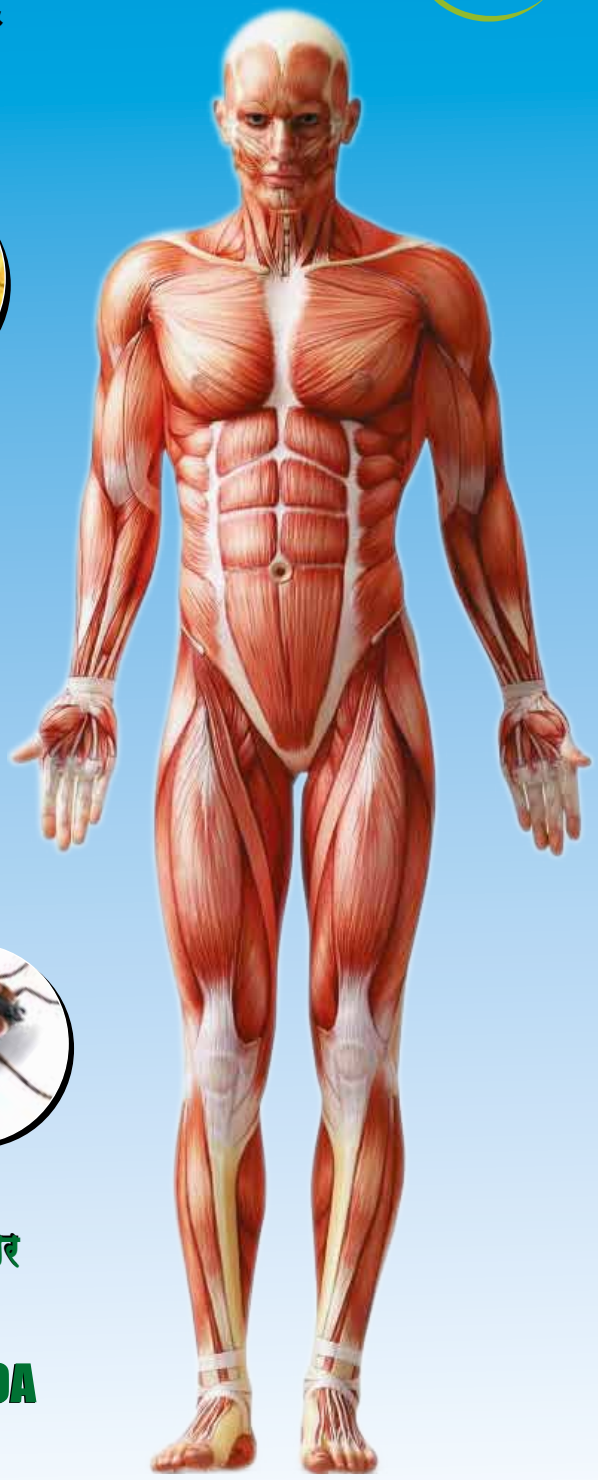
Technical Bulletin No.:39

Important Zoonotic Diseases: Prevention & Control

CELEBRATING 25 Years Silver Jubilee Publication



Z. B. Dubal
S. B. Barbuddhe
N. P. Singh

भाकृअनुप
ICAR

गोवा के लिए भा.कृ.अनु.प. का अनुसंधान परिसर
(भारतीय कृषि अनुसंधान परिषद)
ओल्ड गोवा ४०३ ४०२, गोवा, भारत

ICAR RESEARCH COMPLEX FOR GOA
(Indian Council of Agricultural Research)
Old Goa - 403 402, Goa, India

Technical Bulletin No.: 39

Important Zoonotic Diseases: Prevention And Control

Prepared By

Z. B. Dubal
S. B. Barbuddhe
N. P. Singh

ICAR RESEARCH COMPLEX FOR GOA
(INDIAN COUNCIL OF AGRICULTURAL RESEARCH)
Old Goa - 403 402, Goa (India).

Published by
Dr. N.P. Singh
Director
ICAR Research Complex for Goa
Ela, Old Goa- 403 402, Goa, India

Phone : +91 832 2284678/679
Fax : +91 832 2285649
E-mail : director@icargoa.res.in
Website : www.icargoa.res.in

*

Authors

*

Correct citation:

Dubal Z B, Barbuddhe S B and Singh N P. (2014). Important Zoonotic Diseases: Prevention and control. Technical Bulletin No. 39. ICAR Research Complex for Goa (Indian Council of Agricultural Research), Old Goa- 403 402, Goa, India.

*

Page Layout : Mrs. Sushma S. Gadagi

*

Printed at:
M/s. Impressions, Belgaum

FOREWORD

SINCE prehistoric time, major changes in human disease burden, spatial distribution, and pathogen types have arisen largely owing to human activity. The changes from small hunter gatherer to large agricultural communities was associated with the emergence of human contagious diseases, many of which are animal origin. Out of all microbial pathogens, 61% are zoonotic with 13% species regarded as emerging or reemerging. Among emerging infectious diseases, 75% are zoonotic with wildlife being one of the major sources of infection. In India, agriculture and animal husbandry workers such as farmers, livestock owners, animal handlers, veterinary extension workers and veterinarians contract many zoonotic diseases.

Etiological changes in mans environment and agricultural operations, increased movement or traveling of man, handling of animal byproducts and waste, overcrowding of animals, increased trade in animal products, drug resistant organisms, changing livestock farming practices, climate change and disaster and changes in microbial pathogens due to genetic shift and drift are the principal factors responsible for emergence and re-emergence of pathogens. Many of these factors are interrelated. Increased urbanization allows faster spreading of any new disease between populations and air travels enables to spread the disease all over to world within a short span of time. Emerging pathogens are more likely to be viruses than other pathogen types and more likely to have a broad host range. Interestingly, many of such pathogens are maintained in the environment by wide reservoir host and many of such emerging pathogens have wildlife reservoir as well as domestic

rodent reservoir. These diseases have potentially serious human health and economic impact. In the developing countries, the impact of such diseases are comparatively more than the developed countries because of poor diagnostic facilities, under reporting system, poverty and lack of medical facilities at rural part of developing countries including India. Further, lack of awareness and poor personal hygienic practices as well as poor farming practices continuously add the human and animal disease and death cases. Many infections are associated with poor sanitation, contaminated food, inadequate personal hygiene, or access to safe water and lack of basic health services, which are common in India. Several programme are regularly organized by Government of India for control of such zoonoses. Though Government of India launched many programme for effective control

and prevention of emerging zoonotic diseases, new diseases are still bombarding and the old diseases are still encroaches in the newer geographical areas of India. Thus, India have a big challenge to fight with such emerging and reemerging zoonotic diseases for saving such a huge human and animal population which are always at greater risk.

(Narendra Pratap Singh)

PREFACE

LIVESTOCK is an integral part of farming community in India as 65-70% human population is dependent on agriculture and allied sector especially animal husbandry. The concept of emerging infectious diseases appeared in late 1980s, when major outbreaks occurred around the globe e.g. Hantaan virus in USA. Later many emerging diseases have been reported from several countries which have potentially serious human health and economic impact. Examples are avian influenza, severe acute respiratory syndrome (SARS), Bovine Spongiform Encephalopathy (BSE) and the Nipah virus. The major factors involved in the increase in zoonoses, whether new or old, include population shifts and growth; changes in behavior; group urbanization, poverty and crowding, changes in ecology and climate; evolution of new strains of microbes; inadequacy of the public health infrastructure, modern tourism and liberalized trade. There are several domestic and wild animals as well as birds which are playing important role in transmission and maintenance of zoonotic infections. Wild carnivores and stray dogs are major reservoir of rabies in India. Pig on the other hand also known as "Mixing Vessels" for influenza viruses where, reassortment of various genes of influenza viruses easily takes place. Pig also acts as an amplifying host for Japanese encephalitis. As far as birds are concerned, the entry of migratory birds in wild life sanctuaries as well as water bodies could not be stopped, thus playing a vital role in transmission of avian influenza in India and various other countries in different continents.

The role of rodents in transmission of zoonoses have been accepted worldwide. Rodents exploit a wide variety of habitats

and environment even those with extreme climatic conditions throughout the world. Some of these infections can also be indirectly transmitted by the bite of vectors like mosquitoes, flies, ticks, flea or mites. India is facing problem of Hanta virus infection, Kyasanur forest disease and Crimean Congo Hemmorhagic fever which are recently reported as rodent borne zoonotic infections. Beside this, rickettsial infections like Q fever and scrub typhus are also endemic and big challenged for India. The majority of human population is staying in close proximity with domestic animals as well as with domiciliated animals especially in slum areas of various cities which highlights the outbreaks of many bacterial infections like leptospirosis, salmonellosis, brucellosis etc. and viral diseases like dengue, Nipah virus infection and Japanese encephalitis. Some areas are prone for tick borne viral disease e.g. Ganjam virus and Bhanja virus infection as observed in sheep and goat from Orissa. Most of the emerging viral diseases are surprisingly zoonoses. To tackle these diseases, their control in animal species is essential. For all these diseases or infections, veterinarians were instrumental in their identification, isolation of the causative organisms and understanding of the epidemiology of the infection. A close liaison between veterinarians and medicos is therefore necessary.

(Authors)

CONTENTS

Introduction

THE Joint Expert Committee of WHO and FAO (1959) has defined Zoonoses are "those diseases and infections which are naturally transmitted between vertebrate animals and man". Zoonoses include only those infections where there is either a proof or a strong circumstantial evidence for transmission between animals and man. Zoonoses is the word derived from Greek word "zoo" means animals and "noses" means diseases, the term coined and first used by Rudolf Wirchow who defined it for communicable diseases. There are variety of infectious agents (more than 300) which spreads through various modes of transmission. Of the 1415 microbial diseases affecting humans, 61% are zoonotic with 13% species regarded as emerging or reemerging (Taylor *et al.* 2001). Among emerging infectious diseases, 75% are zoonotic with wildlife being one of the major sources of infection (Daszak *et al.* 2001). More than 20 virus families contain human pathogens of which *Flaviviridae, Bunyaviridae, Reoviridae* and *Togaviridae*, accounting for more than half of the species affecting humans.

The wide range of animals (domestic, pets, companion, synanthropic) acts as a reservoir and carrier of many zoonotic disease agents and organisms are distributed in the natural ecosystem such as soil, water, food and aerosol. The link between human and animals with their surrounding are very close especially in developing countries where animals provide transportation, draught power, fuel, clothing, proteins etc. This can lead to serious risk to public health with severe economic consequences. As the population continue to increase and new area are opened up for food production, both for humans and their livestock, which are more frequently exposed to disease agents as a results of encounters with wild animals, thereby increasing human exposure to rare zoonotic infections. Increased urbanization allows faster spreading of any new disease between populations and also within an area. However, air travels enables to spread the disease all over to world within a short span of time. In the 20th century, the epidemiology of the occurrence of disease has been shifted due to a number of factors and driving force that have converged to create a new era of emerging and re-emerging diseases.

WHO/FAO/OIE joint consultation on emerging zoonotic diseases held in Geneva on 3-5 May 2004, defined an emerging zoonosis as "a zoonosis that is newly recognized or newly evolved, or that has occurred previously but shows an increase in incidence or expansion in geographical, host or vector range". The concept of emerging

infectious diseases appeared in late 1980s, when major outbreaks occurred around the globe e.g. Hantaan virus in USA. These diseases have potentially serious human health and economic impact and their current upward trend are likely to continue. Examples are avian influenza, severe acute respiratory syndrome (SARS), Bovine Spongiform Encephalitis (BSE) and the Nipah virus. A total 3461 cases with 170 deaths of SARS have been reported from 25 countries on five continents. In 2003, the National Institute of Virology confirmed the presence of the new corona virus which causes SARS. More than 60 bacteria families contain human pathogens; however, the enterobacteria and the mycobacteria account for the major emerging and reemerging species. Re-emerging diseases are age-old diseases that have increased their prevalence again. Some of these diseases were previously treatable but have developed resistance to the drugs used to treat them. The increase in migration due to war and international travel has also facilitated the spread of disease. Cholera, for example has increased due to increase in shipping. The TB, which claims one human life every minute in our country in a deadly combination (particularly of the multi drug resistant strains of *Mycobacterium tuberculosis*) with AIDS is first emerging as a major human killer; leptospirosis as an emerging disease claimed many human lives following floods in Maharashtra and Gujarat in recent past. Between 29 March and 10 May 2005, a total of 214 cases of meningococcal disease including 16 deaths (case fatality rate 7.5%) has been reported to WHO. NICD has confirmed *N. meningitidis* serogroup A in 7 cerebrospinal fluid specimens. India has had its share of emerging infections (*Vibrio cholerae* 0139, plague, Gp–B rota virus, HIV/AIDS, Nipah virus, chikungunya fever, Chandipura encephalitis, H5N1 influenza, etc.) laced with successes and controversies.

In recent times, India had to mobilize a sizeable proportion of its precious resources and trained manpower from developmental activities to win the nagging war against bird flu (HPAI caused by H5N1 strain) since 18th Feb., 2006 when it was first reported from Navapur Dist. of Maharashtra, and subsequently from other places including Jalgaon (Maharashtra), Gujarat and MP in 2006; Imphal (Manipur) in July, 2007, West Bengal, Assam and Tripura in 2008, Sikkim on 19th January, 2009, and recently from Tripura in 2011-12.; Brain fever (JE) which has become endemic in the central eastern part of U.P. and NE region of the country had killed approximately 1016 children and left many crippled with permanent neuromuscular retardation in 2005.

