

---

## MORPHOLOGICAL CHANGES OF LISTERIA MONOCYTOGENES IN A FOOD PROCESSING ENVIRONMENT

---

**Sumana Samanta**

Research Scholar

Department of Microbiology, CMJ University, Jorabat, Meghalaya, India.

**Dr. Mousumi Roy**  
(Assistant Professor)

Research Guide

Department of Microbiology, CMJ University, Jorabat, Meghalaya, India.

**DECLARATION:** I AS AN AUTHOR OF THIS PAPER /ARTICLE, HERE BY DECLARE THAT THE PAPER SUBMITTED BY ME FOR PUBLICATION IN THE JOURNAL IS COMPLETELY MY OWN GENUINE PAPER. IF ANY ISSUE REGARDING COPYRIGHT/PATENT/ OTHER REAL AUTHOR ARISES, THE PUBLISHER WILL NOT BE LEGALLY RESPONSIBLE. IF ANY OF SUCH MATTERS OCCUR PUBLISHER MAY REMOVE MY CONTENT FROM THE JOURNAL WEBSITE. FOR THE REASON OF CONTENT AMENDMENT/OR ANY TECHNICAL ISSUE WITH NO VISIBILITY ON WEBSITE/UPDATES, I HAVE RESUBMITTED THIS PAPER FOR THE PUBLICATION. FOR ANY PUBLICATION MATTERS OR ANY INFORMATION INTENTIONALLY HIDDEN BY ME OR OTHERWISE, I SHALL BE LEGALLY RESPONSIBLE. (COMPLETE DECLARATION OF THE AUTHOR AT THE LAST PAGE OF THIS PAPER/ARTICLE)

### Abstract

The primary aim of this study was to examine the growth of *Listeria monocytogenes* L56 (was to investigate the viability and proliferation of IMR isolates). Culture at 37°C Scanning electron microscopy (SEM) and motility media were able to identify morphological changes and motility induced by temperature stressors, respectively. Morphological changes and motility of *Listeria monocytogenes* in broth cultured in 300 ml TSB for 12 h at 37° C. for log phase and 19 h for stationary phase were measured at 2 h intervals. Longitudinal measurements of 180 cells were taken at 9,500x magnification using SEMafore for each temperature treatment. This study revealed that the morphology of *Listeria monocytogenes* cells changed significantly between exponential and stationary phases under stress conditions. At each of the three temperatures he studied, the stationary-phase cells were shorter and more spherical than the extended log-phase cells, while the extended-log phase cells were longer at 55°C, but at 28°C or 4°C. It did not stretch at °C. Loss of motility observed in stressed stationary-phase and unstressed log-phase cells indicates that mobility is critical for organismal survival under temperature stress.

**Keywords:** *Listeria monocytogenes*, temperature stress, morphology, motility, survival

---

## 1. Introduction

*Listeria monocytogenes* is a bacterium that may be found in tainted food. It causes listeriosis, a gastrointestinal illness that can be mild or severe. Among foodborne infections, it has the greatest hospitalization rate of around 99% and a death rate of 19%.

Life-threatening human infections like meningitis, encephalitis, spontaneous abortion, and miscarriage can only be caused by *Listeria monocytogenes* if the bacteria can traverse the intestinal, blood-brain, and fetal-placental barriers. The elderly, young children, pregnant women, and those with compromised immune systems are at greatest risk, though anyone can contract the disease. The number of cases reported in Europe has dramatically risen between 2008 and 2014. The EU notification rate increased by 8.6% from 2012 to 2013 and was 0.44 instances per 100,000 people.

*Listeria monocytogenes* is so normal in climate that contamination of areas, including meal plans, cannot be prevented unless strong areas are implemented. Due to their ability to withstand various stressors of B. food processing plants and their ability to form biofilms that promote persistence Cross-contamination of *L. Monocytogenes* with food is one of the main potential pathways of food contamination when the area where food is produced is contaminated.

