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FOREWORD

The roll-out of the GeneXpert and Xpert MTB/RIF into the National Tuberculosis Control Programme of South Africa represents an opportunity to transform laboratory testing strategies for tuberculosis in a high burden setting. The article in the current Bulletin summarising statistics for MDR and XDR tuberculosis prior to the widespread introduction of these diagnostic tests represents important data on the current situation with regard to drug resistant tuberculosis. As discussed in the article, data derived from the NHLS corporate data-warehouse do have several limitations but also represent a novel platform for the timeous acquisition of strategic information to guide planning.

In South Africa, sexually transmitted infections (STIs) are generally managed syndromically. For this reason, surveillance to determine the relative contribution of different pathogens to the different STI syndromes as well as the drug resistance profiles of these pathogens where relevant is critical. Updated surveillance data from Gauteng Province demonstrate ongoing high levels of resistance to ciprofloxacin amongst *Neisseria gonorrhoeae* isolates and support current recommendations of first line treatment with a 3rd generation cephalosporin.

Additional articles in the current edition include a description of an outbreak of encephalitis in Mpumalanga Province as well as a summary of the molecular epidemiology of human metapneumovirus in the Western Cape, both articles providing relevant local data related to important syndromes of meningitis and pneumonia.

Cheryl Cohen, Editor

CONTENTS

MICROBIOLOGICAL SURVEILLANCE FOR SEXUALLY TRANSMITTED INFECTIONS REPORT ON THE FINDINGS FROM GAUTENG PROVINCE IN 2011

Sexually Transmitted Infections Reference Centre, National Institute for Communicable Diseases

The sexually transmitted infections (STI) microbiological surveillance was undertaken in Johannesburg between January and April 2011. 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 415 consecutive STI patients were recruited (222 VDS, 125 MUS, 68 GUS). 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 (73.0%, 91/125) followed by *Chlamydia trachomatis* (32.0%, 40/125) (Table 1). BV was the most common aetiological agent followed by Candida and *Trichomonas vaginalis* (TV) in women with VDS (55.0%, 121/220; 26.8%, 59/220; and 15.3%, 34/222 respectively) (Table 2). These data were compared to the data from 2010 (Tables 1 and 2).

Table 1: The prevalence of the STI pathogens in patients with MUS in Johannesburg for the 2010 and 2011 surveys.

Pathogen	MUS		
	2010 (n = 212)	2011 (n = 125)	*P* value
Neisseria gonorrhoeae	159 (75.0%)	91 (73.0%)	0.699
Chlamydia trachomatis	42 (19.8%)	40 (32.0%)	0.012
Trichomonas vaginalis	13 (6.1%)	4 (3.2%)	0.306

Table 2: The prevalence of the STI pathogens and bacterial infections in patients with VDS in Johannesburg for the 2010 and 2011 surveys.

Pathogen or conditon	VDS		
	2010 (n =189)	2011 (n =222)	*P* value
Neisseria gonorrhoeae	23 (12.2%)	25 (11.3%)	0.877
Chlamydia trachomatis	31 (16.4%)	31 (14.0%)	0.493
Trichomonas vaginalis	38 (20.1%)	34 (15.3%)	0.241
Bacterial vaginosis	72 (38.1%)	121 (55.0%)	<0.001
Candidiasis	59 (31.2%)	59 (26.8%)	0.325

The number of GUS patients recruited in 2011 was lower than that of GUS patients recruited in 2010 (68 vs 102). In both 2010 and 2011 surveys, herpes was the most frequent cause of genital ulceration accounting for 54.2% (77/142) and 58.8% (40/68) of GUS cases detected by M-PCR respectively. *Lymphogranuloma venereum* accounted for only 1.5% of GUS cases in 2011 while no cases of chancroid and donovanosis were detected.

Table 3: Aetiology of GUS in Johannesburg for the 2010 and 2011 surveys.

Pathogen	**2010 (n = 142)**	**2011 (n = 68)**	***P* value**
Herpes simplex virus	77 (54.2%)	40 (58.8%)	0.555
Treponema pallidum	5 (3.5%)	0 (0%)	-
Haemophilus ducreyi	0 (0%)	0 (0%)	-
Chlamydia trachomatis L1-L3	2 (1.4%)	1 (1.5%)	1.000
Klebsiella granulomatis	0 (0%)	0 (0%)	-
No pathogens	59 (41.5)	28 (41.2%)	1.000

The prevalence of HIV co-infection among patients with MUS, VDS and GUS is shown in Table 4.

Table 4: HIV seroprevalence for patients with MUS, VDS and GUS in Johannesburg (2010 and 2011).

Syndrome	**2010**	**2011**	***P* value**
MUS	67/212 (31.6%)	36/125 (28.8%)	0.625
VDS	95/189 (50.2%)	95/222 (42.8%)	0.137
GUS	96/142 (67.6%)	36/68 (53.0%)	0.047

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, though the relative prevalence decreased by 2% in 2011. Genital herpes continues to be the major cause of GUS. Syphilis was not detected in 2011 as compared to 3.5% in 2010 and LGV prevalence did not change in 2011 (1.5%) and 2010 (1.4%). 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 89 gonococcal isolates isolated and tested for during January to April 2011 were still susceptible to ceftriaxone

and cefixime, which are drugs presently used in public clinics to treat presumptive gonorrhoea. However, in the 2010 survey (32/142, 21.9%) *N. gonorrhoeae* isolates were resistant to ciprofloxacin and in 2011 the resistant pattern did not change significantly (20/89, 22.3%). Ciprofloxacin is no longer used or recommended nationally for the treatment of gonococcal infections. 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 4), 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, Valentia Kekana, Alex Vezi, Lindi Mshibe, Sydney Khumalo b) Mr. Maluleke and staff at Alexandra Health Centre, c) Laboratory staff at the STI Reference Centre, and d) NICD/NHLS and NDOH for their funding of the surveillance.

Report compiled by David Lewis and Frans Radebe

VIRAL MENINGITIS OUTBREAK, MPUMALANGA PROVINCE, 2011

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Introduction
Viral meningitis is a syndrome of meningeal inflammation and can be caused by numerous viruses. The occurrence of viral meningitis is more frequent than bacterial meningitis but is seldom as life threatening. Prior to the introduction of the measles, mumps and rubella vaccine the mumps virus was believed to be the most common cause of viral meningitis amongst children. However, enteroviruses are now the most common cause of viral meningitis.[1]

Enteroviruses are so named as the viruses are able to replicate in the human gastrointestinal tract; humans are the only natural reservoirs for enteroviruses. There are at least ninety immunologically distinct serotypes within the family *Picornaviridae* that are responsible for a broad spectrum of disease in persons of all ages. Enteroviruses are transmitted by the faecal-oral route and acquired through direct person-to-person contact, or contaminated water.[2] Resultant infections are largely mild or asymptomatic; however, in some instances disease may be serious or even fatal. As a result, outbreaks of enteroviral disease can be considered a major public health concern.[3]

On 10 February 2011 the Mpumalanga Province Department of Health notified the National Institute of Communicable Diseases of the National Health Laboratory Service of a suspected outbreak of viral meningitis in a primary school in Skukuza. The infection control practitioner from a private hospital in Nelspruit reported an increase in the number of patients who had lumbar punctures done and whose cerebrospinal fluid (CSF) showed features of viral meningitis.

