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Checking a live rodent trap, RATZOOMAN project, Mapate, Limpopo Province

CONTENTS

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Provisional listing: number of laboratory-confirmed cases in South Africa of diseases under surveillance reported to the NICD, corresponding periods 1 January - 30 September 2005/2006

	Disease/ Organism	Case Definition	Subgroup	Cumulative to 30 Sept, year	EC	FS	GA	KZ	LP	MP	NC	NW	WC	South Africa
VIRAL DISEASES	Acute Flaccid Paralysis	Cases < 15 years of age from whom specimens have been received as part of the Polio Eradication Programme		2005	19	20	27	41	30	14	2	24	21	198
				2006	48	20	55	36	44	35	7	24	29	298
	Measles	Measles IgM positive cases from suspected measles cases, all ages		2005	472	0	40	73	1	5	0	1	14	606
				2006	3	0	19	2	1	9	4	21	1	60
	Rubella	Rubella IgM positive cases from suspected measles cases , all ages		2005	83	9	111	72	6	63	18	36	19	417
				2006	159	37	409	194	336	137	69	139	88	1568
	VHF	Laboratory-confirmed cases of CCHF (unless otherwise stated), all ages		2005	0	0	0	0	0	0	0	0	1	1
				2006	0	2	2	0	0	0	2	0	0	6
	Rabies	Laboratory-confirmed human cases, all ages		2005	4	1	0	0	0	0	0	0	0	5
				2006	2	0	0	3	17	0	0	1	0	23
BACTERIAL AND FUNGAL DISEASES	*Haemophilus influenzae*	Invasive disease, all ages	All serotypes	2005	11	13	115	20	1	8	1	0	31	200
				2006	7	13	109	42	1	3	7	3	35	220
		Invasive disease, < 5 years	Serotype b	2005	4	2	15	3	0	0	0	0	3	27
				2006	2	4	20	7	1	1	1	0	7	43
			Serotypes a,c,d,e,f	2005	0	1	9	2	0	1	0	0	3	16
				2006	1	1	13	2	0	0	1	0	3	21
			Non-typeable (unencapsulated)	2005	2	3	34	2	0	2	0	0	4	47
				2006	1	3	30	3	0	1	0	0	2	40
			No isolate available for serotyping	2005	2	1	12	3	0	0	0	0	8	26
				2006	1	2	14	9	0	0	0	1	6	33
	Neisseria meningitidis	Invasive disease, all ages		2005	9	18	289	15	8	16	6	11	49	421
				2006	20	29	279	18	5	24	13	20	51	459
	Streptococcus pneumoniae	Invasive disease, all ages	Total cases	2005	150	163	1706	346	53	166	27	88	374	3073
				2006	155	161	1611	340	77	166	31	104	358	3003
			Penicillin non-susceptible isolates	2005	33	44	517	109	12	42	7	20	109	893
				2006	40	39	470	108	19	51	8	20	88	843
			No isolate available for susceptibility	2005	20	7	210	25	10	17	3	8	38	338
				2006	26	12	231	39	12	25	3	13	29	390
		Invasive disease, < 5 years		2005	52	55	478	136	15	47	4	20	155	962
				2006	44	55	438	111	13	38	9	22	114	844
	Salmonella spp. (not typhi)	Invasive disease, all ages		2005	31	20	383	82	7	28	0	2	58	611
				2006	37	19	466	94	2	26	0	15	63	722
		Confirmed cases, isolate from a non-sterile site, all ages		2005	128	24	162	150	22	47	3	26	141	703
				2006	68	28	149	139	17	37	11	54	125	628
	Salmonella typhi	Confirmed cases, isolate from any specimen, all ages		2005	22	0	23	8	3	80	0	0	9	145
				2006	38	1	14	12	6	7	0	0	18	96
	Shigella dysenteriae 1	Confirmed cases, isolate from any specimen		2005	0	0	0	0	0	0	0	0	4	4
				2006	0	0	0	1	0	0	0	0	1	2
	Shigella spp. (Non Sd1)	Confirmed cases, isolate from any specimen, all ages	All serotypes	2005	108	27	193	130	14	33	5	4	245	759
				2006	105	36	170	148	10	27	21	14	316	847
	Vibrio cholerae O1	Confirmed cases, isolate from any specimen, all ages	All serotypes	2005	0	0	0	0	0	0	0	0	0	0
				2006	0	0	0	0	0	0	0	0	0	0
	Cryptococcus (*Cryptococcus* spp.)	Invasive disease, all ages	Total cases (incl. *C neoformans*)	2005	333	180	1179	712	102	261	31	157	243	3198
				2006	339	202	1440	984	132	342	50	287	253	4029
			C. gattii	2005	2	1	29	14	16	14	0	8	7	91
				2006	3	4	23	6	16	20	4	9	7	92

