

JAPANESE ENCEPHALITIS ; A MAJOR HEALTH PROBLEM IN SRI LANKA

TISSA VITARANA

Director

Medical Research Institute

Colombo

Introduction

Encephalitis is an inflammation of the brain most commonly due to a viral infection. There are several viruses which can cause encephalitis. Studies done at the MRI show that the Japanese Encephalitis virus is the most important cause and probably accounts for over 80% of cases in Sri Lanka. Rabies is another important cause. Among the other viruses Measles, Mumps, Enteroviruses, Adenoviruses and Herpes simplex in that order, are causes of encephalitis in Sri Lanka.

Japanese Encephalitis (JE) is a serious public health problem with a high mortality, specially in children and old people, in many countries of Asia. The virus was first isolated in Japan in 1935 and the illness it caused was earlier referred to as Japanese B Encephalitis. This was because there was another type of encephalitis which was referred to as Japanese A Encephalitis. However, the latter was found to be identical with Von Economo's disease which was found in other parts of the world, and is not found any longer in Japan, and has been dropped. Therefore, the term Japanese B Encephalitis is no longer used, and this disease is now called Japanese Encephalitis.

The incidence of JE was high in Japan, Korea and China from about 1930 to 1960. A significant reduction was noticed from about 1965 in Japan after the implementation of wide scale immunization with a killed vaccine. With the use of this vaccine there was a drop in the incidence in Korea and Taiwan as well subsequently. However, the incidence in China has continued to

remain high. But in the 1970's the incidence of JE has increased in South East Asia particularly in Thailand, Vietnam and Burma.¹ In South Asia too there has been a significant increase - specially in India and Nepal. This increase has been evident in Sri Lanka after 1980 but it was most evident after the 1985/86 epidemic, in the Anuradhapura and Puttalam districts. Most of the cases of JE in all countries occur among children, but no age is exempt.

The Epidemiology

Japanese Encephalitis is an infection caused by a virus belonging to the genus *Flavivirus*. It is closely related to the Dengue viruses which are also present in Sri Lanka. These viruses are transmitted by mosquitoes and therefore referred to as Arboviruses (arthropod-borne viruses).

In nature, the main reservoir of JE infection is not human beings but various animals. Of these the most important is the pig, which is considered to be an amplifier host. This is because the virus is found at the highest levels for the longest period (3-7 days) in the blood of the pig. Therefore, a mosquito vector biting a pig has the greatest chance of getting infected.

Research done by us at the Medical Research Institute (MRI) showed that more than 80% of pigs tested in Ragama, Tissamaharama Anuradhapura and Chilaw districts had evidence of past infection. In contrast,

1 T. Umenai, R. Krzysko, A. Bektimirov and F.A. Assaad (1985) Japanese Encephalitis: Current Worldwide Status, Bulletin of World Health Organization, 63(4) : 625-631.

among cattle, goats and dogs only about 30-50% were infected. The figure among birds was much lower. Because virus is found for a shorter period and at lower levels in the other animals, the chance of a mosquito getting infected by them is much lower than from a pig.

Although about 140 species of mosquitoes have been described in Sri Lanka only a few appear to be involved in the transmission of JE. The main vector mosquitoes appear to be *Culex tritaeniorhynchus* and *Culex gelidus*. *Culex fuscocephala* and *Culex pseudovishnui* appear to be vectors of lesser importance. *Culex gelidus* appears to be more important in the western coastal belt and the suburban areas where they breed in shallow ditches and pools. *Culex tritaeniorhynchus* and the other vectors are found more in rice fields.

Females of these vector mosquito species get infected when they take a blood meal from an infected animal such as a pig. The virus multiplies in the cells of the mosquito, such as the coelomic epithelium, and then reach the salivary glands in about 9-12 days, after which the mosquito becomes infective.

The mosquito vectors prefer to feed on animals and transmit the infection to them. The spill over of infection to humans occurs when the absolute number of infected mosquitoes increases. Thus water logging of rice fields and pools (specially after rains) and the close proximity of these breeding sites to human dwellings promote the transmission to people. JE is therefore mainly a rural disease, though it can occur in the outskirts of towns which are close to rice fields or pools.

The JE virus does not produce any illness in most of the animals infected, with the exception of encephalitis in horses and abortion in pregnant pigs. Studies done in several villages in Sri Lanka by the MRI team suggest that only about one out of 300 to a 1000 infected people develop encephalitis. The others get a minor fever or do not show any illness at all.

Pathological and Clinical Features

In Japanese Encephalitis, nerve cells in the brain are damaged mainly in the mid-brain, cerebral cortex and

cerebellum. The anterior horns of the spinal cord may be affected. Blood vessels are dilated with occasional bleeding and there may be lymphocytes and other cells around them. Inflammatory cell infiltration of the brain and meningeal coverings may also occur.

After the virus enters the body following a mosquito bite it travels to the liver, spleen, and some lymph glands where the virus multiplies within the macrophages. The virus is then released into the blood stream after 5-12 days (incubation period) and with this fever occurs. The fever may be accompanied by chills (and shivering), severe headache, malaise and sometimes nausea and vomiting. In many cases the illness does not progress beyond this point and the fever comes down in 3-6 days. In a few, the virus may enter the brain and cause encephalitis.

The fever continues to be high with a relatively slower pulse. The face may take on a Parkinsonian look (dull, staring, mask-like), with thick retarded speech. Neck stiffness is common and muscular rigidity may occur. Altered consciousness (clouding of consciousness, excitement, confusion, disorientation, stupor and coma) generally occurs. Tremors and involuntary movements of various types and incoordination are common. Convulsions (fits) too are common specially in children. Paralysis occurs and may be trivial or extensive. In Anuradhapura, weakness of neck muscles was seen in some cases. Changes in reflexes are not prominent. Sensory changes are difficult to demonstrate because of altered consciousness.

Involvement of other systems, such as the myocardium of the heart can occur.

The case fatality (death rate) usually ranges between 20 and 40% but in some epidemics has exceeded 60%. In the recent (1985/86) Anuradhapura and Chilaw outbreaks it was about 20%. Death usually occurs within 10 days of the onset of illness. Death is due to the results of destruction of nerve cells and swelling of the brain. Patients may die of heart or respiratory failure or during fits.

Other causes of death are complications like pneumonia, urinary infection, lack of fluid and food, and bed sores

due to poor nursing care. The death rate has been reduced in the hospitals by reducing the swelling of the brain through the use of mannitol, preventing fits by suitable drugs, providing adequate food and fluid, and preventing and treating the complications like bacterial infections.

Unfortunately, upto 50% of those who survive have some remaining defects, which may clear-up gradually or even remain for life. These defects may vary from minor emotional or psychiatric disturbances to intellectual deterioration and even to complete paralysis and dementia.

It was not easy to establish the diagnosis of Japanese Encephalitis in a patient upto 1985, as two samples of blood or cerebro-spinal fluid (CSF) collected at a time interval of about 10 days had to be tested for Haemagglutination Inhibition (HI) or neutralizing antibodies. Usually the first sample had no antibody or only low levels, while the second sample had a significantly higher level of antibody. As a result, a diagnosis could only be established in those patients from whom two adequately spaced blood or CSF samples were received.

Isolation of the virus can be done using mice, tissue culture or mosquito inoculation techniques. These are time-consuming and require samples that have been maintained at a low temperature to ensure survival of the virus. It is therefore not a routine diagnostic procedure, but it is important from a point of view of research and control (choice of appropriate vaccine).

Since 1985 the development of the IgM capture Enzyme Linked Immunosorbant Assay (ELISA) by Donald Burke has made it possible to detect specific JE IgM antibody in a single sample of CSF (or blood) and make a definite diagnosis.

History of Japanese Encephalitis in Sri Lanka

The virus was first isolated in Sri Lanka from a human patient by Hermon et al. at the Medical Research Institute in 1968.² But its presence was suspected earlier when a child died of encephalitis at the School for the Blind at Mahawewa in June 1960. One of the serum samples sent to the Virus Research Centre at Poona, India was positive for group B arbovirus, probably Japanese Encephalitis.

While encephalitis occurred sporadically throughout the year in most parts of the country, the first major outbreak occurred in the Kurunegala district in 1971. This mainly affected children and in the course of that year 76 cases were admitted to the children's ward at Kurunagala Hospital. There was a high case fatality of 66 deaths and of the 10 who survived 5 had residual neurological deficits. Unfortunately, by the time the Medical Research Institute was informed, it was too late to establish the cause of the outbreak. In 1971 a study was made of encephalitis cases admitted to the Children's Hospital, Colombo and District Hospital, Gampaha.

Of the 31 cases admitted to DH Gampaha, 6 were serologically investigated at the MRI and one was found positive for JE. Out of 50 cases at the Children's Hospital, 4 were serologically investigated but there were no positives.

