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NICD NHLS

FOREWORD

South Africa has the world's largest antiretroviral therapy programme. With widespread use of antiretroviral agents there is concern about the development of resistance to first line agents. Surveillance for the detection of resistance is thus essential. The current bulletin reports on findings from surveillance for transmitted resistance from 2002 through 2007 and reassuringly finds extremely low levels of resistance mutations. It is however critical that we have ongoing surveillance in order to track trends over time.

This bulletin includes two articles describing the recent cholera outbreak in South Africa. This outbreak highlights several issues related to outbreaks of communicable diseases including the role of transborder spread of infectious diseases and the fact that when introduced, infectious diseases spread rapidly if conditions are suitable for transmission.

Our last article describes an outbreak of gastrointestinal illness and highlights travel-related food and waterborne infections. Such outbreaks are under-reported and often not fully investigated. We encourage submission of articles on this theme in future.

Cheryl Cohen, Editor

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SURVEILLANCE FOR TRANSMITTED HIV-1 DRUG RESISTANCE IN SOUTH AFRICA

Lynn Morris[1], Johanna Ledwaba[1], Gillian Hunt[1], Mpho Rakgotho[1], Visva Pillay[1], Beverley Singh[3], Adrian Puren[4] and Lindiwe Makubalo[5]
[1]HIV/AIDS Virus Research Unit, [2]Serology Laboratory, [4]Specialized Molecular Diagnostics Unit, National Institute for Communicable Diseases
[5]National Department of Health

HIV-1 drug resistance is an inevitable consequence of the use of anti-retroviral drugs (ARV) with the majority of patients who fail ARV developing resistant strains. In South Africa there has been a rapid scale-up of ARV use with over 700,000 patients initiated on treatment since the start of the Comprehensive HIV and AIDS Care, Management and Treatment (CCMT) Plan in April 2004. The transmission of resistant viruses to newly infected persons is a public health issue and has been reported in the USA and Europe where treatment has been available for a longer time. In countries where treatment options are more limited, the transmission of resistant viruses could have serious consequences for ARV treatment programs. Since it is not possible in South Africa to perform HIV resistance testing on an individual patient basis, it is imperative that we implement surveillance strategies to monitor for transmitted resistance.

The World Health Organization (WHO) has developed a minimum resource method termed the HIV drug resistance threshold survey (HIVDR-TS) to monitor transmitted

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resistance among drug-naive persons (1). This survey takes advantage of specimens already collected from sites conducting HIV sero-prevalence studies such as in antenatal clinics, sexually transmitted-infection clinics, and other HIV diagnostic sites, although these should be in areas where ARV have been available for at least 3 years. It uses sampling among individuals under 25 years of age and, in the case of women, those with no previous pregnancies as a surrogate for recent infection. The survey requires between 34 and 47 specimens and categorizes the prevalence of transmitted resistance overall and to specific drug classes as <5% (low prevalence), 5–15% (moderate prevalence), or >15% (high prevalence) Recommendations for public health action is provided for each level and only when >15% is confirmed in a follow-up survey or in another area should a change of the first-line regimen be considered.

The annual antenatal survey (ANSUR) conducted by the National Department of Health is an anonymous, unlinked cross-sectional survey which estimates HIV prevalence using blood samples taken from pregnant women attending public health sector antenatal clinics across all 9 provinces in South Africa. This survey provides the best available estimates of HIV infection in the country. Data show that HIV-1 prevalence has increased dramatically since 1991 from <1% to 28% in 2007 and differs by province (2). To date the NICD has conducted 10 HIVDR-TS using ANSUR samples from 3 provinces in South Africa (Table 1). This survey was first conducted in Gauteng in 2002 prior to the start of the CCMT program and annually from 2004. It was expanded to KwaZulu Natal in 2005 since this province has the highest HIV prevalence and to the Western Cape in 2006, which has the longest running treatment program. Consecutive samples were selected based on the inclusion criteria as set out by the WHO guidelines i.e. women <25 years of age and in first pregnancy. Genotyping was performed from plasma viral RNA by sequencing the *pol* (polymerase) gene (both protease and reverse transcriptase) using an in-house assay that has been extensively internally and externally validated.

Using the WHO threshold survey the classification of resistance prevalence overall and for each drug class was <5% for all 3 provinces at all time points (Table 1). A total of 439 sequences have been analyzed and all were HIV subtype C, the predominant subtype in South Africa. A total of 5 samples with 6 mutations were identified, all of which are included in the consensus genotypic definition for transmitted HIV-1 drug resistance (3, 4). The mutation T69D causes low level resistance to all nucleoside reverse transcriptase inhibitors and K70R causes low-level resistance to d4T and AZT. M184I is selected by 3TC and confers low-level resistance to this drug while Y181C is selected by NVP and EFV and causes high-level resistance to both drugs. These mutations do not occur among untreated persons and thus these individuals were either exposed to ARV drugs or were infected with a resistant strain. The M46I mutation found in one individual is a protease inhibitor resistance associated mutation, but is likely to be a polymorphism in this case as this mutation occurs at low frequency among drug-naive persons (4). While the use of these ANSUR serum samples for resistance surveillance has proven useful, the amplification rates varied considerably with unacceptably low rates in two surveys (KZN 2005 and WC 2007). However, since the HIVDR-TS is based on the number of genotypes obtained and does not take into account amplification failures, these low rates are unlikely to have caused any significant bias. Nevertheless, we are currently exploring ways to improve sample collection from these sites, including the use of dried blood spots for HIVDR testing, to minimize this problem.

In addition to the threshold survey the WHO recommends the use of information collected routinely in medical and pharmacy records to monitor the functioning of ART sites for factors potentially associated with HIVDR referred to as HIVDR-early warning indicators (EWI) (5). These EWI are not routinely analysed in South Africa but such information would be useful to guide future threshold surveys. While our data suggest that there is currently a low prevalence of transmitted HIV-1 drug resistance and thus no cause for alarm, the scale of the ARV program in South Africa and the numbers of patients who will fail therapy and develop resistant strains will expand. This combined with the high incidence rates in this country will increase the risks of transmission of resistant strains. It is therefore important to continue population-based drug resistance surveillance to guide strategies aimed at limiting the emergence of transmitted resistance.

References

1. Bennett D, Myatt M, Bertagnolio S, Sutherland D, and Gilks CF. Recommendations for surveillance of transmitted HIV drug resistance in countries scaling up antiretroviral treatment. *Antiviral Therapy* 2008, 13 Suppl 2: 25-36.
2. The National HIV and Syphilis Prevalence Survey South Africa 2007. National Department of Health South Africa 2008. http://www.doh.gov.za/docs/index.html
3. Shafer RW, Rhee SY, Pillay D, Miller V, Sandstrom P, Schapiro JM, Kuritzkes DR and Bennett D. HIV-1 protease and reverse transcriptase mutations for drug resistance surveillance. *AIDS* 2007, 21: 215-223.
4. Bennett DE, Camacho RJ, Otelea D, Kuritzkes DR, Fleury H, et al. Drug Resistance Mutations for Surveillance of Transmitted HIV-1 Drug-Resistance: 2009 Update. PLoS ONE 2009, 4(3): e4724.
5. Bennett D, Bertagnolio S, Sutherland D and Gilks CF. The World Health Organization's global strategy for prevention and assessment of HIV drug resistance. *Antiviral Therapy* 2008, 13 Suppl 2: 1-13.

Table 1: Threshold Surveys for transmitted HIV drug resistance conducted among antenatal clinic attendees in Gauteng, KwaZulu-Natal and the Western Cape

Province	Year	Number specimens	Mean Age	Parity	Mean Age Partner	Amplification Rate	Number of sequences analyzed	HIV-1 Subtype	Number with mutations	Mutational patterns	Threshold Level
Gauteng											
	2002	115	19	0	26	52%	52	C	0		<5%
	2004	113	20	0	26	39%	44	C	2	T69D, K70R	<5%
	2005	86	20	0	26	81%	63	C	0		<5%
	2006	64	20	0	25	94%	55	C	0		<5%
	2007	133	20	0	Unknown	44%	57	C	1	M46I, M184I	<5%
KwaZulu-Natal											
	2005	287	21	0	26	14%	40	C	1	Y181C	<5%
	2006	78	20	0	24	89%	41	C	1	Y181C	<5%
	2007	61	20	0	Unknown	67%	34	C	0		<5%
Western Cape											
	2006	107	22	0	Unknown	42%	45	C	0		<5%
	2007	118	Unknown	0	Unknown	15%	8	C	0		ND*

* Not done due to insufficient sequences available

CHOLERA OUTBREAK IN SOUTH AFRICA: PRELIMINARY DESCRIPTIVE EPIDEMIOLOGY ON LABORATORY-CONFIRMED CASES, 15 NOVEMBER 2008 TO 30 APRIL 2009

Brett N. Archer[1], Ayanda Cengimbo[1], Gillian M. De Jong[1], Karen H. Keddy[2], Anthony M. Smith[2], Arvinda Sooka[2], Genevie Ntshoe[3], Lucille Blumberg[1]
[1]Outbreak Response Unit, Epidemiology Division, [2]Enteric Diseases Reference Unit, [3]South African Field Epidemiology and Laboratory Training Programme, National Institute for Communicable Diseases (NICD)

1. Introduction

Cholera is an acute intestinal infection caused by the ingestion of the bacterium *Vibrio cholerae*. Following a short incubation period (hours to five days), disease typically presents as profuse watery diarrhoea that can rapidly lead to severe dehydration and death if not promptly treated.[1] Two serogroups of *V. cholerae* can cause outbreaks – *V. cholerae* O1 and O139. To date, *V. cholerae* O1 has been responsible for all cholera outbreaks in South Africa (SA) and the majority of cholera outbreaks worldwide.