Factors Influencing The Emergence of Zoonotic Diseases

At least 11 pathogens have emerged or re-emerged in India during 1992-2009, majority of which were of animal origin. In India, agriculture and animal husbandry workers such as farmers, livestock owners, animal handlers, veterinary extension

workers and veterinarians had been found to commonly contract approximately 40 zoonotic diseases. Similarly, people engaged in production and processing of livestock products such as personnel working in abattoir, dairy, poultry enterprises and piggery suffer frequently with about 22 zoonotic diseases including rabies, JE, Kyasanur forest disease (KFD), anthrax, brucellosis, plague, TB, Leptospirosis, Salmonellosis, Campylobacteriosis, listeriosis, verotoxic *E. coli* and Clostridial infections. The following factors are responsible for emergence or re-emergence of bacterial zoonotic diseases…

1. Etiological changes in mans environment and agricultural operations e.g. Leptospirosis, plague, Rift Valley fever, Kyasanur Forest Disease etc.
2. Increased movement or traveling of man e.g. amoebiasis, giardiasis, colibacillosis, slmonellosis, SAARS, Yellow fever etc.
3. Handling animal byproducts and waste e.g. anthrax, chlamydiosis, dermatophytosis, tularaemia
4. Culture anthropological norms e.g. dermatophytosis, food borne infections, brucellosis etc.
5. Increased in density of animal population e.g. dermatophytosis, tuberculosis etc.
6. Increased trade in animal products e.g. anthrax, brucellosis, salmonellosis, Hantaan virus, Bird flu etc.
7. Drug resistant organisms e.g. E.coli, Staphylococcus aureus etc.
8. Changing livestock farming practices e.g. E.coli O157:H7, Salmonellosis, Listeriosis etc.
9. Changing environmental conditions including climate and disaster e.g. plague, Leptospirosis etc.
10. Pathogen changes like genetic shift and drift e.g. Influenza, E.coli, Staphylococcus aureus etc.

Natural animal habitats like national park and sanctuaries are also frequent sources of disease transmission. The major factor involved in the increase in zoonoses, whether new or old, include population shifts and growth; changes in behavior; group urbanization, poverty and crowding; changes in ecology and climate; evolution of new strains of microbes; inadequacy of the public health infrastructure, modern tourism and liberalized trade. Many of these factors are interrelated. There are many examples of zoonoses that were probably prevalent previously with silent

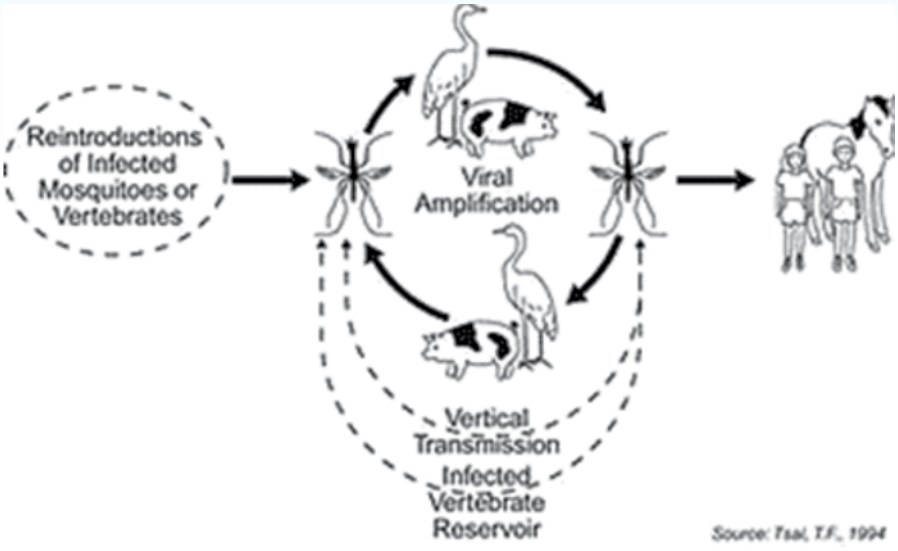
foci but have either surfaced recently or just recognized. On the other hand, many emerging zoonoses are not easily identified because the clinical signs observed are not specific or distinguishable from other clinical infections or the animals are healthy carriers with no apparent clinical signs.

The avian flu has hit our poultry industry severely (in Maharashtra, West Bengal and other states) and despite the most appropriate methods of controlling the disease, new areas keep coming in the grip of the disease. It is because our borders are porous, and at every moment, there is possibility of introduction of new disease from neighboring countries, like avian flu from Bangladesh. New animal disease are not only emerging or re-emerging in our country at an alarming rate but are potentially dangerous to humans such as HAPI (highly Pahogenic Avian Influenza); Nipah, Hendra, Hantaan, norovirus, SARS and recent outbreak of Crimean Congo hemorrhagic fever in Ahmadabad, Gujarat since 18th January, 2011. Vector borne diseases like Japanese encephalitis, dengue, West Nile virus, KFD and Rift Valley fever are also spreading to a much wider areas. Therefore, all challenges of existing diseases coupled with newer challenges increase the responsibilities of researchers, scientists, disease diagnosticians and field veterinarians.

Important viral zoonotic diseases in India

Japanese Encephalitis (JE): Japanese Encephalitis (JE) is a mosquito borne viral zoonotic disease of public health importance with epidemic potential and high mortality rate. Japanese encephalitis virus (JEV) (Family-*Flaviviridae*) is the most common cause of childhood viral encephalitis in the world with an estimated 50,000 cases and 10,000 deaths annually (Monath and Heinz 1996; Solomon *et al.* 2003). The disease was first recognized in Japan and the country was experiencing recurrent outbreaks of encephalitic syndrome since 1871 known as Von Economo's disease or encephalitis lethargic. Several neighboring countries like Russia, China, Malaysia, India, Indonesia, Thailand etc. experience the disease outbreaks. In India, it was first detected in 1955 from Tamil Nadu. There are two subtypes of JE virus such as Nakayama and JoGAr – 01 (Beijing), however GP78 (Nakayama strain) is thought to be the most common Indian strain of JE. Pig is the amplifier host. JE is primarily a disease of rural, semi urban, agricultural areas where vector mosquitoes (mainly *Culex tritaeniorhynchus* and less frequently other species of *Culex* and *Anopheles*) proliferate in close association with pigs and other animal reservoirs.

JE occurs chiefly in three areas: (1) China and Korea (2) the Indian sub-continent consisting of India, parts of Bangladesh, southern Nepal, and Sri Lanka (3) the Southeast Asian countries of Burma, Thailand, Cambodia, Laos, Vietnam, Malaysia, Indonesia and the Philippines. JE was clinically diagnosed for the first time in



India in 1955 at Vellore, erstwhile North Arcot district of Tamil Nadu. A large scale outbreak of JE was reported from Bankura and Bardwan districts of West Bengal in 1973. Since then the disease has been reported from 24 states/Union Territories so far. There was a rise of JE incidence in 1980s and has dropped significantly and maintained till 1995. Later the disease spread to new areas probably due to agricultural development and intensive rice cultivation supported by irrigation schemes that lead to an increase in mosquito population. It is a seasonal disease in temperate areas. Epidemics coincide with the heavy rainfall and or floods during monsoon and post monsoon period (August to December), agricultural practices, due to high density of the mosquito vector (because of stagnant water), and presence of reservoir host (pigs). Northern India, including North-eastern India, receives summer monsoons and as such the transmission season begins from May, with incidence reaching peak in August-October depending on the advancement of monsoon. With onset of winter, JE outbreaks subside. However, in endemic areas, sporadic cases may occur throughout the year due to congenial climatic conditions (e.g., Southern India).

JEV is endemic in the Gorakhpur and Basti divisions of eastern Uttar Pradesh. An abundance of rice fields and a bowl-shaped landscape of this region allow water to collect in pools (Parida *et al.* 2006). Heavy rains saturate the ground providing ideal breeding conditions for mosquitoes. In addition, high temperature and relative humidity provide a suitable environment for JEV transmission. The longest and most severe epidemic of JE in 3 decades occurred from July to November 2005 in Gorakhpur, Uttar Pradesh, India. Overall, 5,737 persons were affected in 7 districts of eastern Uttar Pradesh, and 1,344 persons died (WHO 2005). A warning to the

impending Japanese encephalitis outbreak was given to Tamil Nadu Government in 2006. The disease has been reported in 2007 from Assam (368), Goa (44), Tamil nadu (17), Manipur (11), Karnataka (6), Haryana (6) and Kerala (1) (Anon, 2007. www.promedmail.org). In 2010, most human cases were reported from May to October especially in northern India. The season may be extended or year round in some areas especially in southern India. Human cases reported from all states except Dadra, Daman, Diu, Gujarat, Himachal, Jammu & Kashmir, Lakshadweep, Meghalaya, Nagar Haveli, Punjab, Rajasthan and Sikkim. Highest rates of human disease have been reported from the states of Andhra Pradesh, Assam, Bihar, Goa, Haryana, Karnataka, Kerala, Tamil Nadu, Uttar Pradesh and West Bengal.

Pigs and birds are the most important reservoirs. Though they usually do not manifest the disease, they develop very high titers of virus and infect mosquitoes. Thus, pigs are known for amplifying hosts. However, pregnant sows prematurely give birth to infected and often dead piglets leading to huge economic impact on swine market. Pond heron, grey heron, night heron, cattle egret and little egret are the known birds involved in bird cycle in India. Serological evidence of JEV activity among paddy birds, crow, duck and other water birds were also documented. Susceptible children are infected by infected mosquito bites. The virus enters in the brain and neurological cells through hematogenous route where it causes extensive damage to the brain cells by mechanical means and inflammatory reactions. Later, complementary mediated cytolysis of the infected brains cells have been occurs. The infected person shows symptoms like high fever, severe headache, prosration, neuchal rigidity and altered sensorium. In some cases, incoordication, paralysis and death can be occur in extensive damage of the neurological cells. Frogs, snakes, egrets, bats and most domestic animals like cattle and horses are also infected by the virus. Man is an incidental and dead-end host. Man-to-man transmission does not occur in nature. Cattle also act as dead-end host in the transmission cycle. From Ardeid birds, JE infection is transmitted by mosquitoes to pigs/ducklings. Man or cattle get infected either from birds or pigs/ducklings through mosquito bite. Ardeid bird–mosquito–Ardeid bird and pig/duckling–mosquito–pig/duckling cycle exist in nature.

The virus cannot usually be isolated from clinical specimens, even with the best laboratory facilities, probably because of low levels of viremia and the rapid development of neutralizing antibodies. The diagnosis is therefore usually based on the presence of antibodies. The IgM capture ELISA for serum and CSF has become the accepted standard for diagnosing JEV (Parida *et al.* 2006).

Chikungunya virus: This is also a mosquito borne viral infection characterized by severe, sometimes persistent joint pain (arthritis) with fever and rashes. The structural proteins of CHIK virus from both African and Asian strains were well

isolated and analysed (Simizu *et al.* 1984), while serological cross-reactivity further defined the virus and grouped CHIKV within the Semliki Forest virus (SFV) antigenic serocomplex (Weaver *et al.* 2005). Monkeys, and possibly other wild animals, may serve as reservoirs of the virus. It is mainly caused by the bite of infected *Aedes Aegypti* mosquitoes. The incubation period can be 2-12 days, but is usually 3-7 days followed by clinical signs of fever, debilitating arthralgia (joint pain), swelling of joints, stiffness of joints, myalgia (muscular pain), headache, fatigue (weakness), nausea, vomiting and rash. It is rarely life-threatening. Homeopathy has very effective treatment for Chikungunya. Homeopathic treatment can also be given along with conventional treatment (allopathy) if desired. Rhus Tox, Eupatorium Perf, Bryonia, Arnica are the few homeopathic remedies that help in Chikungunya but more accurate and effective remedy can be chosen by a homeopathic doctor according to clinical picture of that particular case.

CHIKV was first isolated from the serum of a febrile human in Tanzania (formerly Tanganyika) in 1953 during an epidemic of dengue-like illness (Robinson 1955; Ross 1956). Retrospective case reviews have suggested that CHIKV epidemics occurred as early as 1779 but were frequently documented inaccurately as dengue outbreaks (Carey 1971). Numerous large cities in South East Asia particularly Calcutta and Bangkok have been identified as active sites for transmission of disease (Burke *et al.* 1985; Pavri 1964; Sarkar *et al.* 1965). Chikungunya fever has an interesting epidemiology, it shows cyclical pattern of appearance and disappearance, of which major epidemics occur at an interval of 7-8 years and sometimes as long as 20 years between two episodes. Outbreaks were reported from various parts of India such as Vellore, Calcutta and Maharashtra in 1964; Sri Lanka in 1969; Vietnam in 1975; Myanmar in 1975 and Indonesia in 1982. After an interval of more than 20 years, chikungunya fever reappeared in several countries including India, Indonesia, Maldives, Thailand and various Indian Ocean islands. During the 2005–2007 explosive epidemics in the Indian Ocean islands and in India, anecdotal cases of CHIKV-associated deaths, encephalitis and neonatal infections were reported (Powers and Logue 2007). Till 10 October 2006, 151 districts of eight states/provinces of India have been affected by chikungunya fever. The affected states are Andhra Pradesh, Andaman & Nicobar Islands, Tamil Nadu, Karnataka, Maharashtra, Gujarat, Madhya Pradesh, Kerala and Delhi. More than 1.25 million cases have been reported from the country with 7,52,245 cases from Karnataka and 2,58,998 from Maharashtra (Chhabra *et al.* 2008). The envelope 1 gene (E1) sequences of the virus isolated from the *Ae. albopictus* mosquito species showed close genetic relatedness (Kimura 2 Parameter genetic distance=0.0013) to CHIKV-positive isolates from human serum samples from Kerala (Kumar *et al.* 2012). The recent outbreaks of chikungunya in Orissa have been caused by viral strains of IOL group of the ECSA genotype with E1-A226V, E2-I211T and E2-L210Q mutations,

which in turn has favored *A. albopictus* to be the main arboviral vector in this region (Das *et al.* 2012).

