## Communicable Diseases Surveillance Bulletin

November 2004

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Before and after treatment for HIVIAIDS/TB co-infection, March and September 2003. (David Walton/PIH) Source : World Health Organization http://www.who.int/3by5/treatmentworks/en/

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[^0]| EPIDEMIC PRONE DISEASE SURVEILLANCE : JANUARY-OCTOBER |  |  | CUMULATIVE | ECP | FSP | GAP | KZP | LPP | MPP | NCP | NWP | WCP | RSA |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| AFP, cases from whom specimens have been received | < = 15 years |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 25 \\ & 18 \end{aligned}$ | $\begin{aligned} & 12 \\ & 11 \end{aligned}$ | $\begin{aligned} & 32 \\ & 23 \end{aligned}$ | $\begin{aligned} & 34 \\ & 32 \end{aligned}$ | $\begin{aligned} & 63 \\ & 55 \end{aligned}$ | $\begin{aligned} & 9 \\ & 12 \end{aligned}$ | $\begin{aligned} & 4 \\ & 4 \end{aligned}$ | $\begin{aligned} & 20 \\ & 20 \end{aligned}$ | $\begin{aligned} & 21 \\ & 23 \end{aligned}$ | $\begin{aligned} & 220 \\ & 198 \end{aligned}$ |
| Measles, IgM positive results | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 0 \\ & 1 \\ & \hline \end{aligned}$ | $\begin{aligned} & 1 \\ & 0 \end{aligned}$ | $\begin{aligned} & 90 \\ & 488 \end{aligned}$ | $\begin{aligned} & U \\ & U \\ & \hline \end{aligned}$ | $\begin{aligned} & 2 \\ & 2 \\ & \hline \end{aligned}$ | $\begin{aligned} & 32 \\ & 9 \end{aligned}$ | $\begin{aligned} & 2 \\ & 4 \\ & \hline \end{aligned}$ | $\begin{aligned} & 1 \\ & 5 \\ & \hline \end{aligned}$ | $\begin{aligned} & \mathrm{U} \\ & 13 \end{aligned}$ | $\begin{aligned} & 128 \\ & 522 \\ & \hline \end{aligned}$ |
| Rubella, IgM positive results from measles IgM negative patients | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 115 \\ & 87 \end{aligned}$ | $\begin{aligned} & 50 \\ & 2 \end{aligned}$ | $\begin{aligned} & 536 \\ & 170 \end{aligned}$ | $\begin{aligned} & U \\ & U \end{aligned}$ | $\begin{aligned} & 179 \\ & 26 \end{aligned}$ | $\begin{aligned} & 193 \\ & 165 \end{aligned}$ | $\begin{aligned} & 21 \\ & 3 \end{aligned}$ | $\begin{aligned} & 81 \\ & 98 \end{aligned}$ | $\begin{aligned} & \text { U } \\ & 17 \end{aligned}$ | $\begin{aligned} & 1175 \\ & 572 \end{aligned}$ |
| CCHF | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 1 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 1 \end{aligned}$ | $\begin{aligned} & 0 \\ & 2 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 4 \end{aligned}$ |
| Rabies, human | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 9 \\ & 7 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 1 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | 1 0 | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 10 \\ & 8 \end{aligned}$ |
| Haemophilus influenzae, invasive | All ages | All serotypes | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 5 \\ & 6 \end{aligned}$ | $\begin{aligned} & 14 \\ & 11 \end{aligned}$ | $\begin{aligned} & 102 \\ & 114 \end{aligned}$ | $\begin{aligned} & 18 \\ & 21 \end{aligned}$ | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | $\begin{aligned} & 7 \\ & 5 \end{aligned}$ | $\begin{aligned} & 1 \\ & 0 \end{aligned}$ | $\begin{aligned} & 4 \\ & 3 \end{aligned}$ | $\begin{aligned} & 41 \\ & 35 \end{aligned}$ | $\begin{aligned} & 193 \\ & 197 \end{aligned}$ |
