

# GLOBAL AGENDA ON INFLUENZA — ADOPTED VERSION

(Editor's Note: The Global Agenda on Influenza was initiated and finalised during a WHO Consultation on 6-7 May 2002. It is important to increase awareness on the existence and objective of the Global Agenda on Influenza to the "influenza community" as well as to policy and decision makers at national and international levels. For full information and details, please view the web address: [http://www.who.int/emc/diseases/flu/global\\_agenda\\_report/Introduction.htm](http://www.who.int/emc/diseases/flu/global_agenda_report/Introduction.htm))

WHO has a long-established influenza surveillance network, which has contributed greatly to the understanding of influenza epidemiology and which provides an effective basis for regular updating of influenza vaccine formulations. Recognising the importance of further developing the capacity for influenza surveillance and control and for encouraging wider input and participation, the WHO Global influenza programme called for contributions to develop a Global Agenda in July 2001.

After including more than 100 suggestions on its contents and a public discussion on the website, the Global Agenda was finalised and adopted by consensus by the participants of the WHO Consultation on Global Priorities in Influenza held at WHO headquarters in Geneva, Switzerland, from 6-7 May 2002.

## What is the Global Agenda?

A compilation of a limited set of prioritised activities critical to mobilise public health action to reduce morbidity and mortality due to annual influenza epidemics and to prepare for the next influenza pandemic. It has been developed for all those involved in activities to reduce morbidity and mortality from annual influenza epidemics and preparedness for the next influenza pandemic, to coordinate national and international action in influenza surveillance and control, and to advocate and raise funds.

## Why is a Global Agenda needed?

Only 250 million influenza vaccine doses produced annually are used in developed countries. Although cost-efficient control options on vaccine usage exist, these are only available in developed countries. Only about 50 countries worldwide have national influenza prevention policies. In developed countries, vaccination rates in high-risk groups are suboptimal. In developing countries, the health and economic impact or burden of influenza is not completely understood, prioritisation of communicable disease control strategies are hampered, and vaccine and antivirals are not affordable, competing with other health priorities.

Global efforts for the international coordination of activities for prevention and control of influenza are needed for a variety of reasons:

- to strengthen links between influenza surveillance control;
- to renew interest in epidemic influenza prevention and control, and pandemic preparedness;
- to bring together all key players;
- to develop a common strategy focusing on global priorities; and
- to stimulate political commitment and increase financial investment into influenza control.

## Objectives of the Global Agenda

The Global Agenda seeks to provide impartial and prioritised guidance to all parties on regional/district and national/global action for influenza control, develop ownership and support for implementation of identified priority activities, be used as a tool to support coordination of action for

influenza control, and support advocacy for fund raising. Intended for national/international coordination of action in influenza surveillance and control and for advocacy and fund raising, the Global Agenda will also serve as a "working plan" for the WHO global forum on influenza.

## Content

The adopted version of the Global Agenda encompasses:

### A. Strengthen epidemiological and virological surveillance nationally and internationally

There is a special need for support for developing countries.

#### 1. Enhance and integrate virological and disease surveillance

Required key actions:

- Evaluate the activities and physical facilities of the national influenza centres (NICs).
- Develop standardised methods and training for laboratory and disease surveillance (develop reagents and manuals, provide training and proficiency testing).
- Encourage integrated surveillance based on clinical and virological data.
- Facilitate shipment of influenza isolates and specimens.
- Encourage NICs to collect additional data (eg, influenza-related hospitalisation, use of emergency services and mortality) in different age groups.
- Incorporate antiviral resistance monitoring.

Rationale: the WHO influenza surveillance network must be utilised more effectively in order to improve global influenza surveillance.

#### 2. Expand virological and disease surveillance

Required key actions:

- Identify gaps in geographical coverage of the WHO global surveillance network and explore the use of polio and other networks to expand surveillance coverage.
- Investigate and integrate into the WHO surveillance system other sources of samples and information, including rapid tests, commercial testing and clinical trial samples.
- Arrange regional/global meetings to improve laboratory and disease surveillance and support interactions between "sister" laboratories.

Rationale: expansion of the WHO global influenza network is essential to provide comprehensive global coverage for early warning of the emergence of variants and novel strains.

#### 3. Expand animal influenza surveillance and integrate with human influenza surveillance

Required key actions:

- Expand and formalise the WHO Animal Influenza Network (AIN).
- Establish close interactions between OIE (Office International des Epizooties) and WHO influenza networks.
- Encourage studies at the human/animal and domestic/wild bird interfaces and provide training to carry out Studies Develop and distribute reagents for identifying influenza viruses of all subtypes and establish the total gene pool among influenza viruses.

Rationale: extension of animal influenza surveillance and integration with human influenza surveillance is essential for understanding and preparing for threats to human health posed by animal influenza viruses.

<b>Leading Article</b>				
Global Agenda on Influenza	1	Non-Human Sources	4	First Identification of Ciprofloxacin Resistance in <i>Salmonella</i> isolated in NZ
<b>Bacteriology</b>	3	<i>Escherichia coli</i>	4	<b>Virology</b>
Legionellosis and Environmental		<b>Antibiotic Resistance</b>	4	Respiratory Viruses
Legionella isolates	3	Antituberculosis-Drug Resistance	4	Adenoviruses
Special Bacteriology	3	Collated Diagnostic Laboratory		Enteroviruses
<b>Enteric Pathogens</b>	3	Antimicrobial Susceptibility Data	4	Norwalk-like Virus
<i>Salmonella</i>	3	Survey of Non-Multiresistant and Multiresistant MRSA	7	<b>Culture Collection</b>
				8

#### 4. Improve data management, utilisation and exchange

Required key actions:

- Encourage communication between surveillance systems and harmonisation of data.
- Improve collation/analysis/dissemination of existing data, including an electronic bulletin board for special announcements.
- Facilitate and support central databases (eg, FluNet and the Los Alamos National Laboratory (LANL) sequence database) for recording epidemiological, virological and genetic information, and for modelling purposes.