Rabies: Rabies is one of the most important oldest recognized diseases in India. It has been recognized in India since the Vedic period (1500–500 BC) and is described in the ancient Indian scripture *Atharvaveda.* It is a highly fatal zoonotic viral encephalitis caused by rabies and related viruses, Genus- *Lyssavirus* Family- *Rhabdoviridae* that infects almost all warm-blooded animals especially carnivores (dogs, cats, foxes, jackal etc) including wild life and human beings. The disease is most commonly seen in carnivores from which it is transmitted to herbivores and other hosts. Human rabies transmitted from wild animals is rarely reported in India, where nearly 95% deaths occur due to bites from rabid dogs. The dog has been, and still is the main reservoir of rabies in India (Ghosh, 2006). However, wild life rabies is also a big problem and challenged to India. Recently, an incidence of rabid fox bites in a village in southern part of India involving 18 individuals, including 4 children were reported by Madhusudana et al (2013). Out of the 18 individuals, one person was died with the rabid symptoms. The partial Nucleoprotein (N) gene sequencing of the virus isolated from the patient who died of rabies had close homology with species I (prototype rabies) sequences available in GenBank and our own past isolates from dogs and humans, thus confirming that virus spillover from wildlife to domestic dogs continues to occur in India (Madhusudana *et al.,* 2013).

Rabies is transmitted by bites of rabid animals, corneal transplant, kisses or sexual contact, tissues of an infected animal or fresh wound that come into contact with saliva or tissues of an infected animal. The disease is characterized by marked change in behavior like irritability, increase sensitivity to noise and light, increased alertness, restlessness, aggression, depression, hiding in dark place, impaired corneal reflex and attack. In more severe form, the affected animal showed nervous signs like irritability, viciousness, biting and attacking, muscle tremors, incordination, pica, spasm, paralysis, difficulty in swallowing, drooling and frothing of saliva, dropping of jaws, coma and deaths. Both sylvatic and urban cycles of rabies perpetuate in India since ancient times. Rabies is present throughout the country, except in the islands of Lakshadweep and, Andaman and Nicobar. India has the highest incidence of human rabies in the world. India reported about 20,000 human deaths every year (Sudarshan et al. 2008). The Indian Association for the Prevention and Control of Rabies estimates that about 80,00,000 people receive treatment for dog bites every year. Once symptoms of rabies develop, it is almost invariably fatal in humans. The problem with dog rabies in India is that dogs are asymptomatic carriers and they are freely available in every human establishment. The dog population was estimated to be about 25 million, most of which are ownerless and not immunized against

rabies. The estimated animal bite load per year was 2.28 million. A study carried out in four cities of India has reported the annual incidence of animal bites to be 2.1 per 1000 population. The number of animal bites in the capital city of Delhi had increased from 23852 in 1995 to 29905 in 1998 of which over 95% were dog bites (Bansal 2004, www.japi.org/january2004/Correspondence.pdf). Many cases of dog bites have been reported from various states of India. Recently, a British woman bitten by a puppy in Goa has died in a UK hospital from rabies (Navhind Times, 29th May, 2012). Another case of a man from Karnataka and working in Goa was diagnosed in Goa Medical College (GMC) with rabies 25 years after the dog bite (Times of India, 16th September, 2012). Children constitute 30 to 50% of those receiving post-exposure vaccination or dying from the disease. The nuisance of stray dogs seems to have assumed epidemic proportions everywhere. Municipal bodies across India avoid catching these stray dogs due to animal rights activists, for instance the number of stray dogs culled in Delhi had decreased from 50,000 to 2500 a year (Bansal 2004, www.japi.org/january2004/Correspondence.pdf).

The disease has been eliminated from UK, Japan, Malaysia, Taiwan, Portugal and Uruguay while rest of the countries are facing problem of rabies elimination and control. Different strategies have been adopted to control/eliminate rabies broadly viz., those directed at humans (pre exposure treatment) and those directed at animal host species (population control and vaccination). The strategies should be used in combinations to control the rabies in animal as well as in humans. In order to eliminate canine rabies, there are few pre-requisite viz., availability of potent cell culture vaccine, medical and veterinary professional interactions, effective methods for prevention and control with effective diagnostic services, increase awareness in public, implementation of dog control strategies, financial backing and other inter-sectoral cooperations etc.

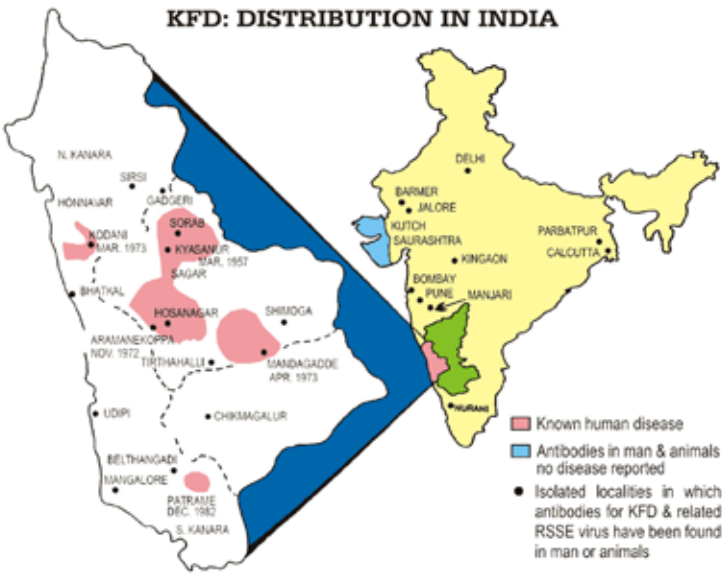
Chandipura virus encephalitis: Chandipura virus (CHPV) is a vesiculovirus, Family *Rhabdoviridae*. Vesiculo viruses were isolated in 1965 in the Chandipura (Nagpur) region of India from two adult patients with febrile illness during an outbreak caused by chikungunya and dengue viruses (Bhatt and Rodrigues 1967). The virus, known to be carried in dormant stage by sandflies, which live near domestic animals like cows and buffaloes, is transferred from the fly to human beings specifically during monsoon. Cases clinically diagnosed as viral encephalitis from Raipur in central India in 1980 showed CHPV aetiology, confirmed by isolation of CHPV virus from the acute sera (Rodrigues *et al.* 1983). Anukumar *et al.* (2013) observed a progressive replication of virus in spinal cord and brain but not in other tissues like kidney, spleen and liver of suckling mice. Histo-pathological lesions noticed in the spinal cord and brain tissues suggested the extensive damages in these tissues. Chandipura virus has been isolated in Nigeria from hedgehogs

(*Atelerix spiculus*). In natural conditions, it has been isolated from a pool of 253 unidentified Phlebotomine sandflies (*Phlebotomus* spp.) in the Maharashtra State of India (Dhanda *et al.*, 1970) and from unidentified *Sergentomyia* in the Karimnagar district in Andhra Pradesh, India (Geevarghese *et al.*, 2005). The data show a wide distribution of the virus and the capacity of two genera of sandflies namely *Phlebotomus* (subgenera *Phlebotomus* and *Euphlebotomus*) and *Sergentomyia* to transmit the virus.

CHPV was incriminated as the etiological agent of large-scale encephalitis outbreaks in children (9 months to 15 yr of age) in various districts of Andhra Pradesh in 2003 with high case fatality rate (CFR) of 55.6% (Rao *et al.* 2004). In a focal outbreak in eastern districts of Gujarat between June and July 2004, CHPV aetiology in 11 of 20 encephalitis cases indicated its importance as an encephalitis causing virus in endemic areas in India (Chadha *et al.* 2005). An outbreak of twenty-six cases of encephalitis with a case fatality rate of 78.3% was investigated among children in Gujarat State, India in 2005. Chandipura virus RNA was present in 9 of 20 acute-phase serum samples, and virus sequences from the present outbreak were closely related to prototype strain (1965) and Andhra Pradesh, India (2003) isolates (Chadha *et al.* 2005). In the Nagpur region of Maharashtra in 2003, 33 encephalitis cases were confirmed as Chandipura with case fatality rate (CFR) of 41%. In 2005, in an outbreak in Bhandara and Nagpur districts, 7 of 21 cases were confirmed as Chandipura (NIV annual report, 2003-2006). In a hospital-based surveillance of acute encephalitis among children from endemic areas of North Telangana region of Andhra Pradesh between May 2005 and April 2006, CHPV aetiology was identified in 25 of 52 cases with seasonality in late summer and early monsoon. An outbreak of acute encephalitis syndrome (AES) in children was reported from Nagpur division, Maharashtra, between June and September 2007. Recently, 17 people died with 29 registered cases in Gujarat in 2010 (http://www.dnaindia.com /india/report_chandipura-virus-kills-17-in-gujarat_1419111) and 17 children died in Nagpur region in July, 2012 (Shrivastav 2012, http://articles.timesofindia.indiatimes.com/keyword /niv/recent/4).

Kyasanur Forest Disease (KFD): This is a tick-borne flavivirus infection, first described by Telford Work and Harold Trapido in 1957 in a small forest region i.e. Sorab taluka of Shimoga district of Karnataka in India. KFD virus belongs to Russian Spring Summer Encephalitis group, a member of family *Flaviviridae*. The virus came to light when scientists heard that, monkeys were dying in large numbers in the area, and a number of local villagers also developed a mysterious illness comprising of severe headache, chills and a high fever. After an incubation period of 2-7 days, the clinical symptoms start with the onset of continuous fever (about 40°C) for about 12 days or more, headache, vomition, myalgia, cephalgia, insomnia, bradycardia,

hemorrhages on mucous membranes, encephalitis, neck stiffness, tremor and mental disorders. Initially, the disease was localized within a small area of Sagar Sorab taluka of Shimoga district which then moved to adjoining areas as a result of deforestation and increased human/animal activity in the forest area. KFD mainly appears during inter-monsoon period i.e. December to June when man enters the forest for collecting wood and plants for fuel and get bitten by larval and nymphal stages of infected ticks, *Haemaphysalis spinigera* which become very active during the period. KFD virus has been isolated from 16 species of ticks. However, *Haemaphysalis spinigera* is considered as main vector. In enzootic state, KFD virus circulates through small mammals such as rodents, shrews, ground birds and ticks. When monkey came in contact with infected ticks, they get infected, amplify and dissimilate the infection creating hot spot of infection.



In 1983, a large scale epidemic of KFD involving 1500 human cases and 150 deaths has been recorded which coincided with the clearing of large tracts of the forest for cashew nut plantations and grazing of cattle (Varma 2000). This has had a two-fold effect on KFD biology and epidemiology. First, significant increase in human presence in surrounding area leads to increased human exposure to the agent and its vectors (ticks). Second, the introduction of cattle to the region has led to a relative shift of host for these adult ticks which prefer to feed on large animals including humans (Varma 2000; Chomel *et al.* 2007). Avoidance from tick infested area; use of tick repellent or wearing protective cloths and use of acaricides are the preventive measures. Immunization with killed vaccine may be done for population at risk.

Swine and Avian Influenza: Swine and avian influenza are acute respiratory infection characterized by fever, cough and dyspnoea. Influenza A viruses are negative sense, single-stranded, segmented RNA viruses under the Family

Orthomyxoviridae. Several subtypes of influenza viruses are exist in nature depending on the hemagglutinin and nerraminidase antigens. So far 17 H and 10 N antigens are present. So, in theory, 170 different combinations of these proteins are possible. These many combinations are not reported till date. However, some subtypes such as H5N1, H7N3, H7N7, H7N9, and H9N2 are very pathogenic to human beings. Pandemic flu viruses have some avian flu virus genes and usually some human flu virus genes. Both the H2N2 and H3N2 pandemic strains contained genes from avian influenza viruses. Avian influenza viruses are normally not known to infect humans but human and avian influenza viruses share the haemagglutinin and neuraminidase antigens. Therefore, it is likely that some of these viruses may have resulted by recombination during infection of avian viruses in mammals or mammalian viruses in birds. An outbreak of avian influenza A (H5N1) was reported in Hong Kong during 1997 among various species of birds including chickens and ducks leading to heavy mortality in chicken. Subsequently, it was isolated from a child who died of Rey's syndrome in Hong Kong in May 1997. Intensive surveillance conducted during Nov-Dec. 1997 revealed that, the subtype has never been isolated from humans before this episode. Epidemiological and laboratory investigations implicated transmission of the virus from infected chickens to human due to antigenic variation. Since then, the outbreaks of H5N1 virus have been reported from different parts of the world with South East Asian region being highly affected.