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

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

0 = no cases reported

STUDYING RODENT-BORNE DISEASES IN AFRICA - THE RATZOOMAN PROJECT

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RATZOOMAN is the acronym for a 3-year, multi-country, European Union-sponsored research project that studied the zoonotic potential of 3 rodent-borne diseases in Africa. Tanzania, Mozambique, Zimbabwe, and South Africa participated in partnership with research entities in Europe. The lead institution was the Natural Resources Institute, University of Greenwich, UK. In South Africa the prime contractor was the National Institute for Communicable Diseases in the form of its Special Bacterial Pathogens Reference Unit; some work was subcontracted to the Plant Protection Research Institute (PPRI), Pretoria (Frikkie Kirsten, Emil von Maltitz and Fanie Malebane) and the Natural Science Museum, Durban (Dr Peter Taylor).

The objective of this project was to measure the prevalence of the three target diseases, namely, plague, leptospirosis and toxoplasmosis. Host ranges and the infection dynamics within the host populations, and transmission from animal hosts to humans were studied. Plague is normally a flea-transmitted bacterial disease, caused by *Yersinia pestis*. The main reservoir is rodents, and plague was an important public health problem in South Africa until around the middle of the last century, when it became quiescent over most of its distribution; a focus was maintained in the Eastern Cape, until the last recognized human outbreak occurred about 25 years ago. The spirochaetal disease leptospirosis is maintained by asymptomatic infection of many animals, including rodents; the organisms are excreted in the urine and survive in surface water under the right environmental conditions. Humans are typically infected by exposure to untreated water via domestic, agricultural, or recreational activities. The disease spectrum ranges from asymptomatic or mild, to life-threatening (Weil's disease). Toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii*, which has cats as definitive hosts, and a very wide range of intermediate hosts including wild rodents and domestic animals, and humans (accidental intermediate hosts). Groups at particular risk are the immunosuppressed (mainly because of HIV, but occasionally transplant- or chemotherapy-related) in whom toxoplasmic encephalitis is the main risk, and the unborn, who can develop brain involvement of varying severity, mental retardation, and eye disease.

Methods used in the project in South Africa were community-based assessments of knowledge and beliefs about rodents, and their economic impact (eg loss of food, crops); trapping of rodents and testing of rodent blood and tissue samples (n>1600) and testing of human blood (n=217) for evidence of exposure to the three target diseases. Capture-recapture studies were done in Mapate to establish rodent population dynamics. Carcasses of trapped rodents identified by the Durban Natural Science Museum or the University of Antwerp/Danish Pest Control Laboratory. Some dogs (n=34) were sampled in Mapate as well. Fleas were collected from trapped rodents for identification and testing for evidence of plague by culture and PCR. All data will ultimately be assembled into a geographic information system (GIS) database, along with climatic, demographic, agricultural etc information required to model the distribution of rodent-borne diseases; eventually this might suggest interventions to control or reduce transmission.

The major testing site in South Africa was the rural village of Mapate, near the town of Thohoyandou in Limpopo Province. Mapate was chosen as a field site for the project because work had already been done there by scientists from PPRI, and was still ongoing at the time of this study, in relation to another project focusing on ways of controlling rats. The area has subsistence farming, performed mainly by women. Maize is the staple crop, but a large percentage of households also grow fruit. Poverty rate in Mapate is high and there are many rats present in most houses and fields.