Four CSF specimens taken at the private hospital were sent to the NICD-NHLS for further testing. One of the specimens was insufficient and could not be tested. One specimen was negative and two positive for enterovirus on polymerase chain reaction (PCR) testing.

In response, an outbreak investigation was undertaken with the objectives of confirming the outbreak and the causative agent, determining the scope of the outbreak, supporting control activities and making recommendations for control and prevention of future outbreaks.

Method
A descriptive cross-sectional study was conducted. A broad case definition was applied for the purpose of identifying further cases by both retrospective medical record review and prospective active case finding. A case was defined as any individual residing in Ehlanzeni and Mbombela districts of Mpumalanga Province reporting to a healthcare facility with the symptoms of fever or headaches and neck stiffness or photophobia, or any individual resident in the same area for whom a lumbar puncture was performed in the management of a febrile illness with a date of onset of illness from 1 January 2011.

Exclusion criteria were: any individual residing in Ehlanzeni and Mbombela districts of Mpumalanga Province reporting to a healthcare facility with the symptoms of fever or headaches and neck stiffness or photophobia, where illness was diagnosed as other than meningitis or encephalitis; or any individual resident in the same area for whom a lumbar puncture was performed in the management of a febrile illness with a date of onset of illness from 1 January 2011 whose CSF specimen was found to be normal or revealed fungal or bacterial infection.

From collected information, we classified the cases into suspected, probable and confirmed based on, (a) clinical

features of viral meningitis, (b) a lumbar puncture results, and (c) microbiological confirmation.

a. S*uspected case:* Any individual residing in Ehlanzeni and Mbombela districts of Mpumalanga reporting to a healthcare facility with symptoms of fever or headache AND neck stiffness or photophobia with a date of onset from 1 January 2011.
b. *Probable case:* Any individual residing in Ehlanzeni and Mbombela districts of Mpumalanga reporting to a healthcare facility with symptoms of fever or headache AND neck stiffness or photophobia with a date of onset from 1 January 2011, who underwent a lumbar puncture with abnormal CSF test results, i.e. raised protein, raised white cell count and no bacterial or fungal identification.
c. *Confirmed case:* Any individual residing in Ehlanzeni and Mbombela districts of Mpumalanga reporting to a healthcare facility with symptoms of fever or headache AND neck stiffness or photophobia with a date of onset from 1 January 2011, who underwent a lumbar puncture with abnormal CSF test results, i.e. raised protein, raised white cell count and no bacterial or fungal identification, and found to be PCR positive for enterovirus by the NICD.

To identify common risk exposures a detailed telephonic questionnaire was administered to a sample of the cases.

Results

Epidemiological findings

Active case-finding identified 87 cases; 10 confirmed, 49 probable and 28 suspected cases (Figure 1). The median age of cases was 8 years (range=4 months to 60 years), with the majority (n=62, 71%) of the cases younger than 15 years of age (Table 1). Fifty-eight percent of cases were male.

The earliest reported date of onset of symptoms was 26 January 2011 (Figure 1). The majority of the cases (n=60, 69%) were residents of Nelspruit and Skukuza. Eight family clusters (≥2 cases in the same household) were identified. The only confirmed cluster associated with an institution was seven cases from a primary school in Skukuza.

Clinical and Microbiological findings

Cases presented with headache (n=87, 100%), fever (n=77, 88%) and neck stiffness (n=49, 77%). A total of 57 (65%) cases was admitted to hospital. Illness was noted to be mild, with no complications reported and no deaths.

Cerebrospinal fluid specimens were taken for 59 (68%) cases. Microscopy and Gram stain showed no bacteria detected and revealed pleocytosis in all cases; 80% (47/59) of these were lymphocyte predominant.

Enterovirus PCR testing was done at the Specialized Molecular Diagnostic Unit at the NICD-NHLS on 17 CSF specimens, and was positive in 10/17 (59%). Further characterization identified the causative organism as one of the human enterovirus group B viruses.

Public health response

In response to the outbreak, environmental health practitioners were deployed to assess hygiene and sanitation facilities at schools reporting cases. Health education material highlighting the practice of good personal hygiene as well as information relating to meningitis was distributed at schools, crèches and at healthcare facilities. The primary school in Skukuza closed their grade R class for a week in an attempt to limit the number of observed cases and spread of illness.

Table 1: Number of cases of viral meningitis by age group, Mpumalanga Province, 26 January to 9 March 2011 (n=87)

Age group (years)	Number of cases	% of total number cases	Cumulative %
0-5	33	37.9	37.9
6-10	22	25.3	63.2
11-15	8	9.2	72.4
16-20	2	2.3	74.7
21-25	3	3.4	78.2
26-30	3	3.4	81.6
31-35	3	3.4	85.1
36-40	3	3.4	88.5
41-45	4	4.6	93.1
46-50	2	2.3	95.4
51-55	1	1.1	96.6
56-60	3	3.4	100.0
Total	87	100.0	N/A

