

COMMUNICABLE DISEASES SURVEILLANCE BULLETIN

NOVEMBER 2009



FOREWORD

South Africa is currently experiencing a widespread measles outbreak. This outbreak began in Gauteng but to date all 9 provinces have reported confirmed measles cases¹. Fellows from the South African Field Epidemiology and Laboratory Programme (SA-FELTP) have been involved in assisting with investigations related to this outbreak as well as other recent outbreaks. In this bulletin we include three outbreak related articles compiled by SA-FELTP fellows (two related to measles from different districts in Gauteng and one related to hepatitis A). These articles highlight the value of such a training programme in producing timeous detailed outbreak reports from the field. In addition we include a third article describing the measles outbreak nationally from national laboratory-based surveillance data. We hope that these articles will provide useful information to guide response to current and future outbreaks.

Reference

1. Malotle M, Nteo MD, Harris BN, Cohen C, McAnerney JM, Mashele M, Mahlaba L, Smit S, Moshime M, van der Gryp R. Measles outbreak, Tshwane, South Africa, 2009. *Comm Dis Surveill Bull* November 2009; 7(4): 10.

Cheryl Cohen, Editor

CONTENTS

Hepatitis A outbreak in Tshwane District, Gauteng Province, South Africa, May-June 2009	1
Measles outbreak in the City of Johannesburg, Gauteng Province, 23 August 2009 to 1 November 2009—Interim report	6
Measles outbreak, Tshwane, South Africa, 2009	10
Measles outbreak, South Africa, 2009, data on laboratory-confirmed cases	15
Table 1: Provisional listing of laboratory-confirmed cases of diseases under surveillance : 01 January—30 September 2009	22
Table 2: Provisional laboratory indicators for NHLS and NICD: 01 January—30 September 2009	23

HEPATITIS A OUTBREAK IN TSHWANE DISTRICT, GAUTENG PROVINCE, SOUTH AFRICA, MAY-JUNE 2009

Motshabi Modise^{1,3}, Thejane Motladiile^{1,3}, Genevieve Ntshoe^{1,3}, Rina van der Gryp⁴, Ayanda Cengimbo², Bernice Harris^{1,3}, Lucille Blumberg²

¹South African Field Epidemiology and Laboratory Training Programme (SAFELTP) and ²Outbreak Response Unit, National Institute for Communicable Diseases, ³School of Health Systems and Public Health, University of Pretoria, South Africa, ⁴City of Tshwane Metropolitan Municipality Department of Health, Gauteng Province, South Africa

Introduction

Hepatitis A is the most common cause of acute viral hepatitis in many parts of the world including South Africa.¹ Although the virus is endemic in southern Africa, the true burden of disease is unknown. While infection in childhood is frequently mild, severe disease can occur in certain high risk groups and disease in adults is often associated with significant morbidity. Localised and more widespread community and institutional outbreaks occur in South Africa and frequently raise challenges for control given limited

resources. South Africa has a unique epidemiological pattern of disease with variation in rates of infection across different socioeconomic groups and provinces.¹

Infection with hepatitis A virus (HAV) causes acute inflammatory disease of the liver.^{2,3,4,9} HAV is transmitted faecal-orally, even in microscopic amounts, either from close person-to-person contact or ingestion of faecally contaminated food or water.²⁻⁴ Signs and symptoms may

(Continued on page 2)

NATIONAL INSTITUTE FOR COMMUNICABLE DISEASES

Requests for e-mail subscription are invited - please send request to Mrs Liz Millington:
lizm@nicd.ac.za

Material from this publication may be freely reproduced provided due acknowledgement is given to the author, the Bulletin and the NICD.

WEB

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



include: fever, fatigue, loss of appetite, nausea, vomiting, abdominal pain, dark urine, clay-coloured bowel movements, joint pain and jaundice.^{5,8} Symptoms usually appear 28 days after exposure, but can occur between 15 and 50 days.^{6,7} Clinical presentation may vary and include asymptomatic infection mostly in children, symptomatic hepatitis with or without jaundice, fulminant hepatitis with acute liver failure, cholestatic hepatitis and relapsing hepatitis¹. Hepatitis A diagnosis is confirmed by a positive serological test for immunoglobulin M (IgM) antibody to hepatitis A virus.^{4,5}

During May 2009, the Gauteng Department of Health informed the National Institute for Communicable Diseases (NICD) of an outbreak of Hepatitis A in Pretoria North, Tshwane District in Gauteng Province. Fourteen cases, of which 10 were laboratory confirmed, were initially reported to the Department of Health. The first case was notified to the Communicable Disease Control Coordinator, City of Tshwane on 11 May 2009 and additional cases continued to be notified. An outbreak investigation was conducted to describe the characteristics and determine the extent and possible source of the outbreak, as well as institute prevention and control measures.

Methodology

Case definitions

- Clinical case definition: a person who presented with symptoms of jaundice, dark urine, abdominal pain, vomiting and/or loss of appetite associated with hepatitis A and/or is notified to the Department of Health as a suspected hepatitis A case.
- Laboratory diagnostic criteria: A positive immunoglobulin M (IgM) antibody to hepatitis A virus (anti-HAV IGM) laboratory result.

Case classification

- Suspected case: a case that meets the clinical case definition
- Probable case: a case that meets the clinical case definition and has an epidemiologic link with a laboratory confirmed case.
- Confirmed case: a case that meets the clinical case definition and is laboratory confirmed

To establish the extent and possible source(s) of the outbreak, a number of activities were carried out: a surveillance system was established for new and missed cases through active case finding and a retrospective record review at laboratory and health care facilities where patients were seen or hospitalised; patients were interviewed telephonically to gather more information relating to past travels and events attended; site visits were conducted to gather additional information regarding patients' clinical diagnoses. A comprehensive line-list was compiled from these activities.

Intervention measures that were instituted include: environmental assessment to identify the possible sources/

exposures, health promotion activities emphasizing good hygiene practices and importance of hand washing, as well as the administration of post-exposure prophylaxis (PEP) in the form of pooled immunoglobulin (intragam).

Results

Descriptive Epidemiology

During this investigation we determined and compared the number of cases reported from March to June from Tshwane since 2005. The cases increased from 24, 27, 31, 36 and 61 in 2005, 2006, 2007, 2008 and 2009, respectively. A marked increase in the number of cases reported from 2008 to 2009 confirmed an outbreak of hepatitis A in Pretoria North. Here we report on 33 cases that were notified and investigated from 2 March 2009 to 9 June 2009.

Thirty three cases with a diagnosis of suspected hepatitis A infection were identified from 2 March 2009 to 9 June 2009 (Figure 1). Of these, 67% (n=22) were laboratory confirmed, 21% (n=7) were probable and 12% (n=4) suspected (Figure 1). Blood specimens were taken in 24 of the 33 cases, and 22 tested positive for anti-HAV IgM. Two suspected cases tested negative for anti-HAV (IgM), and were excluded. Of the remaining 31 cases, 15 were children under the age of 18 years and 16 were adults. Age ranged from 18 months to 40 years with the median of 19 years. The highest proportion of cases (n=8, 24%) were aged 5 to 9 years followed by 25 to 29 years (n=6, 19%). 55% (17/31) of cases were female.

Table 1: Age distribution of cases, hepatitis A outbreak, Tshwane District, Gauteng Province, March to June 2009

Age Group	Frequency	Percentage
0 – 4	3	10
5 – 9	8	26
10 – 14	3	10
15 – 19	2	6
20 – 24	4	13
25 – 29	6	19
30 – 34	2	6
35 – 39	2	6
40 – 44	1	3
Total	31	100

Most cases 48% (n=15) were reported in the Pretoria North area while the rest were from other suburbs in the northwest of Pretoria. Almost 70% of cases (n=19, 68%) had a link to three schools and two crèches (Figure 3). Seven household clusters were identified: one household had four cases, two households had three cases each and another four households had two cases each (Figure 3).

(Continued on page 3)

Post exposure prophylaxis was given to 52 % (n=16) of household contacts of cases using pooled immunoglobulin (Intragam). Among the sporadic cases, one confirmed

case, seen at one facility in Daspoort was lost to follow-up for collection of clinical and epidemiologic information.

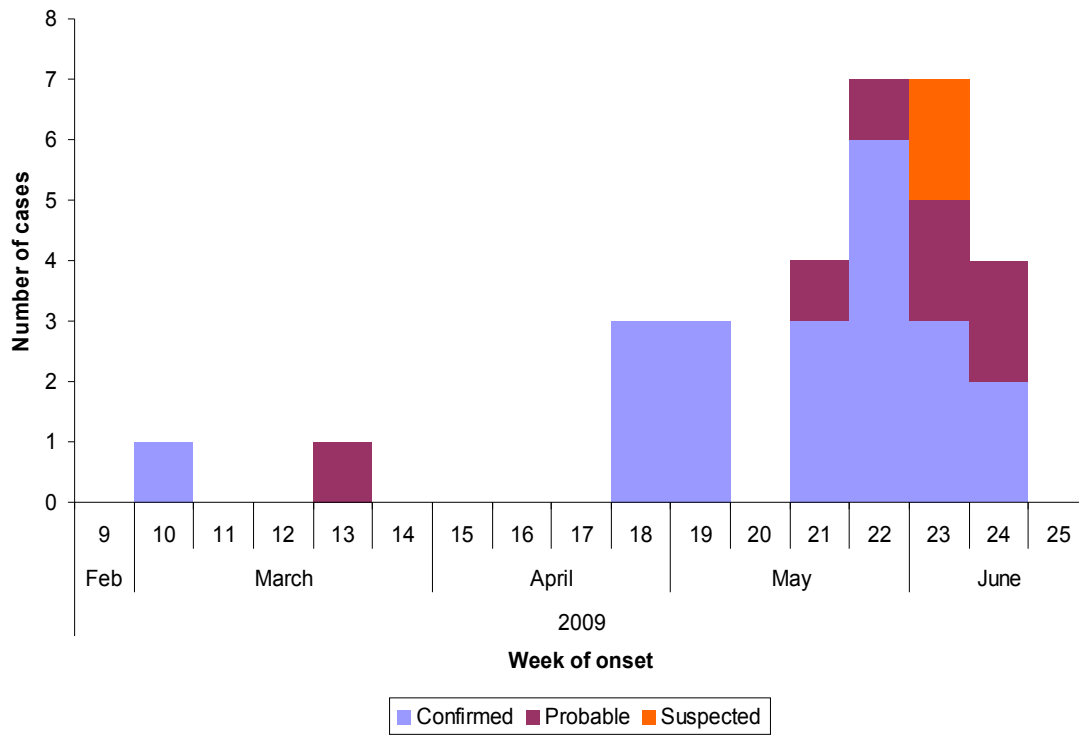


Figure 1: Epidemic curve, hepatitis A outbreak, Tshwane District, Gauteng Province, March to June 2009

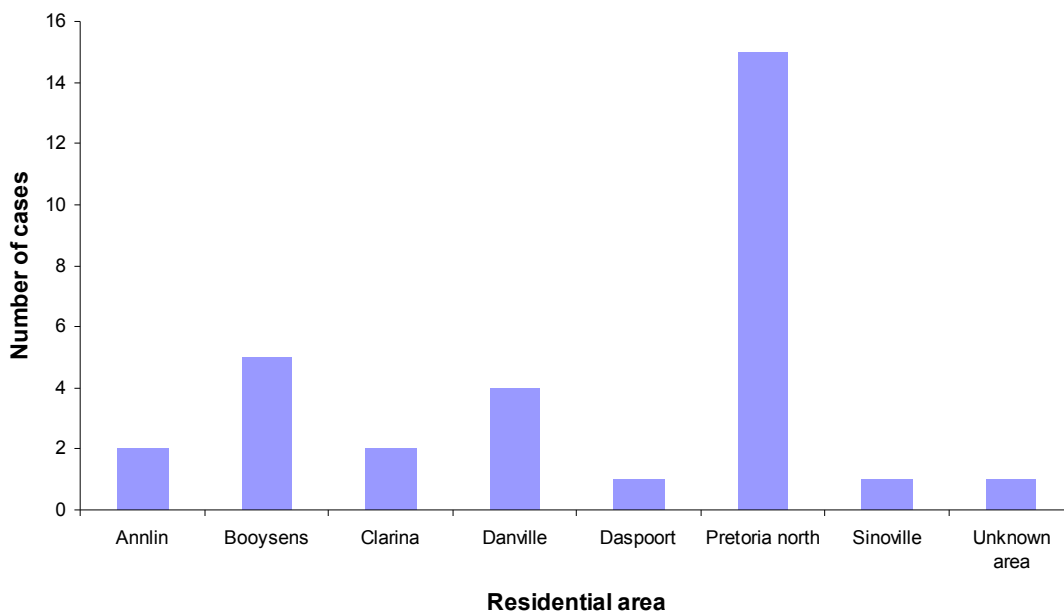


Figure 2: Frequency distribution of cases by area, hepatitis A outbreak, Tshwane District, Gauteng Province, March to June 2009

(Continued on page 4)