There is a great deal of worry in the food business about the presence of *Listeria monocytogenes* in food processing settings and the possibility of cross-contamination with food due to the devastating effects of listeriosis on the general population. This is a major issue for the fast food sector since no heat treatments or other antibacterial procedures are included in the process between manufacture and consumption.

Because listeriosis is food poisoning, it is less common. Food cross-contamination can be reduced, environmental pollution of food processing environment can be avoided, and environmental pollution of food processing environment can be completely avoided . Additionally, a 37% decrease in listeriosis may result from inhibiting *L. monocytogenes* proliferation at retail and beyond.

The purpose of this review is to talk about how the obtained isolates were characterised so that we can better understand this type of contamination and look into ways to lessen it. This research looks at the cases of *Listeria monocytogenes* found in processed foods during the last three years.

### **1.1. Present rules on the Occurrence of *Listeria monocytogenes* under eating material**

Regulation (EC) No. 2073/2005 is the current regulation in the European Union (EU) governing *Listeria monocytogenes*, and it outlines the standards that food items sold in the EU must fulfil. The presence is authorised at varying levels based on the food's potential to sustain bacterial growth; however, absence is necessary for foods meant for babies and other medical applications (10-25 g samples). Below 100 CFU/g is considered an acceptable threshold for *Listeria monocytogenes* in ready-to-eat (RTE) foods to prevent the pathogen from growing during storage (5-25 g sample). To ensure the RTE diet is safe for growth, *Listeria monocytogenes* levels must be 100 CFU/g or less in all 5 g samples taken before the product leaves the facility and throughout its shelf life. At the point when the capacity of an eating regimen to help development has not been shown, it is expected that development will happen and the nonattendance models are utilized. Similar laws exist in New Zealand, Australia, and Canada.

A more stringent rule is in force in the USA. *L. monocytogenes* must never be present in 5–25 g of food or in the area where it is being processed. Small manufacturers and businesses in developing nations that are compelled to comply with US rules and export to the USA face a significant difficulty as a result of these stringent restrictions. Businesses engaged in exporting often follow the laws of the nation to which they are exporting.

### **1.2. Acquired *L. monocytogenes* Isolate Characterization**

#### **➤ Serotyping/Serogrouping**

Somatic (O) and flagellar (H) antigen interactions formed the basis of the agglutination approach used to serotype *L. monocytogenes* subspecies, which was devised by Seeliger and Hohne in 1979. The primary serogroups were divided into five phylogenetic groups using a multiplex PCR-based approach. The five genes used in this test are *prfA*. Nowadays, serotyping and serogrouping are

combined. To distinguish *L. monocytogenes* from other *Listeria* species and to allow serotyping of the organism, Vitullo et al. Developed real-time PCR method in 2013.

### ➤ Other Subtyping Methods

Subtype *L. monocytogenes* strains are useful for tracking contamination pathways throughout the processing environment and gaining an understanding of the source of such contamination beyond the species level. Typeability, power, reproducibility, speed, cost, and lab capacity are the most crucial aspects to consider while deciding on a technique. When it comes to band-based techniques or DNA fingerprinting, pulsed field gel electrophoresis (PFGE) is now the gold standard for identifying *Listeria* strain correlations for monitoring contamination routes in food processing facilities. PFGE produces DNA patterns that differ from strain to strain based on the quantity and size of the DNA fragments collected. Using bioinformatics tools, pulses may be found that represent closely linked strains. While whole genome sequencing is currently being used, the Pulse Net International network still employs standardized PFGE techniques to investigate *L. monocytogenes* (and other harmful bacteria) (WGS).. The reference allele sequences, sequence categories, and epidemiological data for numerous species can be found in an MLST database. There is analysis and interrogation software that enables searching through the databases' allele sequences and sequence kinds.