Checking traps in Mapate, Limpopo Province

Other study sites were in Durban and near Port Elizabeth. The area known as Cato Crest is one of six informal settlements that comprises the community of Cato Manor, Durban. Cato Crest is approximately seven kilometers to the west of the Durban central business district. Illness is often believed to be brought about through witchcraft in Cato Crest. Umuthi (witchcraft medicine) is commonly thought to be the cause of illness, and this is believed to be transmissible through both rats and cats, particularly the latter. Rats often enter the houses. Damp soil and pools of stagnant water are features of most parts of Cato Crest and are risk factors for the transmission of leptospirosis.

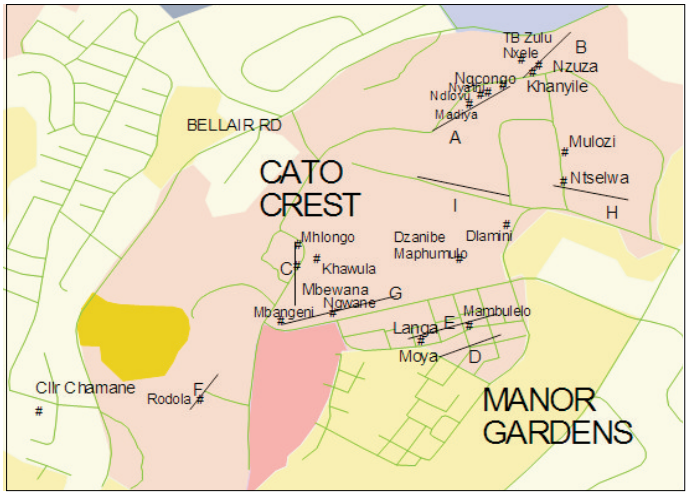
The Coega area near Port Elizabeth was chosen as the third site, as this was the site of the last outbreak of human plague in South Africa in 1982. This site is also where a major port is being built and there is a lot of disturbance of the environment because of construction, road building, etc. This environmental disruption disturbs the natural rodent populations in area, with the potential for increased contact with human communities.

RESULTS

Only limited human specimen testing was possible, due to refusal of Mapate residents to donate blood samples. Fear of witchcraft and HIV were given as the main reasons for this. Two hundred and seventeen human blood samples from Cato Crest were tested for the three diseases. Nearly 20% were positive for leptospirosis on the screening agglutination test, but only one of these could be confirmed by the definitive microagglutination test (MAT titre >1:100). One explanation for this discrepancy is that the spectrum of animal-related serovars detected by MAT may not be the same as for humans. Thirty-five percent of subjects showed evidence of toxoplasmosis exposure, which is consistent with previous serosurveys in South Africa; there were no plague-positive specimens. To date about 5-10% of rodents and dogs have tested positive for *Leptospira* sp. by PCR, but testing has not been completed.

Some of the flea species that were collected are known to be involved in the plague transmission cycle, but all tested negative. This is confirmation that there is no active plague in these areas at present.

Training was given at the start of the project in Tanzania to all the four countries involved in the project.



GIS map of Cato Crest study area showing trapping transects A-I (Courtesy Dr P Taylor)

Lorraine Arntzen, NICD, South Africa, taught the EIA serology test for detection of plague antibodies to all the participants. Dr Rassul Nala and a scientist from Instituto Nacional de Saude, Mozambique, trained in the Special Bacterial Pathogens Reference Unit, NICD, in various techniques used in the studies.

Mirjam Engelberts, KIT Biomedical Research, The Netherlands, taught culture, DriDot and MAT testing for leptospirosis to all the participants.

Herwig Leirs, Danish Pest Infestation Laboratory, taught capture-mark-release (CMR) and general trapping methods to all the participants.

Robert Machang'u, Pest Management Centre, Tanzania, taught identification, weighing, measuring and dissection of the rodents, to all the participants.

CONCLUSION

The RATZOOMAN project has helped to revive the plague surveillance program in South Africa. This surveillance program stopped a number of years ago for various reasons. Despite its present quiescent condition South Africa is a plague endemic country and needs to have ongoing surveillance in place.

