ABSTRACT
Zoonoses have a worldwide distribution and can cause severe disease which clinicians fail to recognize. Zoonotic diseases are responsible for a large burden on the public health, livestock economies, and wildlife of India. Moreover, there is paucity of authentic data regarding occurrence of these diseases and their true impact on public health. Understanding the distribution of common zoonotic diseases, their modes of transmission to humans, how to make a clinical & laboratory diagnosis of them and how to preventive them would be of utmost importance specially in India where 68 % of the work force is in close contact with domestic animals and where unhygienic living conditions, lack of education, poor personal hygiene, poor veterinary and public health services, poverty and malnourishment contribute to dissemination of these diseases.

Keywords: Zoonotic Diseases, Mode of Transmission, Clinical Diagnosis, Lab Diagnosis, Prevention.

INTRODUCTION
Zoonotic diseases are among the most frequent and dreaded risk to which mankind is exposed. Zoonoses occur throughout the world transcending the natural boundaries (Ichhpujani et al., 2000). Zoonotic diseases are of great public health importance in India, where 68% of the workforce relies on farming that is in close contact with domestic animals and poultry with frequent exposure to sick or infected animals. Unhygienic living conditions, lack of education, poor personal hygiene, poor veterinary and public health services, poverty and malnourishment contribute to the dissemination of these zoonoses. Zoonotic diseases are responsible for a large burden on the public health, livestock economies, and wildlife of India (Knobel et al., 2005). A number of zoonoses also cause severe disease and medical clinicians who encounter zoonoses in human patients may either fail to recognize them or concentrate on treating the individual patient rather than thinking of disease control. Morbidity estimates of many zoonoses are unknown. Lack of authentic data and awareness regarding the occurrence of these diseases and their true impact on public health have acted as major obstacles in commencing adequate and effective control measures (Asokan et al., 2011). Therefore it is important to have a thorough knowledge about the common zoonotic diseases, their global morbidity estimates, their common modes of transmission to humans, clinical and laboratory diagnosis and preventive measures.

Common Zoonotic Diseases in India

Anthrax: is caused by the bacterium Bacillus anthracis. Humans acquire infection from cattle, sheep, goats, horses and swine. Worldwide this disease is more prevalent in Africa, Asia, South America and Eastern Europe (James et al., 2012). It is enzootic in India. Anthrax in sheep is prevalent in sheep in Andhra – Tamilnadu border causing cutaneous and meningoencephalitic human infections with a high mortality rate. Outbreaks in Karnataka and west bengal have been seen (Baveja, 2012). There are three clinical types of disease based on the route of infection. Cutaneous anthrax follows entry of spores through abraded skin; the typical lesions are pustules which are more commonly seen on face, neck, hands and back. Pulmonary anthrax occurs due to inhalation of dust of wool characterized by haemorrhagic bronchopneumonia. Intestinal anthrax is transmitted by ingestion of improperly cooked infected meat causes violent enteritis with bloody diarrhea. Any type of anthrax if not treated in time lead to sepsicaemia and death. Swabs, fluid or pus from pustules in cutaneous anthrax, sputum from
Review Article