The last major cholera outbreak in South Africa occurred in 2001 causing approximately 100,000 cases.[2] Although sporadic, localised, outbreaks of cholera continue to occur within the country[3], during mid-November 2008 we witnessed the importation of cases following a significant epidemic in neighbouring Zimbabwe. Within a short period of time local transmission was documented in South Africa, which has been linked to contaminated water supplies, poor sanitation infrastructure and poor access to potable water sources; resulting in explosive outbreaks within two provinces. The following report documents the preliminary findings of the surveillance systems set up to capture laboratory-confirmed cases of cholera within South Africa. At the time of this publication, the current outbreak is ongoing and this report will focus on preliminary data for laboratory-confirmed cases received during the period of 15 November 2008 to 30 April 2009.

2. Methodology

2.1 Case definitions

Clinical (suspect) cholera case definition: Any individual with acute onset of watery diarrhoea in South Africa from 1 November 2008 to date.

Confirmed cholera case definition: Any individual who meets the clinical case definition on whom *Vibrio cholerae* O1 was isolated from stool or rectal swab in South Africa, from 1 November 2008 to date.

2.2 The role of NICD/NHLS and surveillance systems descriptions

Both the NICD and the NHLS have been central to the public health management of the cholera outbreak. The primary role of NHLS/NICD has been the laboratory confirmation of cholera cases (and hence the outbreak), monitoring the spread of the outbreak within South Africa, and monitoring pathogen specific trends (such as antimicrobial susceptibility, serotypes and molecular characterisations). In addition to providing the necessary intelligence for the management of the outbreak, the NICD has supported the establishment of surveillance systems for clinical cholera cases and has advised on case management and the implementation of public health interventions to control the outbreak.

Laboratory-based surveillance for cholera has not followed a constant strategy, but rather the role of the laboratory

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and its testing strategies have changed with the ongoing evolution of the outbreak. During the beginning of the outbreak, laboratory testing focused on confirming the importation of cholera cases into the country from Zimbabwe, and identifying the occurrences of local transmission (i.e. confirming the existence sustained local outbreaks). Initially specimens were collected from all cases meeting the clinical case definition. Thereafter, in provinces with established community-wide outbreaks (Limpopo and Mpumalanga), clinical case definitions alone were used for diagnosis, thus specimen collection and laboratory confirmation was limited to sporadic testing to monitor the situation. However, in areas without such outbreaks (all provinces excluding Limpopo and Mpumalanga), all clinical cases continued to be tested. Finally, with the reduction in the frequency of new infections in recent weeks, a strategy of testing all cases meeting the clinical case definition for cholera has been gradually reinstituted in Limpopo and Mpumalanga provinces.

Processing of stool samples and rectal swabs for bacterial isolation and initial identification to confirm *V. cholerae* O1 is conducted by the many NHLS diagnostic laboratories throughout South Africa. The testing laboratory captures patient identification information, basic demographics, facility details, and laboratory results on a networked laboratory information system (Disa*Lab®). Data is stored and communicated centrally to a central repository within the NHLS. This data is then transferred to the Corporate Data Warehouse (CDW). The CDW provides the first level of laboratory-based surveillance for cholera cases through the regular extraction of detailed line-lists and the automated email notifications to the Outbreak Response Unit (ORU) of the NICD. The ORU has maintained a database of all laboratory-confirmed cholera cases, based upon data received from the CDW, private laboratories, the Department of Health (DoH) and other relevant health authorities. The following preliminary report provides an analysis of this database of laboratory-confirmed cases received during the period of 15 November 2008 to 30 April 2009.

In addition, the Enteric Diseases Reference Unit (EDRU), as part of the Group for Enteric, Respiratory and Meningeal Disease Surveillance in South Africa (GERMS-SA), NICD receives a proportion of *V. cholerae* O1 isolates for further characterisation. Isolates undergo biochemical confirmatory testing and serotyping. Minimum inhibitory concentrations (MICs) were obtained using E-test strips, according to the manufacturer's instructions. PCR was used to determine the biotype of *V. cholerae* O1 and to determine the presence of cholera enterotoxin. The molecular epidemiology of isolates was investigated using pulsed-field gel electrophoresis (PFGE) analysis incorporating *Not*I digestion of genomic DNA. The results of the analysis of isolates received by the EDRU between 15 November 2008 to 30 April 2009 are also presented.

3. Results

3.1 Descriptive epidemiology

A total of 1,144 laboratory-confirmed cholera cases were reported in South Africa for the period of 15 November 2008 to 30 April 2009. For the same period, DoH reported a total of 12,706 cases meeting the clinical (suspect) case definition for cholera. Of the total clinical cases 64 deaths were recorded, resulting in a case fatality rate of 0.5% for South Africa. Mpumalanga (n=387, 33.8%) and Limpopo (n=612, 53.5%) provinces account for the majority of laboratory-confirmed cholera cases, with 5.6% (387/6,855) and 11.2% (610/5,460) of clinical cases confirmed for the two provinces respectively (Table 1).

Table 1: Frequency distribution of clinical and laboratory-confirmed cholera cases by province, South Africa, 15 November 2008 to 30 April 2009, n=1,144

Province	Total cases No. (%total)*	Laboratory-confirmed cases No. (% clinical cases confirmed)†	Deaths No. (CFR%)
Mpumalanga	6 855 (54.0)	387 (5.6)	30 (0.4)
Limpopo	5 460 (43.0)	612 (11.2)	26 (0.5)
Gauteng	286 (2.3)	71 (24.8)	4 (1.4)
North West	91 (0.7)	60 (65.9)	4 (4.4)
Western Cape	8 (0.1)	8 (100.0)	0 (0.0)
Eastern Cape	2 (<0.1)	2 (100.0)	0 (0.0)
KwaZulu Natal	2 (<0.1)	2 (100.0)	1 (50.0)
Northern Cape	1 (<0.1)	1 (100.0)	0 (0.0)
Free State	1 (<0.1)	1 (100.0)	0 (0.0)
Cumulative total	**12 706 (100.0)**	**1 114 (9.0)**	**65 (0.50)**

*Laboratory-confirmed cases and cases meeting the current clinical case definition for cholera (all individuals with acute onset of watery diarrhoea) as reported by the National Department of Health, Situation Report, 22 April 2009.
†Laboratory-confirmed cholera cases reported to the NICD from NHLS and private laboratories.

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The epidemic curve (Figure 1) illustrates the distribution of cases by epidemiological week of specimen collection as a proxy for date of disease onset. During the initial weeks of the outbreak (epidemiological week 47 of 2008 to week 1 of 2009), relatively low case frequencies were observed, with the majority of these occurring in Limpopo Province and sporadic cases in other provinces.

A significant increase in case frequency was observed during epidemiological week 2 of 2009, with acceleration of infection observed primarily in Mpumalanga Province. This rapid increase in Mpumalanga Province may be attributed to an increase in local transmission and infections resulting from contamination of water supplies utilised by local communities; supported by the isolation of *V. cholerae* from both municipal and untreated surface water sources (e.g. rivers) and the documentation of raw sewage spillage into rivers. Laboratory-confirmed case frequencies peaked during week 3 of 2009, with 299 cases confirmed during the epidemiological week.

Thereafter a rapid decrease in case frequency was noted in Mpumalanga Province, and a gradual decrease recorded in Limpopo Province. A relatively small focal increase was observed in North West province during weeks 10 through 12 of 2009, which was attributed to contamination of borehole water supplies (*V. cholerae* was isolated). A second minor increase was observed in week 14 of 2009 in Limpopo Province, which was reported to be linked primarily to transmission of the pathogen at funerals in the Capricorn District Municipality (Limpopo DoH, personal communication, 2009). From weeks 15 to 17 of 2009, sporadic laboratory-confirmed cases have been confirmed with two to seven cases per week. Although data is not included here, small clusters of laboratory-confirmed cases of cholera have continued to occur in Limpopo Province since week 18 of 2009.

