25	7
Limpopo	45	40	63	17	27	7	11
Mpumalanga	128	75	66	34	30	4	4
Northern Cape	23	39	48	31	38	12	15
North West	91	65	71	25	27	2	2
Western Cape	107	260	54	172	36	50	10
South Africa	**1349**	**1653**	**58**	**972**	**34**	**232**	**8**

*2009 CLSI breakpoints for penicillin (oral penicillin V) were used: susceptible, ≤0.06mg/L; intermediately resistant, 0.12-1mg/L; resistant, ≥2mg/L.

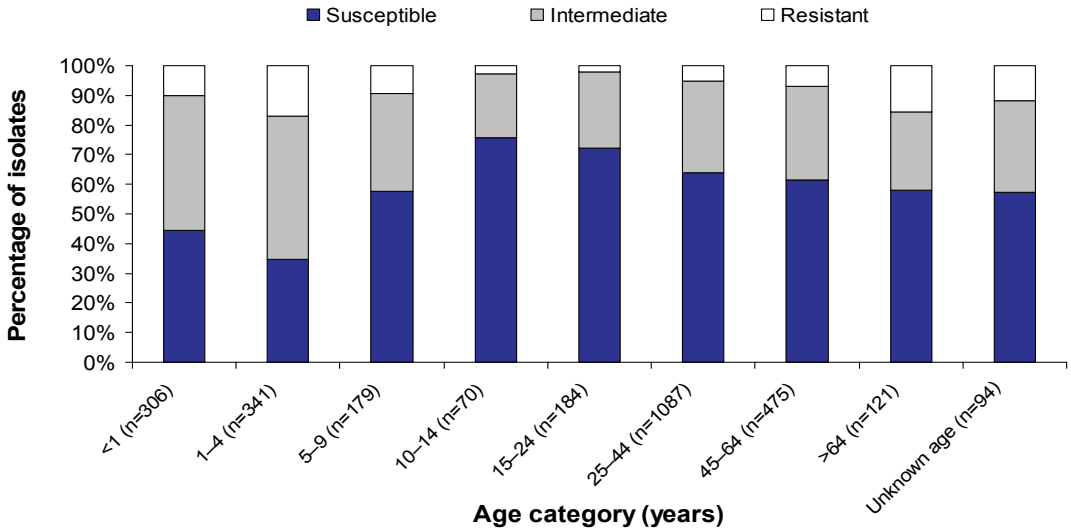

Figure 14: Number of laboratory-confirmed, invasive pneumococcal disease cases, reported to GERMS-SA, by age group and penicillin susceptibility, South Africa, 2010, n=4206 (n=2857 with viable isolates).

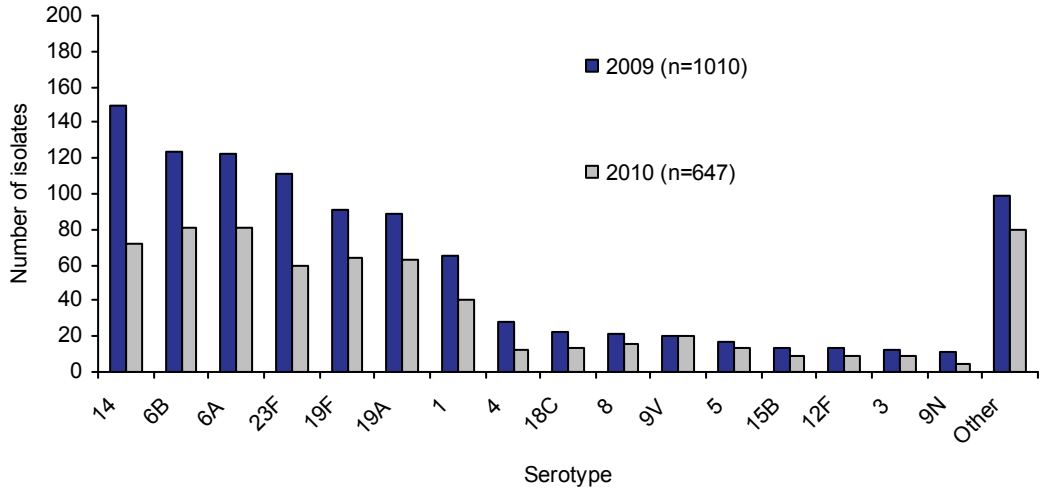

Figure 15: Pneumoccocal serotypes, in descending order, causing laboratory-confirmed, invasive pneumococcal disease, reported to GERMS-SA, in children <5 years, South Africa, 2009-2010 (2009: n=1338, n=1010 with viable isolates; 2010: n=907; n=647 with viable isolates).

