VOLUME 8, NO. 3

COMMUNICABLE DISEASES SURVEILLANCE BULLETIN

AUGUST 2010

NICD NHLS

FOREWORD

The emergence of vaccine-derived polioviruses (VDPVs) has recently been highlighted as an ongoing threat in settings of low vaccine coverage and as a major concern in planning for polio eradication[1]. The current Bulletin illustrates the work done by the NICD Polio Molecular Unit related to monitoring the presence of VDPVs in the African region. Resistant bacterial infections are another group of emerging infections of global concern. We include an article describing the molecular characterization of Methicillin-resistant *Staphylococcus aureus* from the newly formed NICD Satellite Unit for Molecular Epidemiology. Both articles highlight the importance of molecular techniques in monitoring emerging pathogens.

In a surveillance report, results from microbiologic surveillance for sexually transmitted infections (STI) for Gauteng Province for 2010 are presented. These data are critical to guide syndromic guidelines for STI case management in South Africa through monitoring the causative agents of clinical STI syndromes as well as antimicrobial resistance patterns of relevant pathogens.

Cheryl Cohen, Editor

Reference

1. Jenkins HE, Aylward RB, Gasasira A, Donnelly CA, Mwanza M, Corander J et al. Implications of a circulating vaccine-derived poliovirus in Nigeria. N Engl J Med 2010 June 24;362(25):2360-9.

CONTENTS

IDENTIFICATION OF VACCINE-DERIVED POLIOVIRUSES (VDPVs) IN THE DRC FROM 2005 TO 2010

Nicksy Gumede[1,2], Marietjie Venter[1,2], Olivia Lentsoane[1], Jean Muyembe-Tamfum[3], Riziki Yogolelo[3], Adrian Puren[1] and Barry D Schoub[1]
[1]National Institute for Communicable Diseases, Johannesburg, South Africa
[2]University of Pretoria/Tshwane Academic Divison NHLS
[3]National Institute for Biomedical Research (INRB), Kinshasha/Gombe

Oral polio vaccine (OPV) is an important and effective means of control and eradication of wild polio viruses. The consequence of the use of a live OPV is that there is genetic drift as a result of mutations and recombination events with, for example, non-polio enteroviruses that result in the acquisition of transmissibility and neurovirulence properties similar to the wild polio viruse. One outcome of such events is that of circulating (c) Vaccine-derived poliovirus (cVDPV) strains that are transmitted and may cause flaccid paralysis. The VDPVs are Sabin – like viruses that have less than 99% VP1 nucleotide sequence identity to the Sabin Oral Polio Vaccine strains (OPV). cVDPVs may originate following prolonged replication of the vaccine strain as a result of factors such as reduced population immunity, and inadequate vaccine coverage. cVDPVs are likely to circulate for at least 1-2 year before being indentified. VDPV for type one and type two polio are more commonly reported but rare for polio type three. The reasons for this are not clear. For an outbreak of cVDPV at least two linked cases need to be identified. Some of the documented

WEB
This bulletin is available on the NICD website: http://www.nicd.ac.za

cVDPV outbreaks reported include Egypt (1982-1993); Haiti (2000-2001); Dominican Republic (2000-2001); Philippines (2001); China (2004); Cambodia (2005-2006); Indonesia (2005); and, Madagascar (2002 and 2005).[1] An outbreak due to wild type polio virus as well as co-circulation of cVDPV was detected in the Democratic Republic of Congo (DRC) in 2005 for which an immunisation campaign was initiated to interrupt transmission. However, the Acute Flaccid Paralysis (AFP) cases detected between 2005–2010 in the DRCwere shown to be cVDPV.

In total, 481 viral isolates obtained from the DRC during 2003 until 2010 were tested by RT-PCR using pan-enterovirus, pan-poliovirus, serotype specific and Sabin type 1, 2 and 3 virus specific primers. The same isolates were further tested by ELISA and both techniques identified the isolates as Sabin-like poliovirus. All isolates were further screened by Real Time RT-PCR and the genetic variability of eighty isolates was investigated by nucleotide sequence analysis of the VP1 region. Partial sequencing analysis confirmed that 27 isolates from 30 AFP cases had $\leq$ 99% VP1 sequence identity to the parental Sabin strain.

