37th year of publication

Epidemiological News Bulletin

QUARTERLY

APRIL - JUNE 2011 VOL. 37 NO. 2 A PUBLICATION OF THE MINISTRY OF HEALTH, SINGAPORE

CONTENTS

Suggested citation:
Ministry of Health, Singapore. [Article title]. Epidemiol News Bull [Year]; [Vol]:[inclusive page numbers]

MOH Weekly Infectious Diseases Bulletin
http://www.moh.gov.sg/mohcorp/statistic-sweeklybulletins.aspx

Risk analysis on the Fukushima nuclear incident

Background

A 9.0 magnitude earthquake and a powerful tsunami battered the north-east coast of Honshu, Japan on 11 March 2011, leaving more than 28,000 dead or missing. The tsunami destroyed the power supply and cooling systems of reactors at the Fukushima Daiichi nuclear power plant, which led to explosions and release of radionuclides into the environment, resulting in a nuclear emergency of worldwide concern.

The Japanese government ordered the evacuation of nuclear plant workers and residents living within 20-km radius of the Daiichi plant and 10-km radius of the Daini plant. Some 270,000 people were evacuated and potassium iodine tablets were distributed to the evacuees. In addition, people living between the 20-km and 30-km radius of the Daiichi nuclear plant were advised to stay indoors or voluntarily leave the area.

Japan's Nuclear and Industrial Safety Agency (NISA) initially rated the nuclear accident as level 4 based on the International Nuclear and Radiological Event Scale (INES)[1] of 1 to 7. The rating was revised to level 5 on 18 March, and subsequently upgraded to level 7 on 12 April, the same level as the Chernobyl accident, when it was retrospectively estimated that the Fukushima plant had released 10,000 terabecquerels of radioactive material per hour in the early hours after the explosion of the Fukushima reactors.[2]

In response to the nuclear event, the international community imposed a series of measures including travel alerts and advisories against travel to Japan, especially the affected areas, the testing and

ISSN 0218-0103
http://www.moh.gov.sg/mohcorp/publicationsnewsbulletins.aspx

restriction of import of goods from Japan, as well as monitoring of ambient levels of radiation.

Radioactive contamination in Japan

Radioactive contamination in the environment (air, sea water, soil) and essential supplies (drinking water and food) was subsequently detected in Japan, which triggered concerns about the health effects of radiation. Various measures to monitor environmental radiation and ensure food and water safety were implemented by the Japanese government.

On 16 March, the Japan's Nuclear Safety Commission recommended that local authorities instruct evacuees leaving the 20-km area to ingest stable (not radioactive) iodine.[3] Stable iodine pills and syrup (for children) were made available at evacuation centres. The Japanese authorities also conducted examinations of the thyroid glands of 900 children (comprising newborns and children up to 15 years of age) in Kawamata town and Iitate village in the Fukushima prefecture.[4] None of them had exposure readings exceeding the safety level.

The health consequences from Fukushima incident remain to be seen, as the event is on-going and the radiation exposure seems to be continuing at low levels. Studies of the 1986 Chernobyl accident demonstrated that measures such as the intake of iodine pills immediately following the accident (based on the experience in Poland), as well as restriction on the intake of local milk in the immediate aftermath of the accident, could effectively reduce the risk of thyroid cancer.[5] Such measures have been implemented by the Japanese government. The Fukushima prefectural government is planning to conduct regular health checkups for its residents, and monitor the long-term health of the children.[6]

International reaction

The World Health Organization (WHO) said on 13 April there was no need for new public health measures to counter the higher levels or radiation emitted from the Fukushima nuclear plant at the moment.[7] This was because the measures already taken by Japanese government, including the enforcement of an evacuation zone, the relocation of nearby residents and early distribution of potassium iodine pills, were appropriate, although studies would have to be conducted over the next 10 to 20 years to survey for radiation-associated health effects and any related public health issues.

The United Nations agencies (WHO, International Atomic Energy Agency, World Tourism Organization, World Meteorological Organization, International Maritime Organization, International Civil Aviation Organization, International Labour Organization) have been closely monitoring the effects of the Fukushima Daiichi plant incident. Although the radioactive materials have undoubtedly spread into the global atmosphere, these agencies, as of 15 April, remained confident that radiation levels were extremely low that do not present health or transportation safety hazards to passengers and crew.[8]

A number of countries have detected radioactive contamination in their environment and in food products imported from Japan, but at low levels which posed no threat to human health. As of 30 April, Japan's Foreign Ministry said more than 50 countries and territories had banned or restricted food and other products from Japan.[9] Many countries (e.g. Canada,

China and South Korea) have also stepped up their measures in radiation monitoring.

Neighbouring countries which are in earthquake prone areas with nuclear power plants have been proactive in implementing pre-emptive measures to ensure safety at their own facilities, including China and South Korea. The nuclear accident at Fukushima has renewed concerns over the rush by developing countries to join the so-called 'nuclear renaissance' without the necessary infrastructure, personnel, regulatory frameworks and safety culture. The past decade has seen increasing enthusiasm for nuclear energy to meet burgeoning energy demands, enhance energy security and reduce greenhouse gas emissions.

Measures in Singapore

Singapore is located more than 5000 km away from the incident site in Japan. The National Environment Agency (NEA) has assessed that in view of the distance between the two countries, there is minimal risk of Singapore being affected by the radiological plume.[10] The NEA has been tracking the impact on Singapore's radiation levels through its radiation monitoring stations. No abnormality has been detected so far. Singapore's national water agency, PUB, has stepped up monitoring of radioactivity level in its water supplies. No abnormality has been detected, and levels remain well within the safety levels stipulated in the WHO Drinking Water Guidelines. The tap water in Singapore is safe for drinking.

The Agri-Food & Veterinary Authority (AVA) has stepped up its surveillance to test all food products from Japan for radioactivity soon after the Fukushima nuclear incident. Samples of food products imported from Japan were first tested positive for radioactive contamination above Codex guidelines[11] on 23 March and the last positive result was on 2 April 2011. All radioactive contaminated food had been appropriately destroyed and the food that is available in the local market is safe for consumption. AVA lifted the suspension of fruits and vegetables from Shizuoka and Hyogo prefectures with effect from 16 May 2011.[12] This followed investigations by the Japanese authorities, which ascertained that the samples of contaminated vegetables were not from these two prefectures, but from the Saitama and Ibaraki prefectures instead, and the exporters had wrongly declared the origins of the vegetables. To facilitate the traceability of the source of food imported from Japan, Japan started issuing certificates of origin to accompany food products exported to other countries, including Singapore.

The Ministry of Health (MOH) has assessed the risk of immediate health impacts to Singaporeans who were located outside the evacuation zone to be negligible.[11] Singaporeans returning from the evacuation zone who felt unwell were advised to seek medical advice at the emergency department of their nearby public restructured hospital upon return to Singapore for medical consultation. To date, no cases of radiation exposure have been detected and there have been no admissions related to radiation injuries.

The Ministry of Foreign Affairs (MFA) issued an advisory against non-essential travel to Japan on 17 March 2011.[13] Nearly 2 months after the 11 March earthquake, while MFA no longer advises against non-essential travel to Japan, Singaporeans are advised to continue to avoid travel to the coastal areas of Fukushima, Miyagi and Iwate prefectures, which continue to suffer infrastructural disruptions following the 9.0-magnitude quake, tsunami and

threatened nuclear crisis.[14] Travellers are also advised to strictly observe the 30 km exclusion zone around the power plant imposed by the Japanese government, and refrain from going within 80 km of it.

Singapore's response under International Health Regulations (2005)

In response to the drastic increase in international travel and trade, and emergence and reemergence of international disease threats and other health risks, 194 countries have agreed to implement the WHO's International Health Regulations (2005) (IHR). [15] This binding instrument of international law came into effect on 15 June 2007. The stated purpose and scope of the IHR are "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." Under the Regulations, Member States must notify the WHO Secretariat of public-health emergencies of international concern (PHEICs), and better identify and respond to these events.

The IHR mechanism has served as an effective channel for Singapore to engage in regional cooperation concerning core capacity in various areas and information sharing. Under IHR (2005), MOH facilitated timely sharing of information with the WHO and the National Focal Point (NFP) of relevant countries, when samples of food products imported from Japan were found to test positive for radioactive contamination at levels above the Codex guidelines. The NFP network proved to be useful in tracking exported food items from Japan which were found to be contaminated with radioactive material.

Comments

Although the magnitude of the Fukushima Daiichi nuclear incident is rated at level 7, the highest on the International Nuclear and Radiological Event Scale (INES) ("major release of radioactive material with widespread health and environmental effects requiring implementation of planned and extended countermeasures"), the amount of damage it has caused so far is comparatively less than the Chernobyl incident. In addition, the amount of radioactive material released by this incident is estimated to be about 10% of that released at Chernobyl. While the situation at the nuclear plant remains serious, there are signs of recovery.

