

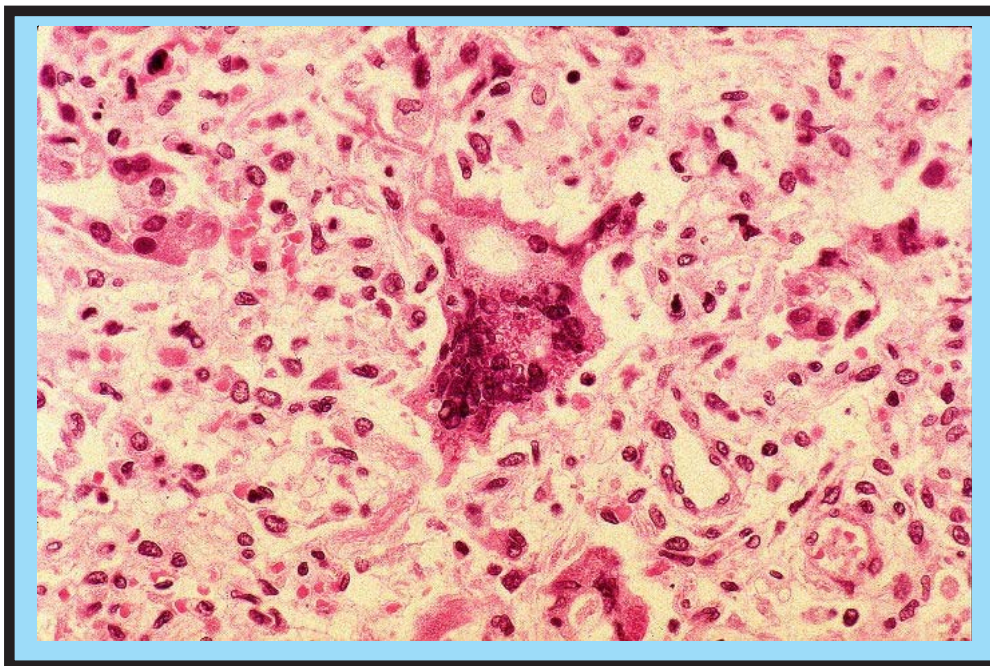


Communicable Diseases Surveillance Bulletin



January 2005

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Diseases (NICD)
of the National Health Laboratory Service (NHLS)**



*Histopathology of measles pneumonia. Giant cell with intracytoplasmic inclusions.
Source : CDC/Dr Edwin P Ewing, Jr.*

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EPIDEMIC PRONE DISEASE SURVEILLANCE : JANUARY-DECEMBER		CUMULATIVE	ECP	FSP	GAP	KZP	LPP	MPP	NCP	NWP	WCP	RSA
AFP, cases from whom specimens have been received	< = 15 years	2003 2004	30 24	13 17	37 28	39 36	72 56	15 14	5 5	25 25	24 25	260 230
	All ages	2003 2004	3 7	1 0	155 561	U 101	2 5	42 11	3 4	2 10	U 33	209 732
Rubella, IgM positive results from measles IgM negative patients	All ages	2003 2004	301 104	63 2	740 245	U 31	251 40	327 285	32 4	163 122	U 20	1877 853
	All ages	2003 2004	0 0	0 1	0 0	0 0	0 0	0 0	0 1	0 2	0 0	0 4
Rabies, human	All ages	2003 2004	1 0	0 0	0 0	9 6	0 0	0 1	0 0	1 0	0 0	14 7
	All ages	2003 2004	8 10	15 13	122 132	22 27	1 2	9 6	1 1	4 3	59 39	241 233
<i>Haemophilus influenzae</i> , invasive	Age < 5 years	2003 2004	1 1	0 0	14 5	1 1	0 0	0 0	0 0	1 0	8 3	25 10
		2003 2004	2 0	1 6	31 30	5 5	0 0	2 0	0 0	0 0	12 10	53 51
	Unknown serotype	2003 2004	3 3	8 2	5 11	10 9	0 0	1 1	1 1	0 1	14 7	42 35
		2003 2004	23 29	20 22	181 175	13 21	1 12	13 11	4 6	26 18	87 58	368 352
Meningococcal disease	All ages	2003 2004	92 160	108 217	2013 2019	255 487	46 68	129 178	14 21	146 113	426 485	3229 3748
	Age < 5 years	2003 2004	41 63	48 77	565 621	94 169	7 18	34 46	2 6	33 32	205 211	1029 1243
<i>Streptococcus pneumoniae</i> , invasive	Penicillin, non-susceptible, all ages	2003 2004	17 31	15 42	431 508	66 134	3 11	13 37	2 1	17 26	94 108	658 898
		2003 2004	21 12	8 24	167 237	41 36	8 10	10 19	0 0	35 9	45 50	335 397
	All ages	2003 2004	16 19	24 14	543 603	18 63	0 15	11 4	1 0	7 6	79 59	698 783
<i>Salmonella</i> species - enteric isolates	All ages	2003 2004	92 117	18 30	219 237	25 140	2 12	15 37	0 0	18 28	229 137	618 738
		2003 2004	2 12	0 0	19 18	2 7	7 9	15 7	0 0	1 0	4 8	50 61
<i>Shigella</i> species	All ages	2003 2004	124 110	28 42	155 214	62 123	12 12	29 33	0 0	7 16	270 344	687 894
		2003 2004	53 26	0 0	3 3	0 0	0 0	83 217	0 0	2 46	1 0	14 292

