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Persistence of *Listeria monocytogenes* in food processing environments: challenges and future directions

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Abstract

Listeriosis, caused by *Listeria monocytogenes*, remains one of the most serious foodborne illness in high-income countries, despite its rarity. This paper addresses the challenge of persistent *L. monocytogenes* in food processing environments, which leads to significant public health and economic impacts. We examine the persistence mechanisms of *L. monocytogenes*, focusing on natural selection due to disinfectant use, genetic drift from periodic population bottlenecks, and recolonization from non-food contact areas. Our analysis highlights the interplay of these dynamics in shaping *L. monocytogenes* populations, emphasizing that genetic drift plays a crucial role in persistence. Understanding these dynamics is essential for developing targeted strategies to mitigate the risks posed by persistent *L. monocytogenes* strains, thereby enhancing food safety and public health.

Keywords: *Listeria monocytogenes, population genomics, persistence, food processing environments, genetic drift*

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1. Introduction

Listeriosis is a rare but severe infectious disease caused by the bacterium *Listeria monocytogenes*. Despite its low incidence, it poses a significant public health challenge due to its high hospitalization and mortality rates, making it one of the most serious foodborne illnesses worldwide—particularly in high-income regions such as the European Union (EU) [1].

In 2023, 27 EU Member States reported 2952 confirmed invasive human cases, resulting in 1497 hospitalizations and 335 deaths. This corresponds to a notification rate of 0.66 cases per 100,000 population—a 5.8% increase compared to 2022 and the highest rate observed since EU surveillance began in 2007. This rise continues a five-year upward trend (2019–2023), with aging populations and the prevalence of chronic diseases identified as contributing factors [1, 2].

Listeriosis remains a global concern. In the United States, data from foodborne outbreaks between 1998 and 2023 recorded 1517 illnesses, 1191 hospitalizations, and 217 deaths, corresponding to an overall case fatality rate of approximately 14% and a hospitalization rate of around 79% [3].

These consistently high rates of hospitalization and mortality, particularly among high-risk groups (pregnant women, newborns, the elderly, and individuals with weakened immune systems or chronic diseases), highlight the urgent need for innovative and effective prevention strategies.

While *L. monocytogenes* is widespread in the environment, infection commonly occurs through consuming contaminated food. High-risk foods include ready-to-eat products such as deli meats, unpasteurized dairy products, and fresh produce contaminated during processing. The pathogen's ability to survive refrigeration and resist common preservation methods further exacerbates the risk [1, 2, 4].

Several studies have reported the existence of *L. monocytogenes*-specific molecular subtypes that reoccur, year after year, in food processing environments (FPEs), despite routine washing and disinfection [5–8]. These persistent strains are also frequently identified in final products such as cheeses [9], sausages [5], and smoked fish [10]. Interestingly, the same strain isolated from people infected with *L. monocytogenes* has been detected in cheese and within the cheese FPE [11–14].

The difficulty in their eradication underscores the urgency for effective identification and prevention methods in food processing. Different hypotheses have been tested in recent years. Some factors include inadequate zoning and hygiene barriers in FPEs, poorly designed equipment, and insufficient cleaning and disinfection, as

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some niches prove challenging for disinfectants to access or result in disinfectant concentrations being reduced to sub-inhibitory levels [5, 6, 15]. Biological explanations range from genetic factors, where *L. monocytogenes* may acquire mechanisms that reduce intracellular disinfectant levels [16, 17], to biofilm formation or resistance to disinfectants, which result in the establishment of protective microenvironments. However, the findings indicate that no single genetic marker was universally responsible for a strain's ability to persist. Both persistent and presumed non-persistent groups exhibited a variety of stress resistance markers, encompassing those for heavy metal resistance [18], oxidative stress, and pH stress [19]. While physical and process-related factors are relevant, the biological characteristics of *L. monocytogenes* play an equally important role in its persistence. A defining characteristic of this pathogen is its cold tolerance, which enables it to survive and even grow at the refrigeration temperatures commonly used in FPEs. Unlike many other foodborne pathogens, *L. monocytogenes* can grow at temperatures as low as −0.4 °C, allowing it to persist in environments where competing microorganisms are inhibited. Several adaptive mechanisms contribute to this cold tolerance. At low temperatures, *L. monocytogenes* changes its membrane composition by increasing the proportion of unsaturated fatty acids to maintain fluidity. Proteins such as CspA and CspB stabilize RNA structures and facilitate protein folding under cold stress. Cryoprotectants such as glycine betaine and trehalose accumulate in cells to protect cell structures from damage caused by freezing or refrigeration [20].