A total of 27 children with AFP were found to excrete VDPVs of serotype 2 in the DRC between 2005 and 2010. These viruses represent at least three lineages and at least two outbreaks. Molecular analysis confirmed 11 of these viruses to be genetically related, representing lineage 1 circulation. Altogether 15 cases occurred in Katanga (KAT) province, 4 cases in Kasai Occidental (KOC), 2 cases in Bandundu (BDD), 1 case in Sud Kivu (SKV), 1 case in Orientale (ORT) and 1 case in Équateur (EQT). The first case was identified in Inongo district of Bandundu province in September of 2005 and the last reported case was in the Kalonda Quest district of KOC province on 24th of February 2010. Partial genomic sequencing of the poliovirus isolates revealed considerable nucleotide sequence divergence of between 1.1% to 2% from the prototype Sabin strain in the VP1 region of the viral genome. The DRC VDPVs formed 3 independent lineages, one represented by 3 VDPVs found between 2005 and 2007 and the other two represented by viruses from 2008 to 2010 (Figure 1). The viruses from the three lineages clustered geographically: (a) KAT and KOC provinces showed 99.20% to 98.3% similarity to Sabin type 2, (b) KAT province in which viruses showed 99.00% to 98.20% similarity to Sabin type 2; and (c) KOC province showed 98.90% to 97.4% similarity to Sabin type 2. The 1%-3% VP1 sequence divergence of the three DRC lineages indicates circulation of approximately 1 to 3.0 years, in accordance with previous published estimates of a rate of 1% per year for PV nucleotide sequence evolution.[2,3] No cVDPVs were identified in DRC in 2006.

