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Headlines

Infectious diseases are capable of affecting the entire country. Often a single individual or a community can suffer from the assault of many infections in a season or a year.

Infectious diseases claim about 22 per cent of all lives in India. Every year millions suffer from different infectious diseases in India.

Malaria alone affects – millions lives and claims - - many lives. The six other major infectious diseases (filaria, Japanese encephalitis, kala-azar, dengue, like – infect – and kill – collectively. Increasingly newer sections of populations are at risk of contracting infections largely due to environmental changes and lack of medical supervision.

New and fast emerging fevers and infectious diseases are reported at an increasing rate from all parts of the country. Since 1995, at least 27 fevers of unknown origin have occurred.

No corner, city or village in India is immune from infectious disease. Many new fevers have become a part of daily existence in cities and villages alike. The medical institutions have no clue on what they are and they are doing nothing about.

The diseases:

What are infectious diseases?

Infectious diseases in India

Diversity of Diseases in India

Where and why do they occur – in India?

History of infectious diseases in India

Burden of infectious

Environmental causes of infectious diseases

Infectious disease control programmes

Major types of infectious diseases in India

Kala-Azar

Dengue

Japanese Encephalitis

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Filaria

Fevers of Unknown Origin



5. Infectious and vector-borne diseases: Where transmission is deadly

5A: The disease challenge

When malaria was nearly eradicated in India in 1965 and smallpox was eradicated from the globe in the late 1970s, many public health experts and scientists presumed that infectious diseases could at long last be conquered. Death rates from infectious diseases had declined in Indian cities through the 1960s - thanks to better immunisation programmes, intensive chemical use and high usage of antibiotics. During this period, scientists extended the theories of how diseases spread, identified their natural history, and created a host of potent antibiotic drugs that helped save millions of lives. This became the silver bullet with which all infectious diseases were to be tackled. But then things began to change. Development started to take place in a haphazard manner, resulting in unforeseen impact upon the environment. This, in addition to widespread corruption at the grassroots, lack of political will to tackle the scourge of diseases, marginal application of public health and sanitation measures, little co-ordination between different organisations and government agencies and diversion of interest from research on the diseases ensured that many of these diseases returned. As a result, infectious diseases that were once thought to be eradicable (and other countries did eradicate many of these diseases), have deeply entrenched in the lifecycles of cities and villages in India and their elimination now seems to be an impossible task.

Infectious diseases i.e. diseases that are capable of being transmitted from one person to another are caused by a pathogen. The pathogen is an infectious agent which is usually a minute organism like a virus or a bacteria that can use a human body to multiply within them. Infectious diseases are of two types – communicable and non-communicable diseases. Infection is transmitted to humans by a parasitising animal like mosquito transmitting malaria called the vector or carrier. Some infections though are transmitted from human to human like the flu. Often other animals, which may remain unaffected from the diseases causing pathogen, serve as a reservoir of the disease. The former type of disease, based on the mode of spread of infection is classified as non-communicable diseases, and specifically termed as vector-borne diseases. The latter are classified as communicable diseases and are transmitted among large populations through human-to-human contact by the spread of the pathogen through mediums like dust, air or water.

A vector is a carrier of a pathogen. Traditionally pathogen or the infectious agent, classically a virus or a bacteria, and the insect vector, have been interchangeable called “parasite”. The term parasite is not suitable as both parasitise of human host, hence a distinction between the two is kept here by using vector for the carrier insect and pathogen for the actual disease-causing agent.

The main problem for a pathogen is how it can infect a host and replicate within it. It needs a prolific vector, one produces an enormous number of offspring, and can adapt in diverse climatic conditions and transfer it in favourable seasons. Both the vector and the pathogen need to breed quickly and produce complete sets of potent offspring. Efficient pathogens are ones that are capable of using more than one vector to transmit themselves. It should find an easy way to jump between hosts without harming the host.



India is virtual hotbed of infectious diseases. Diseases like malaria, kala-azar, cholera and diarrhoea exact social, psychological, and economic costs. They disrupt economic development, and perpetuate the poverty that in turn, fosters the spread of infectious diseases.

There are many kinds of infectious diseases. They can be bacterial, viral or minute worms that cause these infections. Many diseases have not been identified as yet, despite their common occurrence. Such infectious diseases are called fevers of unknown origin or mystery fevers. While some diseases have persisted for a very long time, there has been an emergence of new infectious diseases as well as the re-emergence of old ones. Infectious diseases that have been effectively controlled but emerge again are called emerging and those that occur frequently in cycles are called re-emerging. Many re-emerging fevers remain unknown because public health authorities do not recognise them as separate entities nor do they have the diagnostic capabilities to report their prevalence. Even a well-studied disease like leptospirosis remained unidentified for a painfully long time in Kochi and Mumbai in recent years. These diseases are often misdiagnosed leaving a susceptible population vulnerable to more attacks.

More than 28 "new" or previously unrecognised disease-causing microbes have been identified since 1973, worldwide. Just since 1995, a new strain of cholera (number 0139) has surfaced in the Indian sub-continent and Africa -killing thousands of people across the world. **Plague** re-emerged in Surat and Beed. There have been outbreaks of leptospirosis in Mumbai from 1999 to 2001; dengue and its more serious manifestation-dengue hemorrhagic fever afflicts New Delhi and other industrial cities and various forms of meningitis in parts of the country. In addition deadly infectious fevers like the Siliguri fever in West Bengal continue to remain unidentified and undiagnosed.

Infectious diseases are fairly egalitarian in their spread. The range of infectious diseases remains isolated in small pockets, which on getting the right environmental conditions flare up an epidemic. The entire country is at risk of contracting one infectious disease or another. Malaria is the most prevalent infectious disease and poses a risk to about 80 % of the population. Other major infectious diseases are listed in table –(Table: Most prevalent infectious diseases in India).

5A.1: The environmental link

Humankind's relationship to infectious pathogens is a part of an evolutionary drama. Simply put, micro-organisms have a selfish interest in human survival. In an infectious epidemic, pathogens afflict a few and ensure to keep some survivors as a sustainable option. As a response, humans too evolve resistance to the pathogen's virulence and then the pathogen develops means to evade this acquired immunity. This cat and mouse relationship is central to this drama.

Infectious diseases are designed to spread and affect large populations. In natural conditions, they have a distinct season of emergence when ideal conditions are available for their proliferation. In case of vector-borne diseases, conditions should be ideal for both the pathogen and the vector. Many environmental changes prolong this ideal period, thereby aiding the spread of infection. The mode of transmission of



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infections is an important factor in controlling the disease. Environmental factors play a critical role in this, affecting the maintenance and spread of the disease.

Ecological changes cause rapid evolutionary changes in the ability and intensity of the pathogen to cause the disease. The absence of precise knowledge of the causal process of each outbreak is rarely known.

Change in land use especially irrigation, agricultural intensification and deforestation have caused the release of new diseases or established many less prevalent diseases. Clearance of forests or any undisturbed natural habitat causes vectors and reservoirs to leave their natural and captive hosts and seek new hosts. This is commonly seen in vector borne diseases. Villages especially around forest areas, rice fields or irrigation canals commonly suffer from infectious diseases like malaria and Japanese encephalitis. Often drastic climatic change usher infectious diseases. Floods, earthquakes and cyclones can bring in infectious disease from unexpected quarters. The Surat plague episode was possibly linked to the Latur earthquake, the reintroduction of malaria in Mauritius and Madagascar is attributed to cyclonic winds that carried infected mosquitoes from Africa, and Japanese encephalitis introduced got introduced in Australia by mosquitoes carried over by atmospheric wind (Reynolds DR, Smith AD, Mukhopadhyay S, Chowdhury AK, De BK, Nath PS, et al. Atmospheric transport of mosquitoes in northeast India. *Med Vet Entomol* 1996;10:185-6. , Scott A. Ritchie and Wayne Rochester Wind-Blown Mosquitoes and Introduction of Japanese Encephalitis into Australia, Vol. 7, No. 5, Sep–Oct 2001 (available at - (www.cdc.gov/ncidod/eid/vol7no5/ritchie.htm))

Deforestation and rapid changes in land-use are possibly the most important factors in the emergence of an infectious disease. Human settlement, commercial development, construction of roads, water control systems (dams, canals, irrigation systems, reservoirs), and climate singly and in combination contribute to the emergence of infectious diseases. The nature and extent of change in the incidence of infectious disease depends on the intensity of changes in land-use and settlement, the time interval from one land-use to another/others, changes in type of soil and its degree of water absorption, changes in vegetation characteristics, changes in the types and amounts of bodies of water, their size, shape, temperature, pH (the measure of acidity or alkalinity of a substance), and proximity to vegetation and, changes in climate. All these affect the vector, and in turn, the incidence and prevalence of infectious disease.

Deforestation brings about the most disruptive changes in land ecology and therefore vector populations. This disturbance affects the soil type, moisture and other physical qualities of the soil, vegetation and water content in the forest ecosystem. Cleared tropical forests or encroachment and extraction of timber and fuel-wood from forests causes the forest to degrade and resemble grazing land for cattle, and give way to settlements, small agricultural plots, or are left as open areas. These disturbances change existing ecological balances and niches and provide conditions for proliferation of new vectors or provide condition for dominance of locally existing vectors and their pathogen.



The replacement of forests with crop farming and cattle raising create a supportive habitat for vectors. Introduction of new to-the-area animal species, such as cattle, sheep, pigs, and chickens can result, variously, in either increased or decreased transmission of infectious disease to humans. With livestock to feed up on, the vectors may reduce feeding on humans or, conversely, with the plethora of livestock blood resources, vectors may multiply and seek additional, that is, human, feed sources. With the larger reservoirs of infection, there is increased pathogen transmission and, humans not only become ill, but also further increase the pathogen reservoirs.

Nature and quality of soil and elevation contribute to determining the type of bodies of water formed in disrupted areas. Whereas the forest floor in primary growth tends to be heavily shaded and littered with a thick layer of organic matter that absorbs water and renders it quite acidic, cleared lands are generally more sunlit and prone to the formation of puddles with more neutral pH which can favour specific mosquito larvae called anopheline's development. Lower the water salinity and neutral to mildly alkaline water increase fecundity and growth of freshwater vectors. Terrain affects the manner in which water collects in deforested areas: on steep inclines, streams are more common than large pools. Crops such as sugar cane and rice are water intensive and often submerged in a few inches of water, which provides an ideal habitat for breeding mosquitoes.

When deforested areas are replaced by crop agriculture, the pattern of human settlement usually changes. In addition to an influx of settlers, migrants may come, and if a suitable anthropophilic (or human-preferring) vector is present, the migrants become a reservoir for transmission of infectious disease endemic to their former home grounds, but foreign to the non-immune resident settlers. Indigenous forest dwellers generally have developed immunity to forest-dwelling infections. However, during the process of deforestation, as the forest recedes, new settlers who are drawn to the edge of the forest for agricultural activities, or enter the remaining forest for commercial activity such as logging, are particularly vulnerable as they lack immunity to infections endemic to the area.

Vectors too have their preference for hosts. The changing ecology and the arrival of new human and animals population causes the displacement of the original hosts of the vector. The vector must now adapt to seeking new hosts – which may result in a strong fondness for them or may prove to be a distaste, which may force the vector (and therefore the infection) to migrate from this area and disappear.

The human biting behaviour of vectors are affected by human availability, flight range of the vector, its biting frequency, and biting times in relation to human habits. In adapting to changed environmental conditions, including reduction of animal population and increased human population, some vectors display a conversion from a primarily animal preference to primarily human preferring orientation. Most zoonotic (animal dependent) vectors display three distinctive life cycles: forests dwelling (sylvatic), animals dependent (zoonotic), and human feeding (anthroponotic). A pathogen eradicated from human and domestic animal populations, can survive in forested (sylvatic) habitats of degraded forests and nearby groves and then, subsequently, re-invade human and domestic animal reservoirs.



Reservoirs, irrigation canals, and dams are closely associated with disease. Construction of reservoirs and canals can lead to a shift in vector populations, such as snails and mosquitoes, their larvae and their parasites. In the tropics, during construction of dams and canals, excavation pits provide breeding sites for mosquitoes where they lay buoyant egg masses. Different mosquito species vary in their habitat requirements, in both the larval and adult stages. Some species prefer sunlit pools with turbid water, with little or no emergent vegetation. Some larvae prefer clear water, inhabiting the edges of clean, clear, gently moving streams or, conversely, others thrive in irrigation and hydroelectric reservoirs with their frequent changes in water level, vertical shorelines, and emergent vegetation without organic material or salinity; others inhabit coastal areas with high salinity. Some vector species require extensive vegetation cover and inhabit swamps and relatively permanent water-bodies with organic material, others are hardier and more versatile in adapting to diverse environments. Riverine pools, created by diversion of flow out of river-beds, provide breeding in shallow, stagnant surface water exposed to sunlight. Deeply shaded pools, seepages in forests, footprints, mining pits and irrigation ditches, and excavated depressions in the open sunlight all provide areas for mosquitoes to deposit their eggs.

Climate the aggregate effect of temperature, precipitation, humidity, sun's intensity and wind which are reflected in the type of vegetation in plant communities and the life that it supports. Increasingly, human activities has contributed to considerable local and global-level climate change: fossil-fuel combustion (coal, oil, and gas) increasing the concentration of CO₂ ; deforestation reducing CO₂ , absorbing capacity of forested areas; industrial activity create significant changes in the ambient temperatures. Small changes in the mean climate can produce relatively large changes in the frequency of extreme weather events and frequency and severity of heat waves and altered water cycle. During the twentieth century, there was a rise in average daily temperatures; during the last fifty years, surface temperatures appear to have been warmer than any similar period in the last 600 years. Because warmer air can hold more moisture than cooler air, the hydrologic cycle has changed, particularly affecting waterborne disease vectors, which are sensitive to changes in the hydrological cycles. There were also increases in cloud cover, total precipitation, and frequency in extreme precipitation events, at an intensity of more than two inches per day [44± 46]. It is expected that by the year 2100, there will be a 28C rise in temperatures with accompanying indirect effects: a 49 cm. rise in sea level, an increase in hydrologic extremes and enhanced evaporation, leading to both more droughts and more floods, and prolonged recovery of the ozone hole. Extreme weather has deleterious effects on the population: drought resulting in poor hygiene and reduced food supplies, floods in contamination. Although global distributions of these changes are expected to be unequal, they are expected to result in significant wide- ranging effect on ecosystems. As global warming continues, there is concern for increased transmission of some tropical diseases and potential for their expansion (of debatable extent) into temperate regions. For example, temperature determines the rate at which mosquitoes develop into adults, the frequency of their blood feeding, the rate with which pathogens are acquired and, the incubation time of the pathogen within the mosquito. These influences must be compared with the opposing effects that high temperatures exert in reducing adult mosquito survival.



The length of rainy and dry seasons and the interval between seasons affects larvae and adult vector development and abundance. Transmission of many infectious diseases is confined to the rainy season. Rain provides the breeding sites for mosquitoes and helps create a humid environment, which prolongs the life of vectors.

The El Nino phenomenon, cycling with a frequency of every 2±7 years, is second only to seasonal variability as the strongest driver of weather variability in many regions of the world. During El Nino, weakening of easterly trade winds causes warm equatorial Pacific water to migrate from the western to the eastern Pacific, resulting in drought in some regions of the world and flooding in others. For instance, in India, El Nino variations can result in excessive rainfall and drought.

Urbanisation process transforms the ecology. As it is, clearing forests or building on riverfronts or invading into deserts bring about the change of displacing the natural elements and bringing in human factors. Increasing human activity and growth of cities build pressures on civic services. Most of the pressures are release in one way or another into the natural ecosystem. For example, human and industrial wastes drain into natural water bodies creating poor sanitary conditions. Migration into cities too brings people of different areas together. These people of different age groups, capabilities, physical state and resistance often become hosts to several diseases. Sometimes they bring in diseases to a much larger population.

Most infectious diseases have an ecological basis to their origin. In a poor state like Orissa there is a virtual 'microbial traffic', which has its annual causalities. Diseases have a distinct pattern of ecological succession. Even causalities have a pattern: In summers due to water paucity, tainted water spread diarrhoea, which affects children and older people more. When rains arrive malaria and fevers of unknown origins emerge, all age groups are affected but young women and pregnant women are specially targeted. If a natural calamity like flood or cyclone arrives, then malaria recurs, and cholera and other gastro-intestinal infections follow which affect young adolescents and children more than adults. In winter the paucity of food causes widespread hunger and leads to malnutrition, which is an ideal environment for emerging pathogens to consolidate in their evolution as pathogens. In recent times, many fevers have been released from forests, after severe degradation of forests has taken place in these areas. Clearly there is a distinct pattern of succession of diseases.

The scale of epidemic is eventually determined by the number of susceptible people who can be infected by the pathogen and the timeliness of intervention that can prevent the spread of the infection. Failing health infrastructure, lack of epidemiological surveillance, antibiotics abuse and resistance caused by it has helped the disease and its agent to establish themselves in cities and villages. Inequity to access to health services also ensures that people who suffer from poor health and infections also do not receive the desired medical facility. Inequality to live, grow and work in clean environments make populations susceptible to infections and poor health. Children who grow in these environments are particularly susceptible to environmentally inflicted health problems.

<i>Disease</i>	<i>Season of appearance and frequency</i>	<i>Area</i>	<i>Contribution of environmental factors for emergence of disease</i>
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Malaria	Late summer, monsoon or post-monsoon. Annual	All over India except high altitude areas of the Himalayas (above 1650 mts)	Land-use changes like irrigation, deforestation and agriculture, and urbanisation and migration. 60 per cent of cases emerge due to environmental change
Dengue	Late monsoon and early winter. Annual	Industrial towns of northern India especially Delhi, western UP and Ludhiana	Largely due urbanisation and improper water storage. Almost entirely attributable to environmental factors.
Japanese Encephalitis	Post monsoon event in 11 districts of Andhra Pradesh after outbreaks were restricted to every 2-3 years. Appears every 2-3 years in Bihar	Peninsular India and eastern states. Most prevalent in Andhra Pradesh, Karnataka, Orissa, UP, MP and Bihar.	Rapid change in agriculture practices and livestock rearing especially pigs. Wild local reservoirs like wading birds also contribute to the dispersal of the disease. Significant contribution of environmental factors. ((WHO–CDS Dec, 2000))
Kala azar	Annual	Bihar, pockets of eastern and central India	Animal reservoirs, favourable breeding grounds of the sand fly (the vector) and susceptible populations. Significant environmental reasons for emergence and persistence.
Filariasis	Persistent in susceptible populations	South, east and central states with moist tropical climate	Urbanisation, unhygienic conditions, improper water storage and susceptible populations. Almost entirely attributable to environmental factors.
Other viral infections	Sporadic in occurrence, almost annual	All over the country	Rapid environmental change, poor hygienic conditions and susceptible populations in both rural and urban areas. Almost entirely

5A.2: The poverty link

Infectious diseases and poverty are intimately connected. Infectious diseases are most intractable for states in the poorest areas of India like Orissa, Bihar, Madhya Pradesh. The only parts of India free of malaria are the northern and southern extremes, which either have a geographical advantage of being extremely cold, or the richest and most educated communities as those in Kerala and southern and coastal Karnataka. Bihar has the worst incidence of kala azar in Asia and perhaps the world. It is the poorest state in the country. Infectious disease risks have always been very geographically specific, however the rapid environmental change, adaptation of pathogens and vector nad the



movement of people that facilitate only means that infectious diseases will increase their range. Intensive assaults of infectious diseases are increasing in India.