|  | Age < 5 years | Serotype b | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 0 \\ & 1 \end{aligned}$ | $\begin{aligned} & 4 \\ & 2 \end{aligned}$ | $\begin{aligned} & 12 \\ & 15 \end{aligned}$ | $\begin{aligned} & 1 \\ & 1 \end{aligned}$ | $\begin{aligned} & 1 \\ & 1 \end{aligned}$ | $\begin{aligned} & 2 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 1 \\ & 0 \end{aligned}$ | $\begin{aligned} & 6 \\ & 3 \end{aligned}$ | $\begin{aligned} & 27 \\ & 23 \end{aligned}$ |
|  |  | Non-serotype b | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\frac{12}{2}$ | $\begin{aligned} & 1 \\ & 1 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | 1 0 | $\begin{aligned} & 3 \\ & 3 \end{aligned}$ | $\begin{aligned} & 17 \\ & 6 \end{aligned}$ |
|  |  | Non-typable | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 2 \\ & 0 \end{aligned}$ | $\begin{aligned} & 1 \\ & 3 \end{aligned}$ | $\begin{aligned} & 25 \\ & 26 \end{aligned}$ | $\begin{aligned} & 5 \\ & 2 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 8 \\ & 8 \end{aligned}$ | $\begin{aligned} & 41 \\ & 39 \end{aligned}$ |
|  |  | Unknown serotype | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 2 \\ & 3 \end{aligned}$ | $\begin{aligned} & 7 \\ & 3 \end{aligned}$ | $\begin{aligned} & 4 \\ & 11 \end{aligned}$ | $\begin{aligned} & 9 \\ & 8 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 1 \\ & 1 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \end{aligned}$ | $\begin{aligned} & 1 \\ & 1 \end{aligned}$ | $\begin{aligned} & 11 \\ & 6 \end{aligned}$ | $\begin{aligned} & 35 \\ & 33 \end{aligned}$ |
| Meningococcal disease | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 14 \\ & 23 \end{aligned}$ | $\begin{aligned} & 18 \\ & 17 \end{aligned}$ | $\begin{aligned} & 162 \\ & 141 \end{aligned}$ | $\begin{aligned} & 11 \\ & 21 \end{aligned}$ | $\begin{aligned} & 1 \\ & 9 \end{aligned}$ | $\begin{aligned} & 11 \\ & 9 \end{aligned}$ | $\begin{aligned} & 4 \\ & 4 \end{aligned}$ | $\begin{aligned} & 25 \\ & 17 \end{aligned}$ | $\begin{aligned} & 75 \\ & 50 \end{aligned}$ | $\begin{aligned} & 321 \\ & 291 \end{aligned}$ |
| Streptococcus pneumoniae, invasive | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & \hline 73 \\ & 127 \end{aligned}$ | $\begin{aligned} & 87 \\ & 176 \end{aligned}$ | $\begin{aligned} & 1724 \\ & 1711 \end{aligned}$ | $\begin{aligned} & 187 \\ & 413 \end{aligned}$ | $\begin{aligned} & 36 \\ & 61 \end{aligned}$ | $\begin{aligned} & 104 \\ & 149 \end{aligned}$ | $\begin{aligned} & 14 \\ & 17 \end{aligned}$ | $\begin{aligned} & 122 \\ & 95 \end{aligned}$ | $\begin{aligned} & 341 \\ & 384 \end{aligned}$ | $\begin{aligned} & 2688 \\ & 3133 \end{aligned}$ |
|  | Age < 5 years |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 32 \\ & 53 \end{aligned}$ | $\begin{aligned} & 40 \\ & 56 \end{aligned}$ | $\begin{aligned} & 479 \\ & 490 \end{aligned}$ | $\begin{aligned} & 73 \\ & 143 \end{aligned}$ | $\begin{aligned} & 4 \\ & 17 \end{aligned}$ | $\begin{aligned} & 29 \\ & 39 \end{aligned}$ | $\begin{aligned} & 2 \\ & 4 \end{aligned}$ | $\begin{aligned} & 29 \\ & 27 \end{aligned}$ | $\begin{aligned} & 167 \\ & 170 \end{aligned}$ | $\begin{aligned} & 855 \\ & 999 \end{aligned}$ |
|  | Penicillin, nonsusceptible, all ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 12 \\ & 25 \end{aligned}$ | $\begin{aligned} & 12 \\ & 30 \end{aligned}$ | $\begin{aligned} & 368 \\ & 417 \end{aligned}$ | $\begin{aligned} & \hline 49 \\ & 105 \end{aligned}$ | $\begin{aligned} & 2 \\ & 10 \end{aligned}$ | $\begin{aligned} & 10 \\ & 30 \end{aligned}$ | $\begin{aligned} & 2 \\ & 0 \end{aligned}$ | $\begin{aligned} & \hline 11 \\ & 22 \end{aligned}$ | $\begin{aligned} & 83 \\ & 92 \end{aligned}$ | $\begin{aligned} & 549 \\ & 731 \end{aligned}$ |