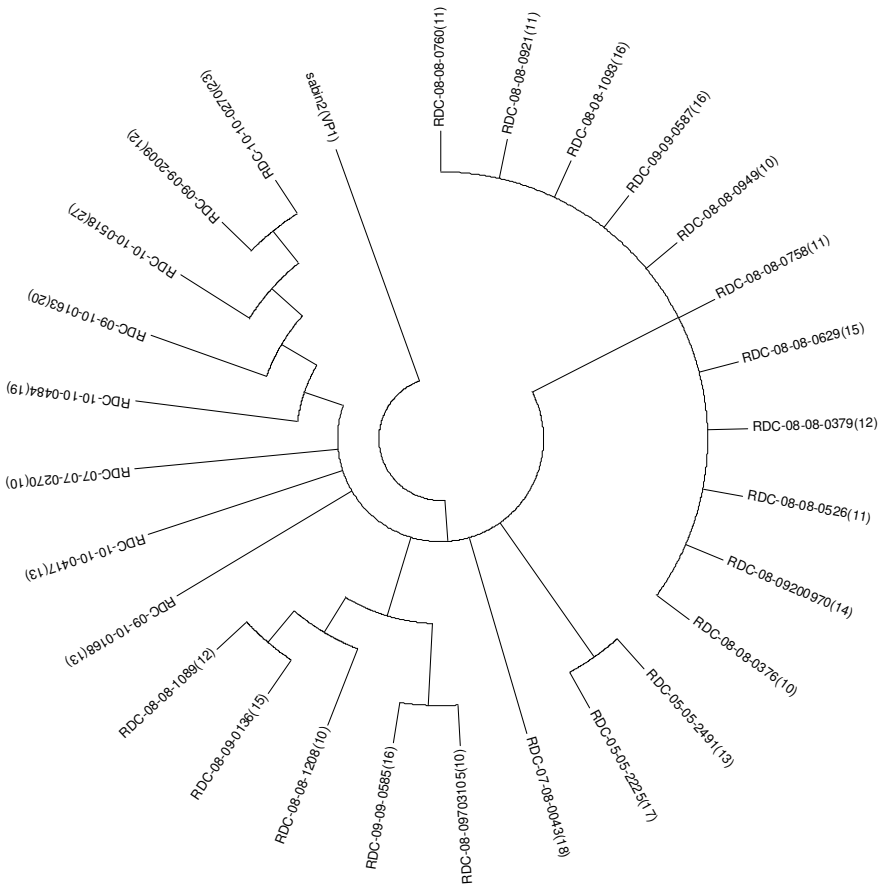

Figure 1: A Mega 4 tree rooted at the Sabin 2 reference strain closest related to the strains isolated in the DRC in 2005 - 2010.Brackets represent number of nucleotide changes from the Sabin 2 reference strain.

(Continued on page 45

The distribution of most of the VDPVs within a single province in DRC, and close genetic relationship indicate co -circulation and co-evolution of these viruses. Follow up investigations revealed that immunisation coverage had been low (70%) for the period 2005 – 2008. This factor as well a possibly other unidentified factors was sufficiently favourable for cVDPV emergence. This is the first time that type 2 cVDPVs has been detected in DRC. The occurrence of WPV outbreaks during the same period emphasizes the need to maintain high OPV coverage and AFP surveillance to minimize the risk of emergence of VDPVs or circulation of imported WPVs. The cVDPV findings have important implications for the Global Polio Eradication Initiative and for future policies regarding OPV immunization.[4, 5, 6]

Inadequate vaccine coverage, limited population immunity, high population density and inadequate sanitation conditions may exist elsewhere in Africa that could lead to further identified outbreaks of cVDPV and improved surveillance for VDPVs will be essential for the achievement of polio eradication in Africa.

Acknowledgement
We would like to thank the staff from National Institute for Communicable Diseases (NICD, Polio Molecular section), Centers for Disease Control and Prevention, Atlanta, Georgia (CDC), World Health Organization (WHO) Polio LabNet and the DRC National Polio Laboratory and EPI team for their contribution. This work was supported in part by a grant from WHO.

References

1. Rakoto-Andrianarivelo M, Gumede N, Jegouic S, Balanant J, Andriamamonjy SN, Rabemanantsoa S, Birmingham M, Randria-manalina B, Nkolomoni L, Venter M, Schoub BD, Delpeyroux F, Reynes JM. 2008. Reemergence of recombinant vaccine-derived poliovirus outbreak in Madagascar. The Journal of infectious diseases 197(10):1427-1435.
2. Alexander JP, Jr., Gary HE, Jr., Pallansch MA. 1997. Duration of poliovirus excretion and its implications for acute flaccid paraly-sis surveillance: a review of the literature. J Infect Dis 175 Suppl 1:S176-182.
3. Jorba J, Campagnoli R, De L, Kew O. 2008. Calibration of multiple poliovirus molecular clocks covering an extended evolutiona-ry range. Journal of virology 82(9):4429-4440.
4. Kew O, Morris-Glasgow V, Landaverde M, Burns C, Shaw J, Garib Z, Andre J, Blackman E, Freeman CJ, Jorba J, Sutter R, Tambini G, Venczel L, Pedreira C, Laender F, Shimizu H, Yoneyama T, Miyamura T, van Der Avoort H, Oberste MS, Kilpatrick D, Cochi S, Pallansch M, de Quadros C. 2002. Outbreak of poliomyelitis in Hispaniola associated with circulating type 1 vaccine-derived poliovirus. Science 296(5566):356-359.
5. Rousset D R-AM, Razafindratsimandresy R, Randriamanalina B, Guillot S, Balanant J, et al.,. 2003. Recombinant vaccine-derived polioviruses in Madagascar. emerging infectious diseases 9:885-887.
6. Yang CF, Naguib T, Yang SJ, Nasr E, Jorba J, Ahmed N, Campagnoli R, van der Avoort H, Shimizu H, Yoneyama T, Miyamura T, Pallansch M, Kew O. 2003. Circulation of endemic type 2 vaccine-derived poliovirus in Egypt from 1983 to 1993. J Virol 77 (15):8366-8377.