Rationale: existing surveillance must be better harmonised and data sets and information must be made more rapidly and widely available in order to maximise their usefulness.

#### B. Increase knowledge on health and economic burden of influenza

##### 1. Capacity strengthening in epidemiological and statistical techniques for studies on influenza disease burden

Required key actions:

- Develop common protocols including case definitions.

Rationale: to enable studies on burden of influenza disease.

##### 2. Evaluation of the clinical and economic burden of disease in countries where there is no recognition of influenza or no control policies are in place

Required key actions:

- Establish comprehensive studies in countries representative of a geographical area and socioeconomic status.
- Conduct additional smaller epidemiological studies for national influenza policy development.

Rationale: to establish the magnitude of influenza as a public health problem. To determine the proportion of acute respiratory infections and febrile illnesses that is due to influenza, and to prioritise influenza in relation to other major infectious diseases.

##### 3. Re-evaluate the clinical and economic burden of influenza in countries where influenza control policies are in place

Required key actions:

- Encourage the evaluation of the burden of disease in different age and risk groups in relation to control policies.
- Provide tools and protocols for evaluating effectiveness of current and alternative strategies for influenza control.

Rationale: the need for better information to sustain and improve influenza control.

#### C. Increase influenza vaccine usage

The recommendations represent a logical but not necessarily sequential progression and support one another in enhancing vaccine coverage.

##### 1. Encourage assessment of disease burden and cost-effectiveness analyses

Required key actions:

- depend on antecedent activities, but considered absolutely essential to improve coverage.

Rationale: required to justify vaccine programmes and establish as a national priority given competing priorities.

##### 2. Encourage countries to establish national policies and set immunisation targets

Required key actions:

- WHO regularly collate and publish policies, immunisation rates and reimbursement mechanisms.

Rationale: each element is an important determinant of vaccination coverage.

##### 3. Promote awareness among policy-makers, health care providers and the public

Required key actions:

- develop, compile and disseminate relevant information, initiate demonstration projects, provide training.

Rationale: an important determinant of vaccination coverage.

##### 4. Encourage countries to identify and develop effective strategies for vaccine delivery

Required key actions:

- develop, compile and disseminate information, initiate demonstration projects, provide training.

Rationale: important determinants of vaccination coverage.

#### 5. Develop and implement methods for measurement and feedback of the progress of national and local programmes

Required key actions:

- develop and standardise approaches to assessing national vaccination rates, rates within target groups and vaccine effectiveness to close the audit loop.

Rationale: measurement and feedback are important determinants of vaccination coverage.

#### D. Accelerate national and international action on pandemic preparedness

##### 1. Increase awareness of the need for pandemic planning

Required key actions:

- Transform scientific message on pandemic preparedness into political action.
- Make pandemic preparedness an agenda item for a future World Health Assembly.

Rationale: authorities must understand the potential impact and threat of pandemic influenza, and thus the importance of pandemic planning and provision of adequate resources to carry it out. With adequate preparation, the morbidity, mortality and social disruption associated with a pandemic should be reduced.

##### 2. Accelerate the development and implementation of national pandemic plans

Required key actions:

- WHO to develop a model national plan and assist with regional planning.
- Develop a tool for country self-assessment of pandemic planning progress.
- Exchange expertise/provide consultations in pandemic planning.
- Publish the progress/status of pandemic planning periodically.

Rationale: WHO published the Pandemic Preparedness Plan in 1999. However, only a few countries have begun pandemic planning. There are many obstacles to planning, especially in developing countries. Providing motivation and assistance will accelerate the planning process and decrease the risk of a world unprepared for the next pandemic.

##### 3. Enhance the utilisation of influenza vaccine and antivirals in the inter-pandemic period

Required key actions:

- Set up a specific working group to develop guidelines for the use of antivirals.
- Enhance the surveillance of antiviral resistance antivirals.
- Provide assistance to countries where there is no existing or limited influenza vaccine manufacturing capacity and there is a wish to produce influenza vaccine.

Rationale: widescale use of antivirals and vaccines during a pandemic will depend on familiarity with their effective application during the inter-pandemic period. The increasing use of these modalities will expand capacity and mitigate the morbidity and mortality of annual influenza epidemics. Studies conducted during the inter-pandemic period can refine the strategies for use during a pandemic.

##### 4. Develop strategies for the utilisation of vaccines and antivirals and securing adequate supplies for a pandemic

Required key actions:

- Develop and rehearse strategies for emergency production, licensing and testing of vaccines and antivirals.
- Develop models and guidelines for the use of vaccines and antivirals when they are in short supply and adjust these at the start of the pandemic.
- Incorporate antiviral stockpiling into pandemic plans.
- Equity of supply between countries should be addressed by a multidisciplinary working group under the auspices of WHO.

Rationale: vaccines will not be available at the start of a pandemic. The only specific intervention possible in the absence of vaccines would be the use of antivirals, but their adequate availability requires stockpiling. It is essential that action is taken to accelerate availability of vaccines and antivirals and to develop guidelines for their use when they are in short supply.