Table 28: Number and percentage of invasive pneumococcal cases reported amongst children less than 5 years of age caused by the serotypes contained in the 7-valent, 10-valent and 13-valent pneumococcal, conjugate vaccines, South Africa, 2010, n=907 (n=647 with viable isolates).

Province	Total isolates available for serotyping	7-valent serotypes *		Serotype 6A#		10-valent serotypes*		13-valent serotypes*	
		n	%	n	%	n	%	n	%
Eastern Cape	45	22	49	6	13	27	60	37	82
Free State	32	19	59	3	9	22	69	27	84
Gauteng	279	135	48	36	13	160	57	227	81
KwaZulu-Natal	96	41	43	10	10	50	52	73	76
Limpopo	12	8	67	0	0	9	75	9	75
Mpumalanga	22	11	50	5	23	11	50	18	82
Northern Cape	32	16	50	3	9	19	59	25	78
North West	15	7	47	3	20	11	73	14	93
Western Cape	114	64	56	15	13	68	60	100	88
South Africa	647	323	50	81	13	377	58	530	82

*7-valent serotypes: 4, 6B, 9V, 14, 18C, 19F, 23F; 10-valent serotypes: 4, 6B, 9V, 14, 18C, 19F, 23F, 1, 5, 7F; 13 -valent serotypes: 4, 6B, 9V, 14, 18C, 19F, 23F, 1, 5, 7F, 19A, 3, 6A.
Cross-protection with 6B has been demonstrated.[1]

Discussion

Differences in IPD incidence by province have been documented for several years, and are partly due to differences in specimen-taking practices and laboratory reporting, however real differences in disease incidence cannot be excluded. The decrease in incidence of disease in children <1 year of age is mostly likely due to the introduction of PCV7 in South Africa. Our data for 2010 show similar prevalences of pneumococcal resistance to penicillin and ceftriaxone compared with 2009. The low levels of penicillin non-susceptibility from blood culture specimens still support the use of penicillin as first-line therapy for community-acquired pneumonia. Vancomycin, together with ceftriaxone, should be considered for the empiric treatment of suspected pneumococcal meningitis (CSF specimens positive for Gram-positive cocci or latex agglutination tests positive for *S. pneumoniae*), especially amongst unvaccinated children. As ceftriaxone-resistant isolates are likely to be serotypes contained in PCV7, we anticipate that the number of resistant isolates causing disease will decrease with wider use of the vaccine.

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Report compiled (in alphabetical order) by Linda de Gouveia and Anne von Gottberg

KLEBSIELLA PNEUMONIAE

Antimicrobial Resistance Reference Unit, National Institute for Communicable Diseases

Results

From July through December 2010, 519 cases of *Klebsiella pneumoniae* bloodstream infections were reported, and an additional 452 cases were identified on audit: a total of 971 cases of laboratory-confirmed bacteraemia caused by *K. pneumoniae* were identified (Table 29). The highest number of cases (n=649; 67%) was detected from Gauteng province (Table 29). Most cases of bacteraemia occurred amongst adults (Figure 16).The highest number of cases was detected during December 2010 (Figure 17). Of the viable *K. pneumoniae* isolates tested for antimicrobial resistance, 295/475 (62%) were extended spectrum β-lactamase (ESBL) producers. The percentage of isolates which were ESBL-producing varied by province (Gauteng, 141/248 (57%) vs. Free State, 37/46 (80%)) (Figure 18).