|  | Susceptibility unknown, all ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 21 \\ & 15 \end{aligned}$ | $\begin{aligned} & \hline 7 \\ & 21 \end{aligned}$ | $\begin{aligned} & 141 \\ & 274 \end{aligned}$ | $\begin{aligned} & 29 \\ & 63 \end{aligned}$ | $\begin{aligned} & \hline 7 \\ & 9 \end{aligned}$ | $\begin{aligned} & 9 \\ & 20 \end{aligned}$ | $\begin{aligned} & \hline 0 \\ & 3 \end{aligned}$ | $\begin{aligned} & 33 \\ & 11 \end{aligned}$ | $\begin{aligned} & 39 \\ & 43 \end{aligned}$ | $\begin{aligned} & 286 \\ & 459 \end{aligned}$ |
| Salmonella species - invasive isolates | All ages | All serotypes excl. <br> S. typhi | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 12 \\ & 7 \end{aligned}$ | $\begin{aligned} & 17 \\ & 12 \end{aligned}$ | $\begin{aligned} & 401 \\ & 409 \end{aligned}$ | $\begin{aligned} & 11 \\ & 45 \end{aligned}$ | $\begin{aligned} & 4 \\ & 9 \end{aligned}$ | $\begin{aligned} & 17 \\ & 7 \end{aligned}$ | $\begin{aligned} & 1 \\ & 0 \end{aligned}$ | $\begin{aligned} & 4 \\ & 5 \end{aligned}$ | $\begin{aligned} & 46 \\ & 33 \end{aligned}$ | $\begin{aligned} & 513 \\ & 51 \end{aligned}$ |
| Salmonella species - enteric isolates | All ages | All serotypes excl. S typhi | $\begin{array}{r} 2003 \\ 2004 \\ \hline \end{array}$ | $\begin{aligned} & 78 \\ & 94 \\ & \hline \end{aligned}$ | $\begin{array}{r} 19 \\ 18 \\ \hline \end{array}$ | $\begin{aligned} & 191 \\ & 173 \\ & \hline \end{aligned}$ | $\begin{array}{r} 10 \\ 79 \\ \hline \end{array}$ | $\begin{aligned} & 2 \\ & 5 \\ & \hline \end{aligned}$ | $\begin{aligned} & 17 \\ & 20 \\ & \hline \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{array}{r} 18 \\ 23 \\ \hline \end{array}$ | $\begin{aligned} & 225 \\ & 100 \\ & \hline \end{aligned}$ | $\begin{aligned} & 560 \\ & 512 \end{aligned}$ |
| Salmonella typhi | All ages |  | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 2 \\ & 6 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{aligned} & 16 \\ & 14 \end{aligned}$ | $\begin{array}{r} 2 \\ 5 \\ \hline \end{array}$ | $\begin{aligned} & 4 \\ & 8 \\ & \hline \end{aligned}$ | $\begin{aligned} & 12 \\ & 4 \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{aligned} & 1 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{aligned} & 2 \\ & 6 \\ & \hline \end{aligned}$ | $\begin{aligned} & 39 \\ & 43 \end{aligned}$ |
| Shigella species | All ages | All serotypes | $\begin{array}{r} 2003 \\ 2004 \\ \hline \end{array}$ | $\begin{array}{r} 99 \\ 77 \\ \hline \end{array}$ | $\begin{array}{r} 24 \\ 25 \\ \hline \end{array}$ | $\begin{aligned} & 117 \\ & 164 \\ & \hline \end{aligned}$ | $\begin{aligned} & 32 \\ & 92 \\ & \hline \end{aligned}$ | $\begin{aligned} & 12 \\ & 8 \\ & \hline \end{aligned}$ | $\begin{array}{r} 26 \\ 21 \\ \hline \end{array}$ | $\begin{aligned} & 0 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{aligned} & 6 \\ & 10 \\ & \hline \end{aligned}$ | $\begin{aligned} & 235 \\ & 224 \end{aligned}$ | $\begin{array}{r} 551 \\ 621 \\ \hline \end{array}$ |
| Vibrio cholerae 01 | All ages | All serotypes | $\begin{aligned} & 2003 \\ & 2004 \end{aligned}$ | $\begin{aligned} & 53 \\ & 26 \\ & \hline \end{aligned}$ | $\begin{array}{r} 0 \\ 0 \\ \hline \end{array}$ | $\begin{array}{r} 3 \\ 3 \\ \hline \end{array}$ | $\begin{aligned} & 0 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{aligned} & 0 \\ & 0 \\ & \hline \end{aligned}$ | $\begin{aligned} & 74 \\ & 217 \\ & \hline \end{aligned}$ | 0 0 | $\begin{aligned} & 1 \\ & 46 \\ & \hline \end{aligned}$ | 1 0 | $\begin{aligned} & 132 \\ & 292 \\ & \hline \end{aligned}$ |