##### 5. Advocate research on pandemic viruses, vaccines, antivirals and other control measures

Required key actions:

- Investigate mechanisms underlying the emergence of pandemic viruses.
- Develop novel vaccines and production strategies/technologies.
- Evaluate the immunogenicity and safety of conventional and novel vaccines.
- Update libraries of seed viruses and vaccine potency reagents.
- Evaluate the effectiveness of antivirals for complications; relative effectiveness and side-effects of M2 and NA inhibitors.
- Feasibility of alternative models of antivirals access.

- Evaluate the effectiveness of community control strategies other than vaccines and antivirals.

Rationale: with more knowledge on pandemic viruses, vaccines, antivirals and other control measures, it will be possible to design more appropriate intervention strategies, and governments will be encouraged to commit resources.

# BACTERIOLOGY

## LEGIONELLOSIS AND ENVIRONMENTAL LEGIONELLA ISOLATES

During January to March 2002, laboratory testing identified 15 sporadic cases of legionellosis, one of which involved the death of a 62 year old male. All cases were confirmed either by the isolation of legionella organisms from the respiratory tract (1 case), or the demonstration of a seroconversion (4 cases), or a four-fold or greater rise in antibody titre (2 cases), or the demonstration of consistently high antibody titres of >256 on two or more occasions (8 cases). All cases had clinical illness consistent with legionellosis. A further two cases were notified on suspicion of legionellosis, but laboratory testing using the indirect fluorescent antibody test could not confirm their legionellosis status.

Table 1. Age and sex distribution of clinical legionellosis cases, January to March 2002

Age (Years)	0-25	26-50	51-75	>75	No. of Cases	Average Age
Male	1	2	8	2	13	59.5
Female	0	0	2	0	2	64.5
<b>Total</b>	<b>1</b>	<b>2</b>	<b>10</b>	<b>2</b>	<b>15</b>	<b>61.3</b>

The causative legionella agent was identified in all of the fifteen cases. The infecting organisms are shown in the table 2. The predominant species causing illness was *Legionella longbeachae*, with a total of eight cases (53.3%) infected with this organism. Handling and using compost was either proven or implicated as the source of infection with these cases.

Table 2. Legionellosis cases and environmental isolates, January to March 2002

Legionella species	Clinical Cases		Environmental Isolates	
	Confirmed	Number	Where isolated – if known	
<i>L. anisa</i>	-	13	11 x potable water system 2 x industrial equipment	
<i>L. bozemanii</i> serogroup 1	1	2	1 x compost 1 x industrial equipment	
<i>L. bozemanii</i> serogroup 2	-	2	1 x compost 1 x industrial equipment	
<i>L. dumoffii</i>	1	0	-	
<i>L. feeleii</i> serogroup 1	-	1	unknown	
<i>L. longbeachae</i> serogroup 1	4	5	5 x compost	
<i>L. longbeachae</i> serogroup 2	1	0	-	
<i>L. longbeachae</i> serogroup unidentified	3	0	-	
<i>L. micdadei</i>	-	3	3 x compost	
<i>L. pneumophila</i> serogroup 1	1	10	1 x cooling tower 9 x industrial equipment	
<i>L. pneumophila</i> serogroup 5	-	2	2 x industrial equipment	
<i>L. pneumophila</i> serogroup 6	-	7	3 - unknown 3 x industrial equipment 1 x cooling tower	
<i>L. pneumophila</i> serogroup 7	-	1	unknown	
<i>L. pneumophila</i> serogroup 8	-	2	unknown	
<i>L. pneumophila</i> serogroup 12	4	0	-	
<i>L. pneumophila</i> serogroup 14	-	2	2 x industrial equipment	
<i>L. santicrucis</i>	-	1	Cooling tower	
<b>Total</b>	<b>15</b>	<b>51</b>		

quarter involved infection with *Legionella pneumophila* serogroup 1. The death involved a housebound 62-year old male and occurred five weeks after admission to hospital with a respiratory complaint and one week after isolation of the legionella from the patient's sputum. The source of the infection, whether nosocomial or domestic, could not be ascertained.

The cases were widespread throughout New Zealand with 2 cases each in the Northland, Hawkes Bay, Wellington, and Canterbury Health Districts; and one case each in the South Auckland, Taranaki, Wanganui, Wairarapa, Nelson-Marlborough, South Canterbury, and Southland Health Districts. During January to March 2002, *Legionella* species were identified in 51 environmental isolates that were either isolated by ESR or referred to ESR for identification. All 51 isolates were identified to the species and serogroup level.

## SPECIAL BACTERIOLOGY

### Interesting Isolates Received in the Special Bacteriology Laboratory

- Brucella* species were received from two patients during the period January to March 2002.

*Brucella* species are slow growing, small gram-negative coccobacilli and give a rapid urease positive reaction. The two cultures were confirmed and speciated at MAF's National Centre for Disease Investigation, Wallaceville, and were identified as:

*Brucella melitensis* from blood of culture M 43y with diskitis. Home-slaughter of a pig in the Auckland area 2-3 weeks prior to illness was suspected to be the source of the infection, but follow-up testing of pigs proved to be negative.

*Brucella abortus* from blood of M 5y who had recently returned from India. Consumption of unpasteurised milk was a possible source of infection.

- Corynebacterium diphtheriae* var *mitis* non-toxigenic strain from sputum of M66y with pneumonia.

### Listeria monocytogenes

Five isolates of *L. monocytogenes* from human cases were referred in the period January to March 2002 (Table 3.) Three of the isolates were from adults who were elderly and/or had underlying illness identified, one case was a premature baby who made a good recovery after antibiotic therapy, and the remaining case was a one-year-old child with fever and diarrhoea.