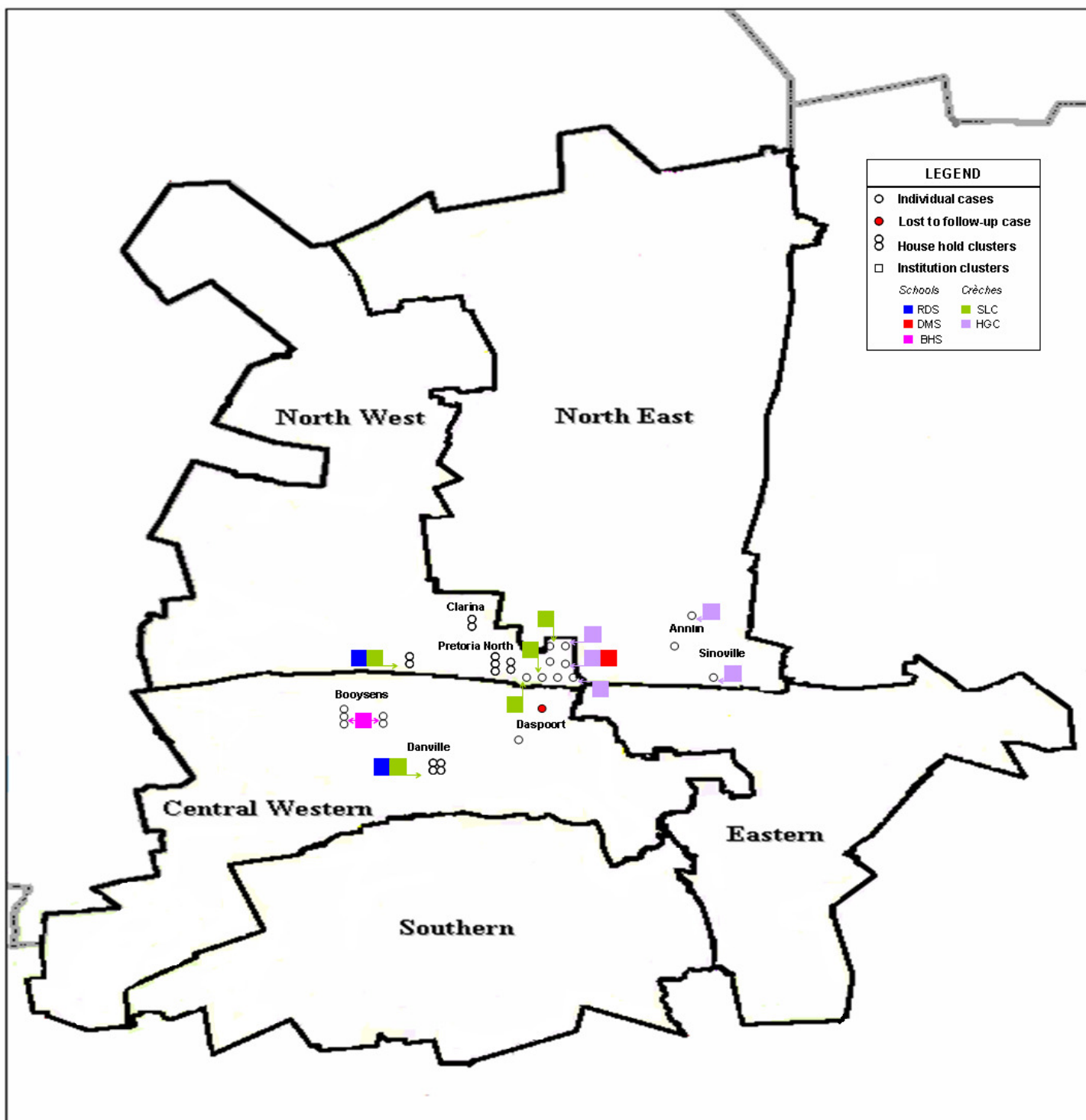


Figure 3: Geographic distribution of cases, hepatitis A outbreak, Tshwane District, Gauteng Province, March to June 2009

Signs and symptoms

Interviews were conducted with 28/31 cases to obtain detailed clinical, demographic and epidemiological information. Among these, the most common symptoms reported were: jaundice (n=21, 75%), dark urine (n=20, 71%), loss of appetite (n=19, 68%) and vomiting (n=18, 64%) (Table 2).

Four (14%) cases were hospitalised, and one (3%) case relapsed and still had jaundice at the time of the interview. One death, an 18 month old baby, was reported and notified to the Department of Health as fulminant hepatitis A (CFR: 3%), and the diagnosis was reportedly confirmed by post-mortem results, however we were not able to access the post mortem report. There were no laboratory results to confirm the diagnosis.

(Continued on page 5)

Table 2: Symptoms reported by interviewed hepatitis A cases, Tshwane District, Gauteng Province, March to June 2009 (n=28)

Clinical signs and symptoms	Frequency	%
Jaundice/yellowing of skin or eyes	21	75
Dark urine	20	71
Loss of appetite	19	68
Vomiting	18	64
Nausea	17	61
Abdominal pain	17	61
Fever	17	61
Arthralgia/Myalgia	9	32
Fatigue	8	29
Diarrhoea	7	25
Grey stool	6	21
Total cases	28	100

During interviews, cases were asked about possible risk factors including gatherings attended, work/school, eating out and water sources. No common epidemiological factor was found that could link all the cases. In addition we were not able to identify the source of the outbreak.

Environmental assessment was done by the investigating team at one school and two day-care centres/crèches and findings were as follows: one day-care/crèche which had three cases, and a school had proper toilet facilities with soap and municipal water, in which children were washing their hands before eating and after toilet use. Another day-care/crèche was found to be over-crowded and had four cases of hepatitis from three distinct households.

48% (n=15) of the household contacts could not receive PEP because they could not afford to pay for it at the private healthcare facilities, and that it was not available within the public healthcare facilities.

Discussion

We described an outbreak of hepatitis A in Tshwane District, Gauteng Province of South Africa, which occurred from 2 March 2009 to 9 June 2009. This outbreak was detected through a mandatory notification supporting the district's surveillance system. The outbreak included a total of 33 cases with 1 probable death due to a rare but devastating complication, fulminant hepatitis A, which results in 70-95% mortality rates¹. Of the total cases, 75% presented mainly with jaundice and 67% were laboratory confirmed. Males and females, and children under the age of 18 and adults were affected by hepatitis A in nearly equal proportions.

We could not identify the source of the outbreak and there were no identified activities linking all of the cases. The possible link among the cases included mainly three crèches and two schools, from which a secondary propagation of infection was noticed at the level of seven households. Thus, we could not identify the source of the

outbreak, and neither could qualify it to either being a common point source or multiple sources.

Limitations

Our investigation had the following limitations. Most cases interviewed could not recall the dates they visited restaurants. Not all of the cases were available to be interviewed. Data were wrongly entered in the notification forms; in some instances date of consultation was recorded/reported as date of onset, and wrong/previous addresses were used.

Recommendations

Laboratories and private health practitioners must be encouraged to report notifiable diseases to the Department of Health. The Department of Health must ensure that PEP is available at public health care facilities, as some cases mentioned that their contacts could not afford private healthcare and PEP was unavailable at the public facilities. Thorough surveillance and prompt reporting all cases of notifiable disease presenting at facilities may have enable the investigating team to identify the source.

Conclusion

We described an outbreak of hepatitis A in Tshwane District that was linked to three schools and two crèches. Seven household clusters as well as sporadic unlinked cases were identified. Although some clusters involving households with children attending the same school and after school care were identified, no common epidemiological factor was found that could link all cases.

Acknowledgements

We would like to thank all the healthcare facilities in the study area; schools and crèches; parents; National Health Laboratory Services (NHLS), Outbreak Response Unit, NICD; Private laboratories - Lancet and Ampath; Mr Leon Van Zyl, Tshwane Local Authority, Department of Health; Mrs Mmampedi Huma, SAFELTP; Dr David Mutonga, TEPHINET/CDC-Fellow.

(Continued on page 6)

References

1. NICD Guidelines for the control of hepatitis A in South Africa. Outbreak Unit, Epidemiology Division, National Institute for Communicable Diseases of the National Health Laboratory Service 2008. Johannesburg – South Africa. http://www.nicd.ac.za/pubs/other/NICD_Guidelines_Hepatitis_A_in_SA.pdf
2. Ngui SL, Granerod J, Jewes LA, Crowcroft NS, Teo CG. Outbreaks of Hepatitis A in England and Wales Associated With Two Co-Circulating Hepatitis A Virus Strains. *J Med Virol* 2008; 80:1181–1188
3. Villar LM, da Costa MCE, de Paula VS, Gaspar AMC. Hepatitis A Outbreak in a Public School in Rio de Janeiro, Brazil. *Mem Inst Oswaldo Cruz* 2002; 97(3):301-305
4. World Health Organization. Hepatitis A. Department of Communicable Disease Surveillance and Response [Online]. [2000][cited 2009 July 07]. Available from: URL: <http://www.who.int/emc>
5. de Souza LJ, Braga LC, de Souza Maciel Rocha N, Tavares RR. Acute Acalculous Cholecystitis in a Teenager with Hepatitis A Virus Infection – A Case Report. *BJID* 2009; 13(1):74-76
6. Department of Health and Human Services, Centers for Disease Control and Prevention. The ABCs of Hepatitis. [Online]. [2009] [cited 2009 July 07]. Available from: URL: <http://www.cdc.gov/hepatitis>
7. Department of Health and Human Services, Centers for Disease Control and Prevention. The ABCs of Hepatitis. [Online]. [2009] [cited 2009 July 07]. Available from: URL: <http://www.cdc.gov/hepatitis/HAV/ProfResourcesA.htm#section1>
8. Wasley A, Grytdal S, Gallagher K. Surveillance for Acute Viral Hepatitis --- United States, 2006. *MMWR Surveill Summ.* 2008; 57(2):1-24
9. Victor JC, Monto AS, Surdina TY, Suleimenova SZ, Vaughan G, Nainan OV, Favorov MO, Margolis HS, Bell BP. Hepatitis A Vaccine versus Immune Globulin for Postexposure Prophylaxis.

MEASLES OUTBREAK IN THE CITY OF JOHANNESBURG, GAUTENG PROVINCE, 23 AUGUST 2009 TO 1 NOVEMBER 2009—INTERIM REPORT

Mokete Phungwayo^{1,3}, Verushka Chetty^{1,3}, Modeste Landoh^{1,3}, Bernard Sawadogo^{1,3}, Amukelani Dlomu^{1,3}, Refilwe Mokgetle^{1,3}, Lungile Mbata^{1,3}, Brett N Archer^{2,3}, Bernice N Harris^{1,3}, Baski Desai⁴, Antonia Barnard⁴

¹South African Field Epidemiology and Laboratory Training Programme (SAFELTP), ²Outbreak Response Unit, National Institute for Communicable Diseases (NICD), ³School of Health Systems and Public Health, University of Pretoria. ⁴Public Health Unit, Communicable Diseases Directorate, City of Johannesburg, Department of Health.

Abstract

The current measles outbreak in the City of Johannesburg (CoJ) began in August 2009. The objectives of this investigation were to describe the outbreak in terms of time, place and person, and to recommend and implement appropriate control measures to contain the outbreak and prevent future outbreaks. From 23 August to 1 November 2009, 1 221 suspected cases have been investigated, of which 13 (1%) tested measles IgM negative and 261 (22%) positive. The remaining 947 (78%) cases are awaiting laboratory results or are in the process of being investigated. Twenty-four percent (294/1 208) of all cases (suspected and confirmed) have been reported in Region A. Fifty-six percent (674/1 208) of all cases were younger than 5 years of age.

1. Introduction

Measles is a communicable disease that is considered a major health problem worldwide with nearly 45 million cases and one million deaths each year.¹ The disease occurs mostly in children, but also affects susceptible adults.² The typical signs and symptoms of measles are fever and a non-blistering rash with or without cough, coryza and conjunctivitis.^{2,3,4} Although an effective vaccine is available, measles is still among the leading causes of vaccine-preventable deaths in children under five years of age, especially in developing countries where it has been implicated in the death of at least four percent of children in this age group.^{5,6,7}

South Africa has provided routine measles vaccinations at nine months of age since 1975. The presence of maternal antibodies at nine months of age may result in a poor immune response to the vaccine. In 1995 the Department of Health, therefore, added a second vaccine dose given at 18 months of age. Vaccine coverage in the CoJ in 2008-2009 was estimated to be 92%, with a dropout rate

of 12.5%⁹ (Figure 1). Vaccination coverage is estimated using data (immunisation doses) from local government, provincial health facilities, two non-governmental facilities and private providers.⁸

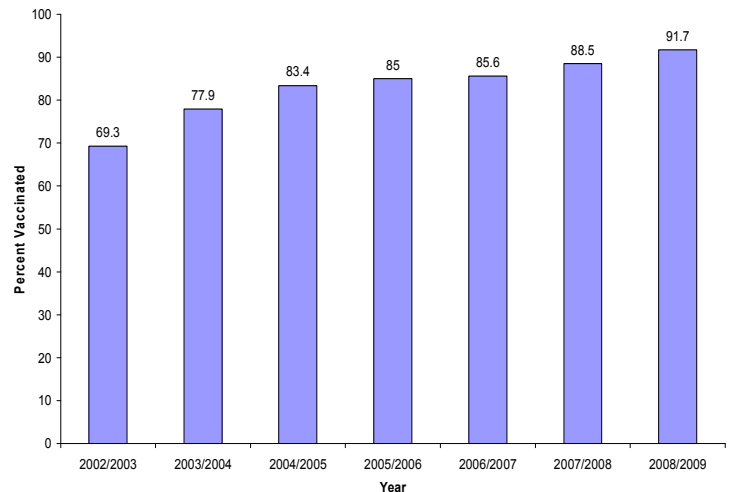


Figure 1: Measles vaccine coverage, children < 1yr, City of Johannesburg, 2002–2009⁸

Since 1995, seven Southern African countries have launched measles elimination initiatives as recommended by the World Health Organization (WHO) Regional Office for Africa.⁹ South Africa has implemented the following strategies, adopted from the WHO:

1. High immunization coverage: the national target is 90% coverage with two doses of measles vaccine at ages 9 and 18 months;
2. Interruption of chains of measles transmission with

(Continued on page 7)

3. periodic mass immunization campaigns;
4. Identification of low coverage and high risk areas with mop up immunisation.
5. Case-based measles surveillance supported by the laboratory for clinical and laboratory investigation for all suspected cases.¹⁰

South Africa introduced case-based measles surveillance in 1998 as part of this measles elimination strategy. Laboratory confirmation, by detection of IgM antibodies using ELISA was introduced due to lack of specificity of a clinical diagnosis of measles, based on the WHO clinical surveillance criteria (rash and fever with one of the “three C’s” - coryza, cough or conjunctivitis).⁹ However, in the setting of a measles outbreak the positive predictive value of the clinical case definition for suspected measles is high.