# WORLD HEALTH ORGANIZATION (WHO) INFLUENZA VIRUS VACCINES COMPOSITION RECOMMENDATION 

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During the period February to September 2004, influenza A(H1N1 and H1N2), A(H3N2) and B viruses circulated in many parts of the world.

Influenza A(H1N1 and H1N2) viruses were isolated from sporadic cases in many countries, while two countries reported outbreaks. Most isolates were antigenically similar to A/New Caledonia/20/99. Current vaccines containing A/New Caledonia/20/99 antigen stimulated HA antibodies against recent $\mathrm{A}(\mathrm{H} 1)$ influenza isolates, which were of similar titre and frequency to those against the vaccine virus.

Influenza A(H3N2) viruses were associated with outbreaks in many countries. While the majority of isolates were similar to A/Fujian/411/2002, an increasing proportion of recent isolates was distinguishable from the A/Wyoming/3/2003 vaccine virus and more closely related to A/Wellington/1/2004. Current vaccines containing A/Wyoming/3/2003 antigen stimulated HA antibodies that were lower in frequency and titre to A/Wellington/1/2004-like viruses than to the vaccine virus.

Influenza B activity occurred sporadically with only one reported outbreak. The majority of recent isolates were antigenically similar to B/Shanghai/361/2002. Current vaccines containing influenza B/Shanghai/ 361/2002-like antigen stimulated HA antibodies to recent B/Shanghai/361/2002-like isolates that were of similar titre and frequency to those against the vaccine virus.

The influenza season in South Africa was mild, with viruses isolated from early May to August. All the
isolates except one were influenza $\mathrm{A}(\mathrm{H} 3 \mathrm{~N} 2)$ and were closely related to the A/Wellington/1/2004 strain. The remaining isolate was influenza $B$ and shared close similarity to strains isolated in Israel and Madagascar.

As in previous years, the national control authorities should approve the specific vaccine viruses used in each country. National public health authorities are responsible for recommendations regarding the use of the vaccine. WHO has published recommendations on the prevention of influenza.

Most of the population is likely to have been previously infected with influenza A(H1N1 and H1N2), influenza A(H3N2) and influenza B viruses. As a consequence, 1 dose of inactivated influenza vaccine should be immunogenic for individuals of all ages except young children. Previously unimmunised children should receive 2 doses of inactivated vaccine with an interval between doses of at least 4 weeks.

Updated epidemiological information is available on WHO's web site at http://www.who.int/influenza.