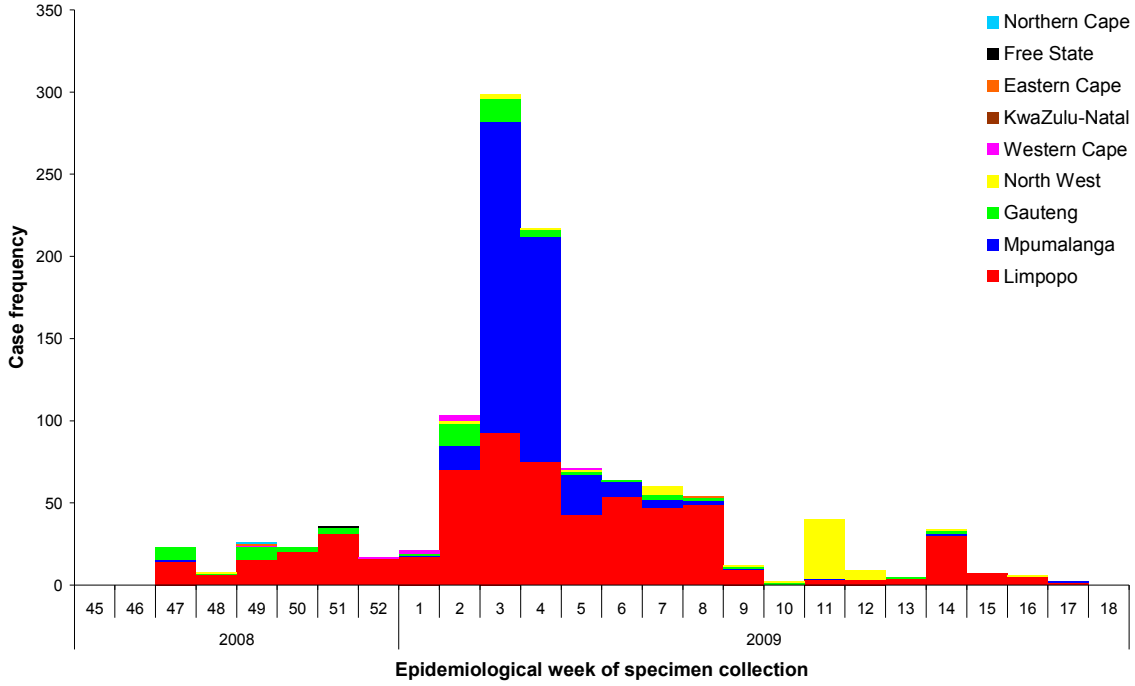

Figure 1: Epidemic curve showing the frequency of laboratory-confirmed cholera cases by epidemiological week of specimen collection and province, South Africa, 2008 week 45 (3 November) to 2009 week 18 (3 May), n=1,144 (5 unknown date of collection)

Description of laboratory-confirmed cases by age and gender (Figure 2) show that young adults, aged 20 to 34 years, are the most affected age groups. Females are more affected than males, accounting for 56% (n=642) and 41% of infections respectively.

Only healthcare facility data was available from the laboratory-based surveillance. Characterisation of cases by sub-district of the health care facility at which a case presented was used as a proxy for describing the spatial distribution of cases by place of residence (Figure 3). Here it may be observed that the majority of cases occurred in the north-eastern areas of South Africa. All areas of Limpopo and Gauteng provinces were affected; however, the outbreak was highly focused within selective sub-districts of Mpumalanga Province. Mbombela Local Municipality (Ehlanzeni District, Mpumalanga Province) accounted for the highest case frequency observed in a single sub-district, with 20% (n=225) of the total laboratory-confirmed cholera cases in South Africa.

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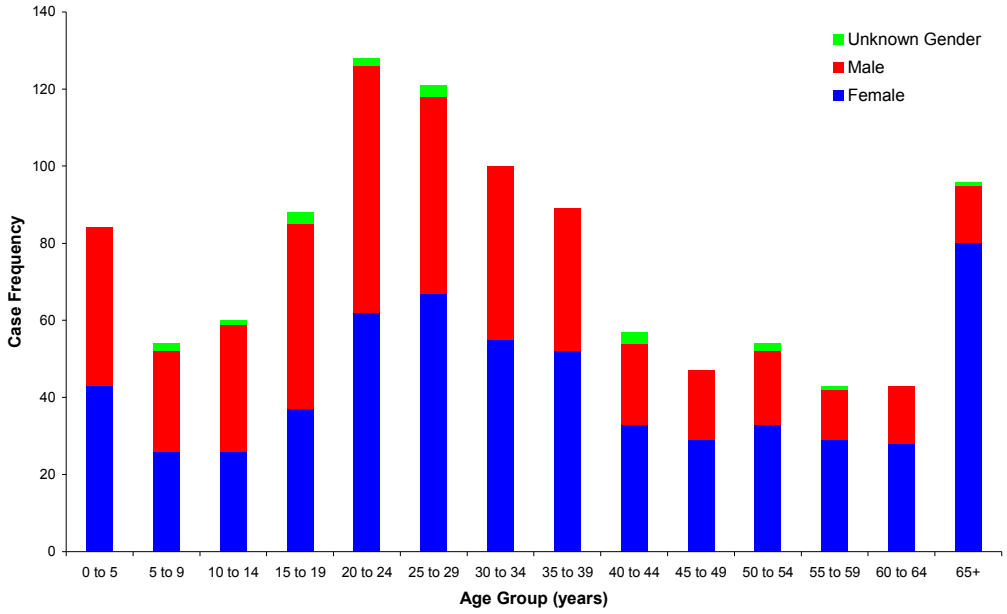

Figure 2: Graph showing the distribution of laboratory-confirmed cholera cases by age and gender, South Africa, 15 November 2008 to 30 April 2009, n=1,144 (70 unknown age)

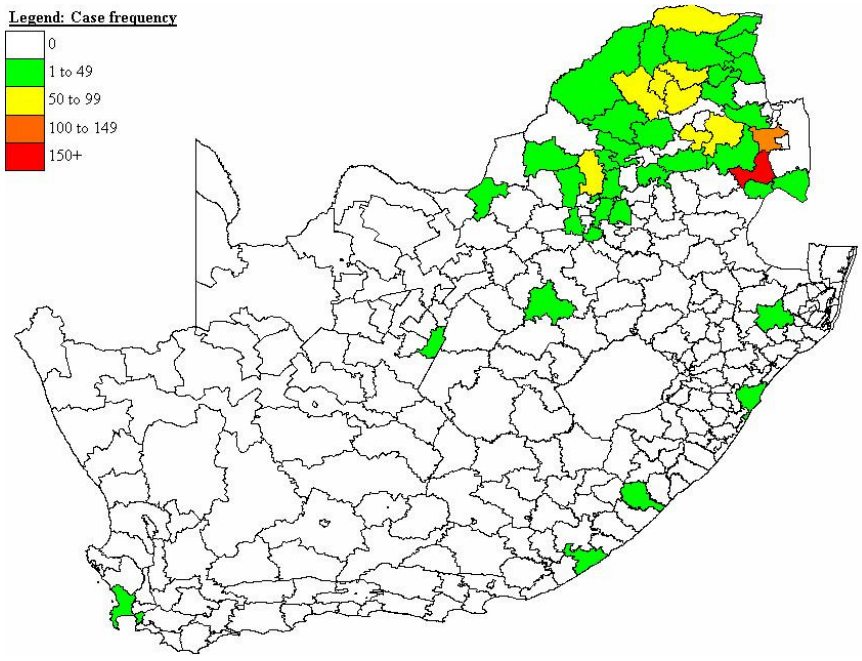

Figure 3: Map showing the spatial distribution of laboratory-confirmed cholera cases by sub-district (estimated by health-care facility), South Africa, 15 November 2008 to 30 April 2009, n=1,144 (4 unknown sub-district)

3.2 Phenotypic and genotypic characterisation and antimicrobial resistance

Six hundred and fifty-four isolates (56.3% of 1,144 laboratory confirmed cases) from the current outbreak have been received and characterised further by EDRU during the period of 15 November 2008 to 29 April 2009. All isolates were confirmed as *V. cholerae* O1. A total of 643/654 (98.3%) were serotyped as *V. cholerae* O1 Ogawa and the remainder (11/654; 1.7%) as *V. cholerae* O1 Inaba. All isolates were confirmed to *V. cholerae* O1 biotype El Tor and contained cholera toxin. Pulsed field gel electrophoresis (PFGE) confirmed that isolates were related at a stringency of >90%.

Antimicrobial susceptibility testing revealed 99.2% (519/523) of isolates to be resistant to cotrimoxazol and produce nalidixic acid (Figure 4). The organism is multi-drug resistant, but remains susceptible to tetracycline (3.1% resistant, 16/523).

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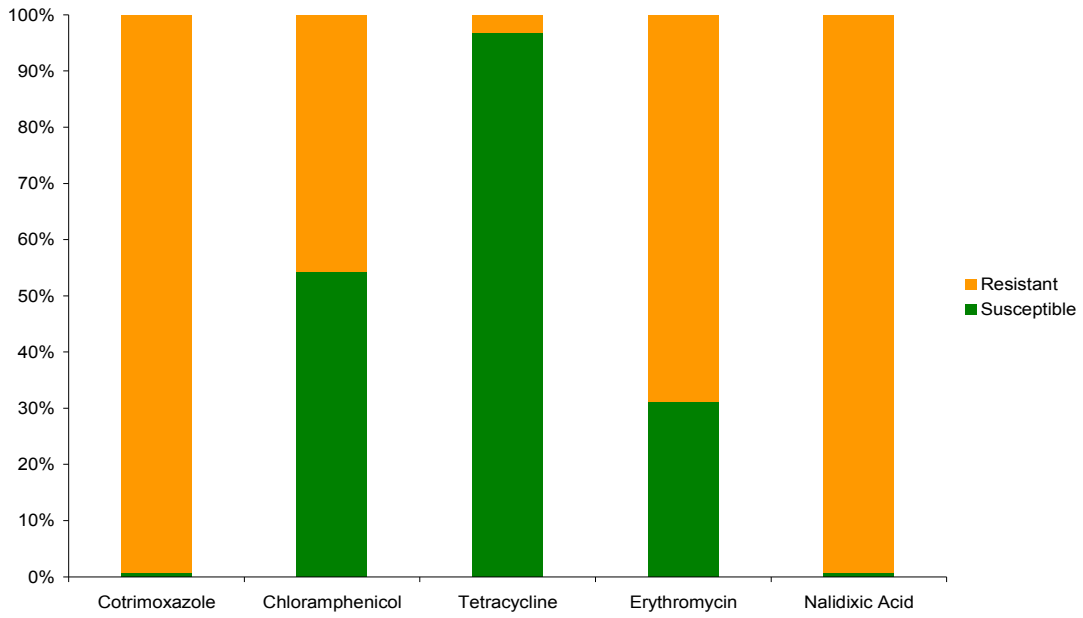