Table 29: Number of *Klebsiella pneumoniae* cases reported to GERMS-SA sentinel sites by province, South Africa, July-December 2010, n=971 (including audit cases)

Province	*Klebsiella pneumoniae*
Free State	82
Gauteng	649
KwaZulu-Natal	36
Limpopo	13
Western Cape	191
All sentinel sites	**971**

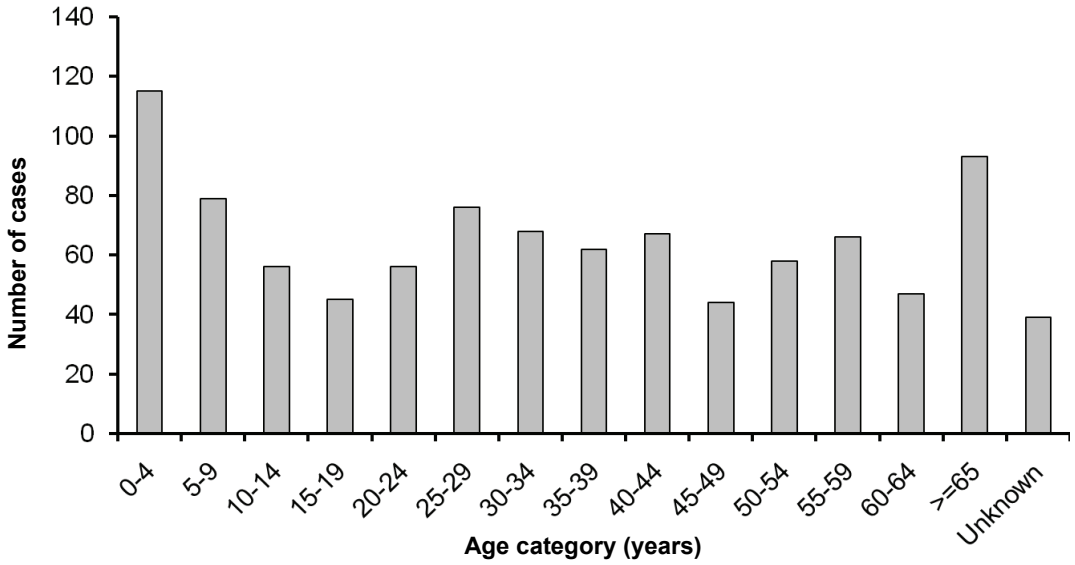


Figure 16: Number of cases of laboratory-confirmed *Klebsiella pneumoniae* bacteraemia reported to GERMS-SA sentinel sites by age category, July- December 2010, n=971.

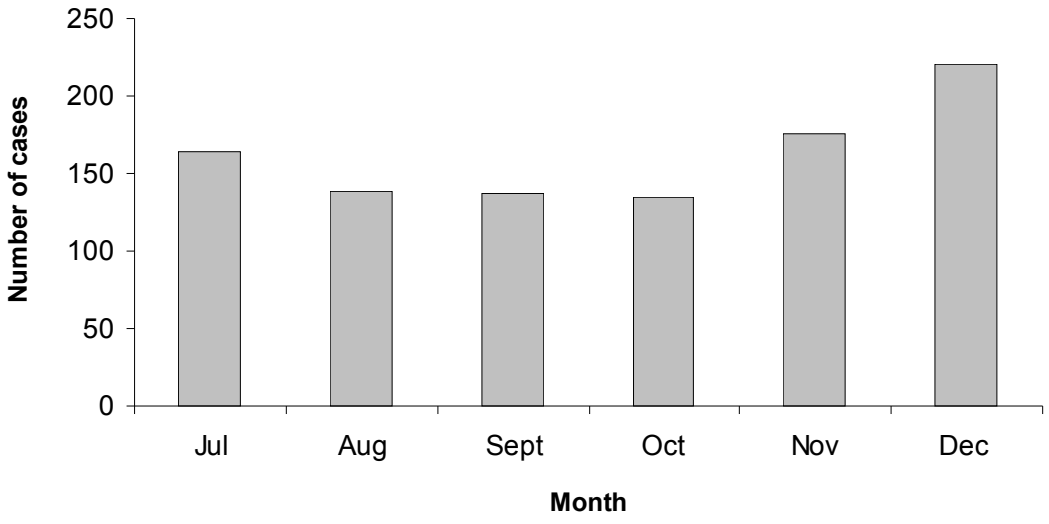
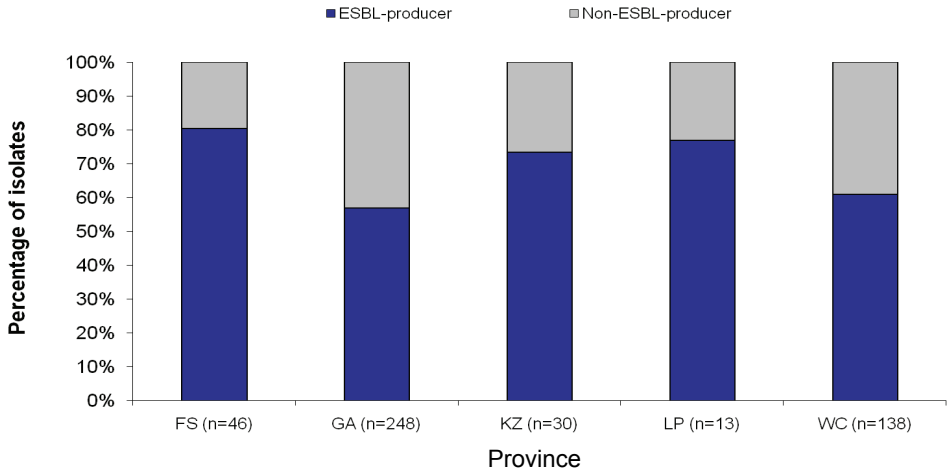