Table 3. Listeria monocytogenes from human cases, January-March 2002

Month isolated or of onset	Health district	Sex/Age	Source	O antigen serotype
January	Waikato	M 1d	BC	4
February	Canterbury	F 72y	BC	1/2
February	South Auckland	M 77y	Knee aspirate	4
February	Hutt	M 50y	BC	1/2
March	Canterbury	M 1 y	BC	1/2

# ENTERIC PATHOGENS

## SALMONELLA

### Human

During January to March 2002, 887 isolates of Salmonella were received compared with 737 for the same period in 2001. This increase in isolates is in part due to several outbreaks of Salmonellosis.

The first in February involved 29 cases which were traced back to a Bakery which supplied potato topped mince pies to a café. The pies were not reheated to an adequate temperature to kill any *Salmonella* present. The serotype was identified as *Salmonella* species 6,7 : k : -.

The second and third were in the Nelson district in March, 10 cases following a school camp and 58 following a church camp. *S. Typhimurium* phage

type 1 was isolated from all cases but was not isolated from any environmental or food source at either camp.

*S. Typhimurium* phage type 1 was also isolated from 17 cases in Christchurch during March. No common source was determined for these cases.

*S. Weltevreden* 15+ was isolated from a family group of eight people who had shared an umu meal brought from Samoa. The strain was also isolated from palusami (taro in coconut milk, wrapped in taro leaves), which was part of the umu meal.

*S. Typhimurium* phage type 160 cases have remained high over the summer months, representing 43.7% January and 32.9% February of total *Salmonella* isolates compared with 24.1% and 19.1%, respectively in 2001.

### Non-Human

There were 287 isolates of *Salmonella* during January-March compared with 306 for the same period in 2001.

The predominant isolates were *S. Hindmarsh* in sheep (43) and *S. Typhimurium* phage type 160 in poultry environmental isolates (39).

There were 64 isolates of *S. Typhimurium* phage type 160 compared with 31 in 2001, including isolates from cattle, dogs, cats, horses, alpaca, birds, and shellfish.

## ESCHERICHIA COLI

There were 13 isolates of *E. coli* O157 confirmed during January to March compared with 18 for the same period in 2001.

Table 5. Isolates of *E. coli* O157, January-March 2002

Month	Sex / Age	District	Comments
January	F 70	New Plymouth	Bloody diarrhoea
January	M 9m	Waikato	Diarrhoea
January	M 76	Auckland	None given
January	M 3	Canterbury	None given
January	M 2	Tauranga	None given
January	F 53	Canterbury	None given
January	F 12	Wellington	Colitis
January	F 7	Rotorua	Abdominal pain
February	M 9m	Waikato	Blood in stools
February	M 73	Auckland	Diarrhoea and vomiting
February	M 6	Canterbury	None given
March	M 66	Wellington	Bloody diarrhoea
March	F 1	Waikato	None given

## ANTIBIOTIC RESISTANCE

### ANTITUBERCULOSIS-DRUG RESISTANCE, 2001

The national surveillance of antituberculosis-drug resistance is based on the results of susceptibility testing of isolates in the Mycobacteriology Reference Laboratories at Auckland, Wellington and Waikato Hospitals. These laboratory data are matched with tuberculosis notifications. In 2001, 377 cases of tuberculosis were notified. The causative organism was iso-

Table 6. Resistance among isolates from tuberculosis cases, 2001

Antimicrobial	Number tested	Number resistant <sup>1</sup>	Percent resistance <sup>1</sup>
Isoniazid	294	18	6.1
Rifampicin	294	1	0.3
Ethambutol	294	2	0.7
Pyrazinamide	294	8 <sup>2</sup>	2.7
Streptomycin	294	19	6.5

<sup>1</sup> includes resistance alone or in combination with other antimicrobials

<sup>2</sup> includes the six *M. bovis* isolates, which are intrinsically resistant to pyrazinamide

lated and identified from 294 (78.0%) cases. Antimicrobial susceptibility testing results were available for all 294 isolates, which comprised 288 *Mycobacterium tuberculosis* and six *M. bovis* isolates.

Resistance to each antimicrobial tested is shown in Table 6.

The majority (86.4%) of isolates were susceptible to all five antimicrobials tested (Table 7). None of the isolates were multidrug resistant, that is, resistant to at least isoniazid and rifampicin.

Table 7. Resistance patterns among isolates from tuberculosis cases, 2001

	Number (%)	Resistance pattern <sup>1</sup>	Number (%) with each pattern
Fully susceptible	254 (86.4)		
Resistant to 1 agent	33 (11.2)	H	12 (4.1)
		Z	7 (2.4) <sup>2</sup>
		S	13 (4.4)
		E	1 (0.3)
Resistant to 2 agents	6 (2.0)	HS	5 (1.7)
		RE	1 (0.3)
Resistant to 3 agents	1 (0.3)	HZS	1 (0.3)

<sup>1</sup> H, isoniazid; Z, pyrazinamide; S, streptomycin; E, ethambutol; R, rifampicin

<sup>2</sup> includes the six *M. bovis* isolates, which are intrinsically resistant to pyrazinamide

Among the 294 isolates tested, 272 (92.5%) were from new cases of tuberculosis and 22 (7.5%) from reactivations. There were no significant differences in resistance among new cases and reactivations.

## COLLATED DIAGNOSTIC LABORATORY ANTIMICROBIAL SUSCEPTIBILITY DATA, 2001

A record number of laboratories (28) submitted their antimicrobial resistance data for 2001. These data were collated to provide estimates of national rates of resistance, which are shown in Table 8.

