Review Article

Listeria monocytogenes: An Emerging Food Borne Pathogen

M. Saha*, C. Debnath and A. K. Pramanik

Department of Veterinary Public Health, Faculty of Veterinary and Animal Science,
West Bengal University of Animal and Fishery Science, 37, K.B.Sarani,
Belgachia, Kolkata-700 037, West Bengal, India
Corresponding author

A B S T R A C T

Keywords

L. mono cytogenes, Listeria spp, Zoonosis, Foodborne

L. monocytogenes is a psychrophilic bacteria recognized as a pathogen of great importance of food. Infection with *L. monocytogenes* is a widespread zoonosis, affecting mainly cattle, sheep and goat herds. Listeria species are ubiquitous bacteria widely distributed in the natural environment. *L. monocytogenes* has been called an "emerging food-borne pathogen" because only recently we have recognized that it can be transmitted through food. It causes listeriosis, a serious infectious disease which occurs as a consequence of consumption of food contaminated with this pathogenic bacterium. It is a psychrophilic bacteria recognized as a pathogen of great importance of food. It is accepted that listeriosis in humans is a disease that is transmitted mainly through food. The series of outbreaks of the 1980s showed that *L. monocytogenes* causes very serious invasion and often life-threatening disease, constituting an economic burden for both public health services and food industry.

Introduction

India is the world's largest producer of dairy products by volume and has the world's largest dairy herd. The country accounts for more than 13% of world's total milk production and is also the world's largest consumer of dairy products, consuming almost all of its own milk production (Singh, 2011).

There are many organisms secreted through milk, one of them is *Listeria monocytogenes* which causes significant public health problem. *L. monocytogenes* is the most important species in the genus

Listeria creating human health hazard and having a worldwide distribution with an extensive host range which includes mammal, poultry, fish, crustacean and ticks. The name of the intracellular organism emphasizes the relationship between infection and the development of monocytosis in the host, which is now considered to be an inconsistent feature of listeriosis in all animal species (OIE Manual, 2014).

L. monocytogenes as an important food-borne pathogen became evident in 1980s,

after several food-borne outbreaks proved to be caused by it (Schlech *et al.,* 1983; Fleming *et al.,* 1985; Linnan *et al.,* 1988). After that, several reports have been recorded of food-borne listeriosis, both epidemics and sporadic cases, almost all kinds of foods (Goulet *et al.,* 1995; Salvat *et al.,* 1995; Loncarevic *et al.,* 1997; Miettinen *et al.,* 1999; Lyytikäinen *et al.,* 2000). The first proof that milk products could be responsible for listeriosis outbreaks was corroborated by Fleming *et al.* (1985) which involved 49 cases, seven of them in the foetus or in infants and 42 in immune- compromised adults.

L. monocytogenes is known to be secreted in milk by both infected and healthy animals (Wagner *et al.,* 2000). Human listeriosis is a food-borne disease, and it has been estimated that 99% of all human listeriosis cases are caused by the consumption of contaminated food products (Mead *et al.,* 1999). Although listeriosis is not common in humans, it is a clinically significant disease because of its high mortality and severity (Atil *et al.,* 2011).

L. monocytogenes has been called an "emerging food-borne pathogen" because only recently we have recognized that it can be transmitted through food. *Listeria monocytogenes* is a ubiquitous bacterium. It causes listeriosis, a serious infectious disease which occurs as a consequence of consumption of food contaminated with this pathogenic bacterium. Listeriosis is a significant public health problem (Rocourt and Catimel, 1985). The first communications/reports of the presence of *Listeria* in food associated with dairy products, where cow milk was mentioned as carrier of the fatal listeriosis (Farber and Peterkin, 1991).

According to many communications, consumption of milk and dairy products

contaminated with *L. monocytogenes* can lead to individual cases of listeriosis or true outbreak of this disease. Of all dairy products, soft cheeses and non-pasteurized milk are most common causes of listeriosis. In the process of production of milk and dairy products, it most commonly occurs as a consequence of post- pasteurization contamination. Listeriosis is serious disease of humans, occurring sporadically or in the form of epidemic, with mortality rate of over 25% (USDA, 1999).

L. monocytogenes is a psychrophilic bacteria recognized as a pathogen of great importance of food. It is accepted that listeriosis in humans is a disease that is transmitted mainly through food. The series of outbreaks of the 1980s showed that *L. monocytogenes* causes very serious invasion and often life-threatening disease, constituting an economic burden for both public health services and food industry. Infection with *L. monocytogenes* is a widespread zoonosis, affecting mainly cattle, sheep, and goat herds. Listeria species are ubiquitous bacteria widely distributed in the natural environment. This specific character of the bacteria inevitably results in contamination of numerous food products. *Listeria* spp. is widely distributed in environment and is the causal agent of listeriosis, a disease that can be serious and is fatal among elderly, very young and immune-compromised persons, with an approximate 20% case–fatality rate (Rocourt and Catimel 1985), that may increase up to 75% in high risk groups, such as pregnant women, neonates, and immune-compromised adults. The incidence of listeriosis in developed countries is about 0.2 to 0.8 cases per 100,000 persons annually (Gellin *et al.,* 1991; McLauchlin 1996; Kela and Holmström 2001; Lukinmaa *et al.,* 2003). The incidence is not high, but as the mortality is about 20% (Gellin *et al.,* 1931), the disease is a public health

concern. Listeriosis usually manifests in the elderly, in foetuses or newborns and in individuals with underlying diseases.

According to Bergey's Manual of Systematic Bacteriology (1994) *Listeria* genus includes: *L. monocytogenes, L. ivanovii, L. innocua, L. seeligeri L. welshimeri and L. gray.* Among these *L. monocytogenes* is pathogenic for humans and animals, and *L. ivanovii* is mainly pathogenic for animals, primarily sheep. Other species are considered to be non-pathogenic.

All *Listeria* species are small, regular rods, 0.5 μm in diameter and 1─5 μm in length that do not form spores or capsule. They produce catalyse but not oxidise. It is a Gram positive, facultative anaerobic bacterium with both psychotropic and mesophillic features. *Listeria* species are found in the intracellular state within monocytes and neutrophils (Grayand Killinger, 1966). *L. monocytogenes* is motile due to one to five peritrichous flagella, which may be lost as the bacteria enter the human cell. Movement is still possible because the bacteria polymerize acting into long acting tails that propel the bacteria through the cytoplasm (Salyers and Whitt, 2002). Filaments, ranging in size of 6-20μm, may develop in old cultures.

Members of the *Listeria* genus are not forming spores and capsules, distributed individually and in form of short chains, sometimes in form of the letters 'V' and 'Y'.

In direct smear they can be coccoid, and therefore mistaken with streptococci (Todar, 2009). In old cultures they form longer rods, with long filaments, and also Gram-negative units can occur. *Listeria* is mobile at the temperature of 20–25°C (they create peritrichous flagella) and immobile

at 37°C. They are catalase positive, oxidase negative, Esculin hydrolysis positive. *L. monocytogenes* during its exponential phase of multiplication creates a toxin called listeriolysin O (hemolysin), which leads to in-vitro hemolysis on blood agar. *L. monocytogenes* has a higher resistance to external environment.

L.monocytogenes is a psychrotroph with resistance to high temperatures. It has wide temperature range for growth. It multiplies at temperatures ranging from 1.5°C to 45°C. Optimal temperature for growth is 30–37°C. The organism reproduces/ multiplies best at pH 7. It is resistant to acidic (pH< 5) and alkaline (pH 9.6) environment.

L.monocytogenes can grow well at the NaCl concentration of more than 10%. In one study it was reported that the organism survived for one year period in concentration of 16% NaCl, at pH 6.0 (Seeliger and Jones, 1984). In another study Conner *et al.* (1986) stated that the resistance of *L. monocytogenes* to common salt intensifies at lower temperatures. It can survive 100 days in concentration of 10.5–30% NaCl, at 4°C. The organism is resistant to UV radiation, gamma rays and x rays which greatly contribute to wide spreading of this bacterium (Bougle and Stahl, 1994).

L. monocytogenes is resistant to most of sanitation preparations/chemicals. Preparations that have proven to be the best in destruction of *L. monocytogens* are iodoforms peracetic and peroctanoic acid, quarternary ammonium compounds and chlorine solutions (Tompkin *et al.,* 1999; Schafer *et al.,* 2000; Eifert and Sanglay, 2002).

The public health importance of listeriosis is not always recognized, particularly since

listeriosis is a relatively rare disease compared with other common food-borne illnesses such as salmonellosis or botulism. However, because of its high case fatality rate, listeriosis ranks among the most frequent causes of death due to food-borne illness second after salmonellosis (Rossmanith *et al.,* 2006). It is responsible for the highest hospitalisation rates (91%) amongst known food-borne pathogens and has also been linked to sporadic episodes and large outbreaks of human illnesses worldwide (Jemmi and Keusch, 1994). Epidemiological data from different countries show that the majority of human outbreaks are associated with three *L. monocytogenes* serotypes (1/2a, 1/2b and 4b) among a total thirteen recognized serotypes.

