



International Journal of Scientific Research Letters

Journal Homepage: <http://cphfs.in/research.php>



Prevalence and risk of listeriosis by ready to eat products

Chaitali Ghosh

Department of Zoology, Gargi
College, University of Delhi, Delhi,
110049, India

ghoshchaitali@gmail.com

Harish Kumari

School of Biotechnology, Gautam Buddha
University, Greater Noida, 201310, India

Jitendra Singh Rathore

School of Biotechnology, Gautam
Buddha University, Greater Noida,
201310, India

Abstract - Listeriosis is one of the most serious food borne diseases of our society due to the harshness of manifestations (septicaemia, meningitis and foetal death) with a case-fatality rate ranging from 20% to 50%. An opportunistic, gram positive, food-borne pathogen named *L. monocytogenes* are universally distributed throughout the natural environment and is also present in many animals and plants. Its omnipresent distribution and psychrotrophic aspect allows the pathogen to persist and develop in different types of refrigerated ready-to-eat products (RTE). The augmentation of the RTE product's use (which do not require cooking before consumption and has an increased shelf-life at refrigerated temperatures), determines the pervasiveness of *Listeria* spp. Mainly the species *Listeria monocytogenes* is found in the RTE foods and because of the susceptibility of a large population, there is an increase in listeriosis in European countries. In pre-treatment processing plants, *Listeria monocytogenes* pathogen can be controlled in ready to eat products by detecting the species using different selective culture media. Both *Listeria* selective agar and PALCAM agar showed low sensitivity and specificity for *L. monocytogenes* compared to CHROM agar. In this review we will discuss about the pre-treatment processing and control of *Listeria monocytogenes* in ready to eat products.

Keywords: Epidemiology, *Listeria monocytogenes*, revealed, persists, listeriosis, ready to eat product (RTE).

I. INTRODUCTION

Listeria is a gram-positive, rod shaped bacteria that are found as single unit and also as short chains. It is a facultative anaerobic, motile, non-spore and capsule forming bacteria. It is an opportunistic intracellular bacterial pathogen. *Listeria* includes six species- *Listeria monocytogenes*, *Listeria innocua*, *Listeria seeligeri*, *Listeria welshimeri*, *Listeria ivanovii* and *Listeria grayi* [1]. The size of *Listeria monocytogenes* is 0.4-2µm. The genome size of *L. monocytogenes* is 2905 kb having a low GC content. This gram-positive bacterium is closely related to the genus *Bacillus*, *Clostridium*, *Enterococcus* and *Staphylococcus* [2]. The presence of *Listeria monocytogenes* has been widely observed in food and ready to eat products and various environmental and clinical samples [3]. Growth is further enhanced by exposure to cold temperature and it grows quite well at 4°C. They are omnipresent in nature and can readily contaminate ready to eat products such as meat, seafood, cheese, cut vegetables, and other dairy products, throughout or subsequent to the processing step. This is the primary step of transmission to humans i.e. by the consumption of contaminated food [4]. There are six virulence factors responsible for the invasion and replication of *L. monocytogenes* inside a mammalian cell. It acts as an

intracellular parasites because of the virulence gene cluster (*prfA*, *plcA*, *mpl*, *hly*, *actA* and *plcB*) which are physically linked in a chromosomal island formally known as the *hly* or *prfA*-dependent virulence cluster or LIPI-1 (*Listeriopathogenicity island 1*). The *Listeria* genus includes three hemolytic species (*L. monocytogenes*, *L. ivanovii* and *L. seeligeri*) all of which contains LIPI-1 but only two of these species (*L. monocytogenes* and *L. ivanovii*) are potentially pathogenic [5].

II. LISTERIA MONOCYTOGENES

These pathogens are widely dispersed through-out the environment especially the species *L. monocytogenes*. They can be isolated from soil plant material, decaying vegetables, silage, animal and marine fish, sea food, animal feed, and food processing environment [6]. It is present in particularly hazardous chilled processed food products with a long shelf life, for example - smoked fish, hot dog, soft cheese, ready to eat meat and partly cooked products. This pathogen is able to grow slowly at very low temperatures and multiply to dangerous level in refrigerated food, unless controlled [7, 8]. This pathogen is also common coloniser in the food factories. They form a biofilm and therefore are very difficult to remove. Asymptomatic carriers are human and animals [9, 10]. *L.*

monocytogenes pathogen can survive and grow saprophytically also, and the primary habitat is decaying vegetable matter [11].