Notably the 2001 data indicated:

- Escherichia coli* from bacteraemia: data for *E. coli* from bacteraemia were collected for the first time. Resistance to cefotaxime/ceftriaxone recorded at 0.6%, gentamicin at 1.1%, and no imipenem resistance.
- E. coli* urinary: resistance to the newly reintroduced mecillinam at 5.1%. There was little apparent change between 2000 and 2001 in resistance to trimethoprim (22.5% in 2001), co-amoxiclav (10.0%), nitrofurantoin (1.5%) or fluoroquinolones (1.6%).
- Klebsiella*: 1.4% cefotaxime/ceftriaxone resistance.
- Pseudomonas aeruginosa*: little change between 2000 and 2001 in resistance to gentamicin (11.9% in 2001), ceftazidime (3.4%) or fluoroquinolones (8.9%). Imipenem resistance increased from 5.2% in 2000 to 7.3%.
- Enterococcus*: 0.7% of 2718 isolates were reported as vancomycin resistant, which equates to 19 isolates. In contrast, vancomycin-resistant *E. faecium* or *E. faecalis* from just six patients were referred to ESR for confirmation during 2001.
- Neisseria gonorrhoeae*: 9.7% fluoroquinolone resistance.
- Streptococcus pneumoniae*: the laboratories that distinguished between penicillin resistance (MIC  $\geq 2.0$  mg/L) and intermediate resistance (MIC 0.12-1.0 mg/L), reported 11.2% resistance and 9.5% intermediate resistance, a total 20.7% penicillin nonsusceptibility. In contrast, the laboratories which did not distinguish between intermediate resistance and resistance reported 28.3% penicillin nonsusceptibility. 10.8% cefotaxime/ceftriaxone resistance (MIC  $\geq 2.0$  mg/L), which is high relative to the prevalence of penicillin resistance (11.2%). This effect may be due to penicillin-nonsusceptible isolates being selectively tested for cefotaxime susceptibility.
- Staphylococcus aureus*: no apparent increase in methicillin resistance in 2001, with 6.9% resistance recorded in 2000 and 6.8% in 2001. Similarly, there was little change in mupirocin resistance (21.5% in 2000 and 19.7% in 2001). Fluoroquinolone resistance increased from 5.2% in 2000 to 9.7%.



Table 8. Antimicrobial resistance data from hospital and community laboratories, 2001<sup>1</sup>

	Percentage resistant (number tested <sup>2</sup> )																	
	amikacin	ampicillin	cefazidime	ceftriaxone/cefotaxime	cefuroxime/cefamandole	cephalothin	co-amoxiclav	co-trimoxazole	fluoroquinolone	gentamicin	imipenem/meropenem	meclizolam	nitrofurantoin	piperacillin	tetracycline	ticarcillin	tobramycin	trimethoprim
<i>Acinetobacter</i> spp. <sup>3</sup>	9.0 (133)		22.3 (211)					18 (600)	17.7 (651)	13.7 (671)	2.0 (346)			39.3 (219)			9.7 (155)	
<i>Citrobacter freundii</i> <sup>4</sup>		92.8 (797)		23.0 (152)	22.0 (118)	82.8 (128)	49.0 (708)	10.9 (348)	3.6 (701)	2.6 (272)	0 (146)							
<i>Enterobacter</i> spp. <sup>3</sup>	0.1 (733)	95.3 (2267)		20.9 (1039)	45.1 (938)	95.8 (927)	84.5 (2196)	7.1 (1540)	4.6 (1641)	4.3 (1535)	0.5 (1027)			9.7 (349)			4.7 (337)	
<i>Escherichia coli</i> from bacteraemia	0 (352)	55.6 (1014)		0.6 (818)	2.8 (823)	28.9 (554)	20.9 (858)		2.7 (969)	1.1 (1015)	0 (489)						2.2 (229)	
<i>E. coli</i> urinary	0 (4384)	55.4 (77932)		0.6 (6228)	2.2 (4761)	19.8 (10945)	10.0 (76164)	21.3 (6670)	1.6 (83600)	1.0 (15726)		5.1 (2866)	1.5 (83481)				1.0 (2109)	22.5 (84517)
<i>Klebsiella</i> spp.	0.2 (895)	96.8 (5316)		1.4 (1598)	8.3 (1530)	11.4 (1565)	9.8 (4650)	7.5 (2821)	2.8 (4460)	0.6 (2866)	0.2 (1607)			6.2 (468)			1.1 (551)	
<i>Morganella morganii</i> <sup>2</sup>	1.3 (152)	97.1 (712)		5.0 (260)	85.2 (256)	99.3 (274)	95.3 (681)	12.9 (403)	9.1 (539)	11.8 (449)	3.3 (242)							
<i>Proteus mirabilis</i>	0.2 (565)	13.6 (5815)		0.3 (1147)	1.9 (827)	5.1 (1387)	2.8 (5578)	9.8 (2379)	0.9 (4822)	1.3 (2100)	0.2 (869)			98.5 (272)			1.5 (336)	
<i>Pseudomonas aeruginosa</i>	10.0 (1116)		3.4 (5506)						8.9 (8717)	11.9 (9337)	7.3 (2519)			3.5 (5095)		16.0 (605)	3.8 (4203)	
<i>Serratia</i> spp. <sup>3</sup>	0.8 (367)	94.4 (1837)		14.8 (723)	89.8 (697)	97.9 (579)	87.6 (1745)	10.2 (1230)	14.9 (1298)	3.2 (1205)	0.2 (625)			64.1 (167)			2.4 (248)	