Environmental radioactivity in air, soil and seawater in Japan is reported to be generally declining. Restrictions on the shipment of implicated food from affected prefectures have also been gradually lifted as radioactive contaminations fall below regulatory limits, or become undetectable. Although the threat of another devastating earthquake and/or tsunami that could seriously jeopardise current efforts to control the nuclear situation remains, the Japanese are actively putting in place measures to pre-empt and mitigate their impact, e.g. sandbagging the shoreline at the plant and building a breakwater on the shoreline.

These developments are reassuring, and a good indication that even if the situation were to deteriorate, Singapore should have sufficient lead time to prepare. The Fukushima nuclear incident has underlined the importance of maintaining core capacity in risk communication and radiation emergencies, and highlighted the importance of preparedness against nuclear events, early warning, as well as the prompt response and implementation of measures to minimise and mitigate potential health impacts of nuclear radiation.

(Contributed by: Public Health Intelligence Branch, Epidemiology and Disease Control Division, Ministry of Health)

References

1. *International Atomic Energy Agency. The International Nuclear and Radiological Event Scale (INES). Available from http://www-ns.iaea.org/tech-areas/emergency/ines.asp (Accessed on 13 March 2011).*
2. *International Atomic Energy Agency. IAEA briefing on Fukushima nuclear accident (12 April 2011, 14:30 UTC). Available from http://www.iaea.org/newscenter/news/2011/fukushima120411.html (Accessed on 13 March 2011).*
3. *International Atomic Energy Agency. Fukushima nuclear accident update (19 March 2011 12:00 UTC) – Corrected. http://www.iaea.org/newscenter/news/2011/fukushima190311.html (Accessed on 22 March 2011).*
4. *Prime Minister of Japan and His Cabinet. Press conference by the Chief Cabinet Secretary, dated 3 April 2011. http://www.kantei.go.jp/foreign/incident/110403_1504.html (Accessed on 24 May 2011).*
5. *World Health Organization. Health effects of the Chernobyl accident and special health care programmes - Report of the UN Chernobyl Forum Expert Group "Health" 2006. Available from http://www.who.int/ionizing_radiation/chernobyl/WHO%20Report%20on%20Chernobyl%20Health%20Effects%20July%2006.pdf (Accessed on 24 May 2011).*
6. *Japan Today. "Periodic health checkups likely for Fukushima residents" dated 23 April 2011. Available from http://www.japantoday.com/category/national/view/periodic-health-checkups-likely-for-fukushima-residents (Accessed on 24 May 2011).*
7. *International Center for Innovation in Technology. "No new measures needed to counter radiation health risks in Japan" dated 13 April 2011. Available from http://japanstrategicinitiative.wordpress.com/2011/04/13/no-new-measures-needed-to-counter-radiation-health-risks-in-japan/ (Accessed on 24 May 2011).*
8. *World Tourism Organization (UNWTO). Press release "UN agencies: current situation poses no risk to travel to and from Japan" dated 15 April 2011. Available from http://unwto.org/en/press-release/2011-04-15/un-agencies-current-situation-poses-no-risk-travel-and-japan (Accessed on 24 May 2011).*
9. *NHK World (Japan Broadcasting Corporation). "Japan steps up efforts to prove its food safe" dated 30 April 2011. Available from http://www3.nhk.or.jp/daily/english/30_02.html (Accessed on 24 May 2011).*
10. *Singapore Government Statement On Fukushima Nuclear Incident. Joint news release by NEA, MICA, MFA, MOH. MHA, AVA and CAAS "Public urged not to be unduly alarmed – Minimal risk of a radiological plume over Singapore" dated 15 March 2011. Available from http://app2.nea.gov.sg/news_release.aspx?year=2011 (Accessed on 24 May 2011).*
11. *Codex Secretariat. Fact Sheet on "Codex guideline levels for radionuclidies in foods contaiminated following a nuclear or radiological emergency" dated 2 May 2011. Available from http://www.fao.org/crisis/27242-0bfef658358a6ed53980a5eb5c80685ef.pdf (Accessed on 24 May 2011).*
12. *Agri-Food & Veterinary Authority. Press release "AVA lifts suspension on import of fruits and vegetables from Hyogo and Shizuoka prefectures" dated 16 May 2011. Available from http://www.ava.gov.sg/NewsEvents/PressReleases/ (Accessed on 24 May 2011).*
13. *Ministry of Foreign Affairs. "MFA spokesman's comments in response to media queries for an update on the situation in Japan" dated 17 March 2011. Available from http://www.mfa.gov.sg (Accessed on 24 May 2011).*
14. *Ministry of Foreign Affairs. Latest release "MFA spokeman's comments on travel to Japan – We no longer advise against non-essential travel to Japan" dated 12 May 2011. Available from http://www.mfa.gov.sg (Accessed on 24 May 2011).*
15. *World Health Organization. International Health Regulations (2005). Available from http://www.who.int/ihr/9789241596664/en/index.html (Accessed on 24 May 2011).*

Epidemiology of locally acquired salmonellosis in 2009

Introduction

Salmonellosis is a bacterial disease which is characterized by acute enterocolitis, with sudden onset of headache, abdominal pain, diarrhea, nausea and vomiting. The incubation period is usually between 12 and 48 hours but it can range from 6-72 hours. Infection can arise from ingestion of the salmonella bacteria in food derived from infected animals or contaminated by feces of infected animals or human. Common implicated food items include processed meat products, inadequately cooked poultry and eggs and dairy products.[1]

Salmonella, the causative agent, is a genus of gram negative, motile, facultative anaerobic rod-shaped bacteria. It is divided into two species, *Salmonella enterica* and *Salmonella bongori*, with *Salmonella enterica* further divided into subspecies, serogroups and serotypes based on their antigenic presentation.[2]

In December 2008, salmonellosis was made a mandatory notifiable disease in Singapore under the Infectious Diseases Act. All clinical and confirmed cases are to be notified by medical practitioners and clinical laboratories to the Ministry of Health (MOH), Singapore, within 24 hours from time of diagnosis. Prior to this, it was notified administratively. This report aims to describe the epidemiology of locally acquired salmonellosis in 2009.

Materials and methods

We reviewed the demographic data, *Salmonella* serogroups and sources of notification of all locally acquired cases of salmonellosis in 2009. For cases with more than one source of notification, the first source of the notification based on date and time was used for analysis.

We used t-test to determine the level of statistical significance for any changing trend in notifications. The chi-square test was used to compare proportions and logistic regression analysis was carried out to identify factors that could have contributed to any difference in the trend. All statistical analyses were performed using SPSS software version 17.0 (SPSS Chicago, IL). A p-value of <0.05 was considered statistically significant.

Epidemiological findings

A total of 1144 laboratory confirmed cases of salmonellosis were notified in 2009 with an average of 22 cases per week (range 4 to 34). Of these, 1054 (92.1%) cases were locally acquired while 90 (7.9%) cases were imported; i.e. infected overseas (*Fig 1*). Among the locally acquired cases, the incidence rate was 21.1 per 100,000 population. There was a statistically significant difference ($p<0.001$) in the average number of cases notified per week (16.7 cases, range 4-31) during the first half of the year (epidemiological week 1-26, Jan-Jun) compared with that in the second half of 2009 (23.8 cases, range 13-31, week 27-52, Jul-Dec). The total number of cases reported increased by 42.3% from 435 to 619, respectively.

The age of the notified cases ranged from 5 days to 98 years with the highest incidence rate in children aged 0 to 4 years (208.4 per 100,000 population), followed by adults aged 55 years and older

(27.9 per 100,000 population) (*Table 1*). These two age groups constituted 42.8% and 21.6%, respectively of the reported cases in the year. More males were affected in comparison with females, with a male to female ratio of 1.3:1.

Singapore residents contributed 80.2% of the locally acquired cases, while foreigners accounted for another 19.8%. Among the 3 major ethnic groups, the incidence rate was highest among the Chinese (22.1 per 100,000 population) and they comprised 72.3% of the cases among local residents.

Among these locally acquired salmonellosis cases, serogroup D (inclusive of *Salmonella enteritidis*) was the predominant serogroup which made up 43.3% of the total cases, followed by serogroup B (inclusive of *Salmonella typhimurium*) at 26.7%. Between the first half and second half of 2009, the number of serogroup D cases increased by 73% from 167 to 289, compared with an overall increase of 42.3% (*Fig 2*).

MOH received salmonellosis notifications from 10 hospitals, three private clinical laboratories and private general practitioner (GP) clinics. The majority of cases were received from hospitals (67.6%), followed by private clinical laboratories (27.3%). Between the two periods, most sites showed an increase in notification. Private laboratory 2 showed the highest increase from 18 to 99 cases (5.5 fold increase) followed by hospital 4 from 85 to 142 cases (an increase of 67.1%) (*Fig 3*).