### **1.3.L. monocytogenes Processing Environment Persistence**

The conventional definition of *L. monocytogenes* persistence in processing environments is undetectable pulsatility regularly isolated from the same facility for 6 months or more. This indicates that the strain can survive this pulsatility in the processing environment despite the best sanitation measures in the factory. It is possible that a strain's repeat in a handling climate is welcomed on by repetitive defilement of the handling office by an outer source, however even in that situation, the strain would in any case should have the option to get by and endure outside. What drives such tenacity is a mystery. Persistency has not been identified as a genetic trait. Potential genes for genetic markers include biofilm formation genes, *qacH*, and *SSI*. These genes have been shown to be present in non-persistent transient strains, but not in persistent strains. A

second explanation is that the shelters and niches in which stubborn strains can live protect them from the depuration process.

#### **1.4.Environmental food processing's**

On the frequency of *L. monocytogenes* in surroundings involved in food preparation, several surveys have been published. It's not always practical to compare survey findings from one study to another, however. The following elements may have an impact on the observed prevalence:

1. Several test techniques are used. B. Varying swab size and test area (varies from 10 cm<sup>2</sup> to 1 m<sup>2</sup>).
2. Several strategies, such as the ISO, BAM, or other methods, are employed to analyze *L. monocytogenes*.
3. Several frequencies of recurrent facility testing, such as Sala et al survey's conducted on a single occasion, another facility's survey conducted on a number of times, and many facilities' survey conducted on a number of occasions.
4. A facility that has shown success is the objective.
5. The findings might be impacted by varying sample sizes and sampling sites.

By examining isolates from multiple locations, PFGE analysis may, to some extent, assist in determining the source of contamination in the processing environment. For example, PFGE profiles between isolates from raw materials and isolates from the food processing environment indicate that raw materials were the source of contamination. During the R uckerl et al. survey, four of the seven genotypes that were initially isolated from the raw materials were found. 's in the food handling climate (FPE) were demonstrated to be available there. Similar PFGE types may also be present in food processing facilities and the surrounding environment, suggesting a potential source of contamination.

## **2. Literature review**

*Listeria monocytogenes* is a Gram-positive, rod-shaped, non-spore-forming bacteria that may be found in water, soil, sewage, decaying plants, silage, and animals (O'Neil and Marquis, 2006;

Gandhi and Chikindas, 2007). Research conducted by Renier et al. These bacteria (2011) are able to thrive under a wide range of extreme circumstances, including high salinity (up to 10% NaCl), low water activity (0.9), low acidity (pH 0-4.5), and high heat (40 °C). increase. Human listeriosis is caused by *Listeria monocytogenes* and is particularly harmful for teenagers, the elderly, pregnant women, and those with impaired immune systems. Meat, fish (raw and smoked), spoiled cheddar, unpasteurized milk, ready-to-eat (RTE) dinners, and new organic products are all high-risk sources of microorganisms for human consumption (Gandhi and Chikindas, 2007).

Mortality is high, with annual rates of listeriosis as low as 0.1–10 per million, which can exceed 11-12 % (WHO), 2019a). Numerous cases of it have reported worldwide, with the highest mortality rate occurring in the U S in 1985. Creamy cheese; Beckers et al., 1987), Canada (2008) Charcuterie; South Africa (to date; Birk-Urovitz 2011, no reports) (Deli Meat, 2017-2018; Smith and other, 2019) the outbreak in South Africa demonstrates that *Listeria monocytogenes* remains a significant food safety threat despite the development of biological controls and treatments. Morphology, virulence, gene expression, and antimicrobial resistance (AMR) of *Listeria monocytogenes* are all affected by environmental stress (Matereke and Okoh, 2020). In (FPE), a better understanding of pathogen adaptation mechanisms can lead to the development of more effective and cost-effective disinfection methods that ensure food safety.

The goal of the review is to gather data on how *L. monocytogenes* responds to stress in FPE. The risk of FPE and the bacterium in food could be reduced, customer safety could be maintained, and effective food preservation and sanitation practices could be developed with a better understanding of how *L. monocytogenes* responds to stress.

