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FOREWORD

The recent outbreak of Rift Valley Fever in South Africa serves as a reminder of the risk of re-emergence of pathogens which have not been identified in South Africa for many years. Influenza is another emerging pathogen and the recent identification of widespread resistance to the antiviral drug oseltamivir in Europe highlights the importance of strong disease surveillance systems. The conjunctivitis outbreak in the Northern Cape Province is an example of the pitfalls of inadequate specimen collection in outbreaks of infectious disease. The article highlighting the Laboratory Programme for the Comprehensive Care, Management and Treatment Programme for HIV and AIDS is an excellent example of how vertical laboratory programmes can provide indirect evidence for the success of public health programmes. This programme provides impressive evidence of how sophisticated laboratory services can be rapidly upscaled to support priority public health programmes. Strong surveillance systems and laboratory infrastructure are necessary to identify emerging diseases and outbreaks, track trends in drug resistance and support programmes promoting the health of all South Africans.

Cheryl Cohen
Editor

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RIFT VALLEY FEVER OUTBREAK IN SOUTH AFRICA, 2008

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Description of the Outbreak

From January to April 2008 multiple, small focal outbreaks of Rift Valley fever (RVF) affecting mostly captive buffalo and cattle, were reported in South Africa from Mpumalanga, Limpopo and Gauteng Provinces. The six different localities where RVF outbreaks were confirmed in animals are shown in Figure 1. Laboratory diagnoses in animals and humans were achieved by immunohistochemistry, reverse transcriptase polymerase chain reaction (RT-PCR), virus antigen detection and/or serology at the Faculty of Veterinary Science, University of Pretoria, Onderstepoort Veterinary Institute and NICD. Recent infection with RVF virus was confirmed in 13 persons: a veterinary surgeon exposed during a necropsy, five farm workers who either handled infected cattle or their tissues, four veterinary students and two technicians exposed during necropsies and a veterinary student who examined an ill calf. All developed fever and myalgia, and encephalitis was noted in two of the patients, while 5/6 patients tested manifested minor liver dysfunction. One patient remains ill, while the others have recovered fully. All infected humans had close contact with infected animals

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and/or animal tissues, which supports the contention that this mode of transmission is more common than mosquito bites.

Discussion

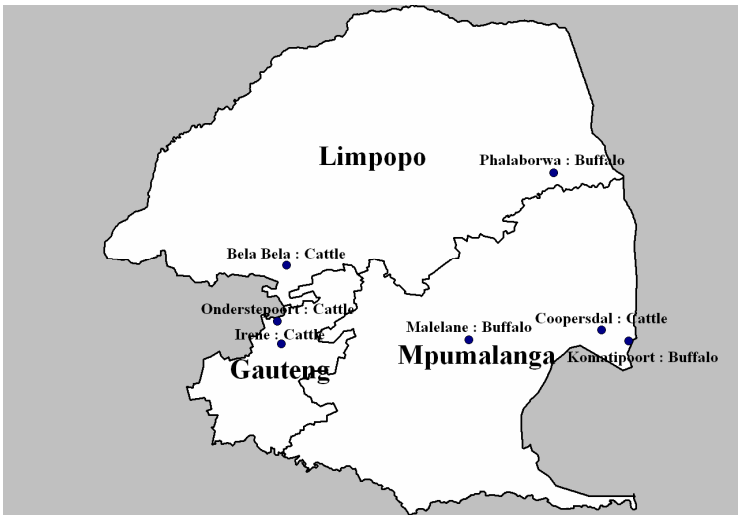
Rift Valley fever (RVF) is a zoonotic disease, caused by a mosquito-borne virus of the *Phlebovirus* genus of the family *Bunyaviridae* that was first isolated in Kenya in 1930 following an outbreak of "enzootic hepatitis" in sheep in the Rift Valley. Epizootics generally occur in 5-15 year cycles and follow droughts broken by heavy rains. Transovarial transmission of virus in the floodwater-breeding *Aedes* mosquitoes is thought to be responsible for maintenance of the virus during interepidemic periods. The zoophilic species, *Aedes mcintoshi* and *Ae. juppi*, appear to be major maintenance vectors in East and Southern Africa respectively, with *Culex* mosquitoes acting as epidemic vectors. RVF causes heavy mortality among newborn animals and abortion in pregnant animals. Recent outbreaks in East Africa, in 2006-2007, caused substantial losses of livestock in Kenya, Somalia and Tanzania.

Rift Valley fever was first reported in South Africa in 1950-1951 and it was estimated that 100 000 sheep died and 500 000 aborted. The second major and more widespread outbreak caused extensive losses of sheep and cattle in 1974-76. More than 10 000 humans were estimated to have been infected, mainly on farms. The potential lethality of the virus for man was first recognized during the 1974-76 RVF epizootic in South Africa when deaths from encephalitis and/or haemorrhagic fever with necrotic hepatitis were observed. In 1981 a small outbreak was recorded on a dairy farm in Mtubatuba, KwaZulu Natal province. In 1999 there was a small outbreak in captive-bred buffaloes in Skukuza in the Kruger National Park.