Between the two periods, there were significant differences in terms of gender (p=0.025) and nationality/ethnicity (p=0.047). There was also a change in the distribution of the serogroups (p=0.004) and sources of notifications (p<0.001). Multivariate analysis by logistic regression indicated there was significant change in the distribution of the cases between the two periods in terms of gender (p=0.016), serogroups (p=0.008) and source of notifications (p<0.001). Further analyses showed that there was a higher

Figure 1
Weekly distribution of salmonellosis cases reported in 2009

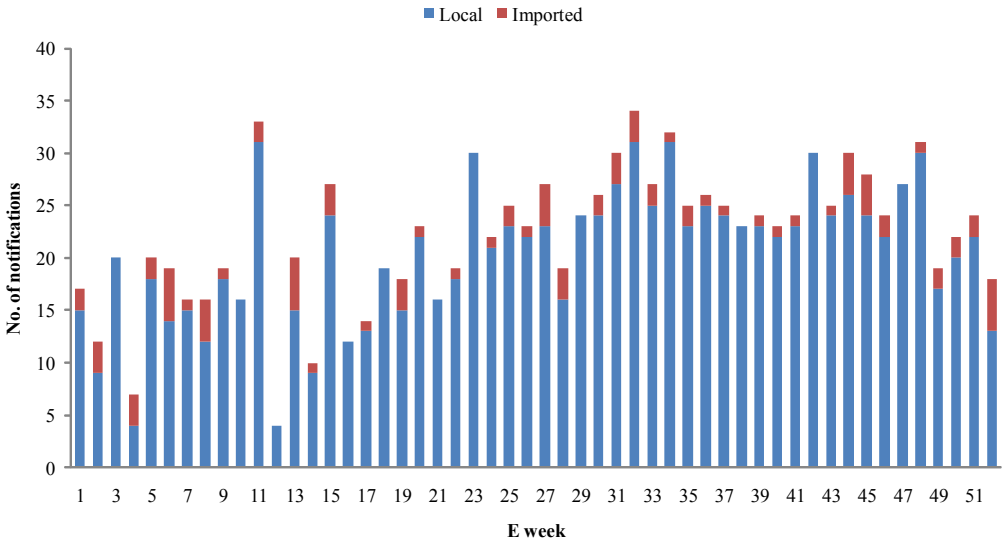


Table 1

Comparison of locally acquired salmonellosis cases reported in the first and second half year, 2009

	First half (%) [wk 1 – 26]	Second half (%) [wk 27 – 52]	Total (%)	Incidence rates (per 100,000 population)	p-value#
All	435 (41.3)	619 (58.7)	1054 (100)	21.1	
Age group (years)					
0-4	179 (41.1)	272 (44.0)	451 (42.8)	208.4	
5-14	26 (6.0)	57 (9.2)	83 (7.9)	16.4	
15-24	28 (6.4)	36 (5.8)	64 (6.1)	8.1	
25-34	29 (6.7)	52 (8.4)	81 (7.7)	7.5	0.123
35-44	36 (8.3)	42 (6.8)	78 (7.4)	8.9	
45-54	36 (8.3)	33 (5.3)	69 (6.5)	10.0	
55 and above	101 (23.2)	127 (20.5)	228 (21.6)	27.9	
Gender					
Male	265 (60.9)	334 (54.0)	599 (56.8)	23.0	0.025
Female	170 (39.1)	285 (46.0)	455 (43.2)	19.1	
Nationality / Ethnicity					
Residents	358 (82.3)	487 (78.7)	845 (80.2)		
Chinese	267 (74.6)	344 (70.7)	611 (72.3)	22.1	
Malay	51 (14.2)	58 (11.9)	109 (12.9)	21.8	
Indian	23 (6.4)	42 (8.6)	65 (7.7)	18.9	0.047
Others	17 (4.8)	43 (8.8)	60 (7.1)	50.0	
Foreigners	77 (17.7)	132 (21.3)	209 (19.8)	16.7	

Chi-square test used to compare differences between the first half and second half of 2009

Figure 2
Serogroup distribution of salmonellosis cases reported in 2009

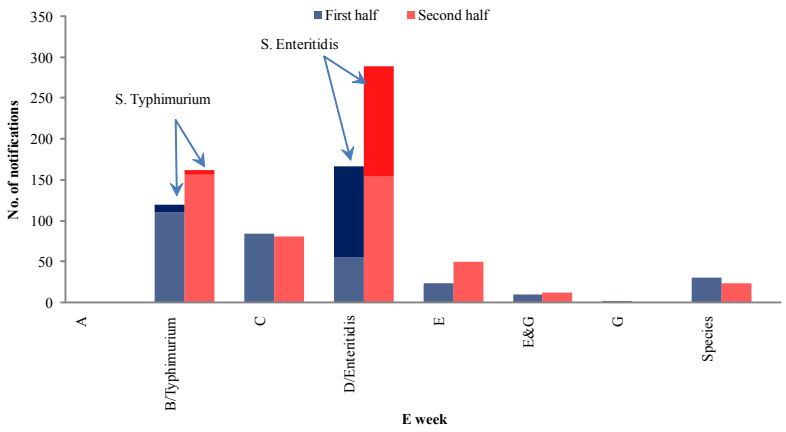
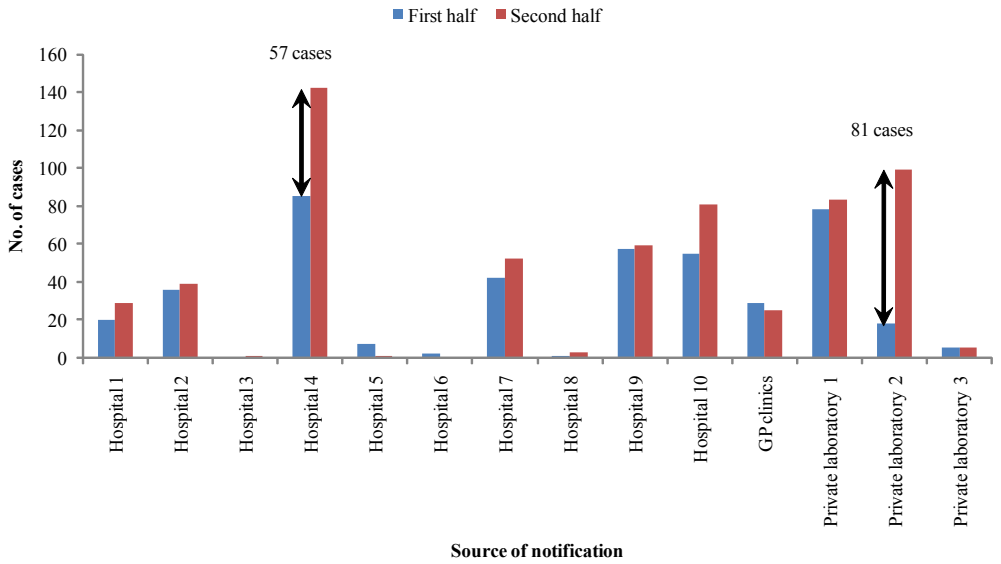


Figure 3
Notifications of salmonellosis cases by institutions in 2009



proportion of female cases (p=0.025), serogroup D (p=0.004) and notifications from private clinical laboratory 2 (p<0.001) in the second half than in the first half of 2009.

Comments

Salmonellosis affects predominantly the infants and young children locally, consistent with the global trend. Adults aged 55 and older formed the other major age-group affected by salmonellosis and this is in accordance with the local trend from 2000 to 2008.[3]

There was a significant increase in salmonellosis notifications in the second half of 2009 compared to the first. Our findings showed that there was a considerable increase in cases reported by private clinical laboratory 2. Further inquiry into the data showed that this laboratory had started notifying salmonellosis cases only from week 18 in 2009 onwards. Nonetheless, the increase could not be attributed to this abrupt contribution by this specific laboratory as multivariate analysis showed that gender, serogroup and notification sources are independent factors affecting the distribution of cases in the two periods. Similarly, it could not be used to explain the increase in proportion of female cases or serogroup D cases.

With the mandatory notification of salmonellosis, the accuracy in assessing the distribution of *Salmonella* serogroups in Singapore has increased. Currently, most *Salmonella* isolates are routinely serotyped for *Salmonella enteritidis* and *Salmonella typhimurium*. Majority of cases not tested positive for these two serotypes were notified in terms of the serogroup.