The CoJ is considered to be the economic hub of South Africa. It is situated in Gauteng Province and covers an area of 1 644km². With a population of 3 590 071 in 1 165 014 households, it is the most densely populated and urbanised municipality in South Africa.¹¹ The city reported a growth of 6.7% per year between 1996 and 2001. By far the largest population growth was amongst the poor.¹¹

The current measles outbreak started in March 2009 in the neighbouring Tshwane district. To contain the outbreak, the Tshwane district embarked on a mass measles immunisation campaign targeting all public and private primary and secondary schools from 24 August 2009 to 4 September 2009. The outbreak spread to the other districts in Gauteng. On 1 September 2009, an increase in the number of measles cases was noted in the CoJ. The South African Field Epidemiology and Laboratory Training Programme (SAFELTP) was requested to assist in the outbreak response.

2. Methods

The SAFELTP residents, as part of the CoJ outbreak investigation teams, were assigned to assist CoJ in meeting the following objectives:

- To capture and analyze data on all measles cases reported in the City of Johannesburg;
- To describe the outbreak in terms of time, place and person;
- To recommend appropriate control measures; and
- To make recommendations aimed at preventing future outbreaks.

A suspected case of measles was defined as an individual with onset of fever (temperature $\geq 38^{\circ}\text{C}$), rash and one or more of the following: cough, coryza or conjunctivitis within the CoJ, from 25 August to 1 November 2009. All suspect cases are required by law to be reported to the Communicable Disease Control Directorate, and were interviewed utilising a standardised case investigation form.

The collection of blood and urine samples from all suspected cases for laboratory confirmation is ongoing. Laboratory testing included the detection of anti-measles IgM antibodies by ELISA test. A confirmed case was defined as any individuals meeting the suspect case definition in whom anti-measles IgM was detected. The majority of laboratory testing was conducted by the NICD; however, confirmations made by private sector laboratories were also included.

A database of all suspected and confirmed measles cases was created and maintained, which included the capturing of newly detected cases as well as cases notified for the period. In addition, the NICD database of all laboratory results and confirmed cases were cross-referenced to

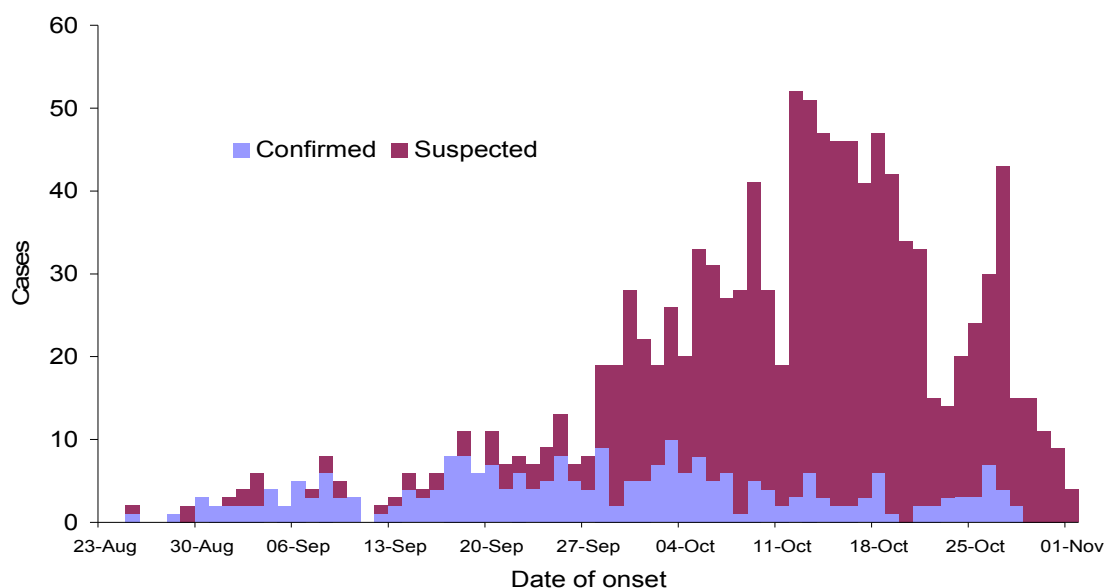


Figure 2: Frequency of suspected and confirmed measles cases by date of symptom onset*, City of Johannesburg, 23 August to 1 November 2009 (n=1 169, 52 with unknown date and 4 sporadic cases prior to 23 August have been excluded)

(Continued on page 8)

obtain missing data. Data were cleaned and original questionnaires were checked in the event of discrepancies. Data analysis was conducted utilising Microsoft® Excel 2003, and EpiInfo 2000 (version 3.51). Incidence rates (IR) were calculated as the number of new cases reported between 25 August and 1 November 2009 per 100 000 persons within the same population group. 2009 mid-year population estimates were acquired from the District Health Information System (DHIS, 2006).¹²

3. Results

Prior to 25 August 2009, increased numbers of cases of suspected and confirmed measles were recorded within CoJ. From 4 September 2009 the frequency of new cases rose steeply (Figure 2).

As of 1 November 2009 a total of 1 221 suspected measles cases have been reported within CoJ. Of these, 13 tested negative and were excluded from further analysis. Of the remaining 1 208 cases, 261 (22%) have been laboratory confirmed (IR=7 per 100 000 persons). Cases have been reported from all seven regions of the CoJ. The highest proportion of measles cases (suspected and confirmed) were observed in Region A (294/1 202, 24%, IR=88 per 100 000 persons) and Region D (267, 22%, IR=21 per 100 000 persons). However, Region F has the second highest incidence (52 per 100 000 persons, 22% of cases) (Table 1, Figure 3).

Table 1: Distribution of suspected and confirmed measles cases by administrative region, City of Johannesburg, 23 August to 1 November 2009

Region	Population*	Suspected ^A		Confirmed ^B		Total Cases ^{A+B}		
		N	%	N	%	N	%	IR
A	332 288	240	25	54	21	294	24	88
B	259 776	40	4	26	10	66	5	25
C	250 246	34	4	26	10	60	5	24
D	1 266 199	211	22	56	21	267	22	21
E	448 683	107	11	29	11	136	11	30
F	476 079	203	21	43	16	246	20	52
G	556 800	109	12	24	9	133	11	24
Unknown	-	3	0	3	1	6	0	-
Total	3 590 071	947	100	261	100	1 208	100	34

N = Number of Cases; % = Percentage of column total; IR = Incidence Rate per 100 000 population
 *2009 Midyear Population Estimates (DHIS 1.4, 2006)

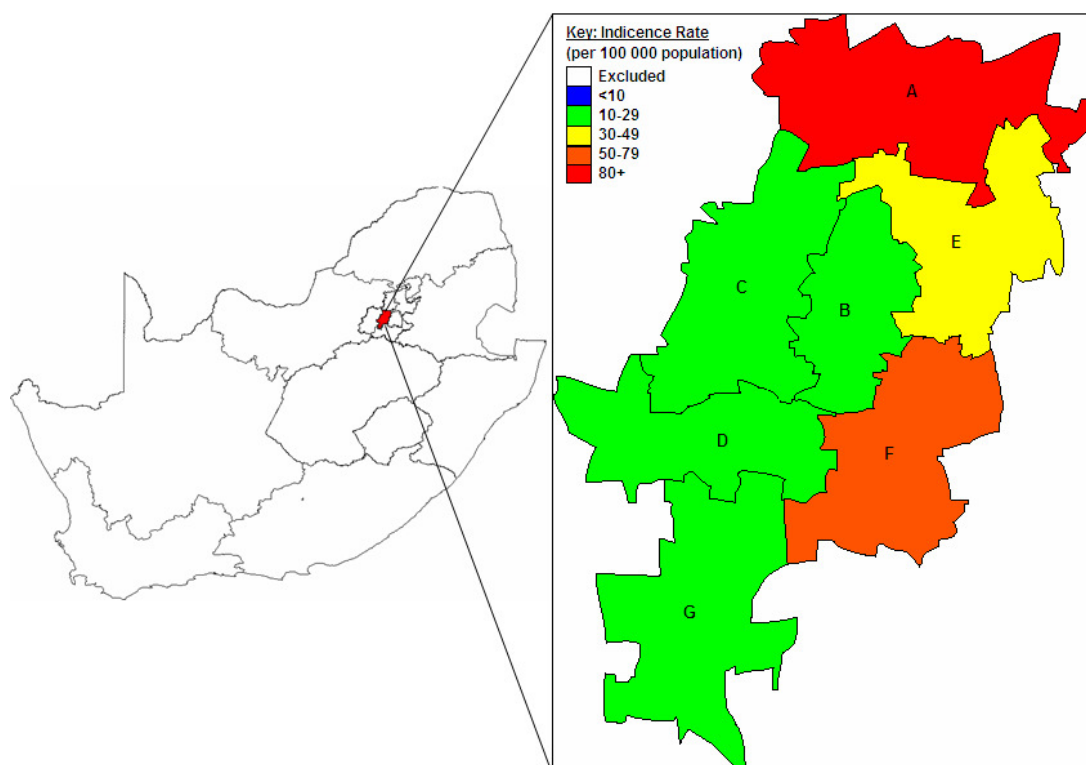


Figure 3: Maps showing the geographical location of City of Johannesburg and the incidence of suspected and confirmed measles cases by administrative region within the district, 23 August to 1 November 2009

(Continued on page 9)

Forty-eight percent (576/1 208) of all cases (suspected and confirmed) were female (suspected 442/947, 47%; confirmed 134/261, 51%). Children under five years of age are the most affected, accounting for 56% (674/1 194, IR=187 per 100 000) of all cases (Figure 4). More specifically, the highest incidence of infection was noted among children aged 6 to 11 months (n=254, IR=663 per 100 000).

CoJ response to reported cases

When CoJ is informed of a suspected case, the sub-district Expanded Programme on Immunisation (EPI)/Communicable Disease Control (CDC) coordinator traces

the case, immunises all their contacts and looks for more cases in that area. A mass measles vaccination campaign was conducted from 12 October to 6 November targeting everyone aged 9 months to 20 years of age. Parents, caregivers and training institutions were asked to ensure that all children receive their additional dose of measles vaccine. Health workers visited clinics, schools and crèches throughout the city for immunisation. Consent letters were sent to all schools in the province and parents were urged to sign consent for administration of an additional dose of the measles vaccine.

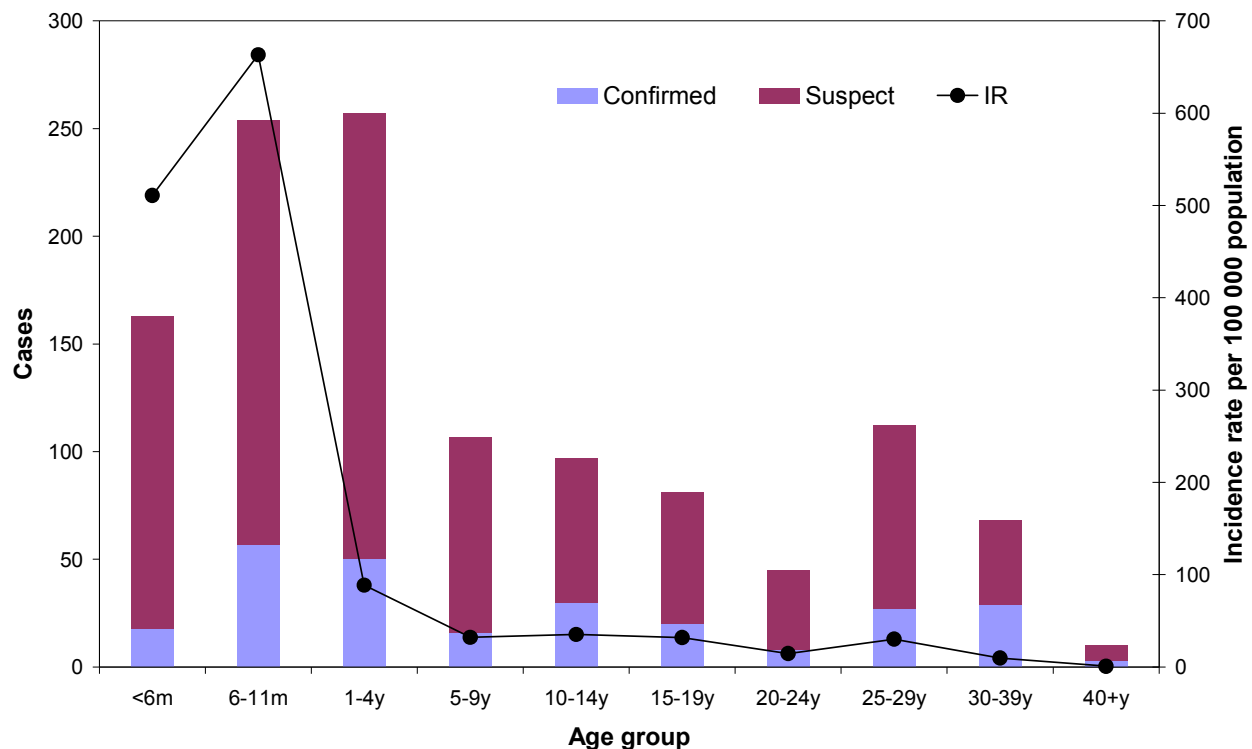


Figure 4: Incidence of suspect and confirmed measles cases by age group, City of Johannesburg, 23 August to 1 November 2009 (n=1 208, of which 14 with unknown age)

4. Discussion and conclusions

A total of 947 suspect and 261 confirmed measles cases have been reported as of 1 November 2009. The epidemic curve presented here suggests a propagated outbreak, with transmission occurring from person to person. Most of the cases were under 5 years of age, which is consistent with typical age groups commonly affected by the disease and findings reported elsewhere.¹³ However the fact that almost half the cases were in persons >5 years indicates that an immunity gap exists in this age group. Factors that could have contributed to the spread of disease in children aged <5 years in CoJ include high population density, nosocomial transmission and outbreaks in children's institutions (such as crèches, hostels or schools).¹³

It should be noted that these findings are subject to limitations due to ongoing investigations, which could have

led to bias in some results. Analysis of vaccination status was not possible due to inconsistent data recording on the case investigation form. A proportion of investigated cases had reported incorrect addresses and could not be traced, decreasing the impact of contact tracing activities.

