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NIPAH VIRUS : AN EMERGING PUBLIC HEALTH THREAT

INTRODUCTION

Nipah virus (NiV), a recently emerged, zoonotic paramyxovirus, was implicated as the cause of a highly fatal, febrile human encephalitis in Malaysia and Singapore in 1999 and in Bangladesh in 2001, 2003, 2004 and 2007. Outbreaks in India have been reported from West Bengal in districts bordering Bangladesh in 2001 and recently in April 2007.

Nipah virus, a new member of paramyxovirus family, was named after one of the affected Malaysian villages, Sungai Nipah. Dr Chua Kaw Bing from the University of Malaya, Malaysia, discovered the new virus on 18 March 1999. Nipah virus mainly affects pigs and humans. It first appeared in 1998 in Malaysia causing significant damage to the local swine industry as well as the loss of over 100 human lives. It was subsequently imported to Singapore via live pigs during March 1999 and led to 11 cases, with 1 death among abattoir workers.

Nipah virus is related to, but distinct from Hendra virus, another paramyxovirus first isolated in 1994 in Hendra in Brisbane, Australia. The two viruses form a new genus, Henipavirus, in the paramyxovirus family. Genetic characterization shows that the viruses vary by about 20%. Their behaviour is also different, in terms of the species they infect and the way they seem to be transmitted. Hendra virus does not transmit readily between animals other than flying foxes, while Nipah virus appears to be easily transmitted between pigs, and possibly to other animals.

GLOBAL SCENARIO INCLUDING INDIA

The first human cases of disease attributed to Nipah virus occurred in late September 1998 in the northern city of Ipoh in Malaysia. The cases were first attributed to the Japanese encephalitis (JE) virus; however, the epidemiology of the disease was not consistent with JE. Most of the cases were in adult males who had direct contact with pigs. In March 1999, Malaysian researchers identified the virus as a previously unknown paramyxovirus which was confirmed by CDC, USA. Scientists now conclude that the role of JE in the outbreak was insignificant. According to Malaysian authorities, of the 146 cases where tests were available, 116 tested positive for Nipah virus, 18 tested positive for both Nipah and JE virus, and 12 tested positive for JE.

Malaysian states in which these cases occurred are Perak, Negri Sembilan, and Selangor). Nipah virus was also confirmed in abattoir workers in Singapore, where many pigs imported from Malaysia were slaughtered.

By late 1998, through movement of infected pigs, the disease reached Sikamat, about 60 kilometres south of the Malaysian capital, Kuala Lumpur. By March 1999, the disease spread to the northern Malaysian, major pig-producing area of Bukit Pelandok.

There have been several outbreaks of Nipah virus in Bangladesh since 2001. In some outbreaks, more than 90% of infected people died. More recently, in Bangladesh, several outbreaks of Nipah virus-associated disease in humans have been described. Between April 2001 and February 2007, the Bangladesh Directorate of Health Services has recognized a total of 105 cases, at least 71 (67.6%)

of which were fatal. In marked contrast to the Malaysian outbreak, infection in humans was not associated with disease in pigs (pigs are uncommon in Bangladesh), and there is evidence of horizontal human transmission. Surveyed flying foxes populations in Bangladesh have shown serological evidence of Nipah virus infection.

Outbreaks of Nipah Virus – Since its discovery in 1999, the outbreaks of NiV have been reported in four countries including India. Some of the details are shown in Table-1 and Map- 1.

Indian scenario

Outbreak of Nipah virus in Siliguri town, West Bengal

An outbreak of acute encephalitis occurred in Siliguri town (Jalpaiguri district of West Bengal) of India between January 31 and February 23, 2001. Siliguri shares borders with Bangladesh, Bhutan, Nepal, and is close to China. The outbreak was investigated by a team drawn from All India Institute of Medical Sciences (AIIMS), New Delhi; National Institute of Communicable Disease (NICD), Delhi; National Institute of Virology (NIV); Indian Council of Medical Research, Pune and WHO Country Office. The outbreak caused widespread panic among the residents. Occurrence of cases and deaths among treating medical, nursing and paramedical personnel further compounded the matter and led to the closure of private health facilities. Absence of drug therapies effective in treating Nipah infection further increased case fatality and fear among the general public.

EPIDEMIOLOGICAL INVESTIGATIONS

A total of 66 probable cases and 45 deaths were reported between January 31 and February 23, 2001. Of the 66 cases, 5 left against medical advice, 45 expired and 16 survived. The case fatality was 68 percent. Study of the information collected during the course of investigations revealed possible epidemiological linkages in 43 cases.