Thereafter, cases occurred sporadically throughout the island and for the 10 year period 1971 to 1980 there was an average of 1,030 hospital admissions each year for encephalitis with a mean case fatality rate of 38% (range of 25% - 45%). During this period etiological studies done at the MRI on a limited number showed that Japanese Encephalitis is the leading cause, accounting for 43.3%.³

2 Y.L. Hermon., M. Anandarajah. (1974) - Isolation of Japanese Encephalitis virus from the serum of a child in Ceylon. Ceylon Medical Journal, 19.02.93-99.

3 Tissa Vitarana (1982) - Viral Diseases in Sri Lanka. Viral Diseases in South-East Asia and Western Pacific - Ed. John S. Mackenzie, Academic Press, Sydney, 198-204.

A serological survey done by us in 1976 and 1977 in various parts of Sri Lanka indicated that Japanese Encephalitis occurred mainly in certain parts of the country (see Map).⁴ These were the Western coastal belt extending from Kalutara in the south upto Puttalam in the north and inland for about 10-15 miles to include Gampaha. Domestic pig rearing is common in this area and 83% of pigs tested had quite high levels of HI and NT antibodies against JE. The other areas affected were those with large rice fields and where wild pigs occur such as the districts of Kurugenala, Anuradhapura, Batticaloa and Tissamaharama. There was some JE activity in the Northern province as well. There was little or no JE in the hill country.

JE Outbreaks

At the beginning of 1985 (January and February), 32 cases of encephalitis were warded at the Anuradhapura Hospital. Of these, 13 were positive for JE by the newly introduced MAC ELISA test for JE specific IgM in the CSF. Two of these were also positive by haemagglutination inhibition (HI) and neutralization (NT) tests.

Commencing in November 1985 and extending to February 1986 there was a major outbreak of JE involving the Anuradhapura and Chilaw districts. In Anuradhapura there were 406 cases of encephalitis with 76 deaths.⁵ An unusual feature of this outbreak was that about 50% of cases occurred in the age group 20-50 years and there was a preponderance of male patients. In Chilaw there were 106 cases with 17 deaths, but this followed the classical pattern and mainly affected children.⁵

On the basis of ongoing field research studies it would appear that the factors contributing to the Anuradhapura outbreak were :

1. The increased area of rice cultivation and the excess rain, which led to pooling, leading to an increase of vector mosquito breeding;
2. Encouragement and increase of pig breeding in the area;
3. An increase of non-immune people for example through migration from areas, such as the hill country, where the JE virus is less active.

In the Chilaw area the main contributory factor appears to be the increase of vector mosquitoes, due to the excessive rains, and a build up of susceptible children.

A bigger epidemic of over 600 cases of JE occurred in the Anuradhapura District at the end of 1987 and early 1988.

Impact of Irrigation Schemes such as the Mahaweli

In the Anuradhapura district most cases during the last outbreak occurred in the 'H' area of the Mahaweli Scheme. Studies done in 1985 by a team from the MRI, AMC and AFC showed that there is past and present JE activity in the Mahaweli System 'C' and in the Lunugamwehera Scheme at Tissamaharama.⁶ However, the level of JE activity was much lower than in the 'H' area of the Mahaweli.

There is clearly a need to carefully monitor JE virus activity and vector mosquito densities in these irrigation schemes.

Control of JE in Sri Lanka

Immunization of children aged 1-10 years was commenced in the middle of 1988. In the highest risk areas

4 U.T. Vitarana, M. Kanapathipillai (1977). - Prevalence of Arbovirus Infections in Sri Lanka. Results of a 1976 survey compared with that of 1966. Proc. Sri Lanka Ass. Adv. Sci. 32,7.
 5 Vitarana, T., Jayasekera, N., Herath, P., et al. Japanese Encephalitis Outbreak in Sri Lanka ; Association with new irrigation schemes. Virus Diseases in Asia (Thongcharoen and Kurstak ed.) (1988) Mahidol University, Bangkok, 193-196.
 6 Tissa Vitarana, Pushpa Herath, Kingsley Kalpage, Nalini Jayasekera, Mervyn Wickramasinghe and Varuni Gunatillake (1985) Study of Mosquito Borne disease in some new irrigation schemes in Sri Lanka - Diseases Aspects. Paper read at seminar organized by IIMI at Digana. 9-13.

i.e. Anuradhapura and Puttalam districts, 2 doses of killed JE vaccine are given at intervals of 2-4 weeks and followed by a booster injection one year later. Studies done by the MRI have shown that the vaccines imported from Japan and Korea are equally effective.⁷ The antibody responses in a group of vaccinated children is being followed to determine when a further booster is required. This may be about 3-4 years after the 3rd dose, as is recommended in Japan.

Though it is desirable that all children aged 1-10 years should be immunized in all the endemic areas (or even the whole country) the government finds itself unable to find the money for this. Therefore, the MRI has conducted antibody surveys in the endemic areas and worked out an order of priority according to the JE virus activity in each area. With the available vaccine from 1989 onwards more and more children in the endemic areas are being brought under vaccine cover.

The benefit of this programme is already evident in that the occurrence of JE cases among children has come down to extremely low levels in the vaccinated areas.

Together with this, other control measures are also being implemented. These are vaccination of pigs and water management to reduce vector mosquito breeding. If the water level in the rice fields is lowered to ground level one day in a week then the mosquito larvae and pupae

die. While this is possible to do in large irrigation schemes with sufficient stored water, it is not possible in smaller irrigation schemes. Preliminary studies done at the MRI show that fish like guppies, are good feeders on vector mosquito larvae.

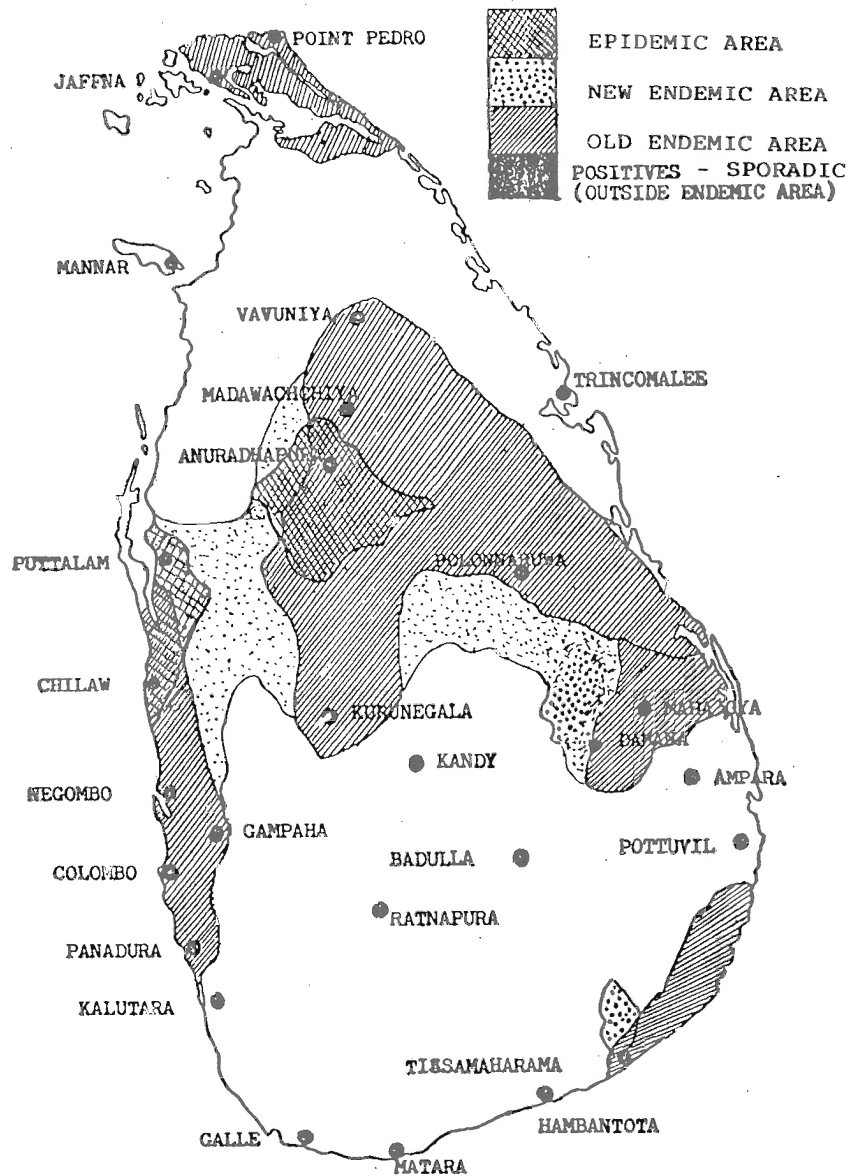
Further research is needed to find out how these fish will resist chemicals and poisons used in agriculture. The MRI has planned out such a research programme with the Agricultural Research Centre at Mahalluppallama. A study on the effects of agricultural chemicals on vector mosquito larvae is also planned.