The RATZOOMAN project has also helped to bring together some of the plague-endemic countries in Africa. This has helped with skills transfer between these countries and their European counterparts, as well as establish valuable communication links. All who were involved in this project are very enthusiastic about future work in this field.

The training that went on during this project enhanced scientific capacity in the four African countries and it would be a pity not to encourage further work in this field. There is a real problem with rodents and rodent-borne disease in Africa and much work needs to be done in the future. The project formally concluded with an international conference held at Malelane, South Africa, in May 2006, but to continue for some time will be the work of laboratory investigations, taxonomic identification, analysis of data and utilization of GIS to model disease transmission and make recommendations regarding control. The conference proceedings and more information about the project can be found on its website: http://www.nri.org/ratzooman

MULTIDRUG-RESISTANT TUBERCULOSIS (MDR-TB) & EXTENSIVELY DRUG-RESISTANT TUBERCULOSIS (XDR-TB)

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During the 1990's multidrug-resistant (MDR) tuberculosis (TB), defined as resistance to at least isoniazid and rifampicin emerged globally. In a national survey conducted in South Africa in 2001/2, MDR-TB was documented in 1.8% of new TB patients and 6.7% of previously treated patients (Tuberculosis Research Unit, Medical Research Council). MDR-TB treatment requires the use of secondline drugs that are less effective, more toxic and significantly more expensive than isoniazid and rifampicin based regimens. Treatment regimens include ethionamide, aminoglycosides (kanamycin or amikacin), 4-fluorinated quinolones, and cycloserine. Limited studies have confirmed that the efficacy of these regimens has ranged from 56% to 83%. Most studies have involved HIV negative patients with MDRTB.

Cases of TB with resistance to virtually all second line drugs have emerged in the past decade many of them as a result of failed MDRTB treatment and amplification of drug resistance. Drug resistant strains are readily transmissible and HIV infected patients are particularly vulnerable to nosocomial spread. A nosocomial outbreak of XDR-TB affecting HIV positive patients was reported from a TB referral hospital in Gauteng, South Africa in 1996, and all these patients died. A study to assess the extent of drug-resistant TB in the Msinga subdistrict of KwaZulu Natal identified fifty three patients with XDR-TB; 67% had a recent hospital admission; all 44 patients who were tested for HIV were positive. Fifty two patients died. Genotyping of isolates showed that 39 of 46 patients had similar strains.

A new definition of XDR-TB was proposed at a WHO meeting in October. XDR-TB is now defined as MDR-TB (*M tuberculosis* resistance to at least rifampicin and isoniazid from among the first line anti-TB drugs) with additional resistance to any fluoroquinolone, and to at least one of three injectable second-line anti-TB drugs used in treatment (capreomycin, kanamycin or amikacin).

The true extent of XDR-TB is not known. In a retrospective survey of 17,690 TB isolates collected from 2000-2004 from an international network of TB laboratories, 20% of isolates were MDR and 2 % were XDR. There are some limitations to these findings: the laboratory drug susceptibility methods used in the different countries was not standardised and there was likely selection bias in the isolates submitted. Despite these limitations XDR-TB clearly poses a serious threat to public health, particularly when associated with HIV and that control of XDR-TB will not be possible without close coordination of TB and HIV programmes and interventions.

Specific recommendations made by the WHO meeting included:

A call for countries to strengthen TB control programme management - the key to preventing TB drug resistance. This should be done in coordination with scaling up universal access to HIV treatment and care.

Technical assistance to improve drug-resistant TB surveillance methods.

Strengthen tuberculosis laboratory capacity as this is the cornerstone of the control programme in all countries utilising DOTS programmes.

Universal access to tuberculosis culture, particularly where HIV co-infection is prevalent.

Universal access to drug susceptibility testing and access to second-line anti-TB and antiretroviral drugs in all countries.

Evaluation and roll-out of rapid testing, particularly for rifampicin resistance. This will improve case detection of all patients suspected of MDR-TB and could prove to be life saving for those infected with HIV

Implementation of infection control measures to protect patients, health care workers and visitors (particularly those who are HIV infected).