A. Pathogenic properties of *Listeria monocytogenes*

L. monocytogenes are intracellular facultative microbes which survive in macrophages and can penetrate a variety of non-phagocytic cells, like epithelial cells, hepatocytes and endothelial cells [12]. When we consume the contaminated food with *L. monocytogenes*, it enters in the primary site i.e. gastrointestinal tract. At a particular time after enrichment *Listeria* is engulfed in a phagocytic vacuole which becomes acidified as soon as it takes up *Listeria*. Within few minutes, phagosome membrane disruption is mediated by the hemolysin and phospholipase of the bacteria, and thus the *Listeria* spreads freely in the cytoplasm [13]. The cytosol *Listeria* is able to multiply, indicating that the cytoplasmic compartment is permissive for *Listeria* proliferation. *L. monocytogenes* are surrounded by a fibrillar material that are composed of actin filaments which are arranged to form an actin tail, which thus allows its movement up to the cell, leading and pushing to the formation of finger like protrusions with the bacterium at the tip [14,15]. The pseudopodia are inserted by disinfected neighbouring cell that in turn are engulfed by phagocytosis, resulting in the formation of secondary phagosomes determined by double membrane. Bacteria outbreak rapidly and admit to a new intracellular cycle [16]. *L. monocytogenes* crosses the intestinal barrier; its cells are dissipated into the host by the lymph or blood, meeting the mesenteric lymph nodes, spleen and liver [17]. The minimum dose required to cause clinical infection in humans has not been determined, but a large number of *L. monocytogenes* detected in food product that are responsible for wide ranging case of listeriosis has 106 CFU/g. Very low doses may also cause infection, specially in the high risk group [18]. The level of contamination as low as 102 or 103 CFU/g have been linked with clinical cases. The infection may vary depending on the virulence of the strain and the host aspects. Some evidences comes from the fact that only 3 of the 13 known serotypes of *Listeria monocytogenes* (1/2a, 1/2b and 4b) are responsible for majority of the human and animal cases of listeriosis worldwide [19]. Mostly some serotypes are found in food product [20].

III. LISTERIOSIS

This disease is transmitted by contaminated food consumption that affects humans and animals. *L. monocytogenes* and *L. ivanovii* are two pathogenic species which penetrate the host cells. The humans and animals are infected by *L. monocytogenes*, *L. ivanovii* is an animal pathogen principally linked to disease in ruminants [21].

A. Types of listeriosis

For both wide ranging and sporadic cases, the major source of infection is contaminated food. [22, 23]. *Listeria* primarily causes infection of the central nervous system (meningitis, meningoenzephalitis, brain abscess, cerebritis). These bacteria infect the immuno-compromised ones, like in the case of pregnant women, newborn and the elderly. *Listeria* is everywhere and these microbes are primarily transmitted by the oral route after ingestion of contaminated food product, which enters the intestinal tract to cause systemic infection. In some cases non-pregnant adults having listeriosis disease are

associated with at least one of the following conditions: malignancies (leukaemia, lymphoma), chemotherapy, immunosuppressant therapy (organ transplantation or corticosteroid treatment), chronic liver disease (alcoholic or cirrhosis), kidney disease and diabetes. The diagnosis of the disease requires the isolation of the organism from the blood and/or the cerebrospinal fluid. The treatment includes prolonged administration of antibiotics, primarily ampicillin and gentamicin, to which the organism is usually susceptible.

B. Two types of infection are caused by *Listeria monocytogenes*

1) *Non-invasive listeriosis* - The *Listeria* microbes cause gastroenteritis disease besides the typical symptoms of fever, diarrhoea and vomits. Basically all the population exposed to this pathogen is capable of developing the disease. This type of infection usually begins relatively 20 h after ingestion of heavily contaminated food [24].