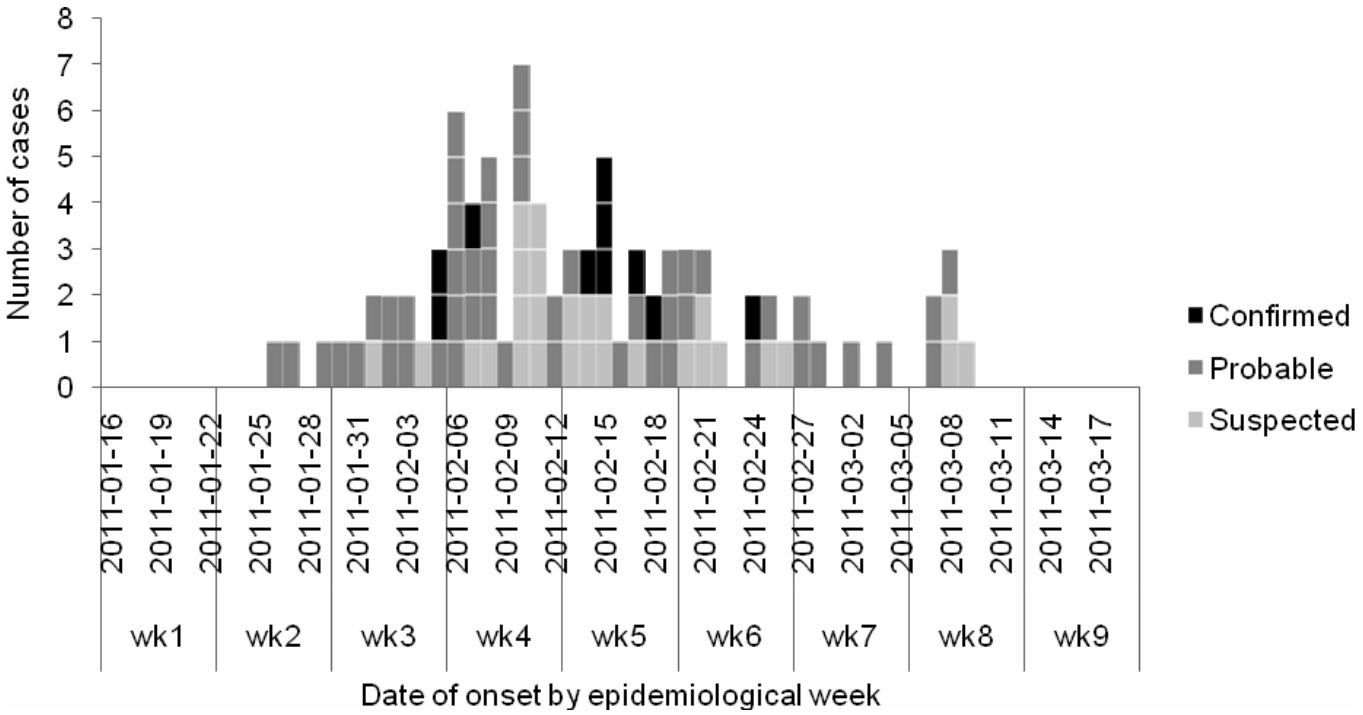

Figure 1: Number of enterovirus cases by date of onset by epidemiological week, Mpumalanga Province, 26 January to 9 March 2011 (n=87)

Discussion

This was one of the largest outbreaks of enteroviral meningitis described in South Africa, alongside the 2001 outbreak of echovirus-3 in Western Cape Province involving 90 children who attended a summer camp.[4] Illness in this Mpumalanga Province enteroviral outbreak was mild with no complications from infection reported.

Apart from the clustering in time and geography, as well as the family and school clusters, no other common risk exposures could be identified in the Mpumalanga Province outbreak. The public health response included re-enforcing good hygiene and other infection prevention and control methods, as the virus is spread primarily by the feaco-oral route.[1] Enteroviruses can survive on environmental surfaces for long periods of time, allowing transmission by touching contaminated surfaces and then exposing the eyes, nose or mouth. Infection is associated with a wide range of clinical syndromes including; asymptomatic infection (majority of cases), aseptic meningitis, encephalitis, myocarditis, myositis, acute hemorrhagic conjunctivitis, herpangina, hand-foot-and-mouth disease, and respiratory infections.[1]

Outbreaks of enterovirus have been documented previously by the NICD in November 2009 in the Western Cape Province[5], as well as in February 2010 in the Northern Cape Province[6], both associated with illness in younger people. The true incidence of enterovirus infection or disease is unknown. The incidence of enteroviral infections is highest in summer and autumn in temperate climates, but year-round in tropical climates.[1]

Recommendations

Prevention of further cases with regard to the outbreak was focused on providing health education and improving hygiene and sanitation. It was recommended that the public health response should include sharing specific guidance on infectious disease control in closed settings and reinforcing public health messages around personal hygiene and hand washing to prevent spread.

Acknowledgements

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MOLECULAR EPIDEMIOLOGY OF HUMAN METAPNEUMOVIRUS, 2009-2010

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Introduction

Ten years ago a Dutch group identified a novel and clinically important respiratory virus, human metapneumovirus (hMPV) belonging to the *Pneumovirinae* subfamily of *Paramyxoviridae*.[1] Human respiratory syncytial virus (RSV) was until then the only known human pathogen of this subfamily. Although not as common as RSV, hMPV is found worldwide with a seasonal distribution and clinical symptoms similar to that of RSV. Occurring predominantly in the winter season, hMPV is responsible for a range of clinical symptoms from mild upper respiratory tract infections (URTI) to more severe lower respiratory tract infections (LRTI), including pneumonia and bronchiolitis that may result in infants and young children being admitted to the ICU.

Genetic analysis of the hMPV genome has identified 2 genotypes, A and B, which differ by 15% at the nucleotide level.[2] These genotypes can be further separated into sub-genotypes, A1, A2, B1, and B2. During seasonal epidemics, different genotypes may co-circulate with one genotype predominating. The predominant genotype varies from season to season. There is conflicting evidence on whether different genotypes have specific disease associations.

A number of molecular epidemiological studies of hMPV have been undertaken in the South African setting, including studies examining its prevalence in hospitalised children, HIV-infected infants with respiratory illness and children with acute wheezing.[3,4,5,6,7,8] But no study has been done since 2005.

In April 2009 the NHLS diagnostic virology laboratory at Groote Schuur Hospital introduced a multiplex RT-PCR assay for screening samples for common respiratory viruses, including hMPV. The assay detected both genotypes A and B of hMPV. This was the first time that routine diagnostic samples were tested for hMPV in South Africa.

This study was undertaken to determine the role of hMPV as a cause of severe acute respiratory infections in hospitalised infants in the Western Cape Province. Data was collected over a 20 month period from April 2009 to December 2010, over 2 consecutive hMPV seasons.

Methods

From April 2009 to December 2010 respiratory samples mainly from infants with severe acute respiratory infections were screened by the commercial Seeplex RV7 RT-PCR assay as per manufacturer's instructions (Seegene Inc., South Korea). hMPV-positive samples detected using the screening assay were amplified using primers to the fusion gene, a phylogenetically informative region, as previously described.[3] The PCR products were sequenced directly using both forward and reverse nested primers by Dye Terminator sequencing (Applied Biosystems, Foster City, CA, USA). A phylogenetic tree was constructed using reference sequences from the Genbank database and rooted with avian metapneumovirus (AMPV).

Results

Of the 4911 samples screened over the 20 month period, 198 (4,0 %) hMPV positive samples were identified. hMPV was mainly found in infants and young children under 3 years of age and 57,8 % infections were in infants less than 6 months of age (Figure1). The prevalence of hMPV was strongly seasonal. Over the 20 month period monitored, hMPV was predominantly detected between the months of August and October although there was sporadic detection of hMPV during other times of the year (Figure 2). Interestingly, the peak for hMPV detection shifted from September/October in 2009 to August/ September in 2010.

Based on phylogenetic analysis of 104 hMPV positive samples (Figure 3), the predominant hMPV genotype detected was A2 (80/104, 76, 9%) followed by B1 (16/104,