MICROBIOLOGICAL SURVEILLANCE FOR SEXUALLY TRANSMITTED INFECTIONS: REPORT ON THE FINDINGS FROM GAUTENG PROVINCE IN 2010

Sexually Transmitted Infections Reference Centre, National Institute for Communicable Diseases

The sexually transmitted infections (STI) microbiological surveillance was undertaken in Gauteng (Johannesburg) between January and April 2010. The aim of the surveillance was to determine a) the aetiology of the male urethritis syndrome (MUS), vaginal discharge syndrome (VDS) and genital ulcer syndrome (GUS), b) the prevalence of HIV co-infection in patients with these syndromes, and c) the antimicrobial susceptibility of *Neisseria gonorrhoeae* isolates to cefixime, ceftriaxone and ciprofloxacin.

1. Aetiological Findings

A total of 530 consecutive STI patients were recruited (189 VDS, 212 MUS, 142 GUS). Thirteen GUS patients presented with VDS at the same time. Pathogens were detected by multiplex polymerase chain reaction (M-PCR) on swabs collected from VDS, MUS and GUS cases. Smears from VDS cases were examined for the presence of bacterial vaginosis (BV) and Candida by microscopy. In men with urethral discharge, *Neisseria gonorrhoeae* was the most common aetiological agent (75.0%, 159/212) followed by *Chlamydia trachomatis* (19.8%, 42/212) (Table 1). BV was the most common aetiological agent followed by Candida and TV in women with VDS (38.1%, 72/189; 31.2%, 59/189; and 20.1%, 38/189 respectively). These data were compared to the data from 2009 (Table 2).

Table 1: The prevalence of the STI pathogens in patients with MUS in Johannesburg for the 2009 and 2010 surveys.

Pathogen	2009 Survey (n = 207)	2010 Survey (n = 212)	*P* value
Neisseria gonorrhoeae	147 (71.0%)	159 (75.0%)	0.358
Chlamydia trachomatis	52 (25.1%)	42 (19.8%)	0.193
Trichomonas vaginalis	15 (7.2%)	13 (6.1%)	0.648

(Continued on page 46)

Table 2: The prevalence of the STI pathogens and bacterial infections in patients with VDS in Johannesburg for the 2009 and 2010 surveys.

Pathogen or condition	2009 Survey (n =207)	2010 Survey (n =189)	*P* value
Neisseria gonorrhoeae	26 (12.6%)	23 (12.2%)	0.906
Chlamydia trachomatis	27 (13.0%)	31 (16.4%)	0.345
Trichomonas vaginalis	55 (26.6%)	38 (20.1%)	0.130
Bacterial vaginosis	68 (32.8%)	72 (38.1%)	0.275
Candidiasis	50 (24.2%)	59 (31.2%)	0.116

The number of GUS patients recruited in 2010 was slightly higher than that of GUS patients recruited in 2009 (142 vs 102). In both 2009 and 2010 surveys, herpes was the most frequent cause of genital ulceration accounting for 93.8% (76/81) and 92.8% (77/83) of GUS cases detected by M-PCR respectively and syphilis was the second most frequent cause of genital ulceration. Lymphogranuloma venereum accounted for only 1.4% of GUS cases in 2010 while no cases of chancroid were detected. No Donovanosis cases were detected in 2010.

Table 3: Aetiology of GUS in Johannesburg for the 2009 and 2010 surveys

Pathogen	2009 Survey (n =102)	2010 Survey (n = 142)	*P* value
Herpes simplex virus	76/81* (93.8%)	77/83* (92.8%)	0.959
Treponema pallidum	5/81* (6.2%)	5/83* (6.0%)	0.968
Haemophilus ducreyi	2/81* (2.5%)	0/83* (0%)	0.150
Chlamydia trachomatis L1-L3	0 (0%)	2 (1.4%)	0.229
Klebsiella granulomatis	1 (1.0%)	0 (0%)	
No pathogens	21 (20.6)	59 (41.5)	N/A