U = unavailable, 0 = no isolates received

Note: The above are NICD laboratory data and do not necessarily reflect a quantitative measure of disease in the country.

HUMAN RABIES CASES IN SOUTH AFRICA, 2004

Felicity Burt, Janusz Paweska & Lucille Blumberg, Special Pathogens Unit (SPU) & Epidemiology Unit, NICD

Two subtypes of rabies virus occur in southern Africa: canid (dog) virus, and vivverid (mongoose) virus. The canid virus causes epidemics in dogs in southern Mpumalanga, KwaZulu-Natal and the Eastern Cape. Most South African cases of human rabies result from dog-bite and occur in these areas. The vivverid virus occurs widely in the rest of the country and is responsible for sporadic cases of rabies in other animals and humans. Over the period 1928-2000 about 36.8% of South African cases in animals have occurred in dogs and 28.4% in vivverids (mongooses, civets and genets). Prompt post-exposure prophylaxis (PEP) using immunoglobulin and vaccine is effective against both viruses, but occasional failures have been reported where, for example, treatment has been delayed or incomplete, or patients are immune compromised.

The 7 cases of human rabies confirmed in South Africa during 2004 represent a significant decrease from the 11 cases confirmed in 2003, and is low in comparison to the 20-30 cases confirmed annually 10-15 years ago. This may reflect improved dog vaccination coverage as in 2004 a major dog vaccination campaign was launched in KwaZulu-Natal as well as a drive to improve PEP for human animal-bite victims in clinics and hospitals.

Of these cases, one occurred in Mpumalanga, where a 57 year old man succumbed to rabies a month after a water mongoose bite in his home in Standerton,

despite administration of adequate PEP. No specific reason for failure of PEP could be identified in this patient. The remaining six cases occurred in KwaZulu-Natal where dog rabies is most prevalent. One incident in the Harding district of KwaZulu-Natal involved 12 persons exposed to a rabid puppy. No PEP was administered to any of the exposed persons resulting in rabies-related deaths in two children.

One additional probable case of rabies that occurred in a dog-bite victim in KwaZulu-Natal is highly unusual as the child is still alive but remains severely neurologically impaired 9 months after the onset of clinical symptoms. Although the clinical features and history of exposure support a diagnosis of rabies, the laboratory diagnosis of rabies in this patient is supported only by the presence of rabies antibodies in the cerebrospinal fluid. There are likely to be a significant number of clinical cases in humans that remain unconfirmed due to failure to perform appropriate diagnostic tests.