Cold tolerance is not uniform among *L. monocytogenes* strains. Strain-specific variations in cold growth capacity have been widely documented and are increasingly being studied using population genomics approaches. Genomic studies have revealed differential expression of cold adaptation genes such as *cspA*, *pgpH*, and the two-component regulatory system *lisRK*, which play an important role in enabling certain strains to better withstand cold stress [21–23]. Strains with enhanced cold tolerance may be more likely to persist in refrigerated environments typical of FPEs, contributing disproportionately to contamination risks.

Despite ongoing efforts, the persistence dynamics of *L. monocytogenes* in FPEs remain unclear. While several studies have focused on strain-specific traits that enhance resistance, increasing attention is being paid to the role of the broader microbial community within FPEs. These microbial ecosystems may influence the survival of *L. monocytogenes*, for example, through competitive or synergistic interactions that affect biofilm formation or disinfectant resistance. FPEs, where microbial communities converge, are ideal for studying the dynamics of bacteria that cause foodborne diseases within the One Health framework. The WHO's One Health concept examines connections between FPEs, animals (like dairy cows), and consumer health, and reinforces the need to understand how microbial ecology in FPEs contributes to the persistence of *L. monocytogenes*.

Current methodologies for studying and tracking foodborne disease pathogens involve whole-genome sequencing data, analyzed through allele calling algorithms, and identification of Single Nucleotide Polymorphisms (SNPs), also known as point mutations. When a new allele emerges, the organism's genome diverges phylogenetically, potentially placing it in a new group. To accommodate this, the multilocus sequence typing (MLST) technique has been continually expanded to include more genes, facilitating the grouping of organisms. Both the core genome, MLST (cgMLST), and whole genome, MLST (wgMLST), incorporate more loci for comparison. While these methods are highly effective for tracking pathogens, they may have limitations when studying persistence.

This article proposes analyzing persistence through population genomics. The evolution of *L. monocytogenes* populations in FPEs is influenced by horizontal gene transfer, natural selection, and genetic drift, which affect bacterial diversity and frequency shifts. They also are exposed to various stressors, promoting rapid evolution.

Tracking sources of contamination relying on molecular typing may not fully capture the evolutionary pressures on these populations. We aim to elucidate these limitations and propose more effective strategies for studying *L. monocytogenes* persistence in FPEs. Specifically, we examine how *L. monocytogenes* populations respond to stressors such as exposure to disinfectants, adaptive advantages favoring certain strains under natural selection, the increase in the frequency of certain strains due to genetic drift from reduced population sizes, and colonization. This perspective paper provides a comprehensive view of *L. monocytogenes* dynamics in response to environmental challenges within FPEs.

2. Persistence through expression of genes involved in stress response and natural selection

In the dynamic environment of FPEs, various strains of *L. monocytogenes* are continuously introduced through raw materials and the workforce, increasing bacterial diversity. Although diversity is expected to be high, specific strains consistently dominate over long periods, posing a threat to food safety. This dominance suggests that their persistence may result from niche adaptation.

FPEs are regularly washed and disinfected to lower the microbial load and reduce the risk of transmitting *L. monocytogenes* through food. In this context, could persistence be linked to the selective pressure from disinfectants? *L. monocytogenes* strains carrying the *bcrABC* gene cassette or the *qacH* gene exhibited significantly higher resistance to benzalkonium chloride (BAC), a commonly used quaternary ammonium compound (QAC), than strains lacking these genes [24]. These genes are often associated with mobile genetic elements, such as the transposon Tn*6188* (*qacH*) or pLM80-like plasmids (*bcrABC*), facilitating their spread by horizontal gene transfer. In Norwegian meat and salmon processing plants, 22% and 8% of isolates were found to carry *qacH* and *bcrABC,* respectively, figures that have been exceeded in more recent surveys, possibly due to environmental selection pressure and gene transfer mechanisms [17, 24]. Furthermore, adaptation to commercial QAC disinfectants has also been shown to induce cross-resistance to certain antibiotics, raising additional concerns about the wider implications of disinfectant use [25]. Taken together, these findings highlight that exposure to these compounds may drive the evolution and prevalence of strains that are resistant to them.