Figure 17: Number of cases of laboratory-confirmed *Klebsiella pneumoniae* bacteraemia reported to GERMS-SA sentinel sites by month, July- December 2010, n=971.



*Sentinel sites may have preferentially submitted antimicrobial-resistant isolates
FS—Free State, GA—Gauteng, KZ—KwaZulu Natal, LP—Limpopo, WC—Western Cape

Figure 18: Number of viable, laboratory-confirmed *Klebsiella pneumoniae* isolates reported by GERMS-SA sentinel sites*, by province and ESBL production, July-December 2010, n=478.

Discussion

Sentinel surveillance for *K. pneumoniae* bacteraemia was initiated in July 2010 through GERMS-SA. Incidence has not been reported. In the start-up phase, over half of the detected cases were only identified through audit; isolates were not submitted for these cases. It is important to recognise that there may have been an inherent selection bias – laboratories may have selectively reported cases with antimicrobial-resistant isolates. Amongst the submitted isolates, almost two-thirds were ESBL producers. Most ESBL-producing isolates were submitted from Free State and Western Cape laboratories.

Report compiled by Olga Perovic

STAPHYLOCOCCUS AUREUS

Antimicrobial Resistance Reference Unit, National Institute for Communicable Diseases

Results

The number of cases of *Staphylococcus aureus* bacteraemia reported to the GERMS-SA from July through December 2010 was 506 while an additional 280 cases (36%) were identified during an audit (total number of cases available for analysis was 786) (Table 30). Of these, the majority of cases were detected from sentinel sites in Gauteng (Table 30). The highest number of cases (n=177) was detected in July 2010 (Figure 19). Most cases (577/786, 73%) occurred amongst patients aged >15 years (Figure 20). Resistance to oxacillin was determined for a subset of isolates (n=348) from 6 sentinel sites; the percentage of isolates which were methicillin-resistant *Staphylococcus aureus* (MRSA): Free State (11//24, 46%), Gauteng (78/182, 43%) and Western Cape (74/179, 41%) (Figure 21).

Discussion

Incidence of *S. aureus* bacteraemia was not calculated. In addition, cases could not be separated into hospital- versus community-acquired categories because only laboratory-based data were available. Most cases of S. *aureus* bacteraemia occurred amongst adult patients. The percentage of *S. aureus* isolates which were MRSA was almost certainly biased by isolate submission practices at some sentinel sites (laboratories may have selectively reported cases with antimicrobial-resistant isolates).