Most patients in South Africa succumb within less than a week after admission to hospital, frequently within 24-48 hours, and the only other patients over the past 20 years observed to have undergone a protracted morbid period were two children in KwaZulu-Natal who received incomplete courses of post-exposure immunisation, as well as four similar cases documented in the international literature.

VIRAL HAEMORRHAGIC FEVER, 2004

Felicity Burt, Janusz Paweska & Lucille Blumberg, Special Pathogens Unit (SPU) & Epidemiology Unit, NICD

The Special Pathogens Unit of the National Institute for Communicable Diseases is responsible for the diagnosis and investigation of diseases associated with the formidable (biohazard class 4) viruses in southern Africa, and operates a maximum security (biohazard containment level 4) laboratory. Class 4 viruses known or considered likely to occur in Africa include Marburg, Ebola, Rift Valley fever (RVF), Crimean-Congo haemorrhagic fever (CCHF), Lassa fever, and hanta viruses. In South Africa, local transmission is only reported with CCHF and RVF.

The number of suspected cases of viral haemorrhagic fever (VHF) in southern Africa referred to the laboratory for investigation has fallen progressively in recent years and most likely relates to under diagnosis due to diminished clinical awareness. In 2003 there were no laboratory confirmed cases of CCHF in South Africa.

In 2004 CCHF was confirmed in 4 patients, of whom two were farmers from North West and the Northern Cape provinces respectively, one was a miner living on a farm in the North-West province, and the fourth a man who lived on the outskirts of Bloemfontein, Free State. Three patients developed disease following the bite of *Hyalomma* ticks, 1 patient was infected after dipping sheep and exposure could therefore be due to either sheep or ticks. Two of the patients died and in two cases, CCHF was only suspected post mortem. An additional case of fatal CCHF was confirmed in a farmer from Namibia following a tick bite,

Laboratory tests were conducted on a number of suspected cases of VHF who were subsequently confirmed to have other diseases, including malaria, meningococcal septicaemia, HIV with opportunistic infections, leukaemia, tick bite fever and herpes hepatitis.

RVF was confirmed in a Windhoek resident who presented with fever, hepatitis and thrombocytopenia following a visit to Katima Mulilo and Rundu in Namibia. RVF is endemic in the area, but no recent outbreaks have been reported. The last major outbreak in southern Africa occurred in 1974-1976 in South Africa and Namibia, and in 1978 in Zimbabwe.

The total number of cases of CCHF diagnosed in southern Africa from the time that the presence of the disease was first recognised in 1981 till the end of 2004 is 176, of which 1 case occurred in the Democratic Republic of the Congo, 1 in Tanzania, 16 in Namibia and 158 in South Africa. Marginally the largest group of South African cases, 77 (40.3%),

arose from a known tick bite or the squashing of ticks; a similar number, 71 (40.3%), arose from known or potential contact with fresh blood or other tissues of livestock and/or ticks; 7 (3.9%) nosocomial infections arose from contact with blood or fomites of known CCHF patients, while in 21 (11.9%) 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 constituted 147 (83.5%) of all cases. The case fatality rate fluctuated around 30% in the first few years after CCHF was initially recognized in southern Africa, but has declined to 26.7% (47/176) probably as a result of better case management.

SUSPECTED MEASLES CASE BASED SURVEILLANCE

Bernice Harris, Jo McAnerney, Epidemiology Unit, NICD

South Africa is in the eradication phase of measles control. The NICD is accredited by WHO to perform measles and rubella IgM testing for national case based surveillance to confirm true measles cases and trace the molecular epidemiology of the virus in South Africa. Blood and urine specimens from each suspected measles case are sent to the NICD for confirmation. Case investigation forms are completed by facility or district personnel and forwarded to the National Department of Health. The numbers presented here represent specimens received by the NICD and may differ from those of the National Department that include epidemiologically linked cases where no specimens were taken.

During 2004 the NICD tested 3 322 blood specimens from cases of rash and fever for suspected measles case based surveillance of which 51% were collected in Gauteng and 20% in Mpumalanga. Of these specimens 22% tested positive for measles and 26% for rubella (epidemic prone diseases table, page 2).