	Percentage resistant (number tested <sup>2</sup> )													
	ampicillin	cefotaxime	clindamycin	co-amoxiclav	co-trimoxazole	erythromycin	fluoroquinolone	gentamicin	methicillin/oxacillin	mupirocin	nitrofurantoin	penicillin	tetracycline	vancomycin
Coagulase-negative Staphylococci (blood isolates)			22.5 (905)		33.1 (1159)	42.1 (2056)	22.9 (1172)	38.0 (1588)	54.5 (2067)			84.7 (2019)	9.6 (1159)	0 (1378)
<i>Campylobacter</i> spp.						1.2 (342)	2.9 (279)							
<i>Enterococcus</i> spp.	2.8 (8418)							17.6 <sup>4</sup> (739)			1.1 (7028)		62.6 (1478)	0.7 (2718)
<i>Haemophilus influenzae</i> (non-invasive) <sup>5</sup>	21.0 (11624)			0.7 (6237)	17.0 (9098)								1.5 (6196)	
<i>Moraxella catarrhalis</i>	93.6 (1415)					2.4 (1011)							2.4 (1217)	
<i>Neisseria gonorrhoeae</i>							9.7 (906)					6.4 (990)	19.4 (509)	
<i>Staphylococcus aureus</i>			2.4 (6006)		0.8 (55969)	12.2 (75491)	9.7 (13625)	1.7 (22178)	6.8 (82600)	19.7 (28033)		88.1 (75771)	2.8 (56548)	
<i>Streptococcus pneumoniae</i> (non-invasive) <sup>5</sup>		10.8 <sup>6</sup> (1076)			40.2 (3765)	18.2 (4875)						11.2 <sup>7</sup> (2542)	13.6 (2535)	
<i>Streptococcus pyogenes</i>						1.3 (15433)						0 (15504)		

<sup>1</sup> data supplied by Auckland, Christchurch, Gisborne, Hawkes Bay, Middlemore, North Shore, Rotorua, Southland, Taurarunui, Waikato, Wairau, Wanganui, Wellington, Whakatane and Whangarei Hospitals; and Auckland Diagnostic Medical, Auckland Southern Community, Christchurch Southern Community, Dunedin Southern Community, Medlab Bay of Plenty, Medlab Central, Medlab South, Medlab Wellington, Nelson Diagnostic, Rotorua Diagnostic, Taranaki Medlab, Valley Diagnostic and Wanganui Diagnostic laboratories

<sup>2</sup> data presented only if available for  $\geq 100$  isolates

<sup>3</sup> ESCAPPM organisms with potential for inducible cephalosporinases and stably derepressed mutants producing high levels of cephalosporinases

<sup>4</sup> high-level resistance

<sup>5</sup> susceptibility of isolates from invasive disease tested at ESR and reported in LabLink 2002; 9(1): 11.

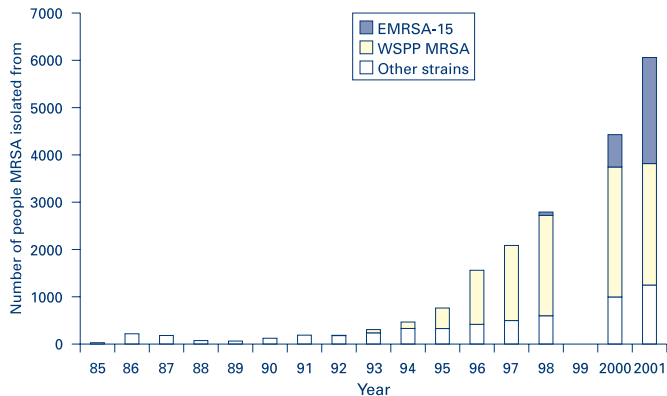
<sup>6</sup> cefotaxime/ceftriaxone resistance (MIC  $\geq 2.0$  mg/L)

<sup>7</sup> penicillin resistance (MIC  $\geq 2.0$  mg/L)

## SURVEY OF NON-MULTIRESISTANT AND MULTIRESISTANT MRSA, AUGUST 2001

The second annual survey of all MRSA (non-multiresistant and multiresistant) was held during August 2001. Over the one-month period, MRSA were referred from 504 people (485 patients and 19 staff). This number of referrals equates to an annual rate of 161.8 per 100,000; a 32% increase on the rate in 2000 (122.4 per 100,000) (Figure 1). MRSA was reported to be causing infection in 88.0% of the 400 people for whom this information was reported.

Figure 1. MRSA isolations, 1985-2001



Among the 485 patients with MRSA, 50.3% were categorised as hospital patients and the other 49.7% as community patients (see LabLink 2000; 8(1): 10 for the definitions of hospital and community patients).

The majority of the MRSA isolates were either WSPP MRSA (42.5%) or EMRSA-15 (37.1%). Over the last 2-3 years, an increasing proportion of the isolates have been the EMRSA-15 strain. Correspondingly, the proportion that are WSPP MRSA has decreased (Figure 1). The majority (72.9%) of WSPP MRSA continue to be isolated from people in the community, whereas most (75.9%) EMRSA-15 are isolated from hospital patients or staff (Table 9).