Table 30: Number of *Staphylococcus aureus* cases reported to GERMS-SA sentinel sites by province, South Africa, July-December 2010, n=786 (including audit cases)

Province	*Staphylococcus aureus*
Free State	40
Gauteng	510
KwaZulu-Natal	26
Limpopo	3
Western Cape	207
All sentinel sites	**786**

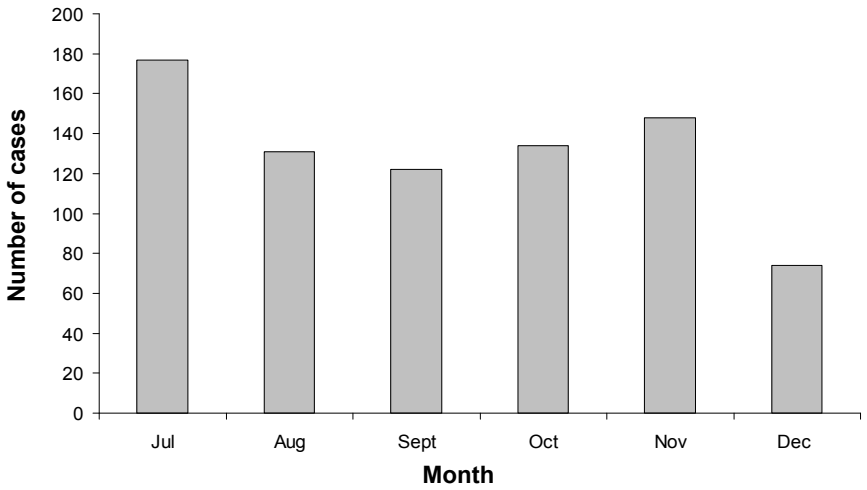


Figure 19: Number of cases of laboratory-confirmed *Staphylococcus aureus* bacteraemia reported to GERMS-SA sentinel sites by month, July- December 2010, n=786.

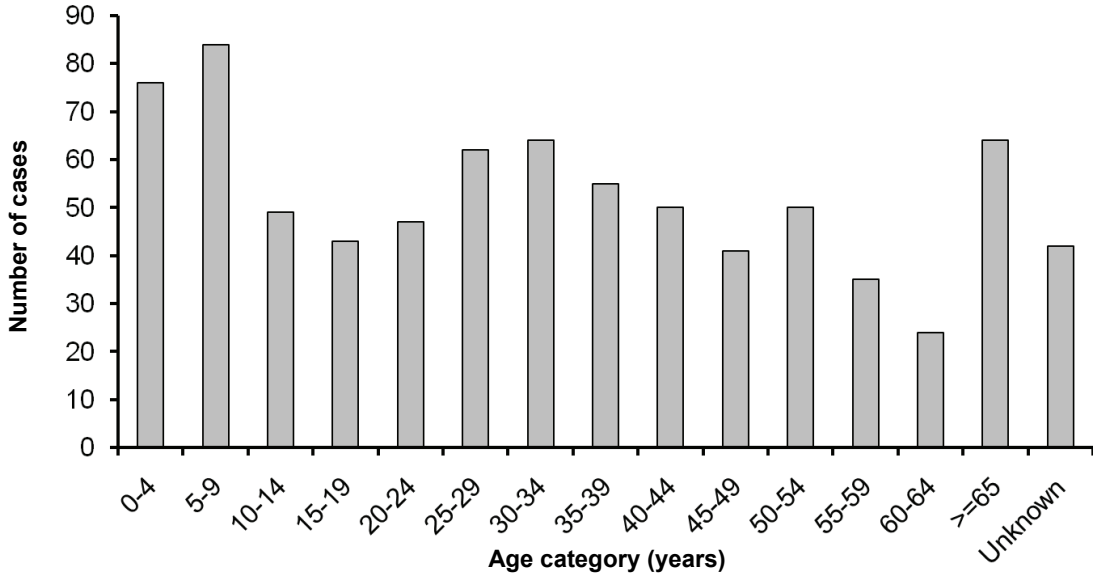


Figure 20: Number of cases of laboratory-confirmed *Staphylococcus aureus* bacteraemia reported to GERMS-SA sentinel sites by age category, July- December 2010, n=786.