Ensure all patients with HIV are adequately treated for TB and started on appropriate antiretroviral therapy.

The laboratory capacity in high prevalence countries must be strengthened to diagnose and survey drug resistance. Previous surveillance focused on the diagnosis of MDR-TB and these should immediately be extended to include XDR-TB in future. Rapid surveys of drug-resistant TB must immediately be performed to roughly estimate the extent of the problem, but cannot take the place of formal well designed national drug resistance surveillance. The association with HIV should also be determined.

Management of patients with XDR-TB is limited by a lack of effective drugs and experience with alternate regimens. Limited studies would indicate a poor prognosis for these patients.

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INSECTICIDE RESISTANCE IN MALARIA VECTOR MOSQUITOES: RESEARCH AND MANAGEMENT POST-1999/2000 EPIDEMIC IN SOUTH AFRICA

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The period 1995 to 2000 saw malaria case incidence in South Africa rise from 8,750 to 64,622, a seven-fold increase in six years (Fig. 1). In 1996, the malaria control programme changed from DDT to pyrethroids (Deltamethrin) for indoor residual house spraying because a) DDT left unsightly marks on the walls, b) pyrethroids do not persist in the environment as long as DDT yet they have a similar residual life on the walls, c) DDT caused bedbugs to bite more frequently, and d) the local vector *Anopheles arabiensis* was as susceptible to Deltamethrin as DDT (Coetzee, 2005). The three-fold increase in malaria cases that same year and for the following two years was attributed to good rains and an increase in cross-border movement of people between Mozambique and South Africa. It was only in 1999 that entomological surveys showed the presence of *Anopheles funestus* in northern

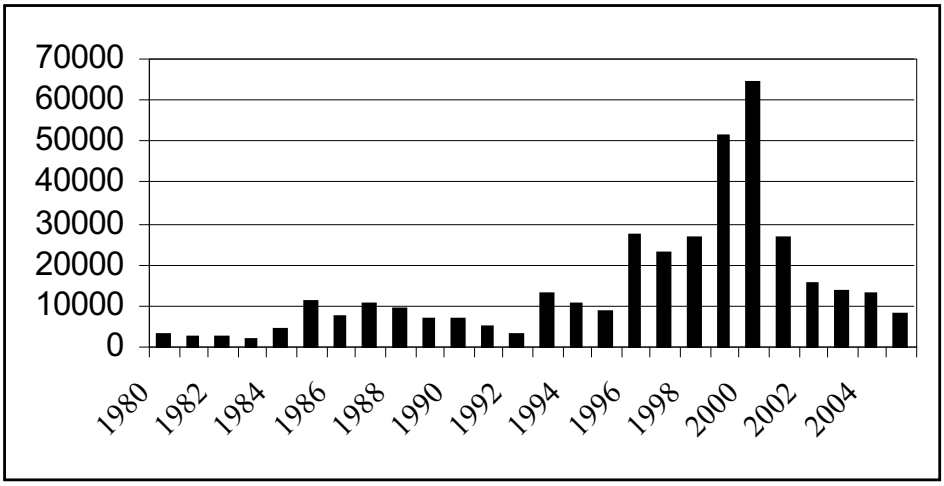

Figure 1. Malaria cases in South Africa from 1990 to 2005 (unpublished Department of Health records).

Kwazulu/Natal and, most importantly, demonstrated pyrethroid resistance in this highly efficient vector of malaria (Hargreaves et al., 2000). At the same time, failure of first-line malaria treatment with sulphadoxine-pyrimethamine (S-P) was detected in Kwazulu/Natal, exacerbating the problem (Vaughan Williams, 2003). Measures taken by the Malaria Control Programme were: (1) to reintroduce DDT for traditional structures while Deltamethrin was still used for western-style structures (Hargreaves et al., 2003), effectively creating a mosaic distribution of insecticides and resulting in *An. funestus* disappearing, and (2) to change the treatment of uncomplicated malaria from S-P to artemether combination therapy. The malaria case incidence subsequently decreased to under 8,000 by 2005 (Fig. 1).