*Listeria monocytogenes*, a bacterium that rapidly contaminates a wide range of food items (Miladi et al.), is frequently the cause of food recalls. 2017). *L. monocytogenes* is subjected to a wide range of harmful stressors in FPE, including low or high temperature, low pH, high salinity, and chemical concentrations that may be sublethal or lethal.

O'Byrne and Karatzas (2008) suggest that virulence and environmental stress tolerance are two features of *Listeria monocytogenes* biology that overlap. According to NicAgain and O'Byrne



(2016), in the absence of a sufficient stress reaction, the virus does not transit through the human gastrointestinal system and stays alive and in her FPE. One of the most significant stress response systems is connected with the alternative sigma factors B, C, H, and L, with B playing a crucial role (Lungu et al., 2009). (Lungu et al., 2009) in *L. monocytogenes*, these elements regulate nearly 300 genes, including stress-related and virulence-related genes. *Listeria monocytogenes* rely on vitamin B to persevere in the face of oxidative, osmotic, and acidic stresses (Figure 2). A complex structure known as the stressosome governs the activity of B and modulates the performance of the overall stress response of *Listeria monocytogenes* (NicAogain and O'Byrne, 2016). Studies of the *Listeria monocytogenes* transcriptome and proteome have revealed a number of proteins that are activated in response to stress. This table provides an overview of the major *Listeria monocytogenes* virulence and stress response genes and proteins.

The transcription of genes related to the stress response is controlled by B. The expression of virulence genes (transcriptional activator of virulence genes) is then controlled by PrfA. A complicated cross-talk mechanism involving proteins B and PrfA regulates gene expression both inside and outside the host, including the suppression of inefficient genes (Gaballa et al., 2019). The adaptive responses of *Listeria monocytogenes* and the stress conditions encountered during food production influence the virulence of the bacteria and pose a greater threat to consumers. Understanding how *Listeria monocytogenes* responds to changing environmental conditions (primarily lethal effects) is necessary for effective control and disinfection of industrial processes. We believe we can stop the spread of resistance and prevent *L. monocytogenes* from becoming more dangerous as a result of this. The challenges faced by *L. monocytogenes* during FPE and the strategies it developed to overcome them are the primary focus of this review.

The general stress response proteins ClpC (ATPase), ClpP (protease), and RelA (generating guanosine pentose phosphate [(p)ppGpp]) as well as the specific stress response proteins, such as the serine protease HtrA (degradation of misfolded proteins), all play critical roles. Role in osmotic resistance without the accumulation of suitable solutes, research indicates that RelA participates in the response to osmotic stress (Burall et al., 2012).

The *Listeria monocytogenes* B regulon contains eight genes, including *hfq*, *dtpT*, LMRG 01658, 00208, 00211-00212, and other proteins potentially associated with osmotic stress. However, its function has not yet been clarified (Liu et al., 2019). Ribeiro et al. (2014), initiation of these properties during salt pressure promotes enhanced formation of exopolysaccharides. The Hfq protein binds to sRNA when the cell swells inside. According to Christiansen et al. (2006), mutants lacking this protein failed to respond immediately to ethanol and osmotic stress. Protection by osmotic stress is highly dependent on the involvement of her DtpT proteins in the transport of di- and tripeptides (Wouters et al., 2005). Kdp, on the other hand, is a transcriptional response regulator that enhances K<sup>+</sup> import to accelerate stress responses. Osmotic resistance is also associated with trehalose, and the *treA* gene from *Listeria monocytogenes*, which encodes phosphotrehalase, was discovered (Ells and Truelstrup Hansen, 2011). According to Burgess et al. (2016), membrane-modifying proteins that respond to osmotic stress include putative peptidoglycan-associated protein (*lmo2085* gene) and putative UDP-glucose phosphorylase (*lmo1078* gene).

According to Arioli et al. (2019), *Listeria monocytogenes* can grow and tolerate temperatures between 0 and 45 °C. According to Smelt and Brul (2014), *L. monocytogenes* is inactivated for 10–12 seconds at 55–65 °C, depending on the cell's physiological state.