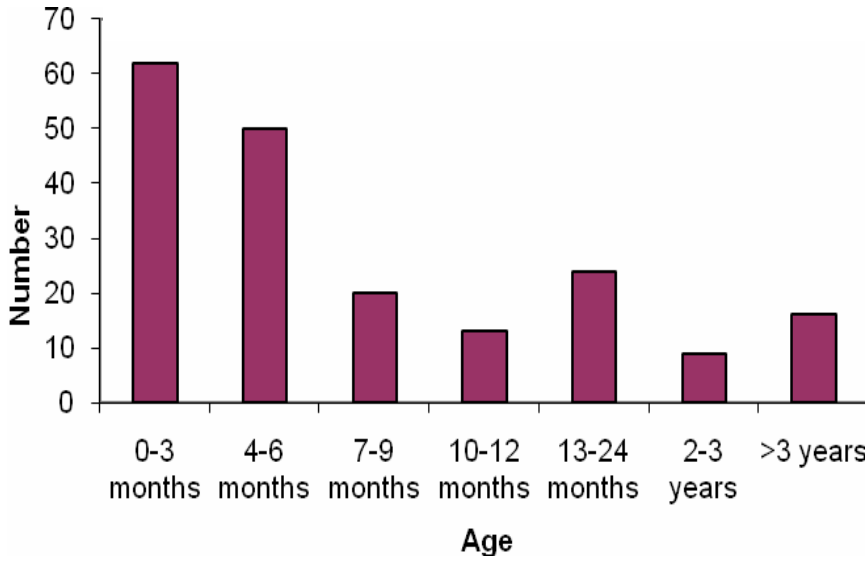

Figure 1: Age distribution of infants infected with human metapneumovirus

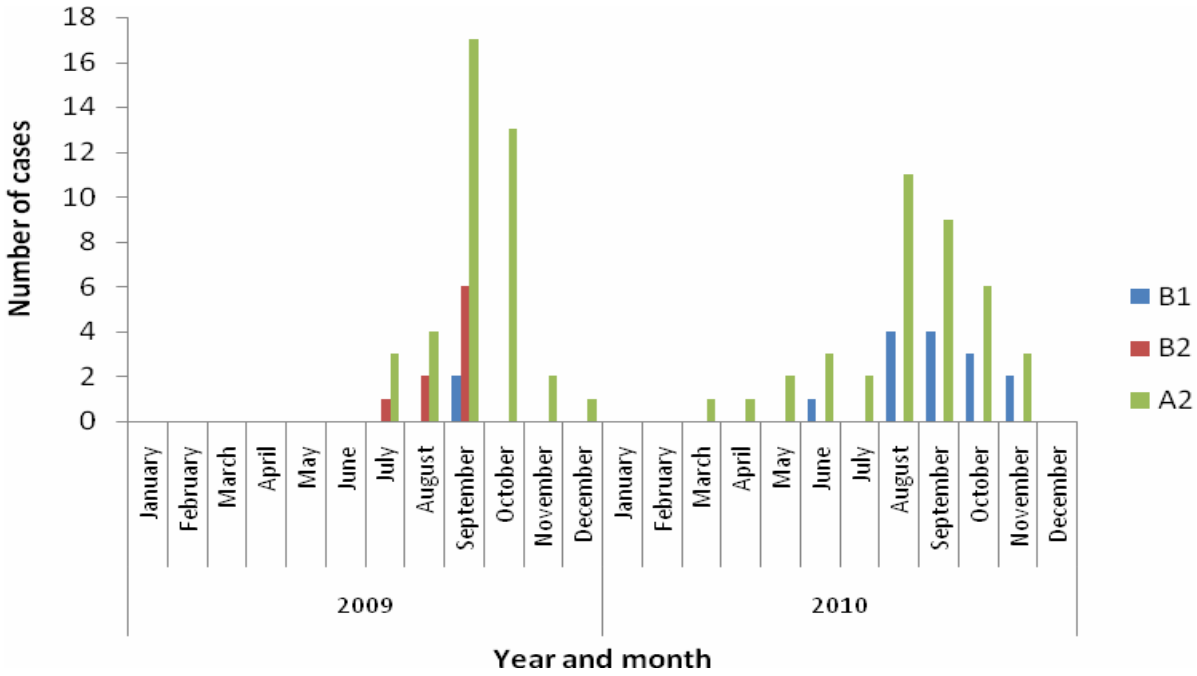

Figure 2: Distribution of human metapneumovirus genotypes (n = 104) from April 2009 and December 2010.

15, 4%) and B2 (8/104, 7, 7%). No genotype A1 was found.

Genotype A2 was detected throughout both the 2009 and 2010 seasons. However, co-circulation of genotype B viruses occurred at low frequency in both seasons. In 2009 both genotypes B1 and B2 co-circulated with A2 early in the season (from July to September), while in 2010 only B1 co-circulated with A2. Genotype B1 was detectable throughout the hMPV season in this year.

Based on the clinical data captured from request forms, the commonest clinical presentation in infants with hMPV was pneumonia, bronchiolitis or lower respiratory tract infection. Clinical information was available on 151/198 (75%) of hMPV-positive patients. Lower respiratory tract infection was specifically indicated in 124/151 (82.1%) instances. Other co-morbidities included HIV infection or exposure, congenital cardiac abnormalities and previous prematurity.

Co-infection with hMPV and one or more other respiratory viruses was identified in 63/198 (32%) cases. Rhinovirus A was the commonest co-pathogen detected, present in 37 instances. Co-infection with adenovirus was detected in 14 infants and hMPV, adenovirus and rhinovirus was detected in 2 infants. In one instance a patient was co-infected with 4 respiratory viruses, namely adenovirus, RSV, influenza A and hMPV.