Poverty too is also geographically specific. The twenty poorest districts of India show the highest morbidity due to infectious diseases. They also show the higher infant and pregnant women mortality. The malaria prevalence rate in these districts range from 26 % to 64 %. Other infectious diseases that are seasonal contribute a significant burden to the communities. Cholera and other gastro-intestinal disease are also prevalent at much higher rates than other parts of the country. In contrast, the richest 20 districts show low incidence and prevalence of infectious disease. Though pockets of infectious disease, and high infant and maternal mortality are observed, the overall incidence is low. For example in Mumbai city, the infant mortality is --, which is comparable to rural -- . Not far from Mumbai, another highly industrialised district, Thane has the most dismal nutrition, child mortality and hunger record. This inequity in access to facilities and inequality in distribution of benefits is common and ignored in even in the most developed states of India.

[chart - Infectious disease and Poverty – five poorest and five]

Globally, the 150 countries with populations over one million in 1995, account for over 99 percent of the world's population. Forty-four of the 150 countries, or 29 percent, have intensive malaria. Thirty-five of these 44 countries are in Africa. The average purchasing-power parity GDP per capita in 1995 for the malarial countries was \$1,526, compared to an average income of \$8,268 in the countries without severe malaria, more than five times higher. (John Luke Gallup and Jeffery Sachs, *The Economic burden of Malaria*; CMH Working paper WG1 10, February 2001, CMH, Geneva).

Infectious disease affects poor people disproportionately. It prevails more in poor people because they are physically susceptible to disease. This does not mean that these diseases do not affect rich people. In cities where slums and shantytowns are situated close to organized housing, infectious diseases breed in the squalor and spread rapidly to these areas. Infections are opportunists that invade into susceptible populations.

Many socio-economic studies on malaria, kala azar and filaria show that they affect the poorest of the poor. In the case of kala azar, the most marginalized poor in Bihar, the Christians and Muslims, are the ones who contract the infection. Malaria affects pregnant women and children first. Poorer sections of rural communities live in the fringes of villages, often close to fields and forests. Such communities show a higher incidence of malaria and other fevers than those who live in larger communes or villages, further away from the forests.

Often a host may not manifest an infection, despite being infected by two or more disease. Such individuals are potent carriers who serve as reservoirs of the disease and transmit it to susceptible individuals like children, pregnant women and the old.

In urban areas too, the poor are the first to face the incidence of disease as they live in margins of cities, which provide them with few civic amenities. In fact, most of them live around sewers, river-banks or drains. Not only are these populations more susceptible to infections because of their poor immunity or physical health, but also the constant



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burden of infectious disease that persists in their surroundings that makes them easy targets.

<i>Disease</i>	Annual cases	Annual deaths	Population at risk	Population covered under control programmes	Possible threats
Malaria			Entire country except higher reaches of the Himachal Pradesh and Jammu and Kashmir (above 1,200 mts.above sea level)	Centrally administered and state collaborated programme. The National Anti-Malaria Programme is the oldest diseases control programme in India. It oversees other vector borne disease control programmes. Aided by the Malaria Research Centre, New Delhi.	
Dengue					
Japanese Encephalitis					
Filariasis	22.5 million clinical annual cases		463.58 million people living in filaria endemic states will be exposed to the risk of filariasis by the end of the year 2000 in 13 States and 5 Union Territories (NAMP, 2000)	Merged with malaria control programme. The components of the revised National Filaria Control Programme have been outlined and the programme is being implemented in 13 districts covering 41	



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				million population using single annual doses of DEC. Another strategy of co-administered drug regimen (DEC+ albendazole) in six selected districts covers 21 million population.	
Kala Azar				Merged with malaria control programme	
Cholera and other gastro-intestinal diseases			Entire country	No specific control programme. Extensive cholera research specially in vaccine development	Linked with climate change
Tuberculosis			Entire country	National programme covers the entire country. Strategic support from international community NGOs and donors. Extremely successful in bringing down the number of cases.	Will rise with increasing incidence of AIDS, smoking and air pollution. Antibiotic resistance also a very serious challenge.
Other respiratory diseases			Entire country	No comprehensive programme except the tuberculosis control programmes	Will increase in cities and small towns alike. Opportunistic infections linked with increasing air



					pollution
Other viral infections			Entire country	No surveillance strategy or treatment protocols	Will emerge and establish as new diseases for which cure will be needed, sooner or later

All figures from World Health Report 1999

5A.3: History of disease control

The British approached disease control in a scientific manner. They kept accounts of medical research and investigation in colonial India studying native races and the impact of climate and diet, apart from the diseases that interacted with them and their indigenous medical systems, (Mark Harrison "The Tender Frame of Man": Disease, Climate, and Racial Difference in India and the West Indies, 1760-1860. The Bulletin of the History of Medicine 70.1, Spring 1996 available at <http://muse.jhu.edu/demo/bhm/70.1harrison.html>). With the foundation of the Grant Medical College in Bombay in 1845, and other hospitals in Kolkatta and Chennai addressed the control of infectious diseases. The Plague Research Institute was opened on August 10, 1899 by the then Governor of Bombay, Lord Sandhurst, with W. M. Haffkine as its first Direction in Chief. Its primary job was to produce the plague vaccine for use all over India. They studied India's indigenous medical systems, developed two mental asylums (in Madras and Lucknow) and leprosy asylums in each zone, devised the technological aspects and social implications of the colonial smallpox vaccination policy, and acted on potential cholera and plague areas like in the pilgrimage centres of Puri and Pandharpur. (Biswamoy Pati and Mark Harrison (eds.), Health, Medicine and Empire (January 2001). New Perspectives in South Asian History, Orient Longman). Possibly the British did most groundwork in controlling infectious diseases and recording mysterious fevers. The famous Sir Francis Younghusband expedition of 1903 recorded a mysterious violent fever enroute to Tibet from Calcutta (later called Karyovirus -II, or Jacobson's disease after the expedition's physician). The Gazette of the Bombay Presidency 1896, narrates tales of mysterious fevers and deaths in the porter colonies in Surat and Bombay dock areas.

But civic administration specially health and hygiene conditions were well monitored during the British Raj. For example, a letter written by a reader of The Statesman Calcutta in 1876 laments the plight of cholera in the city. He complained that this year two people had succumbed to the infection! Apart from the plague epidemic of 1894- 96, Calcutta witnessed few epidemics during the strong British presence in the city. Gradually, when the British moved out the capital from Calcutta to Delhi in 1911, more epidemics took place. Plague, malaria and cholera occurred sporadically became regular features. The Calcutta Presidency Gazette of 1917 reports that kala azar that was restricted to the confines of Upper Assam, Central provinces but had breached into Bengal.

Sanjoy Bhattacharya, health historian based at the Wellcome Trust Centre for History of Medicine at University College London, believes that the real thrust of medical services



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and disease management came at the onset of the wars that the British waged in the eastern borders, especially with the Japanese. The Indian Medical Department (IMD) officials involved in civilian practice were regular inducted into military service. But this proved unsuccessful due to the limited numbers of such officials. The result was a series of new strategies to develop the strength of the military medical framework. For instance, the Indian government decreed that men under 40 years of age, who were fit for military service, not be recruited from the open market to fill vacancies that had arisen from the appointments vacated by those released from military duty. They also ordered the provincial governments to review the needs of their medical and public health services and thereby determine the maximum number of medical graduates and licentiates that these administrations could release for military service.

Investigations about the usefulness of employing medical licentiates into the military was given additional attention after the office of the Secretary of State for India announced that the Medical Personnel (Priority) Committee in Britain would be unable to provide medical personnel for service in the subcontinent. The result of this was the creation of a special committee to investigate the public health and medical situation in India, which arrived from Britain on 13 December 1942 and toured the subcontinent for 3 months. It recommended that the army make greater use of medical licentiates through the creation of a new service, the Indian Army Medical Corps (IAMC), which would allow the authorities to formally accommodate their qualifications. The IAMC was formed with effect from 3 April 1943. The establishment of this new service also assisted the expansion of other wings of the military medical service, especially the anti-malaria units. The malaria control measures before September 1939 had been largely concentrated in and around permanent cantonments, except during periodic military tours of the North Western Frontier Provinces. The war in eastern India forced a change of strategy, involving widespread spraying measures in and around troop encampments and battlefronts. The resultant increase in demand of qualified manpower was dealt with by the inclusion of civilian malaria officers into the newly formed IAMC.

Another major challenge faced by the military medical authorities was the need to increase hospital capacity. Between 1942 and 1945, for instance, 117 general hospitals were raised. These were complemented by a series of ambulance trains capable of running on broad, metre- and narrow-gauge railway networks and of carrying between 196 and 268 patients. The increased scale of war by 1943 forced the military to embark on the construction of new types of hospitals and treatment centres, especially in the frontier regions. This resulted in the setting up of improved varieties of 'staging stations', which were capable of providing emergency surgical assistance and medical treatment for up to 100 patients. The work of these establishments was bolstered by the setting up of 'Malaria Forward Treatment Units', which could hold and treat 600 patients. These special health schemes helped to successfully provide curative and preventive medical services to the Indian army detachments operating in eastern India, even though some infrastructural frailties, especially nursing services, could never be completely banished during or after the war. The overall expansion of the military medical services, and its effects, was marked in all 1163 medical units; 197 539 new hospital beds had been created in the region by October 1945, and this new infrastructure treated 5,000,000 military casualties.



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With the expansion and stabilisation of the production of military supplies, the food situation eased somewhat, but items such as cloth, woollens, mineral fuels, chemicals and medicines continued to be in short supply for civilians. Whereas fuels like petroleum and coal were primarily stockpiled for military transport and industrial production, the rapidly falling stock of medicines, caused by the cutting-off of imports from outside India, were carefully maintained so as to improve the health of the army detachments and military labour in the eastern frontiers. Similar schemes were also targeted at the expanding mining and plantation industries, which were considered critical to the war effort due to the loss of imports. Even though the mines and plantations proved more difficult to provision, mainly due to transport problems, efforts were consistently made to keep up food and medical supplies to these establishments. In Assam, for instance, much attention was paid to the needs of those employed in the petroleum and tea industry, while official initiatives in Bihar and Bengal tended to be targeted at the coal and mica mines, as well as the tea plantations. In addition, concerted official efforts were made to ensure that employers introduced special welfare provisions for their workers and complied with these. Reports from the districts of Bihar often mentioned, for example, how they had regularly resorted to the Tea Districts Emigrant Labour Act (Act XXII of 1932) to force recruiting agencies attached to various companies to offer medical attention, subsidized food and regular wages for those tea garden labourers who had been used in military construction projects in Assam. Similarly, an ordinance was promulgated by the Indian Government in January 1944, which allowed the authorities to intervene in the administration of the coal mines and force the constitution of a fund to provide for a variety of facilities-antimalarial schemes, hospitals, good housing, water supplies and mobile cinema units catering to 'recreational needs'-for colliery labour in Bengal, Bihar and Assam. The importance given to increased coal production, labelled as an 'essential war effort' in 1943, was also underlined by the targeting of special anti-malaria operations at the coal fields, organized by the Indian Government, with military assistance, through the Malaria Institute of India from 1944 onwards. (Sanjoy Bhattacharya Tackling hunger, disease and 'internal security' : Official medical administration in colonial eastern India during the Second World War (part 1), The National Medical Journal of India, vol 15, no.1, 2002)

In the mid-1940s and fifties, the Indian medical system was young and full of ideas. With predecessors like Ronald Ross, JBS Haldane and their Indian successors like Y. Subbarao, Shambhunath Dey, V Ramalingaswami and Vasant Khanolkar, India was set to develop a strong interest in research of on traditional diseases like malnutrition and infectious diseases. The Indian Council of Medical Research (ICMR) established in 1911, its network of 21 permanent research institutes, and six regional medical research centres distributed throughout the country are responsible for all medical research and technical inputs into medical programmes.

Many newly instituted scientific institutions were discovering new diseases and agents and designing programmes albeit with over-dependence on technology. Between 1945 - 1965, at least 12 new viruses and new ranges of old ones were reported by the medical fraternity and reported in journals of repute. By the late 60s, most research on new diseases had died down largely because most diseases were considered conquerable through antibiotics and insecticides.

Programmes were planned at the central level and implemented at district and village level in the states. The Bhore committee in 1946 gave the concept of a primary health



centre as a basic health unit, to provide, as close to the people as possible, an integrated curative and preventive health care to the rural population with emphasis on preventive and social promotion of health care services. The Bhore Committee aimed at having a health centre to serve a population of 10,000 to 20,000 with 6 medical officers, 6 public health nurses and other supporting staff. The Central Council of Health at its first meeting held in January 1953 had recommended the establishment of primary health centres in community development blocks to provide comprehensive health care to the rural population. But in view of the limited resources, the Bhore Committee's recommendations, and the subsequent committees and the National Health Plan (1983) could not be fully implemented, despite considerable investments and effort. Increasingly, these centres came under criticism, as they were not able to provide adequate health coverage, partly because they were poorly staffed and equipped, and partly because they had to cover a large population of 1,00,000 or more, dispersed over a large area.

Health services are covered under the Indian constitution as a subject of the state and not essentially of the centre. Central government intervention and assistance is usually sought and given in the areas of control/eradication of communicable and non-communicable diseases, broad policy formulation, medical and paramedical education and regulation, drug control and prevention of food adulteration. Most infectious disease programmes are covered under the central programme, though the implementation is under the joint supervision of the state and central government agencies and departments. These programs also reinforce the delivery of primary, secondary and tertiary health care system in the respective states. During the year 1998-99, an outlay of 1145 crores including 524 crores foreign assistance has been expended in the central sponsored schemes. About one-half of the commitments towards the health programmes are from donors, since the 70s. In many of these programmes, state share costs with the Centre on an equal basis.

Donors, largely led by the World Bank and World Health Organisation gave the Indian health systems a model to replicate from success stories from elsewhere in controlling the largest single infectious disease – malaria. Most of the programmes were standardised with set targets and achievements. Almost all programmes were designed to meet these criteria. The reasons for the early successes and failures of following a set pattern of programme management are discussed in Table –.

Table : Guiding principles for disease control

Guiding principle	Early stages (1955 – 80)	Later stages (1990s)	Reasons for failure	Current strategy
Successful tropical disease control programs are technology-driven.	Strong adoption of insecticides like DDT to eradicate tropical diseases	Persistence with DDT and other insecticides despite resistance in vectors and pathogens	Too much reliance on technology and no impetus on epidemiology and demography. Simple technology and local innovations	Persistence with medical prophylaxis and other insecticides. Alternatives like bednets, bio pesticides, bioenvironment



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			not implemented	al control etc. neglected
Successful programs are "campaign-oriented"	Initial campaign successful as "technological wonders" like DDT showed immediate results. Little motivation to develop campaign around people or communities.	Campaign fervour waned as broad technological measures failed to show desired result. Widespread corruption at the grassroots and top bureaucracy avoid community involvement	Despite failures, dependence on DDT remained strong. Cheaper alternative like bioenvironmental control methods not adopted despite know-how being available	Several programmes initiated. No shared vision for control or strategic epidemiological research. Duplication in operations especially in insecticide application. NP campaign sentiment missing as people not empowered.
Successful programs rely heavily on expert staff - groups that have the authority to decide on technical matters	Scientific community given bureaucratic powers, making them overconfident in their approach	Initial success of technology made "expert" closed to other ideas. DDT's failure had terminated any active research in seeking alternatives abandoned	Technical staff depended on research done in the West, little contribution from local scientific community	New World Bank Programme of infectious disease controlled proposed (see box)
Successful programs use both vertical and horizontal modes of operation	More vertical than horizontal	Integration with existing health structure at the grassroots fails. State and central bureaucracy	Lack of holistic planning for communities at the grassroots. Urban programmes fail due to lack of interest in malaria control	No effort made to strategise and reorient the set up.
Successful control programs are largely centralised in formulating strategy and decentralised in operation	Both planning and operation centralised, little participation sought from local	No shift in involving communities or other agencies in control operations	Rigid and lack of innovativeness. Ability to manage two or more different infectious diseases difficult.	No new strategies adopted.



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	communities			
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Source : points for column 1 from Bernhard H. Liese, Paramjit S. Sachdeva, L and D. Glynn Cochrane (1991)Organizing and Managing Tropical Disease Control Programs : Lessons of Success World Bank Technical Paper Number 159, The World Bank, Washington DC

5A.4: Current programmes for disease control

Despite a sound institutional framework, infectious disease management in India lacks a policy framework. The decisions concentrate on macro – policy issues rather than micro-needs. The framework of infectious disease control largely based on four disease: malaria, tuberculosis, leprosy and polio. Malaria encompasses all vector borne diseases while others are largely individual projects within the health ministry’s mandate.

Program	Malaria Control	Filaria Control	Kala-Azar	Japanese Encephalitis
Launched in	First launched in 1958 modified in 1977. Received with World Bank assistance in 1994 and 1997.	Launched in 1955	Merged with malaria control programme in --	Part of the NAMP
Current Situation	In 1947 is approximately 75 million cases, currently – million cases occur annually	At present 14.18 million population in north, east and South India exist	In 1998, 13,360 cases and 217 deaths were reported.	In 1998, 1,617 cases and 335 deaths were reported.
Coverage	Initial program covered the entire country and now tribal and problem areas are more focused	At present 49.87 million people affected. Largely, urban population is covered under the scheme.	Program is being covered under National Anti-Malaria Program (NAMP)	Andhra Pradesh, West Bengal, Assam, Kerala, Tamil Nadu and Punjab reported maximum deaths. Covered under NAMP
Achievements	By 1965, malaria incidence was reduced to 0.1 million and deaths completely eliminated. By 1976 it resurged with nearly 6.4 million cases. The modified plan brought it down to 2 to 3 million	Delimitation of the problem in hitherto unsurveyed areas. Control in urban areas through	Provision of insecticides, anti Kala-Azar drugs and technical guidance to the affected states.	Vaccine has been developed indigenously but has not been propagated as it proves too expensive.



	cases a year.	antilarval and antiparasite measures		
Remarks	World's biggest programme against a single communicable disease. Due to the resurgence of malaria new and modified program was implemented from 1977	Training and research centres at Rajahmundry, Calicut and Varanasi.	Serious public health problem in Bihar and West Bengal	High mortality rate of 30 to 45 per cent in many areas.

5A.5: The institutions for disease control

India's health systems have proved to be inept at predicting and preventing the occurrence of infectious diseases. Sudden epidemics overwhelm public health systems, even in cities. Diseases emerge in densely populated cities and in far-flung places, but most escape the attention of medical research and remain unidentified.