The MRI also has established an early warning system by setting up 10 sentinel study points in the endemic areas. At these points, by regular monitoring it is possible to determine when vector mosquito densities begin to increase. When this occurs, the AMC is informed and they do widespread malathion spraying in surrounding areas around the pig-pens. At this time, health education programmes are intensified so that people will take suitable action to avoid being bitten by the vector mosquitoes which bite at night time, mainly between 6.00 p.m. and 10.00 p.m. and 3.00 a.m. and 6.00 a.m.

It is hoped that through a combination of all these control measures, the incidence of this dreadful disease can be reduced to low levels or if possible eliminated.

7 Vitarana U.T., Colombage G., Bandaranayake, V., Gunasekera H.D.N., Hettiarachchi L., Peiris L. - Antibody responses following the use of Japanese Encephalitis Vaccine manufactured in Korea and Japan in a sample of Sri Lanka Children. Proc. Sri Lanka Ass. Adv. Sci (1989), 45, 20.

MAP OF SRI LANKA INDICATING JAPANESE ENCEPHALITIS
 ENDEMIC AREAS



RABIES - A DANGEROUS PROBLEM

A. SATHASIVAM

*Consultant Virologist In-charge
Rabies and Vaccine Departments
Medical Research Institute
Colombo*

Introduction

Rabies is a Zoonosis, that is, an animal disease which is transferable to man. It is primarily a disease of animals (all warm blooded animals are prone to this disease) and man is incidentally infected due to his close proximity to certain animals like the dog and cat.

Disease and Transmission

This disease is caused by a virus and involves the brain and spinal cord of the infected man or animal and produces an encephalomyelitis (brain fever). Transmission is generally through the infected saliva of the animal and occurs in one of three ways:

1. Direct bite of an infected animal
2. Direct lick on an open wound or break in the skin
3. Direct lick on broken or unbroken mucous membranes like the eyes, nose and mouth.

The saliva of a diseased animal is infective for about a week before signs of clinical illness in the animal. There are instances - not in Sri Lanka, where the infection has spread by the respiratory route, that is by inhalation or breathing.

Incubation Period

The incubation period of the illness is the time taken for clinical manifestations to occur after the bite of the animal. This ranges usually from 10 days to 6 months, the average being 1 1/2 to 3 months.

Once the clinical illness (that is the disease) develops, the patient or animal dies within 4-7 days. In Sri Lanka the disease is 100% fatal, although cases have been documented in other parts of the world of recovery from the disease. Hence a dog, an animal or a man suffering from rabies will not live for longer than 10 days at the most.

Infectivity

Not all animals or humans bitten by rabid animals develop rabies. Only 36-40% of animals or man bitten by rabid animals develop the disease. This is mainly because the virus is only intermittently excreted in the saliva and not all the time.

Mode of Spread

The virus multiplies at the site of the bite for about 72 hours after which it travels up the peripheral nerves to the brain and spinal cord where it multiplies and produces the disease. The virus then comes out into all parts of the body so much so that all tissues become infective namely, cornea of the eyes, salivary gland, gastro intestinal tract, genito urinary tract, mammary glands, skin etc. Thus the saliva, faeces, urine, milk, perspiration etc. are infective secretions.

Diagnosis of Rabies

The laboratory diagnosis of rabies is only done at present at the Rabies Laboratory of the Medical Research Institute which is open day and night on all days (whether a normal working day or holiday) to accept specimens

for diagnosis. The specimen to be sent is the head of the animal or brain of human beings. This should be sent preferably in ice within 10-12 hours of the killing or death of the animal. Decomposition sets in after that period and no tests can be performed on such a specimen. Disinfectants or preservatives should not be added. It is best to send specimens by personal messenger and not by post or train. The parcel should be packed in such a way that the highly infective material, namely brain matter and secretions ensuing from it do not escape out and infect the person bringing the parcel or any person accepting the parcel at the MRI. A leak proof polythene bag or a tin with a well fitting lid, is suggested for the purpose.

Usually the result of the first test which is done on the same day (if the specimen comes within working hours) is available on the same day. In first test negative cases a second test is performed. This is done only twice a week and hence the result of this will usually be available within 72 hours of receiving the specimen. For certain scientific reasons, about 30% of first test negative specimens become positive in the second test.

Management of a Case of Dog or Animal Bite

Since there is no treatment, one should remember the old adage with a slight modification namely: "Prevention Is Better Than No Cure".

Management of a case is broadly divided into two:

1. Local treatment of the wound

Immediate and thorough washing of the wound is essential. Copious amounts of water and soap, if available should be used. In this way, a certain amount of the virus could be washed away.

Then, advice should be obtained from the MRI or the nearest government hospital. It has been shown that the incidence of this disease can be markedly reduced with local therapy alone.

2. Specific treatment

If indicated, anti rabies vaccine treatment should be started without delay. In severe exposure anti rabies serum should be given before vaccine treatment.

Tetanus prophylaxis and measures to control bacterial infection by the use of antibiotics is also indicated.

Control of Rabies

The best way to solve this dangerous problem is by controlling the disease in animals especially dogs and cats. This consists of vaccination of pet animals and the destruction of stray dogs and cats.

Prophylactic Immunisation

Those persons who are regularly or frequently exposed to the risk of rabies like veterinarians, laboratory workers etc. should be prophylactically immunised against the disease.

RABIES

WHAT TO DO IN AN EMERGENCY

1 TREAT LOCALLY

Immediate and thorough local treatment of all bite wounds and scratches is perhaps the most effective preventive measure. The wound should be thoroughly cleansed and flushed with soap and water as soon as possible. Tetanus prophylaxis and measures to control bacterial infection should be given as indicated.

2 DECIDE ON TREATMENT

Every exposure to possible rabies infection must be evaluated individually. Primary consideration must be given to the fact that treatment is rarely, if ever, of value once the clinical signs of rabies become apparent.

If the decision is to treat, start immediately, *the sooner the better*. The recommendations given in the table are only a guide. They should be applied in conjunction with knowledge of the animal species involved, the circumstances of the bite or other exposure, the vaccination status of the animal and the presence of rabies in the region.

Bear in mind that approximately 20% of persons who are bitten by animals known to be rabid have developed rabies if not treated with IMOVAX RABIES VERO VACCINE and Rabies Immune Globulin (Human) (IMOGAM RABIES SERUM).

However, rabies rarely develops unless there has been an actual penetration of the skin by teeth, saliva or mucous membrane contact. Prompt treatment is of critical importance where there have been multiple bites of the head, neck or face, as the incubation period is considerably shortened and symptoms may appear as early as nine days following exposure.

Animal Species	Condition of Animal At Time of Attack	Treatment of Exposed Person (1). All bites and wounds should immediately be thoroughly cleansed with soap and water (see preceding text).
Domestic: dog and cat	Healthy and available for 10 days of observation	None, unless animal develops rabies (2).
	Rabid or suspected rabid	IMOGAM RABIES SERUM (3) and IMOVAX RABIES VERO (VERORAB) (4) (6).
	Unknown	IMOGAM RABIES SERUM (3) and IMOVAX RABIES VERO (VERORAB) (4) (6).
Wild: skunk, bat, fox, coyote, raccoon, bobcat, and other carnivores.	Regard as rabid unless proven negative by laboratory test (5)	IMOGAM RABIES SERUM (3) and IMOVAX RABIES VERO (VERORAB) (4).
Others: livestock, rodents, rabbits and hares.	Consider individually: provoked bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice and other rodents or rabbits and hares almost never call for antirabies prophylaxis.	

(1) If antirabies treatment is indicated, both IMOGAM RABIES SERUM and IMOVAX RABIES VERO (VERORAB) should be given as soon as possible, regardless of the interval after exposure. If R.I.G.H. inadvertently was not given when the vaccination was begun it can be given up to the eighth day after the first dose of vaccine was given. Persons who have been previously immunized with Rabies Vaccine and were known to have protective rabies antibody titer should receive only vaccine.

(2) Begin treatment with IMOGAM RABIES SERUM and IMOVAX RABIES VERO (VERORAB) at first sign of rabies in biting domestic animals during the usual holding period of 10 days. The symptomatic animal should be killed immediately and tested.

(3) If IMOGAM RABIES SERUM is not available, use antirabies serum of equine origin. Do not use more than the recommended dosage.

(4) Discontinue vaccine if fluorescent antibody tests of brain section of the animal is negative.

(5) The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

(6) Discontinue vaccinal treatment if animal remains healthy for 5 days.