Figure 4: Susceptibility profile of isolates of *Vibrio cholerae* O1 received at the NICD during the current cholera outbreak in South Africa, 15 November 2008 to 30 April 2009, n=523

4. Discussion

The epidemiology of *V. cholerae* O1 during the current outbreak may be characterised as being dynamic, with both differences and changes observed spatially and over time. The initial cases were directly linked to cholera outbreaks in neighbouring Zimbabwe, with cases crossing the border to seek healthcare.[4] After a short period, the pathogen established itself in local Limpopo communities and transmission likely occurred primarily within households and during gatherings through contaminated food or stored water. At this stage, widespread contamination of community water supplies was not documented. The majority of cases in other provinces could be attributed to importation of infection resulting from recent travel from an area with an established outbreak, with few instances of local transmission (limited to small household clusters).[5]

The introduction of *V. cholerae* O1 into rivers and other surface (untreated) water systems in Mpumalanga, within communities without potable treated water supplies, resulted in a rapid spread of disease in these communities. Shortly after these communities were affected, interventions such as the supply of safe water and health promotion were implemented. This may have resulted in a steady decline in the rate of new infections observed; although we do not have evidence to support this. A relatively smaller focal outbreak has been documented in North West and Limpopo provinces, which may be attributed to a breakdown in water infrastructure in vulnerable communities and transmission at mass gatherings such as funerals respectively. Transmission and importation of cases has been ongoing in Limpopo Province throughout the outbreak period. Sporadic cases in recent weeks may be attributed to the continued importation of infection from neighbouring countries.

The additional burden of infection noted among young adults may be partially attributed to the increased mobility of such individuals. However, the documented demographic trends may be significant biased by differences in healthcare seeking behaviour and practices of specimen collection for laboratory analysis between population groups. For example, females are more likely to seek healthcare than males[6], which may account for the differences in gender observed. The spatial distribution of cases revealed that cholera has been documented over a wide area within the South Africa linked to those areas with limited access to safe potable water and sanitation infrastructure. The majority of infections have been focused in specific areas of Limpopo and Mpumalanga provinces, therefore allowing for targeted interventions.

5. Conclusion

These data represent the descriptive epidemiology of laboratory-confirmed cases only. To establish a comprehensive picture of the full magnitude of this outbreak and its epidemiology, further detailed analysis of all cases (clinical and laboratory-confirmed) is required.

Despite the many limitations of laboratory-based surveillance systems, the laboratory has played a critical role in confirming the existence of a cholera outbreak and the ongoing monitoring of both case and pathogen-specific trends for the provision of information to health authorities and clinicians. In recent weeks, as clinical cases decline, the role of the laboratory and its surveillance systems has changed with the emphasis on re-introduction of laboratory testing for all clinical cases in order to assist in determining when the end of this outbreak can be declared. Cases, however, continue to occur, and it is important for both

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clinicians and public health authorities to continue to be vigilant in the early detection and response to new cases. Emergency interventions for provision of safe water and sanitation in affected communities provide short term relief and must be replaced by the implementation of sustainable solutions in order to prevent further waterborne outbreaks in South Africa in the future.

6. Acknowledgements

We would like to thank and acknowledge the integral contributions and ongoing hard work of the following parties: the District, Provincial and National Departments of Health; the many participating laboratories of the NHLS, with special reference to the OutNet Representatives including Phadishi Mamaila and Greta Hoyland; the NHLS Corporate Data Warehouse team; the staff of the Enteric Diseases Reference Unit; and finally, the South African Field Epidemiology and Laboratory Training Programme (SA-FELTP).

References

1. World Health Organisation. Cholera. [Online]. Available from: www.who.int/topics/cholera/en/ [Accessed 4 April 2009].
2. Keddy KH, Nadan S, Govind C, Sturm AW for the Group for Enteric, Respiratory and Meningeal Disease Surveillance in South Africa. Evidence for a clonally different origin of the two cholera epidemics of 2001-2002 and 1980-1987 in South Africa. J Med Microbiol (2007) 56: 1644-50.
3. National Institute for Communicable Diseases. Communicable Disease Communiqué. 7(11)-8(4). Available from: www.nicd.ac.za.
4. Motlandille TW, Malaza AL, Archer BN, Maimela E, Moetlo P, Tint KS, et al. Trends and characteristics of cholera outbreak in Limpopo Province, South Africa; 15 November 2008 – 01 February 2009. Communicable Diseases Surveillance Bulletin 2009; 7(2). Available from www.nicd.ac.za/pubs/survbull/2009/CommDisBullMar09.pdf.
5. Archer BN, Motlandille TW, Malaza AL, Velen K, Sigudu T, Jacobs C, Malotle M, Modise M, Ntlebi. Update on Cholera in South Africa, 2008/9. Proceedings of the 1st Scientific Meeting of the SA-FELTP; 2009 Mar 11-13; Johannesburg, South Africa.

TRENDS AND CHARACTERISTICS OF CHOLERA OUTBREAK IN LIMPOPO PROVINCE, SOUTH AFRICA, 15 NOVEMBER 2008 TO 01 FEBRUARY 2009

Thejane Wilson Motladiile[1,2], Abraham Lucky Malaza[1,2], Brett Archer[1,3], Eric Maimela[4], Pauline Moetlo[4], Khin San Tint[1], Bernice N Harris[1,2]
[1]South African Field Epidemiology and Laboratory Training Programme (SA-FELTP), National Institute for Communicable Diseases
[2]School of Health Systems and Public Health, Faculty of Health Sciences, University of Pretoria, South Africa
[3]Outbreak Response Unit, Epidemiology Division, National Institute for Communicable Diseases
[4]Division of Epidemiology, Provincial Department of Health, Limpopo Province, South Africa

Abstract

Cholera continues to be a global threat, especially to the African continent. Conducting cholera surveillance in developing countries poses a serious challenge. We investigated the extent and characteristics of the cholera outbreak in Limpopo province, which began on 15 November 2008, to inform public health interventions. A clinical surveillance system was implemented that utilised line-list data routinely submitted from districts to the provincial Department of Health. This article represents data captured from 15 November 2008 to 1 February 2009 using *Epi-Info*™. A total of 2 170 suspected cholera cases with 35 deaths (case fatality rate: 1.6%) were reported. Of these, 400 cases and 15 deaths were laboratory confirmed. The estimated cholera incidence rate was 41 per 100 000 population. Of the affected individuals, 1 887 (88%), were aged more than five years and 1 110 (51%) were female. 493 patients (23%) were identified as Zimbabwean nationals. Improved surveillance systems provided a better understanding of the cholera outbreak in Limpopo Province.

Introduction

Cholera is an acute diarrhoeal disease caused by infection of the intestine, following ingestion of toxigenic gram-negative bacterium, *Vibrio cholerae* – serogroups O1 or O139, present in faecally contaminated water or food[1-5]. The disease is characterised in its most severe form by a sudden onset of acute watery diarrhoea that can lead to death (within hours) by severe dehydration and kidney failure[2-3,5]. Globally, cholera has claimed many lives throughout history and continues to be a threat, especially in Africa[6]. Between 1999 and 2005, there were over 1 million reported cholera cases and over 28 000 reported deaths worldwide. Africa accounted for about 90% of the cases and 96% of the deaths reported globally[6]. Each year, more than 100 000 cholera cases and 2 000-3 000 deaths are officially reported to the World Health Organization (WHO)[7]. The real figures for cholera are, however, thought to be much higher, and may be attributed to underreporting and other limitations of surveillance systems[7]. To our knowledge, a cholera outbreak was last reported in Limpopo province on 8 March 2002[8].

In this preliminary report, we use enhanced surveillance system data to describe the magnitude, characteristics and outcomes of an ongoing cholera outbreak in Limpopo, to guide and inform public health interventions.

Materials and Methods

This study was conducted in South Africa's northernmost province, which borders Mozambique to the east, Zimbabwe to the north and Botswana to the west (Figure 1)[9]. Limpopo Province occupies an area of 123 910 km^2 with an estimated total population of 5.3 million (5 274 800)[10,11] distributed across five districts (Figure 1). A suspected cholera case was defined as any person in Limpopo Province with acute onset of watery diarrhoea from 1 November 2008 to date. A confirmed case was defined as any individual who meets the clinical case definition on whom *V. cholerae* O1 was isolated from stool or a rectal swab. Specimens were initially collected from

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every case but as the outbreak progressed, a strategy of sporadic testing to monitor the continuing outbreak, was adopted.