Plague: Plague is a deadly infectious disease that is caused by the enterobacteria Yersinia pestis, (Pawar KR et al., 2011). Plague is an ancient disease which caused three pandemics since the 6th century but the global transmission has been low in recent years (Madkour et al., 2001). In India, there had been fatal outbreaks in 1994 and 2002 in Maharashtra and Simla. There are four foci of plague in India. Kolar district of Karnataka, beed-latur belt in Maharashtra, Rhoru in Himachal Pradesh and a small pocket in Uttaranchal.(Ananthnarayan et al., ).It is a bacterial zoonosis with rodents being the principle reservoir. The black rat (Rattus rattus) and oriental rat fl flea (Xenopsylla cheopis) are notorious reservoir and transmitting agent for human plague in India (Seal SC et al., 1969). Transmission of Y pestis to an uninfected individual is possible by droplet contact,direct physical contact including sexual contact, by soil contamination, airborne transmission, fecal-oral transmission and vector borne transmission carried by insects or other animals (Plague Manual., 1988). Human infection with Y pestis usually manifests as bubonic, septicemic, or pneumonic plague. The clinical symptoms of bubonic plague include fever, chills, weakness, headache, and swollen, tender lymph nodes (buboes). Which lymph nodes are involved depends on the site of exposure; however, buboes more commonly are found in the inguinal and femoral regions (Perry et al., 1997). Onset of lymphadenopathy is rapid. Marked edema, swelling, and inflammation of tissues overlying the buboes are frequently seen (Smego et al., 1999). The buboes usually are extremely tender. Diarrhea, nausea, and vomiting also are frequent manifestations (Hull et al., 1983 & Von et al., 1977). Primary pneumonic plague is uncommon and results from inhalation of
organisms, usually via respiratory droplets from infected individuals or animals or, in rare circumstances, owing to accidental inhalation of organisms in laboratory and research facilities (Franz et al., 1997). The initial symptoms are flu-like, with rapid progression to pneumonia with bloody, watery sputum production. Symptoms begin approximately 2 to 8 days after exposure. Bacteremia or secondary plague septicemia is a frequent occurrence, and the fatality rate is higher in patients with higher colony counts. Patients with primary septicemic plague have positive blood cultures but lack lymphadenopathy or pneumonia (Perry et al., 1997 & Smego et al., 1999). Plague septicemia is clinically similar to other gram-negative bacterial septicemias and has a 30% to 50% mortality rate, even with antibiotic administration (Crook et al., 1992 & Hull et al., 1988). Disseminated intravascular coagulopathy, meningitis, and multi organ failure are common. Secondary septicemic plague occurs commonly during bubonic and primary pneumonic plague (Craven et al., 1992.). Laboratory confirmation of plague should be sought as quickly as possible so that appropriate therapy is given. The definitive diagnosis of plague requires culture of Y pestis from a clinical specimen of a fourfold rise in antibody titre. Aspiration from a bubo is the material collected and the smears are subjected to Wayson, giemsa or gram stains. Bipolar staining coccobacilli can be demonstrated with rapid identification by immunofluorescence using F1 specific antibodies labeled with fluorescein. Blood, sputum and cerebrospinal fluid also are processed in the same way as bubo aspirates. Culture of the samples is done on Bloodagar and Macconkey agar. Serological diagnosis is useful in culture negative cases. Haemagglutinating antibodies to F1 antigen appear after 1 week and may be detected by passive agglutination test. A single titre of more than or =16 is very suggestive of plague, whereas a fourfold rise in paired sera is diagnostic. EIA have been applied in the detection of serum antibody and f1 antigenaemia, both appear to be useful diagnostic tests. A significant recent advance in the diagnosis of plague has been the development of a hand-held, immunochromatographic test, which detects f1 antigen in a range of sample types, with good sensitivity and specificity. It serves as a rapid bedside test even in remote areas that do not have access to other means of diagnosis. Several methods of DNA detection using PCR have been developed recently but the diagnostic value of PCR on bubo aspirates found that although very specific, it is not as sensitive as culture or F1 antigen detection (Madkour et al., 2001). The control measures require early clinical suspicion, prompt laboratory diagnosis, strict isolation of patients of pneumonic plague, appropriate antibiotic therapy and case contact management. A vaccine is available for persons who have repeated contact with the pathogen. But the best plague prevention strategy is an aggressive rodent population control (Seal SC et al., 1969).

**Leptospirosis** -Leptospirosis is an emerging global public health problem. The disease is caused by Leptospira interrogans naturally seen in rodents. International Leptospirosis Society conducted three recent worldwide surveys and estimates about half a million severe cases of the disease occur worldwide annually. Seroepidemiologic and clinical studies show that the disease is endemic in Andaman Islands and southern states of India (Loganathan N et al.,2012).The seroprevalence is reported to be high (52.7%) among high-risk population in Andaman Islands and 19.8% and 9.3% in Madras and Bangalore respectively. (Seal SC et al., 1969). The disease is endemic in Kerala Tamilnadu, Gujrat, Andamans, Karnataka, and Maharashtra. It has also been reported from Andhra Pradesh, Orissa, West Bengal, Uttar Pradesh, and Delhi & Puducherry. Rodents, domestic & wild animals form the reservoir of infection where domestic animals such as cattle, dogs, and pigs may act as carriers for several months (temporary carrier) while rodents usually remain carrier throughout their life (permanent carrier). Leptospires are excreted in the urine of the animals and they affect man when he comes into contact with urine of infected animals, directly or indirectly, when he is exposed to an environment contaminated by the urine of the infected animals such as soil and surface water following monsoon rains. Leptospirosis can manifest in many ways. The various syndromes of presentation are acute febrile illness, weil’s syndrome characterized by jaundice, renal failure and myocarditis with cardiac arrhythmias, pulmonary haemorrhage with respiratory failure and meningitis / meningoencephalitis (Shivkumar et al., 2008). The usual presentation is an acute febrile illness with headache, myalgia (particularly calf muscle) and
prostration associated with any of the following symptoms/signs: conjunctival suffusion, anuria or oliguria, jaundice, cough, haemoptysis and breathlessness, haemorrhages from the intestines, meningeal irritation, cardiac arrhythmia or failure and skin rash (Heymann et al., 2004). Leptospirosis can be diagnosed only by laboratory tests as the clinical features are nonspecific. The tests depend on the phase of the infection. During leptospiremic phase (< 7 days) leptospires can be isolated by blood culture and PCR, while in the immune phase, rising antibodies can be detected by serological tests. The isolation of leptospirosis by culture of blood, CSF and urine is the most definite way of confirming the diagnosis of leptospirosis. The media used is EMJH medium. Culture of blood does not contribute to an early diagnosis as results come late, weeks or even months after inoculation of culture medium; however it is valuable in critically ill patients who might die in the first week before the development of antibodies. Direct culture of urine is seldom successful. PCR is promising on both sensitivity and specificity, but is complicated and expensive. Its value for rapid diagnosis is being evaluated and is used in higher centres. The serological tests for diagnosis of leptospirosis are classified as serovar specific tests and genus specific tests. Microscopic Agglutination Test (MAT) is a serovar specific test, which is the gold standard test for diagnosis of leptospirosis because of its unsurpassed diagnostic specificity. The main advantage is that serovars can be identified which is of epidemiological importance. But this is a less sensitive test and needs a dark ground microscope and cultures of various live serovars and may not be available in small laboratories. The commonly used genus specific tests are the ELISA, Macroscopic slide agglutination test (MSAT), latex agglutination test, Dipstick tests (Lepto dipstick, Lepto Tek lateral flow) and Lepto Tek Dri-Dot test. The genus specific tests are the tests of choice for the diagnosis of current infection. These tests are simple, more sensitive and become positive earlier than MAT. But these tests are also positive with saprophytic leptospira. These tests become positive early in the disease (5–6th day) as they detect specific IgM antibodies and help in the rapid diagnosis of current infection (Shivkumar et al., 2008). Leptospirosis can be prevented by immunization of domestic farm animals and pets, simple measures like removing rubbish from work and domestic environments reducing rodent population, improved education of people at particular risk like farmers and health care staff, identifying and culturing source of infection like open sewers, contaminated wells, antibiotic prophylaxis of exposed persons in areas of high exposure with doxycycline (Feigin et al., 1973 & Takafuji et al., 1984).