While South Africa has experienced major treatment failures in the past (to chloroquine in the 1980's, Barnes et al., 2005), without a concomitant major malaria epidemic (Fig. 1), this was only the second time in over 50 years that *An. funestus* had been detected in the country. The previous occasion was in 1976 when a small outbreak of malaria occurred outside Tzaneen in Limpopo Province, on a farm that had not been subjected to indoor residual spraying. *Anopheles funestus* was found resting inside the houses and on dissection, sporozoites were detected in the salivary glands of the mosquitoes (unpublished report, National Institute for Tropical Diseases, 1978). This outbreak was brought under control by spraying the houses on this farm with DDT. The 1999/2000 epidemic on the other hand, was widespread and totally unexpected as indoor residual spraying had continued as normal through the years, just with a different insecticide. Before indoor residual spraying with DDT was established as the mainstay of malaria control in South Africa in the late 1940's, *An. funestus* was the major vector in the country with sporozoite rates reaching over 20% in some seasons (De Meillon, 1933). Little wonder, then, that when it returned in 1999 it caused such havoc.

Having reverted to the use of DDT in 2000, monitoring and surveillance of the malaria control programme continued and no specimens of *An. funestus* were found (Hargreaves, pers. comm.). In 2002, however, routine monitoring in northern Kwazulu/Natal detected *Anopheles arabiensis* in DDT sprayed houses (Hargreaves et al., 2003). Standard WHO procedures were used to determine resistance levels and over 37% of wild-caught mosquitoes survived the discriminating dose of 1-hr exposure to 4% DDT. Interestingly, despite the resistance and the presence of large numbers of *An. arabiensis*, no corresponding increase in malaria transmission occurred. Laboratory studies suggest that resistance was present only in very young mosquitoes and disappeared with age (unpublished data). This meant that older mosquitoes were being killed by the insecticide before parasite development in the females could reach the transmission stage. The resistance was therefore not impacting on the control programme operations.

Research on the resistance mechanisms in both *An. funestus* and *An. arabiensis* has been on-going at the NICD for the past five years (Brooke et al., 2001, 2002; Awolola et al., 2003; Masendu et al., 2005; Amenya et al., 2006). In *An. funestus* we have demonstrated that the resistance is caused by a metabolic mechanism mediated by P450 mono-oxygenase enzymes, a large group of enzymes well known to be involved in detoxifying insecticides in a number of different insects. Molecular studies have narrowed it down to a specific gene within the P450 group and currently we are involved in gene expression studies using microarray analysis. A colony of *An. arabiensis* from northern Kwazulu/Natal has been selected to high a level of DDT resistance and studies are on-going to determine the mechanisms involved in this resistance.

The diverse nature of insecticide resistance in malaria vector mosquitoes, not only in South Africa but across Africa, poses a serious problem to vector control programmes. Baseline monitoring surveys for resistance profiles are absolutely essential before decisions are taken as to which insecticide is to be used for control purposes. Research into the mechanisms involved in the resistance will provide crucial information on possible cross-resistance between classes of insecticides and this information must guide policy decisions made by Ministries of Health if malaria vector control operations are to be successful.

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THE NEW INTERNATIONAL HEALTH REGULATIONS - SAME PARADIGM MORE POWER?

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INTRODUCTION

The application of international law in the control of communicable disease has a long history but has arguably never been more relevant than in the current climate of globalisation and emerging and re-emerging infectious disease threats. The first International Sanitary Conference held in Paris in 1851 heralded a new era in health regulations and the current International Health Regulations (IHR (1969)) replaced the International Sanitary Regulations (adopted by WHO in 1951) as an international legal instrument to "ensure maximum security against international spread of diseases with minimum interference with world traffic".[1,2,3]

These regulations (IHR1969) were initially aimed at control of six infectious diseases namely, cholera, plague, yellow fever, typhus, relapsing fever and smallpox. Later they would be modified to include only 3 key diseases yellow fever, plague and cholera. The obvious inadequacy of these regulations has been apparent for decades and coupled with the failure of compliance by many WHO Member States prompted a revision process by WHO which commenced in 1995.[2]

The outbreak of severe acute respiratory syndrome (SARS) in 2003 and its devastating effect on both human health and economies served to expedite the revision process. The new revised International Health Regulations (IHR (2005)) were adopted by the World Health Assembly in May 2005 and will replace the IHR (1969) in June 2007.