2) *Invasive listeriosis* - Is recognized as a severe food borne disease [25]. The growth period for the aggression of diseasedness is primarily much longer than the gastrointestinal form i.e. between 20 to 30 days. The perinatal listeriosis and invasive listeriosis are present in the adult patients, the prevalent forms of proposal responding to display infection (bacteraemia or septicaemia), the confined infection in the central nervous system or local infection in the (meningitis or meningoenzephalitis). There are more conventional forms of the infection such as endocrine, myocarditis, arteritis, localized abscesses or osteomyelitis [26]. In the case of foeto-maternal and neonatal listeriosis, this disease commonly penetrates the foetus via placenta [27]. The symptoms include abortion, birth of a stillborn foetus, granulomas in many internal organs (baby with generalized infection) or meningitis in the neonate. The diseased women usually continue asymptomatic or they can represent flu like disorder with chills, fatigue, headache and muscular pain generally prolonged for 2 to 14 days before abortion.

C. Manifestations of listeriosis

In infected pregnant women, the bacteria's are mostly located in the uterus, the central nervous system or blood. The primary disease of the intestinal tissue by *L. monocytogenes* leads to encouragement of alternatively sterile body sites. The non-pregnancy related to the proportion of bacteraemic forms has increased and produced at least two third of the cases. It regularly occurs in patients with a latent disease, when in fact CNS infection also appears in the previously healthy persons. Sequelae may follow listeriosis disease, but their incidence is rarely estimated. Up to 11% of neonates and 30% of survivors of CNS disease suffer from residual symptoms, and psychiatric sequelae have also been reported [28].

A classification and arrangement has been designed for differentiating the manifestations of syndromes associated with *L. monocytogenes* so that it takes into consideration host status, route of transmission, severity and growth period. It has been shown that approximately 20% of the population may belong to groups with a greater risk for developing listeriosis [29]. These higher-risk people can be divided into non-perinatal and perinatal groups.

D. Non-perinatal infection

The listeriosis usually presents as either CNS infections, efficient in non-pregnant humans along or without bacteraemia, or bacteraemia alone. Cases are confined to the immuno-compromised or elderly. Adding to these clinical manifestations, less common manifestations involve peritonitis, hepatitis and liver abscess, endocarditis, arterial disease, myocarditis, lung and pleural fluid disease, septic arthritis and osteomyelitis, chorioretinitis, endophthalmitis and corneal ulcer [30]. Although the fact that disease can be handled successfully with antibiotics, still between 20% and 40% of the cases are fatal. The case-fatality rate may approach 75%. These are the seriously immuno-compromised patients.

E. Perinatal (prenatal/neonatal) infection

The pregnant women and their foetuses or newborns are considered in the perinatal group. Infected pregnant women will suffer from prodromal influenza-like illness, which involves fever, chills and headache. About three to seven days after the beginning of prodromal symptoms, a woman has abortions or a premature labour. 30% of pregnant women are reported to have sepsis or fever by listeriosis [31].

Although severe infections appear in adults and children, listeriosis is commonly superimposed on another illness. The high-risk cases primarily consist of persons with chronic debilitating illnesses that impairs their immune system, such as cancer, diabetes or alcoholism; HIV/AIDS; persons taking immunosuppressive medication (e.g. immune suppressors taken by transplant patients); and person over the age of 60-65, particularly individuals with pre-existing, debilitating medical conditions [32]. Healthy children and immuno-competent adults have a low risk of severe infection from *L. monocytogenes*. There have also been a number of outbreaks where the majority of cases developed mild symptoms, such as diarrhoea, fever, headache and myalgia. These outbreaks have generally involved the ingestion of high doses of *L. monocytogenes* by otherwise healthy individuals and these gastroenteritis symptoms generally get self-resolved within a few days.