These epidemiological linkage point towards person-to-person transmission and give a clear indication of the incubation period to be around 10 days with a range of 5-20 days. Age of the cases ranged from 12 to 70 years; the median age was 30 years. Fatality was

Table – 1 Outbreaks of Nipah Virus

S.No.	Country	Duration	Cases	deaths	CFR(%)	Remarks
1.	Malaysia	Sept.1998 to April 1999	265	105	40	93% of cases had occupational exposure to pigs. (Perak, Negri, Semblian, Selangor states)
2.	Singapore	March 1999	11	1	9	All were abattoir workers, who handled pigs imported from Malaysia.
3.	India	Jan-Feb 2001	66	45	68	investigations revealed possible epidemiological linkages in 43 cases and person-to-person transmission (Siliguri District, West Bengal)
		Jan-Apr 2007	5	5	100	Nadia District, West Bengal
4.	Bangladesh	Apr-May 2001	13	9	69	Meharpur District
		Jan 2003	12	8	67	Noagaon District
		Jan 2004	23	17	74	Rajbari, Faridpur, Manikganj, Jaibarhat, Naogaon districts. Mainly males and children affected
		Mar-Apr 2004	30	18	60	Faridpur District
		Feb – 2005	12	11	92	Tangail District – Food borne transmission suspected
		Jan-Apr 2007	15	8	53	1st outbreak in Thakurgaon district and later on in Kushtia district

maximum in older age group (>50year). Fifty nine per cent (39/66) of cases were males. There was clustering in space around one Nursing Home as many of its staff who got affected stayed in or around it.

All the cases that could be interviewed had a definite history of exposure to a case. No association with factors like travel outside Siliguri, visit by guest from outside Siliguri, attending funeral, exposure to injections, contact with animals/birds including pigs, exposure to any new or old insecticide or homeopathic remedies during one month prior to date of onset in cases was found.

Clinical presentation: The clinical illness started with fever, generally of mild to moderate degree and usually without chills or rigors. Headache was a prominent associated symptom; other being vomiting, bodyache and generalized weakness. Altered sensorium presenting with confusion and incoherent talks developed on day 3-4 followed by decreasing level of consciousness leading to coma in majority of cases; 34 per cent of cases had convulsions while 54 per cent had associated respiratory symptoms particularly in the later stage of illness.

There was no rash, haemorrhagic manifestation, jaundice, lymphadenitis, urinary or gastrointestinal symptoms, neck rigidity, cranial nerve involvement, hepatosplenomegaly, pleural effusion, or ascitis. Papilloedema was found in the terminal phase of illness in a few cases. Deep tendon reflex were diminished, plantars were extensor and intracranial tension was found to be raised in most cases. Heart was normal with no congestive cardiac failure but ECG suggested myocarditis in late stage of illness in some. In cases with respiratory involvement, tachypnoea, bilateral crepitations and evidence of acute respiratory distress syndrome (ARDS) were present.

Of the 8 recovered cases examined in a follow up visit, about 10 weeks after the outbreak subsided, 7 did not show any neuromotor deficit while one case that had developed paraplegia, had improved and was able to walk with support. However, all the cases continued to complain of varying degree of dizziness and weakness.

Entomological investigations: Entomological investigations did not reveal significant presence of Aedes aegypti (dengue) or Culex vishnui (JE vector).

Zoonotic investigations: An appraisal of the environment of Siliguri town to study its potential for possible zoonoses revealed dogs as seen usually in urban areas of India, very few pigs, few flying foxes (bats) Megachiroptera species and no horses. Cattle rearing were observed in the outskirts of the town. No definitive ecological evidence of an ecological support system for the natural harborage of zoonotic infection, in medium sized mammals, with the possibility of its transmission to man was observed.

Laboratory investigations: Biological samples from cases as well as contacts (blood, cerebro-spinal fluid (CSF), throat swab, urine, rectal swab) and necropsy materials (Brain, liver, lung tissue/ aspirate and blood clot from five fatal cases) were collected and processed at NICD, NIV, AIIMS and Defence Research and Development Establishment (DRDE), Gwalior, and Centre for Disease Control and Prevention (CDC), Atlanta, USA.

Various investigations including ELISA for IgM and IgG antibodies and virus culture were also carried out at various laboratories to rule out JE, dengue, leptospirosis, West Nile, measles, legionellosis, plague, malaria, Hanta virus, Herpes simplex virus and Enterovirus infections.