**Rickettsial Infections:** Rickettsial infections cause irreversible damage to the human host and are associated with a high morbidity and mortality. The mortality rate can be as high as 20 – 50 %. Except Antartica, rickettsial infections are prevalent throughout the world. Rickettsial disease in India has been documented from Jammu and Kashmir, Himachal Pradesh, Uttaranchal, Rajasthan, Assam, West Bengal, Maharashtra, Kerala and Tamil Nadu (Padbidri et al., 1984). The zoonotic diseases considered important in India are Epidemic typhus, Murine typhus, Scrub typhus, Indian tick typhus and Q fever.

<table>
<thead>
<tr>
<th>TYPES OF RIKETTSIAL DISEASE</th>
<th>EPIDEMIC TYPHUS</th>
<th>MURINE TYPHUS</th>
<th>SCUB TYPHUS</th>
<th>INDIAN TICK TYPHUS</th>
<th>Q FEVER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causative agent Vector</td>
<td>R. prowazeki Body louse</td>
<td>R. typhi Rat flea</td>
<td>R. tsutsugamushi tsukugamushi mites</td>
<td>R. conori ixodid ticks</td>
<td>Coxiella burnetti ticks</td>
</tr>
<tr>
<td>Animal hosts</td>
<td>Cattle, sheep, goats</td>
<td>rats</td>
<td>Field mice, rats, shrews</td>
<td>Dogs, rodents</td>
<td>Cattle, sheep, goats</td>
</tr>
<tr>
<td>Mode of transmission</td>
<td>1. louse faeces rubbed through minute abrasions caused by scratching</td>
<td>Inoculation into skin of faeces of infected fleas</td>
<td>Bite of infected larval mites</td>
<td>Bite of infected tick</td>
<td>1. Inhalation of dust contaminated by urine / faeces of diseased animals</td>
</tr>
<tr>
<td></td>
<td>2. Aerosols of dried louse</td>
<td></td>
<td></td>
<td></td>
<td>2. through abrasions,</td>
</tr>
</tbody>
</table>
### Distribution in India

<table>
<thead>
<tr>
<th>Location</th>
<th>Distribution worldwide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kashmir</td>
<td>Kashmir, Lucknow, Mysore, Shimla, Mumbai.</td>
</tr>
<tr>
<td>Worldwide</td>
<td>Mexico, Africa, Eastern Europe, Asia. South east Asian countries. East Asia, Australia. In middle east All countries</td>
</tr>
</tbody>
</table>

### Clinical features

<table>
<thead>
<tr>
<th>Location</th>
<th>Distribution worldwide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kashmir</td>
<td>Same as that of epidemic typhus but milder and rarely fatal.</td>
</tr>
<tr>
<td>Jammu Kashmir and Rajasthan, Assam, West Bengal.</td>
<td>High Fever, chills, headache, malaise, prostration, macular rash, generalized lymphadenopathy, punched out ulcer covered with a blackened scab at the site of mite bite.</td>
</tr>
<tr>
<td>Himalayas, Nagpur, Jabalpur, Kanpur, Pune, Lucknow and Bangalore.</td>
<td>Escher at the site of tick bite, fever, malaise, headache, rash on the extremities spreading to the rest of the body</td>
</tr>
<tr>
<td>Worldwide</td>
<td>Fever, chills, malaise, headache, no rash or local lesion. Later pneumonia, hepatitis, encephalitis, rarely endocarditis.</td>
</tr>
</tbody>
</table>