It is recommended that vaccines to be used in the 2005 season (southern hemisphere winter) contain the following:
— an A/New Caledonia/20/99(H1N1)-like virus;
— an A/Wellington/1/2004(H3N2)-like virus;
— a B/Shanghai/361/2002-like virus.*
*Currently used vaccine viruses include B/Shanghai/ 361/2002, B/Jilin/20/2003 and B/Jiangsu/10/2003

## Reference

World Health Organization, Weekly Epidemiological Record, 2004, 79, 369-373

# THE HIV \& AIDS COMPREHENSIVE MANAGEMENT, TREATMENT \& SUPPORT PROGRAMME 

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In South Africa the statistics quantifying the problems we are experiencing with HIV and AIDS are sobering. It is estimated that we currently have approximately 5 million infected people of whom 250000 are children. Roughly 10\% (500 000) of infected people have AIDS and need access to anti-retroviral treatment immediately. By this time in 2005 , this need will have increased to around 750000 people.

In response to these needs, the National Department of Health announced the launch of
a new comprehensive plan for the management of HIV and AIDS in South Africa on 8 August 2003. This included providing access to anti-retroviral treatment using a phased-in approach nationally for those who have advanced HIV infection in South Africa.

The initial 8 months were spent planning the programme, getting the plan approved by the Minister of Health and the Cabinet, and the initial implementation of price negotiations with, amongst others, suppliers of pharmaceuticals and
diagnostic material. From 1 April 2004 the first clinical sites were ready to implement the programme. A target was set of starting 53000 people on anti-retroviral treatment by the end of March 2005. One hundred and thirteen clinic sites would carry out the administration of this treatment across South Africa. This would increase each year until, by five years into the programme, treatment would be available to $100 \%$ of the population that needed it.

From inception of the programme, the National and Provincial Departments of Health have been collecting data on the numbers of people screened and placed on treatment, adverse events occurring due to drug toxicity, the pharmaceutical system, the nutritional support programme, and the laboratory monitoring. Plans for the development of a National Patient Master Index and Patient Information System have been accelerated in support of the programme. Until this is fully implemented, data is collected locally either on existing IT networks, or manually on a paper-based system, transferred across to district level, and thence to National and Provincial Health Departments.

In compliance with the plan, all people testing positive for HIV are screened clinically and with CD4 testing. When the CD4 count drops to 200 cells $/ \mathrm{mm}^{3}$, or the person develops WHO stage IV disease irrespective of CD4 count, antiretroviral therapy is commenced. Baseline viral load is measured and then repeated at 6 month intervals together with follow up CD4 testing. Improving CD4 counts and decreasing viral load to undetectable levels indicate a good response to therapy. Treatment failure would be suspected if viral load rebounded. This could be due to failed compliance with the recommended regimen, the development of viral resistance to the drugs, or discontinuation of treatment due to toxic side effects.

Within the NHLS, the decision was made to use the PLG CD4 method for CD4 counting. This enabled us to decrease the costs of CD4 tests by $50 \%$. In addition, this method displays excellent reproducibility within and between laboratories. The BioMeriuex EasyQ viral load system was selected as the system for monitoring patients on antiretroviral therapy. A new automated extraction system has now completed validation and will be ready for use from early 2005. The combination of this automated extraction system with the realtime amplification and detection assay should permit rapid turn around times, and high throughput of specimens each day, while minimizing user error and contamination risks.

Six months into the programme, NHLS had already tested in excess of 130000 patient samples for CD4 counts. Approximately 50\% of these samples demonstrated CD4 counts of below 200 cells $/ \mathrm{mm}^{3}$, and 20\% overall had CD4 counts below 50 cells $/ \mathrm{mm}^{3}$. In the same period, 16000 viral load tests were completed with more than $50 \%$ measuring more than 50000 copies/ ml . These statistics stress the urgency of this programme, highlighting the need to accelerate the availability of drugs to the population accessing the clinical services.

HIV contributes in no small way to increasing the effects of poverty on the continent of Africa. The nature of the transmission of this infection determines that it is predominantly the younger, more sexually active sector of the population that is targeted by the disease. These are the parents, the labour force, the professionals, the agricultural producers, and the people who have traditionally supported the needs of the generations that come before and after them. Needless to say, the impact is felt across all age groups, as even those not infected by this virus feel the effects of it. Apart from the increasing burden placed on the health care services, proper management of HIV and AIDS is a financial imperative, particularly as the costs associated with the treatment and monitoring of the disease become increasingly affordable.