The Regional Medical Research Centre for Tribals (sic) in Jabalpur, Madhya Pradesh has been working in tribal areas. In the past 17 years, their work has focussed on yaws (an innocuous form of syphilis) and malaria. Though they admit that there are many other infectious diseases, they have not attempted to control any single one or identify new pathogen that causes them. Says KK Tiwari, director of the centre, " The focus of our centre has been to study malarias and syphilis in tribal communities. All other diseases are simply ignored."

Even with better techniques to aid identification, quite a few epidemics remain unidentified. These indicate that either, entirely new strains or new organisms have emerged. But mostly clinicians have been unable to identify even the common pathogens.

A look into the system of disease surveillance in the country reveals some of its astounding shortcomings. There are no authoritative figures on the numbers of patients affected by diseases, the frequency of the disease or how widespread it is. Many outbreaks of easily identifiable diseases like malaria remain unreported. The major reason for this is the increased dependence on a single drug – chloroquine and analgesics, on the emergence of a fever, during the "fever" season.

Besides the unexpected, some known diseases also create problems in identification. Giving an example of the plague epidemic in Surat, Gujarat, Datta says that in case an old disease has emerged after a long time. People forget how to identify it. According to Kalyan Banerjee, former director of National Institute of Virology, Pune, changes in the pathogen can modify the its characteristics and confuse people and delay diagnosis. "Dengue fever, which now has a hemorrhagic manifestation, is a good example," he



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says. In such cases the correct diagnoses can be made only after laboratory investigation and it remains infectious till the results of the investigation are out.

Epidemiological studies are seldom linked to changing environment, population trends, migration of humans and animals through different areas. Few are willing to state the obvious. "Change in the ecology means change in the character of parasites that cause diseases. This has made diagnosis very difficult," says Debi Prasad Mishra, the state health minister. "And the most vulnerable group is the state's poorest tribal population. As their life is linked to the nature any change in it will hit them badly," he adds. One of the reasons for spread of malaria in the region (seventy per cent of the malaria cases are reported from tribal districts of the state) is the fast deforestation in tribal areas, which is leading to a rise in temperature. "Mosquitoes breed faster at higher temperatures and therefore spread faster," explains Kar. Mosquitoes that carry *Plasmodium falciparum*, causing cerebral malaria live in forests, but with deforestation, they have come to the human habitation. This could be the reason for this high incidences of cerebral malaria," says Amarendra Mahapatra, a doctor working with the RCMR on malaria. Cerebral malaria constitutes 85 per cent of total malaria cases in the state. According to state government statistics, districts with high deforestation rate are also the districts with maximum malaria cases.

"Early detection of a disease is possible only if an organisation like the NICD becomes an autonomous body," feels Datta. This would still leave the problem regarding the control of the disease. As of now, the focus is only on the control of the specific outbreaks and not on taking precautions to contain either the spread of the disease or the control of vectors. Adequate precautions are not taken to prevent the disease, feels AS Daga, assistant professor of preventive and social medicine at the Grant Medical College, Mumbai. "Every year epidemics of chicken pox and mumps always occur when schools reopen in summers but routine vaccinations against these diseases is not carried out," she says. In case of the plague epidemic, all the patients suspected to have the disease were kept together, says Deodhar. This led to the spread of the disease even to those who did not actually have the disease. Even when precautions are taken, they are often irrational. At the time of plague in Surat, Gujarat, Nirman Bhawan and the ICMR buildings in New Delhi were treated with insecticides to contain plague, informs Deodhar. The control measures taken are empirical and not specific and therefore ineffective. "Health officials feel that same control measures can be used to control all the diseases," says Banerjee.

Lack of coordination between the state government and the central government and even between the different ministries at the centre is also one of the major reasons for delays in the identification of the problem. The little infrastructure available in the country is not put to good use, as there are no linkages between the institutes. Lalji Singh, director of Centre for Cellular and Molecular Biology, Hyderabad says that they are fully capable of identifying any microorganism if the organism is isolated and sent to them. But according to Datta, collaboration is difficult. When CCMB was asked to help in the identification of the causal agent of Siliguri fever, the institute asked for the cost of the chemicals, which could not be given as the sample, was already being sent to NIV, which supposedly has more expertise in the field of virus identification.

The only way out seems to be strengthening of the surveillance system and building up on the infrastructure. The (draft) national health policy 2001 accepts the shortcomings in



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the disease surveillance system and hopes to overhaul the system by the year 2005. Unfortunately, once again the focus is on known diseases and their control, while new and emerging diseases are not being considered. Most of the ICMR institutes are working on very specific diseases and are not trained in identification of unknown organisms. "The focus of our centre has been to study malarias and syphilis in tribal communities. All other diseases are simply ignored," says KK Tiwari, director, Regional Medical Research Centre for Tribals (sic) in Jabalpur, Madhya Pradesh. Institutes like the Enterovirus Research Institute, Bombay work only on the poliovirus and therefore have no expertise on other infectious diseases.

Table : Institutes and their specialization

Institutions	Comments
Malaria Research Centre (MRC) Delhi	Malaria control and other vector borne diseases like kala azar, filariasis, Japanese encephalitis and dengue. Four country programmes initiated since 1953.
National Institute of Epidemiology (NIE) Chennai	Few applicable protocols or field research. Limited in scope and operations
Vector Control Research Centre (VCRC) Pondicherry	Vector studies on malaria. Excellent work on bio-environmental control of vectors done but not used extensively in malaria control programmes
Centre for Research in Medical Entomology (CRME) Madurai	Little collaboration on finding with other organisations like VCRC
Enterovirus Research Centre (EVRC) Mumbai	Research focus largely on polio
National Institute of Virology (NIV) Pune	Premier institute responsible for virus research. Recent CAG reports suggest tremendous laxity in research and misuse of funds
Rajendra Memorial Research Institute of Medical Sciences (RMRIMS) Patna	Focus work on malaria and kala azar. Limited research and out reach.
National Institute of Cholera and Enteric Diseases (NICED) Calcutta	Considerable research on cholera and other water-borne diseases. Has limited outreach in the community
National Institute of Occupational Health (NIOH) Ahmedabad	Limited research on few pollutants and occupation related illnesses. No wide scale epidemiology or environmental health research
National Institute of Nutrition (NIN) Hyderabad	Focus on nutrition but not on linkages to ecological and socio-economic linkages
Institute of Cytology and Preventive Oncology (ICPO) Delhi	Little research on environmental reasons or infectious disease induced cancers.
Food and Drug Toxicology Research Centre (FDTRC) Hyderabad	Little work done on food safety and infectious diseases
Other institutions of the ICMR	



Institute of Research in Medical Statistics (IRMS) Delhi, Centre JALMA Institute of Leprosy (CJIL) Agra, Institute for Research in Reproduction (IRR) Mumbai, Institute of Immunohaematology (IIH) Mumbai, National AIDS Research Centre (NARI) Pune, Tuberculosis Research Centre (TRC) Chennai, Institute of Pathology (IOP) Delhi

According to Kalyan Banerjee, surveillance system are not integrated in the public health system. Primary health centres are not equipped to identify the disease and alert the whole country. On the other hand, Deodhar feels that decentralization would do the trick and thinks that strengthening the *panchayat* (village institution) system would help. Till this can be done, the politicians, bureaucrats and the technocrats learn to act quickly on advice of proficient public health consultants. Strict protocols of what should be collected and at what time should be prepared to aid the process of identification. It is also necessary that the country builds up upon the infrastructure. “Even medical colleges do not have facilities to identify causal organisms,” says Daga. Along with investing on the equipment, maintenance and the staff, it is necessary that people be trained in epidemiology quickly. According to Banerjee, an important step towards facilitating the identification of pathogens would be to develop a bank where sera from patients all over the country are stored. At a time of an epidemic, this could be used to identify the disease by matching the antibodies. This way, we would also be able to evaluate whether the disease is new or has already occurred some time earlier, he says.

The National Institute of Virology in Pune had been established in 1952 in collaboration between the ICMR and the Rockefeller foundation, USA. Till 1978 it was called the Virus Research Centre. One of the major functions of the institute has been envisaged as the identification of epidemics in the country and so far, the institute has investigated more than 200 epidemics caused by viruses. According to the handbook of NIV, most have been identified. NIV had also been called in to identify the Siliguri disease but so far has not been able to identify the causal organism.

The premier medical bodies, ICMR and its main agency, the NICD have not made many strides to effectively study the emergence of diseases. Epidemiological studies are seldom linked to changing environment, population trends, migration of humans and animals through different areas.

5A.6: Uncertain future of disease control

The current framework deals with controlling infectious diseases acquired either by community (like malaria) or individuals (like leprosy or tuberculosis). In case of widespread diseases like malaria, the emphasis on eradication and prevention involves both treatment and other control interventions. The control framework in India for vector borne disease was developed solely with the purpose of addressing the population as a group at risk rather than an integrated control of host, environment, and transmission factors. Many large companies which have exert significant environmental pressures may have malaria and other disease control programmes but are seldom integrated with national programmes (see list in box below: Box: Organisations that can contribute to vector control programmes). Thus, the overall framework failed to integrate within the existing PHC system which replicated the same tasks of awareness building, education, motivation and training many times over. Immunisation programmes for many diseases worked well largely due to concerted efforts and targets that were donor led. Also social



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and environmental complexities were fewer as compared to other infectious diseases (B. Aylward et al).

Organisations that can contribute to vector control programmes

National Thermal Power Corporation (NTPC) and other thermal plants, steel plants, National Hydro Power Corporation (NHPC), public sector companies, cantonments, railways, inland waterway authority (IWA), ports, municipality and corporations, irrigation department, public works department, agriculture department, dam projects (like Damodar Valley Corporations), collieries and coal mines, ore mines, airports, forest department, revenue department, dredging corporations, large private plants and factories, industrial associations

The current model of infectious disease control focuses on short-term outcomes (cure rates, distribution of prophylactic medicines etc.) rather than a process (production of capacity of prevent future epidemics, disease surveillance, identifying percentage and mix of susceptible population etc.). Interventions only at clinical or chemical control levels are important rather than social and policy level. Disease control policy are a mix of elements, described below :

Element	Desired intention	Application	Current intervention
Nature of disease	Complete understanding of diseases and infectious agent	Restricted research on nature of disease especially with respect to social and physical environment	Fails to address fundamental of disease control as social, economic and environmental milieu not understood by planners and scientists
Verticality of programme and organisations	Centralised planning and decentralised implementation has effectively controlled diseases but cannot be made an objective for sustainable health improvement	Product and outcome more important than process.	Centralised and bureaucratic planning trickled to the grassroots level reducing people's participation. Low capacity of research and planning organisations in integrating the different dimension of the disease
Standardisation of operations	Desired only if condition are similar – eg malaria control in small island systems, guineaworm eradication in desert areas etc..	Uniformly applied even in extremely heterogeneous climatic, social and epidemiological	No major modification in strategy.



		conditions	
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Infectious disease management is about addressing local needs. Infectious disease control requires local knowledge, local empowerment, and wide and integrated application and coherent planning (see box below of people's participation).

Guinea worm eradication

India and other countries of the South East Asian region were officially certified as free of guinea worm disease (dracunculiasis) by the World Health Organization (WHO) in 1999. This is the second disease to be eradicated from the region smallpox was the first. Although guinea worm disease has been eradicated from India, it still exists in a dozen countries in sub-Saharan Africa, with Sudan having the most cases.

The 4th International Commission for Certification of Dracunculiasis Eradication gave its clearance mainly on the basis of the report of its three member certification team, which visited India in November 1999 and investigated 62 villages in five states where the disease had been endemic.

Guinea worm disease is thought to have existed in India for several thousand years. In 1947 India was estimated to have some 25 million cases. When the Indian government launched its national guinea worm eradication programme in 1983-4, nearly 40000 cases occurred annually in more than 12000 villages scattered over seven states. Sustained campaigns were launched at grassroots levels by agencies such as UNICEF and the WHO, with state and central government collaboration. The programme has been evaluated seven times by independent experts. It was a remarkable achievement of active intersectoral collaboration between the key departments of health, water supply, and rural development and multilateral institutions like UNICEF, which played a crucial part in the eradication of guinea worm disease. People's participation was the key to the success of the programme. The motivation and dedication of the village level workers to the top leadership played a critical part in this success story.

Source : Rohit Sharma, India eradicates guinea worm disease News, BMJ 2000;320:668 (11 March)

Short-term goals without long term objectives and competition between programmes adversely affect the successful control of diseases. Most interventions are only limited to health sector interventions and not control through the involvement of other stakeholders (see typology of disease control). With little understanding of inequality in access to health services and economic inequality created by diseases per se, deficient policy ensures that diseases persist in pockets of populations.

Current mix of methods employed for infectious disease control in India

Attack source	Interrupt transmission	Protect susceptible people
Treatment of cases (+), and carriers (-)	Environmental hygiene (-)	Immunisation (+/-)
Isolation of cases (- -)	Personal hygiene (- -)	Chemoprophylaxis (+/-)
Surveillance of suspect cases (- -)	Vector control (+)	Personal protection (+/-)
Control of animal reservoirs (- -)	Disinfection and sterilisation (-)	Better nutrition (+/-)



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Notification of cases (- -)	Restrict population movement (- -)	
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Key : (+) – practised or implemented, (+/-) moderately practiced, (-) rarely practiced, (- -) never practiced

Source : Vaughn and Morrow 1989 quoted in John Porter, Jessica Ogden and Paul Pronyk (1999) Infectious disease policy: towards the production of health , Health policy and Planning 14 (4) : 322 – 328, Oxford University Press.

Control programmes lack in many of the parameters mentioned in table-- . Each of the three phases requires a mix of skills – scientific, social and managerial. In the attack phase strong surveillance networks and feedback mechanisms. Participation of local communities is vital especially in notification and control operations in animal reservoirs. For interrupting transmission phase strong community mobilisation efforts and participation is required. It also incorporates greater scientific understanding of local environmental and social conditions (environmental hygiene rector control, restrict population movement) and behaviour (personal hygiene, disinfection and sterilisation). To protect susceptible population effective coverage and sustainable monitoring of populations is need. Immunisation, chemoprophylaxis, personal protection and better nutrition can be achieved only through education, effective participation, and developing inter-programme linkages (see box: Prescription --). Successful interventions as those in Sardinia and Peru for malaria, Chagas disease in south America, guineaworm eradication in India were some programmes that effectively used these components effectively to control or eradicate infectious diseases.

<p><u>Prescription</u></p> <p>What the government and the scientific community need to do counter epidemics Map susceptible populations of humans and potential reservoirs like cattle Map for ecological changes and corresponding changes in potential vectors Create special regional units within the ICMR institutions to monitor, predict and prevent epidemics Create a strong network from grassroots to institutions at state and national level to gather information about outbreaks Create local, regional and national level institutions to identify new epidemics and control them Involve medical colleges and physicians to identify local epidemiological development Create databases for information exchange and repositories of cultures Conform to international protocols, standards and best practices as those prescribed by WHO and Centers for Disease Control and Prevention, USA.</p>

5A.7: Global shift in infectious disease policy

The global society has realised that infectious diseases are crossing over boundaries and that there is a need to develop a common strategy for surveillance and develop uniform capabilities and standardised capacity in control operations. The current paradigm is not sufficient to address national, cross-border, regional and global needs.



India's notoriety as a source and hotbed of infectious disease is well known- from the plague scare to introducing Asiatic cholera in South America to re-introducing new strains of tuberculosis in Europe and the USA (RAND Corp report).

Table : Paradigm shift

From: Current public health orientation	To: Infectious disease policy
Disease specificity and verticality	Integrated / horizontal linkages
Standardisation of interventions	Flexibility/ context sensitivity
Short term (ad-hoc) orientation	Long-term objectives / sustainability
Emphasis on product/ targets	Emphasis on process
Limited to health sector	Linking multiple sectors
Focus on individual "risk"	Understanding local vulnerability; risk in the context of everyday life
Operating without reference to global processes	Taking globalisation as referent and context
Working in behalf of populations	Working in partnership with communities.

Source : John Porter, Jessica Ogden and Paul Pronyk (1999) *Infectious disease policy: towards the production of health, Health policy and Planning 14 (4) : 322 – 328, Oxford University Press.*

Nowhere in our health policy of 1963 and (draft) policy of 2001, have new infectious diseases been a focussed. Apart from malaria, other diseases receive scant attention. The weakness of the Indian health system lies in its rigidity and inflexibility to meet the ever-changing challenges. The two policies have not developed a comprehensive and integrated surveillance and response. Even today, the focus remains on therapeutic interventions rather than preventing outbreaks. Seldom have the different departmental agencies and ministries collaborated on projects. Water and sanitation departments, weather bureaus, strategic health units, malaria campaigns, housing boards etc. have no link to each other's work. Even during an outbreak, more deaths occur because the local government agencies are too overwhelmed and clueless. Even the high command in institution of the ICMR and NICD are groping for answers on how best to curtail rising infections.

The poorest states in India are the ones that suffer from most infectious diseases are also inconsistent in their commitments to infectious diseases largely because of inability to provide their share in disease control or have been too inept and corrupt to implement these programmes. This is evident from the payments and contributions towards the malaria control programmes (source: Special Issue on Malaria: A critical review, Journal of Communicable Disease Vol 19 No.3, Sept. 1987. Various Comptroller and Auditor General reports and Lok Sabha Committee reports show that the greatest misappropriations have happened in these states. The vicious circle of mal-administration, poverty, infections and malnutrition endures.

Operationally, India's control programmes have focussed on diseases and heavily relied on chemical pesticides and antibiotics. Though considerable success was achieved in many programmes, the efforts could not be sustained. Donor-led programmes though called campaigns are not translated into people's movements at the grassroots. They are at best good policy statements and country strategies on papers and do not leave country offices. Time and again the National Malaria Eradication Programme (NMEP),



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the largest national control programme has complained that the major cause was sudden withdrawal of assistance without consultation, especially for insecticide purchase. The attitude to the public of the expert committees and failure to gain public support and ownership is thought to have contributed significantly to failure of the campaign (CMH paper – see pdfs for source by two women). Multi-tasking for a large bureaucratic organisation like the malaria programme is difficult. It is not strategic enough in its operations and lacks the vision to come up with a comprehensive disease control programme, including malaria. With virtually no grassroots research and studies and blanket programme strategies, outbreaks appear frequently and epidemics extend their range into new areas. An example of such drawback is evident from the multidrug- and insecticide resistant forms of malarial and other pathogens have appeared, that have made infections harder to treat. Even for a well studied and supported disease like malaria does not have an effective management strategy. Sarla Subbarao and VN Kaul of the Malaria Research Centre insist that chemicals like DDT are still effective and will be deployed to counter malaria in India in the coming years (see box on DDT---). Even vectors have developed resistance to the intensive assault of chemicals. As health officials and scientists from various disciplines examined the forces responsible for these unexpected developments, they realized that the hope of overcoming infectious diseases was premature. They have linked the rise in infectious diseases to a breakdown in public health measures in every corner of the country. They also uncovered portentous signs that deteriorating health infrastructure and environmental conditions make cities and villages predisposed for the re-emergence of old infectious diseases and emergences and establishment of new ones (see box on the proposed disease surveillance programme of the World Bank).

