Food Reviews International

ISSN: 8755-9129 (Print) 1525-6103 (Online) Journal homepage: www.tandfonline.com/journals/lfri20

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To cite this article: Pedro Sousa, Rui Magalhães & Paula Teixeira (07 Jun 2026): Survival and Adaptation of *Listeria monocytogenes* in the Food Industry: Insights into Persistence Mechanisms, Food Reviews International, DOI: 10.1080/87559129.2026.2682346

To link to this article: https://doi.org/10.1080/87559129.2026.2682346

FOOD REVIEWS INTERNATIONAL
https://doi.org/10.1080/87559129.2026.2682346

Survival and Adaptation of *Listeria monocytogenes* in the Food Industry: Insights into Persistence Mechanisms

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ABSTRACT

Listeria monocytogenes is a well-known foodborne pathogen responsible for severe cases of listeriosis, an infection that primarily affects individuals with a compromised immune system or other underlying conditions, such as the elderly or pregnant women. This microorganism can persist in food processing environments due to its resistance to a wide variety of adverse conditions, posing a significant food safety concern. Hence, it is essential to further understand which factors contribute to this persistence, including the pathways by which this microorganism can endure such harsh environments within food processing facilities. This review explores the survival mechanisms of *L. monocytogenes* strains and the current methodologies applied to analyse their distinctive persistent behaviour. Recent advances in genomic and computational approaches are reviewed, highlighting their potential to improve the identification, characterisation, and prediction of *L. monocytogenes* persistence in food processing environments. Understanding these aspects may be crucial for developing an effective control strategy that can be successfully implemented in the food industry.

KEYWORDS

Food processing environment contamination; stress tolerance; persistent strains; foodborne pathogen; genomic analysis; risk mitigation

Introduction

Listeria monocytogenes is a well-documented facultative anaerobic, gram-positive bacillus, commonly found in a wide variety of environments, such as soil and water, and is responsible for listeriosis, an uncommon but severe disease with high mortality rates, particularly among vulnerable populations, directly caused by the ingestion of contaminated food products.[1–3] Able to grow in a temperature range of 0°C to 45°C, pH between 4.1 and 9.6 and water activity (a_w) as low as 0.90, this foodborne pathogen can survive in food products for extended periods of time.[4,5]

L. monocytogenes is an intracellular pathogen capable of invading different cell types, such as epithelial cells, and targeting various organs, such as the liver.[6] In immunocompromised individuals, this pathogen can induce fatal conditions, such as meningitis and sepsis, and in pregnant women it may lead to premature birth or abortion.[7,8] According to the European Food Safety Authority (EFSA) Zoonoses report,[9] 3041 confirmed cases of human listeriosis were observed in 2024, the disease with the highest proportion of hospitalisation and mortality, with a significant rising trend observed since 2020. Based on the same report, at distribution level, fermented meat products (fermented sausage) were identified as the main sources of positive *L. monocytogenes* samples. The ability of *L. monocytogenes* to persist in food and feed processing environments (FFPE) through physiological attributes such as cross-protection, stress-tolerance, and biofilm formation is a significant challenge for food safety and a contributing factor to listeriosis outbreaks.[10]

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Currently, microbial tolerance to common sanitizers used in the FFPE, such as quaternary ammonium compounds (QACs), mediated by the presence of certain tolerance genes or by *L. monocytogenes* biofilm formation capacity, is an increasing concern worldwide.[11,12]

Within FFPE, *L. monocytogenes* is frequently detected on floors, drains, conveyor belts, and utensils such as knives, grinding machines, or slicers.[13–15] The presence of *L. monocytogenes* on these surfaces represents a major pathway for its dissemination within FFPE, contributing to cross-contamination, through the transference of *L. monocytogenes* from niche sites into food contact surfaces or directly to products, causing not only a considerable health hazard for consumers but also significant economic losses for producers.[16,17] Even though different strategies are used to control or prevent contamination, such as the use of lactic acid bacteria, UV radiation, or surfactant agents,[18] the continued presence of *L. monocytogenes* in industrial environments, particularly in areas with difficult access, is an increasing public health concern.

Due to its virulence and threat to high-risk groups, surveillance of *L. monocytogenes*, covering prevalence, contamination sources, and transmission routes, is essential.