While no public health programme on as ambitious a scale as this will ever go perfectly according to plan, much has been achieved through the planning and early implementation phases of the programme, ensuring ever increasing access to this life saving intervention. Criticism will no doubt be levelled at the National Department of Health for all sorts of reasons, some valid and some less so. It is time, however, to practice a measure of advocacy in support of all the work and good that has been achieved. This is more likely to ensure the ongoing commitment of, and assist in maintaining the impetus already gained by the political system of this country than whittling away at the credibility of people who have made considerable personal effort to overcome the inertia of the past. If the fear of criticism is lessened, the doors of communication will be more likely to be opened, and greater transparency of all processes will result. This could result in a self perpetuating cycle of confidence building that can only be of benefit to health care and political credibility in this country overall.

## CHOLERA

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As summer progresses, we are expecting to see an increase in diarrhoeal diseases, including salmonellosis, shigellosis and cholera. Bacterial diarrhoea is very much a disease of the summer months and clinicians and laboratories would be well advised to be aware of any increase above normal incidence rates for the season. As the majority of cholera cases do not present with florid rice water stools, should there be any question as to the aetiological agent, stool specimens should be taken and a specific request for cholera diagnosis should be made on the request slip. Be aware that in cases of mixed bacterial infections, dysentery may rarely be a presenting feature and cholera could be isolated from such cases.

The current epidemic started in 1997 in Mozambique and was primarily due to V. cholerae O1 El Tor Ogawa. The epidemic consisted of imported cases and occasional locally acquired
cases until 2000. The first cases occurred in KwaZulu-Natal and over 300000 cases have occurred to date, the majority between the years 2000 and 2001. Associated cases in neighbouring provinces, both imported and locally acquired, have been identified, the most severely affected provinces include KwaZulu-Natal, Eastern Cape, Northern Province and Mpumalanga. North-West Province experienced a significant outbreak at the beginning of this year where the first case in this province this year presented with dysentery as described above, resulting in an unacceptable delay in the diagnosis of the disease. It is highly likely that the disease will return this coming season. Reports of cholera from other African countries are ongoing and the chances of reimportation of the disease, as well as local recurrence are high. A more complete overview of cholera can be found in the March 2004 issue of the NICD bulletin.

## ELIMINATING MEASLES, SOUTH AFRICA, 2004

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## INTRODUCTION

Measles is caused by a virus of the genus Morbillivirus in the Paramyxoviridae family. Eight different strains of measles viruses ( 23 genotypes) have been identified to date; some of these circulate endemically in certain countries ${ }^{1}$. 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. It is predominantly a childhood disease causing rash, fever and any of the following: cough, coryza and conjunctivitis. Vaccination changes the age distribution of measles cases, depending on the coverage and targeted age groups. It 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.

The most common complications are pneumonia, either due to the measles virus or as a result of secondary bacterial or viral infection, diarrhoea, croup, otitis media, mouth ulcers and eye pathology. Less commonly, encephalitis may complicate measles in 1 in 1000 reported cases resulting in permanent brain damage. Myocarditis, pneumothorax, pneumomedia-
stinum, appendicitis and sub-acute sclerosing pan encephalitis (SSPE), a fatal chronic infection of the brain have all been reported ${ }^{2}$.

## DISEASE BURDEN

In spite of available vaccination, measles remains a heavy public health burden worldwide especially in developing countries with 30-40 million cases, 26 million disability adjusted life years (DALYs) and 745000 deaths for the year 2001. ${ }^{3}$ This represents $50-60 \%$ of the estimated million deaths attributable to vaccine-preventable diseases of childhood. Measles may be ultimately responsible for more child deaths than any other single agent because of complications from pneumonia, diarrhoea and malnutrition. Measles is also the major cause of preventable blindness in the world, affecting the same disadvantaged populations.

Of the deaths attributable to measles, $98 \%$ occur in developing countries, where vitamin A deficiency is common. Case-fatality rates in these countries are usually estimated to be in the range 1-5\% but may reach 10-30\% in some situations.

## ACCELERATED CONTROL, ELIMINATION AND ERADICATION

Measles, like polio and smallpox, can potentially be eradicated as it infects only humans, no carrier state or environmental reservoir exists and an effective vaccine is available.