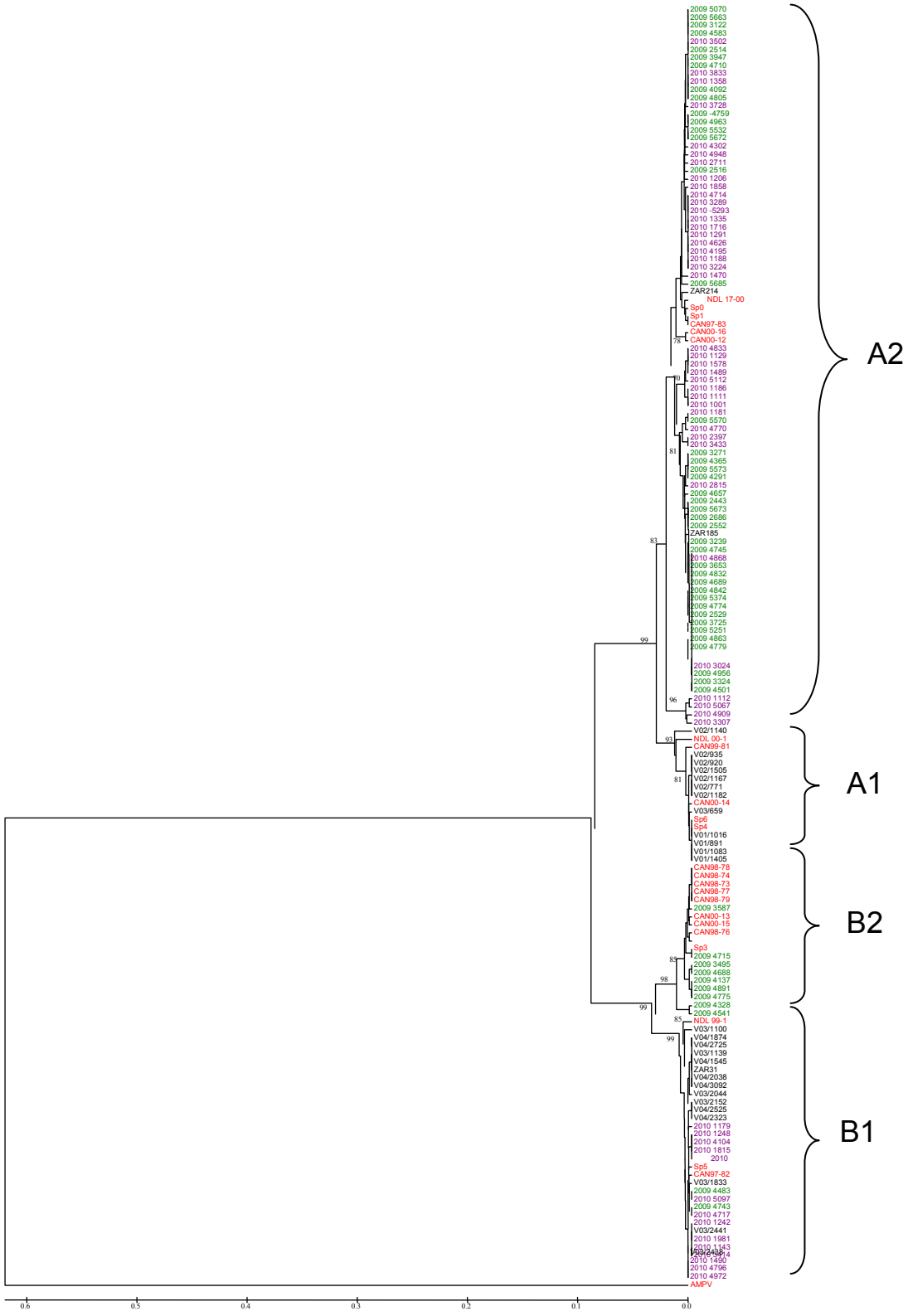

Figure 3: Phylogenetic analysis of nucleotide sequences from the partial fusion gene. Reference sequences from GenBank are indicated in red. Sequences from 2009 (green), 2010 (purple) and South African sequences from other years (black) are indicated. The tree is rooted with avian metapneumovirus.

All hMPV-infected children admitted to ICU were infected with hMPV genotype A2.

Discussion

The epidemiology of hMPV in South Africa is less well understood than for the other common respiratory viruses as this virus has not been easy to detect with routine viral culture techniques and hence we do not have many years worth of diagnostic data to use to determine its pathogenic significance in our context. The advent of molecular testing has made it possible for us to evaluate the prevalence and significance of this important cause of infant respiratory tract disease in our community. Based on data collected over two winter seasons the characteristic features of this virus have emerged. hMPV was an uncommon, but significant cause of severe lower respiratory tract infections in infants, accounting for 4% virus-positive isolates. Infection was most common in young infants with 85% of infections in children under 2 years and >55% of hMPV infections in infants below six months of age. The commonest clinical presentation was lower respiratory tract infection (pneumonia or bronchiolitis). Other reported co-morbidities in hospitalised infants with hMPV included HIV exposure or infection, congenital cardiac abnormalities and previous prematurity.

Co-infection with hMPV and one or more other respiratory viruses was identified in 63/198 (32%) cases. Rhinovirus A was overwhelmingly the commonest co-pathogen detected in hMPV-infected infants. The high association between these 2 viruses is likely to be due to the fact that the seasonal peaks (early spring) of both viruses co-incided during the 2009 and 2010 winter seasons. Adenovirus was the second commonest co-pathogen and other respiratory viruses were also detected at low frequency. While some of these co-infections may have been community acquired, nosocomial transmission of respiratory viruses is very common and it is very likely that some of these infants acquired additional infections during their hospital stay.

In temperate climates, hMPV typically has a seasonal peak, occurring in late winter to early spring. Our data shows a similar epidemiology. In both years in this study isolations began to increase (in July 2009 and May 2010) as the RSV season waned. Detection peaked in September/October in both years. This seasonal epidemiology is very similar to other temperate countries and is compatible with findings of previous South African studies. During any one season multiple hMPV genotypes may circulate, but usually one predominates. The circulation of hMPV genotypes in South Africa has been documented in several studies and data is available over a consecutive six year period, from 2000 to 2005.[3,4,5,8] During this time the predominant genotype changed from B2 in 2000 to A1 in 2001 and 2002, with B1 predominating in 2003, 2004 and 2005. In both 2009 and 2010 (the period of the current study), genotype A2 was the predominant genotype detected in clinical samples. Genotypes B1 and B2 co-circulated with A2, but were infrequently detected. This complex pattern of hMPV genotypes changing over time is mirrored globally, with the same genotype predominating throughout the world in one year and in subsequent years the minor co-circulating genotype, which may vary from region to region, becoming a dominant genotype. Viral evasion of herd immunity may explain these fluctuating circulation patterns.

To conclude, this study based in Cape Town, confirms the characteristic seasonality and disease association, previously reported for this virus: hMPV is an uncommon, but important cause of severe lower respiratory tract infections in local infants. The prevalence of infection was highest in the very young. Confirming the presence of a respiratory virus in the clinical context of lower respiratory infection provides diagnostic information and can assist patient management by reducing the use of unnecessary antibiotics and the need to search for other pathogens to account for the patient's clinical condition.

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UPDATE ON CORPORATE DATA WAREHOUSE-DERIVED MDR– AND XDR-TB STATISTICS FOR EIGHT PROVINCES IN SOUTH AFRICA, JANUARY 2007 TO 30TH JUNE 2011

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In the November 2010 issue of this bulletin[1] an account was given of the impact of the introduction of the of the PCR-based GenotypeMTBDRplus line probe assay (Hain Lifescience GmbH, Nehren, Germany) into the National Tuberculosis Control Programme (NTBCP) of South Africa on the volumes of tuberculosis (TB) culture and drug susceptibility tests (DSTs) performed during the four-year period of roll-out of this line probe assay (LPA). In the same article the latest statistics derived from data generated by the Corporate Data Warehouse (CDW) of the National Health Laboratory Service (NHLS) on laboratory-determined multidrug-resistant TB (MDR-TB) and extremely drug-resistant TB (XDR-TB) were recorded for all the provinces in South Africa except KwaZulu-Natal which up to mid-2011 was not equipped to electronically transfer historical data on laboratory results.

Further attempts to improve CDW statistics on new MDR-/XDR-TB cases occurring annually using a revised algorithm resulted in adjustments of figures given in the earlier bulletin[1] and these amended data on MDR- and XDR-TB prevalence from 2007 to 30th June 2011 will be presented here. The impact of the introduction of the MTBDRplus assay (Hain LPA) for the rapid screening for MDR-TB cases on the performance of culture-based tests including DST used on its own or in combination with Hain LPA in cases where the genetically-based LPA results were confirmed by phenotypic DST will also be illustrated by CDW data used to determine the MDR-TB status of patients. Data related to the roll out of the GeneXpert or Xpert MTB/RIF (Cepheid, Sunnydale, CA) which is being phased into the NTBCP since early 2011 and was targeted to be enrolled, at 23 sites in 10 "hotspot" regions with a particularly high prevalence of TB by World TB day on 24th April 2011 will not feature in the present report.

Methodology of data retrieval

Laboratory-generated data captured on the NHLS laboratory information management system (DISA) have been "cleaned" to provide as accurate as possible information from the Corporate Data Warehouse (CDW) on new cases of smear microscopy-positive, culture-confirmed TB as well as MDR- and XDR-TB cases diagnosed per province during the period 2007 to 30th June 2011 at NHLS laboratories, care being taken to ensure that information is accurate and relates to new TB , MDR- and XDR-TB cases by eliminating as far as possible duplication of cases on the one hand or an underestimation of cases on the other. The latter situation may occur when DSTs have been performed on more than one patient with similar identification characteristics and were erroneously counted as only one case. An improved data processing algorithm was used for this purpose and as a result, annual MDR-/XDR-TB figures reported here would differ to some degree from those reported in a previous issue of the Bulletin[1]. As was the case in 2010, information from KwaZulu-Natal is not included in the present communication but is now being integrated and will be available in future reports.