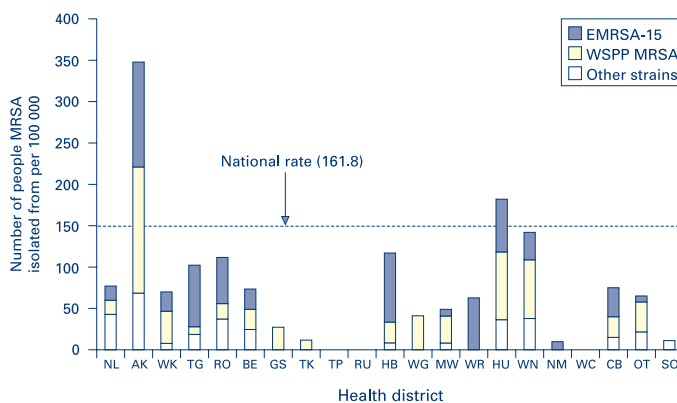
Table 9. Distribution of WSPP MRSA and EMRSA-15 among hospital patients/staff and community patients, August 2001

	Number of people with:	
	WSPP MRSA	EMRSA-15
Hospital patient or staff	58 (27.1%) <sup>1</sup>	142 (75.9%)
Community patient	156 (72.9%)	45 (24.1%)
<b>Total</b>	<b>214 (100%)</b>	<b>187 (100%)</b>

<sup>1</sup> proportion of all isolations of the strain

The geographic distribution of people from whom MRSA was isolated during August 2001 is shown in Figure 2.

Figure 2. Annualised incidence of MRSA by health district, 2001



Due to the increase in the prevalence of multiresistant strains, in particular EMRSA-15, the proportion of MRSA which are multiresistant increased from 25.3% in 2000 to 45.0% in 2001. While most EMRSA-15 isolates are multiresistant to ciprofloxacin and erythromycin, 11.8% were erythromycin susceptible and therefore not categorised as multiresistant. WSPP MRSA remain predominantly non-multiresistant (Table 10).

Table 10. Resistance among MRSA referred during August 2001

Antimicrobial agent (resistance breakpoint, mg/L)	Percentage resistance		
	All isolates (n = 504) <sup>1</sup>	WSPP (n = 212)	EMRSA-15 (n = 186)
Chloramphenicol (MIC ≥32)	0.4	0.5	0
Ciprofloxacin (MIC ≥4)	44.6	0	100
Clindamycin (MIC ≥4)	10.1	2.4	6.5 <sup>2</sup>
Co-trimoxazole (MIC ≥4/76)	5.4	0	0
Erythromycin (MIC ≥8)	45.2	6.6	88.2
Fusidic acid (MIC ≥2)	7.1	1.4	3.8
Gentamicin (MIC ≥16)	6.4	0	0.5
Mupirocin (MIC ≥8) <sup>3</sup>	6.8	2.8	1.0
High-level mupirocin (MIC ≥512)	5.0	1.4	0.5
Rifampicin (MIC ≥4)	1.0	0.5	1.0
Tetracycline (MIC ≥16)	9.1	0.9	1.0
Multiresistance <sup>4</sup>	45.0	1.4	88.2

<sup>1</sup> includes isolates of WSPP MRSA and EMRSA-15, and 106 isolates of other strains

<sup>2</sup> EMRSA-15 exhibits inducible clindamycin resistance by a disc approximation test

<sup>3</sup> includes low-level (MIC 8-256 mg/L) and high-level (MIC ≥512 mg/L) mupirocin resistance

<sup>4</sup> resistance ≥2 classes of antibiotics in addition to β-lactams

## FIRST IDENTIFICATION OF CIPROFLOXACIN RESISTANCE IN SALMONELLA ISOLATED IN NEW ZEALAND

A ciprofloxacin-resistant *Salmonella* was isolated in January 2002. This is the first time ciprofloxacin resistance has been identified among the *Salmonella* routinely monitored by ESR for antimicrobial resistance.

The isolate, *S. Typhimurium* phage type 12a, was multiresistant to ampicillin, ciprofloxacin (MIC 16 mg/L), chloramphenicol, co-trimoxazole, gentamicin, streptomycin, sulphonamides, tetracycline and trimethoprim. The patient was a child who had recently been in China.

## VIROLOGY

### RESPIRATORY VIRUSES

#### Influenza virus

During January to March 2002, eight isolations of influenza viruses were reported from Christchurch (1), Waikato (1), and Auckland (6). Influenza isolations in 2002 were lower compared with 14 isolations of influenza viruses during the same period in 2001. Seven isolations of influenza A and one isolation of influenza B were reported. Four influenza A isolates from Auckland (3) and Waikato (1) were further subtyped as A/Moscow/10/99 (H3N2)-like viruses. One influenza A isolate from Auckland was subtyped as A/New Caledonia/20/99 (H1N1)-like virus. The influenza B isolate was further identified as B/Sichuan/379/99-like virus. The 2002 influenza vaccines should provide good protection against current circulating influenza strains.

#### Respiratory Syncytial Virus and Rhinoviruses

During January to March 2002, eight cases of respiratory syncytial viruses (RSV) were reported from Christchurch (2), Dunedin (1), Waikato (3) and Wellington (2). The cases of RSV infections in 2002 were higher compared

with four cases of RSV during the same period in 2001. Four isolations of rhinoviruses were reported from Waikato (3) and ESR (1). The isolation in 2002 was lower compared with 9 isolations of rhinoviruses during the same period in 2001.

## ADENOVIRUSES

During January to March 2002, a total of 44 adenoviruses were serotyped. This is lower than the 53 adenovirus isolations during the same period of 2001. Adenovirus type 3 was isolated from 17 patients from Auckland (11), Christchurch (2) and Waikato (4). The specimens from these patients were taken between 13 December 2001 and 7 March 2002. The patients ranged in age from 6 months to 39 years (average 18.8 years). Seven isolations of adenovirus type 7 were reported from Auckland (3), Waikato (2), Christchurch (1), and Dunedin (1). The remaining 20 adenoviruses were typed as adenovirus type 2 (4), type 1 (1), type 4 (1), type 21 (1), type 22 (1), type 24 (1) and untypable (11).