Initially health facility cholera data were compiled into line-listings and forwarded to district data managers for capturing. Line-listings comprising patient's personal and demographic information, clinical features, laboratory results and clinical outcomes, were either faxed or transported to the provincial Department of Health (DoH). Due to the challenges of handling large amounts of data manually, an electronic surveillance system was established using *EpiInfo™ version 3.3.2*. District data managers were trained to routinely enter data in *Microsoft™ Excel* and e-mail the spreadsheet to the province for ongoing weekly analyses. Residents of the South African Field Epidemiology and Laboratory Training Programme (SAFELTP) assisted with the retrospective capturing of previously collected data. All the collected data were merged into an electronic database, and the system continues to run to date. Descriptive analysis of cleaned data collected from 15 November 2008 to 1 February 2009 (ending-term of analyses by SAFELTP) was performed using *EpiInfo™*, and is reported here. Rates were calculated as the number of cases per annum per 100 000 population. Univariate analysis of demographic, clinical and outcome variables was carried out to calculate frequencies of occurrence.

Results

Overview of the outbreak for 15 November 2008 to 1 February 2009, Limpopo Province

For this period, 2 170 suspected cholera cases with 35 deaths [case fatality rate (CFR): 1.6%] were reported (Table 1). .Of these, 400 cases and 15 deaths were laboratory confirmed. The estimated cholera incidence rate was 41 per 100 000 population per year (Table 1). Vhembe District recorded the highest number of cases (n=762, 35%) with an incidence rate of 60 cases per 100 000 population per year, whereas Mopani had the least number of cases (n=124, 6%) with an incidence rate of 11 cases 100 000 population per year (Table 1). Cholera incidence was higher (42 per 100 000 population per year) among males (Table 1). CFR was higher among females (1.9%), in Mopani district (4.0%), and among individuals in the 65-74 age group (4.9%) (Table 1).

Descriptive epidemiology

Information on gender was recorded for 2 167 (99.9%) cases of whom 1 110 (51%) were female (Table 1). Of 2 144 (98.8%) cases with recorded age, 258 (12%) were children under the age of five years (Table 1). Age ranged from under one to 98 years, with a median age of 25 years (interquartile range 15 to 41 years). The majority of the cases were aged 15-24 years (n=509, 24%), which recorded 20% (n=7) of the total deaths (Table 1). 1 218 (56%) cases were hospitalised. Travel history within the past seven days was obtained for 94 (4%) of patients of whom 10 (11%) had travelled to Musina and 14 (15%) to Zimbabwe.

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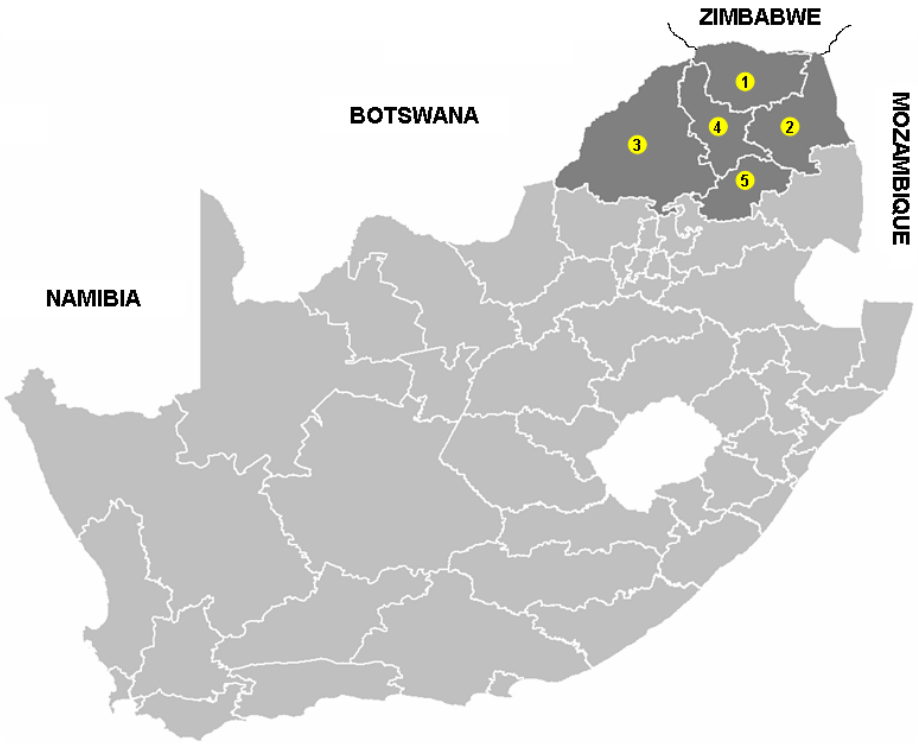

Figure 1: Map of South Africa depicting district health boundaries of Limpopo Province. *Key: 1 = Vhembe, 2 = Mopani, 3 = Waterberg, 4 = Capricorn, 5 = Sekhukhune.*

Table 1: Characteristics of patients with cholera during an outbreak in Limpopo Province, South Africa, 15 November 2008 – 01 February 2009.

Variables			Morbidity			Mortality		
		Population	N	%	†Rate	N	%	CFR %
District	Vhembe	1 276 502	762	35	60	9	26	1.2
	Capricorn	1 281 776	642	30	50	12	34	1.9
	Sekhukhune	996 937	456	21	46	7	20	1.5
	Waterberg	617 152	186	8	30	2	6	1.1
	Mopani	1 102 433	124	6	11	5	14	4.0
	Total	**5 274 800**	**2 170**	**100**	**41**	**35**	**100**	**1.6**
Gender	Female	2 768 100	1 110	51	40	21	60	1.9
	Male	2 506 700	1 057	49	42	14	40	1.3
	Total	**5 274 800**	**2 167**	**99.9**	**41**	**35**	**100**	**.6**
Age	<5	631 300	258	12	41	5	14	1.9
	5-14	1 337 600	248	11	19	-	-	-
	15-24	1 224 100	509	24	42	7	20	1.4
	25-34	770 900	400	19	52	3	9	0.8
	35-44	465 200	261	12	56	5	4	1.9
	45-54	352 200	156	7	44	4	11	2.4
	55-64	248 100	125	6	50	4	1	3.2
	65-74	156 100	103	5	66	5	14	4.9
	≥ 75	89 300	84	4	94	2	6	2.4
	Total	**5 274 800**	**2 144**	**98.8**	**41**	**35**	**100**	**1.6**
Nationality	South African	-	1 634	76.4	-	29	83	1.8
	Zimbabwean	-	493	23.1	-	6	7	1.2
	Other	-	7	0.3	-	-	-	-
	Mozambican	-	2	0.1	-	-	-	-
	Zambian	-	1	0.05	-	-	-	-
	Total	-	**2 137**	**98.5**	-	**35**	**100**	**1.6**

† = Incidence rate per 100,000 population based on the mid-year estimates from Statistics South Africa – 2008.
CFR = case fatality rate in percentages

Nationality of cases

The Limpopo cholera outbreak began on 15 November 2008 (Figure 2). Cases included individuals identified as South African (n=1 634; 76%), Zimbabwean (n=493; 23%), undetermined nationalities (n=7; 0.3%), Mozambican (n=2; 0.1%), and Zambian (n=1; 0.01%) (Table 1). During the first 28 days of the outbreak the majority of the 706 recorded cases were identified as Zimbabwean nationals (n=467, 66%) (Figure 2). From 13 December 2008 to 1 February 2009 the majority of patients were South African nationals (1 402/1 431; 98%). 29 cases (2%) were reported to be individuals of other nationalities during the same period (Figure 2).

Time course of the outbreak by district

The cholera outbreak commenced on the 15 November 2008 in Vhembe district. Within 19, 25, 26 and 55 days cholera was identified in Mopani, Waterberg, Capricorn, and Sekhukhune Districts respectively (Figure 3). Successive peaks were noted within the various districts, with an explosive course in Sekhukhune District towards mid-January 2009. Thereafter, a rapid decrease in case frequencies was noted, followed by another peak within Capricorn District (Figure 3). An increasing and decreasing pattern of spread was observed during the progression of the outbreak across the five districts.

Discussion and Conclusion

In the present analysis we used improved surveillance data to highlight the magnitude, epidemiological trends and characteristics of the cholera outbreak in Limpopo from 15 November 2008 to 1 February 2009. During this period, 2 170 suspected cases of cholera with a CFR of 1.6% were reported across the five districts. Most cholera cases were aged more than five years. In the initial phase of the outbreak the majority of patients were Zimbabwean nationals or residents, and the close proximity of Vhembe district to Zimbabwe is suggestive of an association between the South African and Zimbabwean outbreaks. An increasing and decreasing pattern of transmission observed during the progression of the outbreak across the five districts is suggestive of a propagated outbreak with the occurrence of person-to-person transmission (Figure 3). The peaking of the cases could be the result of a common source of infection present for a short period of time; however, we cannot rule out increased public health awareness and improved surveillance activities during these periods (Figure 3).

In conclusion, an improved surveillance system for clinical cholera cases has provided a better understanding of the real extent of the burden of disease in Limpopo. Cholera cases are still being reported; however, numbers are decreasing. The outbreak of cholera in Limpopo can be linked to the outbreak in Zimbabwe. A computerised system that facilitates routine data entry/transfer can expedite data cleaning, compilation and analysis, as well as report generation and supervisory feedback by Provincial and National Departments of Health, is

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recommended. Training and sensitisation of data managers about the need and the importance of rapid collecting health data are essential.