These products are available in the following Zoellig Pharma Branches:

1. San Fernando, La Union	Tel. No. 41-307
2. Dagupan City	27-80
	21-91
3. San Fernando, Pampanga	61-54-49
4. San Pablo City	41-05
5. Legaspi City	22-23
6. Iloilo City	742-28
7. Bacolod City	2-26-15
8. Cebu City	7-50-14
9. Cagayan de Oro City	33-23
10. Davao City	7-26-12
11. Tacloban City	323-2222

In Manila, also available at:
Manila Sanitarium and Hospital
Makati Medical Center
Mercury Drugstore and selected branches
in Manila & Suburbs
Centro Drug Store in front of MOH



3 DOSAGE AND ADMINISTRATION

INSTRUCTION FOR USE: The freeze-dried vaccine must be reconstituted in its vial with the 0.5 ml. of diluent supplied in the disposable syringe. Gently stir the contents until completely dissolved and withdraw the total amount of the dissolved vaccine into the syringe.

DOSAGE:

RABIES IMMUNE GLOBULIN (HUMAN) IMOGAM RABIES®

20 I.U./Kg. body weight. If possible, up to half the dose should be used to infiltrate the wound.

IMOVAX RABIES VERO (VERORAB)

5 (0.5 ml.) doses given on days 0, 3, 7, 14, 30, (90 optional)

(Doses given on Day 0 (a single dose) / day (one injection))

IMOVAX RABIES VERO (VERORAB)

Now the outstanding choice combining high efficiency with cost effectiveness.

ADMINISTRATION: Rabies Immune Globulin (Human) IMOGAM RABIES® is administered only once at the beginning of antirabies prophylaxis to provide antibodies until the patient responds to vaccination. (IMOGAM RABIES SERUM) after preparation of the injection site, is injected intramuscularly, e.g., in the gluteal region.

IMOVAX RABIES VERO (VERORAB) is administered on days 0, 3, 7, 14, 30, and (90 optional). After preparation of the injection site, immediately inject the vaccine intramuscularly, e.g., in the deltoid region or subcutaneously.

NOTE: NEVER ADMINISTER RABIES VACCINE IN THE SAME INJECTION SITE AS R.I.G.H.

ADVERSE REACTIONS: Local or mild systemic adverse reactions to R.I.G.H. and to Rabies Vaccine are infrequent; they do not contraindicate continuing immunization and may be treated symptomatically.

For further information, please consult I.M. leaflets of Rabies Immune Globulin (Human) and IMOVAX RABIES VERO (VERORAB) IMOGAM RABIES SERUM

To order or for more information on Institute Merieux RABIES VACCINE H.D.C.S. and IMOGAM RABIES:

Write or Call

RHÔNE-POULENC PHILIPPINES, INC.

3rd Floor, ITC Building
337 Gil J. Puyat Avenue
Makati, Metro Manila
Telephone: 816-17-09 to 16

Manufactured by:

INSTITUT MERIEUX
58, Avenue Leclerc
69007 Lyon, France

Distributed by:

Chem. inc.

7224 Malugay Street
Makati, Metro Manila
Telephone: 87-10-51 to 58
87-20-51 to 54

SOME PARASITOLOGICAL ASPECTS OF HUMAN MALARIA IN SRI LANKA

JAYARATNE PINIKAHANA

Senior Lecturer in Sociology

Department of History & Sociology

University of Ruhuna

Introduction

Malaria is caused by a germ called the malaria parasite (protozoa). A parasite is a creature that lives at the expense of another living creature, its host. Human parasites include viruses, bacteria, fungi, protozoa and worms (Wingate, 1985 p 330). The malaria causing parasites are protozoans and four of these malaria parasites of man have been generally recognized.

Plasmodium malariae (Laveran, 1881)

Plasmodium vivax (Grassi and Feletti, 1890)

Plasmodium falciparum (Welch, 1897)

Plasmodium ovale (Stephens, 1922)

The life of the malaria parasite can be divided into two phases; asexual and sexual. The life cycle is shown diagrammatically in figure 1. The asexual phase starts with the injection of the sporozoites into a host by a mosquito. As soon as the sporozoites are transmitted, they make their way in the blood to the liver where they remain for six to twelve days developing schizonts. At the end of this period, these schizonts divide into a large number of merozoites. The nucleus of merozoites invade the red cells and grow first to trophozoites and then into erythrocytic schizonts. After a certain time, some of the parasites in the blood develop into sexual forms called gametocytes (Bruce Chwatt, 1980; Bailey, 1982). The male gametocytes fertilize with the female gametocytes in the mosquito's stomach which result in a zygote (Dutta and Dutt, 1978). The zygote becomes encysted into a small cyst called an oocyst. The oocyst divides a number of times to produce sporozoites. The sporozoites then invade the salivary glands (Imms, 1964).

The malaria parasites need some ecological base in order to continue the life cycle. They are unable to survive inside the mosquito in extreme temperatures, for example below 10°C and above 32°C.

The types of malaria parasites in Sri Lanka

Plasmodium vivax and *Plasmodium falciparum* have been identified as malaria parasites which cause malaria in Sri Lanka (Tams, 1980; Kodisinghe, 1987). Table 1 shows the differentiation of *P.vivax* and *P.falciparum* in Sri Lanka. This table shows a sharp increase in the prevalence of both vivax and falciparum malaria since 1982 except a slight fall in falciparum malaria in 1984 and vivax malaria in 1985. The annual administrative report of the Anti-Malaria Campaign in Sri Lanka in 1985 referred to the fact that the environmental changes such as those known to occur in major irrigation and development projects may possibly favour the transmission potential and vectorial capacity of the mosquitoes. It was also revealed in the annual reports in 1984 and 1985 that the majority of the cases were found in gemming areas and the shifting cultivation areas. An increase in blood filming activities during this period also resulted in reporting more cases throughout the island. Two fold increase in falciparum malaria in 1986 was due to a sudden outbreak which occurred in several areas in Sri Lanka (Administrative Report, 1986).

Although *Plasmodium malariae* was reported in the 1960's, it disappeared with the resurgence of *P.vivax* in 1967. Since 1970 no *Plasmodium malariae* cases have been reported in Sri Lanka. The historical and contemporary data indicate that *Plasmodium vivax* was the predominant malaria parasite in Sri Lanka. This species of malaria parasite of man occurs in large areas of the tropics and is less common in tropical Africa (Bruce Chwatt, 1980). The time taken for this species to develop in a mosquito is 10 days at 25°C and 18 days at 20°C. The incubation period which is the time from infected bite to appearance of symptoms of disease in man is usually between 12 and 17 days, but sometimes it may be

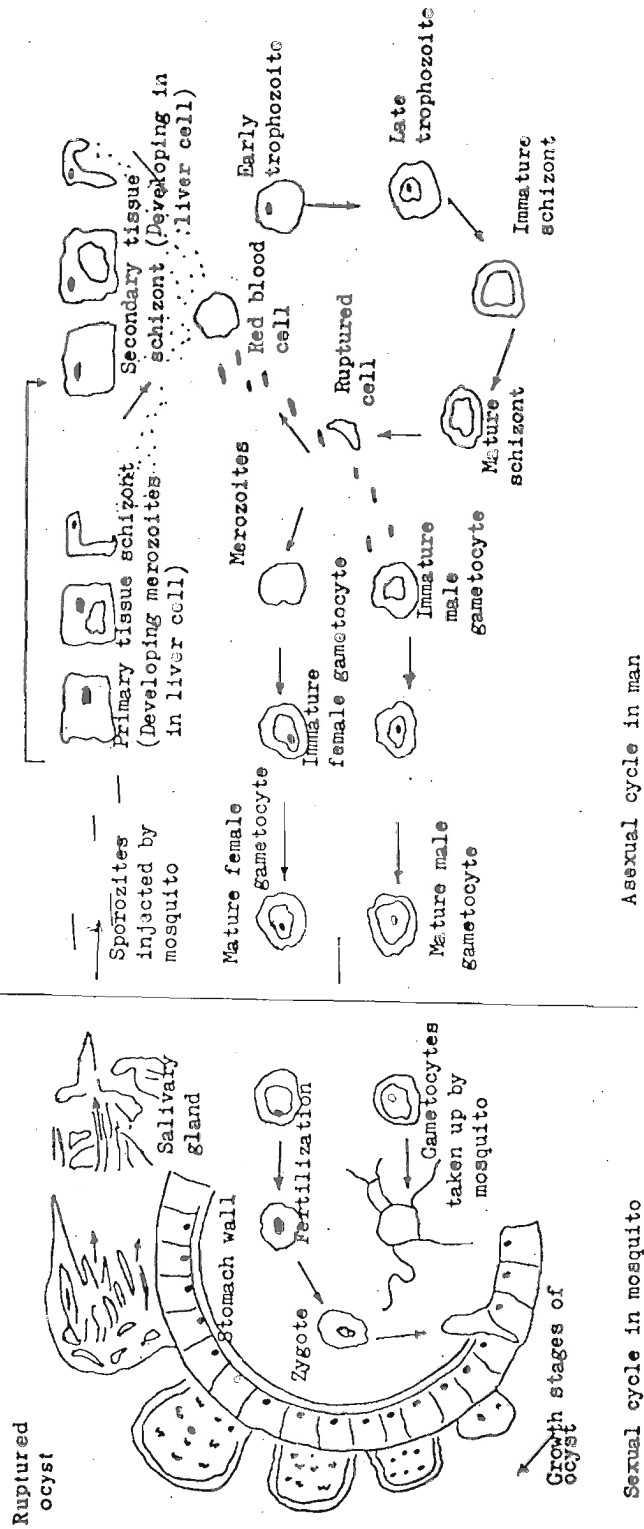


Fig. 1 The Life Cycle of the Malaria Parasite in the Mosquito and in the Human Host. (Balley, 1982)