This information could be used for serogroup surveillance which potentiates the ability to detect possible outbreak. Nonetheless, there are currently

over 2,500 different serotypes identified and are organized into these serogroups depending on their antigenic presentation.[4] In this study, we detected a significant rise in *Salmonella* serogroup D cases. However, majority of these cases were not further serotyped. Hence, establishing serogroup surveillance is only able to provide an overview on the prevalent serogroup and their distributions. It is not discriminatory for monitoring the trend in serotypes, which offers greater resolution of the *Salmonella* situation in Singapore.

To address this problem, the National Public Health Laboratory has embarked on the task in serotyping a sample of *Salmonella* isolates from public hospitals. This will provide better resolution on the distribution of serotypes in Singapore and also support epidemiological investigations in foodborne outbreaks.

(Reported by Lim SK[1], Lai F[2], Tan JD[1], Ling V[1], Toh HY[1], Chan PP[1], Badaruddin H[1], Foong BH[1] and Ooi PL[1], Communicable Diseases Division[1] and Epidemiology and Disease Control Division[2], Ministry of Health)

References

1. Heyman DL (ed). Control of Communicable Diseases Manual. American Public Health Association, 18th Edition, 2004

2. Grimont PAD, Weill FX. Antigenic formulae of the Salmonella serovars*. WHO Collaborating Centre for Reference and Research on* Salmonella*, Institut Pasteur, Paris, France, 9th Edition, 2007*

3. Kita Y, Ye T, Chow A et al. The changing epidemiology of salmonellosis in Singapore. Epidemiological News Bulletin 2006; 32: 21-4

4. WHO. Fact sheet No. 139: Drug-resistant Salmonella*. Available at http://www.who.int/mediacentre/factsheets/fs139/en/*

Hantavirus disease in Singapore: a report of two cases in 2010

Hantaviruses (HTVs) are segmented single-stranded RNA viruses belonging to the *Bunyaviridae* family. Viruses from the *Hantavirus* genus are present in urine, feces and saliva of persistently infected asymptomatic rodents. Human infection is acquired directly through contact with rodent excreta and indirectly through contaminated foodstuff. Infection by the respiratory route from aerosols is believed to be another route of transmission in laboratory setting. Direct transmission by rodent bites has been documented. However; it is not transmitted directly from person to person. The clinical severity of Hantavirus infections ranges from asymptomatic infections to fulminate hemorrhagic shock and death. There are two distinct syndromes caused by certain members of the *Bunyaviridae* family: hemorrhagic fever with renal syndrome (also known as Korean hemorrhagic fever) and hantavirus pulmonary syndrome (also known as hantavirus adult respiratory distress syndrome and hantavirus cardiopulmonary syndrome).[1] Laboratory

findings of the first syndrome which is primarily found in Asia include rise of serum creatinine and blood urea nitrogen, thrombocytopenia, proteinuria and change in serum electrolytes.

In 2010, the Ministry of Health (MOH) received notifications of two laboratory-confirmed cases of hemorrhagic fever with renal syndrome caused by HTV from a hospital and a clinical laboratory in May and October, respectively. These cases were thoroughly investigated upon notification, followed by multi-agency response.

Case reports

Case 1 was a 41-year-old resident who worked as a construction site supervisor. He had no past medical history of note. He was admitted to a general hospital on 7 May 2010 following 3 days of fever with myalgia, lethargy and poor appetite. Physical examination showed scattered petechiae over his ankles and left medial calf region. His temperature was 38.9°C and blood pressure (BP) 104/68 mm Hg. The total white cell and platelet counts were 6.2x 10^9/l (normal, 3.6 -9.3 x 10^9/l) and 13 x 10^9/l (normal, 170 -420 x 10^9/l), respectively.

Initially, he was diagnosed as a suspected case of dengue / viral fever. He was placed on intravenous drip for the management of rising hematocrit and low pulse pressure. He was given two units of platelet transfusion in view of his very low platelet count.

The patient subsequently complained of abdominal bloatedness two days after admission. He was found to have hypertension as well as bilateral pleural effusions and mild ascites. Ultrasound scan of his abdomen showed fatty liver and mildly increased bilateral renal echogenicity which suggested renal impairment. A cyst was also found in the left renal lower pole. .

Laboratory results showed rising creatinine levels from 161µmol/l (normal 44-141 µmol/l) at time of admission to a peak of 520 µmol/. Two weeks later, his renal function returned to normal, though creatinine level was marginally elevated (238 µmol/l) at time of discharge.

There was laboratory evidence of liver impairment with raised total bilirubin (34µmol/l; normal 3-24 µmol/l), alanine aminotransferase (ALT) (466 U/l; normal, 7-36 U/l) and aspartate aminotransferase (AST) (911 U/l; 15-33 U/l), but these levels returned to normal subsequently.

Blood sample collected on 14 May 2010 was positive for HTV by immunofluroescent assay (IFA). HTV IgG and IgM antibodies were also positive. Other possible aetiologies for the infection, including dengue, typhoid, malaria and leptospirosis, were excluded.

Case 2 was another local resident, a 35-year-old IT executive, who presented with a history of fever, mild myalgia and headache on 3 October 2010. On admission, he had a temperature of 38.7°C.He was found to have hepatomegaly, but no rashes or lymphadenopathy. He was treated with intravenous ceftriaxone and oral doxycycline.

Five days later, the patient complained of generalized swelling accompanied with orthopnea and dyspnea on exertion. Patient also complained of lower back pain that radiated to both anterior and

posterior thighs. He was given a trial of amitryptline with resolution of symptoms. Ultrasound of the abdomen showed a small right kidney interpolar cyst and right pleural effusion, suggesting acute kidney injury.

Laboratory findings revealed elevated levels of ALT (from 87 U/l to a peak at 202 U/l), AST (from 130 U/l to 273 U/l) and elevated creatinine (229 µmol/l). The total white cell count was increased (9.5 x 10^9/l; normal, 3.6 -9.3 x 10^9/l) and platelet count decreased (74 x 10^9/l; normal, 170 -420 x 10^9/l).

Blood samples collected on 18 October 2010 and analyzed by IFA confirmed HTV infection. . Other possible causes of the infection, including dengue fever/dengue hemorrhagic fever (DF/DHF), enteric fever, malaria and leptospirosis, were excluded.

Both cases subsequently recovered and discharged well from the hospital.

Epidemiological investigation

Both cases reported recent travel histories to Malaysia (Johore Bahru and Genting Highlands, respectively) 2-4 weeks prior to their onset of symptoms.

Case 1 had observed the presence of rats in the vicinities of his home in Marsiling as well as his work sites at Changi North Crescent, while case 2 also noted rodent infestation around his home residence in Bishan. Environmental investigation jointly conducted with the National Environment Agency (NEA) found evidence of rat burrows in the drains around the residential areas in Marsiling and Bishan. Rat burrows were also detected within the area cordoned off for the lift upgrading works around the residential vicinity of case 2. One rodent excreta sample collected during field investigation tested negative for HTV by PCR.

Rodent control and prevention measures were implemented to eliminate rodent infestation in the affected areas.

Discussion

The incidence of human hantavirus disease is low in Singapore. To date, only three severe cases of hantavirus hemorrhagic fever with renal syndrome (HFRS) have been reported.[2-4]

The two cases described above were probably infected locally through direct or indirect contact with rodent excreta. However, the mechanism by which they acquired the infection could not be determined. They had no direct contact with live rodents. The significance of travel history could also not be ascertained as we have no information of the HTV situation of the places visited.

Rattus norvegicus is the predominant rodent reservoir in Singapore[5]. In a study conducted by Environmental Health Institute in 2009, the hantavirus seroprevalence rate was 34% and two genetically different HTV strains were found in the wild rodent population. However, the pathogenicity of HTV isolated and its impact to public health is still unclear. Further studies to evaluate the epidemiological and epizootical significance of these findings are warranted.[6]

Stringent control measures and vigilance against rodent infestations in and around residential areas and workplaces should be continued to prevent transmission of hantavirus infection in humans.

Editorial comments

Hantavirus infection is present in both rodents and humans in Singapore. Serological studies conducted in the late 1980s and early 1990s showed that 40 (27.8%) of 144 urban rats, 148 (40.3%) of 367 imported laboratory white rats, 5 (1.6%) of 310 patients with glomerulonephritis, 8 (2.5%) of 314 patients with 'leptospirosis', 1 (4.8%) of 21 patients with 'DF/DHF', 14 (19.4%) of 72 patients with 'non-A, non-B hepatitis' and 2 (6.3%) of 32 animal workers had been infected. A seroprevalence survey on hantavirus infection in various population groups in Singapore carried out between 1991 and 1993 showed a prevalence of 4.5% in the general population (8/178), 0.4% in engineering services staff (1/247), 2.7% in sewage workers (16/601), 1.3% in zoo keepers (2/149) and 4.7% in cemetery workers (3/64). All the 76 sand quarry and 43 dumping ground workers tested seronegative.