Farmers are advised to vaccinate livestock routinely but compliance is poor. A live-attenuated virus vaccine based on the Smithburn strain of RVF virus isolated from mosquitoes in Uganda in 1944, is available in South Africa for immunization of sheep and goats. Although the vaccine leads to long-term immunity, its use is associated with abortions in a small proportion of pregnant animals. The Smithburn virus was found to be inadequately immunogenic for cattle and therefore a killed vaccine was developed for use in these animals; it is safe but repeated doses are required. A formalin-inactivated cell culture-derived vaccine was developed in the USA for use in humans but was made available on a limited scale only, mainly for use in laboratory workers. Currently no vaccine is available for humans.

Transmission of RVF to humans occurs through direct contact with the blood and tissues of infected animals, or mosquito bite, and rarely through ingestion of unpasteurised infected milk. The incubation period of the disease varies from 2-8 days. Most infections in humans are asymptomatic while a number of infected persons experience a 'flu-like' illness characterized by acute onset of fever, headache, myalgia and photophobia. Biphasic illness is not uncommon. Severe disease occurs in less than 1% of infected persons, including hepatitis, encephalitis and a haemorrhagic state. Retinitis is a late complication of RVF infection. Treatment is supportive. While ribavirin has been shown to be active against the virus *in-vitro*, some treated patients survived acute illness but succumbed later to encephalitis, and use of the drug is currently considered to be contraindicated for treatment of RVF. Human to human transmission has not been documented, and isolation of patients is therefore not considered necessary. However, transmission of infection through needle stick injuries poses a risk and standard precautions should be followed.

Figure 1: Location of Rift Valley fever outbreaks in animals in South Africa, 2008



In comparison with historical epidemics in South Africa, the current outbreaks appeared to be localized and affected relatively small numbers of animals and humans. This may be due to a low level of endemicity of RVF in the country or due to recent introduction, but nevertheless holds implications for the occurrence of major epidemics should there be heavy rains in the near future. More detailed epidemiological information is needed to clarify the risk of future outbreaks.

Acknowledgements

We would like to acknowledge the support of the SAFELTP Faculty and students, and Communicable Disease Directorates of Mpumalanga Department of Health and Tshwane Metro.

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INFLUENZA UPDATE & RECOMMENDATIONS FOR THE 2008 INFLUENZA SEASON

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Recent findings - oseltamivir resistance in seasonal influenza viruses

In late January 2008 antiviral drug susceptibility testing of seasonal influenza viruses in Europe revealed that some of the A (H1N1) viruses circulating in the 2007/08 winter season were resistant to the antiviral drug oseltamivir due to a mutation in the viral neuraminidase gene.[1] By 31 January, resistant viruses had been detected in 9 countries with the highest incidences in Norway (26 of 36 or 70%) and 15 of 87 (17%) in France. Global surveillance has subsequently shown evidence of similarly resistant viruses in North America, the Far East and Australia. By early May the global incidence of oseltamivir resistant H1N1 viruses was shown to be 15% compared to less than 1% of circulating viruses in previous years.[2,3]

There is no evidence that the appearance of these new viruses are related to use of oseltamivir which is currently seemingly not widely prescribed in most European countries. The 2007/08 winter season is the first time there has been widespread and sustained transmission of such resistant viruses in the community. Low numbers of resistant viruses have been isolated before, but usually following treatment. These viruses previously have not been able to readily transmit and have rapidly disappeared. Clinical experience in Norway suggests that people who become ill with an oseltamivir resistant strain of A (H1N1) have a similar spectrum of illness to those infected with "normal" seasonal influenza A which can cause severe disease or death in vulnerable people i.e. older people, those with debilitating illnesses and the very young. At this stage the significance of these findings remains uncertain. The emergence of drug resistance in the context of limited drug use is unexpected, and the extent of future circulation is difficult to predict.

Influenza vaccination recommendations South Africa 2008[4,5]

WHO recommended vaccine formulation for the 2008 southern hemisphere season:

- A/Solomon Islands/3/2006 (H1N1) - like virus
- A/Brisbane/10/2007 (H3N2) - like virus
- B/Florida/4/2006 - like virus

Indications

1. Persons (adults or children) who are at high risk for influenza and its complications because of underlying medical conditions and who are receiving regular medical care for conditions such as chronic pulmonary and cardiac disease, chronic renal diseases, diabetes mellitus and similar metabolic disorders, and individuals who are immunosuppressed (including HIV infected persons with CD4 counts above 200/mm^3);
2. Residents of old-age homes, chronic care and rehabilitation institutions;
3. Children on long-term aspirin therapy;
4. Medical and nursing staff responsible for the care of high-risk cases;
5. Adults and children who are family contacts of high-risk cases;
6. All persons over the age of 65 years;
7. Women who would be in the second or third trimester of pregnancy during the influenza season. Pregnant women with medical conditions placing them at risk for influenza complications should be immunized at any stage of pregnancy;
8. Any persons wishing to protect themselves from the risk of contracting influenza, especially in industrial settings, where large-scale absenteeism could cause significant economic losses.