The proposed Integrated Disease Surveillance Project

Integrated Disease Surveillance Project proposed by the World Bank and the Ministry of Health and Family Welfare (MOHFW) is proposed to commence in November, 2002.

The major sector issues the project proposes to cover are: overall low health status of the population, particularly the poor institutional arrangements and weak program management, low quality of health services in both the public and private sectors, ineffective targeting of the public funds to the inadequate framework for engaging the private sectors inefficiency and limited financial resources, poor governance.

IDA support for this project will add value by: building on and coordinating across previous investments by the Bank and other donors in disease surveillance; stimulating and accelerating the process of decentralization of disease control efforts to the states; strengthening capacity to respond to emerging changes in disease patterns; and strengthening overall capacity to manage public health programs. Complementing this is the Bank's ability to tap international experience. The Bank has an ongoing partnership with WHO and CDC, who would both be involved in this project. The project proposes to coordinate and decentralize disease surveillance activities by : Integrating and strengthening disease surveillance at the state and district levels; improve laboratory support; train staff for disease surveillance and action.



The GOI would commit 10 million USD, while the World Bank Groups would extend a 60 million USD, The credit will be on standard IDA terms with a maturity of 35 years including a 10-year grace period. The credit will be provided by the International Development Association (IDA), the World Bank's concessionary lending arm. The MOHFW and World Bank so far have not provided any documents or information on project logistics and partners in the programme.

Globally most countries have redesigned their programmes to address the changing needs. More agencies and communities are being asked to participate in managing infectious diseases. In Panama and Costa Rica for example, every agency and civic body responsible for managing water and water resources is also responsible towards the vector control programme. In Chad, the health department has prepared a health matrix of infectious diseases and looked at how it can effectively manage the combined assault in an integrated manner. In India, at least eighty organisations could be involved actively in the control of vector borne diseases (see table—in the malaria chapter). Many of these have budgets and personnel dedicated for this purpose. However there is an absence of a shared vision and working in close coordination with the national programmes.

The current health policy framework does not consider any of these factors in managing the programmes. The Ministry of Environment and Forests does not even consider infectious disease as a part of its responsibility. The least it could do is to include it in the health impact assessments, without which environment impact assessment is a mockery.

Each infectious disease is an evolutionary success story, from oblivion to pandemic proportions, is a remarkably simple. Diffusion of people, mixing of species, poverty, war, squalor, environmental change and susceptible populations, all contribute to the rise of the these pathogens. The best that governments and institutions can do is keep a few paces ahead of the disease through constant monitoring, simple hygiene, collective action and some common sense.

5B: The diseases

5B.1: Japanese encephalitis

Japanese encephalitis (JE) is a leading cause of viral encephalitis in Indian sub-continent. Few countries like China and South Korea have controlled the spread of the disease through effective surveillance and vaccination programs. JE was first described in 1871 when an outbreak occurred in Japan. The disease has been reported from areas as geographically diverse as China, Korea, India, parts of Bangladesh, southern Nepal, Sri Lanka, Myanmar, Thailand, Cambodia, Laos, Vietnam, Malaysia, Indonesia and Philippines. Japanese encephalitis also occurs in Japan, Taiwan, Singapore, Hong Kong and eastern Russia, but with lower frequency.



JE is widespread through India and covers 24 states and union territories (14,15,16,17). An estimated 160 million people are at risk of acquiring JE in India (17). There is a large gap in the numbers of reported cases and expected cases (1,18). One estimate suggests that only 2% of JE cases are recorded (1). Mortality (measured as case fatality rate) apparently increases proportionately as the numbers of cases rise. It is endemic in some regions in India. Every year, eleven districts of Andhra Pradesh brace themselves for another JE epidemic. In 2001 and 2000, few cases -- -- -. In 1999 it claimed over 100 lives - mostly children. The fever was earlier confined to some pockets of Mahabubnagar, Warangal, Medak and Karimnagar districts, but has now spread to most of the Telengana and Mehboobnagar districts. Government data showed that there were 984 cases of Japanese Encephalitis, including 247 deaths in Andhra Pradesh in 1997 and 524 cases, including 201 deaths in 1998. Even though the death toll this year is less compared to the previous years, the Japanese Encephalitis is spreading with multiplier effect to other districts in the State, causing concern to the administration. The medical department in the State is unable to fight the diseases. Hundreds people have died in the tribal communities in Adilabad and other districts in the past due to a 'mysterious fever' that stalked the tribal belt. ((XIII LOK SABHA DEBATES, *Session II (Winter Session)* Monday, November 29, 1999/Agrahayana 2, 1921 (Saka) alfa.nic.in/lsdeb/ls13/ses2/14291199.htm)

It is difficult to eradicate JE because it is transmitted from natural reservoirs like pigs and cattle, and wading birds like ducks, herons and egrets.

Japanese Encephalitis is the only infectious viral disease recognised by NICD and the National Institute Virology, Pune, to *cause epidemics of encephalitis in India* (other viruses cause sporadic cases) (21,22). JE has high mortality and morbidity rates (~1.5/10000 population) (19). It usually affects children of 5-10 years who live in close proximity to cattle, pigs or ducks. Boys are more frequently affected than girls (24). Adults are usually affected either due to fresh introduction of virus as in West Bengal or vaccination of children as seen in Korea, Taiwan (25) and Japan (26).
Ecology of the disease

Many diverse strains occur in India. The North Indian strain of the virus (GP78) is genetically closer to the Chinese SA14 isolate (27). The more common south India strain, the Vellore P20778 isolate, was obtained from various pockets of south India in 1958 (28). JE virus has a complex life cycle. In nature, JE virus is maintained in animals and birds, particularly pigs and wading birds (e.g., Cattle egrets, pond herons etc.) poultry, ducks, frogs, snakes, alligators, and most domestic animals like pigs, horses and cattle (5).

Pigs, like other reservoirs play an intermediate host to the virus, which helps them multiply inside them, without causing them any harm. (3,29,30,31). Countries that do not rear pigs like Pakistan have JE only very rarely with rare epidemics in 1983-84 and 1992 (32,33). High cattle to pigs ratio (400:1) is an important factor for reducing JE infection rate in children (34). Interruption of viral transmission in animal reservoirs, by vaccinating or culling infected pigs or through vector control, is an adjunct strategy, but cannot be solely relied upon to control the disease (35,36). JE virus is carried by female



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mosquitoes from infected pigs (37,38) or water birds like pond herons, Cattle egrets and ducks to susceptible children.

Table 1: JE in India in 2000*

State	Total Cases	Deaths	Case Fatality Rate
AP	343	72	20.99
Assam	158	69	43.67
Bihar	77	19	24.68
Goa	15	3	20.00
Haryana	74	43	58.11
Karnataka	417	34	8.15
Maharashtra	2	0	0
UP	1112	253	22.75
West Bengal	91	29	31.87
Total	2289	522	22.80

* - Source: NICD /NAMP, New Delhi Statistics

Table 2: JE cases and CFR* in Andhra Pradesh from 1998 to 2000^N

Year	Cases of JE	Deaths due to JE	Case Fatality Rate
1998	524	201	38.36
1999 [§]	1036	203	19.59
2000 [§]	343	72	20.99
Total Cases	1903	476	

* - Case Fatality Rate

N - Statistics of Director of Health, Govt. of Andhra Pradesh.

§ - Je Cases in AP showing the drop in Case Fatality Rate after implementation of measures to prevent complications since 1999.

Source: Statistics of Director of Health, Govt. of Andhra Pradesh, Hyderabad 2002.

The main vector, Culex mosquito (*Culex tritaeniorhynchus*, *C.vishnui*, *C.pseudovishnui* and others – totally 8 species) breeds in flooded rice fields, marshes, and standing water around planted fields and can fly up to 5 Kms. It rests on rank vegetation or sugarcane.

This is the reason JE is mostly a rural disease. The spread of JE to new areas is probably due to agricultural development and intensive rice cultivation, increased use of nitrogenous fertilizer (40), supported by extensive disturbance to local ecology by irrigation schemes (see box below) (41,42).

Why blue-green algae is good for rice and bad for mosquitoes

The extensive use of inorganic fertilisers in paddy fields is fuelling mosquito-borne epidemics of Japanese encephalitis, according to Indian researchers. Using environmentally friendly organic fertilisers like blue-green algae could control such epidemics, they say.



The Institute of Vector Control and Zoonoses in Hosur studied the soaring use of inorganic fertilisers in fields of high-yield varieties of rice and correlated the use pattern with the rise in JE. In the state of Tamil Nadu alone, farmers used 791,000 tonnes of the chemicals in 1996-97 compared with just 296,000 tonnes in 1970-71.

"Mosquitoes that transmit Japanese encephalitis mainly breed in rice fields," says researcher, John Victor. So he and his colleague Rachael Reuben investigated whether the large-scale use of fertilisers could influence mosquito populations. Using 60-square-metre plots, they compared the effect of nitrogenous inorganic fertilisers and nitrogen-fixing blue-green algae bio-fertiliser on mosquitoes during the rice-growing season. They found that plots given the recommended dosage of 100 kilograms per hectare of nitrogenous fertiliser had more than double the number of mosquito larvae compared with plots sprayed with bio-fertilisers or with no fertiliser at all. But rice production suffered when only organic fertilisers were used. "We cannot completely avoid nitrogenous fertilisers, because we need a better grain yield also," says Victor.

So the researchers tested plots with a mixture of organic and inorganic fertilisers. These plots had only 50 per cent more mosquito larvae than the controls, but with better yields than before. "Agricultural scientists are now promoting the use of blue-green algae in rice fields in India," says Victor. "We recommend it mixed with lesser amounts of nitrogenous fertilisers."

Some strains of blue-green algae are toxic to mosquito larvae in laboratory conditions. These strains could be added to the fertiliser mix, says Victor, although more work needs to be done to check whether they are effective in rice fields.

But international funding for such research is lacking, says Martin Birley of the Liverpool School of Tropical Medicine, who's also a member of a United Nations panel on environmental control of diseases like Japanese encephalitis. "The agencies that control research are not enthusiastic about funding for environmental methods of mosquito control," he says.

Adapted from Anil Ananthaswamy Why blue-green algae is good for rice and bad for mosquitoes, New Scientist magazine, vol 168 issue 2270, 23/12/2000, page 14

There are several other species of mosquitoes that are possible vectors of the infection. A review of studies done by ICMR suggest that at least 16 species of mosquitoes of the three major genera (Anopheles, Culex and Mansonia spp.) are possible carriers. Yet the mechanisms of the disease spread are unclear in many parts of the country. (ICMR study available at www.icmr.nic.in/buapr2000.pdf)

Epidemics coincide with the monsoon and post monsoon period (August to December) (4). Drying of rice fields resulting in fewer vector habitats, lower temperature, and/or reduction of number of susceptible host animals may be resulting in termination of epidemics (29,43). It is possible that in endemic areas, sporadic cases occur throughout the year due to congenial climatic conditions throughout the year (e.g., Southern India) (6). Between August and December 2000 there was an outbreak of Japanese



encephalitis in the Nawadah district affecting all villages, with 74 reported cases and 19 deaths according to the official report. The media reports indicate a higher number of deaths. In the district there is extensive rice cultivation and a large pig population. The conditions are suitable for the periodic recurrence of epidemics of Japanese encephalitis. The disease occurred in the post monsoon period. The disease occurs as an epidemic every 2-3 years.

Control strategies for JE

There is no concerted effort to prevent JE outbreaks in India. Though a part of the malaria control programme, the initiated rests with individual states where there is severe epidemic threat of the disease. Andhra Pradesh is the only state with some organised effort to combat the disease.

Integrated pest management in Kurnool

Kurnool district in Andhra Pradesh, is endemic for both malaria and Japanese Encephalitis (JE). The incidence of rural malaria is high in this district and outbreaks of the JE have been reported in the past 5-6 years. *Anopheles culicifacies* is the vector of malaria while *Culex tritaeniorhynchus* is the vector of JE, both of which breed in rice fields, mine pits and borrow pits. DDT has been used for indoor residual spraying (IRS) against malaria. Measures used for control of JE vectors are the use of Malathion fogging before and during epidemic season i.e. September to December, each year. To reduce the dependence on chemical pesticides and promote environmental friendly and cost effective methods, it is proposed to field test integrated vector control measures under the framework of integrated agro-ecosystem management at the district level.

Malaria is unstable in Kurnool. In 1999 there were 6771 malaria cases, and in 2000, malaria cases were reduced to 3391. But in 2001 there was a resurgence of JE with 7223 cases, showing the unpredictability of the disease.

Vector breeding is profuse in the rice fields and mining pits, which are found all over the district. Irrigation is the main source of innumerable vector breeding habitats. The source of irrigation is the Srisalam Project on the Tungbhadra River. In Andhra Pradesh, Kurnool is an important rice-producing district. Rice crop is attacked by a number of insects like leafhoppers, stem borers, gall fly etc. for which farmers and agricultural extension workers have been trained to adopt IPM practices since June 2000. Co-ordination committee comprising the health, fisheries, veterinary, Panchayat Raj and Municipal administration has been proposed to work on making integrated pest management a success and with the focus of reducing the dependence on pesticides like malathion. The results are awaited from the project.

Measures to control mosquitoes like monitoring movement of cattle and people, shifting pigsties, environmental sanitation, avoiding mosquito bites and vaccination of the susceptible population together are the best mix of curbing the disease.



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China, Korea, Japan, Taiwan and Thailand faced major epidemics of JE in the past but controlled it primarily with vaccination (1). The only internationally licensed JE vaccine, an inactivated mouse-brain derived vaccine, is efficacious (89) but is problematic from the perspectives of reactions it can cause. Protective immunity induced by the inactivated JE vaccine includes JE virus-specific T cells as well as antibodies with multiple biological activities (90). Mouse brain-derived, Beijing-1 and Nakayama JE vaccines induce high levels of neutralizing antibodies (91). High levels of induced antibodies are maintained at least for 3-4 yr. Immunization is supplemented by booster effect of natural JE infection in endemic areas. This hypothesis is supported by the fact that JE incidence rates have declined in countries with national programs of routine JE vaccination (Japan, Korea, Thailand, China).

Indian JE vaccine is a formalin inactivated vaccine made from the brain of suckling mice inoculated with the Nakayama JE strain, produced at the Central Research Institute (CRI), Kasauli, Himachal Pradesh. The reported cases and deaths due to JE in the affected States like Andhra Pradesh, West Bengal, U.P. Tamil Nadu and Assam according to the Planning Commission had shown considerable decline during the Seventh Plan with the use of indigenously produced vaccine (Planning commission: <http://planningcommission.nic.in/fiveyr/8th/vol2/8v2ch12.htm>). However, the sporadic re-emergence has taken even well an equipped state like Andhra Pradesh by surprise. So far, since there is no evidence of man-to-man transmission, vaccination protocols unlike polio, protect only the vaccinated and does not protect the community at large. JE vaccine is not useful for control of epidemics and is effective only during inter-epidemic periods. Newer vaccines like the single-dose attenuated SA14-14-2 available in China and South Korea, are not yet available in India. It has been administered safely and effectively to more than 100 million children in China since 1988 and Korea. (source : The International Vaccine Initiative (IVI) site : <http://www.ivi.org/jeinasia.htm>)

It is safe for children and effective with one dose costing about Rs.20/- for prevention from JE disease in JE endemic areas. However, production and regulatory standards are unresolved (1). In endemic areas, where sporadic cases occur, throughout the year, the cost effectiveness of vaccination is very low to be considered as the method of choice. To protect infants in endemic districts, 18 million doses of JE vaccine (costing Rs 92 crores) would be required and this has to be followed by boosters every 3 years, which has enormous additional cost of 120 crores (17). The risk of JE is not perceived to be widespread and is limited to some areas, and therefore JE immunization is not included in the National Immunization Program in India. Alternative strategies like bio-environmental control have never been used in vector-borne diseases apart from a few projects for malaria. So much so that traditional agricultural practice of using blue-green algae and *Azolla* in rice fields was eliminated after the use of phosphate-rich fertiliser, which promoted the breeding of vectors. (see box --)

JE virus vaccine is provided as part of the routine childhood immunization programs in some southeast Asian countries. However, in India and other countries in the endemic region, the vaccine is used on a very limited basis in part because of vaccine cost, and a lack of knowledge regarding the severe morbidity and economic impact associated with JE illness and post-JE illness-associated disability. Additional concerns have also arisen regarding the safety and adverse events following immunization with the inactivated,



mouse-brain derived vaccines, although such adverse events have not been systematically documented.

5B.2: Kala-Azar: An overview

Kala azar or visceral leishmaniasis (VL) is an infectious disease, meaning *black sickness* or *black fever* in Hindi owing its name to its characteristic blackening of the entire body associated with low-grade fever (Lainson and Shaw, 1987). The disease is spread by the female sandfly, *Phlebotomus argentipes* that carries the parasite, a single-celled protozoan, *Leishmania donovani*. Kala azar or visceral leishmaniasis (VL) affects 12 million people in 88 countries 16 from developed nations. 72 from developing countries and 13 from the least developed countries. According to the World Health Organisation (WHO, 1999) estimates around 12 million people were infected with the disease worldwide and around 350 million were at risk of contracting it. Currently, the global annual incidence of kala azar is estimated at 1.5-2 million new cases per year, with the distribution of 1.0-1.5 million cases of cutaneous leishmaniasis and 50,00,00 cases of visceral leishmaniasis. Countries like Bangladesh, India and Nepal together account for 90 percent of the cases that occur globally.

Out of the 500 known sandfly species, only 30 are identified as vectors of the disease. Leishmaniasis is the general term given to all the manifestations, and visceral leishmaniasis is the most common and prevalent form in India. Other less common forms are cutaneous leishmaniasis (CL) and mucocutaneous leishmaniasis (MCL) or mucosal leishmaniasis.

In India, the regions of Bihar, West Bengal, eastern districts of Uttar Pradesh, Assam, foothills of Sikkim and some parts of Tamil Nadu and Orissa face major incidences of kala azar. The calculated disability-adjusted life years (DALYs) lost due to the disease in 1990 were 6.8 million for men and 0.5 million for women. This difference is attributed to the fact that men are at more risk than women are, since they come in contact with the sandflies more frequently due to their work outdoor. Graph: No. of cases and deaths in India since 1991

The disease is not new to India and has been known for thousands of years. India recorded the first epidemic of kala azar in 1824-1825 in a place called Muhammadpore in the Jessore district of Bengal (now Bangladesh). It was then named "Jwar-Vikar". In 1832-33 Nadiad district of Bengal reported cases of kala azar. Similar incidences recurred in 1885, 1897, 1913, 1925 and 1944, showing regular intervals of approximately 12-15 years in both Bihar and West Bengal. South India has its first case in the late 19th century.