Acknowledgement

We would also like to extend our sincere gratitude to the South African National Department of Health, the Limpopo Department of Health (Division of Epidemiology and the Outbreak Response Team), the Outbreak Response Unit and the South African Field Epidemiology and Laboratory Training Programme (SAFELTP) in the National Institute for Communicable Diseases (NICD) for making this study possible. We are grateful to Dr Gillian M de Jong for entrusting us with this remarkable assignment as well as her continued support. Dr Cheryl Cohen is thanked for peer-reviewing this manuscript.

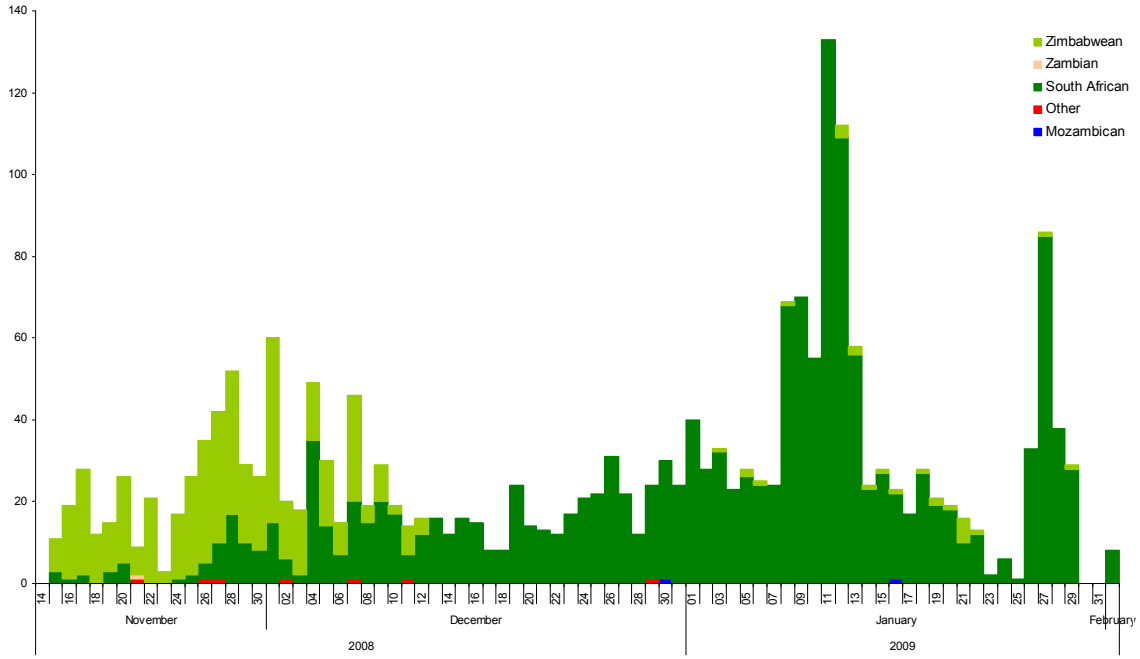

Figure 2: Epidemic curve showing the daily progression of cholera cases reported by nationality in Limpopo Province, South Africa, 15 November 2008 – 01 February 2009.

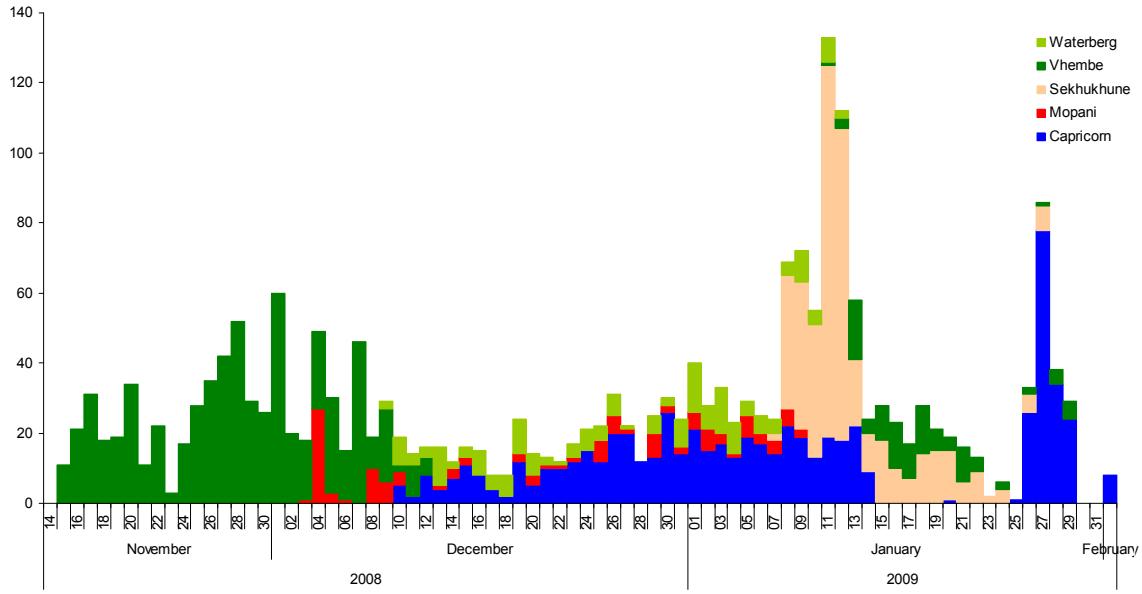

Figure 3: Epidemic curve showing the daily progression of cholera cases reported by district in Limpopo Province, South Africa, 15 November 2008 – 01 February 2009.

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References

1. Nelson EJ, Chowdhury A, Harris JB, Begum YA, Chowdhury F, Khan AI, *et al.* Complexity of rice-water stool from patients with Vibrio cholerae plays a role in the transmission of infectious diarrhea. PNAS 2007; 104(48):19091–19096.
2. Gaffga NH, Tauxe RV, Mintz ED. Cholera: A New Homeland in Africa? Am J Trop Med Hyg 2007; 77(4):705-713.
3. WHO, 2000. WHO Report on Global Surveillance of Epidemic-Prone Infectious Diseases. Geneva: World Health Organization, 1-54.
4. Zuckerman JN, Rombo L, Fisch L. The true burden and risk of cholera: implications for prevention and control. Lancet Infect Dis 2007; 7:521–30.
5. WHO, 2008. Prevention and control of cholera outbreaks: WHO policy and recommendations. Geneva: World Health Organization, 1-4.
6. Osei FB, Duker AA. Spatial and demographic patterns of Cholera in Ashanti region - Ghana. Int J Health Geogr 2008; 7(44):1-10.
7. Wiwanitkit V. Cholera outbreak in Thailand during the past 25-year period, a summary on epidemiology. Rev Esp Enferm Dig 2008; 100(4): 244-252.
8. Chabalala HP. A Report on Cholera Outbreak Response in Limpopo Province [Online]. [2002 April 01] [cited 2009 May 08]. Available from: URL: http://www.doh.gov.za/docs/reports/2002/cholera/limpopo.html
9. Maps of South African municipalities [Online]. [2008] [cited 2009 Feb 23]. Available from: URL: http://commons.wikimedia.org/wiki/File:NP03a2_locator.svg#file.
10. Statistics South Africa. Statistical release P0302. Mid-year population estimates. South Africa, Pretoria; 2008.
11. Lehohla P. Provincial Profile 2004. Limpopo. Statistics South Africa. South Africa, Pretoria; 2006.

DIARRHOEAL DISEASE OUTBREAK DURING A SCHOOL WHITE WATER RAFTING TRIP - ZAMBEZI RIVER, AUGUST 2008

Genevie M Ntshoe[1, 2], A Malaza[1, 2], Elizabeth Prentice[1, 3], Khin San Tint[1], Faustine Ndugulile[1], Bernice Harris [1,2,3]
[1]South African Field Epidemiology and Laboratory Training Program (SAFELTP)
[2]School of Health Systems and Public Health, University of Pretoria
[3]National Institute for Communicable Diseases (NICD)

Introduction

Diarrhoea is a common disease[1], and can be caused by viral, bacterial or parasitic organisms[2]. Bacteria such as *Salmonella* species and *Campylobacter* species are among the common causes of diarrhoea and these cases are likely to be more severe than viral diarrhoeal cases in people greater than 5 years[1]. Infection is acquired and spread through contaminated food or drinking water. Person to person transmission may also occur as a result of poor hygiene[2]. The World Health Organization (WHO) defines diarrhoea as the passage of 3 or more loose/liquid stools per day or more often than is usual for the individual[2].

In October 2008, the South African Field Epidemiology and Laboratory Training Programme (SAFELTP) were informed of a diarrhoeal disease outbreak that occurred during a seven day school trip to the Zambezi River. A total of 99 males which included 85 learners, 7 teachers and 7 parents undertook a river-rafting trip to the Zambezi. They left South Africa (SA) by air on the 22nd August 2008 landing in Zambia at Livingstone Airport the same day (Figure 1). They proceeded across the border by bus to Zimbabwe and visited Bungi Bridge located on the Victoria Falls Bridge. The river-rafting expedition started on day 2 of the trip which took place in the Zambezi gorge for 4 days. The majority of participants developed diarrhoea during their stay in the gorge. On day 6 of the trip, the participants went on a boat cruise. The trip ended on the 28th August 2008 when they returned to SA. The study was conducted to identify possible sources of infection and to institute control measures for future school trips.