Dosage

Adults: Whole or split-product or subunit vaccine: 1 dose I.M.
Children (<12 years): Split-product or subunit vaccine: 1 dose I.M.
Children <9 years who have never been vaccinated should receive 2 doses 1 month apart
Children less than 3 years of age should receive half the adult dose on two occasions separated 1 month apart

Contraindications

1. Persons with a history of severe hypersensitivity to eggs;
2. Persons with acute febrile illnesses should preferably be immunized after symptoms have disappeared;
3. The vaccine should be avoided in the first trimester of pregnancy unless there are specific medical indications—see above indication no. 7

Timing

Vaccines should be given sufficiently early to provide protection for the winter. A protective antibody response takes about 2 weeks to develop. Should vaccination not be done in the autumn, however, it can be done at any time of the season.

Guidelines for the use of influenza antiviral agents

Anti-influenza agents are intended mainly for patients who have become seriously ill with influenza and for those with underlying illnesses. These drugs should be seen as an adjunct to vaccination, as vaccination remains the key means for the control of influenza outbreaks. Currently there are two classes of antiviral drugs, the M1 inhibitors (adamantanes) and the neuraminidase (NA) inhibitors. Recent studies have shown that a high proportion of the

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seasonal influenza A strains are resistant to amantadine and this antiviral agent is no longer recommended for use by countries like the United States of America.

Currently there are two licensed NA inhibitors, oseltamivir (Tamiflu®) and zanamivir (Relenza®) which are available for clinical use. When given early after onset of symptoms (within 48 hours) both agents reduce the duration and severity of symptoms and decrease the rate of complications such as bronchitis, pneumonia and otitis media.

Due to concerns about the possibility of resistance developing due to frequent use, it is recommended that NA inhibitors be reserved for seriously ill patients or for prophylaxis in high-risk individuals. Oseltamivir is available as a capsule (75mg) or syrup (12mg/ml). The recommended dosage for the treatment of influenza is shown in the table below and should be given for 5 days.

Table 1. Dosage of oral oseltamivir for treatment of influenza

Body Weight	Dose
<15 kg	30 mg bd
15 - 23 kg	45 mg bd
23 - 40 kg	60 mg bd
>40 kg	75 mg bd

Prophlyaxis using oseltamivir is recommended only for high-risk patients and is given for a maximum of 6 weeks.

Zanamivir is approved for the treatment of uncomplicated illness due to influenza infection in patients older then 12 years of age.

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CONJUNCTIVITIS OUTBREAK IN NIEKERKSHOOP, NORTHERN CAPE PROVINCE, 5 DECEMBER 2007 TO 14 FEBRUARY 2008

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Introduction

Conjunctivitis is any inflammation of the conjunctiva, generally characterized by irritation, itching, foreign body sensation, and tearing or discharge and is the most common eye disease worldwide.[1] Symptoms of conjunctivitis include a foreign body sensation, with watering, discharge, redness, and swelling of the lids as well as sensitivity to light and blurred vision. The commonest cause of acute viral conjunctivitis is adenovirus which can spread rapidly through communities leading to epidemics.[2] The most common causes of acute bacterial conjunctivitis are *Staphyloccocus aureus, Streptococcus pneumoniae, Haemophilus influenz*ae (especially in children) and *Moraxella catarrhalis*.[3,4] There is no good evidence of the diagnostic usefulness of clinical signs, symptoms, or both in distinguishing bacterial conjunctivitis from viral conjunctivitis.[5]

An outbreak of acute haemorrhagic conjunctivitis was reported from Ghana in 1969. This large epidemic swept over Ghana from June to October, 1969, affecting a total of 3 664 people of all ages.[6] An outbreak occurred in Nigeria in 1981, where 126 people were treated at the Eye Clinic of the Lagos University Teaching Hospital.[7] In 1985 an enterovirus outbreak caused 70 cases of conjunctivitis at a girls boarding school in Nigeria, and later spread to the community when the girls were released from the school.[8] In South Africa an epidemic of conjunctivitis occurred in March 1982, in KwaZulu-Natal, Pretoria and in Johannesburg. The data for this outbreak came from the reports received from the various health local health care services and the source of the outbreak was not verified.[9]

Described modes of transmission include contaminated fingers, medical instruments and swimming pool water.[10] The condition is highly infectious and cases should not share face towels and should wash their hands regularly. [11] Treatment requires good eyelid hygiene and the application of topical antibiotics (as determined by culture where available).[12] Pulsed-field gel electrophoresis (PFGE) is a molecular typing technique that is frequently used to determine whether epidemiologically related bacterial isolates collected during an outbreak are also genetically related.[13]

A conjunctivitis outbreak in the town of Niekerkshoop, Northern Cape Province, was reported to the health authorities in January 2008 when the nurse at the local clinic noticed an increase in the number of patients

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presenting with red and/or painful eyes. An immediate outbreak investigation was instituted with emphasis on control of the outbreak, appropriate management of cases and urgent specimen collection to identify an aetiological agent. Further epidemiological investigations were then conducted to better describe the epidemiology of the outbreak, to identify the source of infection and to identify risk factors.