Laboratory investigations at the time of the outbreak did not identify an infectious agent. Because Siliguri is in close proximity to Bangladesh, where outbreaks of Nipah virus (NiV) infection were recently described, clinical material obtained during the Siliguri outbreak was retrospectively analyzed for evidence of NiV infection. NiV-specific immunoglobulin IgM and IgG antibodies were detected in 9 of 18 patients. Reverse transcriptase–polymerase chain reaction (RT-PCR) assays detected RNA of NiV in urine samples from 5 patients. CDC, Atlanta, USA and National Institute of Virology Pune finally concluded on the basis of extensive laboratory tests that the causative pathogen was Nipah virus. Sequence analysis confirmed that the PCR products were derived from NiV RNA and suggested that the NiV from Siliguri was more closely related to NiV isolates from Bangladesh than to NiV isolates from Malaysia. NiV infection was not previously detected in India.

Outbreak of Nipah virus in Nadia district of West Bengal April 2007

Cases of fever were reported in April 2007 from Nadia district of West Bengal. The cases presented with fever, headache and body ache mainly, with a few cases having episodes of vomiting, disorientation, respiratory distress and 5 cases ended fatally within 3-10 days of onset. None of the cases presented with cough, expectoration, rash, swelling of limbs, jaundice or loose stools.

Initially a team from Calcutta School of Tropical Medicine (CSTM) visited the area and carried out a brief investigation including collection of samples for laboratory analysis. Another team from NIV Pune collected samples from CSTM Kolkatta and carried out lab investigation. The central government also detailed a multidisciplinary team from NICD (National Institute of Communicable Diseases) Delhi which also carried out an outbreak investigation from 02-04 May 2007.

The analysis of the 5 deaths show that 3 belonged to Betai village (index case, wife and brother-close contacts) and two from Krishnanagar town (also with h/o close contact with the index case). The index case did not give any h/o travel to Bangladesh, however the border is just about 5 Km away from the village.

3 samples (including urine, CSF, brain and lung tissue from post mortem) from the 5 fatal cases were positive for Nipah Virus by RT- PCR. All these samples tested negative for flavivirus RNA. Samples also collected and analyzed at NICD turned out to be negative for Dengue. Entomological survey was carried out but significant Aedes breeding was not found.

Epidemiological linkages between cases point towards possible person-to-person transmission and incubation period of around 10 days. There was neither any concurrent illness in animals nor was there any history of exposure of cases to animals.

EPIDEMIOLOGY

Given the relatively recent emergence of NiV, many aspects still remain unclear. However, the available information about epidemiological features is as described below.

AGENT CHARACTERISTICS

Viral Classification

Nipah virus is a new member of Paramyxoviridae family classified under genus Henipavirus along with a similar virus Hendra virus with which it shares 70% to 88% nucleotide homology and 67% to 92% amino acid homology.

Virion Morphology and Genetic Composition

Henipaviruses are pleomorphic (variably shaped), ranging in size from 40 to 600 nm in diameter. Both the Hendra virus and Nipah virus genomes are non-segmented, single-stranded negative-sense RNA.

Environmental Survival

Environmental survival is not well documented. This is a point of concern for prevention and control programmes.

Biocontainment

Nipah virus is classified as requiring level 4 (BSL-4) security, the highest-level biocontainment procedures.

Transmission and Reservoir of the Nipah virus

Natural reservoir of NiV includes three species of Pteropid fruit bat (also referred to as flying foxes). NiV was isolated from fruit bats in Malaysia and Cambodia and sero positive bats have been detected in other parts of South-East Asia.

Nipah, presumably derived from a fruit bat virus, spread to swine and adapted over a few years in closely confined swine herds. Once established in the swine population, the virus had ample opportunity to adapt to human hosts.

Transmission among swine herds is not well documented and much of what is reported is through retrospective study.

Potential seropositive animals includes rats, cats, dogs, goats, and horses. It is unknown whether seropositive animals could be a source of disease transmission.

Close contact with infected animal secretions and/or tissues may transmit the virus between pigs. Pigs and other animals may also get infected by eating partially eaten (by fruit bats) fallen fruits or bat droppings. The apparent source of infection for humans is close contact with pigs. The specific routes of infection are yet to be determined; however it is thought that transmission of the virus is through direct contact with body fluids. Another theory is that humans may become infected via aerosol transmission from respiratory or urinary secretions. There appear to have been no definite cases of human-to-human transmission of the virus in Singapore and Malaysia outbreaks. However, person-to-person spread was reported during 2004 outbreak in Bangladesh and 2001 outbreak in India.