The contamination of food by *L. monocytogenes* occurs along the food chain from farm-to-fork (Farber and Peterkin, 1991). Cross-contamination, which can occur within the environment of food-processing equipment, is considered to be a possible source of *Listeria* contamination in processed food. *L. monocytogenes* is able to attach and survive on various working contact surfaces. One reason may be its ability to form biofilms (Wong *et al.,* 2012).

L. monocytogenes is associated with septicaemia, meningoencephalitis and abortion in humans and animals, primarily affecting pregnant, new- born, and immune-compromised individuals (Choi and Hong, 2003; Rossmanith *et al.,* 2006).

Several outbreaks of listeriosis were proven to be associated with the consumption of milk and causing great concern in the dairy industry due to the number of cases and the nearly 30% overall mortality rate of these outbreaks (Amagliani *et al.,* 2004).

Historical background

Early reports suggest that *Listeria monocytogenes* may have been isolated from tissue sections of patients in Germany in 1891, from rabbit liver in Sweden in 1911, and from spinal fluid of meningitis patients in 1917 and again in 1920 (Reed, 1958; McCarthy, 1990).

However, it was not until 1926 that the microorganism was fully described, when Murray *et al.* (1926) isolated a small, Gram-positive rod shaped bacterium that had caused an epizootic outbreak in 1924 among rabbits and guinea pigs. They named the organism Bacterium monocytogenes. This was a year after listeriosis in sheep was recognized in Germany as a disease syndrome, though the causative agent had not been isolated.

At approximately the same time, Pirie (1927) isolated and described the same organism from gerbils in South Africa. He named the bacterium *Listerella hepatolytica*, and subsequently recommended in 1940 that the name be changed to *Listeria monocytogenes* (Reed, 1958; McCarthy, 1990).

The first report of human Listeriosis was in 1929, and the first perinatal case was reported in 1936 (Gray and Killinger, 1966). The microorganism has been reported to cause disease in a wide range of wild and domestic animals, and has been isolated from numerous species of mammals, birds, amphibians, fish, crustaceans, insects and reptiles (Hird and Genigeorgis, 1990; McCarthy, 1990; Ryser and Marth, 1991). It is now widely recognized that human Listeriosis is largely attributable to food-borne transmission of the microorganism. It was not until several large, common-source outbreaks of Listeriosis occurred in North America and Europe during the 1980s that

the significance of foods as the primary route of transmission for human exposure to *L. monocytogenes* was recognized (Broome *et al.,* 1990; Bille, 1990). While the modes of transmission for *L. monocytogenes* can include vertical (mother to child), zoonotic (contact with animal to man), and nosocomial (hospital acquired), it is generally considered that most cases of human Listeriosis involve food-borne transmission.

Taxonomy

L. monocytogenes was previously in the family Corynebacteriaceae (Stuart and Pease, 1972), but in the 8th edition of Bergey's Manual of Determinative Bacteriology, *Listeria* along with *Erysipelothrix* and *Caryophanon* were grouped as uncertain affiliation. On the basis of DNA-DNA hybridization, Stuart and Welshimer (1974) suggested a new family *Listeriaceae* to accommodate genera *Listeria* and *Morraya*. Today, the genus *Listeria* belongs to the *Clostridium* subbranch together with *Staphylococcus*, *Streptococcus,* *Lactobacillus* and *Brochothrix.*

Listeria includes six species, of which one is divided into two subspecies: *L. monocytogenes, L. innocua, L. welshimeri, L. seeligeri, L. grayii* and *L. ivanovii* subsp. *ivanovii* and L. *ivanovii* subsp. *londoniensis* (Boerline *et al.,* 1992). A seventh species, *Listeria murrayi*, was previously recognized in the *Listeria* genus; however, DNA-DNA hybridization analysis, multilocus enzyme electrophoresis, and rRNA restriction fragment length polymorphism analysis, proved that *L. murrayi* appeared to be subspecies within *L. grayii* (Boerlin *et al.,* 1991, 1992; Rocourt *et al.,* 1992).

Most recently, studies have proposed recognition of two novel species within the *Listeria genus*, including *Listeria marthii* and *Listeria rocourtiae* (Graves *et al.,* 2009). Only *L. monocytogenes* causes disease in both animals and humans. However, occasional human infection with *L. ivanovii* and *L. seeligeri* has been reported (Gilot and Content, 2002). *L. ivanovii* is known to cause spontaneous abortions in sheep.

Genus description

The *Listeria* species are regular Gram-positive non-sporing rods with a diameter of about 0.5 μm and a length of 0.5-2.0 μm. They are facultative anaerobes with no capsule. Moreover, they are catalyse-positive, oxidase-negative and motile at 20-25°C due to peritrichous flagella but non-motile at 37°C (Seeliger and Jones, 1986). *Listeria* produces flagella at room temperature and exhibit a tumbling motion when examined in broth and a swarming motility can be observed in semi-soft agar at 30°C (Roberts *et al.,* 2009), but flagella are not produced at 37°C (Peel *et al.,* 1988).

Morphology

Ritz *et.al., (*2001) studied the morphology of *L. monocytogenes* through electron microscope. They described the observations as the average length and diameter of the organism is 4 micrometer and 0.4 micrometer and the cell surface are smooth.

Growth and Survival characteristics

The growth and survival of *L. monocytogenes* is influenced by a variety of factors. In food these include temperature, pH, water activity, salt and the presence of preservatives (Table 1). The temperature range for growth of *L. monocytogenes* is between -1.5 and 45°C, with the optimal temperature being 30–37°C. Temperatures

above 50°C are lethal to *L. monocytogenes*. Freezing can also lead to a reduction in *L. monocytogenes* numbers (Lado and Yousef 2007).

As *L. monocytogenes* can grow at temperatures as low as 0°C, it has the potential to grow, although slowly, in food during refrigerated storage. *L. monocytogenes* will grow in a broad pH range of 4.0–9.6 (Lado and Yousef 2007). Although growth at pH <4.0 has not been documented, *L. monocytogenes* appears to be relatively tolerant to acidic conditions.

L. monocytogenes becomes more sensitive to acidic conditions at higher temperatures (Lado and Yousef 2007).Like most bacterial species, *L. monocytogenes* grows optimally at a water activity (aw) of 0.97. However, *L. monocytogenes* also has the ability to grow at a_w of 0.90 (Lado and Yousef 2007). Johnson *et al.* (1988) demonstrated that *L. monocytogenes* can survive for extended periods of time at a_w value of 0.81. *L. monocytogenes* is reasonably tolerant to salt and has been reported to grow in 13–14% sodium chloride (Farber *et al.,* 1992). Survival in the presence of salt is influenced by the storage temperature. Studies have indicated that in concentrated salt solutions, the survival rate of *L. monocytogenes* is higher when the temperature is lower (Lado and Yousef 2007). *L. monocytogenes* can grow under both aerobic and anaerobic conditions, although it grows better in an anaerobic environment (Lado and Yousef 2007).

The effect of preservatives on the growth of *L. monocytogenes* is influenced by the combined effects of temperature, pH, salt content and water activity. For example, sorbates and parabens are more effective at preventing growth of *L. monocytogenes* at lower storage temperatures and pH. Also, adding sodium chloride or lowering the

temperature enhances the ability of lactate to prevent *L. monocytogenes* growth. At decreased temperatures (such as refrigeration storage) sodium diacetate, sodium propionate and sodium benzoate are more effective at preventing growth of *L. monocytogenes* (Lado and Yousef 2007). The organism can withstand repeated freeze - thaw cycles and high salt concentrations (0-10% NaCl) (Lovett, 1990).

Doyle (1988) reported that the organism can survive for 5 days in 20 - 30% NaCl. Survival for more than 100 days has been observed in 10.5 - 30% NaCl at 4^0C (Lovett, 1989). The organism can grow over a wide pH range (5.2 - 9.6); and it has been shown to be quite resistant to alkaline pH. The organism is also capable of surviving a low water activity (<0.92) (Garland, 1995).

Serological association of *Listeria* species

Serology is a classic tool for epidemiological studies. It is the analysis of the properties and effects of serums to detect the presence of antibodies against a microorganism. *Listeria* species strains are stereotyped based on the cellular (O) and flagella (H) antigens (Graves *et al.,* 1999; Seeliger and Hone, 1979). The serology description of *Listeria* is described by Seeglier and Donker based on 15 'O' antigens and 5 'H' antigens (Lovett, 1990). Table 2 provides a list of the serovars of *Listeria* species. Table 3 shows the various serotypes of the *Listeria* species. Over 90% of *Listeria monocytogenes* isolates can be serotyped with commercially available sera.