Methodology

The study population for the case-control study was the community of Niekerkshoop. All cases from the line list at the clinic were included in the study. A case was defined as a person of any age residing in Niekerkshoop who presented to the clinic with red and/or painful eye(s) from 1 December 2007 to 14 February 2008. A control was defined as an asymptomatic person who lived with the case or was the next-door neighbour of the case in the same period. A structured questionnaire was used to collect demographic, clinical, and exposure history information such as sharing of towels and access to clean water. Conjunctival swabs for bacterial culture were collected from patients who were still symptomatic during the investigation. Data was entered into and analyzed in Epi info version 3.4. Odds ratios were used to measure the strength of association between risk factors. A p-value of less than 0.05 was considered statistically significant. Conjunctival swabs were submitted for both bacterial and viral culture (in viral transport medium). Specimens for bacterial culture were processed on standard media (as per Standard Operating Procedures for conjunctival specimens) at the National Health Laboratory Service (NHLS) Kimberley Laboratory. Potentially significant bacterial isolates were sent for further characterization to the Respiratory and Meningeal Pathogens Reference Unit at the National Institute for Communicable Diseases. *S. aureus* isolates recovered from swabs were analyzed by PFGE as described previously using Sma1 restriction enzyme (Roche Diagnostics GmbH, Mannheim, Germany).[13] A dendrogram showing strain similarity was constructed from the PFGE fingerprints using GelCompar™ v 4.1 software (Applied-Maths, Kortrijk, Belgium). The Tenover criteria were used to interpret similarities between strains.[14] Briefly, strains were considered to be related if their restriction patterns were identical or differed by up to three bands. Strains were considered to be possibly related or unrelated if they differed by 4-6 bands or >=7 bands, respectively. Selected specimens were sent for culture of adenovirus and PCR for enteroviruses.

Results

The population of Niekerkshoop is approximately 3500 (municipal data, 2007) and there is only one school which is attended by children between the ages of 6 and 18 years, and a day care centre which caters for children under the age of 5 years. There is one clinic situated within the town. There were no baseline data available for the number of cases of conjunctivitis seen in the period preceding the outbreak. For the period 1 December 2007 to 14 February 2008, a total of 29 people from 26 households reported signs and symptoms of conjunctivitis at the local clinic. In total, 47 controls were selected, 30 were from the same households as the cases and 17 were selected from neighbours. The date of onset for the first case was on 5 December 2007, reaching a peak 9 days later around 14 December 2007 and the last case for the period of the outbreak investigation presented on 13 February 2008 (Figure 1). Due to the Christmas holidays the clinic was closed from 16 December 2007 to 1 January 2008.

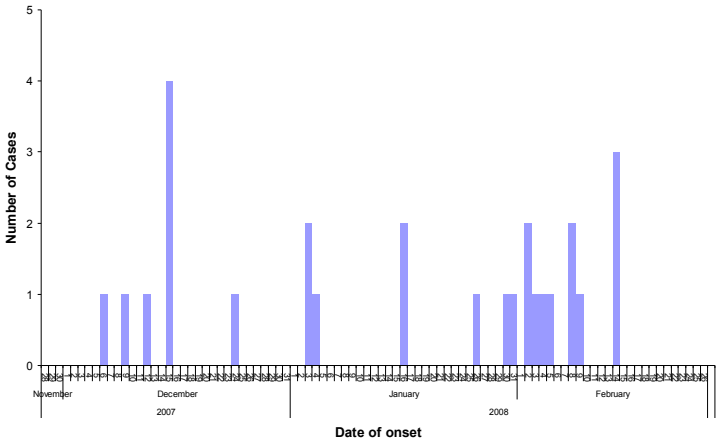

Figure 1: Epidemic curve of conjunctivitis cases according to date of onset of symptoms, Niekerkshoop, 28 November 2007 to 26 February 2008

Eighteen cases (18/29, 62%) were female, and the age group 5 to 15 years was the most affected (12/28 cases with available age data (43%)) (Table 1). All the cases in this age group attended a single school. The most common clinical symptoms in order of frequency were red eye(s), painful eye(s), and eye discharge (Table 2).

Table 1: Conjunctivitis cases according to age group, Niekerkshoop, 5 December 2007 to 14 February 2008.

Age Group	Case	%	Control	%
<5	2	7	4	0
5-15	12	43	17	38
16-25	2	7	6	13
26-35	3	11	6	13
36-45	1	4	3	7
46-55	4	14	5	11
55+	4	14	4	9
Total	28	100	45	100

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Table 2: Frequency of reported symptoms amongst conjunctivitis cases, Niekerkshoop, 5 December 2007 to 14 February 2008.

Symptom	Frequency (n/N)	Percentage (%)
Red eye/s	23/29	79
Painful eye/s	23/29	79
Eye discharge	21/27	77
Itching of eye/s	18/29	62
Swelling of eye/s	17/29	58
Fever	6/29	20
Sensitivity to light	6/29	20

Conjunctival specimens were collected from 15 cases, all were collected in February after the outbreak peak and some were collected from cases several days after the onset of symptoms following the receipt of antibacterial therapy. Of the 15 conjunctival swabs sent for testing, *Staphylococcus aureus* was isolated from 5 specimens, *Streptococcus pneumoniae* from one and coagulase-negative *Staphylococcus* from 4 specimens, and there was no organism cultured in 5 of the specimens.