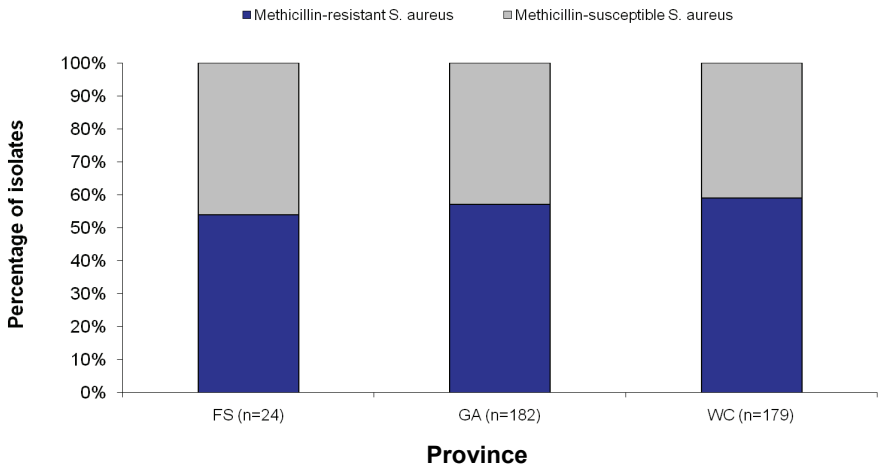


Figure 21: Number of viable, laboratory-confirmed *Staphylococcus aureus* isolates reported by GERMS-SA sentinel sites, by province and oxacillin resistance, July-December 2010, n=385.

Report compiled by Olga Perovic

Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 31 March 2010/2011*

Disease/Organism	Cumulative to 31 March, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Anthrax	2010	0	0	0	0	0	0	0	0	0	0
	2011	0	0	0	0	0	0	0	0	0	0
Botulism	2010	0	0	0	0	0	0	0	0	0	0
	2011	0	0	0	0	0	0	0	0	0	0
Cryptococcus spp.	2010	339	125	531	298	133	219	15	159	115	1934
	2011	329	94	465	246	131	168	15	162	123	1733
Haemophilus influenzae, invasive disease, all serotypes	2010	8	5	41	12	1	0	2	2	21	92
	2011	8	5	26	9	0	4	2	0	17	71
Haemophilus influenzae, invasive disease, < 5 years											
Serotype b	2010	0	1	4	1	0	0	1	1	3	11
	2011	0	1	4	3	0	0	1	0	2	11
Serotypes a,c,d,e,f	2010	0	0	1	0	1	0	0	0	3	5
	2011	0	1	2	0	0	0	0	0	0	3
Non-typeable (unencapsulated)	2010	0	0	13	3	0	0	0	0	5	21
	2011	0	1	2	3	0	1	0	0	2	9
No isolate available for serotyping	2010	3	1	5	0	0	0	0	0	1	10
	2011	2	2	8	0	0	2	1	0	2	17
Measles	2010	900	259	581	1984	177	923	150	474	1069	6517
	2011	1	1	27	12	1	0	7	5	5	59
Neisseria meningitidis, invasive disease	2010	7	6	24	4	2	4	5	2	13	67
	2011	11	1	30	2	2	5	1	0	7	59
***Novel Influenza A virus infections	2010	0	0	0	0	0	0	0	0	0	0
	2011	0	0	0	0	0	0	0	0	0	0
Plague	2010	0	0	0	0	0	0	0	0	0	0
	2011	0	0	0	0	0	0	0	0	0	0
Rabies	2010	1	0	0	1	3	1	0	0	0	6
	2011	0	0	0	0	2	0	0	0	0	2
**Rubella	2010	141	31	47	126	13	62	13	58	107	596
	2011	19	2	40	23	10	18	8	26	26	172
Salmonella spp. (not typhi), invasive disease	2010	11	3	74	15	3	5	2	1	20	134
	2011	15	9	97	22	0	14	3	4	20	184
Salmonella spp. (not typhi), isolate from non-sterile site	2010	50	14	211	52	1	24	3	15	35	405
	2011	63	12	234	38	4	22	11	13	76	473
Salmonella typhi	2010	2	0	11	5	0	4	0	0	2	24
	2011	5	2	8	4	0	3	0	0	5	27
Shigella dysenteriae 1	2010	0	0	0	0	0	0	0	0	0	0
	2011	0	0	0	0	0	0	0	0	0	0
Shigella spp. (Non Sd1)	2010	68	15	219	23	0	9	5	8	110	457
	2011	60	16	242	36	8	5	10	4	187	568
Streptococcus pneumoniae, invasive disease, all ages	2010	73	43	322	81	20	45	21	33	123	761
	2011	55	50	298	61	11	31	15	35	108	664
Streptococcus pneumoniae, invasive disease, < 5 years	2010	15	11	93	23	2	12	12	8	33	209
	2011	8	8	56	12	3	10	3	4	21	125
Vibrio cholerae O1	2010	0	0	0	0	0	0	0	0	0	0
	2011	0	0	0	0	0	0	0	0	0	0
Viral Haemorrhagic Fever (VHF)											
Crimean Congo Haemorrhagic Fever (CCHF)	2010	0	1	0	0	0	0	1	0	0	2
	2011	0	0	0	0	0	0	0	0	0	0
****Other VHF (not CCHF)	2010	7	69	0	0	0	0	11	0	0	87
	2011	7	2	0	0	0	0	2	0	5	16