Based on the epidemiology presented here, it is recommended that CoJ mount an awareness campaign about the value of being fully immunised. Vaccination campaigns should be targeted at the sub-populations and age groups identified as most affected. Surveillance and data management systems within the district must be strengthened; this includes further staff development on systems and case investigations. Finally, clinicians should be encouraged to suspect measles in cases meeting the case definitions, collect appropriate and timely specimens for laboratory testing, and notify and investigate all suspected cases.

(Continued on page 10)

Acknowledgments

We would like to acknowledge the following for their support, advice, and the opportunity to participate in the outbreak investigations and response: The National Institute for Communicable Diseases (NICD), SAFELTP and the City of Johannesburg Department of Health.

References

- World Health Organization (WHO), 1997. Progress towards global control and elimination, 1990–1996. *Wkly. Epidemiol. Rec.*, 72: 349.
- Smith FR, Curran AS, Raciti KA, Black FL. Reported measles in persons immunologically primed by prior vaccination. *J Pediatr* 1982; 101:391–3.
- Heyman DL, editor. *Control of Communicable Diseases Manual*. 19th ed. Washington: American Public Health Association; 2008.
- Griffin DE. Measles virus. In: Knipe DM, et al. *Fields Virology*. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 1401–1441.
- Rima BK, Duprex WP. Morbilliviruses and human disease. *J Pathol* 2006; 208:199–214.
- Bryce J, Boschi-Pinto C, Shibuya K, Black RE. WHO estimates of the causes of death in children. *Lancet* 2005; 365:1147–1152.
- Griffin DE, Bellini WJ. Measles virus. In: Fields BN et al. *Fields Virology*. 3rd ed. Philadelphia: Lippincott-Raven Publishers; 1996. p. 1267-1312.
- City of Johannesburg. Analytical Report On Annual Routine Data (2008/09) (Unpublished data). (8)
- Statistics South Africa. *Community Survey, 2007*.
- Centers for Disease Control and Prevention, Atlanta, USA. Progress toward measles elimination—Southern Africa, 1996–1998. *MMWR* 1999;48: 585–89.
- City of Johannesburg. *Annual Performance Report, 2007/2008*.
- HealthLink. Appendices [homepage on the internet] Place of publication: Publisher's name; [updated yr month day; cited yr month day]. Available from: www.healthlink.org.za/uploads/files/dhb0708_appendices.pdf
- McMorrow M L, Goitom G, van den Heever J, Kezaala R, Harris B N, Nandy R, et al. Measles outbreak in South Africa, 2003 – 2005. *SAMJ* 2009; 99 (5).

MEASLES OUTBREAK, TSHWANE, SOUTH AFRICA, 2009

Malotle M Molebogeng^{1,6}, Dorothy Nteo^{1,6}, Bernice Harris^{1,6}, Cheryl Cohen², Jo McAnerney², Mirriam Mashele³, Londiwe Mahlaba³, Sheilagh Smit⁴, Mpho Moshime⁵, Rina van der Gryp³

¹South African Field Epidemiology and Laboratory Training Programme, ²Epidemiology & Surveillance Unit, ³Viral Diagnostic Laboratory and ⁴Molecular Diagnostics Unit, National Institute for Communicable Diseases, ⁵National Department of Health, ⁶School of Health Systems and Public Health, University of Pretoria

Introduction

Measles is a highly contagious, vaccine preventable, viral disease caused by paramyxovirus of the genus Morbillivirus. Molecular characterization of wild-type viruses has identified eight clades (A – H), which have been divided into 23 identified genotypes. Endemic genotypes D2 and D4 and imported genotypes B2, B3 and D8 have been described in South Africa.¹ The incubation period ranges from 7-21 days. Symptoms include cough, fever, runny nose and red eyes. A few days after the onset of initial symptoms, rash develops on the face and spread downwards to the rest of the body. Transmission occurs through droplets from the mouth, throat, nose and eyes of the infected person or through aerosols. Ninety percent of people without immunity, who come into contact with an infected person, are infected.²

South Africa has undertaken to eliminate measles since 1996, and therefore, blood and urine specimens should be taken from every patient suspected of measles infection. Serological tests performed on serum confirms the presence of measles specific IgM and molecular characterisation for virus isolation is performed on urine to determine whether cases are due to endemic circulation or imported virus. Patients meeting the suspected measles case definition may be infected with rubella and serological testing also confirms presence of rubella antibodies.

In South Africa the last major outbreak of measles occurred in 2003-2005 following introduction of the virus from Mozambique. The outbreak started in Gauteng and Mpumalanga before spreading to other provinces, resulting in 1 676 laboratory-confirmed cases and 7 measles associated deaths nationally.³

In 2009, ten measles cases were reported between 13 and 31 March from City of Tshwane Metro, Gauteng province. No cases were reported thereafter until 25 June 2009,

when three cases were reported. The South African Field Epidemiology Training Programme residents were requested to assist in the outbreak investigation. Investigations were conducted from 7-14 April and 2-23 July 2009. All eleven cases from the first cluster and the first 17 cases from the second cluster were investigated. The outbreak is still on going and as of 22 October, a total of 1 135 cases and four deaths were confirmed throughout South Africa.^{4,5}

We report on the investigation of the first 28 cases. The objectives of the investigation were to: characterise cases by age, sex and place of origin; establish the epidemiological links between the cases; determine the vaccination status, the severity of disease and the genotype of the circulating strain.

Methodology

Case definitions

The following case definitions for measles were used in this investigation:

A suspected case was defined as a person presenting with fever ($\geq 38^{\circ}\text{C}$) and rash and one or more of the following: cough, coryza (runny nose) or conjunctivitis (red eyes);

A probable case was a suspected case epidemiologically linked to a confirmed case and did not have specimens taken for measles diagnosis; and

A confirmed case was a suspected/probable case that is laboratory-confirmed.

Data collection

A line list of confirmed and probable measles cases was compiled using the laboratory surveillance database and the suspected measles case based surveillance case investigation forms. A structured questionnaire was administered telephonically to all confirmed cases. Variables included were: demographic information,

(Continued on page 11)

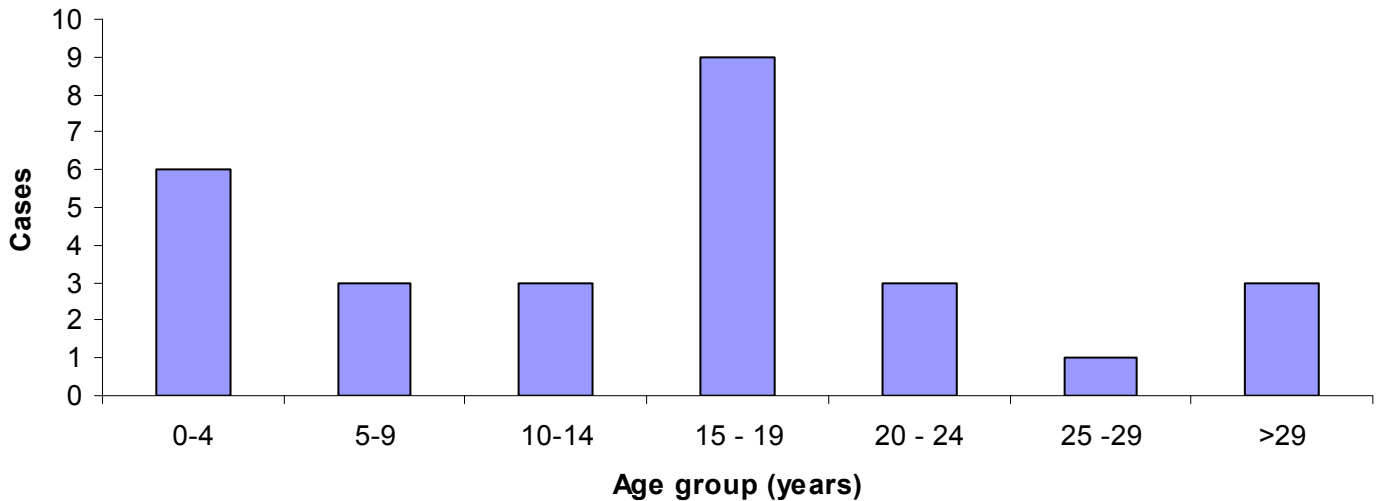


Figure 1: Age distribution of measles cases, City of Tshwane Metro , March-July 2009 (n = 28)

vaccination status, hospital admission, clinical and risk factor information and laboratory results. Data was captured on *Microsoft Excel* and analysed with *EpiInfo*.

Laboratory testing

Enzyme linked immunosorbent assay (ELISA) was used to detect measles specific IgM from serum and reverse transcriptase polymerase chain reaction (RT-PCR) was used to amplify the ribonucleic acid (RNA) of the virus from urine specimens, and the genotype determined through sequencing and phylogenetic analysis.

Results

Demographic characteristics

All 28 cases reported between 13 March and 14 July 2009, were interviewed. Of the total, 20 (71%) were male and

eight (29%) were female. Ages ranged from nine months to 34 years (Figure 1). Twenty seven cases resided in City of Tshwane Metropolitan area and one resided in City of Johannesburg Metropolitan area but acquired infection in Tshwane.

Epidemiological links between cases

Of the total probable and confirmed cases (n=28), eight attended one high school in Pretoria (including the case who resided in Johannesburg), 13 were from six household clusters and eight were not linked to other cases. Cluster 5 had a patient who was a contact of one of the school learners (Figure 2). The 3 probable cases occurred on 15, 23 and 31 March and all formed part of household clusters.

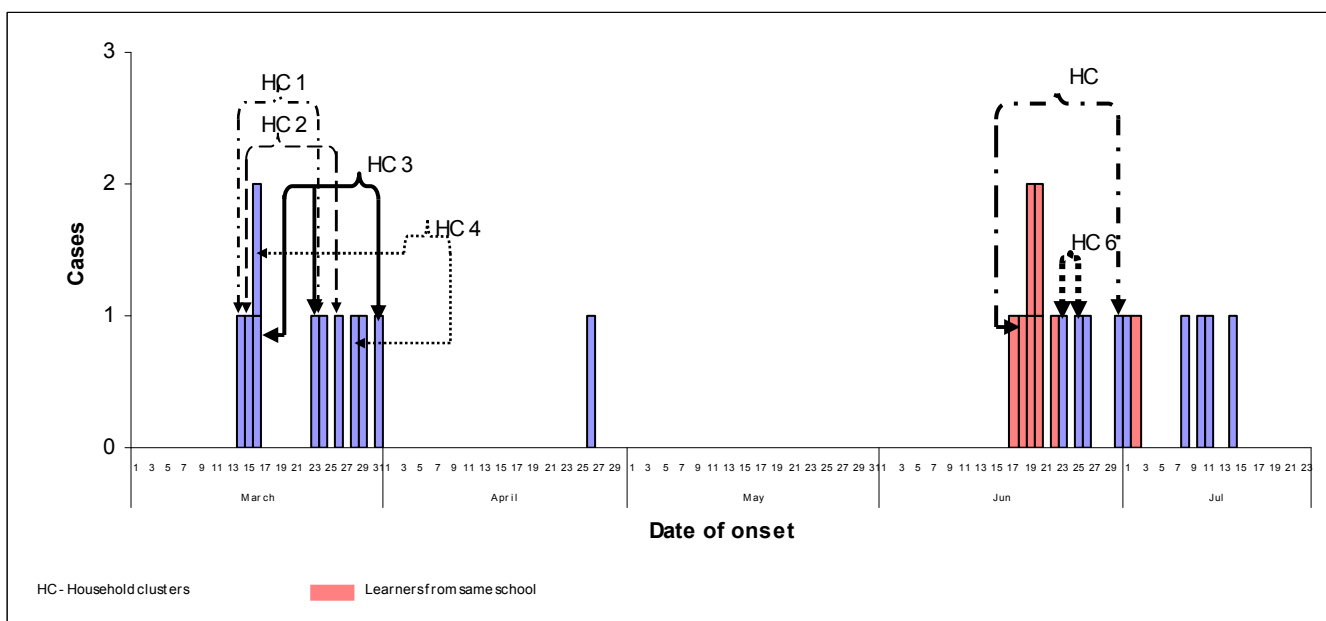


Figure 2: Epidemic curve of measles cases, City of Tshwane Metro, Gauteng, March-July 2009

(Continued on page 12)

Table 1: Vaccination status of measles cases , City of Tshwane Metro, March-July 2009* (n = 28)

Age group	Patients with RTHC						Patients without RTHC					
	Zero dose		1 dose		2 dose		Zero dose		1 dose		2 dose	
	n	%	n	%	n	%	n	%	n	%	n	%
< 9 months	0	0	0	0	0	0	1	4	0	0	0	0
9-18 months	0	0	1	4	0	0	2	7	0	0	0	0
> 18 months - 5 years	0	0	1	4	0	0	1	4	0	0	0	0
> 5 years	0	0	0	0	3	11	3	11	1	4	15	54
Total	0	0	2	8	3	11	7	26	1	4	15	54

Vaccination status

Of the total number of patients (n=28), vaccination status was confirmed in only five patients on a Road-to-Health-Chart (RTHC). Of the five, two were given one dose of the measles vaccine, (aged 10 months and 2 years) and three had received two doses. Sixteen (16/28) reported receiving vaccinations; however this was not confirmed on a RTHC. Of the 16, one reported receiving one dose, and four (5/16) were not sure about the number of doses they received, ten (10/15) reported receiving two doses. Two (2/28) did not know if they were vaccinated. Five (5/28) of the patients, of whom 4 are siblings (two siblings in each cluster), were not vaccinated (Table 1).