PFGE of 4 of the *S. aureus* isolates showed them to be unrelated (Figure 2). All 6 specimens sent for detection of adenovirus and enterovirus (by PCR) were negative. There was no statistically significant association between risk factors evaluated and developing disease (Table 3).

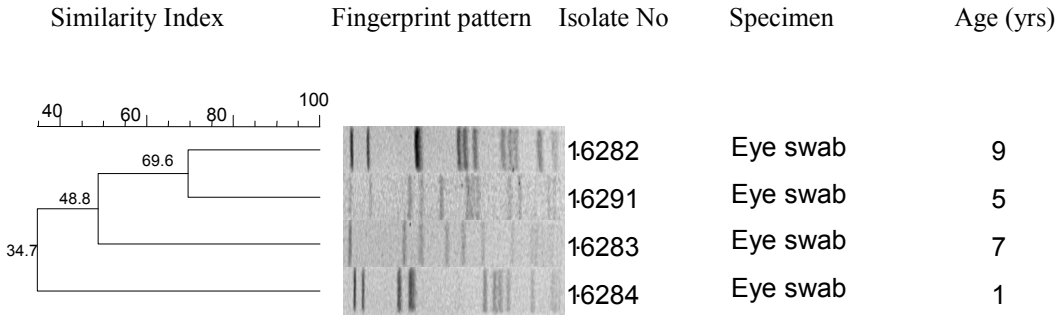

Figure 2: Pulsed-field gel electrophoresis dendrogram of four *S. aureus* isolates recovered from specimens of patients who presented with conjunctivitis at Niekerkshoop, 5 December 2007 to 14 February 2008.

Table 3: Risk factors for conjunctivitis amongst cases and control in Niekerkshoop, 5 December 2007 to 14 February 2008.

Variable	Case n/N (%)	Control n/N (%)	Odds Ratio (95% Confidence Interval)	P-value
Access to clean water	25/29 (86)	35/47 (75)	2.14 (0.62-7.42)	0.12
Recent attendance at large gatherings	2/29 (7)	0/43 (0)	-	0.15
Share facecloth/ handkerchiefs/ towels with household member	10/26 (39)	9/45 (20)	2.50 (0.75-8.42)	0.16
Swim in local dam last three months	4/24 (17)	6/45) (13)	1.3 (0.27-6.10)	0.48
Share eye ointment with household member	4/29 (14)	2/47 (4)	3.6 (0.51-30.87)	0.15

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Information regarding the cause and prevention (personal hygiene) of conjunctivitis was distributed to the community in pamphlets. The community was also informed to report any signs and symptoms of conjunctivitis to the clinic immediately. Existing cases were also given chloramphenicol eye ointment for treatment.

Discussion

The outbreak of conjunctivitis in Niekerkshoop, involved mostly the younger age group of 5 to 15 years, and more than half of the cases were female. All twelve cases in this age group attended the same school. Since this is a small community, and there is frequent interaction among children at school and at home, it is likely that this could have resulted in the spread of infection among this age group.[15] According to the literature, the incidence rate of infectious conjunctivitis is generally high in young children and patients aged over 65 years, compared to other age groups. More episodes are recorded in women than in men.[16] None of the evaluated risk factors were found to be associated with developing conjunctivitis. PFGE confirmed that *S. aureus* isolates were unrelated and thus were unlikely to be the cause of the outbreak. Isolates may have represented sporadic cases of disease or bacterial super-infection of viral conjunctivitis. It is difficult to interpret the significance of coagulase-negative staphylococci as these may represent normal flora of the eye. The source of the outbreak and factors that influenced its spread remains unknown.

Limitations of this study were that the number of controls was limited because selection depended on availability of participants. Also no matching was done on cases and controls, and controls were selected by convenience and thus may not have been representative of the population from which the cases were drawn. Since conjunctivitis is not a notifiable condition there were no baseline data available. This made it difficult to evaluate whether the observed number of cases was truly greater than expected and to exclude the contribution of seasonal factors. The fact that the clinic was closed over the Christmas period means that cases may have been missed. Only a limited number of cases had conjunctival swabs and all of these were collected late during the outbreak from patients who were symptomatic, but were already on chloramphenicol ointment treatment. As a result causative viral and bacterial organisms may have been missed. Throat swabs for viral testing were not taken and this could have revealed more about possible viral causes of infection.

Although the source of the outbreak was not identified, the outbreak resolved following implementation of control measures. The importance of early collection of appropriate laboratory specimens in the outbreak setting should be emphasized.

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THE LABORATORY PROGRAMME FOR THE COMPREHENSIVE CARE, MANAGEMENT AND TREATMENT PROGRAMME (CCMT) FOR HIV AND AIDS

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In 2003, the National Department of Health called together a team to develop a plan for the comprehensive management of HIV infection in South Africa, including the use of antiretroviral medication. Part of the programme involved the use of the National Health Laboratory Services (NHLS) for monitoring patients using CD4 and HIV viral load assays, monitoring toxicity effects of the medication with full blood count, urea and electrolytes, liver enzymes, lipid assays, lactate and arterial blood gas when required, and HIV polymerase chain reaction (PCR) testing for infant diagnosis of HIV infection.