Methods

Initial interviews were conducted on a limited number of cases among the learners; one was done with a hospitalized learner and the two other interviews were conducted via the telephone. Interviews were followed by a retrospective cohort study on the 7th October 2009. Self administered questionnaires were used, and were completed at school under the supervision of the investigating team. Learners and teachers who were part of the trip to the Zambezi participated in this process. Parents were forwarded questionnaires for self-administration. Information gathered from questionnaires included risk factors like food related questions, visit to farms and contact with animals, attendance at large gatherings beside the trip, and activities undertaken during the trip. Food specific relative risks (RR), their 95% confidence intervals and one tailed fisher exact p-values were calculated using EpiInfo version 3.3.2.

Results

Descriptive Epidemiology

The study response rate was 80% (79/99). Out of 79 respondents (77 learners and 2 staff members), 75 reported diarrhoeal disease, with an attack rate of 95%. Of the 75 diarrhoeal cases, 74 were students and 1 staff member. All participants were males, age range from 16 to 44, with a median of 17 years. Seventeen patients (23%) were aged 16 years; 54 (72%) were aged 17 years; 3 (4%) were aged 18 years and 1 was aged 44 years. The outbreak started with 6 patients on day three of the trip and the number increased in the following two days (Figure 1). The most common symptoms reported were watery diarrhoea 75/75 (100%), fatigue 60/75 (80%), nausea 51/75 (68%) and abdominal pains 48/75 (64%); Table 1. Although mostly self-limiting the duration of illness exceeded 7 days in 29% (22/75) of the cases, and ranged from 1-2 days to more than 7 days (Figure 2).

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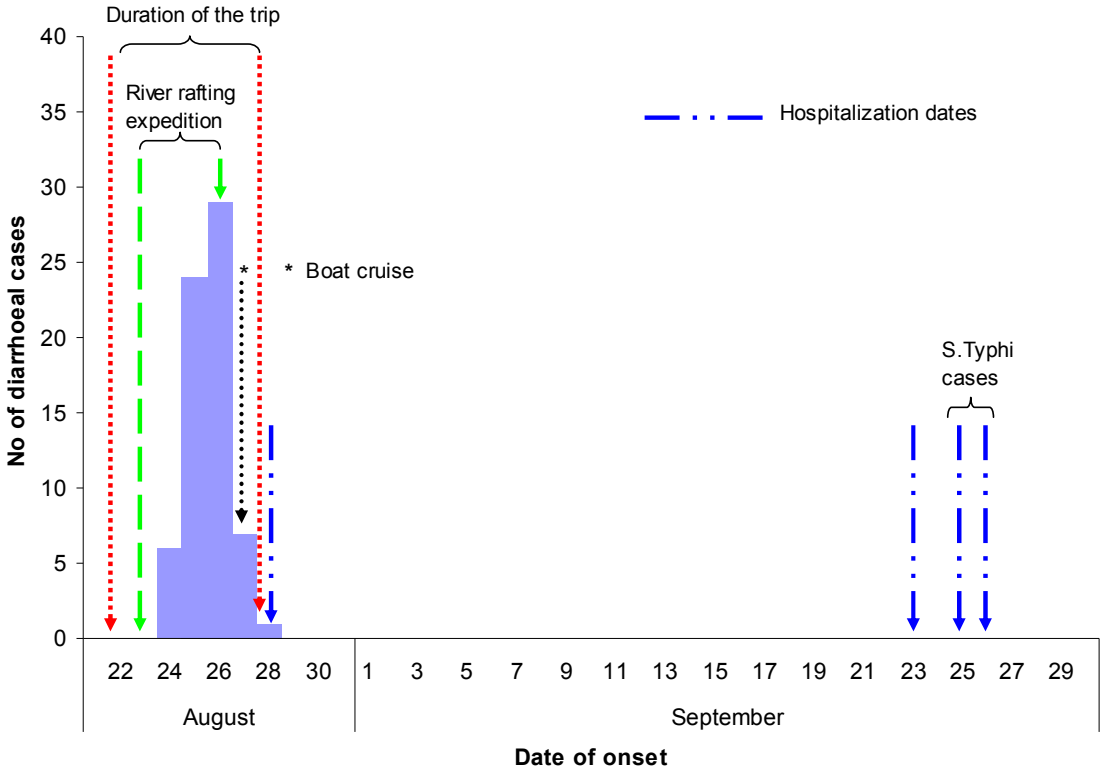

Figure 1: Epidemic curve, diarrhoeal disease outbreak, Zambezi River, August 2008

Table 1: Symptoms reported by those who got ill in Zambezi River, 24-28 August 2008 (n=75)

Symptoms	Frequency	%
Diarrhoea	75	100
Fatigue	60	80
Nausea	51	68
Abdominal pain	48	64
Fever	39	52
Chills	31	41
Vomiting	30	40
Body aches	24	32
Bloody stool	3	4

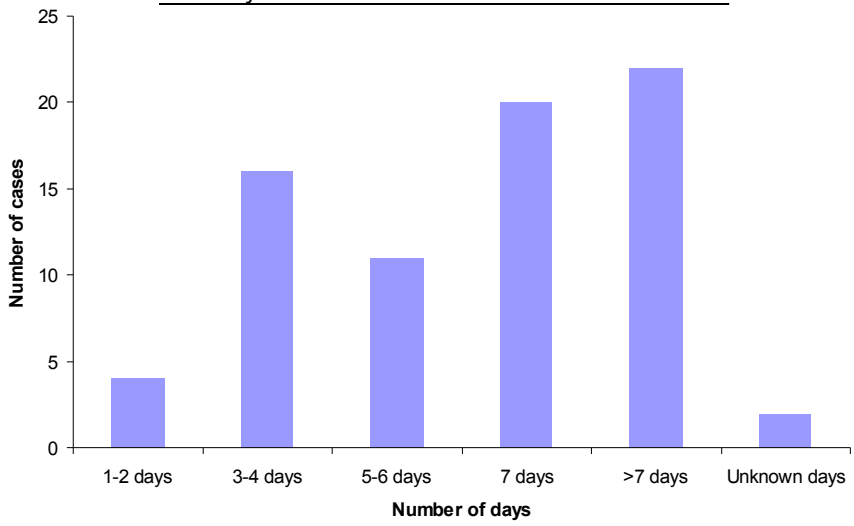

Figure 2: Duration of symptoms among diarrhoeal cases who attended the White Water rafting trip, Zambezi River, with onset 24-28 August 2008.

Immediately after the trip, the school issued letters to the participants advising them to take antibiotic treatment (ciprofloxacin) for their illness. Fifty-five reported that they visited a doctor upon return and of these; 21 were in August, 19 in September and one in October. Only one learner had culture specimen collected immediately after the trip. The specimen was collected on the 29th of August 2008, and the result was negative. Eight cases recovered partially after the trip and then became ill again. These cases consulted their doctors and culture specimens were collected from seven of them; date of collection ranged from 26 September to 6 October 2008. Four learners were hospitalized after their return (Figure 1). One learner was hospitalized immediately after the trip, from the 28 to 29 August 2008. Culture specimen was not collected from this learner. The other three learners became hospitalized from 23 September to 06 October 2008, specimens were taken for culture from two of these. Of the three learners, the first one was hospitalized from 23 to 25 September, the second one from 25 September to 06 October, and the third one from the 26 September to 01 October 2008. Hospitalization days ranged from 1 to 11 days, with mean length of 5 days. *Salmonella* Typhi was cultured in 2 of these patients (one from blood one from stool). Both of these patients received treatment with flouroquinolones and have recovered. *Salmonella enteritidis* was cultured from stool in a further 4 students, one of whom had a mixed infection with *Campylobacter species*. All 75 affected individuals have recovered. A household contact of one of the cases developed similar symptoms and was treated empirically with a fluoroquinolone 24 hours prior to specimen collection. A causative organism could not be isolated and the contact has since recovered. Contact tracing was performed but no further cases were reported.

Investigation into possible sources of infection

It was not clear how often fresh food was supplied and how it was stored throughout the 4 days in the gorge, but some of the participants indicated that food was stored in cooler boxes. We did not have detailed menu of all meals consumed during the trip while conducting the investigation. No food samples were tested to determine the source of infection as the outbreak was conducted after the trip ended. Potable water was provided and boiled river water was also used. During our interviews, lack of toilets and hand washing facilities were highlighted by the learners. This would have further enhanced the transmission of any enteric pathogens. The nature of the activity exposed learners to river water splashes and accidental river-water swallowing. This too could be a possible source of infection.

Analytic epidemiology

There were no significant associations between diarrhoeal disease and consumption of specific foods (Table 2).

Discussion

Salmonella Typhi, *Salmonella enteritidis* and *Campylobacter* species were the only pathogens isolated. Incubation period for *Salmonella* Typhi depends on inoculum size and on host factors; it can be from 3 to more than 60 days, but usually ranges from 8-14 days[3]. The incubation period for *Salmonella enteritidis* ranges from 6-72 hours but is usually 12-36 hours[3]. Incubation period for *Campylobacter species* depend on dose ingested, usually 2-5 days with a range of 1-10 days[3]. *Salmonella enteritidis* was the most common *Salmonella* serotype in diarrhoeal stool specimens taken (4/7), and detected in (4/8) of the specimens cultured. *Salmonella enteritidis* infections are escalating in many developed countries and are been related with consumption of infected poultry, eggs and egg products[4]. This outbreak showed no association between poultry and salmonella infection. The epidemic curve is suggestive of a point source with substantial secondary spread (person to person transmission) or possibly a sequence of exposures. The majority of cases occurred 2 days after the first diarrhoeal cases were reported.