Figure 1(A) illustrates the impact of exposing an *L. monocytogenes* population to a disinfectant, which selectively eliminates susceptible strains through natural selection, thereby reducing their frequency within the population. Continued exposure to disinfectants is thus expected to decrease diversity in the population

by favoring resistant strains, which will gradually dominate and persist over time. Yet, due to architectural barriers, biofilm formation, dilution to sub-lethal concentrations, or other factors, the disinfectants will not be effective against all bacteria. As a result, some susceptible cells may remain in the population, albeit at a lower frequency, and may eventually be eliminated.

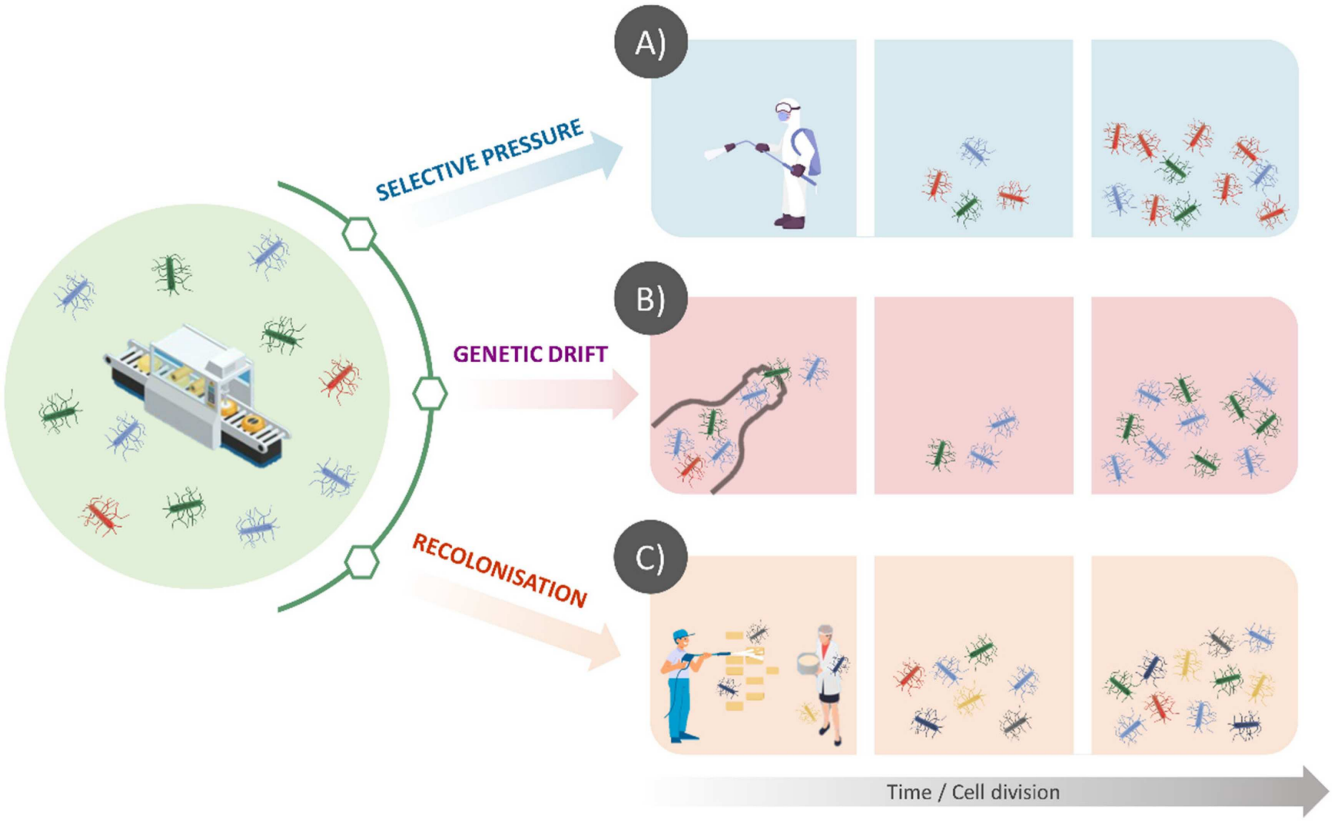

Figure 1 • Events involved in the dynamics of a *Listeria monocytogenes* population in a food processing environment. The green circle illustrates the food processing environment contaminated with *L. monocytogenes* strains, represented in blue, orange, and green. Selective pressure (**A**), genetic drift (**B**), and recolonization (**C**) are events that can occur at the plant level and alter the dynamics of the *L. monocytogenes* population over time. Illustrations are taken from Freepik (Available from: https://br.freepik.cpm, [cited 2024 July 1]).