Both H1N1 and H5N1 are unstable, so the chances of them exchanging genetic material are higher, whereas a stable (seasonal flu) virus is less likely to take on genetic material. The H5N1 virus is mostly limited to birds, but in rare cases when it infects humans it has a mortality rate of 60% to 70%. Experts worry about the emergence of a hybrid of the more virulent Asian-lineage HPAI (highly pathogenic avian influenza) A/H5N1 strain (media labeled 'bird flu') with more human-transmissible Influenza A strains such as this novel 2009 swine-origin A/H1N1 strain (media labeled 'swine flu'), especially since the H5N1 strain is and has been for years endemic in birds in countries like China, Indonesia, Vietnam and Egypt. The most recent poultry outbreaks have been reported from the northeastern part of the country, near the border with Bangladesh: between January and April 2008 from West Bengal, in April 2008 and January, 2012 from Tripura, in November and December 2008 from Assam, and again from West Bengal between December 2008 and May 2009. No human cases have been reported from India to date.

Swine influenza virus was first isolated from pigs in 1930 in the U.S. Many a times, the people have developed swine flu infection when they are closely associated with pigs. The known SIV includes H1N1, H1N2, H3N1, H3N2 and H2N3 strains. However, the strains isolated from Mexico have H1N1 antigens and mainly found

to infect the people. H1N1 was found in Spanish flu in 1918 to 1919, the Asian flu in 1957-1958 and the Hong Kong flu in 1967-1968. Many outbreaks of human influenza caused by Swine influenza (Hsw1N1) virus have also been reported from India. The term 'swine flu' is misleading, but the reason we're caught up in calling it 'swine' influenza is that the H1N1 was first found in pigs. Even though, the virus is a combination of genes from swine influenza, avian influenza and human influenza. It's a real combination of a lot of viruses which, is a common scenario in influenza viruses. The H5N1 was labeled 'avian' influenza because it was first found in birds. The novel H1N1 virus is derived originally from a strain that lived in pigs and this origin gave rise to the common name of 'swine flu', usually used by mass media. Influenza viruses can change its make-up in one of two ways: Antigenic drift is a series of mutations that cause the virus to gradually evolve over time. Antigenic shift is an abrupt change in the surface antigen proteins that suddenly creates a new subtype of the virus. Pigs are thought to be deal 'mixing vessels' for the reassortment of human, swine and avian influenza A viruses. They probably play a crucial role in the emergence of new strains which can start a global pandemic. However, the reassortment could also occur in a human, infected with both human and animal influenza A viruses. The H1N1 virus has adapted itself to humans and is highly contagious. By contrast, the H5N1 'bird flu' was (and is) spreading between birds, only occasionally spreading from a bird to a human, and even more rarely spreading from one human to a second (and maybe a third) and then stopping. So the new H1N1 isn't behaving like a swine flu virus in the sense that H5N1 is behaving like a bird flu virus, even though by the conventions of virology it is still classified as a swine flu virus. The disease was also reported from Goa where 73 samples from Salcete taluka were sent for swine flu testing of which 24 have been reported to be positive in 2010. However, all the patients were recovered completely.

Buffalopox Virus (BPV): Buffalopox is a zoonotic and contagious viral disease, which mostly affects buffaloes, but rarely cows and human beings. It is caused by buffalopox virus (BPV), classified in *Orthopoxvirus* genus of *Poxviridae* family. Occurrence of outbreaks is accompanied with 80% morbidity and significant productivity losses. Human cases of buffalo pox have been reported regularly from different states of India (Bhanuprakash *et al.,* 2010). Nosocomial outbreaks have also been reported among the nurses and burn patients due to contaminated needles (Zafar *et al*., 2007). During 1992-94, epizootics of buffalo pox were reported among buffaloes in Dhule and Jalgaon districts of Maharashtra. The disease is being continuously reported from Uttar Pradesh, Rajasthan, Andhra Pradesh, Gujarat and Karnataka. An outbreak of buffalo pox in domestic buffaloes with high morbidity rate was recorded in Aurangabad district of Maharashtra in November, 2003. The disease was also associated with human infections particularly the milkers working in affected farms (Singh *et al.* 2006). Recently, the outbreak of the disease

in buffaloes and humans has been reported from Solapur and Kolhapur district of Maharashtra which was confirmed by electron microscopy and polymerase chain reaction (Gurav *et al.* 2011).

Pock lesion on neck

Pock lesion on udder

Nipah virus: Nipah Virus infection (NiV) is an emerging infectious disease of public health importance in the South-East Asia Region. The virus is named after the Malaysian village where it was first discovered. This virus along with Hendra virus comprises a new genus designated *Henipavirus* in the subfamily *Paramyxovirinae*. Fruit bats (Genus *Pteropus*) have been identified as natural reservoirs of NiV. There were focal outbreaks of NiV in Bangladesh and India during winter in 2001. Drinking of fresh date palm sap, possibly contaminated by fruit bats (*P. giganteus*) during the winter season, may have been responsible for indirect transmission of Nipah virus to humans (Luby *et al.* 2006). There is circumstantial evidence of human-to-human transmission in India in 2001. During the outbreak in Siliguri, West Bengal, 45 people died including health workers and hospital visitors after exposure to patients hospitalized with Nipah virus illness, suggesting nosocomial infections (Chadha *et al.* 2005). An illness of mysterious fever with high case fatality was reported from district Nadia, north of West Bengal's capital, Kolkata, between 11th and 28th April, 2007. All five cases died within 3-10 days of onset of illness. Initial impressions pointed towards a new strain of dengue. However, blood samples of three dead patients tested positive for Nipah virus by RT-PCR at the National Institute of Virology, Pune. The full genome sequence of Nipah virus (18,252 nt) amplified from lung tissue showed 99.2% nt and 99.8% aa identity with the Bangladesh-2004 isolate, suggesting a common source of the virus (Arankalle *et al.* 2011). Epidemiological investigation revealed that, the index case did not give any history of travel to Bangladesh, the border is just about 5 km away from the village, where an outbreak of Nipah virus was confirmed during the same period. Till date, the human and animal cases of Hendra virus infections have not been reported from India. However, the Hendra virus is closely related to Nipah virus and outbreak of Nipah virus infection has

been reported from Siliguri and nearby areas, there is a need to monitor both the diseases in India.

Ganjam Virus Disease: Ganjam virus (GANV), a tick-borne arbovirus of veterinary importance causing high morbidity and mortality in exotic and crossbred sheep and goats, is widely prevalent in India (Banerjee 1996). It causes an acute febrile illness in sheep and goat characterized by fever, anorexia, lumbar paralysis and high fatality. The virus is antigenically related to Nairobi Sheep Disease virus. Studies at the genetic and serologic levels have demonstrated GANV as an Asian variant of NSDV as the two viruses differed only by 10 and 3 per cent at nucleotide and amino acid levels, respectively (Marczinke and Nichol 2002). Hybridization studies using RNA probes demonstrated that both GANV and NSDV are more closely related to Hazara virus (HAZV), a member of the Crimean Congo haemorrhagic fever virus (CCHFV) group than to Nairoviruses (Marriott *et al.*, 1990). CCHFV is one of the most pathogenic human viruses among Nairoviruses, which has a wide geographic distribution in Africa, Europe and Asia. A few fatal cases due to CCHFV have been reported from Pakistan and India (Athar *et al.*, 2003). The wide prevalence of GANV in different parts of India, its association with these important viruses and its versatility to infect sheep, goat and man makes it an important zoonotic agent. It was first isolated from *Haemaphysalis intermedia* ticks collected from sheep in Ganjam district of Orissa state in 1969. Subsequently, the virus was also isolated from ticks collected from sheep and goat from Shimoga district and also from *Culex vishnui* mosquito from Vellore and acute sera of sheep from Chittoor district of Andhra Pradesh. Besides *Haemaphysalis intermedia* tick, the virus was also isolated from *Rhipicephalus haemaphysaloids* ticks collected in Pune city in 2004-05 and from sheep of Chittoor district, Andhra Pradesh. Gangam virus was also isolated from the acute phase serum of a 12 yr old European boy who was suffering from febrile illness in Vellore, Tamil Nadu (Dandawate *et al.* 1969). During investigation of a disease outbreak in a sheep farm in Veerapuram village of Chengai-MGR district of Tamil Nadu, high morbidity and mortality in sheep was reported (Joshi *et al.* 1998). The wide geographic distribution of the virus, prevalence of antibodies among humans and animals and its ability to infect sheep, goat and humans poses a threat as one of the emerging viral disease of public health importance.

Bhanja virus: The Bhanja virus (Bunyaviridae) was isolated first from adult tick *H. intermedia*, collected from a goat with lumbar paralysis in locality, Bhanjanagar, Orissa state, in December, 1954 (Hubalek 1987). Later, the virus has been isolated from various countries such as Nigeria, Italy, Senegal, Southern USSR, Yugoslavia and Bulgaria. The highest prevalence of antibodies to Bhanja virus has regularly

been observed in herds of ruminants (sheep, goat, cattle), this could be due to their frequent infestation with the virus vector. Antibodies to Bhanja virus have also been detected in sheep and goat sera from Orissa, Gujarat and Karnataka state of India. Generally in adults, the virus causes an unapparent infection but in young ruminants (lamb, kid, calf) it is pathogenic, causing fever and neurological symptoms. Several cases of Bhanja virus febrile illness have been described in humans, with symptoms including photophobia, vomition, meningoencephalitis, and pareses (Calisher and Goodpasture 1975; Vesenjak-Hirjan, 1980). In spite of the presence of virus in small ruminant and ticks, the human cases of bhanja virus has not been reported from India. This might be due to underreporting system and poor diagnostic facilities. The wide geographical distribution and presence of antibody in domestic animals could probably make Bhanja virus as an emerging virus infection.

Important bacterial zoonotic diseases in India

***Leptospirosis* :** Leptospirosis is caused by the organism, a spirochete of genus *Leptospira* that consist of 8 pathogenic species, 23 serogroups and 280 serovars. The disease may appear in acute or chronic form and occurs approximately in 160 mammalian species (Alexander, 1991). The disease in animals is characterized by fever, jaundice, haemoglobinuria, abortion, still birth, repeat breeding, loss of milk etc. The mouse, vole, rat, mongoose, shrew, jackal, dog, pig, cat and cattle are the important reservoirs. Amongst these, rats and small rodent particularly *R. noruegicus* and *Mus musculus* are the most important reservoirs. The farm workers, sewer workers, fishermen and miners are at high risk of infection. In India, the disease is most commonly seen in coastal areas including Andaman and Nicobar islands. The 1980s witnessed a sudden increase in leptospirosis and seroepidemiologic and clinical studies show that the disease is endemic in Andaman Islands and southern states of India. Outbreaks were reported from Mysore, Gujarat, Nagpur and Andamans in 1997. An outbreak of leptospirosis was also reported in 2002 in Mumbai following prolonged water logging due to heavy rainfall. Leptospirosis is widespread in animals in other regions such as West Bengal, Bihar, Punjab, Haryana and Andhra Pradesh and, has been reported to be a common cause of acute renal failure in south India (WHO, 2006). The principal strains of Leptospira are *Leptospira interrogans serovars icterohaemorrhagiae, automnalis, pyrogenes, grippotyphosa, canicola, australis, javanica, sejroe, louisiana* and *Pomona*. Since 1997, the outbreaks of leptospirosis has been reported every year from different places in India viz. Madras and Valsad in 1997, Orrissa in 1999, Mumbai in 2000, Gujrat, Orrissa and Mumbai in 2002, South Gujrat and Chennai in 2003, Gujrat in 2004, Andhra Pradesh, Karnataka and Kerala in 2005-06, Karnataka and Maharashtra in 2006-07 and so on (Jena *et al.,* 2004; Mathur *et*

al., 2009). Mumbai, Gujarat, Kerala and Chennai experiencing Leptospirosis cases almost every year.

Transmission of leptospira occurs through contact with an environment contaminated by urine, aborted fetus and uterine discharge of reservoir host or other infected animals. *Leptospira* can also enter through abraded skin and mucous membrane during bathing or swimming in lake, river or canal polluted with the urine of infected animals. Leptospira are excreted in the urine of infected animals for a long time, often for an entire life time in case of rodents. Venereal transmission also occurs in rodents. Man is considered as "dead end host" but transplacental transmission has been reported. The disease may manifest in human in two phases. 1st phase is septicaemic and 2nd phase is due to immune response. Fever, anorexia, stiffness, jaundice, haemorrhage, neurological signs, abortion, haemataemia, haemoglobinaemia and death may occur. Diagnosis of Leptospirosis can be made by observation of clinical symptoms supported by laboratory investigations where the organism can easily detected by dark field microscopy in clinical material *viz*., urine, faetal tissues, blood etc. The organisms can be isolated by using Ellinghausen-MacCullough-Johnson-Harris (EMJH) medium, Kurthoff's medium and Fletcher's medium in 1 to 6 weeks. Microscopic Agglutination Test (MAT) is the most widely used test for the diagnosis of Leptospirosis. LEPTO Dipstick assay is a newly developed test for rapid diagnosis of leptospirosis and uses a broadly reactive antigen for detecting chiefly IgM antibodies. Besides these, several other immunodiagnostic methods are also available.

Combinations of different approaches like rodent control, sanitation, proper management and immunization have been used to eradicate the disease from dairy and piggeries. Monovalent and multivalent vaccines are available for animal use. The calves are vaccinated at 3-5 months of age to counter passive immunity. The vaccination which offers protection for 6 months should be done by using local strain as the protection offered is serotype specific. In man, the disease control measures include:

1. *Personal hygiene and protection* which consist of (i) drink boiled or chlorinated water, (ii) wash hands with disinfectants, (iii) drink pasteurized or boiled milk (iv) protect food articles and utensils from contamination with urine of rat, (v) encourage use of protective clothings (rubber gloves, goggles, gum-boots particularly when working in water logged areas or handling animals/animal products during slaughterting and parturition etc., (vi) avoid swimming and wading in water of lakes, ponds, swimming pods contaminated with urine of rats and livestock, and (vii) encourage mechanization of agricultural operations.