Hantavirus disease should be suspected in a patient who has been exposed to rodents and their excreta and presenting with fever, muscular pains, hemorrhagic manifestations and proteinuria, leading to complications of shock and renal failure. It should be included in the differential diagnosis of acute nephritis, chronic renal failure, DF/DHF, leptospirosis and patients with hepatorenal dysfunction.

A high standard of environmental sanitation is maintained in all premises, and buildings are rat-proofed to reduce contacts between man and excreta of rodents. Laboratory rats/mice are certified free from hantavirus (as well as lymphocytic choriomeningitis and leptospirosis) before they are imported and mouse colonies and laboratory workers are kept under surveillance. Any batch of laboratory animals found to be infected with hantavirus and other zoonotic diseases is destroyed immediately.

(Contributed by Surveillance & Response Branch, Communicable Diseases Division, Ministry of Health)

References

1. Taylor SL, Altamura LA, Schmalijohn CS Encyclopedia of life sciences www.els.net John Wiley & Sons Ltd 2009.

2. Chan KP, Chan YC, Doraisingham S. A severe case of haemorrhagic fever with renal syndrome in Singapore Southeast Asian J Trop Med Public Health 1996; 27:408-10

3. Wong TW, Chan YC, Lee HW. Hemorrhagic fever with renal syndrome in Singapore: a case report. Southeast Asian J Trop Med Public Health 1985; 16:525-7

4. Chan YC, Wong TW, Yap EH et al. Hemorrhagic fever with renal syndrome involving the liver. Med J Aus 1987; 147:248-9

5. Wong TW, Chan YC, Joo YG et al. Hantavirus infections in humans and commensal rodents in Singapore. Transactions of the Royal Society of Tropical Medicine and Hygiene 1989; 83, 248-5

6. Johansson P, Yap G, Low HT et al. Molecular characterization of two hantavirus strains from different Rattus *species in Singapore. Virology Journal 2010; 7:15*

Risk assessment on West Nile virus

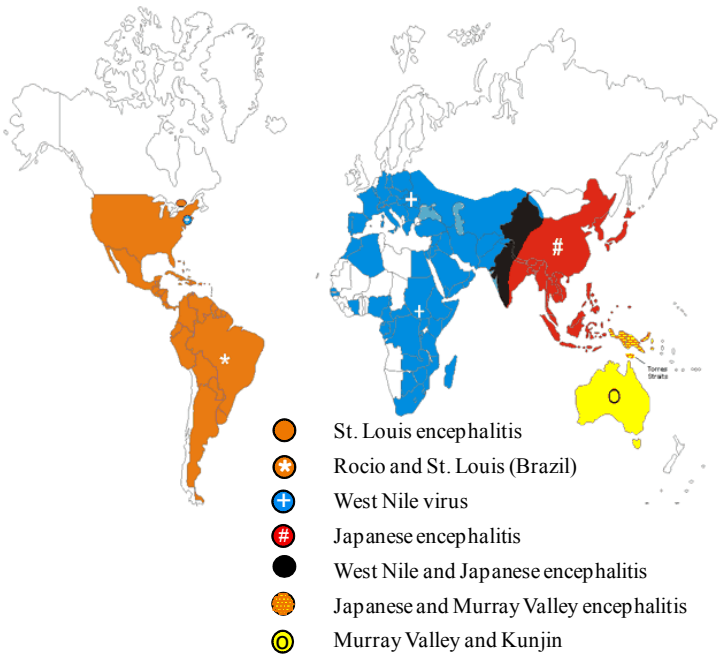
Introduction

History and geography

West Nile virus (WNV) is a mosquito-borne arbovirus whose natural cycle of infection involves birds and mosquitoes.[1] It can also infect humans, horses and some other mammals but these infections are incidental.[2] WNV is a member of the family *Flaviviridae* (genus *Flavivirus*) and serologically belongs to the Japanese encephalitis virus antigenic complex, which includes St. Louis, Japanese, Kunjin, and Murray Valley encephalitis viruses.

WNV was first discovered in 1937 in a patient from the West Nile region of Uganda and has remained enzootic and caused sporadic cases and outbreaks of human WNV infections throughout Africa, parts of Europe, the middle East, West and Central Asia and Australia (in the form of Kunjin virus, a subtype of WNV) (*Fig. 4*). Despite its wide geographical distribution, WNV was considered to be relatively benign until recently as it usually causes a mild illness, with fatal outcomes being very rare.[3] However, the outbreak of WNV infection in Romania in 1996 reported 393 neurological cases (meningitis, meningoencephalitis and encepahalitis) with 17 deaths, a case fatality rate of 4.3%.[4] Furthermore, an unprecedented outbreak of WNV infection involving humans, horses and birds in New York City in 1999 drew attention to WNV as an emerging threat.[5]

Figure 4
The geographic distribution of the Japanese encephalitis serocomplex of the family flaviridae, 2000



Source: http://www.cdc.gov/ncidod/dvbid/westnile/map.htm

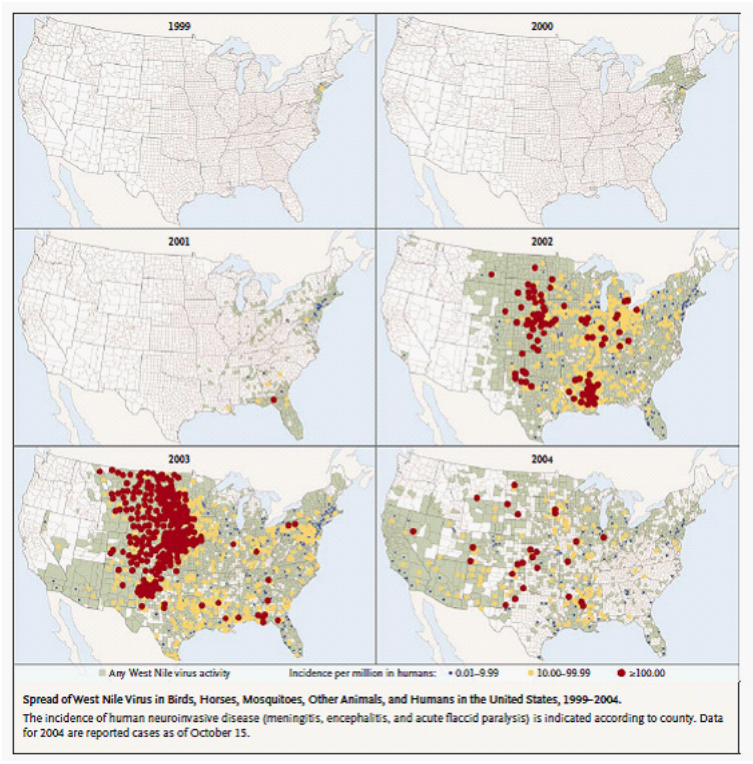
Outbreaks in North America

In the Western Hemisphere, most clinical cases of WNV infection have occurred in the US. The initial outbreak of WNV occurred in New York in August 1999. It has since spread through the US (*Fig. 5*) and into Canada. WNV cases were reported in 40 states in the US in 2002 and 46 states in 2003. Between 1999 and 2009, a total of 12,126 cases with neuroinvasive WNV disease and 1,163 deaths have been reported in the US.[6] In Canada, WNV activity was first reported in dead birds and mosquito pools in southern Ontario in 2001. First human cases were reported from Quebec and Ontario in 2002. Between 2002 and 2009, Canada reported a total of 4,560 clinical cases.[7] Evidence of WNV transmission has also been reported in Latin America and the Caribbean but the paucity of human cases reported in these areas is surprising, considering the ecological conditions that favour the transmission of the virus[8].