### Control measures

<table>
<thead>
<tr>
<th>Location</th>
<th>Distribution worldwide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kashmir</td>
<td>Anti-louse measures and improvements in personal hygiene and living conditions.</td>
</tr>
<tr>
<td>Jammu Kashmir and Rajasthan, Assam, West Bengal.</td>
<td>Residual insecticide like BHC and malathion effective against rat fleas, rodent control measures in affected areas</td>
</tr>
<tr>
<td>Himalayas, Nagpur, Jabalpur, Kanpur, Pune, Lucknow and Bangalore.</td>
<td>Clearing the vegetation where rats and mice live, impregnating clothes and blankets with chemicals like benzyl benzoate and application of mite repellants to exposed skin surfaces.</td>
</tr>
<tr>
<td>Worldwide</td>
<td>Disinfection of dogs, health education about personal protection</td>
</tr>
<tr>
<td></td>
<td>Pasteurization or boiling of milk</td>
</tr>
</tbody>
</table>

Specific diagnosis of rickettsial diseases can be made serologically by indirect IFA and ELISA. Diagnosis can be made in the early acute stage by PCR of blood or tissue, by isolation of rickettsiae by inoculation of blood or tissue in shell vial cell culture and by antigen detection by immunofluorescence on skin biopsy. The diagnosis of Q–fever is frequently made by CFT on paired sera. ELISA and microimmunofluorescence can replace CFT as circulating immune complexes sometimes interfere with CFT. Liver biopsy shows a doughnut granuloma considered typical of Q–fever. (Ori et al., 2008).

**Rabies** is an acute, highly fatal viral disease of the central nervous system caused by Rabies virus. Rabies is more prevalent in the rural areas of Africa and Asia. It is present worldwide except in Australia, New Zealand, UK, Ireland, Scandinavia, Japan and Taiwan. Rabies is one of the oldest recognized diseases affecting humans and one of the most important zoonotic diseases throughout India. In India alone about 20,000 deaths are estimated annually and people are infected following a deep scratch or bite by an infected animal (Sudarshan et al., 2004). Most animal bites in India (91.5%) are by dogs, of which about 20,000 deaths are estimated annually and people are infected following a deep scratch or bite by an infected animal (Sudarshan et al., 2004).
60% are strays and 40% pets. ("New Rabies Vaccine Shows Promise for Prevention, Treatment ", 2009). The incidence of animal bites is 17.4 per 1000 population (Chugh et al., 2008). About 15 million people are bitten by animals, mostly dogs, every year and need post-exposure prophylaxis. The disease begins with headache, malaise, sore throat and mild fever for 3-4 days. Patients complain of pain and tingling at the site of the bite. This is followed by intolerance to noise and bright light. Aerophobia is present. Increased reflexes, muscle spasms, dilation of pupils and increased perspiration, salivation and lacrimation are present. Hydrophobia is pathognomonic of rabies. Paralysis, coma or sudden death may ensue. To – date only three people are on record who has survived after rabies. (Alan C et al., 2003). Rabies can be confirmed in patients early in the illness by antigen detection using immunofluorescence of skin biopsy and by viral isolation from saliva and other secretions. Immunofluorescence of corneal impression smears has proved unreliable and neutralizing antibodies are not usually detectable in serum or CSF before the eighth day. Culture in tissue culture cell lines like WI 38, BHK 21, CER is most successful during the first week of illness from saliva, throat, tracheal or eye swabs, brain biopsy samples, csf and possibly centrifuged urine (Anderson et al., 1984). The method of inoculation of suckling mice yields results in 1-3 weeks but tissue culture isolation in murine neuroblastoma cells takes about 2 days (Rudd rchi et al., 1989). Rapid diagnosis by PCR tests on saliva, CSF and skin biopsy specimens are available in reference laboratories (Crepin et al., 1998). A direct immunofluorescent antibody test rapidly identifies antigen in frozen sections of skin biopsies taken from a hairy area usually the nape of the neck (Bryeson et al., 1975). In unvaccinated patients rabies antibodies often appear during the second week of illness and are diagnostic, but many remain seronegative at death (Hemachudha et al., 2006). In vaccinated people, very high levels of antibody in the serum and especially in the CSF are needed to suggest the diagnosis. [Flaegstad et al., 1991, Lee et al., 2000, Markowitz et al., 1988, Fleischer et al., 1983 & Kleiman et al., 1981). Prevention of human rabies is by eliminating rabies in dogs through vaccination, pre-exposure and post exposure prophylaxis of humans. Pre-exposure immunization is also recommended for people in certain high-risk occupations such as laboratory workers dealing with live rabies virus, veterinarians and dog handlers. Post exposure prophylaxis consists of local treatment of wounds, active immunization with antirabic vaccines and passive treatment with antirabic vaccines.HDC vaccine, PCEC vaccine and PVC vaccine are available which are equally safe and effective. Recombinant vaccines are still in the experimental stage (Ananthnarayan et al., 2013).