Kala azar is a perfect example of a complex life cycle. Fever, enlarged abdomen, general weakness, headache, dizziness, weight loss, sweating and diarrhoea with some characteristic symptoms being blackening of skin and enlargement of spleen and lymph nodes. The major forms of leishmaniasis are:

- Visceral leishmaniasis (VL)
- Cutaneous leishmaniasis (CL)
- Mucocutaneous leishmaniasis (MCL)



Though the sandflies are distributed over Uttar Pradesh, Madhya Pradesh, Gujarat and other states, density that is an important factor for its propagation is not maintained. Bihar has the most ideal conditions. **(See box: Bihar- Reservoir of kala azar)** According to Dhiman, Assistant Director, Malaria Research Centre, "It is important to understand the significance of sandfly density as a transmission factor. The sandfly prefers to feed on animals up to 80 per cent (Zoophilic), very few feed on human beings and even if they do they survive for only 10 days, the minimum period during which infection can take place". The chances of being infected by a patient of kala azar are also less if the patient has previously received some treatment for the disease. Therefore high density of sandflies is a vital factor for indigenous transmission. The infective dose of the sandfly is not known. The density that is an important and contributing factor in the transmission, is the number of sandflies a person collects in one hour. Moisture is a significant component for the sandflies to survive. It is almost impossible to kill the sandflies in their immature and juvenile stage.

Bihar: Reservoir of kala-azar

Ever since the first outbreak of kala azar in 1880, the tale of sandfly infections in Bihar has been one of death, ruin and devastation for millions. Bihar alone accounts for more than 40 per cent cases of the total cases (Bulletin of Tropical Medicine and International Health, Vol 8, No.1 2000). The burden of the disease is alarming as compared with its prevalence in any other state in the country. Bihar being the poorest state in India and very little has been done towards controlling the disease ever since its first appearance the disease has only worsened. One of the major epidemics occurred following an earthquake in the 1934. Over 70 years the situation has become a crisis, with 38 among 42 districts facing kala azar epidemic. (MAP ON DISTRICTS OF BIHAR.) The districts affected have reported an annual average of more than 1000 cases annually in the last ten years. Though in the earlier part of the century Assam and West Bengal were worst affected, strict monitoring over spraying of insecticide, active case surveillance and early treatment resulted in a gradual decrease in the incidence. Ecological changes have further contributed to the decrease in the number of infections, since deforestation has destroyed the sandfly's habitat. GRAPH: CASES IN REST OF INDIA AND BIHAR TILL 1996 (Save as bar graph)

"While North Bihar (now Bihar) is in the grip of kala azar, South Bihar suffers from malaria", says Mahabir Das, former director, Ministry of Health and Family Welfare, Bihar.

The rural-urban migration does not play a significant role in the disease. Alongwith it poverty brings the added burden of diseases. Malnourishment indicative of poverty keeps the people from the rural areas constantly in the vicious cycle of health emergencies. Inaccessibility to Primary Health Centres (PHCs) aggravates the number of fatalities.

The environment link



The sandfly is usually found in places with poor ventilation, moist cracks and crevices. Dams holding large amounts of water play a perfect role in the breeding of the sandflies. Agricultural practices and resettlement programmes bring people into areas of high vector and reservoir concentration. People in occupations such as forestry, mining and farming are at maximum risk. Deforestation destroys the sandfly's natural habitat, thereby facilitates its movement into human settlements. In the absence of wild animals in the deforested areas, sandflies begin their assault on other animals as well as humans. Water-logging, especially in places with continuous rainfall and floods, creates a perfect breeding ground for sandflies.

Affected populations

People living in mud houses give rise to more reasons for the increase of the disease. These houses nurture the sandflies and support them for further transmission.

Poor people tend to be susceptible to infections because of continually compromised immune systems. It has been observed that people with AIDS are even more vulnerable to kala azar adding to the severity of a disease like AIDS. (See box: HIV and kala azar: A growing phenomena) Children of the households are more at risk of the disease because they are involved in domestic as well as external activities. Men spend most of their time in the company of animals, either domestic or wild, frequently encountered in land clearing and cultivation. Men are afflicted with kala-azar two or three times more than women are. Most victims of kala-azar are male children under the age of nine. This is understandable when we consider the sandfly reservoir - the domestic animals that the boys feed, graze and clean. The curious nature of young children often places them in direct and sustained contact with a host animal.

Though it can occur in all age groups, the most vulnerable are children aged between five and ten years. It has been noticed that the incidence of kala azar is the same in both sexes before attaining puberty, incidence of the disease in both the sexes is same but in the reproductive age group males are affected twice as much as the females.

The HIV connection

Many kala-azar infections are self-limited and never progress to cause illness; however, the infection may remain dormant and cause severe disease at a later stage if the immune system is destroyed, in patients with AIDS. Leishmaniasis is one of the opportunistic infections that attacks HIV-infected individuals. Most of the co-infection involves the visceral form of leishmaniasis. Until 1999, 31 countries reported leishmania/HIV co-infection. The overlap in geographical areas with high risk of both HIV and leishmaniasis is increasing, with the spread of leishmaniasis (typically a rural disease) into urban areas and the increased spread of HIV into rural areas. In HIV-infected patients, leishmaniasis accelerates the onset of AIDS by cumulative immunosuppression and by stimulating the replication of the virus. It also may change asymptomatic leishmania infections into symptomatic ones. In addition, since visceral leishmaniasis can be spread intravenously, the sharing of needles by intravenous drug users is a direct way of spreading leishmaniasis, as it is for the spread of HIV. Leishmania and HIV co-infection is considered as an emerging disease, especially in



southern Europe where 25-70 per cent of adult cases are related to HIV infection and 1.5-9 per cent of AIDS cases suffer from newly acquired or reactivated visceral leishmaniasis (WHO 1997). Co-infected patients show severe clinical manifestations including gastro intestinal symptoms.

Treatment of the disease has many limitations to it, be it the diversity in medicines or the lack of vaccines and its progress. (See: Table of the drugs and their merits and demerits)

Drug	Disease	Merits	Demerits	Comments
Pentavalent antimony	visceral leishmaniasis (VL)	Simple to administer.	Due to increased resistance, less than 88 percent of the cases are not cured	Longer use of the drug may increase toxic effects
Pentamidine	Cutaneous leishmaniasis (CL) Visceral leishmaniasis (VL) Mucocutaneous leishmaniasis (ML)	Cures all resistant patients.	Unresponsiveness increasing, larger doses needed	Considered as a second line of therapy because of toxicity and its sub-optimal effectiveness
Amphotericin B	Visceral leishmaniasis (VL)	Cures all general symptoms of leishmaniasis	Toxic, costly and has side effects	–
Lipid formulations of Amphotericin B	Cutaneous leishmaniasis (CL) Visceral leishmaniasis (VL) / Mucocutaneous leishmaniasis (ML) in one.	Very effective without any side effects	Very costly.	Still not clear whether effective for CL-ML; found effective for VL

Source: CP Thakur, Year?

Control strategy for kala-azar

Kala azar is one of the very few diseases that can be controlled if understood and diagnosed properly and at the right time. The system of combating the disease not only involves the control of the vector and the parasite but also calls for the participation of the people and education of the communities. The control methodology usually involves



spraying of insecticides, administering drugs for treatment, bio-environmental (See box: Biological control) methods using biolarvicides or self-protection with insecticide-impregnated nets or mosquito repellents.

Biological control

The increase in the use of the biolarvicides proves effective and useful in the control of the disease. Biolarvicides with bacillus formulations are known to be highly lethal against the immature stages of mosquitoes. Two Russian formulations, Sphericide and Bacticide, were administered against the larvae of *P. argentipes* and *P. papatasi* under laboratory conditions. (Dhiman, (1998) unpublished data) Bacticide was found to be very effective against the larvae when mixed with sandfly larval food, resulting in 100 percent mortality of the first stage larvae of *P. argentipes* within 24 hours. But it has not been administered in the large scale.

It was observed that in the laboratory conditions the larvae of sandflies are susceptible to biolarvicides resulting in 100 per cent mortality from 1-6 days when mixed with larval food. Fourth stage larvae are less susceptible as they may overcome feeding and change into non-feeding pupal stage. But the application of biolarvicides in field conditions (mixing with soil and that too in diverse habitats) seem difficult at present.

Since mass elimination of the parasite from the human host as well as from the reservoir is not both improbable and impossible, most of the control methodologies are based on vector control that contained the reduction of host-vector contact and reduction of the sandfly population using insecticidal spray. *Phlebotomous argentipes* is susceptible to the usual doses of DDT, 100 milligrams per square foot or 1 gram per square meter on all indoor surfaces upto six feet from the ground. Under the kala azar control programme (1990-91) the government of India recommended two rounds of DDT for effective control. The spray seasons and the dosage vary in places depending on the occurrence and severity of the disease in the particular area. For instance, in Bihar the season is from February - March and again May – June, but in West Bengal, it is from May - July and then again from August - October. One of the most effective methods was the plastering of the walls of thatched houses with insecticide. Malathion paint formulation applied to the walls showed encouraging results in Bihar for about two years but due to inconsistency in spraying the vectors promptly became resurgent.

It is necessary to fully understand the ecology of sandflies, their feeding habits, population dynamics, resting behaviour and feeding behaviour if effective controls are to be designed and applied. They breed in moist soil in corners of mud houses, rodent burrows in the houses. They usually prefer resting in shady vegetation especially in peridomestic areas and indoors. This calls for increasing the efficiency of indoor residual spraying (IRS) with insecticides. Routine spraying does have a long-term effect on the vectors. Timely and adequate indoor spraying must be adhered to. One problem that comes with this is that people in villages smear their homes with mud and dung as part of ritual domestic cleaning. Kala azar is a site infection because of short flight range of sandflies, which move by hopping rather than flying. Animal reservoirs such as dogs and rodents could be eliminated as soon as they show signs of being infected to further increase the transmission. Initially, sandflies were considered highly susceptible to insecticides, but indiscriminate spraying created the problem of the parasite's resistance.



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In Bihar, where spraying initiated in 1976, resistance was reported from Patna, Darbhanga, Vaishali and Samastipur districts.

Repellants like diethyltoluamide (DEET) applied to the exposed areas of the body protect against sandfly bites. Though it is not a very reliable method of protection, it could be used as an alternative control. The transmission can be interrupted through better clothing; better housing and use of insecticide impregnated bed nets. The use of these bednets is considered as the most effective and safe strategy of control. There are other methods of control, that is the environmental friendly ways. (See box: Garden plants: more than just ornamental plants)

Garden plants: more than just ornamental plants

Some garden plants can shorten the life-span of the vectors that cause leishmaniasis. A study conducted by researchers of the Hebrew University - Hadassah Medical School, Jerusalem, showed that *Bougainvillea glabra* and some other ornamental plants have the potential to control breeding of sandflies. The vector *Phlebotomus papatasi* that transmits the parasite *Leishmania major* causing cutaneous leishmaniasis, can be considerably controlled by the presence of these plants in or at the risk areas. The continuous exposure of these vectors to the plants led to a significant shortening of the life span from 33 days on a control plant to nine days on *B. glabra*. It was noticed that exposure to plant sugars in a single night, followed by sucrose feeding, had a direct impact on the mortality rate of these sandflies. However, the small or stunted plants had no appreciable effect.

Why control is not working

The basic problem in kala azar control is the lack of diagnostic tools at the field level. Treatment is costly and requires hospitalisation at least for one month. Some persons are not even responsive to the first line of drug i.e. sodium stibogluconate. The second line of drug, Pentamidine is expensive, and therapy may cause diabetes in some cases. Since the initial fever is low grade, poor people usually ignore the disease in the beginning. As there is no method of active case finding in the present health system, such cases go undetected. There is also the problem of asymptomatic cases. Active case detection and treatment is the only solution, so that treated cases are parasitologically free and do not become a reservoir again. Post Kala azar Dermal Leishmaniasis (PKDL), form of kala azar, mostly confined to the skin, is a potent source of infection. In many cases, local customs and beliefs combined with the patients' fears, do not accept or allow for intervention by western methods of diagnosis and treatment. Due to financial constraints and limitation of the government (both the state and the central government) in terms of providing operational cost of the programme implementation further narrows down the efficiency of the programme. C.P. Thakur, Union Health minister, announced of a "kala azar elimination programme" in June 2001. The Union Health Ministry made DDT spraying mandatory for malaria and kala azar for the year 200-01. It has been decided that around 7000 metric tons of DDT would be used for the control of malaria and kala azar.

The WHO recommends building of homes suited to the prevention of sandfly access, removal of rubble and garbage piles to remote dump areas, and the use of fumigation



mats and canisters. Members of a poor village are not likely to be able to afford the repellents and insecticides recommended, and their beliefs may preclude the use of these, even if they were supplied free of charge.

Even if treatment is initiated, and the first round of injections administered, follow-up can be difficult because of the overall inaccessibility of primary health centers (PHC). The unresponsiveness of 40 per cent cases in Bihar to sodium stibogluconate compels the people to depend on costlier and more toxic drugs like Amphotericin B and its lipid formulations.

Future interventions

There are no easy answers in the fight against kala-azar or many other types of diseases endemic to various areas of the world. A change in architecture alone will not halt the spread of the disease, nor will organisational recommendations that cannot be realistically applied. Factors relating to the vector and parasite of the disease in mind while planning the control strategy.

A very simple and practical method of control could be the tilling of soil, which is possible on the fields, and not in the houses but the problem could be curbed somewhere.

Health infrastructure should be able to reach even the remotest corners to evaluate specific needs of the people living in villages, by providing sustained access to the PHC. House - to - house detection, and supplying specific drugs for effective treatment of all cases should become a routine project and should be under strict vigilance.

The anti-vector measures which involve spraying of insecticide should be carried out promptly and the two rounds of DDT recommended by the government of India should be thoroughly implemented. Intensive health care programmes should be carried out regularly.

The current problem India faces is not only the ignorance of the people but also of professionals such as doctors and primary health officials. Intensive training programmes for medical and health officials is a critical step. A customary epidemiological survey to adapt various control measures should be undertaken by the agencies involved in the control programmes. The WHO (1990:56-63) recommends curtailing outdoor activity after dusk, wearing of protective clothing and insect repellent, the use of fine mesh screens on windows, doors, and as bed nets.

5B.3 Dengue: An overview

Dengue is a global infection and remains a potential health problem in areas that are infested by the *Aedes aegypti* mosquito. Until 1970 only nine countries were facing an epidemic but the number has now risen to about 38 countries.

South East Asia and India are prominent dengue sites. Classical dengue is seen in most parts of India except in the Himalayan and other mountainous regions, as high-altitude conditions are not conducive for the propagation of vectors. In the early 1950s the disease was prevalent only along both coasts. But it began spreading rapidly inland within the next few years. It is known as 'breakbone fever' owing to severe muscle and joint pains. It constitutes three major forms—dengue fever (DF), dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS). Of these, DF records more number of cases but has a low mortality rate, while DHF and DSS are fatal.



The disease has been prevalent in India for over a century. Madras (now Chennai) reported the first case in 1780 and in 1872 another case was reported in Calcutta (Kolkata).

Mosquitoes usually acquire the virus through the blood of an infected person. Humans bitten by mosquitoes acquire the infection and their bodies show symptoms of the disease. By and large, humans are the virus' amplifying host, though studies have shown that in some parts of the world monkeys may become infected and serve as a source of the virus for uninfected mosquitoes.

The DEN virus belongs to the class Flavivirus that comprises mainly four strains of the virus - DEN 1, DEN 2, DEN 3 and DEN 4. having distinct characteristics. Dengue fever (DF), the most common form of dengue, is associated with a mild febrile illness lasting about 2-7 days, and is rarely associated with minor haemorrhagic manifestations along with anorexia, constipation, and pains in the joints.

DHF is a complex manifestation characterised by high fever and haemorrhagic phenomena, often with enlargement of the liver. In severe cases it could cause circulatory failure. Due to the failure of the circulatory system and loss of blood from the blood vessels, blood pressure drops leading to clinical shock (DSS). This shock is responsible for the majority of DHF deaths.

Temperatures between 20–28° C are ideal for the breeding of dengue mosquito. At low temperatures, about 16° C, their organs function less and their circulatory system fails, eventually leading to death. Neither can they survive high temperatures for long. About 60-80 per cent of humidity is apt for them.

The larva of the dengue mosquito requires water for its survival and spread. Therefore water is an essential factor.

Uncontrolled urbanisation and the migration of massive number of people from small towns and villages to big cities or from an affected area to an unaffected area is a major factor in the emergence and resurgence of diseases like dengue. Poor populations live in extremely degraded environments, enduring a lack of basic facilities, overcrowding, inadequate sanitation and sewage. The general decline of health services and their inaccessibility contributes to the spread of the disease. The widespread use of fertilisers, pesticides and weed-killers also alter the environment and create health hazards. All these conditions are favourable for the vectors, which thrive in intimate association with human filth and crowds. (See box: Scarcity of water Vs dengue)

Environmental contributions

In terms of the larger ecological cycle, pollutants that are released into the atmosphere due to rapid urbanisation, industrialisation and exploration of natural resources compromise the immunity of affected populations, making them susceptible to infectious diseases. The release of pollutants causes deterioration in air quality and leads to an increase in temperature. Higher temperatures cause greater snow melt, which leads to a rise in water level, and may result in floods. Floodwater and any water body, large or small, of stagnant water, is an ideal breeding ground for mosquitoes.



Cutting down of trees has a major impact on mosquitoes. Like other mosquitoes they start feeding on humans. An interesting factor is the packing of most of the consumer goods in non-biodegradable plastic, an ideal place for larvae to breed.

Major outbreaks of dengue fever have been observed worldwide in large urban areas of Southeast Asia. Alongwith this, other risk areas are Africa, the Americas and Mediterranean regions of Europe. In India there are many places where major outbreaks have been observed in the past and are still observed. (See Table: Major outbreaks in India)

The exact origins of the disease are not clear. It is argued that it originated in Africa and spread worldwide with the slave trade. Another theory says that dengue originated in the Malay Peninsula of Indochina. Regardless, in the wild the virus is passed between primates living in the jungle canopy and mosquitoes that feed on them.} It is believed that the virus was transferred to man by the bite of the tiger-striped mosquito (*Aedes albopictus*) since humans began clearing jungles for their needs and building settlements. The most common vector is now the mosquito *Aedes aegypti*, closely related to *Aedes albopictus*. It is African in origin but has spread throughout the tropics in the Old and New Worlds.

Major outbreaks in India

PLACE	YEAR
Kolkatta	1963
Vishakhapatnam	1964
Asansol	1967
Kanpur	1968
Vellore	1968
Ajmer	1969
Kanpur	1969
Delhi	1970
Jalore	1985
Delhi	1988
Vellore	1990

(Source: Kabra S.K, Verma IC, Jain Y, Kalra.V Dengue Haemorrhagic fever in children in Delhi, *Bulletin World Health Organisation*, 1992 Pg.105-108)

Reported deaths due to dengue in India, 1996-1999

STATE	1996	1997	1998	1999	2000
Delhi	423	1	5	2	0
Haryana	54	0	0	0	1
Punjab	32	3	0	1	-
Karnataka	5	4	5	0	-
Maharashtra	5	5	5	12	-
Tamil Nadu	16	21	5	2	1
Uttar Pradesh	10	1	0	0	-



Gujarat	0	1	0	0	-
Rajasthan	0	1	0	0	-
Orissa	0	0	0	0	-

Source: National Anti Malaria Programme (NAMP), April 2000

The most affected population are the rural population but the disease is slowly spreading its roots even in the urban and peri-urban settlements. Young children are more at risk as they delve into areas that are usually untouched, where the mosquitoes prefer to breed. Their sensitive immune system and susceptibility to diseases keeps them constantly in the vicious cycle of diseases.