Outbreaks in Europe

In the Eastern Hemisphere, human WNV disease has been reported mostly from areas in Mediterranean Basin: Algeria (1994), Morocco (1996), Tunisia (1997, 2003), Romania (1996-2000), Czech Republic (1997), Israel (1999-2000), Russia (1999-2001, 2010) and France (2003).[8,9] By far the largest outbreaks in the Eastern Hemisphere occurred in Bucharest, Romania, in 1996 (393 hospitalised cases, 17

Figure 5
Spread of West Nile virus in the US, 1999-2004



Source: Petersen LR, Hayes EB. Westward ho?--The spread of West Nile virus. NEJM 2004; 351: 2257-9

deaths), Volgograd, Russia, in 1999 (826 hospitalised cases, 40 deaths) and Israel (326 hospitalised cases, 35 deaths); all three regions are located along the major migratory routes of birds that overwinter in Africa.[9,10]

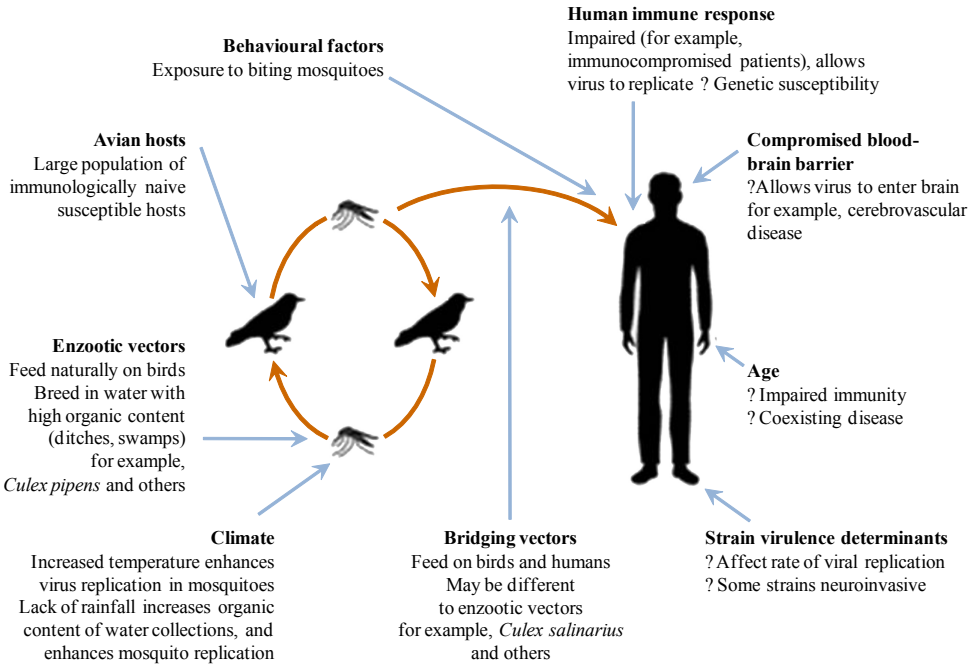
More recently, there was an outbreak of WNV infection in Greece, largely limited to the region of Central Macedonia. The outbreak began in early August 2010 and ended in October 2010 with a total of 261 laboratory confirmed cases and 34 fatalities being reported.[11] In addition, Romania reported 57 cases of WNV infection (54 with neuroinvasive infection and three with fever) with five deaths between July and October 2010; and most cases occurred in the already known endemic area in the south.[12] In Russia, there were 231 cases of WNV infection and 6 deaths reported during the same period and the great majority of cases were reported from Volgograd. Israel has also reported 24 confirmed cases as of 10 August. The Turkish Ministry of Health on 8 September confirmed seven human cases of WNV infection with three deaths.[13]

Epidemiology

Hosts, vectors and transmission

The primary cycle of WNV involves birds and ornithophilic mosquitoes, mainly *Culex* species (*Fig. 6*). The virus can be transmitted by a number of mosquito species, which vary according to the geographical area. In Europe and Africa, the principal vectors are *Cx. pipens*, *Cx. univittatus*, and *Cx. antennatus*; and in India, species of the *Cx. vishnui* complex. In Australia, Kunjin virus is transmitted primarily by

Figure 6
Life cycle of West Nile virus



Source: http://njlmn.rutgers.edu/cdr/docs/Module05-Epi-Part2.pdf

Cx. annulirostris. In the US, *Cx pipiens* is the main vector in the northeast, *Cx tarsalis* in the west, and *Cx quinquefasciatus* in the south. *Aedes albopictus* was found in 2% of the WNV-positive mosquito-pools in the US in 2002.[8]

Birds are the main amplifying hosts for WNV and transmit the infection to biting mosquitoes. WNV has been detected in dead birds of at least 326 species.[14] Although many birds are well adapted and immune to WNV infection, it can cause high mortality in crows and jays. Such bird deaths have been used to monitor the spread of WNV across the US. During the initial outbreaks in New York City in 1999, bird die-offs were investigated separately on top of the investigation into human encephalitis cases.[15]

WNV outbreaks in horses have been documented and approximately 40% of equine WNV cases results in the death of the horses.[14] A serosurvey in New York City of dogs in the 1999 epidemic area indicated that dogs are frequently infected; yet WNV does not appear to cause extensive illness in dogs or cats.[14] Humans, horses and other mammals are incidental, dead-end hosts and they do not appear to exhibit a high viremia of sufficient magnitude to infect mosquitoes.[8]

Clinical manifestation, diagnosis and treatment

WNV infection in humans can result in a range of clinical outcomes from asymptomatic to severe neuroinvasive illness with elderly patients having a higher risk of developing neuroinvasive illness and mortality.[16] About 80% of WNV human infections are asymptomatic and approximately 20% experience a mild or self-limited illness. Symptomatic illness generally follows an incubation period of 2 to 15 days (typically 2 to 6 days) with a variety of nonspecific complaints: sudden onset of high fever (>39°C), headache, myalgia and weakness with gastrointestinal symptoms. These acute symptoms often resolve after a week, although prolonged fatigue is not uncommon. Neuroinvasive disease, developed in <1% of the cases, affects the central nervous system in the form of meningitis, encephalitis and paralysis. WNV infection of spinal motor neurons (anterior horn cells) causes acute, asymmetric flaccid paralysis (AFP) similar to that seen with poliomyelitis. Infection of the brainstem and high cervical spinal cord may cause diaphragmatic and intercostal muscle paralysis with resulting respiratory failure and sometimes death. There were also reports of developing acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barré syndrome). The case-fatality rate among patients with neuroinvasive WNV disease is about 10%, with higher fatality among elderly patients.[17] In addition, many survivors of such neurological complications of WNV infection may have significant disability.

Diagnosis of WNV infection is often based on direct detection of viremia by culture or by serological testing for antibodies. Normally WNV IgM becomes detectable about 8 days after onset of symptoms. WNV infection may be diagnosed by RT-PCR, but the samples must be taken early, soon after infection, as the viremia is transient and starts to drop around the time that antibodies start appearing.[18] There is no specific antiviral therapy; management of WNV infection is generally supportive. No human vaccine is currently available against WNV.

Non-mosquito-borne transmission

Transmission of WNV is primarily by the bite of infected mosquitoes. However, transmissions through (i) conjunctival exposure to infected blood, (ii) blood transfusion, (iii) organ transplantation, (iv)

intrauterine exposure, and (v) breastfeeding have been documented.[8] Since 2003, blood banks in the US routinely screen for the virus amongst their donors. In the UK, the National Blood Services initially ran the WNV test in donors who donate blood within 28 days of a visit to the US, Canada or the North Eastern provinces of Italy. Currently, their policy is to ask donors returning from these areas to wait 28 days before donating.

Surveillance

In human case surveillance for WNV infection, monitoring of encephalitis cases is the highest priority; monitoring other manifestations of WNV infection (e.g. aseptic meningitis, Guillain-Barre syndrome, acute flaccid paralysis, and brachial plexopathy, and fever or rash illnesses) is resource-dependent.[19]

Surveillance strategies in areas known to have WNV / arbovirus activity include both human case surveillance and non-human based surveillance. Non-human based surveillance focuses on the avian and mosquito components of the enzootic transmission cycle. Mosquito-based surveillance is the primary tool for quantifying the intensity of virus transmission in an area. Experience in the US suggests that surveillance of bird mortality, particularly birds from the family *Corvidae*, has been able to effectively track the movement of WNV.[19]

In addition, non-human mammals, particularly equines, also serve as important mammalian sentinels of WNV epizootic activity and potential risk to humans. In the US, the surveillance includes the reporting of equine neurological disease as well as antibody testing of equine specimens (serum and cerebrospinal fluid).

Control

Control of WNV infection is achieved primarily through mosquito control (by elimination of mosquito breeding sites and larviciding active breeding areas). Others include personal protection measures (such as wearing long sleeve clothing, and applying mosquito repellents), and avoiding mosquito-prone areas and vector biting times at dusk and dawn during the peak biting times of *Culex* mosquitoes.

Risk factors

Exposure to infected mosquitoes

The most important risk factor for acquiring WNV infection is exposure to infected mosquitoes. The recent outbreaks in Bucharest and Volgograd occurred in urban areas and were associated with cellars flooded with sewage-polluted water in poorly maintained apartment blocks, a highly productive breeding site for mosquito.[8,20] An analysis of the locations of WNV disease cases during the 1999 outbreak in New York found that cases were clustered in an area with higher vegetation cover, indicating favourable mosquito habitat[8]. A household-based seroepidemiological survey conducted in New York identified that time spent outdoors and failure to apply mosquito repellent are risk factors associated with WNV infection.[21]

Local resident and migratory birds

Birds are probably the most important amplifying hosts of WNV. In laboratory studies, species like song birds, shorebirds, owls and hawks developed viremic levels sufficient to infect most feeding mosquitoes. Field studies in the US confirmed that house sparrows were abundant and frequently infected with WNV.