**Arboviral diseases:** Arboviruses which can cause zoonotic diseases and are of public health importance in India are Japanese encephalitis virus, Dengue virus, Chikungunya fever and Kyasanur forest disease virus. An estimated 50000 cases of JE occur globally each year with 10000 deaths. In India, about 300 million are at risk. Approximately 2.5 billion people live in dengue-risk regions with about 100 million new cases each year worldwide (Nivedita et al., 2012). India accounts for nearly one-third of all dengue cases reported globally (Derek et al., 2011). A report from Indian council of Medical Research states an occurrence of 40–1000 KFD cases each year and a mortality rate of 4-15% (Work et al., 1959). More than 1.3 million people are estimated to be affected by Chikungunya virus prevailed across 150 districts of 8 states in India (Lahariya., 2006).

<table>
<thead>
<tr>
<th>Disease</th>
<th>Japanese encephalitis</th>
<th>Dengue</th>
<th>Chikungunya fever</th>
<th>Kyasanur disease</th>
<th>forest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causative agent</td>
<td>Japanese encephalitis virus</td>
<td>Dengue virus</td>
<td>Chikungunya virus</td>
<td>Kyasanur disease virus</td>
<td>forest</td>
</tr>
<tr>
<td>Reservoir</td>
<td>Wild birds, pigs</td>
<td>? Monkeys</td>
<td>? Monkeys</td>
<td>Forest birds</td>
<td></td>
</tr>
<tr>
<td>Mode of transmission</td>
<td>Bite of Culex tritaeniorhynchus, C. vishnui, C.gelidus.</td>
<td>Bite of Aedes aegypti and Aedes albopictus</td>
<td>Bite of Aedes aegypti</td>
<td>Bite of Tick [Haemaphysalis spinigera]</td>
<td></td>
</tr>
<tr>
<td>Worldwide distribution</td>
<td>Japan, India, southeast Asia</td>
<td>India, far east, Caribbean islands</td>
<td>Sub-Saharan Africa, India, many areas of</td>
<td></td>
<td></td>
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</tbody>
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<table>
<thead>
<tr>
<th>Distribution in India</th>
<th>Clinical features</th>
<th>Complications</th>
<th>Lab diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assam, UP, Bihar, Haryana, Goa, Maharashtra, South India.</td>
<td>Fever, headache, vomiting, signs of increased intracranial pressure, difficulty of speech, ocular palsies, hemiplegia, quadriplegia, tremors, altered sensorium, convulsions, coma</td>
<td>Residual neurological damage, death</td>
<td>1. IgM – capture ELISA to detect specific IgM in the CSF or blood within 7 days of disease. 2. Antibody assays [HI, EIA or IFA] on paired sera</td>
</tr>
<tr>
<td>Whole of India</td>
<td>Saddle back fever, headache, retrobulbar pain, break bone fever, conjunctival injection, lymphadenopathy, maculopapular rash.</td>
<td>Dengue haemorrhagic fever manifesting as reduction of platelets (thrombocytopenia). Minute hemorrhages within skin (petechiae). Plasma leakage. Dengue shock syndrome – Restlessness, irritability. Shock with cold clammy extremities. Rapid respiration, rapid/weak pulse.</td>
<td>1. Dengue NS1 antigen detection can be done in the first week of illness. 2. IgM antibody detection can be done by ELISA within 2-5 days of disease.</td>
</tr>
<tr>
<td>Asia</td>
<td>Fever, chills, cephalalgia, anorexia, lumbago, conjunctivitis and adenopathy, morbilliform rash on trunk and limbs, coffee colour vomiting, epistaxis, petechiae. Prominent symptom in adults is arthropathy pain, swelling, stiffness of the metacarpophalangeal, wrist, elbow, shoulder, knee, ankle and metatarsal joints.</td>
<td>Haemorrhages from nose, gums, stomach and intestine. Meningoencephalitis.</td>
<td>1. Detected in serum in the first 3-4 days with PCR. 2. Ig M can be detected by IFA or EIA in acute sera. 3. Acute infection can be confirmed by rising HI or Serological diagnosis made by rising antibody titres in acute and convalescent sera by IFA, HI, EIA.</td>
</tr>
<tr>
<td>Tamil Nadu, Kerala, Karnataka, Maharashtra, Madhya Pradesh, Gujarat, Rajasthan, Pondicherry, Goa, Orissa, West Bengal, UP and Andaman.</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Karnataka</td>
<td>Fever, headache conjunctivitis, myalgia, prostration, gastrointestinal disturbances.</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
to demonstrate significant rise in JE specific antibody.
3. Dot blot IgM assay for field use.