One of the great strengths of the NHLS is its national data base containing a record of every test performed in the country's public health sector together with patient demographic details. The only province not yet on the laboratory information system is KwaZulu-Natal (KZN) as it has only recently been incorporated into the NHLS. Data reported on in this bulletin from KZN is therefore collected on a different system and collated manually with the overall NHLS data.

Data collected over the past three years for the Laboratory Programme for the Comprehensive Care, Management and Treatment Programme (CCMT) for Quarter 1 (Q1) (January to March) of each year since 2006 is illustrated in Table 1 (overleaf). The percentage increases in test volumes from Q1 2006 until Q1 2008 is as follows: 102% for CD4 testing (from 279 901 to 565 012 tests done), 220% for viral load testing (from 65 617 to 210 090), and 219% for PCR testing (from 13 079 to 41 773) (Figure 1). In order to accommodate this volume of testing, NHLS currently has 56 laboratories performing CD4 testing, 16 for viral load testing, and 8 for HIV PCR testing. Prior to the start of this programme, there were very few laboratories (mainly laboratories associated with universities) performing these tests, and then predominantly for research based purposes. It is clear therefore that there have been tremendous advances in laboratory capacity since the programme started in April 2004.

To some extent, the laboratory data also indirectly measures the impact of the programme on our HIV infected population accessing health care services. For example, the percentage of CD4 specimens tested that measure fewer than 200 cells/mm^3 has decreased from 42% (117306/279901) in 2006 to 36% (204639/565012) in 2008 (Figure 2). This reflects the percentage of patients responding to treatment as well as the fact that increasing numbers of healthy HIV infected people with higher initial CD4 counts are accessing the health services and being monitored using CD4 testing. The number of viral load tests recording undetectable viral load levels have increased from 43% (27996/65617) in 2006 to 58% (121506/210090) in 2008 (Figure 3). This is a measure of good response to antiretroviral treatment, as viral load testing is performed for people with CD4 counts <200 cells/mm^3. Even more gratifying is the decrease in percentage positive PCR tests from 28% (3670/13079) in 2006 to 18% (7417/41773) in 2008 (Figure 4). This is a crude measure of the success of the Prevention of Mother to Child Transmission of HIV (PMTCT) programme as diagnostic HIV PCR tests are generally performed in babies born to HIV infected mothers to determine whether the infants have been perinatally infected with HIV. If we review the percentage positive PCR tests by province, the Western Cape Province (WCP) has the lowest positivity rate of 10.5% (467/4442) in the first quarter of 2008. This is likely in part because this province uses dual drug management in pregnant HIV infected mothers as part of its PMTCT programme, whereas the rest of South Africa is still using single dose Nevirapine. The latest guidelines released from the National Department of Health have now incorporated a recommendation for a dual therapy regimen for all provinces.

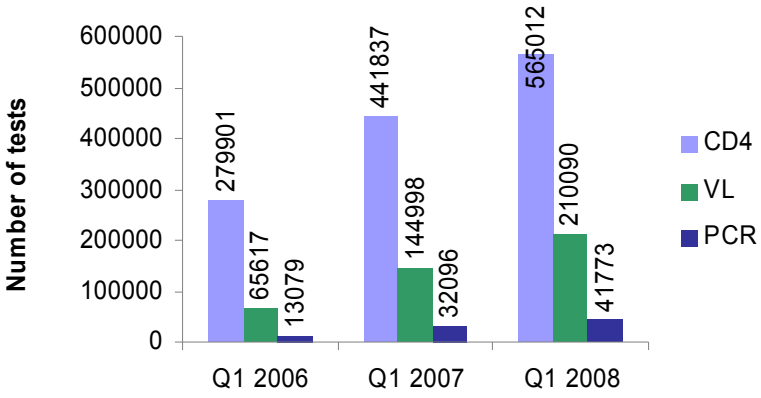

Figure 1: CD4, viral load (VL) and HIV polymerase chain reaction (PCR) test volumes for Quarter 1 (Q1) of 2006, 2007, 2008, South Africa.

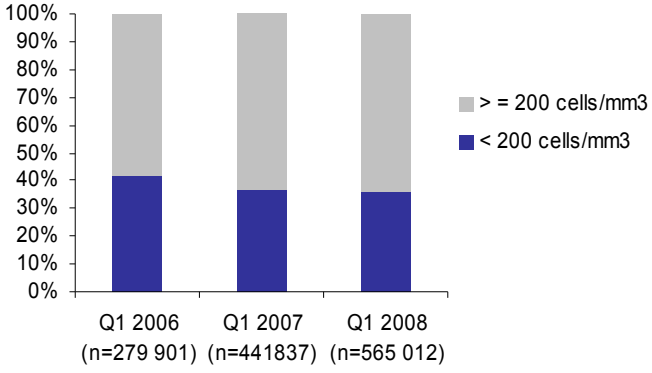

Figure 2: Percentage of CD4 tests performed in NHLS with CD4 count <200 cells/mm^3 for Quarter 1 (Q1) of 2006, 2007, 2008, South Africa.

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Table 1: Indicators for the first quarter (1 January to 31 March) for the Laboratory Programme for the Comprehensive Care, Management and Treatment Programme for HIV and AIDS, 2006-2008.