F. Febrile gastroenteritis

Natural signs and syndrome are associated with febrile listerial gastroenteritis which involves chills, fever, diarrhoea, headache, abdominal pain and cramps, nausea, vomiting, fatigue, joint and muscle pain, and myalgia. *L. monocytogenes* disease manifestation may be limited to these symptoms in alternatively healthy individuals. Mild expression associated with listeriosis has been reported in several countries, and a different variety of foods have been suspected as the vehicle of infection. There are insufficient data available about the incidence of the milder symptoms to allow the impact of this biological end point on public health to be assessed in the current exercise [33].

G. Incidence of human listeriosis

Since the year 2000, there has been a reported increase in the cases of listeriosis in several European countries [34]. Ingestion of *L. monocytogenes* is probably very common, because of the universal distribution of this bacteria and the high number of contamination of raw and industrially processed foods. Still, the tendency of human listeriosis is low in

comparison with other bacteria like *Salmonella* or *Campylobacter*. The European Union announced cases of human listeriosis as 0.3 per 100000 citizens in 2007 [35]. The highest declaration ratio is reported in Denmark (1.1), Finland (0.8), Sweden (0.6) and Luxembourg (0.6).

IV. READY TO EAT PRODUCTS

Ready-to-eat products (RTE) are commercial and processed food and can be defined as a "food designed by the constructor or the manufacturer for direct human consuming without the use of cooking". The *Listeria monocytogenes* have psychrotrophic character and are spread and distributed throughout the environment and thus its high prevalence in different kinds of refrigerated RTE products. Though low temperatures can reduce the growth rate of this pathogen, but it is known that *L. monocytogenes* is able to grow at refrigerating temperatures [36]. Because these RTE products are usually cooked during process, some high risks are present to the consumers due to viable cross contamination with the pathogen and further growth. Consumption of RTE products have increased due to the advance in lifestyle and there are difficulties in controlling the temperature during the global exchange and distribution of these products and thus could be the reason for the observed increment of listeriosis over the past few years. *L. monocytogenes* can recover for long periods of time in a hospitable environment due to its capacity to prevent various stresses and its capability to form biofilm.

A. Foods associated with food-borne listeriosis

Food is the principle route of transmission of listeriosis (WHO, 1988). Listeriosis cases are observed in conjunction with both common-source outbreaks and individual sporadic cases. Foods that are mostly affected include RTE products that (i) support growth of *L. monocytogenes*, (ii) have a long refrigerated shelf-life, and (iii) are consumed without further listericidal activity. These include products that receive a listericidal treatment but are subjected to post-processing recontamination. This also includes cross-contamination in both the retail and home setting. Cross-contamination is also suspected at the distribution level. Common source outbreaks have been associated or linked epidemiologically with the consumption of Hispanic-style soft cheeses (queso fresco); soft, semi-soft and mould-ripened cheese; hot dogs; pork tongue in jelly; processed meats; paté; salami; pasteurized chocolate flavoured milk; pasteurized milk; unpasteurized milk; butter; cooked shrimp; smoked salmon; maize and rice salad; maize and tuna salad; potato salad; raw cut vegetables [37]. In addition, sporadic cases have been linked to the consumption of raw milk; unpasteurized ice cream; ricotta cheese; goat, sheep and feta cheeses; soft, semi-soft and mould-ripened cheeses; Hispanic-style cheese; salami; hot dogs; salted mushrooms; smoked cod roe; smoked mussels; undercooked fish; pickled olives; and raw vegetables. In general, the levels of *L. monocytogenes* in the implicated food have been greater than 103 CFU/g, but there has been instances where the observed level of *L. monocytogenes* in the implicated food has been substantially lower [38]. There is a great deal of uncertainty concerning these estimates because the actual level of the pathogen in a serving of food consumed by an individual could have varied considerably from that observed in other portions of the food during subsequent investigations.