This report records the numbers of new TB cases from the 8 provinces and the rates per province based on numbers of cases diagnosed by smear microscopy and culture, as well as MDR- and XDR-TB detected annually in these provinces. With the exception of one laboratory in the Western Cape which used the Middlebrook agar method, conventional DST to determine isoniazid and rifampicin resistance in patients' isolates was performed by the MGIT 960 system (Becton Dickinson, Sparks, Md, USA). As mentioned earlier, with the introduction of the Hain LPA technology, the MDR-TB status of some patients was determined by LPA on its own or in others by both Hain LPA and conventional DST. In addition, based on resistance to ofloxacin and to one of the injectable agents, kanamycin, amikacin or capreomycin, the numbers of new XDR-TB cases determined by the MGIT 960 system are also presented.

The effect of the introduction of Hain LPA technology into the National Tuberculosis Control Programme (NTBCP) on the utilization of LPA and conventional DST for the determination of MDR status in the NTBCP over the four-and-a-half-year period will also feature in this article, using data obtained from the CDW.

New TB patients diagnosed annually by smear microscopy and culture

CDW data on new patients with laboratory-determined TB during the period January 2007 to 2010 in 8 provinces are recorded in Table 1.

Based on CDW data, more than 700 new cases per 100 00 population were recorded in 2 provinces, Western Cape and Eastern Cape, while all 8 provinces have rates in excess of 300 with a mean rate of 557 per 100 000.

New culture-positive TB patients and MDR- and XDR-TB cases in eight provinces based on data retrieved from CDW

Following the publication of MDR- and XDR-TB findings from the CDW in 2010[1], resistance statistics for the period 2004 to the present were revised through an improved processing algorithm to obtain more accurate statistics. The most recent CDW-generated data are recorded in Table 2.

Table 1: New tuberculosis patients diagnosed by smear microscopy or culture over period 2007-2010 in 8 provinces

Province	Population[2] X 10^6	Microscopy	Culture	Total	Rate* X 10^{-5}
Eastern Cape	6.7	98601	98384	196985	730
Free State	2.8	44524	23927	68451	606
Gauteng	11.2	43629	146231	189860	424
Limpopo	5.4	56839	15578	72417	333
Mpumalanga	3.6	44996	36210	81206	561
North-West	3.2	50654	29416	80070	625
Northern Cape	1.1	898	29890	30788	697
Western Cape	5.2	28914	127901	56815	750
All 8 provinces	39.3	369055	507537	876592	557

*Annual rates of new TB cases per 100 000 population in provinces

Table 2: New culture-confirmed TB, MDR- and XDR-TB patients identified annually in 8 provinces from 2007-2011*

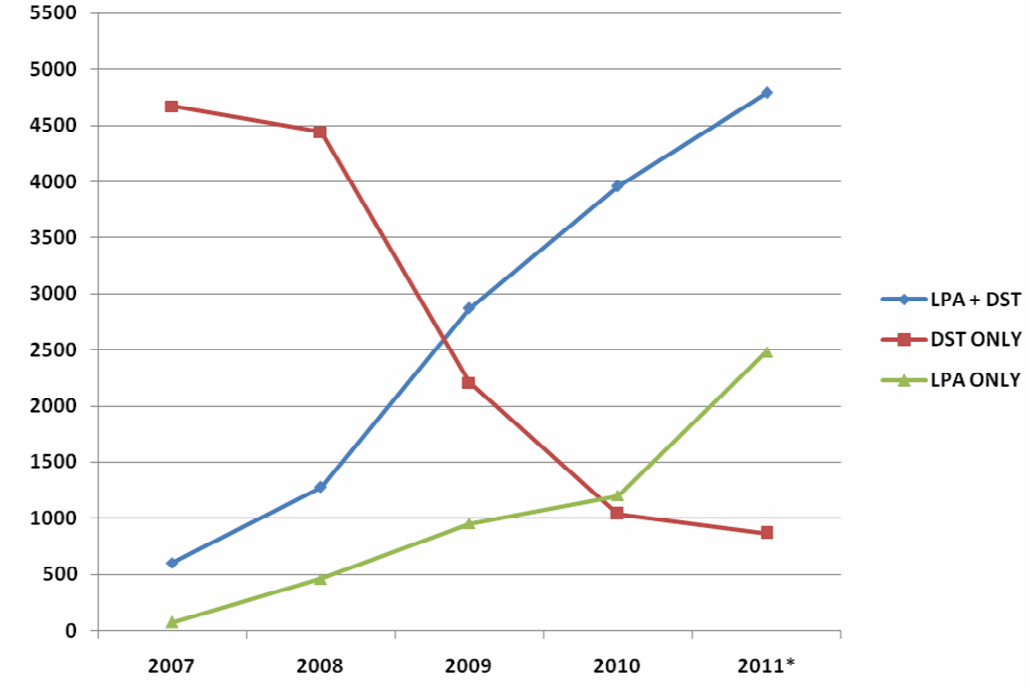
Province	MDR/XDR	2007	2008	2009	2010	2011*	Total(%)
Eastern Cape	New TB†	18169	24723	25486	30006	13808	112192
	MDR	1253	1567	1639	2011	1161	7631 (6.8)‡
	XDR	127	195	196	235	154	907 (11.9)
Free State	New TB	6740	5914	6446	4827	2644	26571
	MDR	179	342	261	367	180	1329 (5.0)
	XDR	5	9	10	7	9	40 (3.0)
Gauteng	New TB	32800	38949	36792	37690	17047	163278
	MDR	1063	1031	1264	1116	794	5268 (3.2)
	XDR	40	39	60	64	29	232 (4.4)
Limpopo	New TB	2754	3490	4276	5058	1931	17509
	MDR	106	176	196	185	151	814 (4.6)
	XDR	3	3	9	2	4	21 (2.6)
Mpuma-langa	New TB	5302	7910	13090	9908	5206	41416
	MDR	549	691	421	591	405	2657 (6.4)
	XDR	13	13	16	27	8	77 (2.9
North West	New TB	6629	7848	7307	7632	3220	32636
	MDR	372	305	387	210	120	1394 (4.3)
	XDR	3	10	12	3	3	31 (2.2)
Northern Cape	New TB	6622	7445	7738	8085	3611	33501
	MDR	203	244	353	306	261	1367 (4.1)
	XDR	15	29	36	33	30	143 (10.5)
Western Cape	New TB	29138	30484	34099	34180	16116	144017
	MDR	1623	1818	1510	1412	999	7362 (5.1)
	XDR	54	62	76	83	44	319 (4.3)
Total	New TB	108154	126763	135234	137386	63583	571120
	MDR	5348	6174	6031	6198	4971	28722 (5.0)
	XDR	260	360	415	454	281	1770 (6.2)

* Numbers of new cases per province in 2011up to 30th June 2011.
† New culture-positive TB cases irrespective of drug susceptibility status identified annually by culture
‡ Percentage of MDR-TB cases identified annually in relation to new culture-positive patients p.a.
§ Percentage of XDR-TB cases identified annually in relation to new MDR-TB cases p.a.