Genes that have been identified as possibly associated with persistence include stress survival islets SSI-1 and SSI-2, genomic islands LGI-1 and LGI-2, genes encoding resistance for heavy metals (cadmium and arsenic), disinfectant tolerance (*qacC, qacH*, and *emrC*), and the efflux cassette *bcrABC*, encoded in mobile genetic elements [26]. These may increase the tolerance to disinfection and/or biofilm formation and provide potential molecular targets for selectively eliminating persistent strains.

Periodic exposure to disinfectants in the factory exerts selective pressure **(A)** on the population. This selection pressure increases the frequency of strains most suited to survive in these conditions. While not all strains may be eliminated by stressors such as cleaning and disinfection, the genetically resistant ones will become more common in the population. Genetic drift **(B)** can randomly favor certain strains, for example, due to a bottleneck effect in the population. In this case, this reduction in population size can occur as a result of washing and disinfecting processing surfaces. It is a random event in which a strain becomes more frequent merely by chance, not because it has a higher fitness. The more prevalent strains have a greater chance of passing through the bottleneck and expanding because of their higher frequency. This factor unbalances the original frequencies of the strains present in the food plant, as the blue and green populations are enriched, and the orange is eliminated. Subsequently, there is clonal expansion of these blue and green strains. Recolonization **(C)** of the factory by strains may occur during cleaning, as *L. monocytogenes* cells may be hidden in zones of difficult access such as cracks in walls or being part of biofilms (represented by the blue and grey strains on the bricks). As these biofilms grow, they can release cells, contributing to recolonization. The hose projects spray water against the wall and these splashes recolonize the previously disinfected food processing machinery. Contaminated clothing worn by machine operators can also contribute to the migration of new strains into the factory population (represented by the yellow and blue strains). This also results in the recolonization of the factory population by bacteria from walls, clothes, and other surfaces. All of these processes can change the population dynamics of *L. monocytogenes* present over time in food processing plants.

Newly introduced bacteria may acquire resistance genes through horizontal gene transfer, enabling them to compete with resident strains already adapted to the FPE, or they may quickly perish. Mutations can also introduce new variant sequences into a population, enhancing genetic diversity and eventually generating a resistant phenotype. New mutations can be synonymous (dS) or non-synonymous (dN) and may occur in coding and non-coding regions. A mutation can occur in a coding sequence and $dN/dS \neq 1$; thus, it affects the bacterial fitness, and it will be prone to natural selection.

A beneficial mutation with fitness 1 + s (where s is the selection coefficient) is much more likely to become fixed in a population when s > 1/N (where N is the effective population size). Conversely, a deleterious mutation with fitness 1 − s has a very low chance of fixation under the same condition [27, 28]. Natural selection reduces the genetic diversity by eliminating alleles associated with lower fitness. However, when the selective pressure is very low (s < 1/N), both beneficial and deleterious mutations are considered nearly neutral.

While the cgMLST method is a benchmark for providing comparable data on prevalent strains, it has limitations in capturing detailed gene-level information for two main reasons. First, adaptive trait genes are often located on mobile genetic elements like plasmids, which are non-species-specific and typically excluded from a bacterial clade's core genome, and thus are not included in cgMLST studies. Consequently, we believe cgMLST is not the most suitable method for studying *L. monocytogenes* adaptation to FPEs. Second, cgMLST algorithms cannot link a genetic profile to a phenotype. Silent (neutral) mutations, such as dS or changes in non-coding regions, can create new alleles or sequence types without any biological impact. Identifying a new sequence type might lead to its classification as sporadic, even if it is phenotypically similar to its predecessor. The "new clone" could dominate the population due to genetic drift, which we will discuss in the next section. This could complicate result interpretation and mask the persistence of a strain in the factory environment.

3. Persistence due to genetic drift and founder effect

The ability to grow is crucial for the persistence of *L. monocytogenes* [29]. Louise Grinyer outlines six steps to control it in manufacturing, catering, and retail settings. To assess the impact of cleaning, consider genetic drift, which causes random changes in allele frequencies, unlike natural selection. Drift plays a key role in bacterial evolution, particularly in low-bio-burden environments like food processing environments [30].