THE KEY CHANGES FROM 1969 TO 2005

The revised IHR (2005) attempts to remedy the immense deficiencies of the IHR (1969) with the overarching aim "to prevent, protect against, control and provide a public health response to the international spread of disease in ways that are commensurate with and restricted to public health risks, and which avoid unnecessary interference with international traffic and trade".[3] Hence at the outset the regulations attempt to broaden their scope and potentially restrict the use of unnecessary trade and travel restrictions in disease control in the context of a global economy.

The IHR (2005) no longer restricts itself to an infectious disease specific approach but requires that WHO Member States report "all events potentially constituting a public health emergency of international concern".[3] To assist with this decision making an algorithm is provided and includes a list of potential diseases (figure 1). In addition WHO Member States must do the following:

- Establish a National IHR focal point accessible at all times for urgent communication, dissemination of information and verification of events.
- Report an event meeting IHR criteria within 24 hours of assessment
- Assess and strengthen the current surveillance capacity for detection and reporting of public health events.

The inclusion of "core capacity requirements for surveillance and response" and "core capacity requirements for designated airports, ports and ground crossings" in the IHR (2005) are significant improvements in the legislation.[4]

Whilst the IHR (1969) included automatic reporting of cases in the WHO Weekly Epidemiological Record the new IHR (2005) makes provision for confidential communication and verification of the threat with WHO prior to disclosure of information. It also allows WHO to obtain disease information from unofficial sources and requires the IHR focal point to verify such rumours on request.[2]

Clearly, many WHO Member States cannot achieve the requirements of the IHR (2005) without assistance and the regulations include an obligation on WHO to provide support to WHO Member States, create WHO focal points for rapid communication with national counterparts and provide the required technical assistance in determining the appropriate control measures during international health emergencies.[2,4]

WILL IT WORK? CHALLENGES TO IMPLEMENTATION

On paper, the IHR (2005) represent a vast expansion of the use of international law to protect global health. However as with the IHR (1969) compliance with this law is likely to be a challenge.

Failure to observe international law may be due to many factors including sovereignty, economic protection and lack of capacity.[3] Although legally binding, there is no clear mechanism of sanction for non-compliance.[5] WHO Member States need to believe in the benefits of full engagement with the new law. This may be hampered by suspicions raised regarding the motivation of the international community in revising the law and in highlighting specific diseases over others. The perception that this is driven more by a need to protect the developed world and its "superpowers" than by an altruistic approach to global health may be supported by the dismal failure of many WHO Member States to meet their promises with respect to the Millennium Development Goals in combating priority diseases.

The movement away from infectious disease specific notification provides an expanded scope of activity and increases the sensitivity of the IHR as an international surveillance system but may also reduce adherence by some countries as they are provided with broad, perhaps more subjective requirements for reporting.[3,6]

By far the most challenging aspect of implementation will be capacity. Whilst the regulations make provision for WHO support of activities this is not accompanied by any financial solutions. Developing countries lacking surveillance capacity and health infrastructure will struggle to comply even with the best intentions. It is essential that economic

assistance is made available to such countries if these regulations are ever truly to protect global health.[3,6,7]

The new IHR (2005) will be implemented in June 2007 by WHO Member States and all reservations to implementation must be submitted by December 2006. However, at the Fifty Ninth WHA in May 2006, a resolution was adopted for immediate voluntary implementation of the IHR (2005) with respect to avian influenza and the pandemic influenza threat.[8]