Table 1
Differentiation of human malaria in Sri Lanka (1980-1986)

Year	Total	P.V.	P.F.	Mixed	% P.V.	% P.F.	% Mixed
1980	47954	46477	1425	52	96.9	2.9	.1
1981	47383	46143	12112	29	97.3	2.5	.06
1982	38566	36967	1541	58	95.9	3	.1
1983	127264	122764	4341	159	96.5	3.4	.1
1984	149470	145711	3593	165	97.5	2.4	.1
1985	117816	104759	12535	522	88.9	10.6	.5
1986	412521	328443	82689	1389	79.6	20.1	.3

Source: Administrative Reports, AMC

prolonged to 8-9 months (Bruce Chwatt, 1980). The time taken for the parasite to develop in human blood cells is about 48 hours. The malaria attack begins with headache, pain in the back and nausea. The primary fever is more severe in non immune than in partially immune persons (W.H.O., 1986). The fever is irregular for 2-4 days and the swings of fever occur between the mornings and the evenings. The cold, hot and sweaty stages become apparent in the afternoon or evening and the temperature may rise up to 40°C or higher. Dizziness and drowsiness may occur and the spleen is palpable during the second week of the attack. The headache, nausea and vomiting are usually less severe in vivax than in falciparum malaria. Relapses may occur in about 60% of untreated cases after a period of recovery (Bruce Chwatt, 1980). The duration of the untreated infection with *P.vivax* is about 2-3 years (Colbourne, 1966; Bruce Chwatt, 1980).

Plasmodium falciparum causes malignant fever, the most dangerous kind of malaria. *Plasmodium falciparum* is the major infection in areas of endemic malaria in Africa, in north west India and Sri Lanka (Bruce Chwatt, 1980). Falciparum parasite takes about 13 days at 25°C and 30 days at 20°C to develop in mosquito and the time from infected bite to the appearance of symptoms of disease in man is usually about 14 days, but it may be as short as 7 or as long as 27 days (Colbourne, 1966). The falciparum malaria attack starts with headaches, pains in the back, chill, nausea and perhaps vomiting. The attacks are frequent and irregular at intervals from a few hours to two days (Win-

gate, 1985). As the disease develops, symptoms increase and the hyperpyrexia with temperature well above 40°C is common together with convulsions. Sweating may occur even when the fever is low and the nausea, vomiting and diarrhoea may increase in severity. The spleen and the liver may become enlarged and slight jaundice may also be present (Bruce-chwatt, 1980). In falciparum infection, the fever may continue to be irregular throughout its course and the hot and cold stages.

The W.H.O (1986) divides the falciparum attack into three distinct stages; the cold stage, the hot stage and the sweating stage. In the cold stage, the temperature rises rapidly to 39°-41°C (102°-106°F) and the headache is often severe. The patient may feel hot and the headache is intense at the hot stage which lasts about 6 hours. The sweating follows the hot stage and continues for an hour or so. After the primary attack of falciparum malaria, there follows an afebrile interval of 48-72 hours. In falciparum infection, the headache, nausea and vomiting are more severe than in *P.vivax* and there is a greater tendency towards the development of delirium, haemolytic jaundice and anaemia (W.H.O., 1986). The falciparum malaria can cause a kind of dysentery and cerebral malaria (Wingate, 1985). The cerebral malaria particularly occurs when non immune persons have remained untreated for 7 to 10 days after development of the primary fever. At this time, the patient's condition deteriorates rapidly with increasing headache and drowsiness with light coma. The mortality of falciparum malaria is much greater than in other forms of malaria and those who survive with continuing falciparum infec-

tion with inadequate treatment or no treatment may suffer a few months with febrile episodes, malarial anaemia and body weakness (W.H.O., 1986). The falciparum infection may persist in man for about 2 years if not treated (Colbourne, 1966).

There are many drugs for treating vivax and falciparum malaria, all highly effective. The oldest, quinine is seldom used because of its toxic effects (Wingate, 1985). The newer synthetic drugs like chloroquine, amodiaquine and primaquine are recommended for vivax and falciparum malaria in different doses at different stages. There are two stages of treatment for both strains of malaria. The presumptive treatment is usually made when the diagnosis is not confirmed and the radical treatment is made after the diagnosis is confirmed in a blood test (W.H.O., 1986).

Summary

Malaria is caused by a parasite. The malaria causing parasites are protozoa and four of these malaria parasites of man have been recognized; *Plasmodium malariae*, *Plasmodium falciparum*, *Plasmodium vivax* and *Plasmodium ovale*. *Plasmodium vivax* and *Plasmodium falciparum* have been identified as malaria parasites which cause malaria in Sri Lanka of which *P.vivax* is the predominant parasite. However, *Plasmodium falciparum* is the most dangerous kind of malaria which is often fatal if the treatments are not taken.

REFERENCES

- BAILEY, N.J.T. (1982)
The Biomathematics of Malaria (London, Griffin)
- BRUCE-CHWATT, L.J. (1980)
Essential Malariology (Heinemann, London)
- COLBOURNE, M. (1966)
Malaria in Africa (Oxford University Press)
- DUTTA M M AND DUTT A K (1978)
Malaria Ecology: A Global Perspective.
Social Science and Medicine Vol 12 p 69-84
- IMMS, A.D. (1964)
General Text Book of Entomology (Methuen, London) p 886
- SAMARASINGHE, L. (1986)
Annual Administrative Report of the Anti Malaria Campaign in Sri Lanka
- TAMS REPORT (1980)
Environmental Assessment; Accelerated Mahaweli Development Programme.
(Colombo) Vol 14
- WINGATE, D. (1985)
Medical Encyclopedia (Penguin Books)
- WHO (1986)
Expert Committee on Malaria (Technical Report Series No. 735)

ZOONOSES

M.C. ATTAPATTU

Deputy Director

Medical Research Institute

Colombo

Diseases spread by animals - generally classified under the broad heading "Zoonoses". Zoonoses have been defined as those diseases which are transmitted to man from vertebrate animals.

Zoonoses are generally considered as an occupational hazard faced by agricultural, industrial and laboratory workers and animal handlers. eg: bovine tuberculosis in farmers, salmonellosis in food processors.

Major Zoonoses include

1. Tuberculosis
2. Brucellosis
3. Leptospirosis
4. Salmonellosis
5. Typhus Fever or Tick Fever
6. Plague
7. Rabies
8. Fungus infections - *M.canis*
M.equinum
M.gallineum
9. Arbovirus infections - eg. Japanese encephalitis

Tuberculosis

This disease is caused by a bacterium known as *Mycobacterium tuberculosis*. This bacterium exists in two forms, the human form and the bovine form. The bovine form is responsible for disease in cattle.

This disease is transmitted from cattle to humans mainly through consumption of milk from infected cows. This bacillus is secreted in the milk.

Prevention

1. Pasteurization kills the tubercle bacillus. Therefore if milk is either pasteurized, or boiled or sterilized, this bacterium is killed. Transmission to humans is

avoided by following these procedures. Raw untreated milk should never be consumed.

2. Spread to humans can also be controlled by routine testing of cattle and isolating the animals which give a positive tuberculin reaction, and ensuring that only tuberculin negative animals are used for milking purposes.

Brucellosis

This disease is caused by bacteria belonging to the genus *Brucella*. There are three strains of *Brucella*. *B.abortus*, *B.mellitensis*, and *B.suis*. *B.abortus* causes disease in cattle, *B.mellitensis* in goats, and *B.suis* causes infection in pigs.

Man get infected accidentally by consumption of infected milk or milk products such as butter and cheese or by handling infected animals. Infected animals excrete these bacteria in their discharges such as urine or milk or vaginal secretions. Thus it is mainly an occupational disease in farming communities, veterinarians, and in butchers. The bacterium can get into the body through minute abrasions in the skin, through the alimentary and respiratory tracts and sometimes through the conjunctiva of these workers.

Prevention of human infections can be achieved by pasteurization of milk and ensuring that other products such as cheese and butter are made from pasteurized milk.

This disease is characterized by intermittent fever accompanied with gland swellings. It is also one of the causes of pyrexia of unknown origin. Can be diagnosed in the laboratory by a test for antibodies produced in the infected humans.