Prevention

Vector control by aerial or ground fogging with ultra-low – volume insecticides, use of mosquito nets, locating piggeries away from human dwellings. Elimination of breeding sites of vector and health education.

Mosquito control measures. No vaccine is available.

Vector control by Carbaryl fenthion spraying in hot spots.

JENVAC vaccine can be administered as a single dose during epidemics for mass vaccination campaigns and also as a two-dose schedule during routine immunisation as part of the National immunisation programme in endemic regions.

Neutralizing antibody titres on paired sera.

RT PCR to detect viral DNA.

Leishmaniasis: it is a complex disease caused by the protozoan Leishmania. It is spread over large geographical areas around the globe. Worldwide it is endemic in Africa, Central and South America, Asia and Mediterranean region. In India, it is endemic in Bihar, Jharkhand, and West Bengal and UP. In India, the disease manifests in two forms: the cutaneous and the visceral (kala-azar) variety. The annual estimate for the incidence and prevalence of kala-azar cases worldwide is 0.5 million and 2.5 million, respectively. Of these, 90% of the confirmed cases occur in India, Nepal, Bangladesh and Sudan. The incidence of kala-azar in India is among the highest in the world (Bora et al., 1999). It is transmitted by the bite of female phlebotomine sandfly. Phlebotamus argentipes transmits visceral leishmaniasis and P.papatasi and P.sergenti transmits cutaneous leishmaniasis. No animal reservoirs are present for visceral leishmaniasis but dogs are the sources and reservoir for cutaneous leishmaniasis. The classical features of kalaazar are fever, splenomegaly and hepatomegaly, anaemia, weight loss, darkening of skin of the face, hands, feet, abdomen and lymphadenopathy. One or several years after apparent cure of kalaazar, post kala-azar dermal leishmaniasis lesions develop consisting of multiple nodular infiltrations of the skin, usually without ulceration. Painful ulcers in the parts of the body exposed to sandfly bites –legs, arms or face are the features of cutaneous leishmaniasis. Bone marrow and spleen aspirations are the most commonly used samples for lab diagnosis. Lymph nodal samples can be used only in foci where lymphadenopathy is frequent. Parasitic detection in peripheral blood is relatively non invasive and can be used both in immunocompetant and immunocompromised patients. Inflammatory edge of an ulcerative cutaneous
lesion is the selected place for parasite detection. The staining method most appropriate for leishmanial detection is one employing panoptic May Grunwald–Giemsa stain. Amastigotes are typically intramonicytic, but frequently extracellular in the smears, nucleus and kinetoplast stain characteristically purple. This method gives a rapid diagnosis, but is less sensitive. Specific staining of biopsy material using mouse anti-leishmania immune serum and peroxidase conjugate can be used for easy detection of parasites. Culture has higher sensitivity than direct detection of parasites in smears. The classical blood agar NNN medium is the most currently used media (Zeledon et al., 1996). Molecular diagnosis can be employed to any form of leishmaniasis. The detection of parasite DNA has high sensitivity and specificity. Other advantages include the possibility of detecting the parasite on contaminated samples or cultures and the relative promptness of the result as well as the possibility to carry out species identification simultaneously or at least using the same DNA extract (Reithinger et al., 2007, Lachaud et al., 2000, Lopez et al., 1993, Pimez et al., 1999). In visceral leishmaniasis, a PCR assay has proved almost 100% sensitive using peripheral blood, avoiding bone marrow sampling making it particularly applicable in immunosuppressed patients as a relatively non invasive diagnostic method. Latest advance is the development of ultrasensitive PCR assay for visceral leishmaniasis which enables detection of asymptomatic carriage in man even in immunosuppressed patients and also allows the determination of parasitemia threshold above which symptoms are likely to occur (Mary et al., 2006). Various serological techniques have been developed to detect circulating specific antibodies for the diagnosis of visceral leishmaniasis but differ in their sensitivity and specificity. They are IFAT, Immuno- enzymatic techniques, counter current immune-electrophoresis, IHA and immune blot. A few tests which are relatively easy to practice in field conditions are DAT, rK39 immunochromatography dipstick, latex particle agglutination, dot – ELISA and fast – ELISA. Immunological diagnosis is a relatively noninvasive approach and useful to combine with direct demonstration of the parasite, which remains the reference technique. Immunodiagnosis should not be used for post treatment follow up and in immunocompromised patients. Immunoblot analysis is a more sensitive technique than IFAT or ELISA, which detect antibodies against specific antigens according to leishmania species. It is a method useful not only for diagnosis confirmation, but also for patient follow up during treatment. And also to detect asymptomatic infections in patients living in endemic areas. A latex agglutination test for detection of leishmania antigens in urine has been developed which appears to be more efficient for initial diagnosis of visceral leishmaniasis before treatment (Kumar, 2002). Control of this disease is by active and passive case detection and treatment of those found to be infected. Mass surveys can be undertaken in endemic areas for early detection of cases (Al-Sous et al., 2004). Phase ITI trials with a first-generation vaccine (killed Leishmania organism mixed with a low concentration of BCG as an adjuvant) produced encouraging results (Bora et al., 1999). A non-human primate model has been developed to evaluate various vaccines/drugs for leishmaniasis caused by Leishmania donovani/ Preliminary studies using autoclaved Leishmania major (ALM) mixed with BCG have been successful in preventing infection with Leishmania donovani," (Anuradha et al., 1992).