### **3. Experimental Method**

#### **3.1. Microbial Origin**

The Institute of Medical Sciences Malaysia (IMR) provided the L56 strain of *Listeria monocytogenes* used in this study. A clinical isolate of contaminated human blood from the Royal Pathological Society of Australia (RCPA) Quality Assurance Program M8/92 Daving Change served as the source of the initial cultures. (4b serotype)

#### **3.2. Certainty of *L. monocytogenes* behavior**



The legitimacy of the isolate was validated by studying its appearance and biochemistry. Before being sub-cultured on PALCAM agar (Merck, Germany), isolates were spiked with 0.6% trypticase soy agar (Merck, Germany) and incubated at 37°C for 24-48 hours (TSAYE). Extract of yeast is visible in the form of streaks. 24-48 hours of incubation at 37 °C on trypticase soy agar (Merck, Germany). Isolates of *Listeria monocytogenes* passed a battery of tests for viability, including Gram staining, catalase activity, methyl red staining, hemolysis, nitrate reduction, rhamnose staining, and xylose staining, at 21 degrees Celsius.

### **3.3.The Ability of *L. monocytogenes* to Withstand High Temperatures**

Single bacterial colonies grown in Trypticase Soy Agar (Merck, Germany) were survival experiments. Prepared the inoculum Three hundred millilitres of TSB containing 10<sup>3</sup> CFU/ml were spiked with 1 millilitre of this broth. Bacteria were grown at 37 degrees Celsius until they entered the log and stationary phases, each lasting 12 and 19 hours, and then subjected to three different sublethal temperature stressors (55 degrees Celsius, 28 degrees Celsius, and 4 degrees Celsius). Prior to the sudden drop in temperature, the inoculum size was 10<sup>8</sup> CFU/ml during the log phase and 10<sup>9</sup> CFU/ml during the stationary phase.

10 ml of the broth containing the stressed *L. monocytogenes* cells was removed from 300 ml TSB and incubated in a sterile environment while stressing *L. monocytogenes* at 37°C-55°C, 28°C, and 4°C, respectively. Placed in a centrifuge tube Tests were performed periodically throughout the exponential and stationary phases of *L. monocytogenes* development. After being centrifuged at 6,000 rpm for 15 minutes at 4°C, the broth containing the stressed *Listeria monocytogenes* cells was collected. A 4% glutaraldehyde solution was then added after the supernatant was drained, and the samples were stored in the fridge until SEM analysis could be performed.

### **3.4. Preparing Samples for SEM's**

The broth containing the stressed *Listeria monocytogenes* cells was collected after centrifugation at 6,000 rpm for 15 minutes at 4°C. Once the supernatant was removed, a 4% glutaraldehyde solution was added, and the samples were placed in the refrigerator until SEM analysis could be performed.

### **3.5. Cell Length Measurement Using SEM**

The number of *L. monocytogenes* cells that were experiencing stress varied from 49 to 100 in each and every SEM image. A total of 451 measurements were taken using triplicate SEM images for each sampling period. This was done in order to ensure accurate results. At 55 degrees Celsius, 48 degrees Celsius, and 7 degrees Celsius, stationary and log-phase cells were cultured for six hours, and samples were collected every two hours. For the course of this experiment, a scanning electron microscope magnification of 22,964x was used.

### **3.6. Impact of Temperature Stress on *L. monocytogenes* Motility**

Before being incubated for three days at 14°C, temperature-stressed *L. monocytogenes* were stably injected into the motility medium (Merck, Germany) at intervals of one hour. According to the growing pattern in the motility that resembles an umbrella medium, the results were classified as either positive or negative.

### **3.7. Statistical Evaluation**

SAS (SAS Institute, Cary, North Carolina, USA) got used for statistically analyze the time that cells in the log and stationary phases were exposed to various stressful temperatures. Across the samples, significant differences (P0.05) were found using one-way ANOVA. The means of the variables in the samples were compared post hoc using Duncan's test.