B. Incidence of *Listeria monocytogenes* in ready to eat meat

As *L. monocytogenes* is psychrotrophic in nature, it is very problematic to control the microbes in RTE products due to refrigerated presentation at the point of sale [39]. There are many varieties of RTE meats available in the market (ham, turkey and chicken), various types of presentations (vacuum, modified atmosphere, sliced at the counter) and abundant brands and thus the common target of government and industry is to control *L. monocytogenes*. The ubiquity of microbes in RTE meat products has been studied by various researchers. Diverse studies have represented that the ubiquity of *L. monocytogenes* in RTE meat products may vary from 0 to 72 %, for dissimilar levels of CFU per gram upon completion of shelf life. In analysis carried out in Spain, the investigators determined different types of brands available at the market and two types of representatives: opened deli meat and vacuum packaged meat [40]. Analysis of the total 601 samples of incises of deli-meats and pate with a low prevalence in vacuum showed prevalence of *Listeria*. Depending on the representation, a higher tendency was displayed in store-packaged products (8.5%), and then in the products packaged by manufacturers (2.7%), implying that the slicing equipment could be the source for the pathogen spread (cross contamination).

C. Isolation and detection of *Listeria monocytogenes* in RTE products

International Standards Organization (ISO 11290) has approved the methods to carry out *L. monocytogenes* isolation and detection in food. The exclusive methods depend on the food matrix. The enrichment broths can be used in these methods, like selective/differential agars. The common characteristic in all *Listeria* spp is hydrolysis of esculin. Selective and differential agar can differentiate between *Listeria* species and other bacteria but to differentiate between *L. monocytogenes* from other *Listeria* species is impossible with these media because both are phosphatidylinositol-specific phospholipase C positive. An alternative medium is the CHROM agar which can differentiate between *L. monocytogenes* and *L. ivanovii* [41]. *L. monocytogenes* and *L. ivanovii* both grow as blue colonies with white-halo around on ALOA plates CHROM agar whereas other *Listeria* species grow as a white colony. Bacteria other than these two species could produce colonies of variable morphologies.

V. CONTROL *Listeria monocytogenes* IN READY TO EAT PRODUCTS

A. Modify the processing plant environment

L. monocytogenes survives extremely well in the processing plant environment. *L. monocytogenes* may reach the processing plants through different variety of routes, like through the entry of raw materials, employees' shoes or clothes, and equipment (boxes, crates, carts etc.). *L. monocytogenes* pathogen can tolerate and continue to grow in conditions (e.g., refrigeration temperatures and high salt levels) that avoid the growth of many other food-borne pathogens. *L. monocytogenes* also has the bias to form biofilms and thus resident populations develop permanent niches in the plant. These local populations form biofilms to enhance their survival and are not easily removed by general purpose cleaners or sanitizers and normal sanitation procedures. Molecular fingerprinting techniques have

extremely contributed to study the ecology, sources, and spread of *L. monocytogenes* and *Listeria* spp. in processing plant environments. While a different variety of *L. monocytogenes* strains are found in mostly processing plants (including seafood plants), individual processing facilities usually harbour unique *L. monocytogenes* populations and strains, that persist for month or years in the plant or its products in spite of sanitation protocols designed to eliminate them. The arrangement of persistent processing plant contamination have been expressed for a variety of food processing environments, containing some smoked seafood, poultry, meat and dairy foods. A few subtypes colonize specific niches in the plant environment and persist over time. Thus screening for the presence and reintroduction of persistent *L. monocytogenes* contamination should be a component of every control planning. *L. monocytogenes* pathogenesis contamination in processing plants shows a major responsibility for the industry and public health. When isolates of *L. monocytogenes* were used for molecular sub-typing, it showed that the subtype(s) persisting in specific plants were accountable for the finished product contamination. The environmental post-processing contamination is thought to have been the source of a 1998/99 multi-state listeriosis outbreak that was related to the consuming of contaminated hot dogs and deli meats. Increased level of *Listeria* contamination in the environment (possibly associated with a construction event in the implicated plant) coincided with the time when product contamination with the outbreak strain first occurred. Reasonably, the environmental contamination was responsible for finished product contamination over a very long time period (>4 months), thus leading to a large outbreak. Elimination of persistent strains in the plant will reduce the risk of finished product contamination from environmental sources.

B. Raw materials

L. monocytogenes exist on raw ingredients also. Though many processing plants have adopted steps to destroy or reduce these organisms. This is the case for cooked in-shell crab products that are generally processed to produce a minimum 6 D reduction of *L. Monocytogenes*. It is fact that any *L. monocytogenes* found on finished product is the result of post process contamination [42]. There is a need to limit the introduction of *L. monocytogenes* into the plant environment, and to implement interruption that will effectively discard *L. monocytogenes* on product contact surfaces, glazing media and packaging materials.