Programme	Indicator	Subgroup	Year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Laboratory Programme for Comprehensive Care, Treatment and Management Programme for HIV and AIDS	CD4 count tests performed	Total tests done	2006	36123	10023	72917	65625	14028	20946	6557	25752	27930	279901
			2007	57901	21594	105053	109845	28142	33904	9998	38847	36553	441837
			2008	69391	28071	122582	160861	41679	41209	10831	45482	44906	565012
		Tests with CD4 count < 200/µl	2006	15430	4582	33014	26677	6155	10098	2275	10890	8185	117306
			2007	22117	8768	41253	38009	13007	12381	3049	14144	9193	161921
			2008	28713	9318	47605	56743	15322	15530	3303	15024	13081	204639
	Viral load tests performed	Total tests done	2006	10150	3744	20270	16265	1739	1548	1477	7642	2782	65617
			2007	21904	7567	39139	31062	8808	9167	3182	13296	10873	144998
			2008	28337	11043	53470	48820	18245	13815	4037	17790	14533	210090
		Viral load undetectable	2006	3145	1775	9795	6591	574	567	573	3397	1549	27966
			2007	8457	3806	20367	16547	3818	4244	1460	7118	8595	74412
			2008	13383	6331	31514	27827	10273	7427	2123	11006	11622	121506
	Diagnostic PCR tests performed	Total tests done	2006	666	606	4405	4611	132	443	346	906	964	13079
			2007	4187	936	9494	8244	1511	1647	633	2349	3095	32096
			2008	5492	2208	11024	9150	3576	2128	716	3037	4442	41773
		Number positive	2006	195	228	1284	1005	66	209	82	402	199	3670
			2007	832	320	1977	1896	417	464	118	614	404	7042
			2008	712	413	1815	2131	682	466	125	606	467	7417

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

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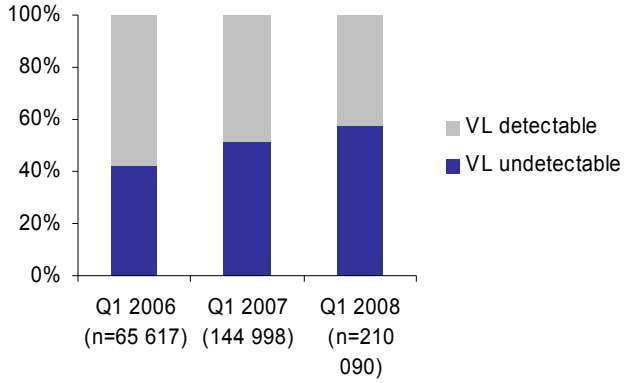

Figure 3: Percentage of viral load tests performed in NHLS that are below the limit of detection for Quarter 1 (Q1) of 2006, 2007, 2008, South Africa.

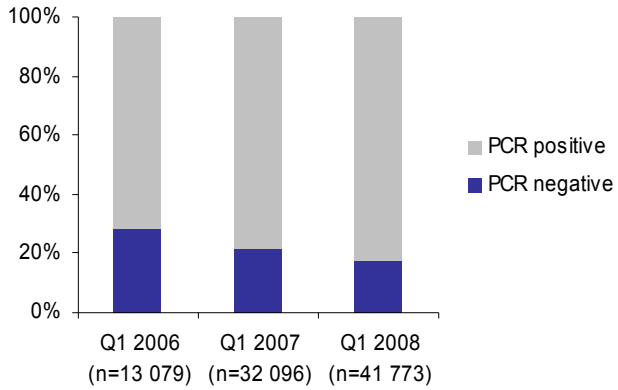

Figure 4: Percentage of HIV PCR tests performed in NHLS that show positive results (indicating a transmission of HIV to infants) for Quarter 1 (Q1) of 2006, 2007, 2008, South Africa.

Data presented in this report are obviously a very crude and indirect measure of the CCMT as it includes repeat testing performed on the same patients, and reflects only laboratory data without clinical input. These limitations however apply to all indicators and remain constant with time. Thus data will still provide some idea of the trends. It is important not to take this as an actual measure of the percentage of people accessing the services and getting treatment as it is only a very crude measure of trends, and should not be used as a critique (either positive or negative) of the programme until more accurate and sensitive data is available. However, data showing improvements of all indicators over the 3 years are encouraging.

The current trends give an indication regarding the numbers of tests for which the NHLS will need to prepare over the remainder of 2008. We can expect approximately 2.3 million CD4 tests, 840 000 viral load tests, and 200 000 HIV PCR tests in the course if 2008. Current annual capacity is sufficient for these needs. To ensure a reliable laboratory service, we aim to perform diagnostic testing for this programme at least 30% below maximum capacity to allow a buffer for instrument down time, power failures, and surge capacity should demand suddenly increase. To cope with the anticipated increase in volumes, laboratory capacity is constantly being increased and the concept of creating super labs to deal with larger volumes of testing more cost-efficiently and with improved staff utilization ratios is being investigated. In addition, increasing automation of laboratory equipment also improves daily throughput and decreases staff time spent on laboratory assays. This factor is critical in this era of global skills shortages.