Taeniasis & Cysticercosis : It is caused by two parasites of medical importance – Taenia saginata and Taenia solium. Taenia solium is endemic in Latin America, Africa, Asia, some parts of Europe and USSR. It is endemic in India and has been widely reported. Taenia saginata is highly endemic in Africa, Eastern Mediterranean countries and in parts of USSR. It is moderately prevalent in India (Gloria M et al., 2013). The disease is transmitted through ingestion of infective cysticerci in undercooked pork (Taenia solium ) or beef (Taenia saginata), through ingestion of food, water of vegetables contaminated with eggs. Cysticercosis refers to tissue infection after exposure to eggs of Taenia solium, the pork tapeworm. This is the more serious public health problem in India than Taeniasis. The disease is spread via the fecal-oral route through contaminated food and water, and is primarily a food borne disease. The most common route is through raw-eaten vegetables which have been grown in fields irrigated with untreated sewage water. Other potential sources of feco-oral contamination are admixture of sewage water with drinking water in pipelines, and through houseflies and cockroaches. Cysts are formed in the brain and muscles.
which may persist for years. Generalised muscle pain, painful nodules in the muscles and seizures when the cysts are located in the brain are the common symptoms. Cysticerci can develop in any voluntary muscle in humans and cause myositis, with fever, headache, eosinophilia, and muscular pseudohypertrophy, which initiate with muscle swelling and later progress to atrophy and fibrosis (Markell et al., 1999). These cysts may block the outflow of cerebrospinal fluid and present with symptoms of increased intracranial pressure (Suri et al., 2008). Neurocysticercosis involving the spinal cord, most commonly presenting as back pain and radiculopathy (Jang et al., 2010). If cysticerci may be found in the globe, extraocular muscles, and subconjunctiva, they may cause visual difficulties that fluctuate with eye position, retinal edema, hemorrhage, a decreased vision or even a visual loss. The traditional method of demonstrating tapeworm eggs or proglottids in stool samples diagnoses only taeniasis (Garcia et al., 1998). Only a small minority of patients with cysticercosis will harbor a tapeworm, rendering stool studies ineffective for diagnosis. Ophthamlic cysticercosis can be diagnosed by visualizing parasite in eye by fundoscopy. Antibodies to cysticerci can be demonstrated in serum by Enzyme Linked Immunosorbent Blot assay and in CSF by ELISA. An immunoblot assay using lentil lectin (agglutinin from Lens culinaris) is highly sensitive and specific. However, individuals with intracranial lesions and calcifications may be seronegative. The diagnosis of neurocysticercosis is mainly clinical, based on a compatible presentation of symptoms and findings of imaging studies. Neuroimaging with CT or MRI is the most useful method of diagnosis. CT scan shows both calcified and uncalcified cysts, as well as distinguishing active and inactive cysts. Cystic lesions can show ring enhancement and focal enhancing lesions. MRI is more sensitive in detection of intraventricular cysts (Prathibra et al., 2011). CSF findings include pleocytosis, elevated protein levels and depressed glucose levels; but these may not be always present. Cysticercosis is potentially eradicable. It is feasible because there are no animal reservoirs besides humans and pigs. Prevention can be achieved by massive chemotherapy of infected individuals, improving sanitation, and educating people, cooking of pork or freezing it and inspecting meat, and by treating or vaccinating pigs (Allan et al., 1997 & Sciutto et al., 2007).

**Echinococcosis:** It is caused by a tape worm called Echinococcus granulosus. It is recognized as a public health problem of nearly global dimensions. It is found in Australia, New Zealand, middle east, Turkey, Greece, USSR, Latin America and far east [Reddy et al., 1968]. Foci in India with high prevalence of echinococcosis exist in Andhra Pradesh and Tamil Nadu. Major problem responsible for considerable morbidity and socio-economic losses in many regions of the world. According to statistics given out by WHO, the annual societal cost of cystic echinococcosis amounts to US $150 million in the subcontinent of India alone. (Reddy et al., 1968). Human echinococcosis is caused by ingestion of eggs of echinococcus with food, unwashed vegetables or water contaminated with faeces from infected dogs. Symptoms of this disease manifest several years after exposure. Cysts in liver, lung, brain, peritoneum, long bones and kidney are seen. Large cysts cause pressure effects and death. Cysts of liver may present as palpable abdominal mass, abdominal pain, nausea, vomiting, portal hypertension and biliary peritonitis. A plain X-ray, US or CAT SCAN permits the location of the cyst. Serological tests with high specificity are indirect immunofluorescent test, and ELISA. Casoni test is still in wide use but lacks specificity (Ray R et al., 2002). Prevention is by control of dogs which involves elimination of stray dogs, deworming of dogs and health education of butchers, animal breeders, shepherds etc. (Martin et al., 2008).