2. *Sanitation:* It includes (i) disinfection of contaminated work areas such as food stores, abattoirs, fish and meat processing plants and animal sheds, (ii) proper collection, transport, treatment and secured disposal of garbage, (iii) proper collection, treatment and secured disposal of animal excreta, (iv) proper disposal of dead and infected animals, (v) disinfection of swimming pool with chlorine, (vi) drainage of wet areas, and (vii) rodent control in the areas of domestic and farm environment.

3. *Animal related care:* It calls for (i) care in handling of laboratory and other animals as they may be carriers, (ii) regular immunization of domestic animals to protect them from disease and from becoming carriers, and (iii) improvement in occupational hygiene standards in cattle and pig farms.

4. *Health education:* General people and particularly the high risk groups should be educated about the disease, and the protective measures to be followed including prohibition of recreational activities in contaminated waters.

5. *Immunization:* Strain-specific killed vaccines for human use, particularly in endemic areas, are available but these have limited use due to many infecting serotypes. Vaccination is valuable when large numbers of workers are exposed to infection in situations (e.g. rice fields) that preclude satisfactory control of reservoir hosts. Immunization of at least 60% of population and 90% of those at risk, must be continued for 3 years to halt epidemics. Vaccine prepared from highly virulent strains appear to afford better cross-protection than those prepared from strains of low virulence. Vaccine produced in protein free (synthetic) media give much less severe reactions. Annual boosters are required to maintain sustained immunity levels.

Plague: It is a highly fatal zoonotic disease caused by a bacterium, *Yersinia pestis*. Plague is an ancient disease which caused three pandemics since the 6th century, but the global transmission has been low in recent years. The disease affects wide variety of species like man, camel, deer, dog, cat, donkey, antelope, rabbit, rat and squirrel. Plague is re-emerging disease as there is a report of re-emergence in Algeria in 2003 after a gap of 50 years. It is a bacterial zoonosis with rodents being the principle reservoir. The black rat (*Rattus rattus*) and oriental rat flea (*Xenopsylla cheopis*) are notorious reservoir and transmitting agent for human plague in India. Recently, one case of human plague was reported in Oregon, US in Times of India in June, 2012 stated that a man after biting by stray cat or mouse developed a symptoms of bubonic plague which was progressed to septicemic plague. It was unclear if it was the cat or the mouse that bit him but the bacterium that causes plague is known to be carried by rodents, cats and other carnivores and passed on to humans via fleas that feed on the infected animal (http://articles.

timesofindia.indiatimes.com/2012-06-16/science/32268631_1_bubonic-plague-mice-stray-cat)

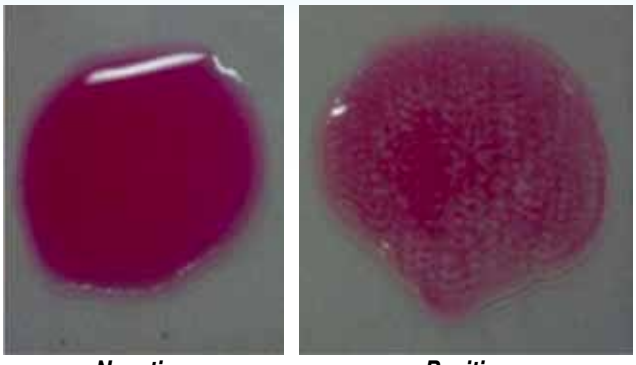
There were 12.5 million deaths in Agra epidemic during 1612. The epidemics were reported in Beed District of Maharashtra (bubonic plague) and in Surat District of Gujarat (pneumonic plague) in 1994. A total of 876 cases (596 in Maharashtra, 151 in Gujarat, 68 in Delhi, 50 in Karnataka, 10 in Uttar Pradesh and 1 in Madhya Pradesh) were presumptively diagnosed in both epidemics, out of which 54 cases were reported as dead. Recent outbreak of 16 confirmed cases of pneumonic plague with 4 deaths seen during 2002 in Shimla District of Himachal Pradesh (Gupta and Sharma, 2007). An outbreak of bubonic plague was also reported in Uttaranchal in 2004.

Clinically plague has three forms: i) Bubonic plague that involves the legs and inguinal lymph nodes causing intense haemorrhagic inflammation; ii) pneumonic plague which is now rare but highly fatal disease involving the lungs; and iii) Septicemia plague that is the terminal stage of both bubonic and pneumonic plague. If bubonic plague is not properly treated then it may lead to meningitis (meningeal plague). The important symptoms are nausea, chill, fever, dysponea, chest pain, pneumonia, epistaxis, haematuria, diarrhoea, constipation, painful and oedematous buboes, cyanosis, staggering gait, delirium, prostration, coma and death. The diagnosis of human plague is made based on clinical features suggestive of plague, demonstration of the pathogen *(Yersinia pestis*) in clinico-pathological material including sputum, CSF or throat specimens, isolation of the etiological agent on CIN agar, laboratory animal inoculation with clinico-pathological material, serodiagnosis by PHA, CFT and IgM capture ELISA and PCR. The basic measures to control the plague include i) reduction of rodent population by rodenticide and eradication of rat flea using insecticide; ii) using killed and live vaccines as prophylaxis, iii) isolation and immediate treatment of affected persons and iv) health education. Tetracycline is the drug of choice followed by chloramphenicol and streptomycin for treatment of plague.

Anthrax: Anthrax is an ancient disease commonly seen in domestic herbivorous animals (cattle, sheep, goats and horses). This is a rapidly fatal infectious disease often characterized by sudden death, exudation of tarry uncoagulated blood from the mouth, nares and anus (pathognomic symptom), spleenomegaly, gelatinous infiltration of subcutaneous and subserous tissues, and malignant pustule or eschar formation on skin. The causal agent of anthrax *Bacillus anthracis* is one of the largest of all bacterial pathogens. It is a Gram positive, endospore-forming, rod-shaped bacterium, with a width of 1-1.2 µm and a length of 3-5 µm. Anthrax spores are transmitted by contact with infected carcasses, hides, hairs or bone meal. It is endemic in countries like India, Pakistan, Iran, Russia, Latin America

and Central Africa. The disease is endemic in Tamil Nadu, Karnataka and Andhra Pradesh (Kumar *et al.,* 2000). Recently, 70 cases of anthrax seen at Christian Medical College, Vellore and 112 cases of anthrax in surrounding places (Sarada *et al.,* 1999). The majority of cases are cutaneous anthrax. However, human cases of intestinal, septicemic, meningeal, pulmonary and gastrointestinal anthrax have also been reported. An outbreak of human anthrax in Mysore with four deaths after consuming diseased deer meat has also been reported recently (Ichhpujani *et al.,* 2004). In June, 2011, human cases of anthrax have been reported from Kandhamal district of Orrissa (near Bhubaneshwar) (http://articles.timesofindia.indiatimes.com/2011-0620/bhubaneswar/296840221anthrax-hit-chief-district-veterinary-officer-veterinary-experts). However, animal anthrax has been recognized by veterinarians in all parts of India. If suspected of anthrax (on the basis of sudden death, pathognomic symptoms and history), the carcass should never opened to avoid contamination of the surroundings. Generally, for quick and fairly reliable diagnosis blood smears prepared from ear clippings or laryngeal oedema are stained for M' Fadyean reaction. On microscopic examination, bacterial capsule appears reddish purple whereas bacilli take deep blue colour. Besides this, there are various methods are available for detection of anthrax bacilli viz. isolation of the pathogen on blood agar, inoculation of laboratory animals, Ascoli's precipitation test employing anthrax hyperimmune serum against attenuated anthrax spores, specific and sensitive enzyme immunoassays based on purified toxin antigens for serodiagnosis and, PCR based diagnosis of *B. anthracis.* Penicillin is one of the promising drug for treatment of the anthrax. B. anthracis is highly susceptible to a variety of antimicrobial agents including penicillin, chloramphenicol, tetracycline, erythromycin, streptomycin, and fluoroquinolones. However, the standard treatment for anthrax is a 60-day course of an antibiotic, such as ciprofloxacin or doxycycline. For prevention, vaccine is available for people at high risk (such as veterinarians, laboratory technicians, employees of textile mills processing imported goat hair, and members of the armed forces). The control of the disease requires coordination between public health, agriculture and animal husbandry departments and the industry.

Brucellosis: The disease is characterized by abortion (usually in 5-8 months of gestation), retained placenta, orchitis, epididymitis, temporarily impaired fertility and, causes considerable economic losses due to loss of progeny, milk yield and animal protein. Brucellosis is caused by members of the genus - *Brucella.* Of different species, *B. abortus* is the most widely spread species whereas *B. melitensis* and *B. suis* are irregularly distributed. *B. neotomae* has its natural foci in the western United States while *B. ovis* is distributed in all sheep raising countries. *B. canis* infection has been confirmed in many countries such as USA, Brazil, Argentina, Mexico, Chechoslovakia, Germany, Japan and India. *Brucella melitensis*



Negative Positive

RBPT Test

is the prevalent species seen in man and causes a more severe form of disease. It is estimated that the true incidence is 25 times higher than the reported cases due to underdiagnosis. Brucellosis affects primarily the livestock and is transmitted to humans by ingestion, close contact, inhalation or accidental inoculation. Major route of infection in animals is through ingestion of contaminated food, milk and water, close contact, upper respiratory tract and conjunctiva. Besides these, contact with aborted fetus and uterine discharge and venereal transmission. Brucellosis is almost invariably transmitted to man from infected domestic animals. However, it has been documented beyond doubt, the possibility of human to human transmission of Brucella infection (Naparstek *et al.,* 1982; Lubani *et al.,* 1988; Mantur *et al.,* 1996; Tikare *et al.,* 2008). Human brucellosis was once thought to be predominantly transmitted through animal contact. Several cases of human brucellosis have been regularly reported from various states in India. Even in small state like Goa, many cases has been reported especially from rural part of Goa. Out of 50 samples collected from suspected human patients, 15 were found to be positive for Brucella (The Hindu, 5th Sept., 2010). However, it is now being realized increasingly that animal products such as milk and meat products also play an important role in the disease transmission. Dairy products prepared from unpasteurized milk such as soft cheeses, yoghurts, and icecreams may contain high concentration of the bacteria and consumption of these is an important cause of brucellosis.

Brucella infected cow (Swollen joints)

In female animals, abortion occurs in late gestation. Sometimes, fever, anorexia, lameness etc. In males, orchitis, epididymitis, testicular hypertrophy and somtimes infertility. In humans, headache, muscular pain, insomnia, anorexia, weakness,

drenching sweets with peculier odour, undulating fever (in the evening). Death may be due to sever toxaemia and endocarditis. Various studies clearly show a high prevalence of the disease in various parts of India. There are several methods are available for detection of *Brucella* pathogens from clinical samples such as Milk ring test for herd surveillance, Serum Tube Agglutination Test (STAT) and Rose Bengal Precipitation Test (RBPT) for individual animal testing. Besides these, several immunological methods viz. ELISA, CFT, EIA, SDTH are also available. Recently, PCR is widely used for its confirmation. Most effective for control of bovine brucellosis, however, due to ban on cattle slaughter in certain states of India, it has been replaced with test and segregation policy. Vaccination of animals with suitable vaccine like *B. abortus* S-19, *B. melitensis* Rev-1 in cattle, sheep and goats, *B. suis* strain 2 in pigs and hygienic measures can protect the persons who are at occupational risk.

Tuberculosis: Tuberculosis (TB) is one of the most widespread infectious diseases and leading cause of death due to single infectious agent among adults in the world. The tubercle bacilli that causes tuberculosis in man belong to the so-called *Mycobacterium tuberculosis* complex which falls under the genus – *Mycobacterium* and has four species namely, *M.tuberculosis, M. bovis, M. africanum* and *M. microti*. Bovine tuberculosis is a chronic bacterial zoonotic disease of cattle and easily spreads to humans through inhalation of aerosols or ingestion of unpasteurized infected milk. Amongst the members of *Mycobacterium tuberculosis* complex (MTBC), *M. tuberculosis* is mainly human pathogen, whereas *M. bovis* is the causative agent of bovine tuberculosis has exceptionally a wide host range and is the principal agent responsible for tuberculosis in domestic as well as wild animals. With 30% of total cases, India has the most TB patient in the world. It is the leading cause of death in India with a total of 4,21,000 deaths annually. About 1000 Indians die from TB per day i.e. one per min (Krishnaswami, 2000) and 95% of new TB cases are seen every year. There is a financial loss of 4-7% in GDP due to TB in Asian countries. TB costs more than Rs. 13,000 crores in India (ICMR Bulletin, 2002). The Office International des Epizooties has classified bovine tuberculosis as a List B disease, a disease which is considered to be of socio economic and public health important within countries and is of significance to the international trade of animals and animal products. Various animal species play an important role in the maintenance of *M. bovis* in wildlife communities and the spread to domestic animals. Badgers (*Meles meles*), brushtail possums (*Trichosurus vulpecula*), deer (*Odocoileus virginianus*), bison (*Bison bison*) and African buffalo (*Syncerus caffer*) are examples of wildlife that are maintenance hosts of *M. bovis*. The test and slaughter polices of tuberculosis control, efficiently used with livestock, are insufficient where wildlife reservoir exist. It will not be possible to eradicate *M. bovis* from livestock until transmission between wildlife and domestic animals

TB infected buffalo

halted. Better diagnostic tests for quick screening of this disease at field level should be developed and made available at the grass-root level (Sandhu, 2011). Control of tuberculosis in these animals is dependent upon the judicious use of diagnostic tests and the application of sound disease control principals. The advance in the development of tuberculosis vaccines for cattle and deer may offer valuable insight into the use of vaccination for control of tuberculosis in a range of captive wildlife species. Such an endeavor will require collaborative efforts between agriculture, wildlife, environmental and political interest. To eliminate the potential zoonotic sources of TB, pasteurization of milk before marketing and organized goat/sheep abattoirs should be made mandatory under law; where milk samples and carcasses can be routinely tested/examined for TB; and the cause of TB possibly traced to the infected herds. Vaccination of our livestock against TB and routine screening of livestock (e.g., on a yearly basis at the farms and also at the animal fairs) should be made mandatory (Sandhu, 2011).