* No of all GUS where a pathogen was detected

Table 4: HIV seroprevalence for patients with MUS, VDS and GUS in Johannesburg (2009 and 2010)

Syndrome	2009	2010	*P* value
MUS	58/ 207 (28%)	67/212 (31.6%)	0.423
VDS	121/207 (58.5%)	95/189 (50.2%)	0.102
GUS	67/101 (66.3%)	96/142 (67.6%)	0.836

Comments:

The relative prevalence of TV in both men and women decreased in the second survey. Gonorrhoea, on the other hand, remained the most common cause of MUS, the relative prevalence increasing by 4% in 2010. Genital herpes continues to be the major cause of GUS with no significant change among the GUS patients where aetiology was found (p=0.959) Syphilis remains an infrequent but important cause of genital ulceration and LGV with a prevalence of 1.4% in 2010 compared to 2009 where it disappeared in the STI patient group. These data confirm that the HIV prevalence observed from the STI patient group in Johannesburg still remains high and these STI patients are still an important group to target for HIV prevention initiatives.

For genital ulceration, it is important to provide high quality counselling and health education around genital herpes, which may be a recurrent and psychologically disturbing condition.

2. Antimicrobial Susceptibility Findings

All isolates of *N. gonorrhoeae* were still susceptible to ceftriaxone and cefixime which are drugs presently used in Gauteng clinics. However, in the 2009 survey (25/87, 20.7%) *N. gonorrhoeae* isolates were resistant to ciprofloxacin and in 2010 there was 1.2% increase (32/142, 21.9%) in the number of ciprofloxacin resistant isolates. This increase was however not statistically significant (*P* = 0.3526).

Given that a) gonorrhoea still remains the most frequent cause of MUS (Table 1), b) MUS is the most common STI presentation in men, and c) the high prevalence of HIV co-infection among MUS patients (Table 3), it is important to ensure the availability of cefixime (in the revised Essential Drugs Programme Primary Care guidelines) as first-line anti-gonococcal therapy. In clinics without oral cefixime, 250 mg of intramuscular ceftriaxone should be used instead. The susceptibility patterns of these new drugs need ongoing surveillance monitoring.

Acknowledgements:

Thanks for the successful completion of this surveillance go to: the Gauteng surveillance clinical team: a) Charles Ricketts, Mr. Alex Vezi, Lindi Mshibe, b) Mr. Maluleke and staff at Alexandra Health Centre, c) Laboratory staff at the STI Reference Centre, and d) NICD/NHLS for their funding of the surveillance.

This report was compiled by Frans Radebe and David Lewis

METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS* CLONAL TYPES CIRCULATING IN HOSPITALS IN CAPE TOWN

Melissa Jansen van Rensburg[1], Andrew Whitelaw[1,2,3], Eliya Madikane[1,2], Gay Elisha[1,2,3]
[1]Division of Medical Microbiology, University of Cape Town, [2]National Health Laboratory Service, [3]Unit for Molecular Epidemiology of the National Institute for Communicable Diseases, the National Health Laboratory Service and the University of Cape Town

Methicillin-resistant *Staphylococcus aureus* (MRSA), a common nosocomial pathogen, is a major public health concern worldwide. The control of MRSA within the hospital setting is facilitated by comprehensive epidemiological data. In this context, a study was carried out to gain knowledge about MRSA clones circulating in hospitals in Cape Town.

One hundred MRSA isolates, collected randomly between the period January 2007 and December 2008, from the academic complex in Cape Town, without duplication, were included in the study. The isolates were cultured from pus and pus swabs (n = 64), urine (n = 9), respiratory tract specimens (n = 3), blood (n = 7) and central venous catheter tips (n = 7). The specimen types were obtained from patients in any one of five hospitals in metropolitan Cape Town: Groote Schuur Hospital (GSH, n = 51), Mowbray Maternity Hospital (MMH, n =19), Red Cross War Memorial Children's Hospital (RCCH, n = 21), Victoria Hospital (VH, n = 4) and University of Cape Town Private Hospital (UCTPH, n = 5).