**Toxoplasmosis:** It is caused by a parasite called Toxoplasma gondii. Humans acquire infection by ingestion of tissue cysts present in raw or undercooked beef, lamb or pork and ingestion of oocysts from soil, water, milk or vegetables. Toxoplasmosis is present in every country of the world and seropositivity rates range from less than 10% to over 90%. Worldwide, over 6 billion people have been infected with *T. gondii* (Klaren et al., 2002). Seroprevalence in India is about 22% approximately (Srirupa et al., 2011). Human infection is acquired by ingestion of tissue cysts in raw, poorly cooked meat of lamb and pork or ingestion of sporocysts derived from cat faeces containing soil or inadequately washed vegetables. Majority of congenital infections are caused when the mother acquires infection during pregnancy.
Infection with Toxoplasma has three stages: During acute toxoplasmosis, symptoms are often influenza-like: swollen lymph nodes, or muscle aches and pains that last for a month or more. Young children and immunocompromised people, develop severe toxoplasmosis. This can cause encephalitis or necrotizing retinochoroiditis. Infants infected via placental transmission may be born with either of these problems, or with nasal malformations. Swollen lymph nodes are commonly found in the neck or under the chin, followed by the axillae (armpits) and the groin (Ling et al., 2011). It is usually found at single sites in adults, but in children, multiple sites may be more common. Enlarged lymph nodes will resolve within one to two months in 60% of cases. However, a quarter of those affected take two to four months to return to normal, and 8% take four to six months. A substantial number (6%) do not return to normal until much later (Dubey et al., 2006). Most infants who are infected while in the womb have no symptoms at birth, but may develop symptoms later in life (Foulon et al., 2000). Skin lesions may occur in the acquired form of the disease, including roseola and erythema multiforme-like eruptions, prurigo-like nodules, urticaria, and maculopapular lesions. Newborns may have punctate macules, ecchymoses, or “blueberry muffin” lesions (Dubey et al., 2006). Laboratory diagnosis includes detection of Toxoplasma-specific antibodies which is the primary diagnostic method to determine infection with Toxoplasma. Antibodies are detected by numerous serologic tests and most of the test kits are commercially available to detect T. gondii specific IgG, IgM, IgA or IgE antibodies. The IFAT, LAT, DAT and ELISA are used more commonly. If more precise knowledge of the time of infection is necessary, then an IgG positive person should have an IgM test performed by a procedure with minimal nonspecific reactions, such as IgM-capture EIA. A negative IgM test essentially excludes recent infection. New born infants suspected of congenital toxoplasmosis should be tested by both an IgM- and an IgA-capture EIA. Detection of Toxoplasma-specific IgA antibodies is more sensitive than IgM detection in congenitally infected babies (Joao et al., 2011). Detection of T. gondii in human blood samples, CSF and amniotic fluid may also be achieved by using the polymerase chain reaction (Webster et al., 2010). The other methods of diagnosis are imaging methods and immunohistochemical studies. Preventive measures include avoiding eating raw or undercooked meat, washing fruit and vegetables before cooking and eating, avoiding drinking unpasteurized goats' milk or eating products made from it, avoiding handling or adopting stray cats. Routine antenatal screening for toxoplasmosis and treatment of infected mothers would be an effective step in protecting the fetuses from toxoplasmosis. No vaccine is at present available for this disease (Joao et al., 2011).

CONCLUSION
Zoonotic diseases which are prevalent in India and which are of serious concern in India are brucellosis, anthrax, plague, leptospirosis, rickettsial infections, rabies, arboviral diseases, leishmaniasis, taeniasis, cysticercosis and toxoplasmosis. All these diseases are transmitted by either contact, inhalation or by ingestion of the pathogens. They usually manifest as an acute infection which becomes chronic and effective various important organs of our body. Most of them are associated with significant morbidity and mortality. Though there are wide arrays of diagnostic tests available for these diseases which range from microscopic detection of the pathogens to the sophisticated molecular diagnostic methods, serological tests would be the main stay of laboratory diagnosis in the Indian scenario as they would be cost effective. Dipstick methods and card tests are quick and aid in rapid diagnosis. These diseases can be prevented by health education, animal vaccination, and case contact management with appropriate antibiotics, antibiotic prophylaxis and vector control. Vaccines for humans use are under trial for most of the zoonotic diseases except a few like vaccines for Japanese encephalitis, Plague, KFD, Rabies and Q Fever.

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