Sera contain antibodies that have been obtained from an animal which has been immunized either by antigen injection or infection with microorganisms containing the antigen. Most *L. monocytogenes* isolates obtained from patients and the environment are type 1 or 4 (Hitchin's, 2003). All non-

pathogenic species, except *L. welshimeri*, share one or more somatic antigens with *L. monocytogenes*.

It is due to this reason that the FDA suggests that stereotyping alone without the completion of identification procedures is inadequate for identification of *L. monocytogenes*. *L. monocytogenes* is the only species that causes infections in humans while *L. ivanovii* is sporadically associated with abortions in animals.

Greater than 95 % of human infections caused by strains of *L. monocyt*ogenes belong to the serotype ½ a, ½ b, and 4b (Graves *et al.,* 1999).

Biochemical characteristics of *L. monocytogenes* and other *Listeria* spp.

All the isolates were subjected to standard biochemical tests such as Gram staining, Catalase test, motility at 25 °C and 37 °C, and acid production from mannitol, rhamnose, and xylose. For further confirma-tion of *Listeria* isolates, other biochemical reactions like β-haemolytic activity, and Christine-Atkins-Munch-Petersen (CAMP) were measured according to Bergey's Manual of Systematic Bacteriology (Seeliger and Jones, 1986). *L. ivanovii* can be differentiated biochemically from *L. Monocytogenes* and other *Listeria* species by its production of a wide, clear or double zone of haemolysis on sheep or horse blood agar, a positive Christie–Atkins–Munch-Petersen (CAMP) reaction with *Rhodococcus equi* but not with haemolytic *Staphylococcus aureus*, and fermentation of D-xylose but not L-rhamnose and D-mannitol (Rocourt and Catimel, 1985). The species are distinguished by the haemolysis test, CAMP test and sugar fermentation (Seeliger and Jones 1986).

Listeriosis worldwide and in India

Listeriosis, a zoonotic bacterial disease has emerged as a major food borne disease during the past two decades, since it has a high case fatality rate (approximately 20 to 30%) (Doyle, 1988). Listeriosis, as an important cause of severe illness account to 3.8% of food-borne hospitalization and 27.6% of food-borne deaths in the united states (Adak *et al.,* 2002). The severity of the disease includes meningitis, septicaemia and abortion. It also has a long incubation time and a predilection for individuals who have an underlying condition, which leads to impairment of (T-cell mediated) immunity.

In 1981, the first documented and confirmed food-borne outbreak occurred at Nova Scotia, Canada. The outbreak was linked to a food produce, locally prepared coleslaw (Schlech *et al.,* 1983). The route of transmission of *L. monocytogenes* by food was evident only in 1980's after a series of outbreaks (Schelch *et al.,* 1983). Nearly all cases of Listeriosis are food-borne (Schwartz *et al.,* 1988; Mead *et al.,* 1999; Adak *et al.,* 2002). Recently the proportion of Listeriosis cases due to food-borne transmission has been estimated to be 99% (Mead *et al.,* 1999).

Due to poor net working, reporting system on the disease outbreak and food-borne disease surveillance, very limited information is available on the prevalence of food-borne disease in India. However, despite, the poor information management system on the incidence and epidemiology of diseases in the Indian subcontinent, reports on sporadic cases of both animal and human Listeriosis could be traced which dates back to the 1930's.

Animal Listeriosis in India traces back to 1935 in Hyderabad states in sheep as

circling disease (Mahajan *et al.,* 1935). The first documented report was in 1950 from sheep in madras (Vishwanathan and Lyyar, 1950).Subsequently there have been a few other reports on the disease in India (Bhujwala and Hingorani, 1975).

Human Listeriosis cases have also been reported in India. Though not phenomenal, the number of human Listeriosis case in India, have been in the rise with reports on sporadic cases and incidence in clinical samples. Probably, it is for this reason that Chugh (2008) has quoted *L. monocytogenes* to be a growing threat and Listeriosis emerging food-borne diseases in India.

A few of the significant clinical reports on the incidence of Listeriosis in the Indian sub-continent is detailed below.

Usha *et al.* (1966) were the first to discuss on human Listeriosis in India.

Bhujwala *et al.* (1973) reported a pilot study on the genetic Listeriosis in Delhi. Again, in 1975, Bhujwala and Hingorani, reported on the perinatal Listeriosis in India.

Thomas *et al.* studied neonatal Listeriosis in north India in 1981, in the same year Gogate and Deodhar, reported a case of meningitis caused by Listeria infection.

With a break for about 15 odd years, reports on Listeriosis, emerged yet again in 1977 with the report of Gupta *et al.* They reported on opportunistic Listeria infection of an infant Gupta *et al.,* in the year 2003 made a report of a sporadic case of Listeriosis in the second trimester of pregnancy.
Srivasta *et al.,* in the year 2005 reported on neonatal Listeriosis.

Lately, Peer *et al.* (2010), reported Meningoencehalitis caused by *L.* *monocytogenes* in an immunocompetent previously healthy 20 month old female child.
Though *Listeriosis* and *L.monocytogenes* may not be seen as potential clinical threat in India today, the probable risk that it might pose in the year to come, cannot be ignored. As against the global trend in *Listeria* and Listeriosis research, the contribution from the Indian subcontinent is still naive.

Mode of transmission

The most common transmission route of *L. monocytogenes* to humans is via the consumption of contaminated food. However, *L. monocytogenes* can be transmitted directly from mother to child (vertical transmission), from contact with animals and through hospital acquired infections (Bell and Kyriakides 2005). Healthy individuals can be asymptomatic carriers of *L. monocytogenes*, with 0.6–3.4% of healthy people with unknown exposure to *Listeria* being found to shed *L. monocytogenes* in their faeces. However, outbreak investigations have shown that Listeriosis patients do not always shed the organism in their faeces (FDA/USDA/CDC, 2003; Painter and Slutsker, 2007). Therefore the role of healthy carriers in the transmission of *L. monocytogenes* is unclear.

Pathogenesis

Infection with *L. monocytogenes* usually follows ingestion of contaminated feed and may result in septicaemia, encephalitis or abortion. Organisms probably penetrate the M cells in Payer's patches in the intestine.

Spread occurs via lymph and blood to various tissues. In pregnant animals, infection results in transplacental transmission. There is evidence that the organism can invade through breaks in the

oral or nasal mucosa. From this site, migration in cranial nerves is thought to be the main route of infection in neural Listeriosis. Lesions in the brain stem, often unilateral, are composed of micro abscesses and peri-vascular lymphocytic cuffs. *L. monocytogenes* has the ability to invade both phagocytic and non-phagocytic cells, to survive and replicate intracellularly and to transfer from cell-to-cell without exposure to humoral defence mechanisms. Specific surface proteins, internalins, facilitated both the adherence of organisms to host membranes and their subsequent uptake. Virulent strains also possess a cytolytic toxin, listeriolysin, which destroys the membranes of phagocytic vacuoles allowing *Listeria* to escape into the cytoplasm. In the cytoplasm, the organisms utilize cellular microfilaments to generate tail-like structures which confer motility. The motile Listeria contacts the internal surface of the cytoplasmic membrane and induces pseudopod-like projections. These projections containing the bacteria are taken up by adjacent cells. The entire process is then repeated following replication of species in domestic animals (Chakraborty and Wehland, 1997).

Clinical Listeriosis

In susceptible people and animals *L. monocytogenes* can cause a life threatening, invasive disease (Vazquez-Boland *et al.,* 2001; Silk *et al.,* 2012). The main predisposing factor is decrease in cell-mediated immunity because of underlying disease or pregnancy, and the risk of Listeriosis is increased also in neonates and the elderly (Wilesmith and Gitter, 1986; Unanue, 1997; Denny and McLauchlin, 2008; Dalton *et al.,* 2011; Silk *et al.,* 2012). About 20% of invasive Listeriosis cases are fatal (Vazquez-Boland *et al.,* 2001; Silk *et al.,* 2012).

a. Human Listeriosis

In humans, 99% of Listeriosis cases are food-borne (Mead *et al.,* 1999). The clinical symptoms of invasive Listeriosis typically begin 20─30 days after the ingestion, even though incubation period can be up to 72 days (Vazquez-Boland *et al.,* 2001).

Most cases are sporadic, leading to meningitis, encephalitis, sepsis, and abortion, and reported in people with another severe underlying disease (Denny and McLauchlin, 2008; Dalton *et al.,* 2011; Silk *et al.,* 2012). Physiological reduction in cell-mediated immunity in pregnant women may result in Listeriosis with influenza-like symptoms and miscarriages (Silver, 1998). In people with no predisposing factors, invasive Listeriosis is rare, and the most typical symptom is mild gastro-enteritis with fever, headache, nausea, diarrhoea, and abdominal pain (Ooi and Lorber, 2005; Goulet *et al.,* 2012). Cutaneous and eye infections have rarely been reported, mainly in farmers and veterinarians in direct contact with afterbirths and infected foetuses (McLauchlin and Low, 1994; Regan *et al.,* 2005; Tay *et al.,* 2008). About 1% of asymptomatic humans occasionally excrete *L. monocytogenes* in their faeces (Lamont and Postlethwaite, 1986; Grif *et al.,* 2001, 2003).