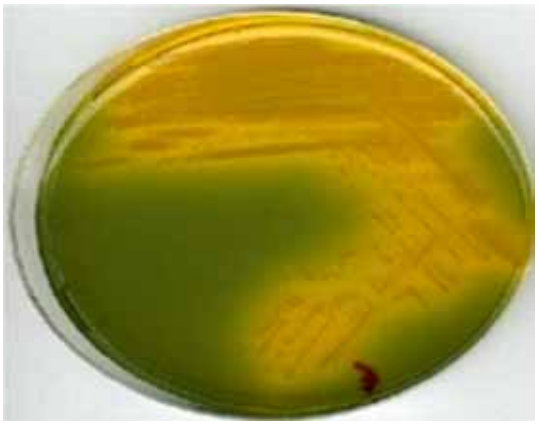
Toxoplasmosis: The disease is caused by *Toxoplasma gondii* (protozoa) and affects all warm blooded animals like cattle, buffalo, sheep, goat, pig, horse, camel, dog, cat, monkey, rodent and man. The principal host is cat in which the organism reproduces sexually and the cysts are shed in faeces of cat and spread the disease to other animals and man. The disease may be transmitted through contaminated food, undercooked meat and meat products, milk, water, inhalation, through semen and by transplacental routes. Congenital as well as acquired infection occurs in man. There may be abortion, premature birth,

hydrocephaly, microcephaly, hepatomegaly, spleenomegaly, fever, icterus, blindness, epileptic convulsion, cerebral calcification and mental retardation in congenital infection. In case of acquired infection, there may be pyrexia, lymphadenopathy, headache, stiff neck, pain in joints and meculopapular rash. The disease is more important in immunocompromised persons as the dormant cyst in the body of such individuals can alive again, may show cerebral illness and ultimately death. Toxoplasmosis is also not well studies in India even though numbers of abortions were reported in humans as well as in animals particularly in cats. There are many difficulties in toxoplasmosis diagnosis, because it is an asymptomatic infection, causing significant morbidity and mortality, for this reason the "diagnostic-space" is very important (Rodrigues *et al.,* 2009). The use of molecular biology techniques is useful in pregnant women with recent seroconversion, to exclude the presence of *T. gondii* DNA in amniotic fluid. In addition, the RT-PCR, for detection *T. gondii* DNA, also appears an easy, accurate and rapid diagnostic technique, whose application is also useful in cases of borderline serologic results (Pignanelli, 2011).

Foodborne infection

Vibrio cholera: This is one of the oldest recorded infectious diseases. The first six pandemics of cholera originated in Bengal and occurred between 1817 to 1923 and the seventh pandemic started in 1961 in Indonesia. The last decade of the 20th century witnessed a sharp increase in the global re-emergence of cholera. In September 1992, *V. cholerae* O139 Bengal (the first non-O1) appeared in south India. This spread to the rest of India and Bangladesh. Subsequent outbreaks due to this organism were reported in Nepal, Pakistan, southeast Asian countries and Japan. More than 200 serogroups (O1–O200) exist (Chatterjee and Chaudhuri, 2004), but only toxigenic strains of the serogroups O1 and O139 cause cholera (Nelson *et al.,* 2009). The genome of *V. cholerae* is in a state of constant flux, resulting in the reemergence and displacement of serotypes Inaba and Ogawa. Several biotic and abiotic factors, viz., salinity, temperature, rainfall and plankton, have been proven as important factors in the ecology of *V. cholerae* that influence the transmission of the disease (Huq *et al.,* 2005; Constantin de Magny *et al.,* 2008). There is a vast diversity of *V. cholerae* isolated in Delhi during 1992–2000 (Das and Gupta, 2005) and also a changing patterns of *V. cholerae* in Sevagram, Maharashtra seen, between 1990–2005 (Narang *et al.,* 2008). Cholera is endemic in northern and Eastern India, which includes Punjab, Haryana, Chandigarh and West Bengal. It has a freshwater environment with salinity close to zero and subtropical climate conditions. This region experiences hot, humid summers and chilly winters. The annual rainfall is moderate (<1000 mm). Cholera occurs in certain pockets of this region in seasonal outbreaks. The disease is on the rise in this part of India, with frequent sporadic cases and outbreaks (Taneja *et al.,*

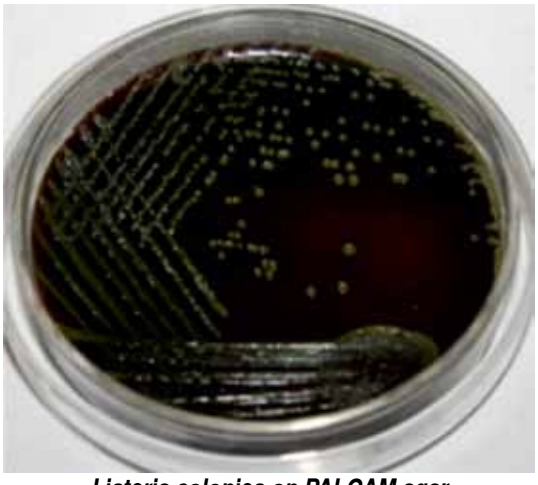
2009), particularly with a peak only during the monsoon months. The ecology of *V. cholerae* in freshwater environs is poorly understood, making it difficult to gauge the exact incidence of disease.



Colonies of Vibrio on TCBS medium

Antimirobial sensitivity test of the Vibrio spp.

Listeria monocytogenes: Listerosis is an emerging zoonotic disease. It is estimated that *L. monocytogenes* is responsible for 28% deaths due to food borne illnesses in the United States. The organism is unbiquitous and inherently robust and can thus survive food-processing and refrigeration of contaminated meat and dairy products. Although 13 serotypes have been identified, only 1/2a, 1/2b, and 4b are frequently isolated from clinical samples, with serotype 4b causing by far the most cases of human listeriosis (Raybourne, 2002; Borucki, and Call, 2003). However, serotype 1/2a is the most prevalent serotype in food. The occurrence of listeric infections in the Indian subcontinent has been extensively reviewed (Malik *et al.*, 2002). Listeriolysin O based ELISA and interferon-g assay has been developed for



Listeria colonies on PALCAM agar

CAMP test for detection of L. monocytogenes

diagnosis of listeriosis in animals (Barbuddhe *et al.,* 1999 & 1998). *L. monocytogenes* has been isolated from cases of mastitis, reproductive disorders and septicaemia in animals (Shakuntala *et al.,* 2006; Rawool *et al.,* 2007). Carriage of the pathogen has been reported in feces and genital tract of 5-10% humans.

***Campylobacter* spp.:** This is a significant zoonotic poultry pathogen and leading cause of bacterial gastroenteritis. Poultry remain healthy carriers and transmit by fecal shedding. Around 2.5 million human infections are reported annually in the United States. The disease is more common in children and there is an emergence of fluorquinolone resistance. In developing countries it is reported in 5–20% in childhood diarrhoea. *C. jejuni* isolated from 13.5% of the diarrhoea patients and this was more frequent than combined *Salmonella* and *Shigella* infections (4.3%; $P < 0.001$) (Jain *et al.,* 2005). Campylobacter jejuni has now become the most frequently reported organism from cases of bacterial gastroenteritis in humans in many countries (Crushell *et al .,* 2004, Iovine, 2008). In India, it was reported from all parts of the country.

***Enterohemorrhagic Escherichia coli* (EHEC):** In last two decades, Enterohaemorrhagic *Escherichia coli* (EHEC) have emerged as an important foodborne enteropathogen of humans which often leads to bloody diarrhea, and occasionally to kidney failure. *Escherichia coli* O157:H7 was first recognized as a cause of human illness in two separate outbreaks of hemorrhagic colitis in Michigan and Oregon in 1982 (Riley *et al.,* 1983). The organisms were transmitted in both cases by undercooked beef. Increasing numbers of diseases related to *E. coli* O157:H7 have been reported since 1982, most have been sporadic (Ostroff *et al.,* 1989) but many institutional and community-wide outbreaks (Swerdlow *et al.,* 1992) have occurred in nursing homes, schools and day care centers or have been related to eating at fast food restaurants (Bell *et al.,* 1994), drinking untreated municipal water or fresh-pressed apple cider (Swerdlow *et al.,* 1992), or swimming

E. coli isolate on EMB agar

Antimirobial sensitivity test of the E. coli isolates

in lake water (Keene *et al.,* 1994). It is estimated that 0.6% to 2.4% of all cases of diarrhea and 15% to 36% of all cases of bloody diarrhea or hemorrhagic colitis are associated with *E. coli* O157:H7. In a nursing home outbreak, the estimated attack rates from both food-borne and person-to-person transmission were 33% among the nursing home residents and 13% among the staff. The attack rate was reported to be as high as 67% (42 of 63 persons) in a kindergarten outbreak involving unpasteurized milk.

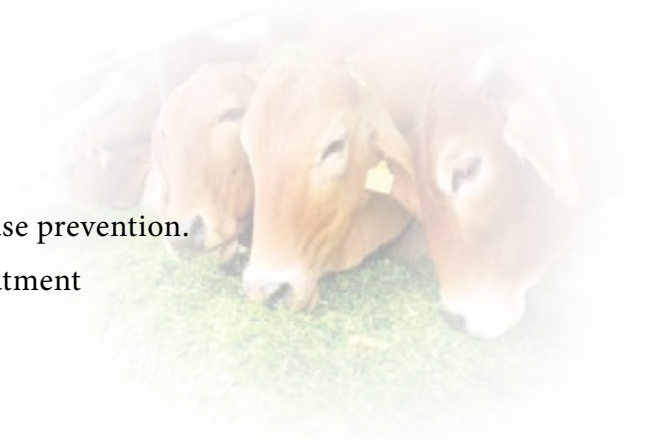
It causes no signs of illness in its natural host, cattle and sheep, but has a low infectious dose in humans where it causes haemorrhagic colitis and haemolytic uraemic syndrome. The manure-contaminated irrigation was the source of the largest recorded outbreak affecting more than 7000 children who had consumed contaminated sprouts in Japan. Non-O157 EHEC were reported in 1.4% of stools from cases of bloody diarrhoea in Kolkata (Ministry of Health and Welfare 2006). EHEC O157 sorbitol phenotype have been isolated from the Ganga River, Varanasi (Hamner *et al.,* 2007). The detection of potentially pathogenic O157:H7 in river water is alarming.

Prevention and control of zoonotic and foodborne infections

Veterinarians play an important role in prevention and control of zoonoses by virtue of their ability to destroy or treat the diseased animals and also controlling the movement of domestic animals. There are several factors involved in the causation and transmission of disease. Correct epidemiological investigation will help to identify these various factors in order to control them during outbreak. The basic approach in controlling or preventing a disease is to identify the weak linkages in the chain of their transmission. This requires sound knowledge of epidemiology of disease, i.e. its magnitude, spatial and temporal distribution, multifactorial causation, sources of infection and the dynamics of transmission.

Prevention: Prevention implies all measures taken to exclude a disease or to protect the animals and/or humans from acquiring an infection. Prevention can be done by following measures….

1. Quarantine
2. Immunization
3. Environmental hygiene
4. Chemoprophylaxis.
5. Education of people about disease prevention.
6. Early diagnosis and prompt treatment



Control: This is a strategy which employs all tactics useful for reducing the frequency of illnesses which are already present in a population or in other words measures to reduced the incidence of disease. It aims to reduce the morbidity and mortality caused by the disease. Control can be done by following measures….

1. Effective and early diagnosis and prompt treatment
2. Isolation of disease animals from healthy stock
3. Vaccination of healthy stock
4. Chemoprophylaxis including deworming
5. Awareness campaign
6. Separation/culling/slaughtering of disease animals or animals at risk

Approaches for prevention and control of disease:

Quarantine: Quarantine is the restraint placed upon the movement of animals, man, plants, or goods which are suspected of being carriers or vehicles of infections or of having been exposed to infectious agent(s). Quarantine may be international, interstate or local. Office Internationale de Epizootics (OIE) was established in Paris in 1924 with a view to make uniform procedures for veterinary quarantine and develop appropriate regulations that are applicable throughout the world. The period of quarantine depends on the incubation period of the agent, the time taken for the infection to be confirmed, i.e., isolation and identification of the pathogen and the time taken for an infected animal to become infectious.

Test and Slaughter: If a disease is infectious, affected animals can be a source of infection to others. In such circumstances it may be economically and technically expedient to slaughter an ill minority of animals to protect a healthy majority. A recent outbreak of avian influenza in different states of India was tackled by depopulation of several poultry birds. Several animals in India are infected with brucella. However, in our country, cow slaughtered is banned. Therefore, there is test and segregation policy to be followed. It is a big challenge to our country to prevent the spreading of Brucella to other animals. The disease is highly contagious and dangerous.