Control strategy for dengue

The control of dengue need not revised as far as the strategy is concerned. The common chemical methods such as spraying with insecticides and fogging have become an integral part of any control technique. Proper treatment once the disease has set in is in any case a must. DHF fatality rates can be reduced to less than 1 per cent.

Community based approach directed towards species sanitation and drawing from the experience of countries where dengue has been effectively controlled is a sensible practice to begin with. Efficient case management of the disease and through a double-pronged attack on mosquito vectors - insecticide spraying by government agencies and elimination of mosquito breeding by family and community action to reduce larval breeding sources.

The most effective means of dengue vector control is environmental management, through physical transformation of largely man-made vector habitats, within and around human dwellings, community participation in planning, execution and evaluation of these control measures is the critical factor. It is also essential to educate people about the means of transmission and preventive measures. A common-sense approach is needed. Aerial spray is often used, as in Delhi now, where extensive areas must be treated in a short period, but spectacular results may not be expected as the *Aedes* mosquito rests indoors. Spraying external areas with insecticides to kill adult mosquitoes is a much-used measure everywhere, but its effectiveness is in doubt. In fact, this approach must be complemented by larvicidal application in and around homes where containers cannot be emptied or covered. The most effective way to control *Aedes* mosquito is larvicidal source reduction by eliminating water holding in containers that serve as larval habitats. The 1981, the *Aedes* eradication campaign in Cuba, which was spectacularly successful, depended upon reduction of larval habitats in artificial and natural containers of water, intense public education, and biological and chemical control methods.

The vector is chiefly peridomestic in its breeding habits, and has a very limited flight range. Therefore, people’s participation is extremely important in any control effort. Without community input, municipal and government authorities can achieve only minimal success.



The two key measures for eliminating man-mosquito contact are the elimination of mosquito breeding and the prevention of mosquito bites.

This can be done by carefully eliminating breeding sites, coupled with larvicidal measures. Since this mosquito breeds chiefly in the artificial collection of clear water/rain water, in and around houses, the following measures are recommended:

- To have tight covers on water storage containers, overhead tanks etc.
- Emptying of coolers and flower-vases etc. at least once a week.
- Covering and sealing of septic tanks and soak-away pits.
- Removal of rubbish and garbage. It was observed that any garbage (even ordinary plastic carrier bags besides broken tins, utensils, crockery etc.) can collect rain water and facilitate mosquito breeding. The garbage may be collected by the municipal corporation, or burnt.

There is no *aedes* control programme in India. During epidemics, manpower is drawn from malaria and **filarial control programmes**. These personnel have neither expertise or equipment to control the disease. Malaria is endemic in Delhi, and the vector *Anopheles stephensi* and *Aedes aegypti* largely share similar breeding habitats. DHF outbreaks coincide with *P. falciparum* prevalence, making diagnosis a problem. The Dip stick and ICT(full form) test specific for *P. falciparum* would be desirable, particularly when laboratory services are inadequate and differential diagnosis for malaria and dengue symptoms is critical. As in the case with malaria, in dealing with DHF preventive vector control is the key to success. A comprehensive control strategy for control of urban malaria and dengue fever needs to be developed, since control of one infection will provide control of other. The control of *Aedes aegypti* by chemical methods like spraying and fogging or ultra violet rays application is not productive and sustainable, except during epidemic control. Among the molecular approaches *Aedes aegypti* refractory to the dengue virus has been produced in the laboratory, and a lot of ecological work has to follow this line of research before any promising outcome can be predicted.

Regrettably, there is a crisis approach to public health in India – intervention is largely launched from crisis to crisis. There are no sustained programmes for the prevention of epidemic transmission. Surveillance systems are inadequate, it will probably be another five years before an effective dengue vaccine will be available for public use. In response to the recommendation of TAC report on plague, a National Apical Advisory Committee for disease control through surveillance and response has been established by the government. This Committee named dengue as a priority.

5B.4: Plague: An overview

A disease that claimed more than 12.5 million people in India in the early part of the 20th century resurfaced in the country after a gap of 28 years in 1994 in Beed (Maharashtra) and Surat (Gujarat).^{i ii} It became the most reprehensible episode in modern India. Specially when one of its busiest industrial town, Surat and other industrial cities like Ahmedabad, Mumbai and even Delhi were gripped by a state of panic. The final official tally for the 1994 epidemic stood at 876 cases of presumed plague of which 596 were in



Maharashtra, 151 in Gujarat, 68 in Delhi, 50 in Karnataka, 10 in Uttar Pradesh and 1 in Madhya Pradesh.ⁱⁱⁱ The last reported plague epidemic prior to Surat in India was in 1966.

Plague is caused by a bacterium, *Yersinia pestis*, which is transmitted by a rat-flea bite to humans. Rat fleas use rats and other rodents to propagate in the wild and domestic environment. Plague epidemics are widely prevalent in Africa, Asia, or South America. In the United States during the 1980s plague cases averaged about 18 per year.

There are three types of plagues. *Bubonic plague* is seen after 2-6 days after the bite of a rat flea the patient gets a sudden onset of fever, chills and headache. A swelling (or bubo) develops in the lymph node around the groin or neck region within 24 hrs. *Pneumonic plague* is the most feared complications of bubonic plague. It is a contagious infection, which reaches the lungs as the bacteria are transmitted from the bubo to the lungs through blood, which spreads through cough droplets in the air. *Septicaemic plague* is caused by the rapid multiplication of the plague bacteria in blood causing fever and death.

The first recorded outbreak of plague in India was in 1612 in Agra and India was the country to suffer most from the Hong Kong epidemic, which originated in lower China in 1855. It took 43 years to reach the port of Bombay in the year 1896 when more than 10 million people died.^{iv} In 1907 the British government's second Indian Plague Commission proved that rat fleas were responsible for spread of the disease. This discovery was followed by widespread use of DDT and other insecticides and the incidence of plague saw a rapid decline. The last epidemic was in Pune and last case in India was in Kolar in Mysore in 1976. ^v Bombay city recorded the last case in 1952. India was considered free of plague in 1967 as there was no laboratory proven case after 1966.^{vi} Thereafter plague outbreaks continued up to 1968 with final disappearance in 1969.^{vii} In 1950 alone plague took a toll of 18,813 lives.^{viii} (See table) Plague resurfaced in 1994, soon assuming the size of an epidemic.

Ecology of plague

The tri-junction of Maharashtra, Karnataka and Tamil Nadu, foothills of Himalayas, watersheds of Vindhyas and Maikal mountain ranges are endemic zones of plague in India but the disease extends into areas like West Bengal in the east and Kerala in the south.

The network of irrigation canals in Rajasthan like the Indira Gandhi Canal has changed the desert ecology. Wild rodent population interact with field and domestic rats, which has increased the possibility of transmission of the plague bacteria. Extensive irrigation, large agricultural fields, massive granaries, and grain markets, as those in States like Punjab and Uttar Pradesh, all create a conducive environment for rats and mice. The house mouse that comprises 18% of the total rodent population now frequents sugarcane fields, where it is possibly interacts with wild rats. ^{ix}

In cities, slums and shantytowns offer the ideal habitat for rodents. Sanitary conditions not only in major metros but also in small towns and suburbs of the country present a grim scenario. The bandicoot (*Bandicota bengalensis*) has now invaded the most populated metropolitan towns like Calcutta and Mumbai, finding the environment



conducive for its survival. Bandicoots are prolific reservoirs of rat fleas that carry the plague bacilli. Garbage and poor sanitation directly influences bubonic plague but acts only as a secondary influence on the pneumonic form.^x Man built structures like air fields and embankments of railway tracks provide rodents a place to burrow and breed while the adjoining agricultural lands provide unabated supply of food.^{xi xii}

Deforestation helps rodent population to grow.^{xiii} Rodents show tremendous resilience in adapting to adverse conditions. A study conducted by Ishwar Prakash and his colleagues in 1993-94 in the Abu hills of the Aravalli ranges, found there was more species richness and abundance in the rocky habitats with sparse vegetation as compared to hills with dense vegetation. In denuded hilly grasslands of Dungarpur in Madhya Pradesh also revealed that the rodent population is rich with many species and relatively high than they had been earlier when the region was covered by dense forests.

The onset of the flowering season of bamboo in northeast India causes a rapid explosion of rodents in the area. Mizoram experienced an upsurge of *Rattus* species, which coincided with the bamboo flowering in that region.^{xiv} Probably the continuous supply of bamboo seeds serve as food for the rodents trigger the rapid reproduction.^{xv} Laboratory tests conducted by RN Saxena, professor at the Department of Zoology, Delhi University show that bamboo seeds make rat population multiply faster when they feed on bamboo seeds, as they are rich with steroids.^{xvi}

Natural calamities favour the growth of rodent populations as ecological space is created. In Andhra Pradesh, cyclones caused a four-fold increase in the rodent population.^{xvii} The dislodge crops provide nutrition to rodents, who virtually have no competition for food and space.^{xviii} The Latur earthquake in August 1993 and outbreak of plague in Maharashtra are related.^{xix} After the earthquake, abandoned houses provide enough food and space for the domestic rodent population, which multiplied and spread to other areas. When new shelters or resettlement were made for the victims of the earthquake, they encroached on new areas, which possibly brought them in contact the wild rodent population, increasing the chances of transmission of the plague bacterium to man through domestic rats.^{xx} Floods and drought too favour rodent ecology.

Floodwaters logging in fields and other areas compel the residing wild rodent population to seek refuge in upper reaches, often near human habitation. This increases the interaction of wild rodents with domestic rats facilitating flea transfer.^{xxi} Once the floodwaters recede there is plenty of decaying food and space created by other animals to invade. During the floods, groups of rodents take shelter and huddle together in dry places. This effectively transfers fleas to those who are not infected. Heavy rains in Surat in August-September 1994 were possibly responsible for the plague outbreak in Surat in September, the same year.^{xxii} (See box on Surat epidemic below).

What happened in Surat

On September 19, 1994, a patient was rushed to the New Civil Hospital in Surat with very high fever, soon two more followed, symptoms were similar. They were treated for malaria and released. By September 20, 1994, two of these patients were dead. The next day the third patient was put through X-rays and lung congestion and pneumonia was diagnosed, as more patients poured in and reports of similar deaths came from other hospitals. He however failed to respond to treatment for malaria and pneumonia. This was when talk about a possibility of plague started gathering ground.^{xxiii}



A week later, a third of the 2.2 million population had fled the city. The Surat epidemic reported 68 death cases of which 48 were young men. Though the disease assumed full blown proportions in Surat there was a small prelude to the affair in village Mamla of the Beed district of Maharashtra. This suspected bubonic plague outbreak occurred in August 1994.^{xxiv} By August 5, 1994 flea nuisance was reported which ensured transfer of pathogen from infected wild rodents to house rats. In mid-August the warning sign of ratfall (domestic rats falling from rafters onto the floors of dwellings and dying there) was noted in Mamla village (John, T. J. (1994). Learning from plague in India. *Lancet*, 344, p. 972). Bubonic plague was quite intense by 22-27 August, 1994 in Mamla and nearby villages, with people showing antibodies to the organism.^{xxv} Reuters and AP news agencies reported "doctors in Mamla village in the southern state of Maharashtra found 35 people with bubo under their armpits and groins, symptoms of bubonic plague." By mid-September, 10% of the population of that village had bubonic plague.

Surat, lies on the western coast in Gujarat and does not figure in the endemic zones of Western India. The plague outbreak in the city in 1994 compelled the authorities to have a second look at the ecology of the city and human association with rodent habitats.^{xxvi} The Centre of Social Studies found that Surat was one of the fastest growing cities in Asia and its population quadrupled between 1971 and 1991. Only 30% of the city's population, covering 12% of the land area, had sewage facilities. On an average 1 toilet was available between 150 families and only 40% of the population had access to drinking water through a pipeline.^{xxvii}

When the epidemic emerged, confusion prevailed over the identity of the epidemic. Experts argued whether it was viral pneumonia or pneumonic plague and even melioidosis.^{xxviii} The National Institute of Virology in Pune claimed that they had isolated and confirmed the organism to be *Pseudomonas pseudomallei* – a causative agent of melioidosis. The NICD and Defence Research Development Establishment (DRDE), Gwalior found that the symptoms matched that of *Yersinia pestis*, the causal organism of pneumonic plague.^{xxix} A Technical Advisory Committee (TAC) constituted by the government to investigate the reasons for the outbreak under the chairmanship of V Ramalingaswami found plague. Studies by WHO International Collaborating Centres for Plague, Pasteur Institute, Paris, Centres for Disease Control and Prevention, in the US and Plague Surveillance Centre in Russia were also being done independently.^{xxx} One group studied the plague of Surat that showed that the bacterium had evolved genetically. An extra protein band of 25 kilodalton (KD) and ribotype S (a type of DNA found in bacteria) had been added to its arsenal.^{xxxi}

The most plausible explanation for the Surat plague was the unusually heavy monsoon during the first week of September 1994, a record in the past 25 years. Floodwaters entered the city and remained stagnant for 5 days resulting in the death of a sizeable number of animals including rodents.^{xxxii} The carcasses released fleas in the floodwater, which infected domestic and other wild rats.^{xxxiii} Migrant labour force from central Maharashtra moved into Surat could have brought in the disease.^{xxxiv} Soon after the floods receded in Surat, the *Ganeshotsav*, the festival of mass frenzy and intermingling of people, ensured easy passage of the infection.^{xxxv} Surat after the flood



was filthier than ever before. The city administration suspecting that the floods and unsanitary conditions in the city was playing havoc, decided to go for a cleanliness drive. The city's garbage weighed to about 5000 tonnes while the removal capacity of the municipal corporation is about 500 tonnes per day. Surat generously sprayed gamaxene, DDT and malathion on mounds of garbage. In fact all guidelines formulated after the DDT overspraying spree of the 60s and 70s were ignored. Rats started to die even at homes in several colonies of the city, complicating the problem further.^{xxxvi} The epidemic in Surat was of the very infectious pneumonic plague. According to Dinesh Shah, Medical Superintendent at the Surat Civil Hospital, susceptible population, the squalor and the emergence of the disease were in right proportions for the disease to erupt into epidemic proportions. He credits the municipal clearing operations for averting from becoming a pneumonic one.^{xxxvii}

In Surat, pneumonic cases appeared suddenly with very high initial mortality but surprisingly without bubonic cases. The only largely known epidemic of pneumonic plague in the 20th century had occurred in Manchuria in China and Mongolia. Other pneumonic plague outbreaks reported in India earlier had occurred as a part of bubonic epidemics and pneumonic cases constituted a proportion of only 2-13% of all cases. Primary pneumonic cases are extremely rare and have usually been reported in people handling infected animals. Young adults, most of them employed as diamond cutters, were the ones who first encountered the infected animals. According to NP Gupta, a former director of the National Institute of Virology, Pune, a unusual feature of the Surat epidemic was that the pneumonic outbreaks are characteristic of colder climates and Surat usually maintains a temperature around 35°C which puts a question on the mode of origin of the disease. Contrary to experience elsewhere in the world the alleged outbreak of pneumonic plague was not preceded by an outbreak of bubonic plague. Bombay in fact escaped without a single case inspite of an influx of 50,000 odd panic stricken migrants from Surat.^{xxxviii} Guneael Rodier, an epidemiologist with WHO says, "We are 100% sure of the involvement of rats in Beed but Surat is a big question mark."^{xxxix}

On September 23, Maharashtra state authorities (five weeks after first bubonic plague cases were diagnosed in that area) summoned about 100 expert snaking-catching Irula tribal from Tamil Nadu to the plague affected areas in Maharashtra to catch rodents. In New Delhi, large numbers of extra city workers and garbage collectors were hired to spray inside houses in poor neighbourhoods, and trap rats. Large numbers of garbage collectors were hired there, as well. Citizens asked why officials had allowed the conditions to worsen to such a degree as to allow plague to break out. Ecologists had raised an alarm over the spreading of poison on the farmlands to kill the rats. Extra bounties were paid to rat killers to prowl and hunt in the back streets at night. The spraying of insecticide would continue. This killing of first the fleas, then the rats was appropriate (killing rats first leaves many hungry fleas looking for a meal, and in the absence of their preferred rats, they will bite humans.) However, the next generation of rats may be infected by underground fleas, and start a new epizootic.

Control strategy for plague



Plague as a disease needs greater surveillance than monitoring. Because of its sporadic occurrence and rapid spread, plague outbreaks can be predicted by studying rodent populations and behaviour. Of greater importance is the watchfulness required after flood, earthquakes, unprecedented rains and heavy snow fall. The recent Himachal plague outbreak signifies that regions previously considered free of the diseases are increasingly coming under the epidemic range (see box below).

Rattled: The Himachal Pradesh episode

On 19 February, 2002, India's Health Minister CP Thakur confirmed that four people in the northern state of Himachal Pradesh have died of pneumonic plague. The sequence of tests collectively confirmed that the cases related to infection with *Yersinia Pestis*, commonly known as pneumonic plague.

The remote village of Chopalkot hamlet in Rohru village of Shimla district, from where the four victims hailed, was sealed. Subsequently another 12 people are undergoing treatment at a government hospital in Chandigarh city of which three succumbed to the disease. Although no fresh cases were reported over the next few weeks, panic has been reported in and around the affected villages and precautions were taken in the neighbouring state of Uttaranchal, where a woman contracted the disease from a relative in Himachal Pradesh.

If nothing else, the incidence of plague in Himachal Pradesh served to focus attention on the entire gamut of disease management in the country. At the end of it, at least one message was clear — plague is really no big deal. Given a good surveillance system and prompt medical attention, there is no reason why even a highly contagious disease cannot be contained. But then, are we really equipped for it or was this time round just a lucky one.

As India's WHO representative Robert Kim Farley said at the official confirmation of the Himachal plague, there is no reason why the disease should make headlines or set in panic. Unfortunately, the mere mention of its name evokes medieval images of horror and death accompanying an invasion of dirty sewer and rats. In fact, though potentially fatal, plague is completely curable if diagnosed early. Yet, in our country, panic buttons are immediately triggered, and reactions take on a fevered pitch. This is where awareness and education would help. After all, diseases like plague are completely curable. Provided they are tackled on time. In the us, for instance, improved sanitation and surveillance technology have limited the disease to areas with wild rodent population.

Yet again a firefighting strategy was adopted in Himachal and subsequently there is absence of any long term strategy prevent future outbreaks. The health minister was quick to take the credit for successfully "containing" the outbreak in Himachal, the extremely cold weather and snow are the lucky factors. But we may not be so lucky the next time around.