Retrospective investigations of Nipah virus encephalitis outbreaks in Meherpur (2001) and Naogaon (2003) in Bangladesh suggested that transmission may occur through close contact with other patients or from exposure to a common source. Although bats in the region had serologic evidence of infection, person-to–person spread may have been an important mode of transmission.

In outbreak of Nipah virus encephalitis in Bangladesh in January 2004, 23 cases and 17 deaths (CFR 74%) were reported from six districts (Map 1 shows only 2 districts). No link could be established between these cases and sick pigs or other mammals.

Another cluster of 30 encephalitis cases with 18 deaths (CFR= 60%) was reported during 13 March to 14 April 2004 in Faridpur district of Bangladesh. Laboratory investigations at CDC, Atlanta confirmed Nipah virus infection in 16 cases. Direct contact with ill patents is suspected to have played a role in the transmission of the diseases and spread of this outbreak.

HOST CHARACTERISTICS

The natural host of the virus is believed to be Pteropid fruit bats (flying foxes). Transmission of the virus is thought to occur through close contact with contaminated tissues or biological fluids (such as urine or saliva) either of the fruit bat host or more usually other infected animals. Affected hosts like pigs, humans and possibly dogs have been reported with clinical illness caused by Nipah virus. NiV has also been shown to cause clinical disease in swine as well as serologic changes in several common farm animals and in various bat species.

Viral Replication

Replication takes place in the tonsils and the respiratory epithelium. This suggests that the virus may be transmitted in part by pharyngeal and bronchial secretions.

CLINICAL PICTURE

In Animals

In pigs, NiV is associated with clinical signs and death, although the morbidity and mortality rates are not yet known. Infection in pigs in the Malaysian outbreak was highly contagious. Clinical disease was characterized by acute fever (>104 F), rapid and labored breathing; an explosive and non-productive cough (loud barking); and neurological changes. Crude case fatality rate was low (<5%), and notably, a large proportion of infected pigs were asymptomatic. Evidence of infection was also found in dogs, cats and horses with lethargy and aggressive behavior.

The clinical symptoms/signs of Nipah virus in humans

In Malaysian outbreak majority of human cases had an H/o direct contact with live pigs. They presented with following symptoms:

- Fever
- Migraine
- Vomiting
- Emphysema
- Myalgia
- Encephalitis (may relapse after recovery)
- Meningitis
- Disorientation
- Neurologic deficits (may persist after recovery)
- Coma
- Death

Case-fatality rate: 40%*

*From APHIS Center for Emerging Issues 1999.

In Humans

Nipah viruses have neurological and pneumonic tropisms. With Nipah virus, the predominant clinical syndrome in humans is encephalitic (whereas in pigs, the infection was characterized by acute fever and respiratory involvement with or without neurological signs). The pathogenesis of Nipah infection is associated with its ability to infect blood vessels and extravascular parenchyma in many organs, particularly in the central nervous system. The incubation period in Nipah virus encephalitis ranges between 4 and 18 days. The onset is usually with influenza-like symptoms and the disease may progress to encephalitis with disorientation, convulsions and coma. About 50 per cent of clinically apparent cases die.

LABORATORY DIAGNOSIS

Diagnosis should be undertaken as quickly as possible.

- Procedures for the laboratory diagnosis of Nipah virus infections include serology, histopathology, immunohistochemistry, electron microscopy, polymerase chain reaction (PCR), and virus isolation.
- The recommended initial screening test is ELISA serology which is a safe test to carry out in the laboratory as it does not amplify infectious virus.

➢ Sample for detection of human cases

Acute phase blood specimens, throat swabs, saliva and urine need to be collected from suspect cases. When possible, CSF should be collected from hospitalized patients.

For diagnoses post mortem, tissue samples (brain, tonsils, lungs, kidney, spleen and spinal cord) need to be collected for antigen detection and virus isolation.

➢ Sample collection, packaging and transport

For serology serum needs to be removed from the clotted blood samples within 24 hours before transportation to avoid haemolysis. Virus can be isolated from the CSF, respiratory secretions, and urine of the patient.

For histopathology and immuno histochemistry, formalin-fixed tissues especially multiple lung and airway samples are recommended.