Environmental hygiene: Implementation of farm hygiene practices improves the sanitary environment of animals. The practices include excrement treatment and disposal, ventilation, availability of clean water, pest control, improvement of housing and general cleanliness. Environmental hygiene plays an important role in control of mechanical vectors, for example, house flies, mosquitoes, ticks, mites, fleas and lice. Systematic antemortem and postmortem examination of slaughter animals has led to a substantial reduction in the risk of transmitting meat borne

pathogens. Disposal of carcasses in the fields needs to be checked out for preventing the spreading of the various disease. Deep buried or burn are the correct methods for disposal of carcasses as well as farm waste.

Mass immunization: Immunization reduces the number of susceptible in the population and thus augments herd immunity making the infection more difficult to spread. Immunization programme must be epidemiologically relevant, immunologically effective, operationally feasible and socially acceptable. The effective immunization program depends on several factors such as type of vaccine used, genotypic diversity of the pathogen in immunization area, appropriate storage facilities, proper dissemination of vaccine, acceptability by the people, mass awareness, availability of vaccine and so on.

Vector control: Vector control is very important aspect in prevention of disease from spreading. It has become extremely popular among the public health experts. The approach consists of activities concerned with environment management and source reduction, chemical control, biological control, genetic control, personal protection and health education.

a) ***Environmental measures:*** Measures to be applied for environmental management are source reduction, filling and drainage operations, planned water management, and proper disposal of refuse and other wastes.

b) ***Chemical measures:*** A wide range of insecticides belonging to organochlorine, organophosphate and carbamate groups are available for vector control. Indiscriminate use of insecticides results in the development of insecticide resistance by vectors.

1. Contact poisons:
   Natural : pyrethrum, rotenone, derris, mineral oils
   Synthetic : organochlorine - DDT, HCH, lindane
   organophosphates - chlorthion, dichlorvos, parathion.
   carbamates - carbaryl, dimetilan
2. Stomach poisons : paris green, sodium fluoride
3. Fumigants : hydrogen cyanide, methyle bromide, sulphur dioxide

c) ***Biological measures:*** (i) Fish : Several species of fish such as *Gambusia affinis*, *Aplochilus panchax*, *Paecelia holbrooki* are effective as predators of anopheline (ii) Fungi: Extremely studied fungi is *Coelomomyces* (iii) Protozoa : *Nosema algerae* , *Thelohania,* and *Vorticella (*iv) Bacteria : *Bacillus thuringiensis*, *B. sphaericus.*

d) ***Genetic manipulation of insect vectors:*** Transgenic technology, controlled manipulation of genome of an insect by the direct introduction of DNA into the germ line offer a challenging potential to exploit genes and gene contents across species barriers and the ability to introduce particular sequences without the genome disruption caused by conventional cross-breeding. Transgenic technology may be useful in controlling vector borne diseases for example, by inducing insecticide susceptibility and temperature susceptibility.

Reservoir control: Reservoir is defined as "any person, animal or non-living thing in which infectious agent lives and multiplies and can be transmitted to a susceptible host". For e.g. foxes and doges are main reservoir for rabies. It is helpful against rats, stray dogs and other noxious reservoir hosts of infections such as leptospirosis, plague, typhus and rabies. Poison baiting and trapping have been among the most commonly employed techniques against reservoir hosts. Anti-rodent measures include environmental sanitation, use of rodenticides and fumigation.

Early diagnosis: In veterinary medicine the techniques of early detection have been successfully applied in the diagnosis of tuberculosis, brucellosis, mastitis, glanders and salmonellosis. Early diagnosis of disease is very important and necessary for immediate treatment and control measures over the disease. Some diseases are difficult to diagnose because of common flu like symptoms for e.g. many viral infections. Application of tuberculin skin test in domestic animals and human population, and mallein test for glanders are classic examples in this regard. The development of various immunological and molecular techniques such as enzyme linked immunosorbent assay (ELISA), radio immunoassay (RIA), immunofluorescence (IF), restriction fragment length polymorphism (RFLP), sequence analysis, DNA probes and polymerase chain reaction (PCR) has revolutionized diagnostic procedures with their wide applicability.

Treatment: Mass treatment of an affected population may be carried out under an emergency or when the disease prevalence is very high. Mass treatment is given either prophylactically or curatively. Use of coccidiostats to poultry in drinking water and routine incorporation of anthelmintics in the ruminant salt licks or feeds are some of the examples of mass treatment.

Genetic improvement: It has been shown that the incidence of some infectious diseases can be reduced by selective breeding. For example, certain breeds of cattle in tsetse zone of Africa are known to be tolerant to trypanosomosis. In general, the local breeds of animals are resistant to various common diseases while the exotic breeds like HF and Jersy are more prone to infections. Therefore, development of new animal breeds particularly resistant to various disease and high milk/meat producing are essential.

Health education: The health education envisages making community aware of the cause and mode of disease transmission, prevention and treatment of disease, and the role of community in combating diseases. Health education through mass media such as newspapers, radio, cinema, wall slogans, television can be very effective.

Epidemiological diagnosis: In epidemiological diagnosis the frequency of the population event (disease), its time and place pattern of occurrence and associated factors or circumstances are ascertained. The primary purpose of epidemiological diagnosis is to determine immediate and long term needs for purposeful action against the disease. Molecular epidemiology that relates to the use of molecular techniques namely RFLP, PCR, and DNA probes is a promising field for interactions between epidemiology and the laboratory.

References:

Alexander A.D. (1991). Leptospira. In: Balows, A., W.J. Hausler, K.L. Hermann, H.D. Isenberg, S.J. Shadomy. Eds. *Manual of Clinical Microbiology.* 5th Edn. Washington D.C.: American Society of Microbiology; 1991.

Anukumar B., Amirthalingam B.G., Shelke V.N., Gunjikar R. and Shewale P. (2013). Neuro-invasion of Chandipura virus mediates pathogenesis in experimentally infected mice. *Int J Clin Exp Pathol.* 6(7): 1272-1281.

Arankalle V.A., Bandyopadhyay B.T., Ramdasi A.Y., Jadi R., Patil D.R., Rahman M., Majumdar M., Banerjee, P.S., Hati A.K., Goswami R.P., Neogi D.K. and Mishra A.C. (2011). Genomic Characterization of Nipah Virus, West Bengal, India. *Emerging Infectious Disease* 17(5): 907-909.

Athar M.N., Baqai H.Z., Ahmad M., Khalid M.A., Bashir N., Ahmad A.M., et al. (2003). Short report: Crimean Congo hemorrhagic fever outbreak in Rawalpindi, Pakistan, February 2002. *Am J Trop Med Hyg* 69: 284-287.

Banerjee K. (1996). Emerging viral infections with special reference to India. *Indian J Med Res* 103: 177-200.

Bansal 2004: www.japi.org/january2004/Correspondence.pdf

Barbuddhe S.B., Malik S.V. and Gupta L.K. (1998). Effect of *in vitro* monocyte activation by *Listeria monocytogenes* antigens on phagocytosis and production of reactive oxygen and nitrogen radicals in bovines. *Vet Immunol Immunopathol.* 64(2):149-59.

Barbuddhe, S.B. Malik, S.V.S. and Prahlad Kumar (1999). High seropositivity against anti-listeriolysin O in humans. *Annals of Trop. Med. and Parasitol.* 93: 537-539.

Bell B.P., Goldoft M., Griffin P.M., Davis M.A., Gordon D.C., Tarr P.I., Bartleson C.A., Lewis J.H., Barrett T.J., Wells J.G., et al. (1994). A multistate outbreak of *Escherichia coli* O157:H7-associated bloody diarrhea and hemolytic uremic syndrome from hamburgers. The Washington experience. J.A.M.A. 272(17): 1349-1353.

Bhanuprakash V., Venkatesan G., Balamurugan V., Hosamani M., Yogisharadhya R., Gandhale P., Reddy K.V., Damle A.S., Kher H.N., Chandel B.S., Chauhan H.C., Singh R.K. (2010). Zoonotic Infections of Buffalopox in India. Zoonoses and Pub. Helth. 57(7-8): 149–e155.

Bhatt, P.N. and Rodrigues F.M. (1967). Chandipura: A new Arbovirus isolated in India from patients with febrile illness. *Indian J. Medical Res.* 55: 1295-1305.

Borucki M.K. and Call D.R. (2003). *Listeria monocytogenes* serotype identification by PCR. *J Clin Microbiol.* 41(12): 5537-5540.

Burke, D.S., Nisalak, A. and Nimmannitya, S. (1985). Disappearance of Chikungunya virus from Bangkok. *Trans R Soc Trop Med Hyg* 79: 419–420.

Calisher C.H. and Goodpasture H.C. (1975). Human infection with Bhanja virus. *Am. J. Trop. Med. Hyg.* 24: 1040–1042.

Chadha M.S., Arankalle V.A., Jadi R.S., Joshi M.V., Thakare J.P., Mahadev P.V. and Mishra A.C. (2005). An outbreak of Chandipura virus encephalitis in the eastern districts of Gujarat state, India. Am. J. Trop. Med. Hyg. 73(3): 566-570.

Chatterjee S.N. and Chaudhuri K. (2004). Lipopolysaccharides of *Vibrio cholerae* II. Genetics of biosynthesis. *Biochim. Biophys. Acta.* 1690(2): 93-109.

Chhabra M., Mittal V., Bhattacharya D., Rana U. and Lal S. (2008). Chikungunya fever: a re-emerging viral infection. *Indian J. Med. Microbiol.* 26(1): 5-12.

Chomel B.B., Belotto A. and Meslin F.X. (2007). Wildlife, Exotic Pets, and Emerging Zoonoses. *Emerg Infect Dis.* 13(1): 6–11.

Constantin de Magny G., Murtugudde R., Sapiano M.R., Nizam A., Brown C.W., Busalacchi A.J., Yunus M., Nair G.B. *et al.* (2008). Environmental signatures associated with cholera epidemics. *Proc. Natl. Acad. Sci. USA* 105**:** 17676–17681.

Crushell E., Sinead H., Sharif F. and Bourke B. (2004). Enteric Camplylobacter: Purging its secrets? Review. *Pediatr. Res.* 55(1).

Dandawate C.N., Work T.H., Webb J.K. and Shah K.V. (1969). Isolation of Ganjam virus from a human case of febrile illness: a report of a laboratory infection and serological survey of human sera from three different states of India. *Indian J. Med. Res.* 57: 975-982.

Das S. and Gupta S. (2005). Diversity of *Vibrio cholerae* strains isolated in Delhi, India during 1992-2000. *J. Health. Popul. Nutr.* 23: 44-51.

Das B., Swain S., Patra A., Das M., Tripathy H.K., Mohapatra N., Kar S.K. and Hazra R.K. (2012). Development and evaluation of a single-step multiplex PCR to differentiate the aquatic stages of morphologically similar Aedes (subgenus: Stegomyia) species. *Trop. Med. International Helth*. 17(2): 235–243.

Daszak P., Cunningham A.A. and Hyatt A.D. (2001). Anthropogenic environmental change and the emergence of infectious diseases in wildlife. *Acta Trop.* 78:103–116.

Dhanda V., Rodrigues F.M. and Ghosh S.N. (1970). Isolation of Chandipura virus from sandflies in Aurangabad. *Indian J. Med. Res.* 58(2): 179-180.

Geevarghese G., Arankalle V.A., Jadi R., Kanojia P.C., Joshi M.V. and Mishra A.C. (2005). Detection of chandipura virus from sand flies in the genus Sergentomyia (*Diptera: Phlebotomidae*) at Karimnagar District, Andhra Pradesh, India. *J. Med. Entomol*. 42(3): 495-496.

Ghosh T.K. (2006). Rabies. Proceedings of the IX National Conference of Pediatric Infectious Diseases; 2006; Chennai, India.

Gupta M.L. and Sharma A. (2007). Pneumonic Plague, Northern India, 2002. *Emerg. Infect. Dis.* 13(4): 664–666.

Gurav Y.K., Raut C.G., Yadav P.D., Tandale B.V., Sivaram A., Pore M.D., Basu A., Maurya D.T. and Mishra A.C. (2011). Buffalopox outbreak in humans and animals in Western Maharashtra, India. *Prev. Vet. Med.* 100 (3-4): 242-247.

Hamner S., Broadaway S.C., Mishra V.B., Tripathi A., Mishra R.K., Pulcini E., Pyle B.H. and Ford T.E. (2007). Isolation of potentially pathogenic *Escherichia coli* O157:H7 from the Ganges River. *Appl. Environ. Microbiol.* 73(7): 2369–2372.

Hubalek Z. (1987). Geographic distribution of Bhanja virus. *Folio Parasitologica* 37: 77-86.

Huq A., Sack R.B., Nizam A., Longini I.M., Nair G.B., Ali A., Morris J.G., Khan M.N.H., Siddique A.K., Yunus M., Albert M.J., Sack D.A. and Colwell R. R. (2005). Critical factors influencing the occurrence of *Vibrio cholerae* in the environment of Bangladesh. *Appl. Environ. Microbiol.* 71: 4645–4654.

Ichhpujani R.L., Rajagopal V., Bhattacharya D., Rana U.V., Mittal V., Rai A., Ravishankar A.G., Pasha S.T. *et al* (2004). An outbreak of human anthrax in Mysore (India); *J. Commun. Dis.* 36 : 199–204.