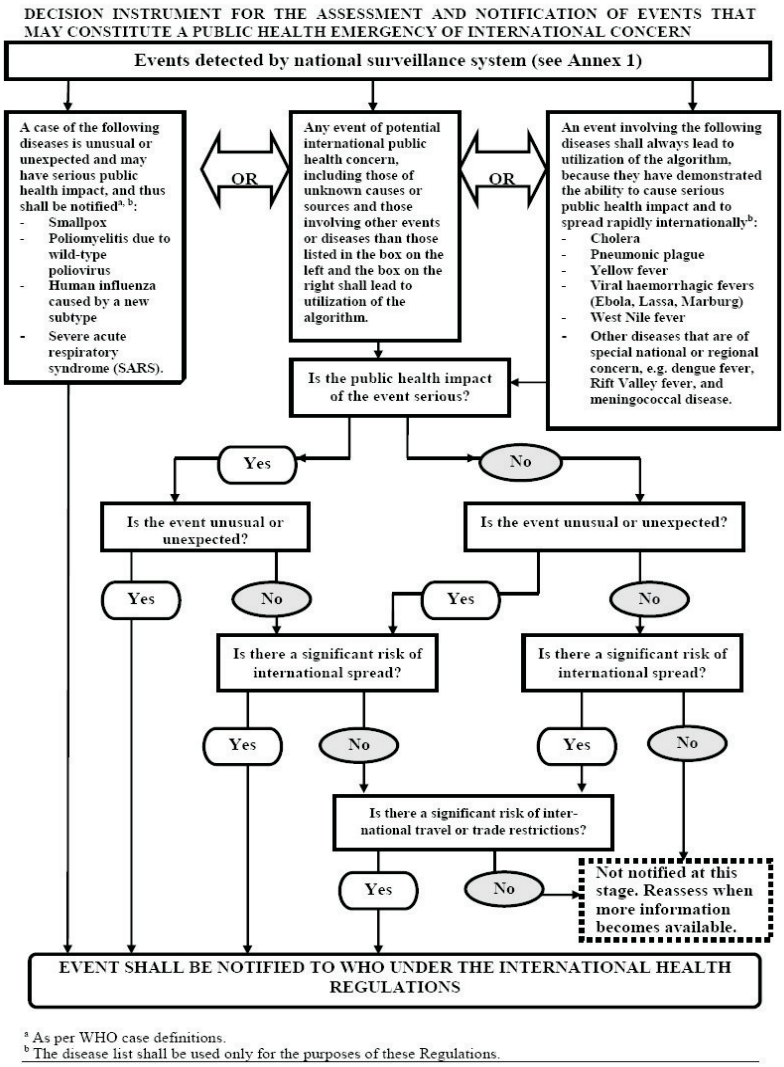
SOUTH AFRICA AND THE NEW IHR

As a WHO Member State, South Africa has agreed to implement the IHR (2005) by June 2007. This will require an assessment of current surveillance capacity and strengthening of existing systems. The national Department of Health will be responsible for implementation and will therefore be required to ensure the cooperation of all provinces in fulfilling its reporting obligations. Systems for rapid communication between provincial and national government will need to be strengthened and any political obstacles overcome. One of South Africa's many challenges will be the building of port health capacity at international airports and ports and the revision of current legislation in this regard. South Africa has an opportunity to use the IHR (2005) to obtain full political commitment for improved surveillance and public health control and to access the necessary resources for this task. It will also be an opportunity to improve cross-border communication and collaborative disease control efforts in the region.

CONCLUSIONS

The revised IHR (2005) have the potential to achieve better protection of global health and has significantly expanded to include both communicable and non-communicable disease threats. However there are likely to be several obstacles to successful compliance. WHO Member States battling overwhelming infectious disease threats such as TB, HIV and malaria within their borders may find it impossible to make provision for additional resources for these activities. It may be wiser for the global community to start by assisting in control of these massive epidemics not traditionally viewed as a "public health threat of international concern" as the most effective means of ensuring poorer WHO Member States are able to comply with the obligations of the IHR(2005). It is not the absence of a legally binding document but rather resources (financial and technical) that primarily hinders effective outbreak detection and response. Perhaps the next revision will also be accompanied by a fundamental shift in paradigm that is more likely to achieve global health benefits for all.

Figure 1



Source: World Health Assembly. Revision of the International Health Regulations, WHA58.3. 2005

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ADDITIONAL RECOMMENDED READING

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