Measles

At the end of August and September 2004, a national mass polio and measles vaccination campaign for

children younger than 5 years of age was conducted in all provinces. Measles cases continued to occur as a vaccination coverage of 95% is needed to interrupt transmission.

95% of all the confirmed measles cases occurred in Gauteng (77%), KwaZulu-Natal (14%) and the Western Cape (5%). The NICD only started receiving specimens from KwaZulu-Natal from mid November 2004. The measles epidemiology in the respective provinces depended on the distribution of susceptibles resulting in a large prolonged outbreak in very young children in low income, high population density suburbs of Gauteng, two limited outbreaks in adults and older children in the Western Cape and an outbreak originating in an inaccessible, low vaccination coverage area in KwaZulu-Natal. Sporadic measles cases occurred throughout the year in Mpumalanga, North West and Limpopo. The Eastern Cape and Northern Cape experienced small isolated outbreaks. No cases were detected in the Free State.

Genotyping was performed on 192 specimens (1 CSF, 6 throat/nasal swabs, 25 sera, 159 urines) from IgM

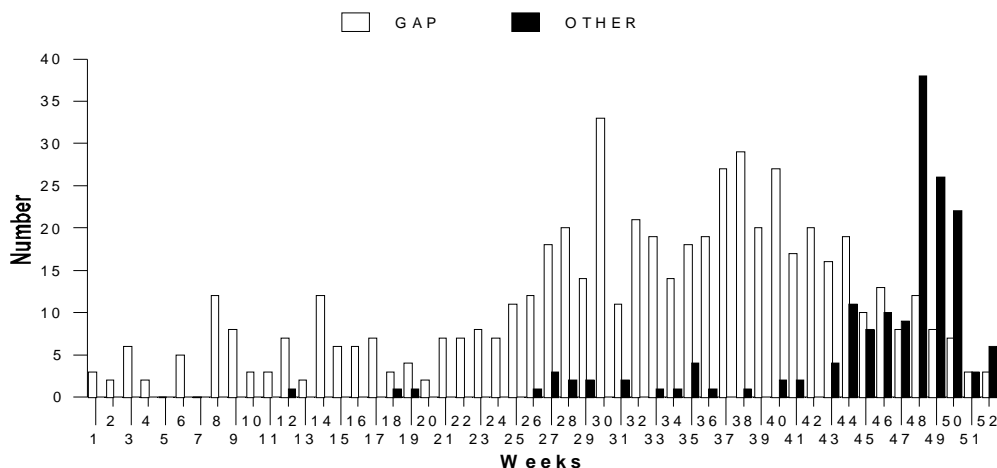


Fig 1: Confirmed measles cases by week of onset, South Africa, 2004

2003 N= 209

2004 N=720

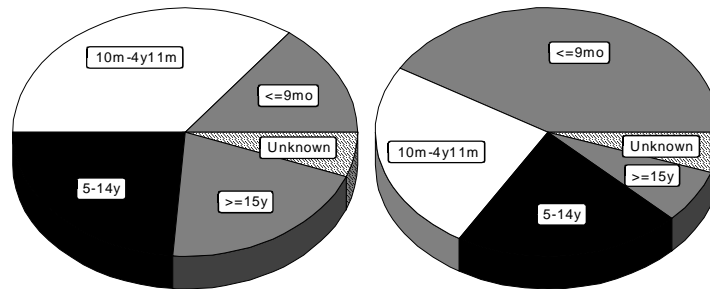


Fig 2: Age distribution of confirmed measles cases, South Africa, 2003 and 2004

positive measles cases. 110 specimens (57%) were positive by PCR and all were identified as D2 genotype.

Strategies employed to interrupt virus circulation included re-introducing immunisation of all children from 6 months to 15 years of age on admission to a health facility or institution to interrupt nosocomial spread, immunisation of contacts of sporadic cases, and extensive mop up campaigns in areas where cases were clustered. The age of the target populations for mop ups depended on the age distribution of cases and varied from all ages in an outbreak involving non-immune adults to children aged 6 months to 15 years in areas with wide spread cases.