Pulsed-field gel electrophoresis (PFGE) separated the 100 isolates into 6 PFGE clusters or clonal types (A-F) and 8 sporadic isolates. Clusters C (n = 35) and E (n = 33) were the largest clusters, collectively accounting for 68% of the isolates. The remaining isolates were assigned to clusters A (n = 4), B (n = 2), D (n = 7) and F (n =11).

When clusters were stratified by hospital, clusters A, B and F included isolates from 2 hospitals; clusters C and D comprised MRSA from 3 hospitals, and cluster E contained isolates from all 5 hospitals (Table1), demonstrating the transmission of strains within and between hospitals. With the exception of MRSA in cluster C there was no obvious clustering by ward or date of isolation of strains. Cluster C comprised 35 MRSA. The levels of genetic similarity within the cluster were high, ranging from 89% to 100%. Significantly, 18 of the 19 MRSA from MMH were included in cluster C. An additional 8 were from the maternity service at GSH. Thus, 74% of MRSA in cluster C were isolated from either women or neonates in the maternity services at MMH and GSH. Further, a dendrogram, indicating the levels of genetic similarity between the isolates suggested clustering of strains isolated within short time frames. This data is strongly suggestive of transmission of MRSA strains within the maternity services in Cape Town hospitals.

The antibiotic susceptibility profiles of MRSA strains within each of the clusters were inspected. Although all of the strains were resistant to β-lactam antibiotics and susceptible to vancomycin, there were striking differences between the antimicrobial susceptibility profiles of the clonal types (Table 2). By and large, the susceptibility profiles of isolates within a PFGE cluster were similar, if not identical. Strains in cluster C tended to be resistant to only erythromycin and clindamycin, whereas some MRSA isolates in clusters B and F were additionally resistant to ciprofloxacin and gentamicin. There were similarities between the susceptibility profiles of strains in clusters A, D and E. Resistance to erythromycin, clindamycin, gentamicin, co-trimoxazole and ciprofloxacin was widespread in these clusters. However, all MRSA strains in clusters D and E (n = 40) were resistant to rifampicin. Thus, the most widely disseminated cluster, E, contains MRSA strains resistant to several classes of antibiotics.

This pilot study has demonstrated the power of molecular epidemiological techniques to detect transmission of resistant bacteria within the hospital setting and highlights the need for improved infection control measures in these hospitals.

Table 1: Stratification of Pulsed-Field Gel Electrophoresis (PFGE) Clusters by Hospital of Origin

PFGE Cluster (n)	Distribution of strains across hospitals (n (%))				
	GSH [a]	RCCH	MMH	UCTPH	VH
A (4)	2 (50)	2 (50)	-	-	-
B (2)	1 (50)	-	-	1 (50)	-
C (35)	13 (37.14)	4 (11.43)	18 (51.43)	-	-
D (7)	4 (57.14)	2 (28.57)	-	-	1 (14.29)
E (33)	19 (57.58)	9 (27.27)	1 (3.03)	1 (3.03)	3 (9.09)
F (11)	8 (72.73)	-	-	3 (27.27)	-

[a] GSH, Groote Schuur Hospital; RCCH, Red Cross War Memorial Children's Hospital; MMH, Mowbray Maternity Hospital; UCTPH, University of Cape Town Private Hospital ; VH, Victoria Hospital.