Mutations occur randomly in bacterial genomes, but new alleles do not always have significant effects on phenotypes or population dynamics. Neutral changes (dN/dS ≈ 1) and mutations in non-coding or non-regulatory regions do not impact fitness and are not targeted by natural selection. When selective pressure is very low (s < 1/N), both beneficial and deleterious mutations are nearly neutral, with random drift being more influential than selection in their fixation probability. In such cases, new alleles can either become fixed or disappear randomly due to genetic drift. Most will be lost within a few generations due to their low numbers, even if beneficial [27]. Occasionally, a mutation becomes fixed, spreading and creating new allelic variants, a process utilized in cgMLST analysis.

The probability of fixation for a neutral mutation is 1/N [28]. Thus, population size plays a crucial role in the impact of genetic drift. In small populations, genetic drift exerts a greater effect as random events can lead to large changes in allele frequencies. In larger populations, the impact of genetic drift is diluted as more individuals buffer against random fluctuations.

Genetic drift tends to be a stronger evolutionary force in small populations such as *L. monocytogenes* in FPEs. As already mentioned, FPEs are regularly washed and disinfected, leading to periodic reductions in the size of bacterial populations. This reduction constitutes a genetic bottleneck. The bottleneck effect occurs when a population experiences a dramatic depletion in size due to an event such as a natural disaster, disease outbreak, or human activities [31]. This reduction in population size can significantly alter allele frequencies and reduce genetic diversity. The surviving population may not be genetically representative of the original population, leading to a loss of genetic variation and potentially reducing the population's ability to adapt to new selective pressures.

Figure 1(B) illustrates the impact of a population size reduction in *L. monocytogenes* through a bottleneck mechanism. This scenario exemplifies genetic drift, which reduces genetic diversity and can thus explain the persistence of certain strains. The founder effect and population bottlenecks are demographic events that affect the intensity of drift. Strains that are more frequent in the population have a higher probability of passing through the bottleneck, continue to increase in frequency, and thus persist over time. The founder effect occurs when a small group of individuals breaks away from a larger population to establish a new one. The allele frequencies of the original population may not be reflected in the genetic makeup of this new "founder" population. Consequently, the new population may exhibit reduced genetic diversity and altered allele frequencies, leading to the rapid fixation or loss of alleles purely by chance.

It is important to note that the more frequent strains are more likely to become founders, and consequently, are more likely to persist in this specific niche.

4. Persistence by repopulating through migration and recolonization

The persistence of *L. monocytogenes* in FPEs can also result from recolonization caused by splashes during the washing process from non-food contact areas. It is not difficult to envisage how this is facilitated by the founder effect when the bacterial load is low. Random drift should therefore play a more important role in the establishment of an incoming strain than natural selection and environmental adaptation.

Figure 1(C) represents the effect of the systematic recolonization of the resident *L. monocytogenes* population in FPE through splashes and aerosols generated during cleaning, originating from areas outside the processing line, such as floors or walls, as well as from the entry of personnel and biofilms [32]. This re-inoculation of populations with low diversity, resulting from ongoing evolutionary processes such as natural selection by disinfectants and other stressors, and genetic drift, provides ideal conditions for the rapid proliferation of these strains. They can then be classified as persistent.

5. Conclusions

The persistence of certain *L. monocytogenes* strains in FPEs poses a significant public health risk, especially to vulnerable populations. Addressing this challenge is crucial for reducing foodborne

illness risks. We propose that examining persistence through population dynamics offers valuable insights, particularly the roles of adaptation to disinfectants and genetic drift. Genetic drift, which has a greater impact in small populations, may significantly influence persistence. By considering these dynamics, we aim to enhance understanding and improve strategies for reducing health risks associated with *L. monocytogenes*.

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Conceptualization, T.N. and G.A.; writing—original draft preparation, C.P.F.D. and T.N.; writing—review and editing, C.P.F.D., G.A., P.T. and T.N.; supervision, T.N.; project administration, P.T.; funding acquisition, P.T. All authors have read and agreed to the published version of the manuscript.

Conflict of interest

The authors declare no conflicts of interest.

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Data supporting these findings are available within the article, at https://doi.org/10.20935/AcadMolBioGen7715, or upon request.

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