In non susceptible people, food containing 1.9×10^5 colony forming units/g has been reported to cause gastroenteritis, although clearly higher infectious doses have also been reported (Vazquez-Boland *et al.,* 2001; Ooi and Lorber, 2005). Symptoms of gastroenteritis typically begin 24 hours after ingestion of the bacterium (Vazquez-Boland *et al.,* 2001; Ooi and Lorber, 2005). In the stomach, the bacterium is exposed to low gastric pH, which reduces the number of viable cells (Vazquez-Boland *et al.,* 2001).

The surviving cells pass into the intestine, passively cross the intestinal wall, proliferate mainly in Peyers patches, and spread to neighbouring enterocytes basolaterally (Vazquez-Boland *et al.,* 2001; Ooi and Lorber, 2005). The massive invasion of *L. monocytogenes* to epithelial cells is thought to cause the symptoms of gastroenteritis (Vazquez-Boland *et al.,* 2001; Ooi and Lorber, 2005). Following passage through the intestinal barrier, the bacterium enters the liver and, less extensively, the mesenteric lymph nodes and spleen through the lymph and blood (Marco *et al.,* 1992; Melton-Witt *et al.,* 2012). Kuppfer cells destroy most of the *L. monocytogenes* cells, and surviving cells start to proliferate and spread into hepatocytes (Cheers *et al.,* 1978; Gregory and Liu, 2000).

In healthy humans, the immune system destroys *L. monocytogenes* in the liver (Cheers *et al.,* 1978; Gregory and Liu, 2000). Disturbed cell-mediated immunity may enable the passage of the bacterium from the liver to the central nervous system and placenta, leading to the appearance of symptoms of invasive Listeriosis (Cheers *et al.,* 1978; Gregory and Liu, 2000; Vazquez-Boland *et al.,* 2001).

Exceptionally, other *Listeria* spp.have been reported to cause human Listeriosis (Guillet *et al.,* 2010). In these cases, symptoms have been similar to those of invasive Listeriosis cases caused by *L. monocytogenes* (Vazquez-Boland *et al.,* 2001; Guillet *et al.,* 2010). In most cases, the foetus or newborn is more likely than the mother to be affected by Listeriosis associated with pregnancy (Silver, 1998).

b. Listeriosis in Animals

Listeriosis has been detected in nearly all domestic animals (Gray and Killinger, 1966). Most Listeriosis cases have been reported in sheep, among which *L. ivanovii*

is also a significant cause of Listeria infections also in cows and goats, causing encephalitis, abortion, or septicaemia (Beauregard and Malkin, 1971; Wilesmith and Gitter, 1986; Low and Donachie, 1997; Chand and Sadana, 1999; Wesley *et al.,* 2002).

In sheep and cows, subclinical mastitis and gastroenteritis caused by *L. monocytogenes* have also been reported (Jensen *et al.,* 1996; Clark *et al.,* 2004; Rawool *et al.,* 2007). In monogastric animals, listeriosis is rare, and large epidemics with generalized listeriosis and acute deaths have been reported only in farmed chinchillas (Wesley, 2007).

In swine, the primary manifestation of Listeriosis is septicemia, whereas in horses, abortions and encephalitis is also typical (Wesley, 2007). Listeriosis of fowls is probably secondary to viral infections, and typically causes septicemia with accompanying cardiac lesions (Cummins *et al.,* 1988; Wesley, 2007). The sensitivity of pregnant animals to Listeriosis has led to epidemics in which the only symptoms were abortions (Wilesmith and Gitter, 1986). Symptomless faecal carriage of *L. monocytogenes* has been reported in primates, other mammals, and birds (Lyautey *et al.,* 2007; Hellström *et al.,* 2008; Esteban *et al.,* 2009).

Listeria monocytogenes as a food borne pathogen

The trait that makes *L. monocytogenes* difficult to control during food processing is that it can multiply over a wide range of temperatures, especially at refrigerator temperatures (Salyers and Whitt, 2002).

The classical way of enriching for *Listeria* in samples that contain other bacteria is to incubate the sample for prolonged periods of time in the refrigerator. Some studies

suggest that 1–10% of humans may be intestinal carriers of *L. monocytogenes* without exhibiting signs of illness. It is estimated by the Centres for Disease Control (CDC) that approximately 2,500 cases of Listeriosis are reported each year with 500 of the cases resulting in death (Mead *et al.,* 1999). The incidence of *L. monocytogenes* is relatively low, but the consequences of infection may be severe. Levels of *L. monocytogenes* have been recovered at greater than 10^3CFU/g. Foods containing 100 CFU/ g or greater should be considered adulterated (Robert and Greenwood, 2003).

L. monocytogenes has been found in at least 37 mammalian species, both domestic and feral, as well as at least 17 species of birds and possibly some species of fish and shellfish. Studies have identified sheep as a major reservoir of *Listeria* in nature (Rodriguez *et al.,* 1984). *L. monocytogenes* can be isolated from soil, silage, and other environmental sources. *L. monocytogenes* is quite hardy and resists the deleterious effects of freezing, drying, and heat remarkably well for a bacterium that does not form spores. *L. monocytogenes* are pathogenic to varying degrees based on the different serotypes (Anonymous, 1992). The production of sulfahydryl-activated hemolysin, listeriolysin O (α-listeriolysin) is associated with the pathogenic potential of *L. monocytogenes* (Geoffroy *et al.,* 1989; Gaillard *et al.,* 1987).

Antibiogram of *L. monocytogenes* Isolates

Yu Shu-Bing *et al.* (2004) studied the status of food contamination with *L. monocytogenes* and its resistance to antibiotics. *L. monocytogenes* strains were isolated using PCR technique. The sensitivity of *L. monocytogenes* was determined using Kirby-Bauer technique. Results showed that the contamination rate

was 5.18% (16/309). The contamination rates of uncooked meat, aquatic products, cooked meat products, and fresh milk were 8.0, 8.0, 11.51 and 3.51%, respectively.

No *Listeria* was detected in milk products and vegetables. The detection rate of *L. monocytogenes* was 1.29% (4/309). Sensitivity tests of *L. monocytogenes* to 12 antibiotics including gentamicin, vancomycin, kanamycin B, norfloxacin, ofloxacin, erythromycin, chloramphenicol, tetracycline, cephalothin and cefazolin, were carried out. The study revealed that *L. monocytogenes* was resistant to enrofloxacin and nitrofurantoin.

Chen HuiYan *et al.* (2006) examined a total of 45 food samples were examined for the presence of *Listeria* spp., of which 7 were positive using the international standard method and 9 using a modified method. All strains were identified as *L. monocytogenes* by the API method. Antibiotic susceptibility testing revealed that all strains were susceptible to vancomycin, erythromycin, midecamycin, cefazolin, sulfamethoxazole, gentamicin, and ampicillin, but were all resistant to nalidixic acid, cefotaxime, cefixime, optochin and polymyxin B.

Forty-one isolates of *L. monocytogenes*, which were obtained from raw burger patties, were tested for their susceptibility against eleven antibiotics by using standard disc diffusion method Wong *et al.* (2012). In particular, 31.7% of the isolates were found to be not resistant to any of the antibiotic tested while the rest showed resistance to at least one antibiotic. The result showed that resistance to tetracycline was the most common (46.3%), followed by erythromycin (36.6%), amikacin (31.7%), and sulfamethoxazole-trimethoprim (17.1%).