Severity of disease

All patients (n=28, 100%) met the suspected case definition before confirmation: 20 (71%) had runny nose, 24 (86%) were coughing and 27 (96%) had red eyes (table 2). All patients sought medical attention and 14 (50%) were admitted to hospital. Among the patients who were hospitalised some of the diagnoses made were: ear infection, pneumonia, suspected meningitis, diarrhoea, tracheal infection, urinary tract infection and tonsillitis. One patient was admitted to the intensive care unit, and in addition to measles the patient was diagnosed with severe bladder infection.

Table 2: Symptoms experienced by confirmed measles cases, City of Tshwane Metro, March-July 2009 (n = 28)

Signs and symptoms	Frequency	%
Fever	28	100
Rash	28	100
Cough	24	86
runny nose	20	71
Red eyes	27	96

Laboratory results and molecular epidemiology

Of the 28 patients, 24 tested positive for measles IgM antibodies; one of the 24 also tested positive for rubella IgM antibodies and three of the patients did not have specimens submitted to the laboratory. Molecular characterisation was possible for 14 patients and this revealed genotype B3 from 13 and D8 from one patient. Of the 13 patients from whom B3 genotype was detected, one was from the first cluster and 12 were from the second cluster. The D8 genotype was isolated from a 34 year old female with a travel history from India. The patient travelled

to India on 2 April 2009 and returned to South Africa on 14 April 2009. She developed signs and symptoms on 22 April 2009.

Spatial distribution of cases

Cases were widely distributed geographically in the City of Tshwane. (Figure 3)

Limitations

It was not possible to confirm vaccination status of the majority of patients as they could not produce their Road-to-Health Charts. Recall bias was observed as many patients could not remember all the places they visited before the onset of illness. Genotyping of the virus was only possible in some of the patients due to the following reasons: lack of specimens (both serum and urine) and delays in forwarding urine specimens to the National Institute for Communicable Diseases.

Intervention measures instituted

The cases in the two clusters were widespread geographically and affected a wide age range. This made it difficult to identify a target group to vaccinate. Therefore a number of interventions were instituted.

The following interventions were instituted in the first cluster:

- Guidelines were circulated in hospitals that all children from 6 months to 15 years in Tshwane district must be immunised upon admission with immediate effect, until the outbreak is over.
- Active case finding was conducted through retrospective record review in health facilities where confirmed measles patients consulted or were hospitalised
- Prevention of nosocomial infections by strengthening infection control practices through:
 - ⇒ isolation of patients presenting with fever and rash in the health facilities
 - ⇒ emphasis on adherence to basic standard infection precautionary measures in place
 - ⇒ follow guidelines in place regarding transmission based precautionary measures
- Strengthen awareness/communication in public and private health facilities:
 - ⇒ circulars on case management, prevention

(Continued on page 13)

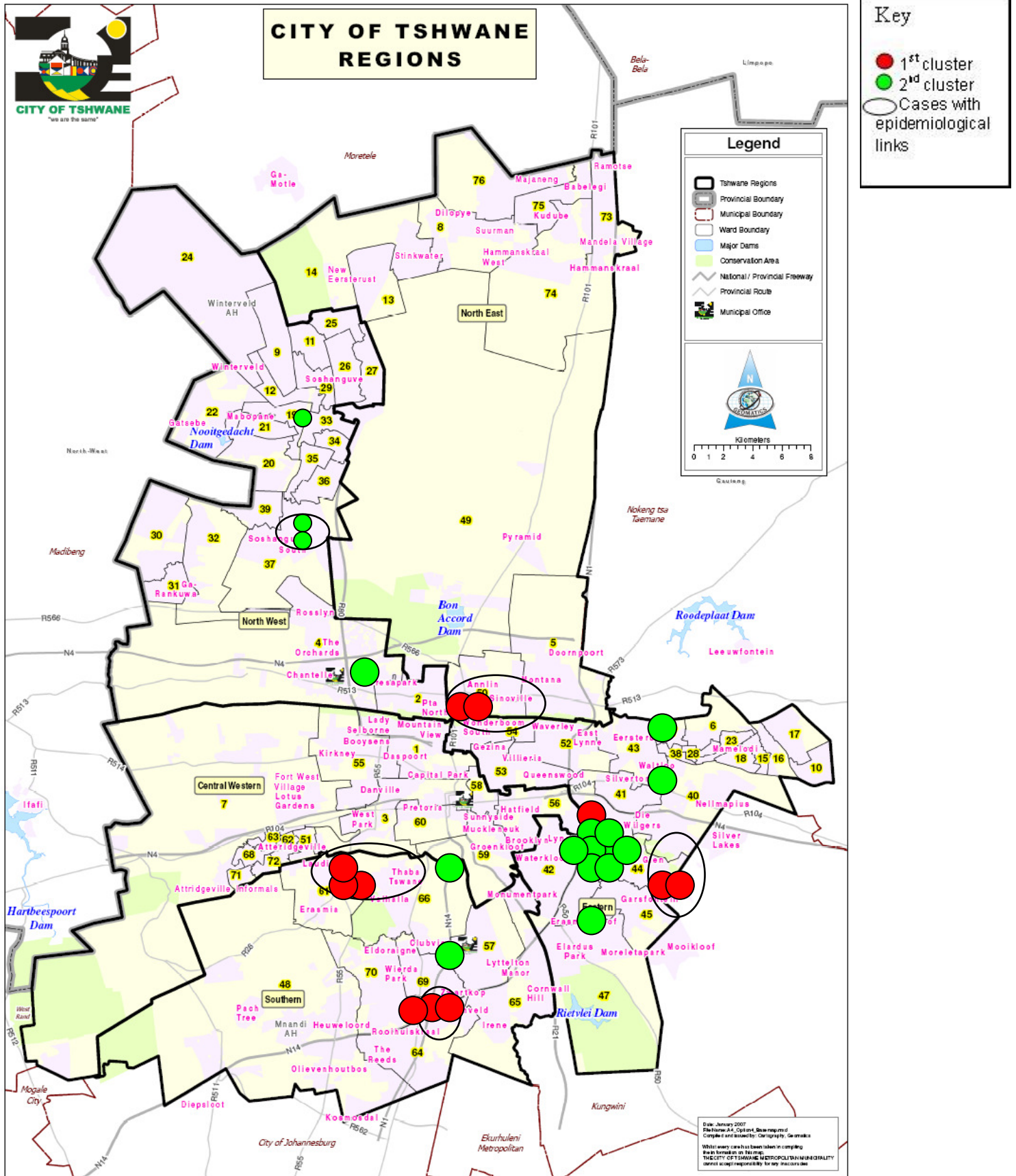


Figure 3: Spatial distribution of measles cases, City of Tshwane, March-July 2009

(Continued on page 14)

- and control of measles cases were issued to health facilities
- ⇒ alert private and public laboratories to be on the look out for specimens with query measles diagnosis and send them to NICD for testing
- Community awareness through workshops and media communication:
 - ⇒ provided Information, Education and Communication (IEC) materials to the public
 - ⇒ health talks and information workshops on measles were conducted at the clinics, hospitals, crèches and the community.
 - ⇒ information on measles to be cascaded to the community through community radio stations and local newspapers (e.g. Record)
 - ⇒ loudhailing in the community.

For the second cluster the following intervention measures were instituted:

- A measles campaign in Tshwane was conducted targeting primary and secondary schools from both public and private sectors. The campaign was conducted for two weeks in August and two weeks and five days in September. Only two private schools in Central sub-district refused to be included in the campaign.
- In-service training of 400 heads of schools in Tshwane was conducted on measles before the campaign.
- Information session on measles was also conducted in 109 clinics, 29 hospitals and for 600 military residents at the National Department of Air Force.
- All health care workers in the clinics and public hospitals were advised to give measles vaccine to medical personnel, nursing staff, emergency medical service staff and other personnel who are in contact with patients.
- Children from 6 months to 15 years were vaccinated against measles on admission in the hospitals.
- Parents of children who missed the opportunity to be vaccinated during the school campaign were advised to take the children to the nearest clinics for measles vaccination.
- Contacts of all reported suspected and confirmed cases were traced and vaccinated.

Discussion and conclusion

The City of Tshwane Metro experienced two clusters of measles cases with more than a two month interval between the two clusters. It is likely that these cases were related as the same genotype was detected in both clusters. The fact that we were unable to identify epidemiological links between the two clusters suggests that some cases in the chain of transmission may have been missed by surveillance or were not reported. The incubation period ranges from 7 to 21 days therefore the gap of 2 months between the clusters means there must

have been at least two generations of cases missed. The only epidemiological links identified were among the cases from learners at high schools in Pretoria and amongst the household clusters.

Genotype B3 was identified from both clusters; however, in the first cluster, one imported case due to D8 genotype was also identified. Both genotypes have not previously circulated in South Africa, and are thus imported.¹ Genotype B3 circulates endemically in West and Central Africa, while D8 was first described in the United Kingdom and is currently circulating on the Indian sub-continent.¹ Epidemiologic information (travel history from India and incubation period) from the patient infected with genotype D8 supports the hypothesis that this patient acquired infection outside of South Africa and was a sporadic case unrelated to the main outbreak.

The isolation of imported strains is an indication that South Africa may indeed have interrupted indigenous transmission of measles. However, the current widespread and ongoing measles outbreak demonstrates that even in a setting of low reported case numbers, populations remain vulnerable to measles importation. To prevent outbreaks it is important to maintain high population immunity even in the absence of a circulating virus.³ The relatively large proportion of adult patients may be related to the accumulation of susceptible individuals over several years. The World Health Organization and UNICEF recommends that vaccine coverage be maintained at 95% for measles elimination and in 2008 vaccine coverage in South Africa was reported to be 85%.⁶ Efforts to strengthen routine vaccination should be emphasized.

References

1. WHO WER. Global distribution of measles and rubella genotypes—update 2006; 81: 474-9.
2. WHO, UNICEF . Joint statement. Global plan for reducing measles mortality 2006-2010. World Health Organization [Accessed on 11 Sept 2009]. Available from: <http://www.doh.gov.za/facts>.
3. McMorrow ML, Gebremedhin G, Van den Heever J, Kezaala R, Harris BN, Nandy R, et al. Measles outbreak in South Africa. JAMA. 2009 May; 99 (5): 314 – 9.
4. Measles outbreak, National Institute for Communicable Diseases [Accessed 20 October 2009] Available from http://www.nicd.ac.za/pubs/communique/2009/NICDCommApr09Vol08_04.pdf
5. Measles outbreak kills 4 News24 [Accessed 20 October 2009]. Available from http://www.news24.com/Content/SouthAfrica/News/1059/dec871912a114abd9084c1e5a33e55ca/16-10-2009-12-42/Measles_outbreak_kills_4
6. WHO-UNICEF Joint Statement on Strategies to Reduce Measles Mortality World Wide [data base on the internet] UNICEF Publications [homepage] [Accesses at 16 Oct 2009] Available at http://www.unicef.org/publications/index_4444.html

MEASLES OUTBREAK, SOUTH AFRICA, 2009, PRELIMINARY DATA ON LABORATORY-CONFIRMED CASES

Jo McAnerney¹, Cheryl Cohen¹, Adrian Puren², Sheilagh Smit², Mirriam Mashele³, Johann van den Heever⁴

¹Epidemiology & Surveillance Unit, ²Molecular Diagnostics Unit, ³Viral Diagnostics Laboratory, National Institute for Communicable Diseases, ⁴Expanded Programme on Immunization, National Department of Health

Measles is caused by a virus of the genus *Morbillivirus* in the Paramyxoviridae family. Measles virus is transmitted by infected droplets during coughing, sneezing, through direct contact with nasal or throat secretions of infected persons or by touching contaminated objects. Measles is predominantly a childhood disease and is highly infectious and spreads rapidly amongst people who are not immune, leading to significant morbidity and mortality as a result of prolonged induced immune suppression.¹

Because of the demonstrated lack of specificity of a clinical diagnosis of measles, case based measles surveillance in South Africa was started in 1998, based on the WHO clinical surveillance criteria (rash and pyrexia and one of the three C's - coryza, cough or conjunctivitis). All cases meeting this case definition should have specimens submitted to the laboratory. Between 1998 and 2002 the annual number of specimens in South Africa ranged from 529 to 1008, and the number of positive measles IgM results from 6 to 39. From 2003 through 2005 widespread measles outbreaks were experienced with 1636 laboratory-confirmed cases of measles reported over this period. Outbreaks affected Gauteng, Mpumalanga, Western Cape, KwaZulu/Natal and Eastern Cape Provinces². Between 2006 and 2008 the annual number of patients with positive measles IgM results decreased again ranging from 30 to 82 confirmed cases each year.^{3,4}

Reported vaccination coverage in South Africa for the first dose of measles vaccine at 9 months of age has increased from 62% to 91% from 2004-2008 and coverage with the second dose has increased from 53% to 75% over the same period (Personal communication Dan Kibuuka, National Department of Health, South Africa). The recommended coverage for measles elimination is coverage of 95% of children for both doses.¹

During 2009 a sharp increase in confirmed measles cases was seen. A number of outbreak response activities were conducted including a measles vaccination campaign in Tshwane during a two week period starting in the last week of August (week 35).⁵

We aim to present the descriptive epidemiology of laboratory confirmed cases in this outbreak.