Some patients developed acute diarrhoea followed by after partial recovery and then worsening clinical picture. S. typhi was identified from two of these patients. This type of picture may occur in outbreaks following consumption of food or water contaminated with multiple pathogens with differing incubation periods.

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Table 2: Measures of association between food consumed and disease during the trip to Zambezi River, August 2008

	Ate		Did not eat		RR*	95% CI**	p-value
	ill	Well	ill	Well			
Fruit	73	3	1	0	1.92	0.48-7.68	NS†
Meat	71	3	4	0	0.96	0.92-1.01	NS
Milk	62	2	11	1	1.05	0.89-1.26	NS
Poultry	68	3	7	0	0.96	0.91-1.01	NS
Salad	69	2	5	1	1.17	0.81-1.67	NS
Beef	72	3	3	0	0.96	0.92-1.01	NS
Raw eggs	4	0	52	3	1.06	0.99-1.13	NS
Pork	38	3	21	0	0.93	0.85-1.01	NS
Vegetables	69	2	4	1	1.21	0.78-1.89	NS
Mayonnaise	49	1	18	1	1.03	0.92-1.16	NS

* Relative Risk
** Confidence Interval
† Not significant, p >0.05

Limitations

Since the river-rafting was in the gorge for 4 days, it was not clear how often fresh food was supplied. Recall bias was a major limitation in this investigation. Most of the participants could not recall what food they ate during the trip. Recall bias may also have affected the reporting of symptoms, duration of illness and food consumed. These biases could lead to differential misclassification. Differential misclassification can either overestimate or underestimate the true association, depending on the circumstances. This further hampered the investigators in their identification of the source of infection. Furthermore, the risk associated with drinking water could not be assessed since everybody drunk water, thus there was no unexposed group. High attack rate meant that there were few unaffected individuals this reduced the ability to detect significant associations

Conclusion

No source of infection was identified. Limitations of this study were the lack of a detailed menu of all meals eaten during the trip as well as the recall bias. Many factors may be associated with the occurrence of this outbreak: a combination of possibly poor food and personal hygiene, lack of proper food storage, unsafe water usage, and inadequate toilet facilities. Exposure to potentially contaminated river water also poses a risk during recreational activities. We recommended the guidelines for safe handling of food; safe drinking water and proper hand washing using approved antiseptic hand gels after toilet use when tap water is not available. When traveling, access to safe food and water is essential in preventing transmission of enteric pathogens. If self-catering, proper refrigeration of foods and adequate cooking temperatures must be achieved. Boiling of untreated water for at least three minutes is required to render it safe for drinking. Manual chlorination of drinking water may also be used.

Acknowledgements

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References

1. Virtual medical centre. Diarrhoea. Available at http://www.virtualmedicalcentre.com. Cited on 27/11/2008
2. World Health Organization (WHO). Available at http://www.who.int/topics/diarrhea/en/. Cited on 26/11/2008
3. Heymann DL. Control of Communicable Diseases Manual. 18th Ed, 2004.
4. Simango C, Mbewe C. Salmonella enteritidis diarrhea in Harare, Zimbabwe. *Tropical Medicine & International Health*. 25 Dec 2001; 5 (7): 503-06

Erratum

Communicable Diseases Surveillance Bulletin, March 2009

Page 18: Table 3. Results of antimicrobial susceptibility testing for all *Salmonella* Typhi isolates (n = 80) received by EDRU, 2008, excluding isolates identified by audit. One isolate of *Salmonella* Typhi was incorrectly reported as resistant to ciprofloxacin. The correct data are that all isolates identified that were fully susceptible to ciprofloxacin.

Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 31 March 2008/2009*

Disease/Organism	Cumulative to 31 March, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Anthrax	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	0	0	0	0	0
Botulism	2009	0	0	0	0	0	0	0	0	0	0
	2008	0	0	0	0	0	0	0	0	0	0
Cryptococcus spp.	2009	410	175	578	390	119	265	16	217	192	2362
	2009	393	135	631	342	160	242	21	209	164	2297
Haemophilus influenzae, invasive disease, all serotypes	2008	9	5	48	9	2	3	0	1	17	94
	2009	6	2	39	16	0	8	1	3	25	100
Haemophilus influenzae, invasive disease, < 5 years											
Serotype b	2008	2	3	3	1	0	0	0	1	4	14
	2009	0	1	5	6	0	0	1	0	7	20
Serotypes a,c,d,f	2008	0	1	6	0	0	0	0	0	0	7
	2009	0	1	7	0	0	1	0	0	3	12
Non-typeable (unencapsulated)	2008	0	0	5	0	0	0	0	0	1	6
	2009	0	0	3	3	0	0	0	0	3	9
No isolate available for serotyping	2008	4	0	18	2	1	2	0	0	6	33
	2009	1	0	7	4	0	3	0	1	3	19
Measles	2008	1	0	3	0	0	1	0	1	2	8
	2009	2	0	7	1	0	2	0	1	2	15
Neisseria meningitidis, invasive disease	2008	3	4	44	2	0	7	2	0	9	71
	2009	5	1	33	12	0	4	0	2	18	75
Novel Influenza A virus infections	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	0	0	0	0	0
Plague	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	0	0	0	0	0
Rabies	2008	2	0	0	2	3	0	0	0	0	7
	2009	3	0	0	3	0	0	0	0	0	6
**Rubella	2008	29	1	9	9	6	4	0	11	3	72
	2009	26	1	5	23	6	20	10	7	11	109
Salmonella spp. (not typhi), invasive disease	2008	17	15	159	24	3	8	4	6	17	253
	2009	22	4	99	28	0	13	3	7	25	201
Salmonella spp. (not typhi), isolate from non-sterile site	2008	59	10	105	41	2	27	4	3	39	290
	2009	53	16	144	19	10	37	16	19	56	370
Salmonella typhi	2008	1	1	10	0	1	7	0	0	2	22
	2009	2	1	9	1	0	1	0	0	3	17
Shigella dysenteriae 1	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	0	0	0	0	0
Shigella spp. (Non Sd1)	2008	53	19	148	35	1	21	6	3	131	417
	2009	73	24	169	32	2	28	9	13	166	516
Streptococcus pneumoniae, invasive disease, all ages	2008	61	46	384	92	15	52	13	24	120	807
	2009	84	62	415	106	12	39	17	28	152	915
Streptococcus pneumoniae, invasive disease, < 5 years	2008	12	21	127	37	6	23	4	5	52	287
	2009	31	22	121	37	4	12	8	7	57	299
Vibrio cholerae O1	2008	0	0	0	0	0	1	0	0	0	1
	2009	0	0	35	0	389	61	0	18	4	507
Viral Haemorrhagic Fever (VHF)											
Crimean Congo Haemorrhagic Fever (CCHF)	2008	0	1	0	0	0	0	2	0	0	3
	2009	0	0	0	0	0	0	0	0	0	0
Other VHF (not CCHF)	2008	0	0	6	0	2	2	0	0	0	10
	2009	0	0	0	3	0	0	0	0	0	3

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

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

U = unavailable, 0 = no cases reported

Table 2: Provisional laboratory indicators for NHLS and NICD, South Africa, corresponding periods 1 January - 31 March 2008/2009*

Programme and Indicator	Cumulative to 31 March, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
Acute Flaccid Paralysis Surveillance											
Cases < 15 years of age from whom specimens received	2008	12	6	22	9	14	6	1	2	8	80
	2009	9	2	16	28	6	14	3	4	7	89
Laboratory Programme for the Comprehensive Care, Treatment and Management Programme for HIV and AIDS											
CD4 count tests											
Total CD4 count tests submitted	2008	72483	30020	130860	160861	44637	42623	48346	11477	45667	586974
	2009	93514	32605	166076	210960	61084	60795	60878	5447	57895	749254
Tests with CD4 count < 200/µl	2008	29925	9940	50933	56743	16432	16139	15893	3458	13307	212770
	2009	31653	9634	57524	66946	19815	20573	18635	867	16779	242426
Viral load tests											
Total viral load tests submitted	2008	30049	12114	56715	44902	19380	14338	19092	4341	14776	215707
	2009	35969	12544	81517	94304	27262	24050	27165	2475	21071	326357
Tests with undetectable viral load	2008	14190	6961	33383	25529	10867	7691	11880	2283	11823	124607
	2009	19644	9618	51299	58915	16990	13636	17188	1496	17117	205903
Diagnostic HIV-1 PCR tests											
Total diagnostic HIV-1 PCR tests submitted	2008	8238	3327	18017	9150	5194	3059	4597	1041	6124	58747
	2009	7588	2907	14575	16146	4166	4085	4552	110	4743	58872
Diagnostic HIV-1 PCR tests positive for HIV	2008	796	471	2171	2139	747	491	670	133	473	8091
	2009	861	381	1649	1791	607	557	632	66	430	6974

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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