Table.1 Limits for growth of *L. monocytogenes* when other conditions are near optimum (Lado and Yousef, 2007)

Minimum

	Minimum	Optimum	Maximum
Temperature(°C)	-1.5	30–37	45
PH	4.0	6.0–8.0	9.6
Water activity	0.90	0.97	–

Table.2 Serovar of genus *Listeria*

Serotype	'O' antigen	'H' antigens
½ a	I, II, (III) a	A, B.
½ b	I, II, (III) a	A B C
½ c	II, (III) a	B D
3a	II, (III), IV	A B
3b	(III), IV a, (XII) a, (XIII) a	A B C
3c	(III) a, IV a, (XII) a, (XIII) a	B D
4a	(III)a, (V) a, VI, VII, VIII b, IX, X b, XI b, XV b	A BC
4ab	(III) a, V a, VI a, VII a, IX a, X a	A BC
4b	(III) a, V, VI, VII b, VIII b, IX b, X b c, XI b, XV b	A BC
4c	(III) a, V,VI, VII b, VIII b, IX b, X b, XI b, XV b	A BC
4d	(III) a, V,VI, VII, VIII, IX b, X b, XI b, XV b	A BC
4e	(III) a, V a, VI a, (VIII) a, (IX) a	A BC
5	(III), V,VI, VII b, VIII, IX b, X, XI b, XV b	A BC
6a	(III) a, V, VI, VII, VIII b, IX, X b, XI b, XV	A B C
6b	(III) a, V,VI, VII, VIII b, IX, X, XI, XV b	A BC
7	(III) a, XII a, XIII a	A B C

( ) means antigen not always present; a O antigen reported by Lovett (1990) and not Graves *et al.* (1999)
b O antigen reported by Graves *et al.* (1999) and not by Lovett (1990)
c Identified from outbreak in United Kingdom (McLauchlin *et al.,* 1991)

Table.3 Serotype of the *Listeria* spp. (Adapted from Hitchins, 2003)

Listeria species	Serotypes
Listeria monocytogenes	½a, ½b, ½c, 3a, 3b, 3c 4a, 4ab, 4b, 4c, 4d,4e, "7"
Listeria ivanovii	5
Listeria innocua	4ab, 6ab, 6b, Un
Listeria welshimeri	6a, 6b
Listeria seeligeri	1/2b, 4c, 4d, 6b, Un

Un: undefined

Table.4 Principle characteristics of *Listeria* species

Test	*Listeria* spp. Reaction
Gram stain	Positive
Cell morphology	Short (0.4-0.5 μm × 0.5-2.0 μm) non spore forming rod with a few peritrichous flagella
Growth conditions	Aerobic and facultative anaerobic
Motility	Positive tumbling motility at 25°C, negative at 37°C
Catalase	Positive
Oxidase	Negative
Aesculin hydrolysis	Positive
Indole	Negative
Urease	Negative

Table.5 Differentiation of *Listeria* species

Species	Beta haemolysis	Production of acid from			Christie, Atkins, Munch- Petersen (CAMP) reaction on sheep blood with	
		L-Rhamnose	D-Xylose	D-Mannitol	*S. aureus*	R. equi
L. monocytogenes	+	+	–	–	+	–
L. innocua	–	V	–	–	–	–
L. ivanovii subsp. Ivanovii	+	–	+	–	–	+
L. ivanovii subsp. Londoniensis	+	–	+	–	–	+
L seeligeri	(+)	–	+	–	(+)	–
L. welshimeri	–	V	+	–	–	–
L. grayi subsp. Grayi	–	–	–	+	–	–
L. grayi subsp. Murrayi	–	+	–	+	–	–

V- Variable, (+): weak reaction; +: >90% positive reactions; –: no reaction.
(Reference- OIE Terrestrial Manual 2014)

A surveillance study carried out by Rahimi *et al.* (2012) to determine the prevalence of *Listeria* spp. in traditional dairy products in Chahar Mahal and Bakhtiyari province, Iran. From February 2009 to February 2010, a total of 290 samples of various traditional dairy products were obtained from randomly selected retail stores located in 6 major cities of the province. Using cultural method, 21 samples (7.2%) were found positive for *Listeria* spp. The highest prevalence of *Listeria* was found in traditional ice-cream (16.7%), followed by cheese (15.0%), butter (7.5%), and kashk (2.2%) samples. The overall prevalence of *Listeria* was 7.2%, in which *L. innocua* was the most commonly recovered species (66.6%); the remaining isolates were identified as *L. monocytogenes*

(23.8%), *L. murrayi* (4.8%) and *L. seeligeri* (4.8%).

All *5 Listeria* strains identified as *L. monocytogenes* were also positive using polymerase chain reaction (PCR). Susceptibilities of the 21 strains to nine antimicrobial drugs were determined using the disk diffusion assay. All isolates were resistant to one or more antimicrobial agents. Six strains (28.6%) were resistant to a single and 5 strains (23.8%) showed resistance to two antimicrobial agents. Multi-drug resistance was established in 23.8% of Listeria strains. Resistance to nalidixic acid was the commonest finding (85.7%), followed by resistance to penicillin (47.6%), and tetracycline (33.3%).

Altuntas *et al.* (2012) aimed a study to determine the antibiotic sensitivity of *L. monocytogenes* strains isolated from animal derived foods. With disc diffusion assay, all fourteen *L. monocytogenes* strains were susceptible to the antibiotics, including penicillin G, vancomycin, tetracycline, chloramphenicol, rifampicin, erythromycin, gentamicin and trimethoprim. However, the percentages of fosfomycin and streptomycin resistances were 92.9% and 7.1%, respectively. Multiple resistances were not observed among the tested strains.

Enurah *et al.* (2013) studied the prevalence of *L. monocytogenes* in fresh raw milk and abattoir effluents in the six zones of Nigeria. Antibiotic resistant profile of the isolates was examined using the Bauer- Kirby disc diffusion assay. A total of 626 food and environmental samples were cultured on selective media out of which 54 (8.6%) were positive for *L. monocytogenes*. Chloramphenicol was the most effective antibiotic against the isolates with the least resistance (3.70%) while nalidixic acid proved to be least effective with resistance

of 90.74%. The multiple-antibiotic resistant pattern of the isolates showed nalidixic acid/cloxacillin (35.2%), nalidixic acid/colistin (31.5%) and cloxacillin/ colistin/ nalidixicacid (29.6%) to be most prominent. The least value was observed in chloramphenicol/ nitrofurantoin/ cotrimoxazole with 5.6%. The moral values of the minimum inhibitory concentrations (MICs) of the antibiotics to the isolates ranged between 4.0 and >16.0 µg/ml. Chloramphenicol, nitrofurantoin and gentamycin recorded the highest MIC compared with other antibiotics. This study has demonstrated that a wide and rapidly expanding range of undesirable and, in some cases, multi-resistant determinants is currently present in *L. monocytogenes*.

Reference

Adak, G.K., Long, S.M., O'Brien, S.J. 2002. Trends in indigenous food borne disease and deaths, England and Wales: 1992 to 2000. *Gut.,* 51: 832–841.

Altuntas, E. G., Kocan, D., Cosansu, S., Ayhan, K., Juneja, V. K., Materon, L.(2012). Antibiotic and bacteriocin sensitivity of *Listeria monocytogenes* strains isolated from different foods. *J. Food Nutr. Sci.,* 3(3): 363–368.

Amagliani, G., Brandi, G., Omiccioli, E., Casiere, A., Bruce, I.J., Magnani, M. 2004. Direct detection of *Listeria monocytogenes* from milk by magnetic based DNA isolation and PCR. *Food Microbiol.,* 21: 597–603.

Anonymous, 1992. *Listeria monocytogenes*. Bad Bug Book. U.S. Food and Drug Administration Center for food safety and applied nutrition. Foodborne pathogenic microorganisms and natural toxins handbook.

Atil, E., Ertas, H.B., Ozbey, G. 2011. Isolation and molecular characterizationof Listeria spp. from animals, food and environmental samples. *Veter. Med.,* 56: 386–394.

Bauer, A.W., Kirby, W.M., Sherris, J.C. 1966. Antibiotic susceptibility testing by a standard single disc method. *Am. J. Clin. Pathol.,* 45: 493–496.

Beauregard, M., Malkin, K.L. 1971. Isolation of *Listeria monocytogenes* from brain specimens of domestic animals in Ontario. *Can. Vet. J.,* 12: 221–223.

Bell, C., Kyriakides, A. 2005. *Listeria*: A practical appraoch to the organism and its control in foods, 2nd edn. Blackwell Publishing, Oxford.

Bhujwala R.A., Hingorani, V., Chandra, R.K. 1973. Genital Listeriosis in Delhi-A pilot study. *Ind. J. Med. Res.,* 61: 1284–1288.

Bhujwala, R.A., Hingorani, V. 1975. Perinatal Listeriosis: A bacteriological and serological study. *Indian J. Med. Res*., 63: 1503–1508.

Bille, J. 1990. Epidemiology of Listeriosis in Europe, with special reference to the Swiss outbreak. Miller, Smith and Somkuti. Pp. 71–74.

Boerlin, P., Rocourt, J., Grimont, F., Grimont, P.A.D., Jacquet, C., Piffaretti, J.C. 1992. *Listeria ivanovii* subsp. lindoniensis subsp. nov. *Int. J. Syst. Bacteriol.,* 42: 69–73.

Boerlin, P., Rocourt, J., Piffaretti, J.C. 1991. Taxonomy of the genus Listeria by using multilocus enzyme electrophoresis. *Int. J. Syst. Bacteriol.,* 41: 59–64.

Bougle, D.L., Stahl, V. 1994. Survival of *Listeria monocytogenes* after irradiation treatment of camembert. *L. Food Protect.,* 57(9): 811–813.

Broome, C.V., Gellin, B., Schwartz, B. 1990. Epidemiology of Listeriosis in the United States. Miller, Smith and Somkuti. Pp. 61–65.