(Continued on page 48)

Table 2: Antimicrobial Resistance Profiles across Pulsed-Field Gel Electrophoresis (PFGE) Clusters

PFGE Cluster (n)	Antimicrobial Agents (n (%) resistant)								
	PEN [a]	OXA	ERY	CLI	RIF	SXT	CIP	GEN	VAN
A (4)	4 (100)	4 (100)	4 (100)	4 (100)	0	4 (100)	4 (100)	4 (100)	0
B (2)	2 (100)	2 (100)	2 (100)	2 (100)	0	0	1 (50)	1 (50)	0
C (35)	35 (100)	35 (100)	32 (91.43)	32 (91.43)	1 (2.86)	1 (2.86)	0	10 (28.57)	0
D (7)	7 (100)	7 (100)	5 (71.43)	5 (71.43)	7 (100)	7 (100)	1 (14.29)	5 (71.43)	0
E (33)	33 (100)	33 (100)	13 (39.39)	11 (33.33)	33 (100)	31 (93.94)	28 (84.85)	31 (93.94)	0
F (11)	11 (100)	11 (100)	11 (100)	11 (100)	0	0	10 (90.91)	3 (27.27)	0

[a] PEN, penicillin; OXA, oxacillin; ERY, erythromycin; CLI, clindamycin; RIF, rifampicin; SXT, co-trimoxazole; CIP, ciprofloxacin; GEN, gentamicin; VAN, vancomycin.

NICD Satellite Unit for Molecular Epidemiology

The NICD has recently established a satellite Molecular Epidemiology Unit at the NHLS Groote Schuur/University of Cape Town. The Divisions of Medical Microbiology and Medical Virology are jointly responsible for the functioning of the unit. The unit will focus on the molecular epidemiology of hospital-associated infections, both sporadic and outbreak-related, the tracking and identification of emerging pathogens and managing the activities of the WHO human papillomavirus Labnet laboratory for the Africa region. The Unit will work closely with other units of the NICD, most notably, the Epidemiology and Outbreak Response Unit in order to build capacity in the Coastal region.

Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 30 June 2009/2010*

Disease/Organism	Cumulative to 30 June, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Anthrax	2009	0	0	0	0	0	0	0	0	0	0
	2010	0	0	0	0	0	0	0	0	0	0
Botulism	2009	0	0	0	0	0	0	0	0	0	0
	2010	0	0	0	0	0	0	0	0	0	0
Cryptococcus spp.	2009	708	244	1160	763	333	448	43	380	311	4390
	2010	755	241	1216	584	309	398	35	307	264	4109
Haemophilus influenzae, invasive disease, all serotypes	2009	17	10	78	22	1	17	4	7	47	203
	2010	24	10	95	18	3	7	7	5	48	217
Haemophilus influenzae, invasive disease, < 5 years											
Serotype b	2009	3	4	9	10	0	1	1	0	12	40
	2010	3	3	13	1	1	3	3	1	9	37
Serotypes a, c,d, e, f	2009	0	1	12	0	0	1	0	1	6	21
	2010	1	0	4	0	1	1	0	0	5	12
Non-typeable (unencapsulated)	2009	1	0	14	6	0	1	0	0	7	29
	2010	1	1	31	3	0	0	1	1	10	48
No isolate available for serotyping	2009	2	1	10	3	1	4	1	3	1	26
	2010	6	2	10	0	1	1	0	1	3	24
Measles	2009	6	1	33	8	2	7	0	7	6	72
	2010	1262	536	933	3613	270	1733	262	688	1521	10818
Neisseria meningitidis, invasive	2009	13	3	96	14	1	15	2	7	31	182
	2010	12	10	72	6	4	7	13	5	24	153
Novel Influenza A virus infections***	2009	0	0	0	0	0	0	0	0	0	0
	2010	0	0	0	0	0	0	0	0	0	0
Plague	2009	0	0	0	0	0	0	0	0	0	0
	2010	0	0	0	0	0	0	0	0	0	0
Rabies	2009	7	0	0	4	2	2	0	0	0	15
	2010	2	0	0	3	3	1	0	0	0	9
**Rubella	2009	84	2	36	47	8	42	22	10	19	270
	2010	194	61	101	242	26	116	26	120	150	1036
Salmonella spp. (not typhi), invasive disease	2009	28	16	176	60	1	20	5	11	45	362
	2010	25	12	197	34	8	9	7	5	44	341
Salmonella spp. (not typhi), isolate from non-sterile site	2009	121	20	356	73	3	79	13	32	123	820
	2010	124	31	380	122	7	46	4	25	88	827
Salmonella typhi	2009	4	1	12	2	0	3	0	0	7	29
	2010	3	1	18	7	1	6	0	0	4	40
Shigella dysenteriae 1	2009	0	0	0	0	0	1	0	0	0	1
	2010	0	0	0	0	0	0	0	0	0	0
Shigella spp. (Non Sd1)	2009	129	45	341	87	2	43	10	11	234	902
	2010	133	34	413	79	6	25	14	13	224	941
Streptococcus pneumoniae, invasive disease, all ages	2009	199	136	1056	251	41	106	38	80	327	2234
	2010	190	117	832	209	45	106	44	67	291	1901
Streptococcus pneumoniae, invasive disease, < 5 years	2009	68	37	308	86	10	35	21	16	117	698
	2010	35	25	211	48	7	25	25	13	82	471
Vibrio cholerae O1	2009	1	1	45	0	639	396	0	55	5	1142
	2010	0	0	0	0	0	0	0	0	0	0
Viral Haemorrhagic Fever (VHF)											
Crimean Congo Haemorrhagic Fever (CCHF)	2009	0	1	0	0	0	0	0	0	0	1
	2010	0	1	0	0	0	0	2	0	0	3
Other VHF (not CCHF)****	2009	0	0	0	5	0	0	0	0	0	5
	2010	17	122	0	0	0	0	72	7	3	222