Measles elimination refers to interruption of transmission in a sizable geographic area in which vaccination would nevertheless need to continue because of the continued threat of reintroduction of the virus. Eradication, defined as the global interruption of measles transmission, represents the sum of successful elimination efforts in all countries. Once eradication is achieved, vaccination could be stopped without risk for measles outbreaks.

Successes in interrupting indigenous transmission of measles virus in the Americas and in the United Kingdom prompted the World Health Organization (WHO), Pan American Health Organization (PAHO), and CDC to convene a meeting in July, 1996 to consider the feasibility of global measles eradication. Presentations at the meeting included an overview of global measles control and elimination efforts; detailed reviews of successful measles elimination efforts in Latin America, the English-speaking Caribbean, Canada, and the United States; surveillance for clinical disease; laboratory tools for antibody detection and virus identification; and other factors that might influence the feasibility of disease eradication.

The meeting concluded that although measles eradication is a logical addition to and extension of the poliomyelitis eradication initiative, the effort should build on the success of poliomyelitis eradication. Consequently, measles eradication should not be undertaken immediately and simultaneously in all parts of the world. Measles eradication efforts should await maturation of the poliomyelitis eradication program in each region of the globe, and should be implemented as countries and regions become free of poliomyelitis. Because of the rapid accumulation of persons susceptible to measles, the implementation phase of an eradication effort should be compressed into as brief a time as possible. Research into the molecular pathogenesis of measles and the immune response to measles virus infection should continue. ${ }^{4}$

## SA OUTBREAKS, 2004

From January to October 2004, 522 laboratory confirmed measles cases have been detected in South Africa, of which 488 (93.5\%) occurred in Gauteng Province. Sporadic cases have occurred in all provinces except the Free State with localised limited outbreaks in KwaZulu-Natal, Mpumalanga, the Western and Northern Cape (figure 1).

Cases occurred during all epidemiological weeks except week 5 and 7. Cases increased in the winter weeks from week 25. The highest number of cases occurred in week 30.

The age distribution of cases has ranged from 1 month to 31 years with the median age of 10 months. However, the age distribution in the various outbreaks has reflected underlying contributing factors with mostly young adults affected in the Western Cape related to low immunisation coverage in migrant, mobile populations and mostly young infants in Gauteng probably due to accumulation of susceptibles, mobile populations and high population density.
$10.4 \%$ of cases had no recorded age, most of whom were seen in hospital out patient departments. These patients were most likely not sick enough to be admitted and may differ in age distribution from the cases with known ages, demonstrating the importance of completing all fields of case investigation and laboratory request forms.

The national mass measles and polio vaccination campaign for children under 5 years of age took place in the last week of July (week 31). Although numbers did not decrease, the proportion of cases in the age group targeted by the campaign significantly decreased (figure 2 and figure 3 ).

Genotyping of the virus has shown that the same virus strain, probably introduced from a neighbouring country in mid 2003, has been responsible for all the measles cases.

## STRATEGIES TO CONTAIN MEASLES OUTBREAKS

The national Expanded Programme on Immunisation (EPI-SA) has identified the following measures to interrupt transmission of indigenous measles in South Africa based on internationally agreed strategies. These measures are discussed in the document Strategies to contain measles outbreaks in South Africa, Department of Health, 2003.

Internationally recommended strategies to eliminate measles include:

- High routine coverage to ensure a high level of herd immunity
- Mass immunisation campaigns that provide a second opportunity for measles immunisation
- Mop up campaigns in areas of low coverage
- Case based surveillance for all suspected cases with laboratory confirmation

These strategies may be divided into three broad activities:

- Primary prevention
- Enhanced routine coverage of both the first and second dose of measles vaccine and administration of vitamin A
- Social mobilisation, health promotion and community awareness, including promoting routine immunisation and responding to issues raised by anti immunisation lobbies.
- Secondary prevention
- Strengthen the implementation of integrated disease surveillance and response
- Early diagnosis and treatment of measles cases
- Strengthen notification at public and private health facilities including general practitioners
- Strengthen the laboratory network and collection of blood and urine specimens from every suspected case
- Prompt outbreak response to prevent spread. Strengthening surveillance, information management and monitoring is essential for timeous response
- Monitoring and evaluation
- Strengthen routine health information systems and implement integrated disease surveillance. Routine EPI coverage should be calculated at health facilities as well as all levels of the health department so that prompt action can be taken when monthly coverage targets are not reached
- Strengthen case based surveillance so that the chain of transmission can be established and corrective action taken
- Review surveillance status. All districts should detect at least 1 suspected case per 100000 population. Non- and underreporting districts should be Identified and reasons established for lack of reporting or cases
- Strengthen laboratory involvement and feedback to ensure prompt reporting of results and identification of outbreaks

South Africa adopted the measles elimination strategy in 1997 and case based surveillance with laboratory confirmation of suspected measles cases was introduced in 1998. The National Department of Health pays for the testing of blood and urine specimens of all suspected measles cases tested at the NICD.

Due to the non specific clinical presentation and concurrent rubella outbreaks, it is essential that all suspected cases have a blood and urine specimen taken to confirm the diagnosis and guide intervention strategies.

Addressing nosocomial spread is critical in any measles outbreak and all admissions to institutions should be immunised from 6 months of age. This dose is additional to the routine 9 and 18 month doses and should be recorded on the road to health card in the space provided for other immunisations. Patients with rash and fever or suspected measles cases should be seen promptly and not wait in queues and crowded waiting areas, and isolated if admitted. Hospitals and institutions such as children's homes must provide measles vaccination to all children and staff that have no evidence of previous vaccination.

## CONCLUSION

It has been widely demonstrated that measles elimination is achievable. The last indigenous measles case in the Americas occurred on 20 September 20025. Transmission of indigenous measles has been mostly interrupted in Canada since 1998 ${ }^{6}$. Finland achieved elimination through high immunisation coverage in 12 years $^{7}$ and Albania through high coverage and targeted mass campaigns in less than 3 years.

High routine immunisation coverage is a critical part of achieving and maintaining interruption of indigenous transmission and limiting outbreaks due to importation of measles virus. This can only be reached through optimising all opportunities to immunise children at any visit to a health facility, whether for growth monitoring or accompanying an adult for a blood pressure check-up.

## References

${ }^{1}$ Rota PA, Bellini WJ. Update on the global distribution of genotypes of wild type measles viruses. JID 2003: 187(Suppl 1):S270-6.
${ }^{2}$ Benenson AS. Control of Communicable Diseases Manual, American Public Health Association, Washington 1995;293-9.
${ }^{3}$ World Health Organization. World Health Report 2002;192-7.
${ }^{4}$ CDC. Measles Eradication: Recommendations from a Meeting Cosponsored by the World Health Organization, the Pan American Health Organization, and CDC. MMWR 1997:46(RR11);1-20.
${ }^{5}$ de Quadros CA, Izurieta H, Venczel L, Carrasco P. Measles eradication in the Americas: progress to date. J Infect Dis. 2004 May 1;189 Suppl 1:S227-35.
${ }^{6}$ King A, Varughese P, De Serres G, Tipples GA, Waters J; Working Group on Measles Elimination. Measles elimination in Canada. J Infect Dis. 2004 May 1;189 Suppl 1:S236-42.
${ }^{7}$ Peltola H, Heinonen OP, Valle M, Paunio M, Virtanen M, Karanko V, Cantell K. The elimination of indigenous measles, mumps, and rubella from Finland by a 12-year, two-dose vaccination program. N Engl J Med. 1994 Nov 24;331(21):1397-402.
${ }^{8}$ Bino S, Kakarriqi E, Xibinaku M, Ion-Nedelcu N, Bukli M, Emiroglu N, Uzicanin A. Measles-rubella mass immunization campaign in Albania, November 2000. J Infect Dis. 2003 May 15;187 Suppl 1:S223-9.

Fig 1 : Confirmed measles and rubella cases per province, South Africa, 2004


Fig 2 : Confirmed measles cases per epidemiological week, South Africa, 2004


Fig 3 : Age distribution of patients with measles before and after the mass immunisation campaign, South Africa, 2004
Jan-Aug N=325
Sep/Oct N=197



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