## **4. Results And Discussions**

#### 4.1. Average cell length of *Listeria monocytogenes* and its response to temperatures below lethal

*L. monocytogenes* exhibits a variety of morphogenesis in response to sublethal stress of temperatures, as shown under a scanning electron microscope. Cell elongation, coccoid, and entire cells are among the phenotypic changes that have been seen. Cell width was ignored since the alterations in this study's results were so minimal.

Table 1: Log and stationary phase's average cell length (m) *Listeria monocytogenes* subjected to stimuli at a sub lethal temperature

Bacterial phase	Temperature and hour of stress	Mean ( $\mu\text{m}$ ) $\pm$ Standard deviation
Log	55°C (2 h)	0.94 $\pm$ 0.46 <sup>a,a</sup>
Stationary	55°C (2 h)	0.87 $\pm$ 0.27 <sup>a</sup>
Log	55°C (4 h)	2.07 $\pm$ 0.97 <sup>a</sup>
Stationary	55°C (4 h)	0.74 $\pm$ 0.27 <sup>b</sup>
Log	55°C (6 h)	1.33 $\pm$ 0.77 <sup>a</sup>
Stationary	55°C (6 h)	0.84 $\pm$ 0.30 <sup>b</sup>
Log	28°C (2 h)	0.83 $\pm$ 0.24 <sup>a</sup>
Stationary	28°C (2 h)	0.89 $\pm$ 0.25 <sup>b</sup>
Log	28°C (4 h)	0.88 $\pm$ 0.24 <sup>a</sup>
Stationary	28°C (4 h)	1.01 $\pm$ 0.31 <sup>b</sup>
Log	4°C (2 h)	1.02 $\pm$ 0.35 <sup>a</sup>
Stationary	4°C (2 h)	0.72 $\pm$ 0.20 <sup>b</sup>
Log	4°C (6 h)	0.89 $\pm$ 0.29 <sup>a</sup>
Stationary	4°C (6 h)	1.01 $\pm$ 0.30 <sup>b</sup>

The log phase cell length variance (in mm) following exposure to various temperature stressors is shown. At the point when log stage cells were warmed to 55°C, fibers formed. The mean cell length increased from 6.36 mm to 1.47 mm after two and four hours, but decreased to 6.22 mm after four hours. In contrast, at 47°C, log phase cells had significantly shorter lengths than cells at 2°C. However, over the printed timeframe, cells at 7 °C showed a predictable decrease in cell length. Cell length did not change in his first 6 hours of exposure at 35°C.



Fig. 1: At 55°C, SEM of log phase cells of *L. monocytogenes* showed elongation

In contrast to other stress circumstances, bacteria subjected to moderate heating temperatures (55°C) grew more quickly and developed filaments. It was determined that the size of bacterial cells It seems that growth rate is an exponential function at a certain time temperature and that the longer they develop, the quicker they do so. Another theory is that bacteria might adapt to "thermal expansion" if they were mildly heated. Serway asserts that its volume grows as growing temperature rises. A bacterial cell's total thermal expansion is a result of variations on the whole distance across the spaces between its molten atoms and molecules. Yet, *L. monocytogenes*' stationary phase revealed a distinct feature (Table 1). The majority sizes ranging from 4.36 to 8.65 m. Cells shrunk after being exposed to 55°C. The cells length grew to 1.36 m; however the mean cell length fell from 1.36 m (2 hours) to 2.45 m (4 hours) (6 h). Whereas stationary phase cells at 4°C ranged in length from 0.72 mm to 1.05 mm, those at 28°C ranged in length from 0.89 mm to

1.01 mm., stationary phase cells at 55°C for 4 hours and 4°C for 2 hours showed lower cell length than other stress conditions.