Chakraborty, T., Wehland, J. 1997. The host cell infcetcd with *Listeria monocytogenes.* In: Kaufmann, S.H.E. (Ed.), Host response to intracellular pathogens. Springer, New York. Pp. 271–290.

Chand, P., Sadana, J.R. 1999. Outbreak of *Listeria ivanovii* abortion in sheep in India. *Vet. Rec.,* 145: 83–8.

Cheers, C., McKenzie, I.F., Pavlov, H., Waid, C., York, J. 1978. Resistance and susceptibility of mice to bacterial infection: course of Listeriosis in resistant or susceptible mice. *Infect. Immun.,* 19: 763–770.

Chen HuiYan, Hong ChengJi, Li Yi 2006. The detection of Listeria in food and results of drug sensitivity test. *China Trop. Med*., 6(5): 770–772.

Choi, W.S., Hong, C.H. 2003. Rapid enumeration of *Listeria monocytogenes* in milk using competitive PCR. *Int. J. Food Microbiol*., 84: 79–85.

Chugh, T.D. 2008. Emerging and re-emerging bacterial diseases in India. *J. Biosci.,* 33(4): 549–555.

Clark, R.G., Gill, J.M., Swanney, S. 2004. *Listeria monocytogenes* gastroenteritis in sheep. *N.Z. Vet. J.,* 52: 46–47.

Conner, D.E., Brackett, E.R., Beuchat, R.L. 1986. Effect of temperature, sodium chloride, and pH on growth of *Listeria monocytogenes* in cabbage juice. *Appl. Environ. Microbiol.,* 52: 59–63.

Dalton, C.B., Merritt, T.D., Unicomb, L.E., Kirk, M.D., Stafford, R.J., Lalor, K., OzFoodNet Working Group, 2011. A national case-control study of risk factors for Listeriosis in Australia. *Epidemiol. Infect.,* 139: 437–445.

Denny, J., McLauchlin, J. 2008. Human *Listeria monocytogenes* infections in Europe--an opportunity for improved European surveillance. Euro Surveill 13: pii=8082.

Doyle, M.P. 1988. Effect of Environmental and Processing Conditions on *Listeria monocytogenes*. *Food Technol.,* 42: 169–171.

Eifert J.D., Sanglay G.C. 2002. Chemistry of chlorine sanitisers in food processing. *Dairy Food Environ. Sanitation,* 22: 534–538.

Enurah, L.U., Aboaba, O.O., Nwachukwu, S.C.U., Nwosuh, C.I. 2013. Antibiotic resistant profiles of food (fresh raw milk) and environmental (abattoir

effluents) isolates of *Listeria monocytogenes* from the six zones of Nigeria. *Afr. J. Microbiol. Res.,* 7(34): 4373–4378.

Esteban, J.I., Oporto, B., Aduriz, G., Juste, R.A., Hurtado, A. 2009. Faecal shedding and straindiversity of *Listeria monocytogenes* in healthy ruminants and swine in Northern Spain. *BMC Vet. Res.,* 5: doi: 10.1186/1746-6148-5-2.

Farber J.M., Peterkin, P.I. 1991. *Listeria monocytogenes*, a food-borne pathogen. *Microbiol. Rev.,* 55: 476–511.

Farber, J.M., Coates, F., Daley, E. 1992. Minimum water activity requirements for the growth of *Listeria monocytogenes*. *Lett. Appl. Microbiol.,* 15: 103–105

FDA/USDA/CDC, 2003. Quantitative assessment of relative risk to public health from foodborne *Listeria monocytogenes* among selected categories of ready-to-eat foods. US Food and Drug Administration, Silver Spring.

Fleming, D.W., Cochi, S.L., MacDonald, K.L., Brondum, J., Hayes, P.S., Plikaytis, B.D., Holmes, M.B., Audurier, A., Broome, C.V., Reingold, A.L. 1985. Pasteurized milk as a vehicle of infection in an outbreak of Listeriosis. *New Eng. J. Med.,* 312: 404–407.

Gaillard, J.L., Berche, P., Mounier, J., Richard, S., Sansonetti, P. 1987. In vitro model of penetration and intracellular growth of *Listeria monocytogenes* in the human enterocyte-like line Caco-2. *Infect. Immunol.,* 55: 2822–2829.

Garland, C.D. 1995. Microbiological quality of aquaculture products with special reference to *Listeria monocytogenes* in Atlantic Salmon. *Food Aust.,* 47(12): 559–563.

Gellin, B.G., Broome, C.V., Broome, W.F., Bibb, W.F., Weaver, R.E., Gaventa, S., Mascola, L. 1991. The epidemiology of Listeriosis in the United States – 1986. *Am. J. Epidemiol.,* 133: 392–401.

Geoffroy, C., Gailllard, J., Alouf, J.E.,

Berche, P. 1989. Production of thiol-dependent hemolysins by *Listeria monocytogenes* and related species. *J. Genetic Microbiol.,* 135: 481–487.

Gilot, P., Content, J. 2002. Specific identification of *Listeria welshimeri* and *Listeria monocytogenes* by PCR assays targeting a gene encoding a fibronectin-binding protein. *J. Clin. Microbiol.,* 40: 698−703.

Gogate, A.A., Deodhar, L.P. 1981. Meningitis due to *Listeria monocytogenes*: (a case report). *J. Postgrad. Med.*, 27: 240–2.

Goulet, V., Hebert, M., Hedberg, C., Laurent, E., Vaillant, V., De Valk, H., Desenclos, J.C. 2012. Incidence of Listeriosis and related mortality among groups at risk of acquiring Listeriosis. *Clin. Infect. Dis.,* 54: 652–660.

Goulet, V., Jacquet, C., Vallant, V., Rebière, I., Mouret, E., Lorente, C., Maillot, E., Stainer, F., Rocourt. J. 1995. Listeriosis from consumption of raw-milk cheese. *Lancet,* 345: 1501–1502.

Graves, L.M., Helsel, L.O., Steigerwalt, A.G., Morey, R.E., Daneshvar, M.I., Roof, S.E., Orsi, R.H., Fortes, E.D., Millilo, S.R., Henk C. den Bakker, Wiedmann, M., Swaminathan, B., Sauders, B.D. 2009. *Listeria marthii* sp. nov., isolated from the natural environment, Finger Lakes National Forest. *Intern. J. Syst. Evol. Microbiol.,*

Graves, L.M., Swaninathan, B., Hunter, S.B. 1999. Subtyping *Listeria monocytogenes*. In: Ryser, E.T., Marth, E.H. (Eds), *Listeria,* Listeriosis, and Food Safety. Marcel Dekker, Inc, New York. Pp. 279−297.

Gray, M.L., Killinger, A.H. 1966. *Listeria monocytogenes* and Listeric infections. *Bacteriol. Rev.,* 30: 309–382.

Gregory, S.H., Liu, C.C. 2000. CD8+ Tcell-mediated response to *Listeria monocytogenes* taken up in the liver and replicating within hepatocytes. *Immunol. Rev.,* 174: 112–122

Grif, K., Hein, I., Wagner, M., Brandl, E., Mpamugo, O., McLauchlin, J., Dierich,

M.P., Allerberger, F. 2001. Prevalence and characterization of *Listeria monocytogenes* in the feces of healthy Austrians. *Wien Klin Wochenschr,* 113: 737–742.

Grif, K., Patscheider, G., Dierich, M.P., Allerberger, F. 2003. Incidence of fecal carriage of *Listeria monocytogenes* in three healthy volunteers: a one- year prospective stool survey. *Eur. J. Clin. Microbiol. Infect. Dis*., 22: 16–20.

Guillet, C., Join-Lambert, O., Le Monnier, A., Leclercq, A., Mechai, F., Mamzer-Bruneel, M. F., Bielecka, M. K., Scortti, M., Disson, O., Berche, P., Vázquez-Boland, J., Lortholary, O., Lecuit, M. 2010. Human Listeriosis caused by *Listeria ivanovii*. *Emerg. Infect. Dis.,* 16: 136–138.

Gupta, R., Chandra, R.K., Hingorani, V. 1977. Listeriosis: an opportunistic infection. *Indian Paediatrics,* 34: 732–734.

Gupta, V., Gautam, V., Mehta, N., Kumari, I., Joshi, R.M. 2003. Listeriosis in second trimester of pregnancy: Case report from India. *J. Infect. Dis*., 56: 60–61.

Hellström, S., Kiviniemi, K., Autio, T. And Korkeala, H. 2008. *Listeria monocytogenes* is common in wild birds in Helsinki region and genotypes are frequently similar with those found along the food chain. *J. Appl. Microbiol.,* 104: 883–888.

Hird, D.W., Genigeorgis, C. 1990. Listeriosis in food animals: Clinical signs and livestock as a potential source of direct (non foodborne) infection for humans. Miller, Smith and Somkuti. Pp. 31–39.