Methods

The National Institute for Communicable Diseases (NICD) of the National Health Laboratory Service (NHLS) is accredited by the World Health Organisation (WHO) to perform measles and rubella IgM testing for national case-based measles surveillance.

Blood and urine specimens from suspected measles cases nationally are submitted to NICD for measles and rubella

testing. Approximately 60% of suspected measles cases from Free State Province are tested at the NHLS Universitas Academic Laboratory at the University of the Free State (UFS). The numbers presented here represent specimens received by the NICD and UFS and do not include information on patients where no specimens were taken.

All blood specimens were tested by Enzygnost (Dade-Behring, Marburg, Germany) diagnostic kits for the presence of anti-measles and anti-rubella immunoglobulin M (IgM). Amplification of ribonucleic acid (RNA) for genotyping was attempted on all cases testing positive or equivocal for anti-measles IgM. For molecular analysis RNA was extracted directly from clinical specimens (urine or throat swab if available, otherwise serum) and tested for the presence of Measles Virus by reverse transcriptase polymerase chain reaction (RT-PCR). Selected specimens were chosen to be representative of different outbreak timing and location (data until beginning of October). Phylogenetic analysis of the terminal 450 nucleotides of the MV nucleoprotein gene was performed using the neighbour-joining algorithm of MEGA software.⁶

Results

During the first five months of 2009 there were 27 confirmed cases of measles (compared to 15 for the same period of 2008) (Table 1). Thirteen of these cases were from Gauteng, 11 from the Tshwane district. In June there were a further 12 confirmed cases in Tshwane, and an additional 23 cases in July. By the end of August (week 35-36) there were a total of 173 confirmed measles cases in South Africa, 136 (79.2%) of which were from Tshwane district. From September onward cases have been confirmed in all nine provinces with 654 cases in September, and 1587 in October, bringing the total number to 2414 (Table 1; Figure 1). Patients with confirmed measles have mainly been concentrated in the Northeast areas of South Africa as well as the metropolitan areas of Durban and Cape Town (Figure 2) with the highest incidence of cases reported from Gauteng province (Figure 3). Nationally, patient's ages ranged from 1 month to 77 years with a median of 8 years (Figure 4).

During the first three months of the outbreak, the median age of patients from Tshwane was 14 years (range 4 month to 38 years) with 51% (58/114) of patients in the 10-19 year age group. However, since September the median age of patients in Tshwane has decreased to 11 years (range 1 month to 77 years) with 35% (255/721) in the 10-19 year age group (Figure 5). At the same time the percentage of patients aged <1 year has risen from 13% (15/114) to 22% (160/710).

(Continued on page 16)

Since August the number of cases in other districts of Gauteng, notably Ekurhuleni and Johannesburg, have started to increase markedly with totals of 383 and 643 respectively. Smaller numbers of cases have also been detected in Metsweding, Sedibeng and the West Rand (Figure 6). The median ages for patients from Ekurhuleni and Johannesburg are 3 (range 2 months to 44 years) and 2 years (range 2 month to 64 years) respectively (Figure 7). Combining patients from all the districts in Gauteng, by far the highest incidence of disease was in children aged <

1 year (Figure 8a). The highest incidence country-wide was also in the <1 year age group. (Figure 8b).

Sequencing of PCR-positive specimens from Tshwane district, other districts in Gauteng and other provinces showed that all of these specimens had identical sequences. Subsequent phylogenetic analysis identified the cluster as genotype B3 and identical to some of the strains of Measles Virus circulating in West Africa in 2009 (Figure 9).

Table 1: Measles IgM positive results per province per month: South Africa 2009

Month	EC	FS	GA	KZ	LP	MP	NC	NW	WC	TOTAL
January	0	0	2	0	0	1	0	0	2	5
February	0	0	1	0	0	1	0	0	0	2
March	2	0	2	1	0	0	0	1	0	6
April	1	0	6	1	0	2	0	0	0	10
May	0	0	2	1	0	1	0	0	0	4
June	0	0	14	0	0	0	0	0	2	16
July	0	0	24	1	0	1	0	0	0	26
August	1	1	95	0	3	2	1	1	0	104
September	8	13	599	6	2	11	1	13	1	654
October	8	16	1356	63	25	20	5	80	14	1587
TOTAL	20	30	2104	73	30	39	7	92	19	2414

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

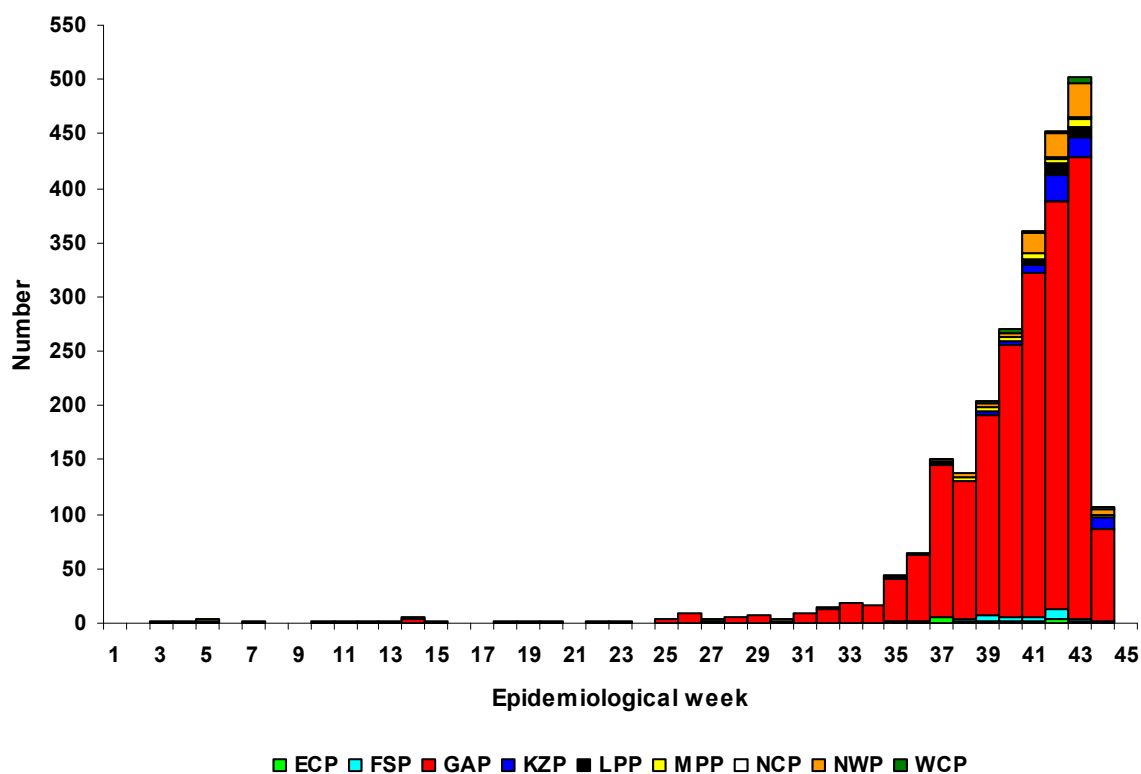


Figure 1: Number of patients with laboratory confirmed measles by week and province: South Africa, 2009

(Continued on page 17)

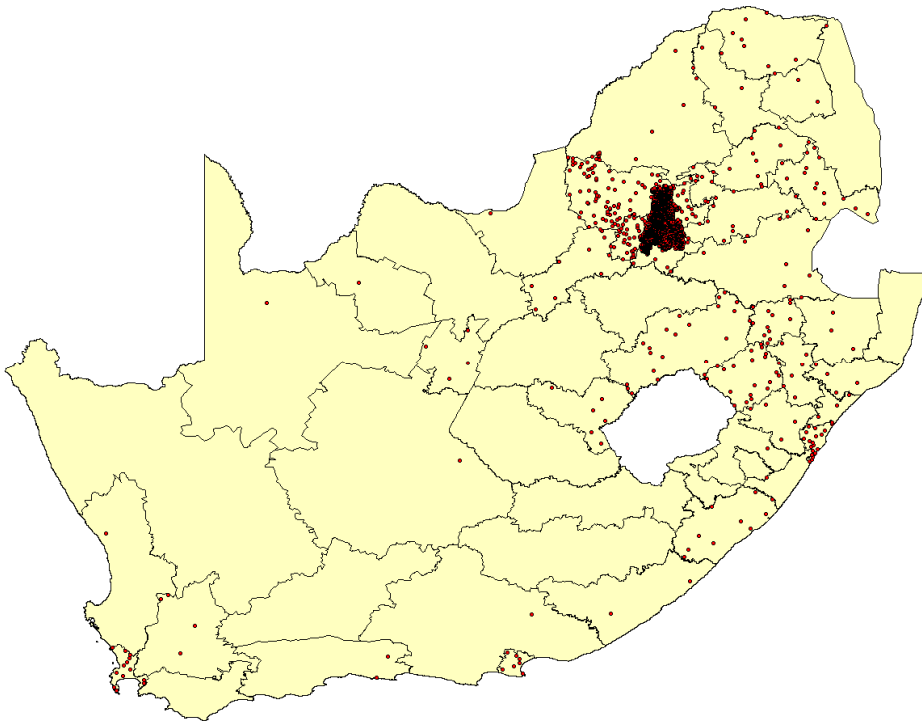
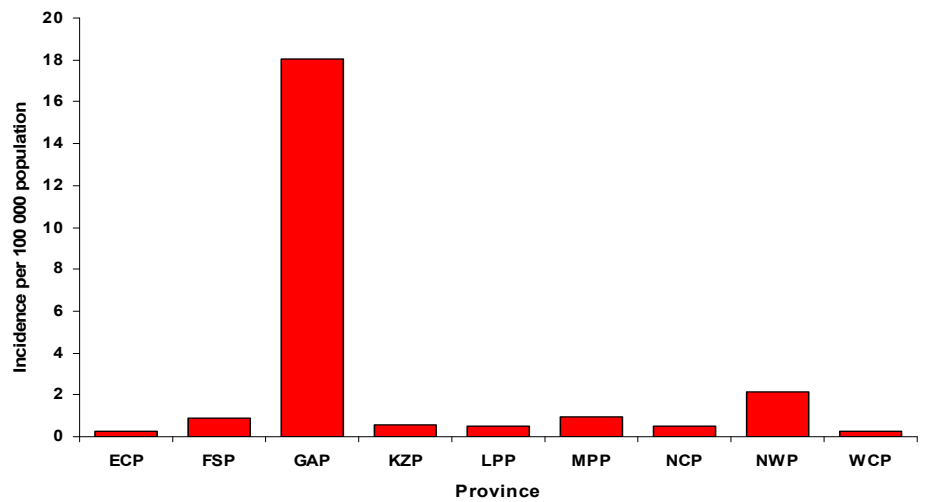


Figure 2: Map of patients with laboratory confirmed measles by district: South Africa, January to October 2009. Each red dot represents one case.