All samples have to be collected and packed according to internationally recognized standards. For air transport to a laboratory (for example, an overseas reference laboratory), the serum samples should be packed by a trained person in accordance with International Air Transport Association. The recipient country lab will require a valid import permit, so prior consultation with the reference laboratory is necessary.

➢ Serological tests in human samples

1. ELISA

Several enzyme-linked immunosorbent assay (ELISA) systems including both indirect IgG ELISA and IgM ELISA have been developed for serological tests after initial investigations of Nipah virus outbreaks. ELISA serology can be conducted safely and quickly without access to BSL-4 facilities, and can be a most useful diagnostic tool. Both serum and CSF samples should be tested.

2. Serum neutralization tests

Neutralizing antibodies to Nipah virus can be detected in the sera using serum neutralization test (SNT) which is the accepted reference serological test, biosafety considerations require that this work be carried out in a BSL-4 facility. In developing diagnostic and surveillance capabilities for Nipah virus, a partnership with an international reference laboratory with BSL-4 capabilities is strongly recommended.

3. Virus isolation

Ideally, to confirm any new Nipah virus outbreak, virus should be isolated and this work be carried out only in BSL-4 facility. Nipah virus grows well in Vero cell line, with development of characteristic cytopathic effects (CPE) i.e. syncytia with the nuclei arranged around the periphery of the multi-nucleated cell. This arrangement differs from most syncytia seen in cell cultures with the closely related Hendra virus. Virus can be isolated from the CSF, respiratory secretions, and urine of the patient. For diagnosis post-mortem, brain, lungs, kidneys and spleen should be cultured. CPE usually develops within 3 days, but two 5-day passages are recommended before declaring it negative. Identification of virus isolates may be done by -

- Immunostaining of fixed infected cells.
- Neutralization with specific antisera.
- PCR of culture supernatants, and
- Electron microscopy.

Suspected new isolates should be sent to an international reference laboratory for molecular characterization.

4. PCR

Diagnostic assays for Nipah (and Hendra) virus are in routine use based on the M and N genes and can give the result very quickly. Real time PCR is the test of choice and can be performed directly on specimens or on the virus isolates.

➢ Post-mortem findings in humans

1. Immunohistochemistry

Immunohistochemistry (IHC) is highly recommended for initial Nipah virus diagnosis. It is one of the safest of tests as it is performed on formalin-fixed tissues. Since the primary pathology occurs in the vascular endothelium, viral antigen can be detected in a range of tissues. Thus it is important that laboratory submissions include a wide range of tissues, and not just lungs.

2. Histopathology

The main histopathological findings in affected human cases include a systemic vasculitis with extensive thrombosis and parenchymal necrosis, particularly in the central nervous system and lungs. Endothelial cell damage, necrosis, and syncytial giant cell formation are seen in affected vessels. Characteristic viral inclusions can be seen by light and electron microscopy. IHC analysis shows widespread presence of Nipah virus antigens in endothelial and smooth muscle cells of blood vessels. Abundant viral antigens can also be seen in various parenchymal cells, particularly in neurons. Infection of endothelial cells and neurons as well as vasculitis and thrombosis seem to be critical to the pathogenesis of this new human disease.

➢ Nipah virus diagnosis in pigs

The local veterinarian or animal husbandry departments should be alerted following suspected Nipah virus activity in humans or animals.

Nipah virus infection is highly contagious in pigs and by the time a farm is suspected to be infected, it is likely that a substantial proportion of pigs will have antibodies.

Necropsies should be conducted on recently dead and euthanized acutely diseased pigs and should always be conducted with integral protective suits being worn.

PREVENTION AND CONTROL

Treatment

- There is no specific treatment available for Nipah infection. Intensive supportive care is required for infected humans.
- Ribavirin, an antiviral drug, may have a role in reducing mortality among patients with encephalitis caused by Nipah virus, although further research is needed

Risk assessment in field investigations - general principles

One should follow/look for following:

- Review the situation prior to commencement of any examination of live or dead animals. Consider differential diagnoses based on the species involved, clinical syndromes, previous diagnostic tests and epidemiological features of the disease.
- Inquire whether (about):
  - The area has a history of particular zoonoses.
  - Presence of any assistants, farm workers or other people at the investigation site, and their likely proximity to potential sources of infection.
  - Investigation site in relation to any environmental features, which may increase the spread of infection as a result of the investigation (such as proximity to water sources, dams, public thoroughfares and other farming establishments).
- IEC: Communicate clearly any concerns or advise precautions to assistants and other people at the investigation site. Manage the investigation site in accordance with a duty of care.
- Avoid contact with secretions, excretions and body fluids of potentially infected animals while conducting clinical examinations or collecting specimens.
- Wear suitable protective clothing, including examination gloves. It should be preferably disposable.
- Keep the use of sharps to a minimum and be sure to dispose of scalpel blades and needles in an appropriately designed "sharps" container.
- Vector control measures should be followed.
- During examination and sampling of live animals, ensure adequate restraint to reduce the risk of accidental infection of personnel.
- Wash hands and equipment(s) after examinations or specimen collection. Disinfect protective clothing, refuse and biological waste or otherwise and dispose off safely.