Footnotes

*Numbers are for cases of all ages unless otherwise specified. Data presented are provisional cases reported to date and are updated from figures reported in previous bulletins.

**Rubella cases are diagnosed from specimens submitted for suspected measles cases.

*** Confirmed cases. Excludes pandemic influenza H1N1. See weekly influenza reports on www.nicd.ac.za.

**** All Rift Valley fever . For 2010 the total includes 1 case from an unknown province.
Provinces of South Africa: EC – Eastern Cape, FS – Free State, GA – Gauteng, KZ – KwaZulu-Natal, LP – Limpopo, MP – Mpumalanga, NC – Northern Cape, NW – North West, WC – Western Cape

U =unavailable, 0 = no cases reported

Table 2: Provisional laboratory indicators for NHLS and NICD, South Africa, corresponding periods 1 January - 31 March 2010/2011*

Programme and Indicator	Cumulative to 31 March, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Acute Flaccid Paralysis Surveillance											
Cases < 15 years of age from whom specimens received	2010	14	3	18	26	9	10	0	9	5	94
	2011	12	4	25	25	23	14	4	4	4	115
Laboratory Programme for the Comprehensive Care, Treatment and Management Programme for HIV and AIDS											
CD4 count tests											
Total CD4 count tests submitted	2010	104,726	71,065	205,053	265,677	71,132	79,235	16,343	64,023	59,594	936,848
	2011	117,309	57,210	220,686	289,528	77,029	92,118	17,565	72,350	72,426	1,016,221
Tests with CD4 count < 200/µl	2010	33,984	20,316	66,587	75,514	21,410	24,238	5,139	18,815	14,582	280,585
	2011	32,355	15,234	67,399	57,222	24,378	26,817	4,509	19,704	14,732	262,350
Viral load tests											
Total viral load tests submitted	2010	38,555	17,877	91,879	88,160	26,416	26,381	6,368	26,267	27,470	349,373
	2011	39,128	21,352	83,545	120,831	25,521	27,122	6,269	27,244	32,479	383,491
Tests with undetectable viral load	2010	25,624	11,977	67,980	65,609	19,168	20,467	3,860	18,583	21,465	254,733
	2011	25,996	16,835	58,035	92,113	17,949	18,650	4,286	17,964	25,238	277,066
Diagnostic HIV-1 PCR tests											
Total diagnostic HIV-1 PCR tests submitted	2010	7,902	3,544	15,599	20,920	5,155	5,919	1,153	4,452	4,311	68,955
	2011	8,795	3,870	16,707	20,318	5,715	6,574	1,323	5,096	4,644	73,042
Diagnostic HIV-1 PCR tests positive for HIV	2010	658	350	1,619	1,778	605	642	115	429	305	6,501
	2011	604	262	1,082	1,245	390	368	77	326	201	4,555

Footnotes

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