Migratory birds play an important role in the transmission of the virus to humans, and have long been suspected to be the principal introductory hosts of WNV into new regions.[22] Studies showed that migratory birds have been linked with the spread of WNV in the Western Hemisphere. Also, antibodies to the WNV have been found in the blood of many migratory bird species in Eurasia, and the virus has been isolated from some species of active migratory birds (e.g. the barred warbler in Cyprus, the turtle dove in Slovakia). Viremia in some bird species can last long enough to infect vector mosquitoes. Migration places substantial physiological stress on birds and stress tends to promote immunosuppression and thus enhance replication of the virus.

Factors related to more severe outcomes

Cases in the US indicated that persons of all ages appear to be equally susceptible to WNV infection, but the risk of developing neuroinvasive form of the disease and mortality increases with age and is slightly higher for males than females.[8,23] The severe outcomes of WNV infection has also been shown to be associated with immunosuppression after organ transplant.[8]

A genetic factor appears to be associated with an increased susceptibility to West Nile disease. A mutation of the gene CCR5 gives some protection against human immunodeficiency virus (HIV) but leads to more serious complications of WNV infection. Carriers of two mutated copies of CCR5 made up 4 to 4.5% of a sample of West Nile disease sufferers while the incidence of the gene in the general population is only 1%. [24,25]

Risk to Singapore

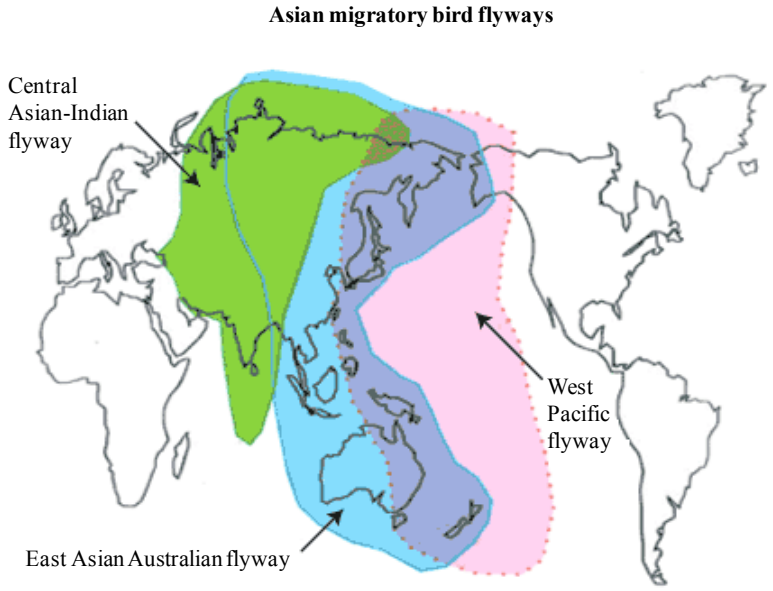
WNV may be introduced into Singapore through (a) migration of infected birds, (b) importation of infected birds or other animals, and c) international travel of infected persons to Singapore. However, humans, horses and most other mammals are incidental "dead-end" hosts and are not involved in the primary cycle of WNV. The building up of a virus reservoir in the local bird populations is necessary to allow the disease cycle to continue locally. Thus, the risk of introduction of WNV leading to local transmission is mainly through migratory birds and the import of infected birds. In addition, the high proportion of asymptomatic cases may pose a risk of WNV transmission through blood donation or organ transplants.[16]

Bird migration in Singapore

Infected migratory birds seem to be the most important introductory hosts of WNV into a new area. Singapore is along the East Asian-Australasian flyway (*Fig. 7*) and Sungei Buloh Wetland Reserve, with an area of 130 hectares, is a stop-over point for migratory birds. As many as 60 different species can be spotted in a single day during the annual migratory season between September and March. The East Asian-Australasian flyway is one of the major migratory flyways around the globe. It expands from within the Arctic Circle in Russia to Alaska in the north to Australia and New Zealand in the south. Between these extremes, the flyway covers East and South-East Asia, including China, Japan, Korea, and the western Pacific.[26]

Kunjin virus (a subtype of WNV) is endemic in the tropical north of Australia, but fortunately this virus seems to be rather avirulent. Other countries along the pathway are not known to have had major outbreaks of WNV. Since June 2010, the National Environmental Agency (NEA) has started to screen

Figure 7
Central Asian, East Asian-Australasian and West Pacific migratory bird flyways



Source: http://en.wikipedia.org/wiki/Flyway#Flyways_of_Eurasia.2C_Africa.2C_and_Australasia

migratory birds and resident birds (crows) for the presence of the WNV using serology and PCR. To date, no WNV has been confirmed in these birds.

Importation of infected birds into Singapore

Since the outbreak of highly pathogenic avian influenza (HPAI) in late 2003, the bird trade through Singapore has declined substantially and the number of countries exporting birds to Singapore has also been reduced. The Agri-food Veterinary Authority (AVA) has implemented pre-border and border controls over importation of captive birds for the pet trade or as personal pets. To import birds into Singapore, an AVA licence from the Import and Export Regulation Division is required. Each consignment of birds must be accompanied by a veterinary certificate dated within seven days of import signed by a government veterinary authority. Birds are also required to be kept in isolation in a facility since they were hatched or for 21 days prior to shipment and examined on the day of export certifying no clinical signs of infectious diseases. WNV viremia in birds lasts at most 7-8 days[9], the isolation requirement of 21 days is deemed sufficiently long to forbid importation of birds infected with WNV into Singapore. Thus, the risk of introduction of WNV through importation of infected birds is low.

Presence of *Culex* mosquitoes

The principal mosquito vector of WNV is *Culex* with *Cx quinquefasciatus, Cx tritaeniorhynchus*, and *Cx gelidus* present in Singapore.[27] *Cx quinquefasciatus* has been reported as the predominant vector of WNV infection in the South of the US and is generally

considered a moderate-to low-efficiency vector[8]. It breeds mainly in polluted waters, in septic tanks, cesspits, drains, ditches, and so on and is associated with increasing urbanization, poor drainage and sanitation.

In Singapore, the NEA has put in place mosquito surveillance to screen mosquitoes captured from areas where migratory birds are present. No WNV has yet been detected via PCR. The NEA also has plans to further strengthen the programme to start screening mosquitoes trapped in urban residential areas as this would ascertain the risk of WNV transmission in urban areas.

Transmission through blood transfusion

Transmission of WNV through blood transfusion in the US has been documented, particularly during periods of high prevalence. WNV testing of the US blood supply was implemented based on nucleic acid amplification test (NAAT) technology. Other than the US, blood supply screening in other countries is based on donor deferral strategies involving pre-donation questioning of donors alone.

The Blood Services Group (BSG), Health Sciences Authority (HSA) has periodically reviewed the need to implement measures to assure blood supply safety with regard to WNV. In view of the absence of reported clinical cases of WNV in Singapore, the current measures taken are based on donor deferral and WNV testing is not performed at this point in time.

Since December 2004, BSG has adopted the following policies to minimize the risk of transmission through blood transfusion:

- Donors who have travelled to areas at risk of WNV transmission and developed a fever in the past 28 days are deferred for 4 weeks from date of recovery;
- Donors who have travelled to areas at risk of WNV transmission in the past 28 days but in good health may be accepted for blood donation. Blood components prepared from these donations are quarantined for 6 months and only released for transfusion after confirmation that the donors have not been diagnosed with WNV or experienced any symptoms of WNV during that period.

The list of areas at risk is regularly updated to reflect the latest disease activity.

To assess the need to introduce WNV testing for the blood supply, BSG tested 7,716 samples from blood donors in 2010 for WNV RNA using NAAT technology. None of the samples tested positive for the virus. This result also suggested that the risk of transmitting WNV through blood transfusion is low.

In addition, the NEA began screening dengue-negative febrile patient samples collected from general practitioner (GP) clinics as part of their *flavivirus* surveillance programme from June 2009. No WNV positive sample has been detected by PCR so far. Both the surveillances conducted by BSG and NEA suggested the virus is unlikely to be circulating in the community. Therefore, the risk of WNV transmission through blood transfusion is low.

Sporadic cases

Singapore is a travel hub with rapid, onward connections to other parts of the world. WNV is endemic in Europe and the US. The volume of travel between Singapore and these two areas is significant. Although the present surveillance programmes reveal no sign of circulation of WNV in the community, sampling strategies are always incomplete and sporadic cases may occur in travellers returning from the affected regions.

Laboratory testing for WNV infection is not routinely available in the hospitals in Singapore. Unidentified causes of hospitalized cases of encephalitis are not uncommon. While encephalitis can be caused by many infective agents, the possibility that some of these cases may be due to undiagnosed WNV infection cannot be excluded. Currently, clinicians do not routinely send samples for WNV testing as the perceived risk of disease is low. Dengue is endemic in Singapore and the WNV antibody assays may cross-react with dengue and other flaviviruses.