Where Nipah virus infection is suspected as a possible differential diagnosis, appropriate protective clothing (PPE) and safe work procedures should be adopted.

Active case search for cases with acute febrile illness with respiratory or neurological symptoms and isolation of suspected patients and use of barrier nursing during care of these patients.

Health awareness regarding patient handling and patient care with special reference to possible contagiousness of the disease needs to be disseminated in the affected area immediately on reporting of the outbreak.

Community awareness through IEC regarding possible risk factors for the disease like consumption of raw date palm juice, close personal contact with patients, contact with domestic animals especially pigs, dogs, cattle and other animals.

Segregation of pigs in enclosures outside human habitation and reduction of contact of the general population with pigs.

Frequent hand washing with soap and water immediately after handling animals.

Safe disposal of potentially infected material

Travellers to the areas affected should be aware of the small but possible risk of infection with Nipah virus and follow any local guidance issued to minimize this risk.

Control Strategies following Nipah Outbreak

Upon the discovery of Nipah, Malaysia and Singapore developed national plans to help control the disease.

- **Phase I:** Immediate eradication by mass culling of pigs (this resulted in loss of over 1 million swine and had a significant impact on the pig industry in Malaysia but no cases reported since then)
- **Phase II:** Antibody surveillance of high-risk farms to prevent future epidemics

Other preventive actions included the following:

- A ban on transporting pigs within the country
- A total ban on porcine production (which has since been lifted)
- Education about contact with pigs
- Use of personal protective equipments for persons exposed to pigs

METHODS OF CONTROL

(A) Preventive measures

- Health education: about measures to be taken and the need to avoid fruit bats.
- Precaution by animal handlers: protective clothing, boots, gloves, gowns, goggles and face shields; washing of hands and body parts with soap before leaving pig farms.

(B) Control of patients, contacts and the immediate environment

- Report to local health authority: case report should be obligatory where ever disease occurs.
- Concurrent disinfection: Slaughter of infected pigs, horses or swine with burial or incineration of carcass under the supervision of health authority.
- Quarantine: Restrict movement of pigs and horses from infected farms to other areas.
- Investigation of contacts and source of infection: Search for missed cases.

Other Considerations for Prevention and Control

- Since the original source of transmission by various species of fruit bats, it may be possible to reduce the transmission of Nipah to pigs by removing the fruit source on a farm, import/export caution, and biosecurity planning should increase.
- Increased hygiene and updated protocols on pig operations are essential.
- Although the risk of transmission is considered low, healthcare professionals, research investigators, veterinary personnel, and individuals in close contact with pig production need to take special precautions considering the limited epidemiologic knowledge to date.

SUCCESS STORY IN MALAYSIA

A control program was developed by the Malaysian Cabinet taskforce, together with international experts. From 28 February 1999 to 26 April 1999, more than 900,000 pigs from affected areas were culled.

Ongoing surveillance demonstrates the success of the control program, with no new cases detected since May 1999.

NIPAH AS A BIOLOGICAL WEAPON

Nipah virus is important as a potential biological weapon (targeted to animals, humans, or both) for the following reasons:

Even a small outbreak in pigs could result in mass culling of affected herds, thereby causing substantial economic loss to the industry or to the national economy.

- Nipah virus can infect humans and the case-fatality rate may be as high as 50%.
- There is no effective treatment or vaccine for the disease in either pigs or humans (although ribavirin may reduce mortality in humans with encephalitis).
- Little is known about Nipah virus, so an outbreak in animals or humans could cause substantial fear and social disruption.
- The Centers for Disease Control and Prevention (CDC) has listed Nipah virus as a Category C, critical biological agent. Category C agents are emerging pathogens that could be engineered for mass dissemination in the future because of availability; ease of production and dissemination; potential for high morbidity and mortality rates and major health impact.

...about CD*Alert*

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