In conclusion, the laboratory data presented in this bulletin offers some indicators of the success of the CCMT for HIV and AIDS. The large increase in the volume of tests processed by the NHLS indicates the improvement in access to care for HIV infected people across South Africa. The changes noted in certain parameters such as the number of CD4 tests below 200 cells/mm^3, undetectable viral load results obtained, and PCR positivity (or more important, negativity) rates all show encouraging trends. New data tools are being developed to monitor more sensitively the trends in individual patient results in order to detect virological failure enabling NHLS to more sensitively survey for drug resistance patterns in our population. As additional results become available, they will be reported in this forum.

Provisional listing: number of laboratory-confirmed cases in South Africa of diseases under surveillance reported to the NICD, corresponding periods 1 January - 31 March 2007/2008

	Disease/ Organism	Case Definition	Subgroup	01 January to 31 March year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
VIRAL DISEASES	Acute Flaccid Paralysis	Cases < 15 years of age from whom specimens have been received as part of the Polio Eradication Pro-		2007	7	7	22	17	8	10	5	9	7	92
				2008	13	4	24	9	14	6	1	2	8	81
	Measles	Measles IgM positive cases from suspected measles cases, all ages		2007	2	0	1	0	0	1	0	1	1	6
				2008	1	0	3	0	0	1	0	1	2	8
	Rubella	Rubella IgM positive cases from suspected measles cases , all ages		2007	31	0	12	8	14	7	4	10	16	102
				2008	29	1	9	9	6	4	0	11	3	72
	VHF	Laboratory-confirmed cases of CCHF (unless otherwise stated), all ages		2007	0	0	0	0	0	0	0	0	0	0
				2008	0	1	0	0	0	0	2	0	0	3
	Rabies	Laboratory-confirmed human cases, all ages		2007	2	0	0	3	0	0	0	0	0	5
				2008	2	0	0	3	2	0	0	0	0	7
BACTERIAL AND FUNGAL DISEASES	*Haemophilus influenzae*	Invasive disease, all ages	All serotypes	2007	10	5	41	10	1	3	0	2	14	86
				2008	7	5	47	7	2	2	0	0	15	85
		Invasive disease, < 5 years	Serotype b	2007	0	0	6	1	0	0	0	1	9	17
				2008	2	3	3	0	0	0	0	0	2	10
			Serotypes a,c,d,e,f	2007	0	0	2	1	0	0	0	0	0	3
				2008	0	1	5	0	0	0	0	0	0	6
			Non-typeable (unencapsulated)	2007	0	0	9	3	0	0	0	0	0	12
				2008	0	0	4	0	0	0	0	0	1	5
			No isolate available for serotyping	2007	7	0	10	2	1	1	0	0	3	24
				2008	4	0	18	2	1	2	0	0	9	36
	Neisseria meningitidis	Invasive disease, all ages		2007	1	2	21	4	0	4	0	5	12	49
				2008	2	4	43	2	0	7	2	0	9	69
	Streptococcus pneumoniae	Invasive disease, all ages	Total cases	2007	65	76	407	73	23	55	11	39	127	876
				2008	56	41	377	93	14	50	12	24	114	781
			Penicillin non-susceptible isolates	2007	9	23	138	32	6	14	1	10	39	272
				2008	21	14	97	35	1	15	6	6	32	227
			No isolate available for susceptibility testing	2007	46	17	114	15	10	21	6	17	25	271
				2008	16	12	126	12	11	22	3	12	24	238
		Invasive disease, < 5 years		2007	26	24	138	34	11	12	5	12	49	311
				2008	10	21	127	36	6	23	4	5	45	277
	Salmonella spp. (not typhi)	Invasive disease, all ages		2007	5	18	104	26	1	6	1	7	18	186
				2008	17	15	159	24	3	8	4	6	17	253
		Confirmed cases, isolate from a non-sterile site, all ages		2007	45	7	81	34	12	36	5	4	24	248
				2008	59	10	105	41	2	27	4	3	39	290
	Salmonella typhi	Confirmed cases, isolate from any specimen, all ages		2007	4	0	6	1	1	4	0	1	5	22
				2008	1	1	10	0	1	7	0	0	2	22
	Shigella dysenteriae 1	Confirmed cases, isolate from any specimen		2007	0	0	0	0	0	0	0	0	0	0
				2008	0	0	0	0	0	0	0	0	0	0
	Shigella spp. (Non Sd1)	Confirmed cases, isolate from any specimen, all ages	All serotypes	2007	39	24	123	43	7	12	8	8	86	350
				2008	53	19	148	35	1	21	6	3	131	417
	Vibrio cholerae O1	Confirmed cases, isolate from any specimen, all ages	All serotypes	2007	0	0	0	0	0	0	0	0	0	0
				2008	0	0	0	0	0	0	0	0	0	0
	Cryptococcus (*Cryptococcus spp.*)	Invasive disease, all ages	Total cases (incl. *C neoformans*)	2007	310	143	550	350	86	169	19	146	114	1887
				2008	158	122	442	361	55	104	9	116	120	1487
			C. gattii	2007	0	0	9	3	7	6	0	5	1	31
				2008	2	0	2	3	2	3	0	1	1	14

Abbreviations: VHF - Viral Haemorrhagic Fever; CCHF - Crimean-Congo Haemorrhagic Fever

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

0 = no cases reported