Human infection by *B.suis* is almost entirely an occupational infection arising through contact with infected pigs or pig meat.

Leptospirosis

This is caused by a fairly large spiral bacterium known as *Leptospira*. The disease can occur in a number of animal species such as cattle, pigs, goats, horses and rodents. This is a disease transmitted most often by infected rodents. Epidemics occur in Sri Lanka from time to time. This disease is endemic in certain parts of Sri Lanka. Leptospirosis is normally spread through contaminated water. The bacterium responsible is shed to the outside through the urine of the animal having this organism. The leptospira survive in the contaminated water and get into the human body through minute cuts and abrasions in the skin and through the mucus membrane of the mouth, lips, and nose and through the conjunctiva. Thus the disease can be contacted by working in infected water or by washing in infected water. Generally affects persons who work in marshy lands, like farmers, fisherman and fish handlers who work in contaminated water, or in abattoir workers when they handle infected animals.

Symptoms

Fever, body pain, conjunctival infection, usually accompanied by jaundice are the symptoms of the disease. It is locally known as "Kahauna" and is well known and recognized in certain areas in Sri Lanka. Jaundice may not always be present.

Laboratory diagnosis

The disease can be diagnosed conclusively by testing the blood for antibodies using a test known as Leptospira Agglutination Lysis test. In the acute stage, the organism can also be cultured from the blood, urine or CSF of the patients. This is done by injecting the samples of blood etc. into hamsters or guinea pigs and isolating the organism from these animals.

Typhus fever or Tick fever

Transmitted to humans through the bite of a tick that lives in rodents. This is caused by a small organism called *Rickettsia*.

These are neither bacteria nor viruses. This is primarily an infection of rodents and man is accidentally infected.

Diagnosis

It is diagnosed in the laboratory by detection of antibodies in the blood of infected persons. The test is known as the Weil Felix test and the disease is known as Weils disease.

Salmonellosis

Another disease that spreads from a variety of animals is salmonellosis which is generally contracted by eating food or food products made from infected animals. It is transmitted mainly through eggs, milk, meat and meat products.

Symptoms

Eating contaminated food causes abdominal pain, diarrhoea and vomiting accompanied by fever and headache. Symptoms appear a few hours after eating contaminated food.

Prevention

The disease can be prevented by eating food that is thoroughly cooked and avoiding eating uncooked or inadequately cooked food. It is also important to bear in mind that food can be contaminated after cooking (even if the cooking has been carried out properly). This can happen if the kitchen hygiene is not maintained. i.e. if utensils such as knives used in cleaning of infected food is used to process cooked food.

Control and prevention of infection in animals is also an important factor in prevention.

Rabies

Rabies is a very dangerous and fatal zoonosis which spreads from infected animals. Spread to man especially from infected dogs. But infections can also occur from the bite of infected rats bandicoots, and other domestic or wild animals.

This disease is fatal and is caused by a virus called rabies virus. It is introduced into man by the bite of an infected animal. The virus travels along the nerves to the brain and causes an inflammation of the brain. Patient invariably dies of the disease which is called hydrophobia.

This name is given to the disease because infected patients are unable to drink water due to a spasm of the throat muscles.

Prevention

1. By preventing infection among animals through vaccinations of the domestic dog population.
2. Advise regarding the management of any person bitten by an animal suspected of being rabid should be obtained immediately.
3. As a first aid measure the wound should be cleaned with soap and water immediately after a bite.

Anthrax

This is mainly a disease of cattle and is spread to man accidentally. Human infections are rare and are generally found in farm workers handling infected animals.

The anthrax bacillus can thrive for long periods in animal products such as hides, hair and wool. The main source of infection to humans are infected carcasses, wool, hair and hides.

Plague

Caused by a bacterium known as *Pasteurella pestis*. This disease is not very common today. In the past plague has been responsible for many epidemics and has accounted for thousands of deaths. It is a disease of rodents where the bacterium is transmitted to man through a flea. It has

now been controlled to a great extent and no epidemics have occurred in the recent past.

Fungal Infections

Certain types of ringworm infections are found in dogs, cats, cattle and fowl. These are transmitted to humans through direct contact. Cattle, fowl, and horse ringworm are generally common among farm workers. In the case of ringworm from dogs and cats, these are generally found among children who come into contact with infected pets. Each of these animals are infected by a different species and therefore it is possible to find out the source of infection by studying the infecting species in humans. For example *M.canis* caused infection in cats and dogs, *M.equinum* caused infection in horses, and *M.gallinae* caused infection in fowl.

Arbovirus Infections

The most important arbovirus infection transmitted to man from animals is Japanese encephalitis. This is transmitted from infected pigs to man by the bite of a mosquito. This disease is especially prevalent in pig breeding areas. Prevention is achieved by preventive vaccination of pigs in endemic areas, and by vaccination of the susceptible population.

Zoonoses therefore present a major health hazard and the community at large should be aware of and be informed of the modes of spread and control. With proper precautionary measures, the majority of these diseases can be prevented.

AIDS-THE SINISTER THREAT TO MANKIND*

MANOHARI SANDANAYAKE

Scientific Officer

NARESA

Colombo 7

All newspaper readers are well aware of AIDS, the dangerous disease now fast turning into a threat for all mankind. In this context, it is quite essential for us to have some understanding of the nature of this dreaded disease and also why it is named AIDS.

Human beings are susceptible to many illnesses. Even a commonplace illness, usually considered a minor ailment, can prove itself fatal if it develops into a chronic stage.

However, normal human beings do not contract each and every disease that is there around them. Nor do they succumb to any illness easily. This is because human beings (and animals) have an inherent ability to resist many diseases. This ability is termed immunity. If by some means or the other, this immunity is destroyed, any minor illness can prove fatal to such immunity deficient persons.

There is a particular virus capable of destroying this inherent immunity in man. Once the virus enters the body of a victim and multiplies itself to a dangerous level, a certain disease condition becomes manifested. The chief characteristic of this condition is the inability of the patient to resist any disease or the weakening (or loss) of immunity. This condition is denoted by the scientific term "Acquired Immuno Deficiency Syndrome". The two important aspects to be noted are that it is acquired and that it is a syndrome (and not a positive disease by itself). AIDS is an acronym of the scientific term and in common usage, AIDS is referred to as a disease.

The first indication of the existence of AIDS came from Uganda when in the 1950's, several young males of that country died of a mysterious disease (or of several diseases).

Scientists were puzzled, but the level of scientific knowledge at that time did not permit a satisfactory diagnosis. Recorded evidence seems to indicate the recurrence of a similar attack in 1978.

It was only in 1981 that AIDS and the virus that was causing the disease were definitely identified. Once the positive identification was made in the U.S.A., doctors were very keen to assess the extent to which AIDS had spread worldwide. Their investigations showed that AIDS had spread throughout Central Africa and the Caribbean and revealed the presence of a large number of AIDS victims worldwide.

The disease had spread with such speed that by February 1990, WHO was in possession of 223,000 recorded cases of AIDS victims in 153 countries. The actual number might have been much more, as it is highly probable that many cases could have escaped detection and that many AIDS victims would also have deliberately avoided diagnosis.

As stated earlier, the first cases of AIDS were reported from Africa and it has spread very fast in that continent. Recorded instances of AIDS from Asia are comparatively new. Although the speed of infection in both continents may be similar, we may derive some consolation from the probability that the number of AIDS victims in Asia is much less than that of Africa.

The AIDS virus does not turn its victim into a diseased person overnight. It takes some time for the infected person to become a positive case of AIDS. However, throughout that period, the infected person acts as a carrier. This stage is obviously much more dangerous than the advanced stages because, the infected person would be playing the role of an AIDS carrier without his

* *Translation of a Sinhala Article*

being aware of it at all. It is suspected that there are about 0.5 to 1.0 million such carriers moving freely throughout the world.

The AIDS virus takes about four years to produce the first minor ailments and AIDS proper manifests itself after another four years.

Thus, it will be obvious that by the time a single positive AIDS victim is identified, he would have already infected several hundred others and each one of them is capable of spreading the disease to many more. This is the deadliest feature of AIDS.

In Sri Lanka, 12 AIDS sufferers and 20 carriers have been reported to date. However, an examination of the case histories of infected persons seems to suggest the existence of over 1000 undetected AIDS carriers in the country. AIDS appears to be spreading in Sri Lanka tragically fast.

Mode of infection

The AIDS causing virus destroys the internal immunity mechanism of its victim and thereby exposes him to serious illnesses.

The mode of transmission is usually through body fluids. The virus can exist only in living cells and it is found in high concentrations in secretions and liquids such as cerebral spinal fluid, amniotic fluid, sperms and vaginal fluid.

There are several ways in which AIDS can be transmitted to a healthy person.

1. Sexual Intercourse

The chances of contracting AIDS by a healthy person through sexual intercourse with an AIDS carrier are considered to be as high as 100%.