The response of authorities to the Surat and Himachal epidemics can be taken as an index to judge their overall attitude to the combat the disease. The first response to the outbreak of plague in Surat was scepticism, even flat denial on behalf of the state government that it was faced with a very serious challenge.^{xi} This was solely responsible for the delay in taking stock of the situation and acting accordingly. Meanwhile thousands of people fled the city that included government officials also. That goes to show how the much vaunted sense of bureaucratic responsibility vanishes instantly.^{xii} The epidemic, first in 28 years focussed the spotlight on India's disease surveillance system. State authorities ignored what can be referred as the first signs of an impending epidemic; a rise in rodent population.^{xiii} What the authorities possibly failed to notice was that though human plague was last recorded in 1966, animal focii of plague continued to exist in the wild and could easily be transmitted to humans.^{xiii} In periodic surveys over the past three years before the epidemic India's Plague Surveillance Unit in Bangalore reported a gradual rise in the number pf rats found to host the plague bacterium. A meeting with the National Plague Coordination Group in 1993 urged states with a high rodent population to strengthen their systems to control plague. Maharashtra in fact had already dismantled its plague control unit in 1987. ^{xiv}A surprising fact is that the only plague clinic in Calcutta downed its shutters in 1992 and when the epidemic erupted in Surat there was no plague monitoring unit in the city though a small team was supposed to monitor the port areas for rodent population. With health care system being the worst among the four metropolises, in the event of a plague outbreak, people in Calcutta will not even find enough hospital beds.^{xv} Public health system is at an equally bad shape in Surat. Over one third of Surat's population does not have access to basic health facilities and there were only two hospitals established about 35 years ago that were treating plague patients in 1994, though ill equipped to do so.^{xvi} (see box below)

The first response from Surat

The first response to the outbreak of plague in Surat was scepticism, even denial by the state government that it ever faced a serious challenge.^{xvii} Thousands of people fled the city that included government officials also. Bureaucratic responsibility and government machinery had failed to control the panic.^{xviii} The epidemic, first in 28 years focussed the spotlight on India's disease surveillance system. State authorities ignored what can be referred as the first signs of an impending epidemic; a rise in rodent population.^{xix} Surveillance methods did not recognise that though human plague was last recorded in 1966, animal focii of plague continued to exist in the wild and could easily be transmitted to humans.ⁱ In periodic surveys over the past three years before the epidemic India's Plague Surveillance Unit in Bangalore reported a gradual rise in the number pf rats found to host the plague bacterium. A meeting with the National Plague Coordination Group in 1993 urged states with a high rodent population to strengthen their systems to control plague. Maharashtra in fact had already dismantled its plague control unit in 1987. ⁱⁱThe only plague clinic in Calcutta had closed down in 1992. ⁱⁱⁱ T. Jacob John (Lancet, 1994, p. 972) found that the key problem in India's response to the plague epidemic was a "lack of epidemiological alertness, skill and interventions." Eswar Krishnan (Lancet, 1994, p. 1298) observed that it was the failure of administration to support scientific staff."

The Surat episode was not only a story of personal losses; it meant a huge loss to the economy. In financial terms, however, the plague's toll was much greater, costing the



Indian economy in excess of \$600 million. More than 45,000 people cancelled their travel plans to India, and the country's hotel occupancy rate dipped to 20 to 60 percent. Many countries stopped air and ship traffic to India altogether. In total, exports from the country suffered a \$420 million loss (Kai Friese, "The Morning After," *India Today* (October 31, 1994), pp. 30-39.). Even before a single case had been confirmed in Calcutta, air links between the city and Bangladesh, Thailand, Gulf countries and Pakistan had been severed; border crossings had been sealed for Indian citizens. International trade was disrupted and the Indian tourism industry was set to suffer a storm of cancellations. The Bombay Stock Exchange share index nose-dived as the epidemic progressed. ⁱⁱⁱ

The situation could have been tackled since India has the capability in terms of scientists and laboratories. "The whole thing should have been accepted as a national issue, mobilising all laboratory facilities and scientific capability. But this was not done. It was entrusted to a single laboratory which had its own limitations, it carried out some physiological examinations but could not determine the causal organism." Says Prof. V Ramalingaswami, then National research professor and chairperson of the committee formed to investigate the Surat episode. ^{iv} "We are facing a number of uncertainties for the future. But underlying all this is the resource crunch that is getting worse now. The health infrastructure was never something to be proud of in our country," he adds further. In view of the challenges in a contradictable future there is an absolute need to increase resource allocation. It is also imperative that we utilise the resources in a cost-effective manner. There is a lot of wastage in this sector. Lot more can be accomplished even with the existing resources. ^v

5B.5 Filariasis

More than 1.1 billion people in 76 countries or 20 % of the world's population live in areas where they are at risk of infection from lymphatic filarial parasites. In south Asian region, *Wuchereria bancrofti* causes 90% of infections; while *Brugia malayi* causes the rest.

Filariasis is a major public health problem next only to malaria. As per recent estimates, about 454 million people are at risk of being affected, with 29.2 million carriers and 22.5 million clinical annual cases are spread in 13 States and 5 Union Territories. India contributes about 74% of endemic population and 81% of the disease burden in the south-east Asian region. There has been increasing trend of the infection during the last three decades. *W.bancrofti* is the most predominant infection comprising 99.4% of the problem in the country while *B.malayi* is confined to the western coast of Kerala and a few pockets in six other States.

Clinical filariasis is widespread in the developing countries of Africa, Asia and South America but are of different types. In Africa, another species causes the debilitating onchocerciasis (river blindness), which affects approximately 18 million people, and blinds about 300,000. Other known filarial parasites of humans are *Loa loa*, *Acanthocheilonema ozzardi*, *A.perstans* and *A.streptocerca*. The magnitude of infection in children has become much better understood in recent years; indeed, most infections appear to be acquired in childhood, with a long period of subclinical disease that progresses to the characteristic overt clinical manifestations of adults. The re-evaluation of the previous underestimations of lymphatic filariasis in children will certainly play a



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role in redefining the global burden of the disease. (WHO, WEEKLY EPIDEMIOLOGICAL RECORD, NO. 20, 18 MAY 2001)

Filarial infection and in particular lymphatic filariasis has a wide spectrum of clinical disease that affects individuals of endemic regions. The manifestations are diverse within individuals having no clinical manifestations.

Although lymphatic filariasis causes death very infrequently, it is a major cause of clinical suffering, disability and handicap.

WHO has estimated the burden of the disease as 4 918 000 disability-adjusted life years (DALYs) -- the highest of all tropical diseases after malaria. A study quoted in WHO's Mortality and Morbidity Weekly suggests that in India alone economic losses due to lost man-days of work and decreased productivity approach US \$1 billion annually.

Lymphatic filariasis is unevenly distributed in the Indian subcontinent, but it is largely more common in rural areas. Due to vastness of some of the states and poor accessibility of many areas, coverage of filariasis surveys have been inadequate hence the actual number of cases have never been estimated correctly.

A study in Pondicherry compared the trend of prevalence and spectrum of manifestations of Bancroftian filariasis disease for 1957, 1986 and 1992, showed the overall prevalence of filarial disease had increased from as 4.7 to 6.7 to 9.9% respectively, showing that it was still a significant health problem. In other areas too, the infection is fast spreading to new areas (ICMR - -)

Ecology of the disease

The thread-like, parasitic filarial worms that cause lymphatic filariasis live almost exclusively in humans. These worms lodge in the lymphatic system, the network of nodes and vessels that maintain the delicate fluid balance in the tissues and blood, and an essential component of the body's immune defense system. They live for years, producing millions of immature microfilariae that circulate in the blood; these are picked up by mosquitoes that then transmit the infection to others. Adult filarial worms are vector borne, obligate extracellular parasites of their definitive hosts. In both cases microfilariae (a small, almost microscopic worms) are ingested by blood-sucking vectors in which they moult twice to become infective larvae, these are transmitted to the new host when the insect bites again.

The vector therefore is the only controllable factor apart from limiting the proliferation of the worm in human reservoirs. Sanitation and hygiene practices can ensure that mosquitoes do not breed close to human habitation and regular. The physical impairment affects the ability of the poor to perform basic chores and work. This adversely impacts their ability to pay for medicines and receive healthy food and safe water (see box below)

Annual cost of lymphatic filariasis in India

The World Health Report in 1995 identified lymphatic filariasis as the second leading cause of permanent and long-term disability worldwide. In fact, the true extent of illness and disability due to this infection are only beginning to be quantified. What is



clear, however, is that in addition to the direct costs of treating lymphatic filariasis, the enormous indirect losses resulting from incapacitation and loss of labour severely stress household, local and national economies. To this huge economic burden will be added the yet-unquantified effects of the newly-discovered subclinical pathology of the renal and lymphatic systems, affecting all those who have the disease. Almost half of all people with lymphatic filariasis have overt clinical disease. The remainder harbour infections with hundreds of thousands – even millions of on tiny, transparent and delicate worms in their bodies, but with the internal damage undetected and untreated.

The annual economic losses caused by lymphatic filariasis in India have been conservatively estimated at US \$1.5 billion. Contrasted to this huge amount is the relatively small cost of community-wide annual treatment programmes for filariasis, where a single dose of DEC would be administered for 4-5 consecutive years. Total programme costs for implementing this revised strategy, which began on a limited scale in 1996, averaged Rs 1 per person. If this programme is extended to include all 420 million endemic area residents, the total yearly investment, for the 4-5 yrs required for elimination, approximates Rs 420 million, or US \$12 million. This is less than 1% of the economic burden of lymphatic filariasis to the country. (Source : WHO (1997)

Lymphatic filariasis: Reasons for hope, Division of Control of Tropical Diseases, WHO, Geneva). A study from a Bancroftian filariasis endemic area in Pondicherry and lymphatic filariasis

in Tamil Nadu, South India showed that 66% patients of filariasis confirmed that their lives had been adversely affected. Thus the economic effects and productivity loss of this disfiguring disease has been greatly underestimated.

Unhygienic conditions that promote the growth of vector population is the most important reason for the prevalence of the disease in India. Migration of people has spread the disease in areas where there was little or no filaria. This has not been particularly well studied in India but lymphatic filariasis due to *W.bancrofti* and *B.malayi* did not exist in Pakistan prior to partition in 1947, except for a few isolated cases. It was however endemic in Bangladesh where infection rate in some parts is 16.8% with clinical manifestations present in 10.1%, which suggested that filariasis was a significant public health problem in Thakurgaon region, Dinajpur District, in Bangladesh. Similarly in Rajasthan and Madhya Pradesh was rarely reported (James Cowell (1904) Spread of elephant's foot in the Indian subcontinent, Madras Presidency Gazette Vol XI, no 7)

In Pakistan, during mass immigration from former East Pakistan in 1974, many immigrants from endemic areas settled in urban areas of Sind particularly Karachi. Rapidly changing political and economic conditions in the region and the continuous rural - urban drift of population to the major cities may have created a new focus of transmission. In addition, *Culex quinquefasciatus*, the ubiquitous vector for bancroftian filariasis, is abundant in Pakistan. The dangers of imported filariasis and more importantly the establishment of a self-sustained foci of disease which was likely to depend on the presence of carriers and a susceptible population of vector mosquitoes. A study of repatriated Biharis from Bangladesh, showed that in a sample of 1,101 people above one year of age, 9.0% were infected with *W.bancrofti*. The infection rate was significantly higher in males 10.2%, than in females 6.7%. Most importantly the mosquitoes, *Culex pipiens fatigans* collected near the camps were positive for infective



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larvae and transmission was observed in the hottest and driest months. Thus, favourable climatic conditions coupled with a constant source of microfilariae provided the perfect conditions for filarial transmission to occur. In a study done in a village in Punjab, these concerns were tested. It found that no one was found positive for human microfilaria parasite in this non-endemic area, thus negating any indigenous filariasis transmission in this town. These findings were compared to earlier surveys in the same area and other non-endemic areas and showed that non-endemic areas continue to be non-endemic for filariasis despite considerable increase in MF rate among migratory population and vector density (Singh S, Bora D, Sharma RC A study of filarial transmission in a non-endemic area of Pathankot (Punjab). J Commun Dis 2000 Mar 32:61-4).

Several irrigation programmes have been responsible for the spreading the infection. The Sardar Sarovar Narmada Project "Studies on Ecology and Environment" by Maharaja Sayajirao University (MSU), Vadodara in 1983 found filaria to have emerged but confined to the coastal areas of Saurashtra and South Gujarat. Filaria has also been reported in Surat, near to the reservoir site, but the study concluded that the disease was unlikely to spread to the reservoir area. (http://www.nca.nic.in/env_findings1.htm)

Control strategy for filaria

Control of the infection is largely through medical intervention rather than vector control. There are 206 National Filaria Control Units, 27 Survey Units and 199 Filaria Clinics. The country adopted a revised strategy in 1997 for elimination of LF. A National Filaria Day (NFD) is being observed with single dose mass DEC therapy once a year and morbidity control through IEC in 13 endemic districts covering about 41 million population. (source : WHO (2000) Elimination Of Lymphatic Filariasis Report Of Informal Consultative Meeting On Lymphatic Filariasis In Sea Region, Bhubaneswar, Orissa, India 23-25 February 2000, World Health Organisation - South-East Asia Regional Office Indraprastha Estate, New Delhi, India).

Even before organised control measures like application of bio-pesticide could be used against the vectors, a large population of mosquitoes have already acquired resistance to it. A paper by S. Poopathi and others shows that a low level of cross tolerance in three strains of *Bacillus thuringiensis* when used on *Culex quinquefasciatus* which was already resistant to another biopesticide, *B. sphaericus*. (S Poopathi et al (1999) Investigations on cross-resistance to *Bacillus thuringiensis* H14 in the Bancroftian Filariasis vector, *Culex quinquefasciatus* Resistant to *Bacillus sphaericus*, Journal of Parasitic Diseases, Vol. 23, December 1999, pp. 121-124)

Unhygienic conditions promote the growth of the vector, which is the most important reason for the prevalence of the disease in India. Children are particularly susceptible to the infection. Migration of people has spread the disease in areas where there was little or no filaria. Several irrigation programmes like the Sardar Sarovar Narmada Project have been responsible for the spreading the infection.

A worldwide coalition, the Global Alliance to Eliminate Lymphatic Filariasis, was created in 2000 to include many organizations, each with a different mandate but all having the common goal of tackling the wide-ranging and complex process of science and practice that will result in the elimination of lymphatic filariasis as a public health problem globally. The partnership, initially formed around the contributions of GlaxoSmithKline (including



all the albendazole required worldwide) and of Merck & Co., Inc. (ivermectin in countries where onchocerciasis is co-endemic), has since broadened to include 37 organizations to date from various sectors of society including the public and private sectors, academia, government bodies and non-governmental development organizations. Indeed, the Global Alliance to Eliminate Lymphatic Filariasis can now envisage the elimination of the disease as the focus of a widely beneficial public health intervention organized through existing or strengthened national health infrastructures.

What happened in the control programme

Only 1/10th. of the population at risk is catered to under the National Filaria Control Programme (Health information of India – pg 202-203). As per 1996 estimates there were approximately 21.23 million filaria cases, and 28.01 million micro-filaria carriers in the country. About 463.58 million people living in filaria endemic states will be exposed to the risk of filariasis by the end of the year 2000 (NAMP, 2000). Control measures include recurrent anti-larval measures, using larvicides in the mosquito breeding places, anti-parasitic measures by detection of micro-filaria carriers, and treatment with D-ethyl carba-mazine (DEC), IEC activities and management of acute and chronic filariasis through referral services. (Rajya Sabha Unstarred Question 337, dt. 29 May 1998.) In March 2002, the anti-filaria campaign in Orissa received a setback following the death of three people after administering albendazole and DEC. The two drugs are the most advanced and simple strategies to minimise drug resistance in the pathogen. The campaign organised by the WHO, the Orissa government and the Regional Medical Research Centre (RMRC), Bhubaneswar had to suspend operations and conduct tests to check for spurious drugs (The Pioneer, New Delhi Feb 1, 2002 pg8). Even many clinicians and programme professionals are disillusioned by the single approach of using therapeutic interventions while ignoring sanitation, hygiene and bio-environmental control options. Drug treatment options for lymphatic filariasis have focused almost entirely on ridding populations of micro-filaraemia, in order to interrupt transmission. While 'simple' hygiene-oriented means of treating individual patients to minimize and even reverse the progression of elephantiasis and other expressions of lymphatic pathology have been developed, with unexpectedly good results for the patient, curing the infections of individuals (by killing the adult worms) has received very little attention to date. Indeed, current recommendations on how best to treat individual patients are inadequate. (E.A. Ottesen, M.M Ismail and J. Horton The Role of Albendazole in Programmes to Eliminate Lymphatic Filariasis, Parasitology Today, vol 15 no 9., 1999 pg 385).

5B.6 Fevers of unknown origin

Unknown, unidentified and lethal fevers are striking India. Leaving a trail of deaths and misery. A system caught unawares gropes in the dark. Whether it is Siliguri or Rae Bareli, Delhi or Mumbai, the medical fraternity has neither responded to the challenge nor shown scientific temper in taming this breed of viruses and bacteria that are on the prowl. The fevers may be due to viruses or bacteria assuming a virulent character or it could be the resurgence of old diseases long forgotten. Or they could be new ones surfacing to assert their existence. Just what are they? The answer is as mysterious as the system that is supposed to demystify it. But one thing is clear: ecological changes



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are the prime catalyst for this resurgence and the scientific community is doing very little to tackle the problem.

The **mystery fever** that attacked Siliguri, a small town in West Bengal, this year is yet to be identified, but it definitely unmasked the rot in India's medical system. What began as sporadic incidences of strange fevers gradually gripped the entire town. Schools were closed. People refused to leave their homes and when they left, they used handkerchiefs as protection — even the bride and bridegroom covered their faces. The panic was unprecedented. Medical practitioners fled the town fearing the fever was contagious. The fear of the unknown had struck. But there was little respite. Over 37 people lost their lives. "What do we do? We don't even know the cause," rued a helpless Bikash Ghosh, the mayor of Siliguri.

Meanwhile, the soothsayers of the medical fraternity were having a field day. All sorts of causes made it to the front pages of newspapers. From Japanese encephalitis, cerebral malaria and plague to mutating measles and what not! The panic also triggered a political turmoil in the state. The opposition Trinamool Congress in the state demanded the resignation of the state health minister even as the death count continued to rise. Inside parliament, the country's medical system virtually crumbled as a hesitant health minister admitted the government's inability to identify the fever. Even after eight months, the government claims to be close to identifying the fever. But nagging questions on the credibility of the identification process and the system are just coming to the fore.

The Centres for Disease Control (CDC), Atlanta, USA, was called in. It analysed samples and a detailed report was sent in early September. However, the Union government has not made the report public. W J Bellani of CDC, in a letter to Robert Kim-Farley of the World Health Organisation (WHO), New Delhi, has narrowed the causal pathogen to be either Nipah or Hendra-like virus. D Neogi, head of the virology department, Calcutta School of Tropical Medicine corroborates this fact. The Nipah virus epidemic took place in Malaysia in 1999, where pigs were reservoirs. The Hendra occurs in Australia in horses and people. Siliguri and its neighbouring areas have a large population of pigs. But how the virus surfaced here is not known.

The lack of coordination among the various government departments is apparent. Neogi, who was a member of state fact-finding team visiting Siliguri during the crisis, claims that he had suspected the pathogen to be Nipah right from the onset. He apparently had told the director of the National Institute of Communicable Diseases (NICD), New Delhi. But NICD experts negated his view, instead propagating the theory of mutating measles.