There was an overall increase of 15.9% from 5348 to 6198 MDR-TB cases from 2007 to 2010 and a 74.6% increase in XDR-TB cases from 260 to 454 in eight of the nine provinces of South Africa during the same period. Similar increasing trends in numbers of cases with fluctuation in some provinces were generally evident in all 8 provinces for both MDR- and XDR-TB cases (Table 2).

Laboratory approaches used for determination of MDR- TB status following introduction of Hain LPA

Hain LPA technology used for the rapid diagnosis of MDR-TB in smear-positive patients was phased into the NTBCP since the second half of 2006 and the utilization of laboratory methods as a result is illustrated in the figure below.



*Projected numbers of tests for 2011 based on numbers performed up to 30th June 2011
LPA = Line probe assay
DST = Drug susceptibility tests

Figure: Drug susceptibility tests performed for MDR-TB detection in 8 provinces in South Africa following the introduction of rapid molecular-based Hain LPA technology.

The increasing numbers of MDR-TB cases detected since 2006 by Hain LPA on its own or in conjunction with confirmatory MGIT DST, and the overall effect of its introduction into 8 provinces on the time consuming MGIT DST is shown in the figure while the trends in the Eastern Cape, Gauteng and Western Cape feature in Table 3. Also shown is a corresponding decline in the number of patients diagnosed by MGIT DST performed on its own. There was however no real decrease in the overall numbers of DSTs performed for MDR-TB identification as in many instances resistance results on initial screening with Hain LPA were confirmed by phenotypic DST performance (see blue and green graphs in Figure and data in Table 3 [see later]).

In Table 3 the utilization of the culture- and molecular-based tests in 8 provinces is recorded to illustrate trends in the use of methods for MDR-TB detection, as well as the numbers of tests performed in the Eastern Cape, Gauteng and Western Cape for this purpose (these provinces detected the largest numbers of MDR- and XDR-TB cases in the country [excluding KwaZulu-Natal]).

Trends similar to those illustrated in Figure 1 for the 8 provinces combined are demonstrated in Table 3 for 3 individual provinces with high TB burdens in the country.

Although the total numbers of LPAs per annum in the 8 provinces increased as expected, the total numbers of conventional DSTs performed annually in these provinces and in the 8 provinces collectively did not change much during the survey period.

Changing volumes of cultures, MGIT drug susceptibility tests and molecular-based line probe tests performed for diagnosis and determination of drug resistance status since the introduction of Hain LPA.

The effect of the introduction of the Hain LPA is illustrated in Table 4 and shows the escalation of Hain LPA tests performed over the 2007 – 2010 period from 5980 to 92634, an increase in the use of cultures from 608993 to 917855 and a decrease in DSTs from 67241 to 33684. The changes as reported in this bulletin in 2010[1] are also shown in Table 4 (numbers in brackets). The original CDW figures for this period showed very similar trends compared with the present revised 2011 version although the projected numbers for 2010 in the 2010 bulletin[1] clearly underestimated the final numbers of cases recorded for that year (see Table 4).

Table 3: Numbers of MDR-TB cases detected in selected provinces according to laboratory test(s) performed for demonstration of MDR status from 1st January 2007 to 30th June 2011.

Method(s)	Year	Numbers of MDR-TB cases diagnosed by method(s) in Eastern Cape	Gauteng	Western Cape	South Africa§
DST only	2007	1212	938	1176	4671
	2008	1316	834	676	4439
	2009	699	577	46	2208
	2010	187	183	9	1039
	2011*	113	157	22	433
LPA only	2007	0	6	68	74
	2008	129	32	288	459
	2009	499	87	293	954
	2010	543	235	248	1202
	2011*	422	300	180	1243
LPA + DST†	2007	41	119	379	603
	2008	122	165	854	1276
	2009	441	600	1171	2869
	2010	1281	698	1155	3957
	2011*	626	337	797	2395
Total DST¶	2007	1253	1057	1555	5274
	2008	1438	999	1530	5715
	2009	1198	1177	1187	5077
	2010	1468	881	1164	4996
	2011*	739	494	819	2828
Total LPA¶	2007	41	125	447	677
	2008	251	197	1142	1735
	2009	940	687	1464	3823
	2010	1824	933	1403	5159
	2011*	1048	637	977	3638
All methods	2007	1253	1063	1623	5348
	2008	1567	1031	1818	6174
	2009	1639	1264	1510	6069
	2010	2011	1116	1412	6198
	2011*	1161	794	999	4071

§ All provinces excluding KwaZulu-Natal; * Numbers of tests performed up to 30th June 2011; † DST by

Table 4: Numbers of cultures, drug susceptibility tests and Hain LPAs performed collectively by eight provinces over period 2007- 30th June 2011

Year	Cultures (from reference[1])*	Drug susceptibility Tests	Hain LPA tests
2007	608993 (603131)	67241 (65809)	5980 (5963)
2008	780338 (774651)	60696 (60147)	23600 (236128)
2009	842539 (837338)	39877 (40205)	62280 (61575)
2010	917855 (687642)**	33684 (22840)**	92634 (65175)**
2011***	440838	7942	45115

* Figures in brackets throughout this table are CDW data published in reference[1]
** Figures in brackets for 2010 were projected from numbers of tests performed up to 15th October 2010[1]
*** Numbers of tests performed up to 30th June 2011

Discussion

The annual rates of new TB cases of >300 per 100 000 recorded here for each of the 8 provinces, and the >700 per 100 000 for the Western and Eastern Cape provinces are high but lower than the 2009 WHO figure of 970 (789 – 1168) for South Africa.[3] The corresponding 2009 figures of the present report are 784, 750 and 748 per 100 000 respectively for the Eastern, Northern and Western Cape provinces with a mean of 588/100 000 for the 8 provinces combined.

The annual MDR-TB prevalence figures for the 8 provinces combined were >6000 for each of the years 2008 to 2010. During 2009, 6031 MDR-TB cases were recorded here for the 8 provinces (see Table 2), compared with South African figures of 5200 (4300 – 6600) for 2009 reported in the WHO Global Tuberculosis Control Report 2010[3]. The figures of the present report are higher than the South African statistics published by WHO especially if one takes into consideration that the present statistic does not include cases from KwaZulu-Natal. Of interest is the ratio of MDR-TB patients to new TB cases of 5.0% which is appreciably higher than the 1.8% (1.5% – 2.3%) of the 2002 drug resistance survey for South Africa[4] suggesting a deteriorating situation which may be due to deficient adherence to treatment and/or transmission of MDR-TB with consequent increase in primary MDR-TB cases, or as a result of increased drug susceptibility testing.

More XDR-TB cases have been captured in the present report compared with that of 2010 [1]. This is the result of the improved CDW algorithm used to retrieve cases with the correct resistance profiles denoting XDR-TB status. There was also a steady increase in the number of XDR-TB cases over the study period while the XDR-TB/MDR-TB ratio of 6.2% suggests problems with the management of MDR-TB which requires treatment with less than optimal second-line anti-TB drugs in hospital and clinic settings where close contact with patients with drug-resistant TB, some co-infected with HIV, would be conducive to transmission, as was the case in the Tugela Ferry outbreak in KwaZulu-Natal.[5]

The reliability of the prevalence figures for TB, MDR-TB and XDR-TB recorded in the present report is subject to limitations inherent to CDW information systems and will need to be verified with carefully collected prospective reporting. At this stage caution should be exercised in the use and interpretation of the present CDW-derived statistics.