Hitchins, A.D. 2003. Detection and enumeration of *Listeria monocytogenes* in foods. Bacteriological analytical manual online. U. S. department of Health and Human Services, U.S. Food and Drug Administration Center for Food Safety and Applied Nutrition. Chapter 10.

International Organization for Standerdization, 1996. Microbiology of food and animal feeding stuffs-Horizontal method for detection and enumeration of *Listeria monocytogenes*-part-1: Detection method. Internatinal Standard ISO11290-1, Geneva, Switzerland.

Jemmi, T., Keusch, A. 1994. Occurrence of *Listeria monocytogenes* in Freshwater Fish Farms and Fish - Smoking Plants. *Food Microbiol.,* 11: 309–316.

Jensen, N.E., Aarestrup, F.M., Jensen, J., Wegener, H.C. 1996. *Listeria monocytogenes* in bovine mastitis. *Int. J. Food Microbiol.,* 32: 209–216.

John G. Holt (Ed.), 1994. Bergey's manual of determination of bacteriology, 9th edn. Williams and Wilkins, Baltimore.

Johnson J.L., Doyle M.P., Cassens R.G. and Schoeni, J.L. (1988). Fate of *Listeria monocytogenes* in tissue of experimentally infected cattle and in hard salami. *Appl. Environ. Microbiol.,* 54(2): 497–501

Kela, E., Holmström, P. 2001. Infectious diseases in Finland 2000. Publications of the National Public Health Institute. KTL B 10/2001. 27

Lado, B., Yousef, A.E. 2007. Characteristics of *Listeria monocytogenes* important to food processors, Chapt. 6. In: Ryser, E.T., Marth, E.H. (Eds), Listeria, listeriosis and food safety, 3rd edn, CRC Press Taylor and Francis Group, Boca Raton. Pp. 157– 213.

Lamont, R.J., Postlethwaite, R. 1986. Carriage of *Listeria monocytogenes* and related species in pregnant and non-pregnant women in Aberdeen, Scotland. *J. Infect.,* 13: 187–193.

Linnan, M.J., Mascola, L., Lou, X.D., Goulet, V., May, S., Salminen, C., Hlird, D.W., Yonekura, M.L., Hayes, P., Weawer, R., Audurier, A., Plikaytis, B.D., Fannin, S.L., Kleks, A., Broome, C.V. 1988. Epidemic Listeriosis associated with Mexican-style cheese. *N. Engl. J. Med*., 319: 823–828.

Loncarevic, S., Danielsson-Tham, M.L.,

Mårtensson, L., Rigner, Å., Runehagen, A., Tham, W. 1997. A case of foodborne Listeriosis in Sweden. *Lett. Appl. Microbiol.,* 24: 65–68.

Lovett, J. 1989. *Listeria monocytogenes*. In: Doyle, M. P. (Ed.), Foodborne bacterial pathogens. Marcel Dekker Inc., New York, NY. Pp. 284–310.

Lovett, J. 1990. Taxonomy and general characteristics of *Listeria* species. In: Foodborne listeriosis, Miller, A.J., Smith, J.L., Somkuti, G.A. (Eds). Society of Industrial Microbiology, Elsevier, New York. Pp. 9–12.

Low, J.C., Donachie, W. 1997. A review of *Listeria monocytogenes* and listeriosis. *Vet. J.* 153: 9–29.

Lukinmaa, S., Miettinen, M., Nakari, U. M., Korkeala, H., Siitonen, A. 2003. *Listeria monocytogenes* isolates from invasive infections: variation of sero and genotypes during an 11-year period in Finland. *J. Clin. Microbiol.,* 41: 1694–1700.

Lyautey, E., Hartmann, A., Pagotto, F., Tyler, K., Lapen, D. R., Wilkes, G., Piveteau, P., Rieu, A., Robertson, W. J., Medeiros, D.T., Edge, T.A., Gannon, V., Topp, E. 2007. Characteristics and frequency of detection of fecal *Listeria monocytogenes* shed by livestock, wildlife, and humans. *Can. J. Microbiol.,* 53: 1158–1167.

Lyytikäinen, O., Autio, T., Maijala, R., Ruutu, P., Honkanen-Buzalski, T., Miettinen. L., Hatakka, M., Mikkola, J., Anttila, V.J., Johansson, T., Rantala, T., Aalto, H., Korkeala, Siitonen, A. 2000. An outbreak of *Listeria monocytogenes* serotype 3a from butter in Finland. *J. Infect. Dis.,* 181: 1838–1841.

Marco, A.J., Prats, N., Ramos, J.A., Briones, V., Blanco, M., Dominguez, L., Domingo, M. 1992. A microbiological, histopathological and immune-histological study of the intra gastric inoculation of *Listeria monocytogenes* in mice. *J. Comp. Pathol.,* 107: 1–9.

McCarthy, S.A. 1990. Listeria in the environment. Miller, Smith and Somkuti. q.v. 25–29.

McLauchlin, J. 1996. The role of Public Health Laboratory Service in England and Wales in the investigation of human Listeriosis during the 1980s and 1990s. *Food Control,* 7: 235–239.

McLauchlin, J., Hill, S.M., Velani, S.K., Gilbert, R.J. 1991. Human Listeriosis and Pâté: A possible association. *Br. Med. J.,* 303: 773–775.

McLauchlin, J., Low, J.C. 1994. Primary cutaneous Listeriosis in adults: an occupational disease of veterinarians and farmers. *Vet. Rec.,* 135: 615–617.

Mead, P.S., Slutsker, L., Diets, V., McCaig, L.F. Bresee, J.S., Shapiro, C., Griffin, P.M., Tauxe, R.V. 1999. Food- related illness and death in the United States. *Emerg. Infect. Dis.,* 5: 607–625

Melton-Witt, J.A., Rafelski, S.M., Portnoy, D.A., Bakardjiev, A.I. 2012. Oral infection with signature-tagged *Listeria monocytogenes* reveals organ specific growth and dissemination routes in guinea pigs. *Infect. Immun.,* 80: 720–732.

Miettinen, M.K., Siitonen, A., Heiskanen, P., Haajanen, H., Björkroth, K.J., Korkeala, H. 1999. Molecular epidemiology of an outbreak of febrile gastroenteritis caused by *Listeria monocytogenes* in cold-smoked rainbow trout. *J. Clin. Microbiol.,* 37: 2358–2360.

Murray, E.G.D., Webb, R.A., Swann, M.B.R. 1926. A disease of rabbits characterised by a large mononuclear leucocytosis, caused by a hitherto undescribed bacillus Bacterium monocytogenes (n.sp.). *J. Pathol. Bacteriol.,* 29(4): 407–439. doi: 10.1002/path.1700290409

National Committee for Clinical Laboratory Standards, 1993. Performance standards for antimicrobial susceptibility tests, 4th edn, NCCLS. 10(7).

OIE Terrestrial Manual, 2014.

Ooi, S.T., Lorber, B. 2005. Gastroenteritis due to *Listeria monocytogenes*. *Clin. Infect. Dis.,* 40: 1327–1332.

Painter, J., Slutsker, L. 2007. Listeriosis in humans, Chapt. 4. In: Ryser, E.T., Marth, E.H. (Eds), Listeria, listeriosis and food safety, 3rd edn. CRC Press Taylor and Francis Group, Boca Raton. Pp. 85–109.

Peel, M., Donachie, W., Shaw, A. 1988. Temperature-dependent expression of flagella of *Listeria monocytogenes* studied by electron microscopy, SDS-PAGE and western blotting. *J. Gen. Microbiol.,* 134: 2171–2178.

Peer, M.A., Nasir, R.A., Kakru, D.K., Fomda, B.A., Wani, M.M. and Hakeem, Q.N. 2010. *Listeria monocytogenes* meningo-encephalitis in an immunocompetent, previously healthy 20-month old female child. *Indian J. Med. Microbiol.,* 28: 169–171.

Pirie, J.H.H. 1927. A new disease of veldt rodents "Tiger Rover Disease". *Publ. S. Afr. Inst. Med. Res.,* 3: 163–186.

Rahimi, E., Momtaz, H., Sharifzadeh, A., Behzadnia, A., Ashtari, M.S., Zandi Esfahani, S., Riahi, M., Momeni, M. 2012. Prevalence and antimicrobial resistance of *Listeria* species isolated from traditional dairy products in Charar Mahal and Bakhtiary, Iran. *Bulg. J. Vet. Med.,* 15(2): 115−122.

Rawool, D.B., Malik, S.V., Shakuntala, I., Sahare, A.M., Barbuddhe, S.B. 2007. Detection of multiple virulence associated genes in *Listeria monocytogenes* isolated from bovine mastitis cases. *Int. J. Food Microbiol.,* 113: 201–207.

Reed, R.W. 1958. Listeria and Erysipelothrix. In: Dubos, R.J. (Ed). Bacterial and mycotic infections in man, 3rd edn. J.B. Lippincott, Philadelphia, PA. Pp. 453–462.