The turn of events from early February in Siliguri clearly reveal the level of under preparedness and the lack of expertise and coordination within all the stakeholders — be it the government or scientific fraternity.

The Siliguri incident is just the tip of the iceberg in more ways than one. It got widespread media attention and the virus close to being identified. But there are many more cases across India that go unreported and in some cases wrongly diagnosed.



Rae Bareli: mystery

Rae Bareli in Uttar Pradesh has a tryst with strange monsoon fevers. Doctors say every year there is a sudden rise in the number of fever cases. This year it claimed 18 children. Though the district administration put the body count at two, the district hospital's records show seven children died of 'suspected brain fever' between mid-August and first week of September. In this period, more than 60 fever cases were registered in the Rana Beni Madhav Singh hospital, the district hospital of Rae Bareli.

But hospital officials claim the issue has been blown out of proportion. "Only a few cases went undiagnosed, the rest were natural deaths," says R N Bharadwaj, chief medical superintendent of the hospital. Officials say that the patients complaining of fevers died within half-an-hour to one hour of admission. To ascertain the cause is, therefore, impossible. "Twenty-six blood serum samples were sent to Sanjay Gandhi Post-Graduate Institute of Medical Sciences (SGPGI), Lucknow, and four were sent to NICD," says R S Pandey, deputy chief medical officer (Epidemics), Rae Bareli. The results are intriguing.

SGPGI says that 14 out of 26 cases tested positive for Japanese encephalitis. "Interestingly, all 14 persons who tested positive are alive, though a 30-40 per cent death ratio is generally recorded in such cases," says Pandey. Then what was the cause? "It is difficult to say. We tried to repeat the tests, but could not coordinate with the patients," adds Pandey. Another way could have been to analyse post-mortem results. But they were not carried out. So till the time a detailed study is carried out, Rae Bareli's cup of woes will keep overflowing.

Meerut: 'Japani viral'

Situated in the rural backyard of the industrial city of Meerut, Khiwai witnessed a major outbreak of a mysterious fever with 'Japani viral' (possibly a local name for Japanese encephalitis) in 1997. According to Rohshanlal, chief medical officer of Meerut, a large number of children, between the ages of 2-8 years, mostly boys, suffered from continuous fever and coughing, vomiting and shivering. This viral infection is suspected to be waterborne. "There is no protocol, except for the use of blood slide to diagnose malaria. Hence, mysterious fevers remain undiagnosed. NICD too has not submitted its report on the incident," says Rohshanlal. Shabbir Ali's four-year-old son had vomiting, behavioural changes and loss of appetite and sight. He succumbed to the fever 14 days later. Fourteen children died in all. S K Singh, medical officer-in-charge for Khiwai village says the village is densely populated and there is little awareness about health and hygiene, and their only source of water is a stagnant pond that is poorly managed. However, the cause of the 1997 outbreak is not understood according to local medical officers and the NICD.

Wadrafnagar

A nondescript and remote block of Sarguja district, Wadrafnagar in Chattisgarh, made headlines in July this year for all the wrong reasons. A strange outbreak affected 66 villages and claimed more than 100 lives within a span of two months. Local people allege that this is a conservative estimate and the actual figure could be more than 300.



According to statistics of the district health officer, M H Parmar, a total of 14,333 cases were reported by the end of August, out of which 1,288 cases were due to gastroenteritis. Deaths were reported from 24 villages. Out of the total of 115 deaths that took place, 46 were due to gastroenteritis. "Most of the other cases were categorised as viral fevers of 'different nature'," says Parmar. He, however, admits that no one is quite sure of the type. NICD director K K Datta says that it was an attack of cholera and the local officials had been informed. Local officials, however, say that they have not heard anything from NICD.

Asked if there is any protocol for the sampling or identification of the virus, Parmar said: "We neither have the facilities nor am I aware of any such protocol. We are expected to take control measures to check the epidemic and to cure the cases. Given the extremely difficult conditions, our doctors are doing remarkable work."

Delhi's frequent fevers

"It is now a trend. There is nothing to be alarmed," says J N Pande, head of medicine, All India Institute of Medical Sciences, (AIIMS). "Every day our out patients department receives a sizeable number of patients who suffer from frequent fevers." Delhi is particularly notorious for viral fevers — since 1986 three major peaks of viral fevers were seen during the change of season from winter to summer to monsoon and again to winter. These fevers have no distinct morbidity patterns but have a differing pattern of virulence. Children and adults, women and men suffer from varying intensity of the symptoms. According to most physicians at any given time, 8-12 per cent of Delhi's population is suffering from viral or unknown fevers, and peaks of viral fevers are spreading out evenly to become a regular feature of the city's woes.

So far, no one in the city of Delhi has succumbed to any of these widely prevalent viral fevers claim doctors. Doctors too admit an increased prevalence and incidence of the fevers and some also perceive that the fevers are gaining virulence. But no one knows what are the types of fevers and what causes them.

Mumbai's leptospirosis

The city that never sleeps boasts of some of the oldest medical institutions in the country. The local people claim that they have the best infrastructure and municipal services. But all this confidence withers away each time an epidemic strikes. Every monsoon, people residing in the slums brace themselves for waterborne fevers and other mystery diseases. One such disease is Leptospirosis. Public health experts complain that patients register first in private hospitals that have no clue in diagnosing and treating patients. The disease is often confused with other diseases like jaundice and malaria. Hence the first few affected are the early victims of the disease. Municipal authorities and government hospitals remain secretive about the emergence and hope that the disease is actually a stray incident rather than an epidemic.

This year like any other year, Mumbai was greeted with the disease. Leptospirosis is a febrile illness caused by infection due to *Leptospira interrogans* — which results from contact with the urine of infected animals, mainly rodents. The disease has struck earlier



— Patna (1985), Chennai (1988), Andamans (1993), Mysore (1997), Surat (1994), Mumbai (1999, 2000 and 2001), Kerala (since 1993) and all along the Malabar belt.

Gurgaon's tryst with fevers

Between April and May 1998, the suburban town of Delhi experienced a “mystery illness” that claimed the lives of more than 15 children. Tests conducted by NICD failed to establish the cause. As in many cases in the past (see box: *The X-files*). Children below the age of four complained of vomiting, loose motions and fever to certain private physicians. Broad-spectrum antibiotics and anti-pyretic drugs were administered. But these led to complications: the children could not pass urine and later developed renal failure. They were then shifted to hospitals in Delhi, where they were put under peritoneal dialysis. A few days later they slipped into coma and died within the next few days.

A team from NICD collected samples, but their analysis has established very little. Doctors and medical experts are at loggerheads about the cause of this illness. Some say that it was due to indiscriminate and inappropriate use of drugs. But NICD has still a lot to answer.

Ecology of the disease

Mystery fevers are an indicator of the evolutionary success of microorganisms. Pathogens that cause unknown fevers are usually viruses, and exceptionally bacteria or other microbes. They remain undetected because they have an extremely short and rapid life cycle and it is difficult to isolate, culture and grow these viruses in laboratories. Viruses are lifeless organisation of a genetic material (RNA OR DNA, never both) and a protein coat, and show signs of life only when they enter a host. They can develop extreme virulence from being benign organisms in a matter of a few (microbial) generations. Microorganisms, especially viruses, can mutate their heritable genetic material more quickly because they are relatively small in size. They can add genetic material from the host's genome and become extremely diverse in matter of a single generation. Their success as a group relies on the high rates at which genes are shuffled by recombination, producing virtually every perceivable permutation and combination of genetic arrangement.

However, only a few of these new variants may have a virulent form. Often associations with other organisms help. The cholera bacterium is more virulent to humans when infected by a particular virus.

On an average a virus has about 4,000 bases (the simple building blocks of genes), which means that a few million permutations in their genetic arrangements are possible. Viruses either have single and double stranded DNA OR RNA; the rates of mutation vary vastly. Size of the genome and infectivity, numbers of hosts infected and number of generations produced too affect this greatly. Thankfully, not all mutations are possible — some of these mutations are lethal for the virus itself.



Most diseases have an ecological basis to their origin. Seasons and changing climate too assist the emergence of diseases. In poor states like Bihar and Orissa there is a distinct 'microbial traffic', a virtual succession of infectious microbes.

Even causalities have a pattern: due to water scarcity during summer, contaminated water leads to diarrhoea, which particularly affects children and older people. During the rainy season, malaria and fevers of unknown origins emerge, all age groups are affected but the effect on young women and pregnant women is more. If a natural calamity like flood or cyclone arrives, then malaria recurs, and cholera and other gastro-intestinal infections follow which affect young adolescents and children more than adults. In winter the paucity of food causes widespread hunger and leads to malnutrition, makes people more susceptible to infections, thereby providing an ideal environment for the spread of diseases. There are distinct patterns of succession of diseases with changing seasons.

In cities of north India, when the winters end abruptly, a spate of viral infections descends on the cities and the villages alike. There is little climate variability in the rest of the country except the coming of the monsoon. Winters are milder and summers like the rest of the country is hot. Forest and rural areas in India have their fair share of viral diseases. Kyasanur forest disease seen in Uttara Kannara and Shimoga districts of Karnataka is a tick borne fever that causes fever and body ache.

Recently, the viral meningitis in Bellary claimed a dozen lives. Its origins and causes remain unknown. Recurrent febrile illnesses emerge sporadically in central India, especially in central Maharashtra, Madhya Pradesh, Andhra Pradesh, Tamil Nadu , Orissa, Bihar and eastern Uttar Pradesh.

Threats from ecological changes in neighbouring countries leads to migration. For example, rural families from eastern Nepal migrate to the forests and the plains of upper Assam every year. With them they bring their livestock, especially buffaloes, each infected by a benign virus called the Karyovirus-II. When these families settle around the forest fringes, a chance encounter with a new vector increases the possibilities of its spread to humans. The fever causes severe haemorrhagic fever, bleeding and death.

Like the Karyovirus of Nepal, there are many fevers — Crimean Congo Haemorrhagic fever in Pakistan and the numerous diarrhoeal diseases in Bangladesh — waiting to nurture their virulent ambition in India.

What is more significant in disease ecology is not how mutated and rapidly evolving viruses and other pathogens become established in humans, but how ecological change contributes to its emergence. Causal factors — changes in land use, human activities and urbanisation — are integrated with ecological factors to cause mutation, changes in the pool of hosts and reservoirs and other elements. These in turn affect both human and animal hosts, as well as vectors. Moreover, air, water or soil pollutants or other stresses affect humans and other species in the ecosystem. Polluted habitats are an ideal environment for these pathogens. Pollution reduces human immunity. And some pollutants can promote the growth and proliferation of some pathogens.

Reasons for prevalence



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Most doctors in India do not believe that there are unknown pathogens that cause these fevers. “There is nothing like an unknown or a mystery fever, they just remain undiagnosed,” contends B K Ram, G T B Hospital, New Delhi. “Fever is a symptom that is meant to be treated with a variety of antibiotics. Only serious infections such as meningitis call for pathological tests,” says Ram. “When we fail to control a fever, we prescribe medication for malaria or tuberculosis depending on the symptoms,” says A K Singh, deputy superintendent, Patna Medical College Hospital, and claims, “this is the World Health Organisation (WHO) protocol for tropical countries.”

ARVIND LAL of Delhi’s Lal Path Labs has been in the pathological diagnostics business for the past three decades. “Prescriptions instruct what tests should be conducted. If a pathogen is absent, we do not report the presence of another. So, many diseases remain undiagnosed, though they can be easily identified,” says Lal. “Twenty per cent of the cases that we receive remained undiagnosed and are presumed to be fevers of unknown origin,” says J N Pande, head of medicine at the All India Institute of Medical Sciences, New Delhi.

Warding off the threat of an epidemic by using a broad spectrum of antibiotics may appear to be a potent option. But the risks attendant on this course of action are many. For one, it is probable that the medication will suppress the symptom without treating the condition. Another alarming possibility is that it may induce resistance in the pathogen even before it is identified. Hard-pressed for time as they are in view of the large number and heterogeneity of their patients, doctors too do not anticipate mysterious fevers. Most experts aver that identification of pathogens is quite elementary. But complications arise because diseases go unreported at the grassroots level. “They remain unidentified because samples are collected without considering where the pathogen is likely to exist,” says K K Datta, director, National Institute of Communicable Diseases (NICD). If the disease is not reported, it does not find mention in the documents published by the institutes, thus remaining a mystery. NICD, which ‘occasionally’ publishes annual reports, brought into focus several mystery fevers raging across the nation in the 1980s.

While these unknown ailments struck Sabarkantha and Baroda in Gujarat in 1982, they surfaced in Sikar, Rajasthan, in 1983. Two state capitals — Shimla (Himachal Pradesh) and Jaipur (Rajasthan) — were afflicted in 1984. The fevers were detected in Ahmedabad in Gujarat in 1996, while Vizianagaram (Andhra Pradesh) and Meerut (Uttar Pradesh) experienced them in the following year.

Let alone unidentified fevers, even some known diseases pose problems in detection. Take, for instance, the cases of the plague in Surat and leptospirosis in Mumbai. Datta says that some diseases fail to appear on pathologists’ ‘radar’. So when they strike, diagnosis is difficult. Kalyan Banerjee, former director of National Institute of Virology, Pune, believes changes in the pathogen can modify the symptoms and delay diagnosis. This was demonstrated by the many strains of dengue. Such an outbreak necessitates laboratory investigation, only after which can diagnosis be done.

“The tendency to term a fever ‘unknown and mysterious’ shows our inability to identify a particular change in a pathogen’s character,” says S K Kar, director of the Regional Centre for Medical Research (RCMR), Bhubaneswar. To identify the disease quickly, there must be an enduring pursuit of the knowledge which the country lacks, rues Banerjee. Some experts like N S Deodhar, former director of the All India Institute of Hygiene and Public Health in Kolkata, feel that unknown diseases are directly proportionate to the ignorance of the people identifying the pathogen.



CSE DRAFT DOSSIER: HEALTH AND ENVIRONMENT>>

A. ENVIRONMENT AND DISEASES

5. INFECTIOUS AND VECTOR-BORNE DISEASES

When an unidentified fever strikes, the usual cover-ups are resorted to. Even if the local media brings it to the attention of the public, medical agencies dismiss these reports and make the ailment out to be malaria (especially cerebral malaria), Japanese encephalitis, dengue, cholera or gastroenteritis. If, however, the symptoms don't match any of these diseases and higher-ups are not convinced, it is promptly termed a mystery fever.

One of the reasons why unidentified fevers go undetected is that research bodies undertake disease-specific studies. Most Indian Council for Medical Research (ICMR) institutes are working on very specific diseases and are not trained to identify unknown organisms. The Regional Medical Research Centre for Tribals in Jabalpur, Madhya Pradesh, which has been active for the past 17 years, has only focused on yaws (a form of syphilis) and malaria. Though its director, R S Tiwari, admits that unknown and mysterious fevers exist, his institute is yet to identify a single one.

Neeru Singh, field director of Malaria Research Centre (MRC), Jabalpur, also acknowledges that mystery fevers are lurking but clarifies that MRC is focusing on malaria only. P K Sinha of Rajendra Medical Research Institute, Patna, says that among the infectious diseases his research is restricted to kala azar.

There is infrastructure to tackle such diseases. BSL-4 lab (Biosafety level 4) is one such facility to study and control known and unknown viruses. There are around 28 BSL-4 labs in the world, some even in developing countries such as Brazil and Iraq. But India lacks one. The National Institute of Virology (NIV), Pune, was established in 1952 precisely for this purpose. It was earlier called Virus Research Centre. The institute claims to have investigated more than 200 epidemics caused by viruses and identified most of them. It is another matter that NIV has failed to predict, prevent or control an epidemic. It launched a project in 1977 to develop a BSL-4 level laboratory. But even after 23 years and Rs 12.87 crore spent, the project is yet to take off.

"As compared to the global infectious disease surveillance system of the WHO, the indigenous mechanism is poor. The Union ministry of health gets to know about epidemics through media reports instead of the public health workers," says Deodhar. Even then scientists and health officials usually wait for the affected state's government to contact them.

According to Deodhar, surveillance studies are not conducted properly as diagnosis is descriptive rather than analytical. WHO uses a semi-automated web crawler that scans news available on the Internet to identify epidemics. After verifying these epidemics, it disseminates the information through the global outbreak alert and response network.

WHO is trying to map epidemic-prone areas through computers. It has also developed an Early Warning and Response Network (EWARN) to detect seven major diseases and ensure rapid response for their control. The US-based Centres for Disease Control (CDC) has also evolved a method to report and identify infectious diseases. Unfortunately, such collaborations are not taking place in India, thanks to the bureaucratic set-up. "Early detection of a disease is possible only if an organisation like the NICD becomes an autonomous body," feels Datta. This would still not address the issue regarding the control of diseases.

The national health policy, 2001, accepts the shortcomings in the disease surveillance system and hopes to overhaul the system by the year 2005. Unfortunately the focus is once again on known diseases and their control, while new and emerging diseases have not even been mentioned.

Precautions to either contain the spread of the disease or the control of vectors are also not taken. "Every year chicken pox and mumps epidemics occur when schools reopen in



the summer. But routine vaccinations are not carried out,” says A S Daga, assistant professor of preventive and social medicine, at the Grant Medical College, Mumbai. Deodhar highlights another glaring case. All patients suspected to have plague in Surat were housed together. This led to the spread of the disease to even those who had not contracted it earlier, he says. Even when precautions are taken, they do not have a scientific basis. For example, during the plague outbreak, the Nirman Bhawan and ICMR buildings in New Delhi were treated with insecticides, reveals Deodhar. These control measures are empirical and not specific and, therefore, ineffective.

The lack of coordination between the state and Union governments and even between various ministries at the Centre is one of the major reasons for the delay in identification and control of diseases. Consequently, even the decrepit infrastructure that is available lies idle. Lalji Singh, director of Centre for Cellular and Molecular Biology (CCMB), Hyderabad, says that they are capable of identifying any microorganism if it is isolated and sent to them.

But Datta says collaboration is difficult. When CCMB was asked to identify the causal agent of the Siliguri fever, the institute asked for the cost of the chemicals. This could not be given as the sample was already being sent to NIV, which is said to have more expertise in the field of virus identification.

According to Banerjee, the surveillance mechanism should be integrated with the public health system so that an outbreak can be reported as soon as it occurs. Deodhar believes that decentralisation of healthcare systems is the solution to the problem. Strict protocols of ‘what, how and when’ samples should be prepared and collected. “At present, even medical colleges do not have adequate facilities to identify causal organisms,” says Daga.

Experts feel that along with investment in equipment, the staff must be supplemented with training people in epidemiology. An important step towards facilitating the identification of pathogens would be to develop a repository of pathogens.

From oblivion to pandemic proportions, pathogens make it look remarkably simple. Diffusion of people, mixing of species, poverty, war, squalor, environmental change and susceptible populations, all contribute to their rise. These influences need to be built into a strong disease surveillance system. The best that governments, institutions and communities can do is to keep a few paces ahead of the disease through constant monitoring, simple hygiene and some common sense.

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