In Gauteng, mop up campaigns mostly involved children aged 6 months to 5 years as the median age of cases was much lower than in the other provinces. The national age distribution of cases differed markedly from that of cases in 2003 with proportionally more cases aged ≤ 9 months and fewer older than 5 years in 2004 (figure 2).

Rubella

89% of all rubella cases occurred in Mpumalanga (33%), Gauteng (29%), Northwest (14%) and Eastern Cape (12%). The median age of cases was 7 years of age. Most cases occurred in the late winter and spring.

AFP SURVEILLANCE, 2004

Jo McAnerney, Bernice Harris, Epidemiology Unit, NICD

AFP surveillance, as part of the worldwide campaign to eradicate poliomyelitis, has continued throughout the year. All cases of acute flaccid paralysis in children < 15 years of age, including Guillain-Barré syndrome, or a patient of any age diagnosed as polio by a medical doctor must be regarded as possible polio cases until proven otherwise.

Although the last South African case of polio occurred in 1989, national polio free certification can only be achieved if we can demonstrate sufficient surveillance. This implies the ability to detect 1 AFP case per 100 000 children < 15 years of age (at least 147 cases during 2004), to have at least 80% of reports from AFP active surveillance sites throughout the country received on time, at least 80% of the AFP cases should have two stool specimens taken within 14 days of onset of paralysis at least 24 hours apart reaching the laboratory in good condition, and all virological investigations must be conducted in laboratories accredited by the Global Polio Laboratory Network.

In South Africa the only accredited laboratory is the NICD which also serves as national isolation laboratory for six other Southern African countries i.e.

Angola, Botswana, Lesotho, Mozambique, Namibia, and Swaziland.

During the year 1 118 stool specimens were received from AFP patients. Of these 95 were from patients with onset of paralysis prior to 2004, or patients who were subsequently considered not to have AFP. Of the remainder 480 were from South African cases and 543 from cases in the six other countries (figure 1). A further 32 specimens were received in the first two weeks of January 2005 from patients with onset of paralysis in 2004.

The provincial case detection rate for South Africa ranged from 1.0 to 2.7, with a national rate of 1.5. Two or more specimens taken within 14 days of onset were received from 174/230 patients. The percentage of adequate stool specimens per province ranged from 47.2% to 87.5% with an national rate of 75.7% (figure 2).

Non-polio enteroviruses were isolated from one or more specimens of 27 South African and 62 non-South African cases. Sabin-like poliovirus was isolated from 21 specimens of 12 South African patients and from 11 non-South African cases. In a seven year old boy from Botswana the isolate was identified as wild type polio 1.