Of interest and relevant to the present study is the extent to which the data following the introduction of a new algorithm for providing more reliable information from this source compares with those from the 2010 report [1]. Table 4 above and Table 5 provide examples of differences between the two CDW-based reports.

With only 2 exceptions (drug susceptibility testing 2009 [Table 4] and new MDR-TB cases for 2009 [Table 5]) application of the new algorithm resulted in more cases/ tests being captured. This may have relevance to possibly less efficient capturing of new microcopy-determined new cases of TB, explaining the lower CDW statistics (see Table 1) compared with those reported for South Africa by WHO[3].

The present system which generated the statistics of the present report, constitutes an attempt at improving the previously used algorithm and is part of an ongoing process of “cleaning” CDW information The differences illustrated above could be regarded as an indication of the extent of improvement achieved. In previous years problems were encountered with accessing reliably information on XDR-TB cases which hopefully have now been resolved and this probably accounts for the changes encountered with this group of patients.

Despite the concern expressed above, there is no doubt that the CDW facility has tremendous potential for providing extremely useful information to the NTBCP and to other interested persons and institutions including agencies involved in TB research. Future improved use of the CDW will include accessing information on basic demographics, age and sex breakdowns with trends and confidence intervals, as well as construction of maps of the geographical distribution of TB, MDR-TB and XDR-TB cases. Such data could be linked to HIV status of patients. It is also intended to construct more detailed trend data from 2004 with indicators to mark impact of introduction of new technologies and policy changes. The recent incorporation of the NHLS TB laboratories of KwaZulu-Natal province into the CDW network will add significantly to the benefits of the CDW information systems.

Table 5: Example of the extent of differences between the 2010 and the present CDW-based information

Total MDR- and XDR-TB cases	2007	Difference (%)	2008	Difference (%)	2009	Difference (%)
MDR cases 2011	5348	511	6174	556	6031	-631
MDR cases 2010	4837	(10.6%)	5618	(9.9%)	6662	(-9.5%)
XDR cases 2011	260	26	360	74	415	78
XDR cases 2010	234	(11.1%)	286	(25.9%)	337	(23.1%)

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Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 30 June 2010/2011*

Disease/Organism	Cumulative to 30 June, 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	665	227	1015	577	262	364	29	293	228	3660
	2011	572	183	938	524	246	310	26	294	195	3288
Haemophilus influenzae, invasive disease, all serotypes	2010	24	12	97	18	3	7	7	5	50	223
	2011	14	11	71	18	1	14	5	1	45	180
Haemophilus influenzae, invasive disease, < 5 years											
Serotype b	2010	3	3	13	1	1	3	3	1	9	37
	2011	2	3	10	7	0	1	3	1	7	34
Serotypes a,c,d,e,f	2010	0	0	5	0	1	1	0	0	6	13
	2011	0	1	8	1	0	0	0	0	3	13
Non-typeable (unencapsulated)	2010	1	1	33	3	0	0	1	1	9	49
	2011	0	2	12	3	0	1	0	0	7	25
No isolate available for serotyping	2010	5	2	10	0	1	1	0	1	3	23
	2011	3	2	15	1	0	4	0	0	2	27
Measles	2010	1251	533	921	3605	272	1717	262	683	1514	10758
	2011	1	2	32	23	1	1	8	6	6	80
Neisseria meningitidis, invasive disease	2010	12	12	72	10	4	8	13	5	24	160
	2011	17	7	68	8	3	7	5	2	17	134
***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	2	0	0	3	3	1	0	0	0	9
	2011	0	0	0	0	3	0	0	0	0	3
**Rubella	2010	193	59	95	239	21	110	21	110	153	1001
	2011	50	3	123	69	66	69	33	50	50	513
Salmonella spp. (not typhi), invasive disease	2010	25	12	197	34	8	9	7	5	44	341
	2011	8	11	135	36	3	18	4	6	35	256
Salmonella spp. (not typhi), isolate from non-sterile site	2010	124	31	380	122	7	46	4	25	88	827
	2011	80	17	307	107	10	34	16	11	128	710
Salmonella typhi	2010	3	1	18	7	1	6	0	0	4	40
	2011	7	1	12	6	1	5	0	1	10	43
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	133	34	413	79	6	25	14	13	224	941
	2011	100	24	284	70	6	12	16	6	239	757
Streptococcus pneumoniae, invasive disease, all ages	2010	195	118	837	208	46	107	44	69	295	1919
	2011	142	101	650	160	29	83	33	82	286	1566
Streptococcus pneumoniae, invasive disease, < 5 years	2010	34	26	218	52	9	27	26	14	88	494
	2011	25	22	144	33	6	23	9	13	65	340
Vibrio cholerae O1	2010	0	0	1	0	0	0	0	0	0	1
	2011	0	0	0	0	1	0	0	0	0	1
Viral Haemorrhagic Fever (VHF)											
Crimean Congo Haemorrhagic Fever (CCHF)	2010	0	1	0	0	0	0	2	0	0	3
	2011	0	0	0	0	0	0	0	0	0	0
****Other VHF (not CCHF)	2010	17	122	0	0	0	0	72	7	3	222
	2011	17	3	0	0	0	0	2	0	14	36

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 2010/2011*

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	2010	26	8	33	31	20	18	1	12	10	159
	2011	33	14	55	45	46	25	5	7	10	240
Laboratory Programme for the Comprehensive Care, Treatment and Management Programme for HIV and AIDS											
CD4 count tests											
Total CD4 count tests submitted	2010	216,965	110,014	423,996	552,115	151,054	178,220	34,253	136,044	140,798	1,943,459
	2011	200,687	117,596	393,564	495,013	135,340	154,255	31,773	124,976	116,997	1,770,201
Tests with CD4 count < 200/µl	2010	58,784	28,221	123,151	106,374	44,582	49,653	8,362	35,462	28,527	483,116
	2011	63,390	34,063	127,355	121,698	41,934	46,291	9,240	35,880	28,995	508,846
Viral load tests											
Total viral load tests submitted	2010	73,509	41,215	173,694	237,347	49,325	56,764	12,796	51,419	65,965	762,034
	2011	68,818	34,686	170,324	165,651	44,341	48,149	12,271	49,263	53,586	647,089
Tests with undetectable viral load	2010	48,650	32,776	120,236	173,393	34,590	39,244	8,859	33,854	51,153	542,755
	2011	45083	22593	124459	121803	31774	37454	7346	33666	41414	465,592
Diagnostic HIV-1 PCR tests											
Total diagnostic HIV-1 PCR tests submitted	2010	17,239	7,838	33,296	41,600	12,116	13,247	2,630	10,198	9,452	147,616
	2011	14,537	6,960	30,073	38,504	10,772	11,306	2,275	8,489	8,964	131,880
Diagnostic HIV-1 PCR tests positive for HIV	2010	1,059	467	2,049	2,322	727	707	134	607	385	8,457
	2011	1,234	653	3,088	3,270	1,127	1,138	218	807	553	12,088

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

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