C. Employees and processing personnel

The employees and processing personnel are also a potential source for the introduction of *L. monocytogenes* in the processing plant environment. Shoes, clothing, hands, etc of the employees can also be a source of transferring *L. monocytogenes* inside the plant from one area to another area. Further, it may also serve as direct sources of contamination if they are involved in post-processing treatment of products. It has been exposed that 1-10% of healthy adult's faecal matter may also be carriers of *L. monocytogenes*.

D. Verification of control

To verify *L. monocytogenes* control, plants should implant appliance on environmental monitoring program for an indicator such as *Listeria* spp. This program, specific to the

plant, should specify the areas to be sampled for *Listeria* spp., the frequency of sampling, and the action to be taken if *Listeria* spp. is detected [43].

CONCLUSION

An increase in the number of listeriosis cases, which is predominantly a food-borne disease, has been observed in several European Union countries since 2000. This increase in the cases, concerns person over 60 years of age and especially those who are immuno-compromised. The number of large cases of listeriosis outbreak has declined since the late 1990s and the large majority of listeriosis cases are sporadic. The low virulence of some strains of *L. monocytogenes* has been demonstrated in vitro and in vivo, albeit not over the complete oral route of infection on relevant animal models. At present, no routine methods permit the differentiation between virulent and avirulent strains of *L. monocytogenes*. The ability of the food to support growth of *L. monocytogenes* and information on the stage of sampling is generally not provided. It is not possible to assess the impact of contaminated samples on the risk for consumer health. Growth of *L. monocytogenes* is a function of the type of food, the storage time and the storage temperature. Storage temperature at retail and domestic refrigerators can vary significantly, especially for the domestic refrigerators. New tools to predict growth have been developed and can be used to determine if the product will or will not support growth of *L. monocytogenes* and estimate the extent of growth during the shelf life.