Regan, E.J., Harrison, G.A., Butler, S., McLauchlin, J., Thomas, M., Mitchell, S. 2005. Primary cutaneous listeriosis in a veterinarian. *Vet. Rec.,* 157: 207.

Ritz, M., Tholozan, J.L., Federighi, M., Pilet, M.F. 2001. Morphology and physiological characterization of L. monocytogenes subjected to high hydrostatic pressure. *Appl. Environ. Microbiol.,* 67(5): 2240–2247.

Roberts, A.J., Williams, S.W., Wiedmann, M., Nightingale, K.K. 2009. *Listeria monocytogenes* outbreak strains demonstrate differences in invasion phenotypes, in lA transcript levels and motility. *Appl. Environ. Microbiol.,* 75(17): 5647–5658.

Roberts, D., Greenwood, M. 2003. *Listeria monocytogenes*, In: Practical food microbiology, 3rd edn. Blackwell Publishing, Massachusetts. Pp. 273–274.

Rocourt, J., Boerlin, P., Grimont, F., Jacquet, C., Piffaretti, J.C. 1992. Assignment of Listeria grayi and Listeria murrayi to a single species, Listeria grayi, with a revised description of Listeria grayi. *Int. J. Syst. Bacteriol.,* 42: 69–73.

Rocourt, J., Catimel, B. 1985. Biochemical characterization of species in the genus Listeria. *Zentralble Bakteriologika Mikrobiologi Hygiene,* 260: 221–231.

Rodriguez, L.D., Fernandez, G.S., Fernandez, J., Garayzabel, F., Ferri, E.R. 1984. New methodology for the isolation of *Listeria monocytogenes* from heavily contaminated environments. *Appl. Environ. Microbiol.,* 47: 118–1190.

Rossmanith, P., Krassnig, M., Wagner, M., Hein, I. 2006. Detection of *Listeria monocytogenes* in food using a combined enrichment/real-time PCR method targeting the prf A gene. *Res. Microbiol.,* 157: 763–771.

Ryser, E.T., Marth, E.H. (Eds), 1991. Listeria, listeriosis, and Food Safety. Marcel Dekker, New York, NY. 632 Pp.

Salvat, G., Toquin, M.T., Michel, Y., Colin, P. 1995. Control of *Listeria monocytogenes* in the delicatessen industries: lessons of a Listeriosis outbreak in France. *Int. J. Food Microbiol.,* 25: 75–81.

Salyers, A.A., Whitt, D.D. 2002. *Listeria monocytogenes*, A doubly motile pathogen. In: Bacterial pathogenesis, a

molecular approach, 2nd edn. ASM Press, Washington, D.C.

Schafer, M., Murphy, M., Boyle, L. 2000. Controlling *Listeria* in your facility. Meat Processing News, Dept. Animal Science and Industry, Kansas State University, United States, Summer/Fall Issues.

Schlech, W.F., Lavigne, P.M., Bortolussi, A.C., Allen, A.C., Haldane, E.V., Wort, A.J., Hightower, A.W., Johnson, S.E., King, S.H., Nicholls, E.S., Broome, C.V. 1983. Epidemic Listeriosis: Evidence for transmission by food. *N. Engl. J. Med.,* 308: 203–206.

Schwartz, B., Ciesielski, C.A., Broome, C.V. 1988. Association of sporadic Listeriosis with consumption of uncooked hot dogs and undercooked chicken. *Lancet,* 2: 779–782.

Seeliger H.P.R., Hohne, K. 1979. Serotyping of *Listeria monocytogenes* and related species. *Methods Microbiol.,* 13: 31–49.

Seeliger H.P.R., Jones, D. 1984. Genus Listeria. In: Sneath, P.H.A., Maier, N.S., Sharpe, M.E., Holt, J.G. (Eds.), Bergey's manual of systematic bacteriology. Williams and Wilkins, Baltimore, U.S.A.

Seeliger, H.P.R., Jones, D. 1986. Genus Listeria. In: Sneath, P.H.A., Mair, N.S., Sharp, M.E., Holt, J.G. (Eds.), Bergey's manual of systematic bacteriology. Williams and Wilkins, Baltimore. Pp. 1235–1245.

Silk, B.J., Date, K.A., Jackson, K.A., Pouillot, R., Holt, K.G., Graves, L.M., Ong, K.L., Hurd, S., Meyer, R., Marcus, R., Shiferaw, B., Norton, D. M., Medus, C., Zansky, S.M., Cronquist, A.B., Henao, O.L., Jones, T.F., Vugia, D.J., Farley, M.M., Mahon, B.E. 2012. Invasive listeriosis in the foodborne diseases active surveillance network (FoodNet), 2004–2009: further targeted prevention needed for higher-risk groups. *Clin. Infect. Dis.,* 54(S5): S396–40.

Silver, H.M. 1998. Listeriosis during pregnancy. *Obstet. Gynecol. Surv.,* 53: 737–740.

Singh, R. 2011. Indian Dairy and Products Annual Report 2010. USDA Foreign Agricultural Service: Global Agricultural Information Network. Retrieved 16 June 2011, from static.globaltrade.net/files/pdf/20110226 231255627.pdf.

Stuart, M.R., Pease, P.E. 1972. A numerical study on the relationships of Listeria and Erysipelothrix. *J. Gen. Microbiol.,* 73: 551–565

Stuart, S.E., Welshimer, H.J. 1974. Taxonomic re-examination of Listeria Pirie and transfer of Listeria grayi and Listeria murrayi to a new genus Murrayi. *Int. J. System. Bact.*, 23

Tay, E., Rajan, M., Tuft, S. 2008. *Listeria monocytogenes* sclerokeratitis: a case report and literature review. *Cornea,* 27: 947–949.

Thomas, S., Verma, I.C., Singh, M., Bhujwala, R.A. 1981. Study of neonatal Listeriosis in North India. *Ind. J. Med. Res.,* 73: 28–32.

Todar, K. 2009. "*Listeria monocytogenes*". Todar/ online textbook of bacteriology. n page, http:// textbookof bacteriology.net/Listeria.html

Tompkin, R.B., Scott, V.N., Bernard, D.T., Sveum, W.H., Sullivan, G.K. 1999. Guidelines to prevent post-processing contamination from *Listeria monocytogenes*. *Dairy Food Environ. Sanitation,* 19: 551–562.

Unanue, E.R. 1997. Studies in Listeriosis show the strong symbiosis between the innate cellular system and the Tcell response. *Immunol. Rev.*, 158: 11–25.

USDA, 1999. United States Department of Agriculture. Consumer information from USDA, Food Safety Education Office, Food Safety and inspection service, USDA, Washington, D.C 20250–3700, USA.

Usha, K. Desai, M.W. and Daftary, V.G. (1966). Listeriosis-A clinical and bacteriological study. *J. Obst. Fr. Gynec. India,* 16: 304–306.

Vazquez-Boland, J.A., Kuhn, M., Berche, P. 2001. Listeria pathogenesis and molecular virulence determinants. *Clin. Microbiol. Rev.,* 14: 584–640.

Vishwanathan, G.R., Lyyar, V.V. 1950. Circling disease of sheep in the Madras states. *Indian Vet. J.,* 26: 395.

Wagner, M., Podstatzky-Lichtens, L., Lethner, A., Asperger, H., Baumgarther, W., Brand, E. 2000. Prolonged excretion of *Listeria monocytogenes* in a subclinical cases of mastitis. *Milchwisschaft,* 55: 3–6.

Wesley, I.V. 2007. Listeriosis in animals. In: Ryser, E.T., Marth, E.H. (Eds), Listeria, listeriosis and food safety. 3rd edn. CRC Press, Boca Raton, FL, USA. Pp. 55–84.

Wesley, I.V., Larson, D.J., Harmon, K.M., Luchansky, J.B., Schwartz, A.R. 2002. A case report of sporadic ovine listerial menigoencephalitis in Iowa with an overview of livestock and human cases. *J. Vet. Diagn. Invest.,* 14: 314–321.

Wilesmith, J.W., Gitter, M. 1986. Epidemiology of ovine Listeriosis in Great Britain. *Vet. Rec.,* 119: 467–470.

Wong, W.C., Pui, C.F., Tunung, R., Ubong, A., Noor Hidayah, M.S., Farinazleen, M.G., Noorlis, A., Cheah, Y.K., Son, R. 2012. Antibiogram pattern among cultures of *Listeria monocytogenes* isolated from frozen burger patties in Malaysia. *Pertanika J. Trop. Agric. Sci.,* 35(4): 793–804.

Yu Shu-Bing, Liang Jing Tao, Zhou Qiang Zhong, 2004. Isolation of *Listeria monocytogenes* from food and test of drug susceptibility. *J. China Trop. Med.,* 4(4): 515, 516, 534.