Iovine N.M., Pursnani S., Voldman A., Wasserman G., Blaser M.J. and Weinrauch Y. (2008). Reactive nitrogen species contribute to innate host defense against *Campylobacter jejuni*. *Infect. Immun*. 76: 986-993.

Jain D., Sinha S., Prasad K.N. and Pandey C.M. (2005). *Campylobacter* species and drug resistance in a north Indian rural community. *Trans. R. Soc. Trop. Med. Hyg.* 99: 207–214.

Jena A.B., Mohanty K.C. and Devadasan N. (2004). An outbreak of leptospirosis in Orissa, India: the importance of surveillance. *Trop. Med. & International Helth* 9(9): 1016–1021.

Joshi M.V., Elankumaran S., Joshi G.D., Albert A., Padbidri V.S., Murali Manohar B., *et al.* (1998). Post-epizootic survey of rift valley fever-like illness among sheep at Veerapuram, Chennai, Tamil Nadu. *Indian J. Virol.* 14: 155-157.

Keene W.E., McAnulty J.M., Hoesley F.C., Williams L.P., Hedberg K., Oxman G.L., Barrett T.J., Pfaller M.A. and Fleming D.W. (1994). A swimming-associated outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7 and *Shigella sonnei*. *N. Engl. J. Med.* 331: 579–584.

Krishnaswamy G.M., Chi D.S.P., Kelley J.L., Sarubbi F.M., Smith K.J..M. and Peiris A.M. (2000). The cardiovascular and metabolic complications of HIV infection. *Cardiology in Review.* 8(5): 260–268.

Kumar A., Kanungo R., Bhattacharya S., Badrinath S., Dutta T.K. and Swaminathan R.P. (2000). Human anthrax in India: urgent need for effective prevention. *J. Commun. Dis.* 32: 240–246.

Kumar S., Jaffar-Bandjee M.C., Giry C., de Kerillis L.C., Merits A., Gasque P. and Hoarau J.J. (2012). Mouse macrophage innate immune response to chikungunya virus infection. *Virol. J.* 9: 313.

Lubani M.M., Dudin K.I., Sharda D.C., AbuSinna N.M., Al-Shab T., Al-Refeai A.A., Labani S.M. and Nasrallah A. (1988). Neonatal brucellosis. *Eur. J. Pediatr.* 147:520–522.

Luby S.P., Rahman M., Hossain M.J. et al. (2006). Foodborne transmission of Nipah virus, Bangladesh. *Emerg Infect Dis.* 12: 1888–1894.

Madhusudana S.N., Mani R., Ashwin Y.B. and Desai A. (2013). Rabid fox bites and human rabies in a village community in southern India: epidemiological and laboratory investigations, management and follow-up. *Vector Borne Zoonotic Dis*. 13(5):324-329.

Malik S.V.S., Barbuddhe S.B. and Chaudhari S.P. (2002). Listeric infections in humans and animals in the Indian subcontinent: a review. *Trop. Anim. Health Prod.* 34(5): 359-381.

Mantur B.G., Mangalgi S.S. and Mulimani M. (1996). *Brucella melitensis* - a sexually transmissible agent?; *Lancet.* 347: 1763[Erratum in: Lancet; 348:970]

Marczinke B.I. and Nichol S.T. (2002). Nairobi sheep disease virus, an important tick-borne pathogen of sheep and goats in Africa, is also present in Asia. *Virol*. 303: 146-151.

Marriott A.C., Ward V.K., Higgs S. and Nuttall P.A. (1990). RNA probes detect nucleotide sequence homology between members of two different nairovirus serogroups. *Virus Res* 16: 77-81.

Mathur M., De A. and Turbadkar D. (2009). Leptospirosis outbreak in 2005: LTMG hospital experience. *Indian J. Med. Microbiol.* 27: 153-155.

Monath T.P., Heinz FX*Flaviviridae* In: Fields B.N., Knipe D.M., Howley P.M., editors. Fields virology. 3rd ed. Philadelphia: Lippincott–Raven; 1996. p. 961–1034.

Naparstek E., Block C.S. and Slavin S. (1982). Transmission of brucellosis by bone marrow transplantation; *Lancet* 1: 574–575.

Narang P., Mendiratta D.K., Deotale V.S. and Narang R. (2008). Changing patterns of *Vibrio cholerae* in Sevagram between 1990 and 2005. *Indian J. Med. Microbiol.* 26: 40–44.

Nelson E.J., Tanudra A., Chaudhury A., Kane A.V., Qadri F., Calderwood S.B., Coburn J. and Camilli A. (2009). High prevalence of spirochetosis in cholera patients, Bangladesh. *Emerg. Infect. Dis.* 15: 571-573.

Ostroff S.M., Tarr P.I., Neill M.A., Lewis J.H., Bean N.H. and Kobayashi J.M. (1989). Toxin genotypes and plasmid profiles as determinants of systemic sequelae in *Escherichia coli* 0157:H7 infections. *The J. of Infect. Dis.* 160(6): 994-998.

Parida ,M., Dash ,P.K., Tripathi, N.K., Ambuj, Sannarangaiah ,S., Saxena ,P., Agarwal ,S., Sahani ,A.K., Singh ,S.P., Rathi ,A.K. ,Bhargava ,R., Abhyankar ,A., Verma ,S.K., Rao ,P.V. and Shekhar, K. (2006). Japanese Encephalitis Outbreak, India, 2005. *Emerg Infect. Dis.,* 12: 1427–1430.

Pavri K.M. (1964). Presence of chikungunya antibodies in human sera collected from Calcutta and Jamshedpur before 1963. *Indian J. Med. Res.* 52: 698–702.

Pignanelli S. (2011). Laboratory diagnosis of *Toxoplasma gondii* infection with direct and indirect diagnostic techniques. *Indian J. of Pathol. and Microbiol.* 54(4): 786-789.

Powers A.M. and Logue C.H. (2007). Changing patterns of chikungunya virus: re-emergence of a zoonotic arbovirus. *J Gen Virol.* 88(Pt 9): 2363-2377.

Rao B.L., Basu A., Wairagkar N.S., Gore M.M., Arankalle V.A., et al. (2004) A large outbreak of acute encephalitis with high fatality rate in children in Andhra Pradesh, India, in 2003, associated with Chandipura virus. *Lancet* 364: 869–874.

Rawool D.B., Malik S.V.S., Shakuntala I., Sahare A.M. and Barbuddhe S.B. (2007). Detection of multiple virulence associated genes in pathogenic *Listeria monocytogenes* from bovines with mastitis. *International J. of Food Microbiol.* 113(2): 201-207.

Raybourne R.B. (2002). Virulence testing of *Listeria monocytogenes*. Journal of AOAC International 85: 516–523.

Riley L.W., Remis R.S., Helgerson S.D., McGee H.B., Wells J.G., Davis B.R., Hebert R.J., Olcott E.S., Johnson L.M., Hargrett N.T., Blake P.A. and Cohen M.L. (1983). Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. *New England J. Med.* 308(12): 681-685.

Robinson M.C. (1955). An epidemic of virus disease in Southern Province, Tanganyika Territory, in 1952-53 I: Clinical features. *Trans. R. Soc. Trop. Med. Hyg.* 49: 28-32.

Rodrigues J.J., Singh P.B., Dave D.S., Prasan R., Ayachit V., et al. (1983) Isolation of Chandipura virus from the blood in acute encephalopathy syndrome. *Indian J. Med. Res.* 77: 303–307.

Rodrigues I.M., Castro A.M., Gomes M.B., Amaral W.N. and Avelino, M.M. (2009). Congenital toxoplasmosis: evaluation of serological methods for the detection of anti-*Toxoplasma gondii* IgM and IgA antibodies. *Mem. Inst. Oswaldo. Cruz.* 104: 434-440.

Ross R.W. (1956). The Newala epidemic. *III*. The virus: isolation, pathogenic properties and relationship to the epidemic. *J Hyg (Lond)* 54: 177–191.

Sandhu G.K. (2011). Tuberculosis: Current situation, challenges and overview of its control programs in India. *J Glob Infect Dis.* 3(2): 143–150.

Sarada D., Valentina G.O. and Lalitha M.K. (1999). Cutaneous anthrax involving the eyelids; *Indian J. Med. Microbiol.* 17: 92–95.

Sarkar J.K., Chatterjee S.N., Chakravarti S.K. and Mitra A.C. (1965). Chikungunya virus infection with haemorrhagic manifestations. *Indian J. Med. Res.* 53, 921–925.

Shakuntala I., Malik S.V.S., Barbuddhe S.B. and Rawool D.B. (2006). Isolation of *Listeria monocytogenes* from buffaloes with reproductive disorders and its confirmation by polymerase chain reaction. *Vet. Microbiol.* 117: 229-234.

Simizu B, Yamamoto K, Hashimoto K, Ogata T. 1984. Structural proteins of Chikungunya virus. *J. Virol.* 51:254–8.

Singh R.K., Hosamani M., Balamurugan V., Sathesh C.C., Shingal K.R., Tatwarti S.B., Bambal R.G., Ramteke V. and Yadav M.P. (2006). An outbreak of buffalo pox in buffalo (Bubalus bubalis) dairy herd in Aurangabad, India. *Rev. Sci. Tech.* 25: 981-987.

Solomon T., Ni H, Beasley D.W.C., Ekkelenkamp M., Cardosa M.J. and Barret A.D. (2003). Origin and evolution of Japanese encephalitis virus in Southeast Asia. *J Virol.* 77: 3091–3098.

Sudarshan M.K., Bhardwaj S., Mahendra B.J., Sharma H., Sanjay T.V., Ashwathnarayana D.H. and Bilagumba G. (2008). An immunogenicity, safety and post-marketing surveillance of a novel adsorbed human diploid cell rabies vaccine (Rabivax) in Indian subjects. *Hum Vaccin.* 4(4):275-9.

Swerdlow D.L., Woodruff B.A., Brady R.C., Griffin P.M., Tippen S., Donnell H.D. Jr, Geldreich E., Payne B.J., Meyer A. Jr., Wells J.G., et al. (1992). A waterborne outbreak in Missouri of *Escherichia coli* O157:H7 associated with bloody diarrhoea and death. *Ann. Intern. Med.,* 117: 812-819.

Taneja N., Mishra A., Sangar G., Singh G. and Sharma M. (2009). Outbreaks caused by new variants of *Vibrio cholerae* O1 El Tor, India. *Emerg. Infect. Dis.* 15: 352–354.

Taylor L.H., Latham S.M. and Woolhouse M.E. (2001). Risk factors for human disease emergence. *Philos Trans R Soc Lond B Biol Sci.* 356: 983–989.

Tikare N.V., Mantur B.G. and Bidari L.H. (2008). Brucellar meningitis in an infant – evidence for human breast milk transmission; *J. Trop. Pediatr.* 54: 272–274.

Varma M.G.R. (2000). The encyclopedia of arthropod-transmitted infections. New York: CABI Publishing; 200: 254-260.

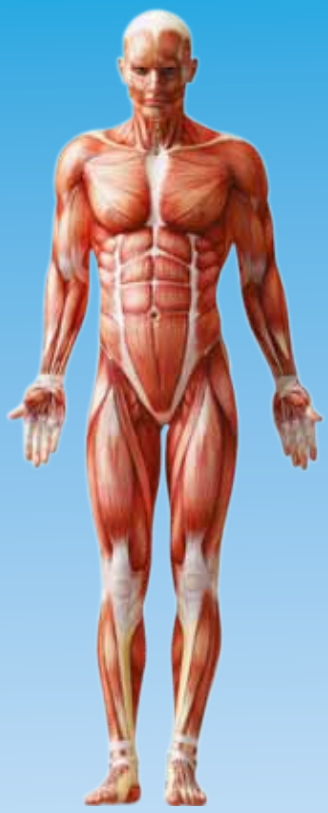
Vesenjak-Hirjan J. (1980). Arboviruses in Yugoslavia. In: J. Vesenjak-Hirjan et al. (Edn.) Arboviruses in the Mediterranean countries, G. Fischer, Stuttgart-New York: 165-177.

Weaver S.C., Frey T.K., Huang H.V., Kinney R.M., Rice C.M., Roehrig J.T., Shope R.E. and Strauss E.G. (2005). *Togaviridae*. In Virus Taxonomy: Eighth Report of the International Committee on Taxonomy of Viruses, pp. 999–1008. Edited by C. M. Fauquet, M. A. Mayo, J. Maniloff, U. Desselberger & L. A. Ball. Amsterdam: Elsevier Academic Press.

World Health Organization Outbreak encephalitis 2005: cases of Japanese encephalitis in Gorakhpur, Uttar Pradesh, India. 2005. Core Programme Clusters. Communicable Diseases and Disease Surveillance. 2005 Oct 21 [cited 2006 Jul 11]. Available from http://w3.whosea.org/en/Section1226/Section2073.asp.

World Health Organization (2006). Zoonotic diseases of public health importance (Zoonotic Division, National Institute of Communicable Diseases, New Delhi, India) pp 34–46

Zafar A., Swanepoel R., Hewson R., Nizam M., Ahmad A., Husain A., Grobbelaar A., Bewley K., Mioulet V., Dowsett B., Easterbrook L. and Hasan P. (2007). Nosocomial buffalo pox virus infection, Karachi, Pakistan. *Emerg. Infect. Dis.* 13(6): 902-904.



भाकृअनुप
ICAR

ICAR RESEARCH COMPLEX FOR GOA

(Indian Council of Agricultural Research)

Old Goa - 403 402, Goa, India