REFERENCES

- [1]. J. Rocourt, and C. Buchrieser, "The genus *Listeria* and *Listeria monocytogenes*: Phylogenetic position, taxonomy and identification. In: Ryser TE, Marth HE, eds. *Listeria*, Listeriosis and Food Safety", New York: CRC Press Taylor and Francis Group;120, 2007.
- [2]. J. A. Vázquez-Boland, et al, "*Listeria* pathogenesis and molecular virulence determinants", *Clinical Microbiology Reviews*; 14:584-640, 2001.
- [3]. S. M. Shantha, and S. Gopal, " Incidence of *Listeria* species in food and food processing environment: A Review", *Research and reviews: journal of microbiology and biotechnology*, Vol. 3, Issue 1, January - March, 2014.
- [4]. Q. Zhu, R. Gooneratne, and M. A. Hussain, "*Listeria monocytogenes* in fresh produce: Outbreaks, prevalence and contamination levels", *Department of Wine, Food and Molecular Biosciences, Lincoln University*, 9 March 2017.
- [5]. H. C. den Bakker, et al, "Comparative genomics of the bacterial genus *Listeria*: Genome evolution is characterized by limited gene acquisition and limited gene loss", *BMC Genomics*, 11: 688, 2010.
- [6]. K. K. Nightingale, et al, "Ecology and transmission of *Listeria monocytogenes* infecting ruminants and in the farm environment", *Appl Environ Microbiol.*, 70(8): 4458-4467, Aug 2004.
- [7]. M. P. Lessing, G. D. Curtis, and I. C. Bowler, " *Listeria ivanovii* infection", *The Journal of Infection*, 29:230-231, 1994.
- [8]. F.S. Southwick, and D. L. Purich, "Intracellular pathogenesis of listeriosis", *The New England Journal of Medicine*, 334:770-776, 1996.
- [9]. M. Lecuit, "Understanding how *Listeria monocytogenes* targets and crosses host barriers", *Clinical Microbiology and Infection*, 11:430-436, 2005.
- [10]. T. J. Adams, S. Vartivarian, and R. E. Cowart, "Iron acquisition systems of *Listeria monocytogenes*", *Infect Immun.*, 58:2715-2718, 1990.
- [11]. P. Cossart, J. Pizarro-Cerdá, and M. Lecuit, "Invasion of mammalian cells by *Listeria monocytogenes*: functional mimicry to subvert cellular functions", *Trends in Cell Biology*, 13:23-31, 2003.
- [12]. H. Réglier-Poupet, E. Pellegrini, A. Charbit, and P. Berche, "Identification of LpeA, a PsaA-like membrane protein that promotes cell entry by *Listeria monocytogenes*", *Infect Immun.*, 71(1): 474-482, Jan 2003.
- [13]. L. S. Burrack, J. W. Harper, and D. E. Higgins, "Perturbation of vacuolar maturation promotes listeriolysin O-independent vacuolar escape during *Listeria monocytogenes* infection of human cells", *Cell Microbiol.*, 11(9): 1382-1398, Sep 2009.
- [14]. M. D. Welch, and M. Way, "Arp2/3-mediated actin-based motility: a tail of pathogen abuse", *Cell Host Microbe.*, 11; 14(3): 242-255, Sep 2013.
- [15]. L. G. Tilney, D. J. DeRosier, and M. S. Tilney, "How *Listeria* exploits host cell actin to form its own cytoskeleton. I. Formation of a tail and how that tail might be involved in movement", *J Cell Biol.*, 118(1):71-81, Jul 1992..
- [16]. K. Ireton, "Molecular mechanisms of cell-cell spread of intracellular bacterial pathogens", *Open Biol.*, 3(7): 130079, Jul 2013.
- [17]. J. A. Melton-Witt, S. M. Rafelski, D. A. Portnoy, and A. I. Bakardjiev, "Oral infection with signature-tagged *Listeria monocytogenes* reveals organ-specific growth and dissemination routes in guinea pigs", *Infect Immun.*, 80(2): 720-732, Feb 2012.
- [18]. "Opinion of the scientific committee on veterinary measures relating to public health on the evaluation of microbiological criteria for food products of animal origin for human consumption", *European commission health & consumer protection directorate-general*, 23 september 1999.
- [19]. A. Vines, and B. Swaminathan, "Identification and characterization of nucleotide sequence differences in three virulence-associated genes of *Listeria monocytogenes* strains representing clinically important serotypes", *Curr Microbiol.*, 36(5):309-18, May 1998.
- [20]. L. Gorski, C. T. Parker, A. S. Liang, S. Walker, and K. F. Romanolo, "The Majority of Genotypes of the Virulence Gene *inlA* Are Intact among Natural Watershed Isolates of *Listeria monocytogenes* from the Central California Coast", *PLoS One*, 11(12): e0167566, 2016.
- [21]. P. A. Gill, J. G. Boulton, G. C. Fraser, A. E. Stevenson, and L. A. Reddacliff, "Bovine abortion caused by *Listeria ivanovii*", *Aust Vet J.*, 75(3):214, Mar 1997.
- [22]. F. Allerberger, and M. Wagner, "Listeriosis: a resurgent foodborne infection", *Clin Microbiol Infect.*, 16(1):16-23, Jan 2010.
- [23]. D. Montero, et al, "Molecular epidemiology and genetic diversity of *Listeria monocytogenes* isolates from a wide variety of ready-to-eat foods and their relationship to