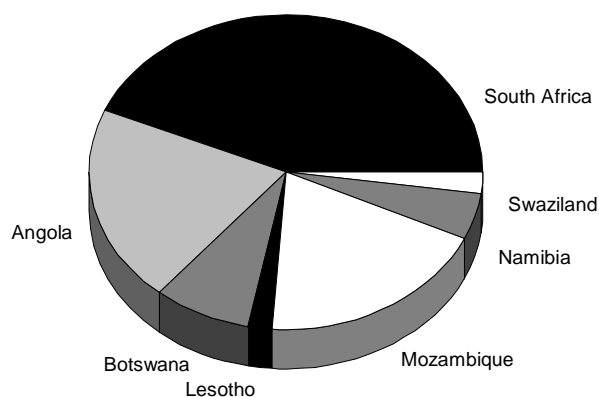


Fig 1: AFP surveillance specimens, NICD, 2004

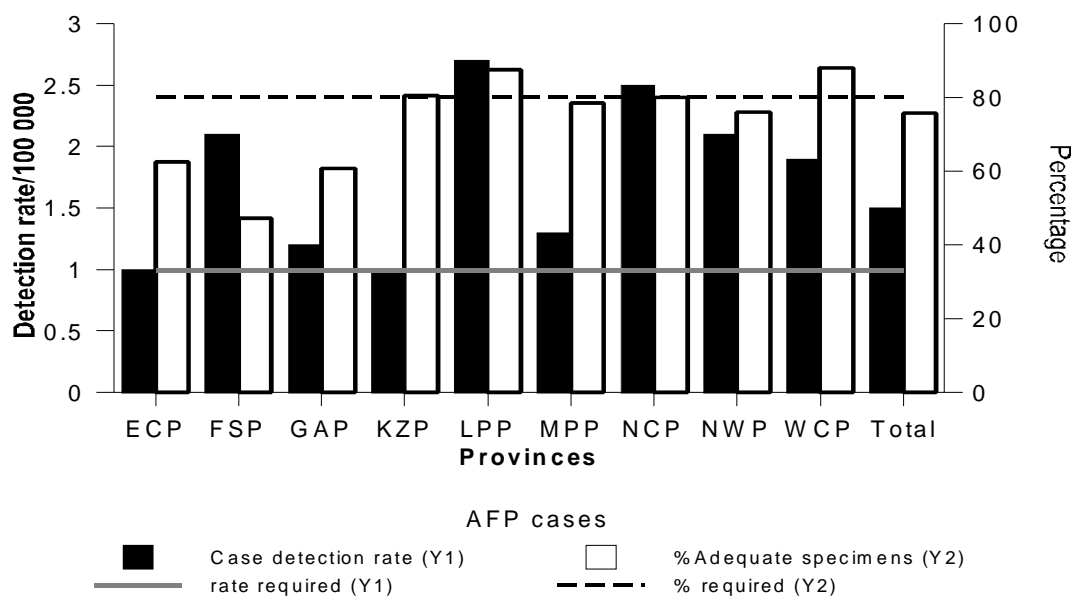


Fig 2: Provincial detection rate and stool adequacy, South Africa, 2004

ENTERIC DISEASE SURVEILLANCE, 2004

Karen Keddy, Enteric Disease Research Unit (EDRU), NICD

Salmonellosis - *Salmonella Typhimurium* was the commonest non-typhoidal isolate received by the EDRU from 2000 - 2004 and is consistent with findings in other African countries. Usually *S. Enteritidis* was the second commonest isolate but was replaced by *S. Isangi* in 2002 and 2003. This resulted from a large national nosocomial outbreak, often co-circulating with *S. Typhimurium* and *S. Muenchen* and complicated by the HIV epidemic. In 2004, total numbers of *S. Isangi* decreased, hopefully indicating more stringent infection control measures.

No seasonal trends were noted in the isolation of *S. Typhimurium* due to the association with HIV. *S. Typhimurium* and *S. Isangi* most frequently caused invasive disease and were mostly isolated from blood cultures. In contrast, *S. Enteritidis* primarily caused gastro-enteritis and was marginally commoner in the summer months.

The majority of *S. Typhimurium* and *S. Isangi* isolates were resistant to five or more antibiotics. Resistance to extended spectrum cephalosporins is high and many express an extended spectrum beta-lactamase (ESBL). This reflects the nosocomial transmission of these isolates. Although nalidixic acid resistance was observed in a significant proportion of all isolates, fluoroquinolone resistance was rare. Quinolone resistance is generally increasing.

Greater numbers of *S. typhi* isolates were received during 2004. Only one isolate from the Western Cape showed multi-drug resistance and was probably acquired elsewhere, as the resistance pattern differed from other isolates from this area.

Shigellosis - Large numbers of isolates from many different serotypes were received in 2003 and 2004 although annual numbers do not differ significantly. Since surveillance was started at the end of 1999 the

incidence of *Shigella dysenteriae* type 1, most frequently associated with major clinical complications and with the greatest epidemic potential, appears to be decreasing. Two isolates were received in 2003 (1 KwaZulu-Natal adult and a Free State child), and one from a KwaZulu-Natal adult in 2004. *S. flexneri* serotypes generally appear commoner in rural areas, possible reflecting inadequate provision of safe water, whereas *S. sonnei* is more commonly seen in urban centres and reflect patterns seen in developing and developed countries.