2. Blood Transfusion

AIDS virus can be transmitted from an infected to a healthy person when blood or blood products from an AIDS infected person is transfused to a healthy person. Use of infected instruments also could have the same effect. (Even a very small quantity of blood such as 1.4

micro litres is considered sufficient for the transmission of the virus). Although there is only a very low chance (of 10%) for the transmission of AIDS via this method, once contracted, the victim advances towards the terminal stages rather quickly.

3. There is a 50% chance that a baby born to an AIDS mother would also be an AIDS victim. In the majority of cases, the pregnancy of an AIDS infected woman ends in a miscarriage. It has also been found that pregnancy has a tendency to accelerate the development of the disease in an AIDS infected woman. However, feeding the milk of an infected mother poses only a mild threat due to very low concentrations of the AIDS virus in human milk. The incidence of AIDS among infants was first detected in U.S.A in 1983 and according to available data, 75% of such infants had contracted the disease at the foetus stage or at birth. Blood transmission accounts for the balance 25%.

4. Organ Transplant

It has been established that a 50% chance for AIDS transmission exists when an organ from a diseased person is transplanted in a healthy person.

No Reason to Panic

Although AIDS is mainly transmitted through bodily contact, activities related to ordinary social and human relationships (for example, parents and children; intimate friends; persons who stay together share many things in common and very often, come into close physical contact) do not constitute a serious threat. However, activities, a few examples of which are given below do not bring about transmission of AIDS from one person to another.

1. Touching or shaking hands with an infected person.
2. Sharing common accommodations.
3. Bathing in a pool used by an AIDS victim or washing cloths in the same place.
4. Embracing an AIDS patient.
5. Common use of crockery, cutlery and other utensils as well as household linen.
6. Common use of toilets.

Human parasites such as head lice, mosquitoes and bugs do not transmit AIDS. This is because the AIDS virus can survive only in the living cells of mammals. Of

course, it is quite possible for the virus to enter the body of a parasite such as a mosquito when it sucks blood from an AIDS victim. It is equally possible for a certain amount of this infected blood to enter the body of a healthy person on a subsequent attack by the parasite. However, the chances are that the AIDS virus would have died in the meantime and what may enter the second victim's body is only the dead virus.

In blood transfusion, it is absolutely essential that the donor should be a perfectly healthy person. If the donor's blood is tested for AIDS, it will go a long way in arresting the spread of this disease. It is also equally essential that all equipment used in blood transfusions are thoroughly sterilised (before use).

Persons who receive vaccinations and inoculations as well as those from whom blood samples are taken should ensure that the syringes used for the purpose are absolutely clean and sterilised.

Persons Most Likely to Become Victims

1. Homosexuals and those indulging in extra marital sex
2. Drug addicts using intra vascular infections
3. Patients who need frequent blood transfusions
4. Male and female prostitutes

This types of persons run a very high risk of contracting AIDS and also act as carriers of the disease, threatening the entire society.

Symptoms

AIDS symptoms takes some time to manifest after the infection. In most cases, the time space may be as long as ten years. However, the disease related anti-bodies are present in the blood after about 6-12 weeks. Sometimes, the time lag may be longer, about 6-8 months. After this period, the patient may suffer minor ailments such as fever, headache, sore throat, sweating in the night and inflammation of lymph glands. Very often, these symptoms disappear on their own after about two weeks. While this stage of AIDS is known as Sezoconversion Disease, only about 10-15 per cent of the infected persons appear to display the related symptoms. For the

next 8-10 years, the infected persons could have a perfectly healthy life without any symptoms whatsoever. This only indicates that the disease has entered its latent stage. After the expiry of the latent stage, patients' lymph glands begin to swell. The extent of swelling may be more than two centimetres and it lasts for about three months. By this time, the patient would be susceptible to a host of other ailments.

In the terminal stage of AIDS, the infected person develops unmistakable symptoms. These may include chronic infections, nervous disorders and rare types of cancer, all of which are necessarily fatal. Some of the complications that can arise in the terminal stage are:-

1. Thaposisacoma

This is an ailment mainly characterised by dark blue or dark violet patches slightly protruding from the skin. They first appear profusely on the torso and arms. Later they will be visible on the ears and around the navel. These patches are not painful, nor do they cause itching. After the patches make their appearance, the disease spreads to internal organs.

2. Tuberculosis

Normally, tuberculosis attacks the lungs, but in AIDS patients, it can also infect other parts of the body.

3. Herpes Simplex

Herpes Simplex is a viral disease. Infection leads to the appearance of wounds inside the mouth, on the penis or around the anus. AIDS victims are highly susceptible to this disease and it can infect healthy persons as well.

4. Candidiasis

Caused by a fungal infection, it takes two forms in AIDS victims.

i. Oral Candidiasis

Symptoms are the occurrence of wounds, whitish in appearance, inside the mouth. However, the wounds are not painful.

- ii. **Oesophageal Candidiasis**
Causes difficulty or pain in swallowing.

The above diseases can be treated, but no cure has yet been found for their root cause - AIDS.

Ways and means of destroying the AIDS virus

AIDS causing virus belongs to a class of viruses named 'retrovirus'. In common with all viruses, it can reproduce only within live cells. Its chief feature is the ability to transform its genetic structure within a very short period of time. Therefore, it has been found impossible to develop an "antivirus".

However, AIDS viruses that exist outside the living cells can be destroyed very easily, because a living cell is essential for their reproduction. Proper sterilisation is an effective method of destroying AIDS viruses.

1. Immersion in boiling water for about 20 minutes. This is sufficient for instruments such as needles and syringes used in vaccinations and inoculations.
2. Application of Dry Heat
Appropriate instruments can be sterilised by subjecting them to a very high temperature of 340°F (170°C) within an electric furnace.

3. Application of Steam

A pressure cooker or a modernised cooling pot can be used for sterilising syringes etc.

4. Use of Chemical Agents

Certain types of virus de-activating chemicals are employed in this method. (e.g. Hydrogen Peroxide 6%, Ethanol 7%, Glutarol 2%). To be 100% effective, the chemicals used should be absolutely fresh.

Treatment

A complete cure for AIDS has not yet been discovered. However, treatment should be given for the control of other ailments arising out of the reduced immunity of the AIDS victim. Without such treatment, the AIDS victim is liable to die within a short time. Azidothiamidine is a widely used drug in the treatment of AIDS. This drug has been found to have beneficial effects on the condition of the patient and his lifespan. However, certain adverse side effects of AZT have recently come to light. These include complications such as contraction of bone marrow.

(This article is based on the data presented at the Seminar on AIDS, sponsored by the Health Education Division for the benefit of creative writers.

I wish to thank Mr. Thusitha Malalasekera for the encouragement he has given me in compiling this article.)

A BOOK REVIEW

ANIMALS TELL PEOPLE ABOUT POLLUTION

An encyclopaedic, exhaustive, pioneering study of how animals tip off humans about pollution has just appeared here.

The author, a British scientist named Yusaf Samiullah, who holds a Ph.D. in ecotoxicology from King's College, University of London, describes it as "a book about animals and their accumulation of environmental pollutants. It is also a book about the effects of contaminants such as 'heavy metals', pesticides, polychlorinated biphenyls (PCBs), polyaromatic hydrocarbons (PAHs) and radionuclides can have on animals.

"The widest possible spectrum of animal group is considered, from the lowest invertebrates to fish, birds and large mammals. These may exist in terrestrial, fresh-water or marine habitats". Dr Samiullah says that the book "Biological Monitoring: Animals", critically review systematic investigations into animals as sensors of environmental pollution.

The book is 767 pages long, reports on the finding and reading of close to 20,000 scholarly references, of which 5,000 to 6,000 are cited, and deals with 569 species of invertebrates, 153 species of vertebrates, 315 of birds, 308 of fish, and 92 species of plants and microorganisms, making a total of 1,437 species.

To do all this Dr. Samiullah has devoted about 14% of his life from the cradle to the tome. The publishers of the massive volume are the Monitoring and Assessment Research Centre (MARC) of King's College London, University of London. It was made possible by the financial support of an organization with an equally long name, the Global Environment Monitoring System (GEMS) of the Nairobi-based United Nations Environment Programme (UNEP).

"It is a quite remarkable scientific work", says Professor Peter Peterson, Director of MARC. "When you get over the initial psychological shock of the sheer size and weight of the book, and start leafing through it, you become passionately interested. While it is primarily intended for scientists, researchers and university stu-

dents, anyone with a good O-level education in biology or science can read, appreciate and use it".

In short, you can hardly hold the book up because it's so heavy, but it's so interesting that you can't put it down. One of the most timely chapters treats marine oil pollution. Its relevance to today's environmental problems in the Middle East is obvious.

The author says that "where oil persists and the effects are severe, saltmarshes, mangrove ecosystems and coral reefs are among the most vulnerable habitats. In these areas, remedial measures may prove futile and may compound the damage attributable to oil alone".