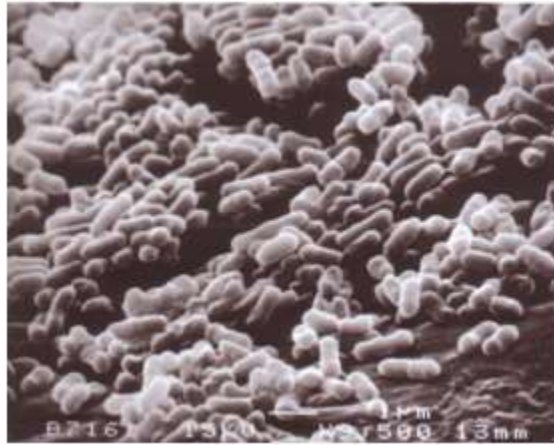


Fig. 2:L. Monocytogenes cells in log phase, SEM, at 28 °C

At 28°C, there were no discernible variations in average size (below 1.01 m), as seen in Fig. 2. Although being shorter, stationary phase and log cells had statistically significant differences at P0.05. The fact that cell development was more robust at 28°C shows that temperature stress was not a factor at this temperature, which is close to the ideal range for growth (30–37°C). According to a research, juvenile cultures of *L. monocytogenes* are made up of short rod organisms with ends it could be somewhat pointed, short chains, and diplo-forms that are often observed as V or Y-shaped. These organisms range in size from 0.5 by 1 to 2 m.

According to the statistical evaluation, the current research showed that the environment of the bacterial development phase affected alterations in *L. monocytogenes*' morphology. It is hypothesized that *L. monocytogenes*' altered and changed shape is an aspect of the microbial cells' adaptability for their continuous survival and prosperous expansion.

#### **4.2.B. *L. monocytogenes*' motility is affected by temperatures that are less than lethal.**

Whether cells from temperature shocks might affect listeria motility when exposed to sub lethal stress of temperatures, it was determined via studies utilizing motility agar. *L. monocytogenes*'

molecular characteristics were influenced by the pathogen's development phase (Table 2). At the three stressful temperatures examined, a decrease in motility was observed in stationary phase cells in comparison to log phase cells.

Table 2: Findings of the *L. monocytogenes*' motion in the log and stationary phases after exposure to sub lethal temperatures

<b>Bacterial phase</b>	<b>Temperature and hour of stress</b>	<b>Motility</b>
Log	55°C (2 h)	Motile
Stationary	55°C (2 h)	Non-Motile
Log	55°C (6 h)	Motile
Stationary	55°C (6 h)	Non-Motile
Log	28°C (2 h)	Motile
Stationary	28°C (2 h)	Non-Motile
Log	28°C (4 h)	Motile
Stationary	28°C (4 h)	Non-Motile
Log	4°C (2 h)	Motile
Stationary	4°C (2 h)	Non-Motile
Log	4°C (6 h)	Motile
Stationary	4°C (6 h)	Non-Motile

Nothing is known about how environmental stress affects the motility characteristics of listeria. The present study's findings demonstrated that listeria's distinctive motility was lost in stationary phase cells, although listeria motility in log phase was unaltered. Although little is known about how temperature affects the mobility recent research indicated that 30 54.3% of 66 tested strains were capable of moving at 22°C and that 4 (9.1%) of them were also capable at 12°C. They discovered that only at 22°C was swimming motility visible, proving that *Listeria* species produce flagellums in response to temperature.



The findings of this research strongly showed that stress may have an impact on several phenotypic traits of organisms as a survival adaptation. Given that the majority of food systems are in stressful environments, the findings of this research are highly helpful in reducing *L. monocytogenes* misidentification in food systems. To improve eating material security, it's crucial to understand how *L. monocytogenes* forms filaments and coccoid under stressful environments.

## 5. Conclusions

The sub lethal temperature stress test revealed that *L. monocytogenes* cells may proliferate and survive while undergoing morphological and motility modifications to adapt to the new environment.