Antimicrobial resistance to WHO recommended antibiotic treatment is rapidly increasing and many isolates are resistant to first line treatment. Resistance to nalidixic acid is increasing and

fluoroquinolones would now be the drug of choice for bacillary dysentery. Two isolates, one each from Western Cape and KwaZulu-Natal, expressed an ESBL and may reflect nosocomial transmission or previous hospitalisation.

Cholera - Compared to 2003, *Vibrio cholerae* isolates more than doubled in 2004. The majority were received in the first six months, with no cases since September. There were also clinical reports of cases in Eastern Cape. The decrease in cases is due to vigilance and early response by local health authorities and increasing population immunity following the epidemic. Sustained vigilance is mandatory as cases continue to occur on our borders and surveillance with rapid outbreak response is the surest way to prevent further epidemics.

RESPIRATORY AND MENINGEAL PATHOGENS SURVEILLANCE, 2004

Elizabeth Prentice, Linda de Gouveia, Vanessa Quan, Anne von Gottberg and the the Group for Enteric, Respiratory and Meningeal Disease Surveillance in South Africa (GERMS-SA)

The following is a summary of the trends in 2003 and 2004 of invasive disease due to three bacterial pathogens occurring in South Africa as reported to the Respiratory and Meningeal Pathogens Research Unit (RMPRU).

Haemophilus influenzae

The number of cases of invasive *Haemophilus influenzae* disease caused by all serotypes in all age groups was similar to those seen in 2003 (Epidemic-prone disease surveillance table, page 2). In children aged less than 5 years *H. influenzae* type b (Hib) disease did not decrease further. This trend may be due to better reporting and case detection as our surveillance enters its fifth year.

Neisseria meningitidis

The number of cases of disease caused by *Neisseria meningitidis* in 2004 was similar to the number in 2003 (figure 1). As was the case in 2003, approximately half of these cases were reported from Gauteng, with an annual incidence of meningococcal disease in 2004 of 1.8/100 000. The Western Cape had the second highest incidence at 1.2/100 000, reduced from the previous year. The increase in cases reported from Limpopo might reflect better reporting of sporadic cases—unfortunately 5 cases had no viable isolates for serogrouping, but the other 7 isolates were identified as belonging to serogroups B, W135 or Y (figure 2).

In some provinces the distribution of serogroups changed (figure 2). This was most marked in Gauteng where serogroup W135 disease had become more prevalent. In 2003 49% of viable isolates received by RMPRU for the Gauteng region were serogroup A and

14% were serogroup W135. In 2004 these percentages changed to 33% and 37% respectively. Of these isolates a large proportion (approximately 90% of those tested) belonged to the Hajj-strain complex. We hope to elucidate the epidemiological factors responsible for this during 2005.

Streptococcus pneumoniae

In 2004 we received an increased number of specimens from cases of invasive pneumococcal disease, especially from the Eastern Cape, Freestate, KwaZulu Natal and Limpopo (figure 3). This might be due to improved case detection. The burden of invasive pneumococcal disease in Gauteng in children aged less than 5 years has not changed over the 2 years: an annual incidence of 75.1 and 81.2 per 100 000 in 2003 and 2004 respectively. The proportion of isolates testing non-susceptible to penicillin increased. Nationally 28% of viable isolates had intermediate or high-level resistance to penicillin (figure 4). In 2003, 23% of the isolates were non-susceptible to penicillin. A similar number of cases were reported from Gauteng in 2003 and 2004 and the level of resistance in this province had risen from 23% in 2003 to 30% in 2004. Mpumalanga and North West Province also had increases in the proportion of non-susceptible pneumococcal isolates.

Surveillance data are improving as more laboratories throughout the country report to the RMPRU. Although some trends are difficult to interpret with this improved case detection, these data will still be useful to clinical, public health and laboratory staff.