Figure 3: Incidence of laboratory confirmed measles by province, South Africa, January to October 2009



N=2372

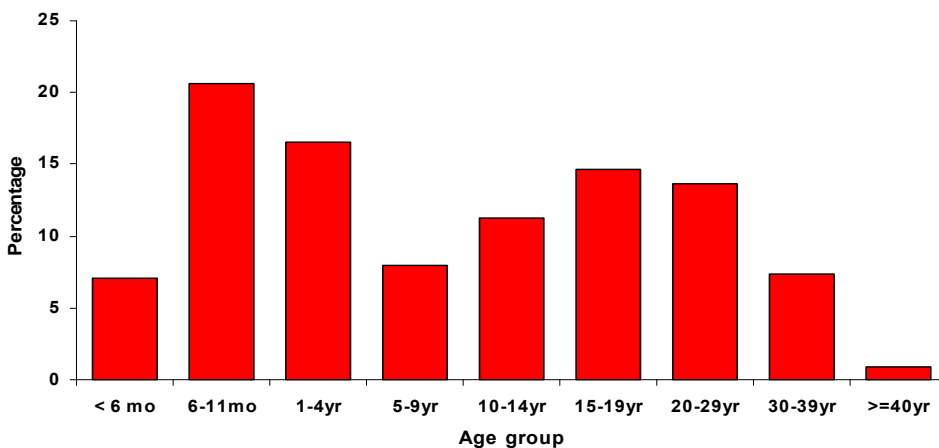


Figure 4: Age distribution of patients with laboratory-confirmed measles: South Africa, January to October

(Continued on page 18)

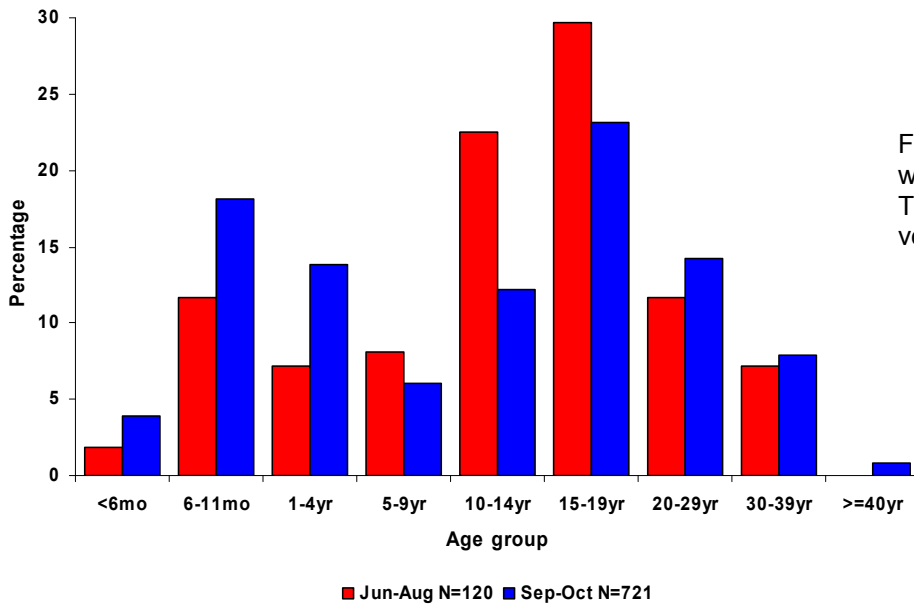


Figure 5: Age distribution of patients with laboratory-confirmed measles, Tshwane district: June to August versus September to October 2009

Figure 6: Patients with laboratory-confirmed measles by week and district, Gauteng South Africa 2009 (arrow indicates vaccination campaign in Tshwane in weeks 35 & 36)

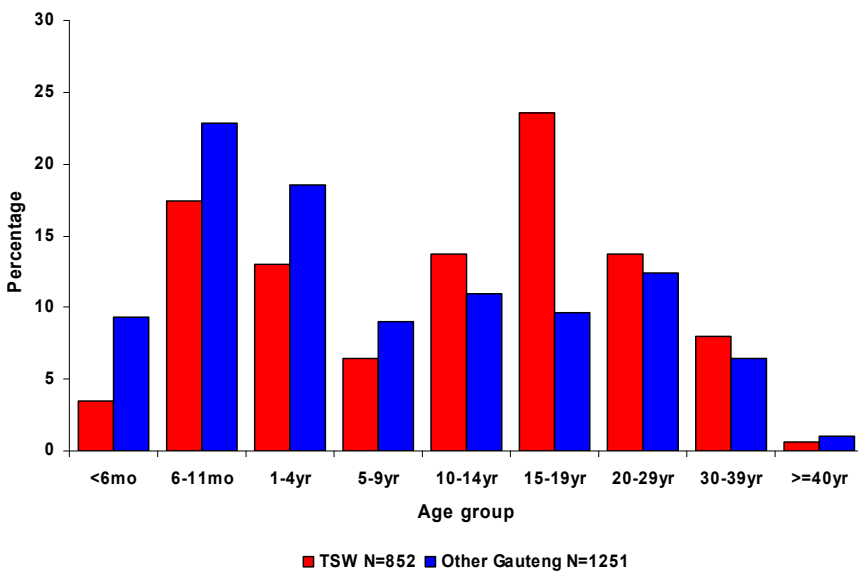
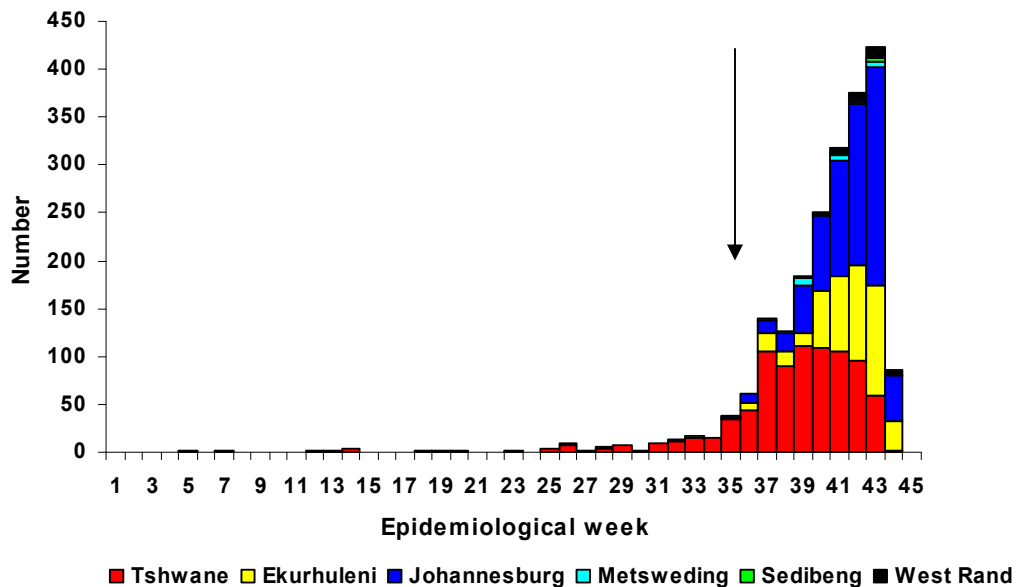


Figure 7: Age distribution of patients with laboratory confirmed measles from Gauteng: Tshwane district (TSW) versus other Gauteng districts, January to October 2009

(Continued on page 19)

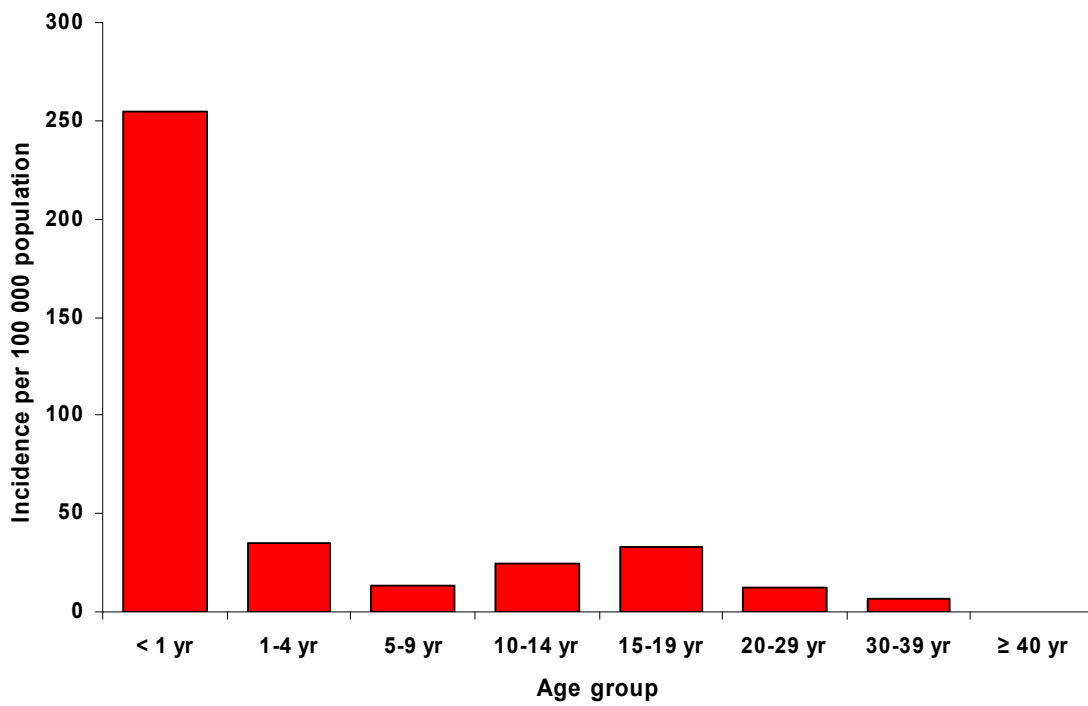


Figure 8a

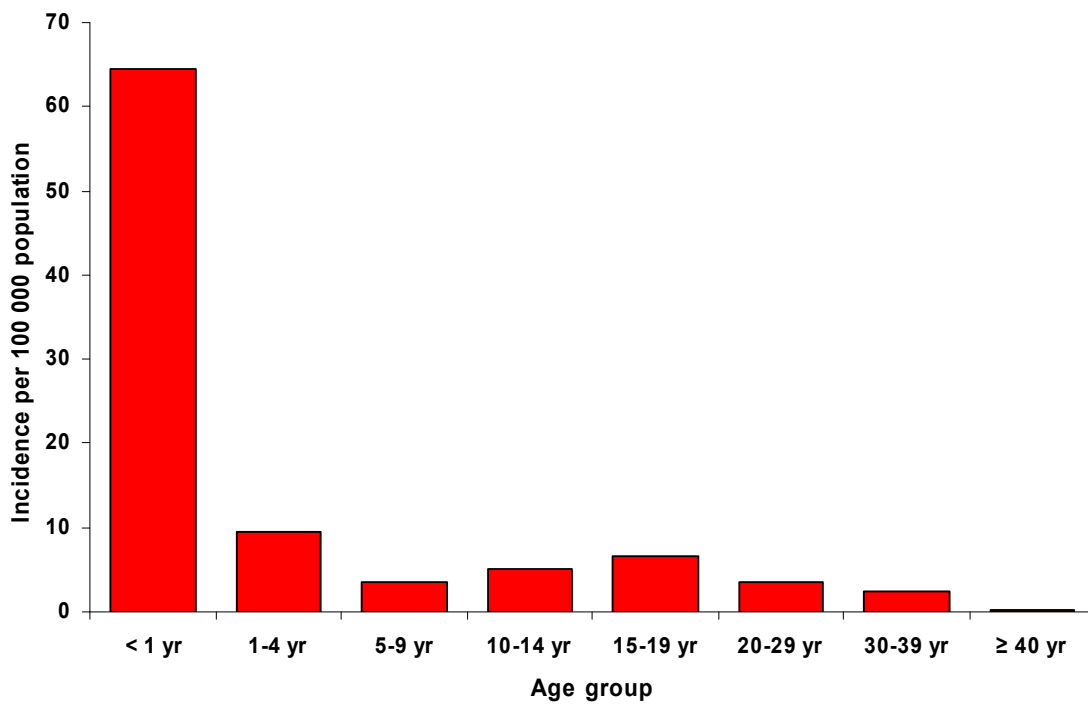


Figure 8b

Figure 8: Incidence of laboratory confirmed measles cases by age group, January to October 2009. a) Gauteng Province and b) South Africa

(Continued on page 20)

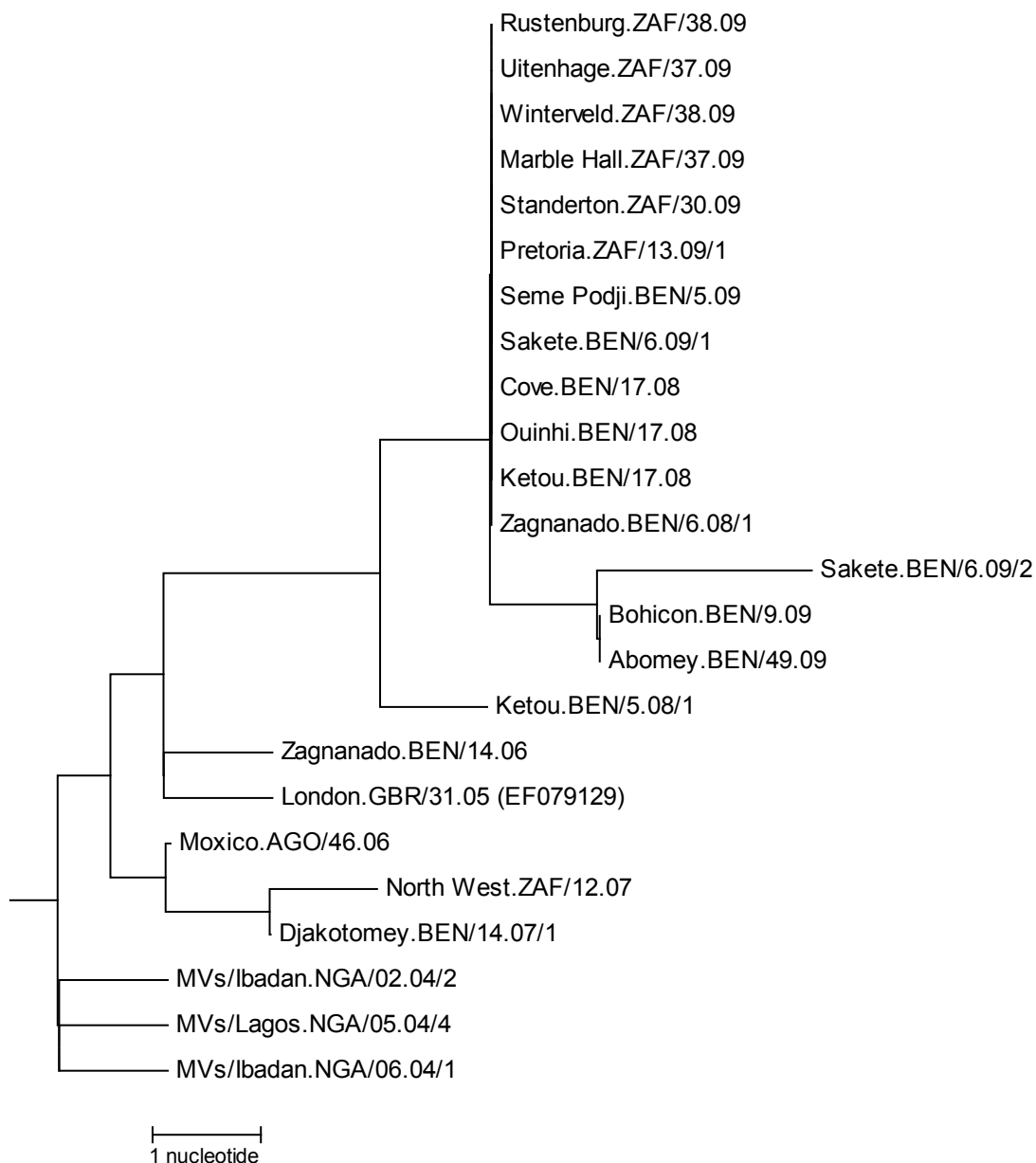


Figure 9: Phylogenetic analysis of the terminal 450 nucleotides of the MV nucleoprotein gene using the neighbour-joining algorithm of MEGA software. This figure depicts representative sequences during the course of the outbreak in South Africa, 2009, relative to closely related strains of genotype B3.