- clinical strains from listeriosis outbreaks in Chile", *Front Microbiol.*, 6: 384, Apr 30, 2015.
- [24]. D. A. Drevets, and M. S. Bronze, "Listeria monocytogenes: epidemiology, human disease, and mechanisms of brain invasion", *FEMS Immunol Med Microbiol.*, 53(2):151-65, Jul 2008.
- [25]. R. W. Pinner, et al, "Role of foods in sporadic listeriosis, II. Microbiologic and epidemiologic investigation, The Listeria Study Group", *Journal of the American Medical Association.*; 267:2046-2050, 1992.
- [26]. M. J. McCue, and E. E. Moore, "Myocarditis with microabscess formation caused by *Listeria monocytogenes* associated with myocardial infarct", *Hum Pathol.*, 10(4):469-72, Jul 1979.
- [27]. V. Janakiraman, "Listeriosis in pregnancy: Diagnosis, treatment, and prevention", *Rev Obstet Gynecol.*, Fall; 1(4): 179-185, 2008.
- [28]. T. Mateus, J. Silva, R. L. Maia, and P. Teixeira, "Listeriosis during pregnancy: A public health concern", *ISRN Obstet Gynecol.*, 851712, 2013.
- [29]. M. Doganay, "Listeriosis: clinical presentation", *FEMS Immunol Med Microbiol.*, 35(3):173-5, Apr 1, 2003.
- [30]. M. Lecuit, "Human listeriosis and animal models", *Microbes Infect.*, 9(10):1216-25, 7 Aug, 2007.
- [31]. R. F. Lamont, et al, "Listeriosis in human pregnancy: a systematic review, *J Perinat Med.*, 39(3): 227-236, May 2011.
- [32]. A. C. Camargo, J. J. Woodward, D. R. Call, and L. A. Nero, "*Listeria monocytogenes* in food-processing facilities, food contamination, and human listeriosis: The Brazilian scenario", *Foodborne Pathog Dis.*, Aug 2, 2017.
- [33]. J. Pichler, et al., "An outbreak of febrile gastroenteritis associated with jellied pork contaminated with *Listeria monocytogenes*", *Wien Klin Wochenschr*, 121(3-4):149-56, 2009.
- [34]. V. Goulet, C. Hedberg, A. L. Monnier, and H. de Valk, "Increasing incidence of listeriosis in France and other European countries", *Emerg Infect Dis.*, 14(5): 734-740, May 2008.
- [35]. J. Denny, and J. McLauchlin, "Human *Listeria monocytogenes* infections in Europe - an opportunity for improved European surveillance", *Euro Surveill.*;13(13):pii=8082, 2008.
- [36]. C. Escolar, D. Gómez, M. Del Carmen Rota García, P. Conchello, and A. Herrera, "Antimicrobial resistance profiles of *Listeria monocytogenes* and *Listeria innocua* isolated from ready-to-eat products of animal origin in Spain", *Foodborne Pathog Dis.*, 14(6):357-363, Jun 2017. .
- [37]. B. D. Sauders, and D. J. D'Amico, " *Listeria monocytogenes* cross-contamination of cheese: risk throughout the food supply chain", *Epidemiol Infect.*, 144(13):2693-7, Oct 2016.
- [38]. C. A. Hwang, and S. Sheen, "Modeling the growth characteristics of *Listeria monocytogenes* and native microflora in smoked salmon", *J Food Sci.*, 74(3):M125-30, Apr 2009.
- [39]. A. Lianou, and J. N. Sofos, "A review of the incidence and transmission of *Listeria monocytogenes* in ready-to-eat products in retail and food service environments", *J Food Prot.*, 70(9):2172-98, Sep 2007.
- [40]. V. Garrido, A. I. Vitas, and Garcia-Jalon, " Survey of *Listeria monocytogenes* in ready-to-eat products: Prevalence by brands and retail establishments for exposure assessment of listeriosis in Northern Spain", *Food Control*, Volume 20, Issue 11, Pages 986-991, November 2009.
- [41]. S. Loncarevic, M. Økland, E. Sehic, H. S. Norli, and T. Johansson, "Validation of NMKL method No. 136--*Listeria monocytogenes*, detection and enumeration in foods and feed", *Int J Food Microbiol.*,124(2):154-63, May 3, 2008 .
- [42]. R. M. Dillon, and T. R. Patel, " *Listeria* in seafoods: A Review", *Journal of Food Protection.*, Vol. 55, No. 12, pp. 1009-1015, December 1992.
- [43]. R. Ismaïl, "Methods for recovering microorganisms from solid surfaces used in the food industry: A Review of the Literature", *Int J Environ Res Public Health.*, 10(11): 6169-6183, Nov 2013.