## ENTEROVIRUSES

### Echovirus type 13 outbreak

In the previous issue of Lablink (2002, March 9(1):13-14), the outbreak caused by Echovirus type 13 was described for the period from 27 February 2001 to 24 January 2002. This report describes the outbreak during 25 January 2002 to 3 April 2002 (the end of the outbreak). A total of 35 E13 isolates were identified from Waikato (11), Wellington (12), Otago (9), Christchurch (2) and Manawatu (1). The outbreak peaked in January and then declined as indicated by the monthly isolations at the end of January (23), February (5), March (6) and April (1). Patients ranged in age from 13 days to 37 years (average 3.5 years). Main symptoms included rash, fever, headache and viral meningitis.

### Echovirus type 30

During January to March 2002, a total of 23 isolates of echovirus type 30 (E30) were identified from Otago (21), Waikato (1), and Auckland (1). It appears that this is the continuation of the E30 outbreak from November 2001 with the isolations in January (16), February (4) and March (3). The outbreak occurred mainly in the Otago health district. The patients ranged in age between 11 years to 39 years old (average 33). It is very interesting to note that this outbreak peaked in an age group of 19 to 39 years, whereas most of enterovirus outbreaks peak in the age group of 0 to 19 years. The clinical symptoms included rash, fever to meningitis.

## NORWALK-LIKE VIRUS

### Characterisation of Norwalk-like virus (NLV) strains from gastroenteritis outbreaks occurring between January and June 30 2002.

There were 46 outbreaks or clusters of laboratory-confirmed NLV-associated gastroenteritis to 30 June 2002. As in 2001, there was no seasonal peak in 2002, with outbreaks being reported during all months. Twenty-nine of the 46 outbreaks (63%) occurred in April, May and June. Seven institutional outbreaks in rest homes and hospitals were reported; four of these were caused by the Lordsdale virus 'global strain cluster', GII/1,4,8. Three outbreaks occurred in children's play/child care centres, two in school camps and one in a school hostel. These outbreaks demonstrate the extensive spread of NLVs through institutionalised settings. Other settings included restaurants, cafes, takeaway bars and catered functions, and several family groups around the country. The extent of NLV infection originating in the home is unknown.

A wide range of genotypes has circulated during the January to June period. The predominant genotype was the common 'Global strain', GII/1,4,8 (19/46 outbreaks, 41.3%). This genotype was identified in a range of settings. The other common genotypes were GII/2 (Melksham virus)

and GII/6,7,9 (Napier, Florida and Gwynedd viruses). Genogroup I strains GI/3, (Desert Shield virus) and GI/4, ('Cruise Ship virus') are still circulating in New Zealand. A strain of Mexico virus was associated with consumption of Korean oysters. This genotype has been linked with several oyster-related outbreaks in previous years but has not been identified in New Zealand since December 2000. White River virus (GII/5), previously identified once before in 1997, was identified in a single case. Two distinct NLV strains were identified in three outbreaks. For the majority of outbreaks, person to person transmission was the likely transmission route, with food and food handling implicated in at least nine outbreaks.

Table 11. NLV Genotypes occurring between January and June 2002

NLV Strain	Genotype	Number of Outbreaks
Lordsdale virus 'Global strain' cluster	GI/1,4,8	19
Napier / Florida / Gwynedd / Idaho Falls virus cluster	GII/6,7,9	7
Melksham virus	GII/2	9
Mexico virus	GI/3	1
White River virus	GII/5	1
'Cruise ship virus'	GI/4	2
Desert Shield virus	GI/3/3b	2
Genogroup II, possible new subtype	GII	2
Identification in progress	Unknown	6
<b>Total</b>		<b>49*</b>

\* Two distinct NLV strains were found in three outbreaks

## CULTURE COLLECTION

Recent accessions to the Collection are shown in Table 12.

Table 12. NZRM new accessions

Name	NZRM No.	Source, Strain	Comments
<i>Buttiauxella agrestis</i>	4043	NZ isolate, 2001	Wound, human
<i>Enterococcus faecalis</i>	4061	Strain POW 1994	Control strain for CDS method of antibiotic susceptibility testing
<i>Haemophilus influenzae</i>	4059	NCTC 11315	Beta-lactamase producer. Used in CDS method of antibiotic susceptibility testing
<i>Pseudomonas aeruginosa</i>	4034	NZ isolate, 2000	MIC (mg/L) ceftazidime 256.0; piperacillin >256.0. Useful as a control culture in multiple combination bactericidal testing (MBCT)
<i>Staphylococcus aureus</i>	4055	NCTC 10442	MRSA
<i>Streptococcus pneumoniae</i>	4060	Strain ARL 10582	Control strain for CDS method of antibiotic susceptibility testing

### Safety in Microbiology Laboratories

The revised standard, AS/NZS 2243.3:2002 *Safety in laboratories - Microbiological aspects and containment facilities*, has recently been released. This edition, which replaces the 1995 edition, revises the requirements, responsibilities and general guidelines for laboratories dealing with infectious disease, and the classification of microorganisms into the four risk groups. It contains descriptions of the four levels of physical containment for microbiology laboratories and laboratories dealing with genetically modified organisms.

This useful and important standard may be ordered from Standards New Zealand. Tel 04 498 5991; Fax 04 498 5994, e-mail snz@standards.co.nz, website www.standards.co.nz

ESR LabLink is produced quarterly by the Institute of Environmental Science & Research Limited (ESR),  
Kenepuru Science Centre, Kenepuru Drive, PO Box 50-348, Porirua, Wellington.  
Telephone: 04-914 0700, Facsimile: 04-914 0770.

Editorial Team: Philip Carter, Sue Huang, Carolyn Nicol, Elizabeth Sneyd.

The content of this publication does not necessarily reflect ESR or Ministry of Health policy.