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 - 30 June 2009/2010*

Programme and Indicator	Cumulative to 30 June, 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	2009	30	3	29	51	32	27	6	10	11	199
	2010	30	9	40	42	26	23	1	14	16	201
Laboratory Programme for the Comprehensive Care, Treatment and Management Programme for HIV and AIDS											
CD4 count tests											
Total CD4 count tests submitted	2009	182,245	71,289	329,161	389,536	103,122	127,183	27,074	108,146	113,856	1,451,612
	2010	203,841	91,292	393,628	495,013	132,285	153,887	31,942	124,832	124,400	1,751,120
Tests with CD4 count < 200/µl	2009	59,322	21,620	112,121	122,230	33,920	42,323	8,329	33,678	31,317	464,860
	2010	64,539	27,267	127,373	121,698	41,279	46,146	9,318	35,769	30,522	503,911
Viral load tests											
Total viral load tests submitted	2009	77,837	26,172	156,978	169,414	43,428	45,906	11,871	46,365	45,069	623,040
	2010	70,366	34,740	164,853	165,651	42,221	47,475	12,288	49,064	57,178	643,836
Tests with undetectable viral load	2009	44,871	20,046	103,328	112,522	27,238	28,776	7,115	30,612	37,303	411,811
	2010	46,018	22,608	121,363	41,776	29,943	36,955	7,328	33,538	44,436	383,965
Diagnostic HIV-1 PCR tests											
Total diagnostic HIV-1 PCR tests submitted	2009	14,969	6,336	29,445	35,226	8,516	9,672	1,876	8,578	9,214	123,832
	2010	15,325	7,100	30,712	40,259	10,869	11,503	2,367	8,755	9,118	136,008
Diagnostic HIV-1 PCR tests positive for HIV	2009	1,618	766	3,399	3,639	1,152	1,308	231	1,060	761	13,934
	2010	1,244	654	3,124	3,270	1,135	1,151	221	831	553	12,183

Footnotes

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

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

U = unavailable, 0 = no cases reported

The Communicable Diseases Surveillance Bulletin is published by the National Institute for Communicable Diseases (NICD) of the National Health Laboratory Services (NHLS), Private Bag X4, Sandringham, 2131, Johannesburg, South Africa.

Suggested citation: [Authors' names or National Institute for Communicable Diseases (if no author)]. [Article title]. Communicable Diseases Surveillance Bulletin 2010; 8 (3): [page numbers]. Available from http://www.nicd.ac.za/ pubs/ survbull/2010/CommDisBull August 2010.pdf