In terms of diagnostic capability in Singapore, the Environmental Health Institute (EHI) and the Defence Medical and Environmental Research Institute (DMERI) of Defence Science Organisation (DSO) have the capability to do the WNV PCR test; while the National Public Health Laboratories (NPHL) can provide pan-*flavivirus* PCR test.

Risk assessment

Likelihood of WNV outbreak in Singapore

Presently, the likelihood of a WNV disease outbreak is considered 'unlikely'. Singapore has put in place human, bird and mosquito surveillance systems to track the activity of WNV. From these, there are no current indications of importation or circulation of the virus in Singapore.

(1) Singapore has had no clinical cases reported so far. The Ministry of Health (MOH) monitors the number of hospitalized encephalitis cases and there has not been any unusual increase in the number of cases.

(2) MOH has implemented the Severe Illness and Death from Possible Infectious Diseases (SIDPID) project in collaboration with the restructured hospitals, which focuses on investigation of unexplained deaths and critical illnesses with suspected infectious disease aetiology. The project serves as a surveillance system for emerging infections caused by novel pathogens such as WNV that could cause outbreaks. Under the protocol, people who have severe neurological illness or who die from encephalitis with unknown aetiology, would have their cerebrospinal fluid (CSF) tested for WNV, if there is strong clinical indication. So far, there has been no WNV case detected.

(3) NEA has been screening dengue-negative, febrile patient samples collected from GP clinics around Singapore since June 2009. More than 700 samples have been tested and no WNV positive samples have been detected so far.

(4) Since June 2010, NEA has started screening migratory birds and resident birds (crows) for the presence of WNV. To date, no WNV positive samples have been confirmed in the 38 samples tested.

(5) In addition to human and bird surveillance, NEA also screens mosquitoes trapped in areas where migratory birds are present. No WNV has been detected as yet.

(6) AVA has implemented strict pre-border and border controls and licensing requirements to minimize the risk of importation of the virus through infected birds to Singapore.

(7) BSG, HSA has implemented strict policies for blood donors who have recently travelled to areas at risk of WNV transmission.

(8) A recent survey of over 7000 samples from blood donors in 2010 showed that none of them were positive for WNV.

Consequence of a WNV outbreak in Singapore

The consequences of WNV infection at the population level is considered 'moderate to minor'. Given that less than 1% of the infected will develop neuroinvasive diseases and the case fatality rate among such neuroinvasive diseases is about 10%, the overall case fatality rate is less than 0.1%.

(Contributed by: Epidemiology and Disease Control Division, and Communicable Diseases Division of Ministry of Health, Agri-food and Veterinary Authority, Health Sciences Authority and National Environmental Agency)

References

1. *Calistri P, Giovannini A, Hubalek Z et al. Epidemiology of West Nile in Europe and in the Mediterranean Basin. Open Virol J 2010; 4; 29-37.*
2. *Dauphin G, Zientara S, Zeller H et al. West Nile: worldwide current situation in animals and humans. Comp Immunol Microbiol Infect Dis 2004; 27: 343-55.*
3. *Hubálek Z, Halouzka J. West Nile fever - a reemerging mosquito-borne viral disease in Europe. Emerg Infect Dis 1999; 5: 643-50.*
4. *Tsai TF, Popovici F, Cernescu C et al. West Nile encephalitis epidemic in southeastern Romania. Lancet 1998; 352: 767-71.*
5. *Solomon T, Cardosa MJ. Emerging arboviral encephalitis: newsworthy in the West but much more common in the East. BMJ 2000; 321: 1484-5.*
6. *Centers for Disease Control and Prevention. West Nile virus. Available from http://www.cdc.gov/ncidod/dvbid/westnile/surv&control.htm*
7. *Public Health Agency of Canada. West Nile virus. Available from http://www.phac-aspc.gc.ca/wn-no/hist-eng.php*
8. *Hayes EB, Komar N, Nasci RS et al. Epidemiology and transmission dynamics of West Nile virus disease. Emerg Infect Dis 2005; 11: 1167-73.*
9. *Reiter P. West Nile virus in Europe: understanding the present to gauge the future. Euro Surveill. 2010; 15: 19508.*
10. *Weinberger M, Pitlik SD, Gandacu D et al. West Nile fever outbreak, Israel, 2000: epidemiologic aspects. Emerg Infect Dis 2001; 7: 686-91.*
11. *Hellenic Centre for Diseases Prevention and Control. West Nile virus infection, http://www.keelpno.gr/index.php?option=com_content&view=article&id=140%3Awest-nile-virus-infection&catid=45%3A2010-06-28-08-41-59&Itemid=1.*
12. *Sirbu A, Ceianu CS, Panculescu-Gatej RI et al. Outbreak of West Nile virus infection in humans, Romania, July to October 2010. Euro Surveill. 2011; 16: 19762.*
13. *European Centre for Diseases Prevention and Control. Update on West Nile virus transmission in Europe 10 Sep 2010. Available from http://www.ecdc.europa.eu/en/activities/sciadvice/Lists/ECDC%20Reviews/ECDC_DispForm.aspx?List=512ff74f%2D77d4%2D4ad8%2Db6d6%2Dbf0f23083f30&ID=940&RootFolder=%2Fen%2Factivities%2Fsciadvice%2FLists%2FECDC%20Reviews*
14. *Centers for Disease Control and Prevention. West Nile virus - vertebrate ecology. Available from http://www.cdc.gov/ncidod/dvbid/westnile/birds&mammals.htm*
15. *Report of the Minority Staff, Senate Governmental Affairs Committee to Senator Joseph I. Lieberman, Ranking Member. Expect the unexpected: the West Nile virus wake up call. Dated July 2000. Available from http://hsgac.senate.gov/wnvfinalreport.pdf*
16. *Zeller H, Lenglet A, Bortel W Van. West Nile virus: the need to strengthen preparedness in Europe. Euro Surveill 2010; 15 :34.*

17. *Hayes EB, Sejvar JJ, Zaki SR et al. Virology, pathology, and clinical manifestations of West Nile virus disease. Emerg Infect Dis 2005; 11: 1174-9.*

18. *Tang JW, Chan PKS. Emerging infections II (West Nile virus, dengue, severe acute respiratory syndrome-associated coronavirus). In: Shetty N, Tang JW, Andrews J, editors. Infectious Disease: Pathogenesis, Prevention, and Case Studies. Chichester, UK; Hoboken, NJ: Wiley-Blackwell, 2009; p. 583-98.*

19. *Centers for Disease Control and Prevention. Epidemic/epizootic West Nile virus in the US: guidelines for surveillance, prevention, and control. Available from http://www.cdc.gov/ncidod/dvbid/westnile/resources/wnv-guidelines-aug-2003.pdf*

20. *Han LL, Popovici F, Alexander Jr JP et al. Risk factors for West Nile virus infection and meningoencephalitis, Romania, 1996. J Infect Dis 1999; 179: 230–3.*

21. *Mostashari F, Bunning ML, Kitsutani PT et al. Epidemic West Nile encephalitis, New York, 1999: results of a household-based seroepidemiological survey. Lancet 2001; 358: 261–64.*

22. *Rappole JH, Derrickson SR, Hubálek Z. Migratory birds and spread of West Nile virus in the Western Hemisphere. Emerg Infect Dis 2000; 6: 319-28.*

23. *O'Leary DR, Marfin AA, Montgomery SP et al. The epidemic of West Nile virus in the United States, 2002. Vector Borne Zoonotic Dis. 2004; 4: 61-70.*

24. *Glass, WG; Lim JK, Cholera R et al. Chemokine receptor CCR5 promotes leukocyte trafficking to the brain and survival in West Nile virus infection. J Exp Med 2005;202: 1087–98.*

25. *Glass, WG; McDermott DH, Lim JK et al. CCR5 deficiency increases risk of symptomatic West Nile virus infection. J Exp Med 2006; 203: 35–40.*

26. *Partnership for the East Asian-Australasian Flyway. Available from http://www.eaaflyway.net/index.php*

27. *Christina L. Mosquitoes of public health significance in Singapore. Available from http://www.nea.gov.sg/cms/sei/EHI1slides.pdf (Accessed on 30 Aug 2010).*

The Epidemiological News Bulletin is published quarterly by the Ministry of Health, Singapore

Any comments or questions should be addressed to:

The Editor
Epidemiological News Bulletin
Communicable Diseases Division, Ministry of Health
College of Medicine Building, 16 College Road,
Singapore 169854
E-mail : Goh_Kee_Tai@moh.gov.sg
Lyn_James@moh.gov.sg