## REFERENCES

1. Archer, D.L. (1996). *Trends Food Sci. Tech.* 7(3), 91-97.
2. Cartwright, E.J., Jackson, K.A., Johnson, S.D., Graves, L.M., Silk, B.J., Mahon, B.E. (2013). *Emerg. Infect. Dis. CME.* 19(12), 1-9.
3. Mook, P., O'Brien, S.J., Gillespie, I.A. (2011). *Emerg. Infect. Dis.* 17, 38- 43.
4. Goulet, V., Hebert, M., Hedberg, C., Laurent, E., Vaillant, V., De Valk H. (2012). *Clin. Infect. Dis.* 54, 652-660.
5. Busta, F.F., Foegeding, P.M., Adams, D.M. (1981). in H.S. Levinson, A.L. Sonenshien, D.J. Tipper, eds., *Sporulation and Germination.* Amer. Soc. Microbiol., Washington, USA.
6. ICMSF. (1996). *Microbiological specifications of food pathogens.* Blackie Academic and Professional, London.
7. Morange, M., Hevin, B., Fauve, R.M. (1993). *Res. Immunol.* , 144, 667- 677.
8. Gulam, R.R.A., Aziah, I., Fatimah, A.B. (1991). *Pertanika*, 14 (3), 249-255.
9. Brzin, B. (1973). *Zbl. Bakteriolog. Hyg. I. Abt. Orig.* , A. 225, 80-84.
10. Brzin, B. (1975). *Zbl. Bakteriolog. Hug. I. Abt. Orig. A.* , 232, 287-293.
11. Isom, L., Khambatta, Z. S., Moluf, J.L., Akers, D.F., Martin, S. E. (1995). *J. Food Prot.* , 58, 1031-1033.
12. Vail, K.M., McMullen, L.M., Jones, T.H. (2012). *J. Food Prot.* 75, No. 12, 2142-2150.

13. Zarei, M., Borujeni, M.P., Khezzadeh, M., Kazemipour, S., Hesami, G., Bemani, E. (2012). *J. Biol. Environ. Sc.*, 6, No. 16, 99-104.
14. Lani, M.N. (2002). Master Thesis, UPM.
15. Neidhardt, F.C. (1963). *Ann. Rev. Microbiol.* 17, 61-86

## Author's Declaration

I as an author of the above research paper/article, hereby, declare that the content of this paper is prepared by me and if any person having copyright issue or patentor anything otherwise related to the content, I shall always be legally responsible for any issue. For the reason of invisibility of my research paper on the website/amendments/updates, I have resubmitted my paper for publication on the same date. If any data or information given by me is not correct, I shall always be legally responsible. With my whole responsibility legally and formally I have intimated the publisher (Publisher) that my paper has been checked by my guide (if any) or expert to make it sure that paper is technically right and there is no unaccepted plagiarism and hentricontane is genuinely mine. If any issue arises related to Plagiarism /Guide Name /Educational Qualification /Designation /Address of my university/college/institution/Structure or Formatting/ Resubmission / Submission /Copyright / Patent/Submission for any higher degree or Job/Primary Data/Secondary Data Issues. I will be solely/entirely responsible for any legal issues. I have been informed that the most of the data from the website is invisible or shuffled or vanished from the data base due to some technical fault or hacking and therefore the process of resubmission is there for the scholars/students who finds trouble in getting their paper on the website. At the time of resubmission of my paper I take all the legal and formal responsibilities, If I hide or do not submit the copy of my original documents (Aadhar/Driving License/Any Identity Proof and Photo) in spite of demand from the publisher then my paper may be rejected or removed from the website anytime and may not be consider for verification. I accept the fact that as the content of this paper and the resubmission legal responsibilities and reasons are only mine then the Publisher (Airo International Journal/Airo National Research Journal) is never responsible. I also declare that if publisher finds any complication or error or anything hidden or implemented otherwise, my paper maybe removed from the website or the watermark of remark/actuality maybe mentioned on my paper. Even if anything is found illegal publisher may also take legal action against me

**Sumana Samanta**  
**Dr. Mousumi Roy**

\*\*\*\*\*