We acknowledge all collaborators, laboratory and clinical staff who make this surveillance possible.

Figure 1: Annual incidence of meningococcal disease reported to the NICD, South Africa, 2003 and 2004

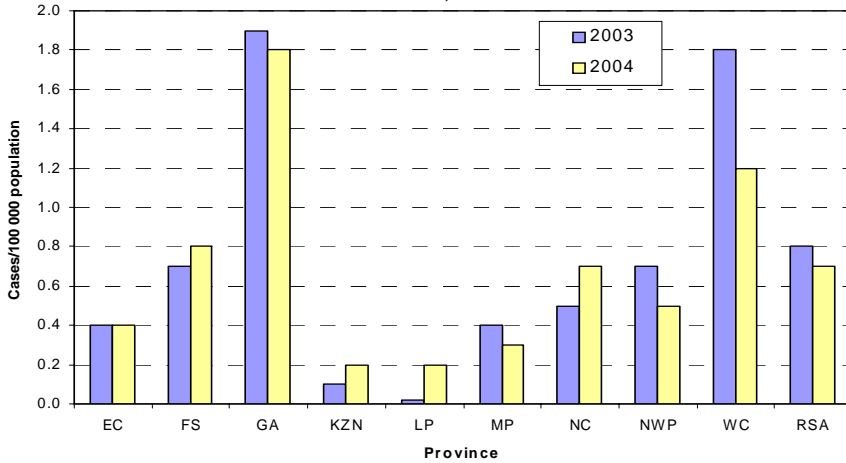


Figure 2: Most common meningococcal serogroups causing disease in South Africa by province for 2003 and 2004 as confirmed at the NICD

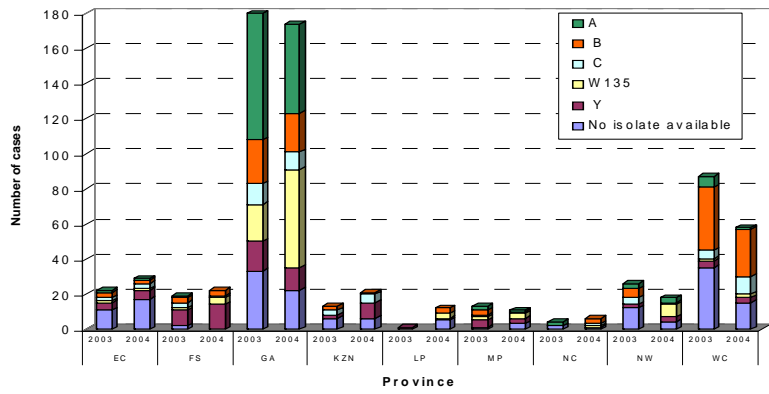


Figure 3: Annual incidence of invasive pneumococcal disease in children <5 years of age as reported to the NCD in 2003 and 2004

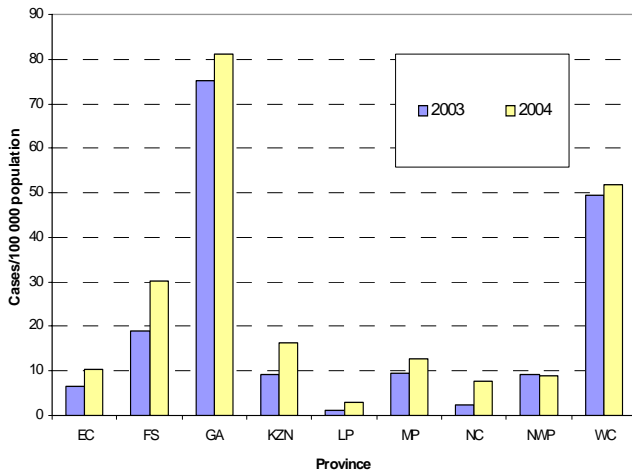


Figure 4: Proportion of penicillin non-susceptible (MIC >0.06 mg/l) isolates (one isolate per invasive pneumococcal disease episode)