Limitations

This report is subject to several limitations. Data presented relate to laboratory confirmed cases and limited epidemiologic data was available on these cases as case investigation forms are seldom submitted to the laboratory. For this reason we were unable to comment on the clinical presentation or severity of cases. In addition we do not have information on measles deaths. Importantly we were also not able to present data on vaccination history. Due to the high burden of specimens currently submitted to the laboratory it is likely that the numbers of laboratory confirmed cases will continue to increase thus this report should be regarded as preliminary.

Discussion

To date Gauteng province has been the most severely affected by this outbreak but in recent weeks cases have been reported from all provinces. This suggests that there is the potential for large outbreaks to occur in these provinces. The most affected age group early on in the outbreak was persons aged 15 to 19 years, this then shifted to affect children < 5 years of age as the outbreak progressed. However to date (October 2009) approximately 20% of cases remain aged > 20 years. The early epidemic in older children and adolescents may have been a combination of an immunity gap in this group related to inadequate measles coverage with low level of circulating

(Continued on page 21)

measles more than a decade before. In addition, a large proportion of the early patient clusters occurred in high schools where conditions may have been favourable for measles transmission.⁷ The later shift to involve children aged < 1 year with a substantial proportion of patients aged < 6 months may be related to decreased levels of maternal antibodies in infants (particularly those born to HIV-infected mothers) as well as nosocomial measles transmission contributing to spread in young children.^{8,2} The molecular analysis is not able to distinguish whether the outbreak in South Africa represents a single introduction of the virus with subsequent infection of susceptible individuals, or whether there were several introduction events.

Several outbreak response vaccination activities have been conducted in Tshwane district and City of Joburg in Gauteng Province.^{7,5} Despite these activities the number of cases continues to increase. It does however appear that the rate of increase in case numbers declines following outbreak response vaccination activities in Tshwane. Outbreak response vaccination may fail to interrupt measles transmission especially if the coverage is not sufficiently high or if response activities are conducted late once the disease has spread widely.⁹ However in certain settings vaccination in response to outbreaks has been shown to reduce morbidity and spread of measles.^{10,11} Where conducted outbreak response vaccination is most effective if started early, covers a wide age range and achieves a high coverage.¹¹

Since the start of case-based surveillance, nationwide supplementary measles immunisation activities have been carried out in 2000, 2004 and 2007. The next national mass campaign is planned for April of 2010. However it seems that this campaign has come too late to avert a large outbreak. The fact that we are experiencing this outbreak just 2 years after the last mass campaign suggests that a substantial immunity gap may still exist. Despite the fact that there have been improvements in coverage of the first and second dose of measles, these still fall short of elimination goals.

This outbreak highlights the fact that South Africa remains vulnerable to large measles outbreaks. Maintaining high routine vaccination coverage should be emphasized as well as periodic timeous mass immunisation campaigns and mop up activities in high risk areas.¹¹ In the current widespread outbreak clinicians should focus on adequate clinical case management including provision of vitamin A and antibiotics where indicated as well as rapid notification and outbreak vaccination of close household and community contacts for suspected cases. More widespread outbreak response vaccination activities should be considered especially in districts where case numbers remain relatively low but coverage gaps exist.

References

1. Heyman DL. Control of Communicable Diseases Manual. 2004;18:111-113.
2. McMorrow ML, Gebremedhin G, van den Heever J, Kezaala R, Harris B N, Nandy R, Strebel P, Jack A, and Cairns, KL. Measles outbreak in South Africa, 2003-2005. *S.Afr.Med.J.* 2009;99:314-319.
3. McAnerney J, Cohen C, Smit S, Singh B, Masango M, Mashele M, Kekana E and Puren A. Suspected measles case-based surveillance, South Africa, 2007. *Communicable Diseases Surveillance Bulletin* 2008;6:2-4.
4. McAnerney J, Smit S, Cohen C, Singh B, Masango M, Hlaletsoa D and Buys A. Suspected measles case-based surveillance, South Africa, 2006. *Communicable Diseases Surveillance Bulletin* 2007;5:1-2.
5. Phungwayo M, Chetty V, Landoh M, Sawadogo B, Dionu A, Mokgetle R, Barnard A, Archer B, Harris B, Desai B and Mbata L. Measles outbreak in the City of Johannesburg, Gauteng Province, 23 August 2009 to 1 November 2009 - interim report. *Communicable Diseases Surveillance Bulletin* 2009;7:6-10.
6. Kumar S, Tamura K and Nei M. MEGA3: Integrated software for Molecular Evolutionary Genetics Analysis and sequence alignment. *Brief.Bioinform.* 2004;5:150-163.
7. Matlote M, Nteo D, Harris B, Cohen C, McAnerney J, Mashele M, Mahlaba L, Smit S, Moshime M and van der Gryp R. Measles outbreak, Tshwane, South Africa, 2009. *Communicable Diseases Surveillance Bulletin* 2009;7:10-14.
8. Scott S, Moss WJ, Cousens S, Beeler JA, Audet SA, Mugala N, Quinn TC, Griffin DE and Cutts FT. The influence of HIV-1 exposure and infection on levels of passively acquired antibodies to measles virus in Zambian infants. *Clin.Infect.Dis.* 2007;45:1417-1424.
9. Aylward RB, Clements J and Olive JM. The impact of immunization control activities on measles outbreaks in middle and low income countries. *Int.J.Epidemiol.* 1997;26:662-669.
10. Goodson JL, Wiesen E, Perry RT, Mach O, Kitambi M, Kibona M, Luman ET and Cairns KL. Impact of measles outbreak response vaccination campaign in Dar es Salaam, Tanzania. *Vaccine.* 2009;27:5870-5874.
11. The Department of Immunization, Vaccines and Biologicals and World Health Organisation. Response to Measles Outbreaks in Measles Mortality Reduction Settings, 2009.

Table 1: Provisional number of laboratory confirmed cases of diseases under surveillance reported to the NICD - South Africa, corresponding periods 1 January - 30 September 2008/2009*

Disease/Organism	Cumulative to 30 September, 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
<i>Cryptococcus spp.</i>	2009	1040	413	1646	1094	341	625	37	593	470	6259
	2009	937	398	1845	1217	391	551	69	584	482	6474
<i>Haemophilus influenzae</i> , invasive disease, all serotypes	2008	25	21	129	30	3	19	4	5	71	307
	2009	26	18	125	34	3	21	4	7	63	301
<i>Haemophilus influenzae</i> , invasive disease, < 5 years											
Serotype b	2008	5	7	18	5	1	3	2	2	10	53
	2009	3	6	15	12	1	1	1	0	16	55
Serotypes a,c,d,f	2008	1	1	11	0	0	1	0	0	5	19
	2009	0	1	12	1	0	1	0	1	22	38
Non-typeable (unencapsulated)	2008	3	3	14	1	0	1	0	0	8	30
	2009	1	0	22	6	0	2	0	0	8	39
No isolate available for serotyping	2008	9	0	33	6	1	7	0	2	12	70
	2009	4	3	21	6	2	5	1	3	3	48
Measles	2008	4	1	7	4	1	1	2	4	3	27
	2009	15	14	774	13	5	24	2	16	9	872
<i>Neisseria meningitidis</i> , invasive disease	2008	20	18	177	25	4	31	8	18	58	359
	2009	24	12	164	23	2	43	7	17	57	349
Novel Influenza A virus infections***	2008	0	0	0	0	0	0	0	0	0	0
	2009	682	312	5572	2256	545	500	134	465	2106	12614
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	7	0	0	5	3	0	0	0	0	15
	2009	7	0	0	3	1	1	0	0	0	12
**Rubella	2008	258	9	135	248	107	117	9	62	23	968
	2009	122	5	145	88	34	82	40	24	35	575
<i>Salmonella spp. (not typhi)</i> , invasive disease	2008	79	27	418	95	11	35	16	20	66	767
	2009	57	25	312	82	4	32	10	32	71	625
<i>Salmonella spp. (not typhi)</i> , isolate from non-sterile site	2008	177	45	376	150	38	96	17	39	127	1065
	2009	167	38	632	129	28	134	22	63	184	1397
<i>Salmonella typhi</i>	2008	9	1	17	9	2	17	0	0	8	63
	2009	7	1	19	2	0	5	0	1	10	45
<i>Shigella dysenteriae</i> 1	2008	0	0	0	0	0	0	0	0	0	0
	2009	0	0	0	0	0	1	0	0	0	1
<i>Shigella spp. (Non Sd1)</i>	2008	120	48	347	103	8	39	18	9	300	992
	2009	169	59	481	118	5	49	10	12	321	1224
<i>Streptococcus pneumoniae</i> , invasive disease, all ages	2008	256	221	1724	423	81	190	58	140	425	3518
	2009	291	229	1781	415	82	222	69	133	475	3697
<i>Streptococcus pneumoniae</i> , invasive disease, < 5 years	2008	74	79	488	146	15	55	22	25	150	1054
	2009	85	54	489	128	16	72	35	20	160	1059
<i>Vibrio cholerae</i> O1	2008	0	0	2	0	0	32	0	0	0	34
	2009	2	0	47	0	618	310	0	28	4	1009
Viral Haemorrhagic Fever (VHF)											
Crimean Congo Haemorrhagic Fever (CCHF)	2008	1	2	0	0	0	1	2	0	0	6
	2009	0	0	0	0	0	0	1	0	0	1
Other VHF (not CCHF)****	2008	0	0	4	0	10	4	0	0	0	18
	2009	0	0	0	5	0	0	0	0	0	5

Footnotes

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

**Rubella cases are diagnosed from specimens submitted for suspected measles cases

*** Confirmed cases from NHLS and private laboratories nationally, province unknown for 42 cases

**** Rift Valley 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

U =unavailable, 0 = no cases reported

Table 2: Provisional laboratory indicators for NHLS and NICD, South Africa, corresponding periods 1 January - 30 September 2008/2009*

Programme and Indicator	Cumulative to 30 September, 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	43	15	45	45	40	30	3	10	29	260
	2009	46	8	49	84	52	33	6	17	16	311
Laboratory Programme for the Comprehensive Care, Treatment and Management Programme for HIV and AIDS											
CD4 count tests											
Total CD4 count tests submitted	2008	216199	90389	398140	592883	141196	130771	76022	104807	136294	1886701
	2009	283872	99849	482474	616415	161122	179607	40341	173095	163400	2200175
Tests with CD4 count < 200/µl	2008	82926	31656	152103	165422	50814	48693	24860	34359	39476	630309
	2009	91115	29763	161087	193389	50841	55902	12212	52534	44217	691060
Viral load tests											
Total viral load tests sub- mitted	2008	91323	39190	180367	206377	61467	46192	31477	42811	45346	744550
	2009	113308	33811	223183	253110	67178	64181	16303	71117	61920	904111
Tests with undetectable viral load	2008	44962	22940	107002	118207	35721	25108	19199	26123	36523	435785
	2009	67786	25765	150465	176064	42155	41854	10213	48905	50884	614091
Diagnostic HIV-1 PCR tests											
Total diagnostic HIV-1 PCR tests submitted	2008	18123	7323	39034	47162	11153	7545	2320	10534	12551	155745
	2009	22306	8029	48413	55573	12189	12972	2714	12902	12882	187980
Diagnostic HIV-1 PCR tests positive for HIV	2008	2374	1325	5649	7711	2033	1511	332	1818	1180	23933
	2009	2329	976	5206	5451	1592	1651	320	1556	1055	20136

Footnotes

*Numbers are for all ages unless otherwise specified. Data presented are provisional numbers reported to date and are updated from figures reported in previous bulletins.
Provinces of South Africa: EC – Eastern Cape, FS – Free State, GA – Gauteng, KZ – KwaZulu-Natal, LP – Limpopo, MP – Mpumalanga, NC – Northern Cape, NW – North West, WC – Western Cape

U = unavailable, 0 = no cases reported

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

Suggested citation: [Authors' names or National Institute for Communicable Diseases (if no author)]. [Article title]. Communicable Diseases Surveillance Bulletin 2009; 7 (4): [page numbers]. Available from <http://www.nicd.ac.za/pubs/survbull/2009/CommDisBullMay09.pdf>

Editorial and Production Staff

Cheryl Cohen
Editor

Liz Millington
Production

Editorial Board

Lucille Blumberg
John Freaan
Nelesh Govender
David Lewis
Terry Marshall
Lynn Morris
Adrian Puren
Barry Schoub