In his chapter on radionuclides, Dr Samiullah points out that "because of their potentially mutagenic, carcinogenic and teratogenic effect, the behaviour, fate and pathways of radionuclides in the environment are matters of great concern to humans. The unique pathway from atmosphere to lichen to reindeer *Rangifer tarandus* and so to humans offers the opportunity for radioecological investigations".

The lichen *Cladonia alpestris* is a major food source for Swedish reindeer during the winter when radionuclides deposited from the atmosphere are thereby transferred to the animals. Lapps who breed reindeer eat a lot of their meat, and so it is not surprising that high concentrations of radionuclides have been found in Lapps.

In the early 1980s in the Thames Estuary U.K. researchers observed a sharp increase of smelt *Osmerus eperlanus* in the fish catch. Smelt are closely related to salmon *Salmo salar* and similarly sensitive to changes in water quality. The increase was attributed to improvements to London's major sewage facilities.

Anglers use lead shot to weigh down their lines and submerge fishing hooks. Even though tin and tungsten are now popular alternatives to lead as fishing line weights, the MARC book reports that "none the less, the number of mute swans *Cygnus olor* dying from ingesting lead weights is still high. In 1987, for example, 24% of all birds rescued or given autopsies in the Thames area were

victims of lead poisoning, although the overall trend was down".

Every year in the U.S.A two million waterfowl hunters expend more than 2,700 tonnes of lead shot. Much of it is swallowed by waterfowl, resulting in elevated tissue lead levels and chronic-to-acute lead poisoning. A study cited by Dr Samiullah estimates that annual losses due to lead poisoning are 2% to 3% of the autumn population of all freshwater aquatic birds.

The death of waterfowl in the Coeur d'Alene river valley in Idaho was traced to lead from mining wastes, while high liver lead and kidney cadmium levels in green-backed heron *Butorides striatus* in southeastern Missouri led investigators to discover erosion and seepage from large tailings dams from inactive lead mines.

"That is a good example of how contaminated animals lead us to avoidable environmental pollution, isn't it?" comments Professor Peterson.

Polar bears *Ursus maritimus* are widely distributed throughout Arctic circumpolar regions and feed mainly on the year-round resident ringed seal *Phoca hispida* and bearded seal *Erignathus barbatus*. Since polar bear tissue specimens can be readily obtained from regular Inuit hunting, this carnivore may be used to monitor the distribution of contaminants such as organochlorine pesticide residues which are biomagnified in the Arctic marine ecosystem.

PCB and DDT contamination in polar bear tissues was noted in 1975, while in 1988 examination of polar bear liver and fatty tissues disclosed the presence of dieldrin, chlordanes, DDT, PCBs, and chlorobenzenes. The book goes on to say that "chlordanes and PCBs accounted for more than 80% of the total organochlorines in adipose tissue. Concentrations of most organochlorines were lowest in animals from the high Arctic, intermediate in Baffin Bay and highest in Hudson Bay".

"These results illustrate how human activities have led to the dispersion of pesticides and toxic chemicals to the furthest regions of the globe", says Professor Peterson.

The peregrine falcon has proved a very useful bird in pointing the claw at DDT. During World War II about 600 peregrines were shot in Britain because it was feared they would prey on messenger pigeons from the French Resistance. Their post-war recovery was rapid, testifying to the resilience of the species. So, the sudden, simultaneous losses in major peregrine falcon breeding populations throughout Europe and North America in the early 1950s suggested a new and unknown agent. That led to the identification of DDT as the causal factor.

In Poland the antlers of roe deer *Capreolus capreolus* have been excellent biological monitors of industrial pollution. In a study of 357 roe deer antlers obtained from the Niepolomice forest over a 53-year period from 1920 to 1973, it was clear from their weight, length, volume and general quality that a marked deterioration in the value of these antlers had occurred two to three years after the commissioning of a nearby large iron and steel works.

"Bees clearly have a useful role in biological monitoring studies," Dr Samiullah says, "particularly as their foraging activities enable the researcher to sample large tracts of territory effectively". Citing an example, he says that "different concentrations of aluminium, barium, calcium, copper, magnesium, nickel, palladium and silicon were found in honey, depending on the distance bees foraged from a major highway in New York State".

"Biological Monitoring: Animals" sells for £45. "At that price," remarks Yusuf Samiullah, "it is a money-saver and a time-saver for anybody contemplating an on-line bibliographic search on any one of the hundreds of topics reviewed. The book provides an informational stepping-stone to the nineties".

Source: UNEP - Monitoring and Assessment Research Centre (MARC) circular letter.

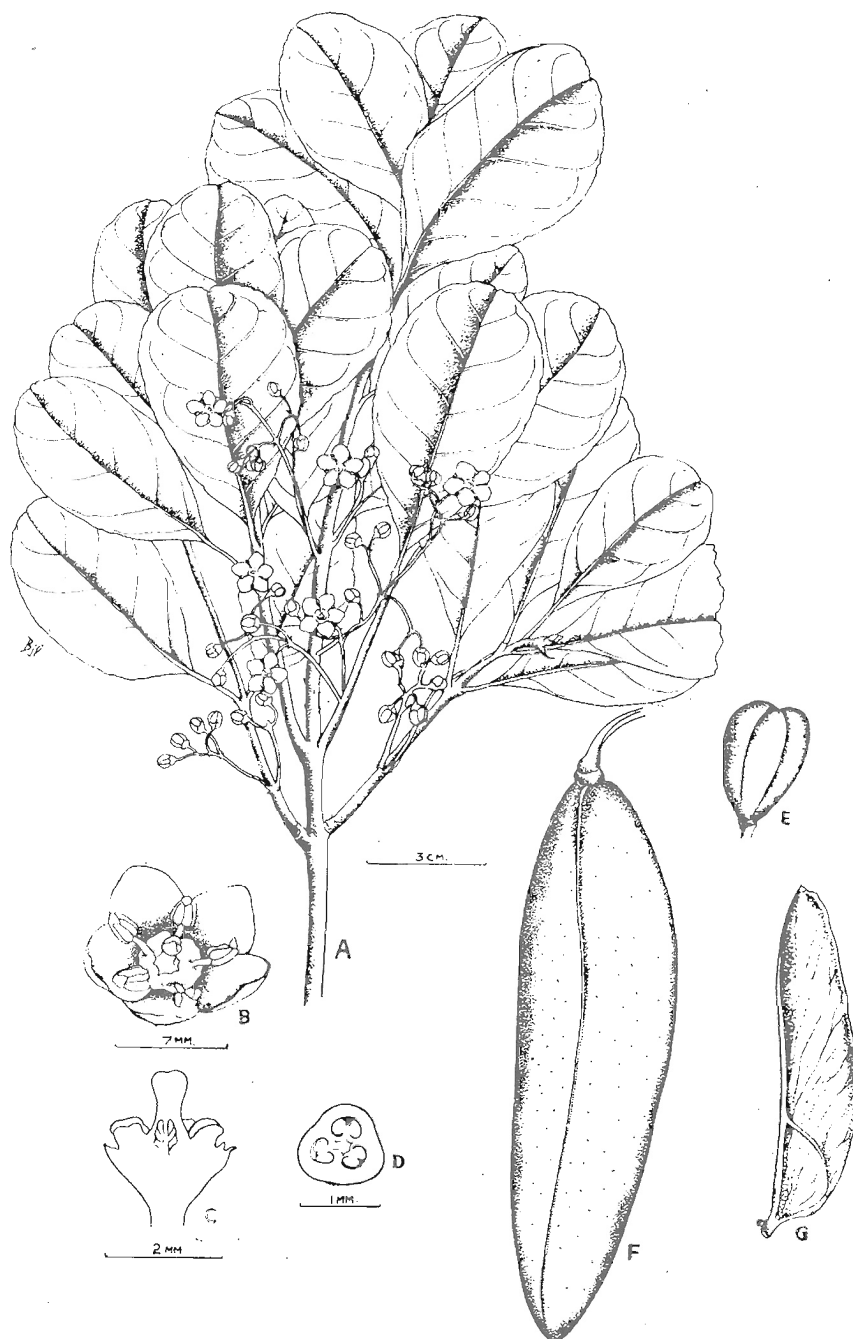


FIG. 111. *Kokoonia zeylanica*. A, branch with leaves and flowers. B, flower from above. C, longitudinal section of the ovary and disc. D, transverse section of ovary. E, young fruit. F, mature fruit. G, winged seed.

Uses: The inner bark, which is used in the treatment of diabetes, snake-bites, swollen joints, eye diseases and headaches. With other drugs, it is used in treating framboesis pimples and diseases of the skin. The bark may also be used to lighten the colour of the skin and for removing marks from the face. It is often used as snuff for severe headaches. The oil from the seeds is used as a leech repellent.