Understanding the mechanisms that confer resilience is also critical. Therefore, this review article integrates physiological, genomic, and ecological perspectives to better understand persistence as a multifactorial phenomenon, organized into several sections to provide a structured overview of *L. monocytogenes* persistence. It outlines key points associated with the definition of *L. monocytogenes* persistence, the adaptive mechanisms that may contribute to modulation and adaptation of this microorganism to food industry environments, as well as the genomic methodologies and bioinformatic tools currently being used to support the analysis of *L. monocytogenes* persistent strains. Ultimately, this review work converges on highlighting critical points and current challenges to be considered in future research directions, emphasizing the need for integrated, ecology-driven approaches to better understand and control *L. monocytogenes* persistence in industrial settings.

Listeria monocytogenes persistence

L. monocytogenes has a remarkable capacity to sustain harsh environmental conditions, such as high salt concentrations, low temperatures, low pH, and the presence of biocides or heavy metals, along with the potential induction of persister cells formation under these stresses.[19–21] These mechanisms may be linked to the extraordinary capacity of some *L. monocytogenes* strains to survive for extended periods in FFPE, a pattern usually recognised as persistence (Fig. 1). It is important to note that, even though "persister" and "persistent" (or "persistence") are often used interchangeably, they refer to distinct concepts and are frequently, but incorrectly, merged.

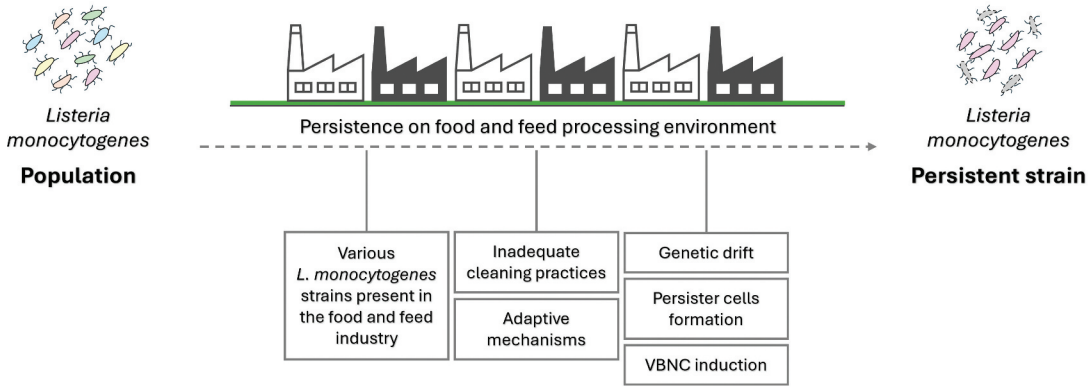

Figure 1. Occurrence of a persistent *Listeria monocytogenes* strain from an initial heterogeneous population through adaptive mechanisms, such as persister cells formation, within FFPE.

Persister cell formation

As recently revised by Serrano et al.[22], "persister cells" are defined as a subpopulation of bacteria capable of surviving exposure to bactericidal concentrations of a given antimicrobial agent. Importantly, these cells remain in a non-replicative state under such conditions, enabling their persistence regardless of exposure to lethal treatments. This is unlike "resistant" bacteria, whose populations can both survive and replicate in the presence of the drug, up to concentrations determined by its minimum inhibitory concentration (MIC).[23] Contrary to resistance, which can be inherited or acquired by various genetic mechanisms from the parent population, persister cells remain as susceptible as the parent population when re-grown in the absence of the antimicrobial agent.[21] Also, upon exposure to increasing antibiotic concentrations or a prolonged exposure to a specific concentration, persister cell formation can be identified by biphasic killing curves, in which sensitive cells die rapidly while persister cells establish as the dominant population, and the overall killing rate reaches a plateau.[24,25] Persister cells formation can be mainly initiated through two separate mechanisms, referred to as type I and type II sub-populations. Type I persisters are described as non-growing and metabolic dormant cells generated in stationary phase in response to environment factors (e.g., antibiotic presence) while Type II persisters are slowly growing cells continuously generated within the total population without the influence of external factors.[22,26,27]

The formation of *L. monocytogenes* persister cells has been observed following exposure to high levels of certain antimicrobial agents, including gentamicin, norfloxacin, and nisin.[21,28,29] Other food-related pathogens have also been reported to form persister cells upon exposure to an inducing stimulus, including *Bacillus cereus*, *Staphylococcus aureus*, or *Escherichia coli*.[22]

Hence, *Listeria* persister cells may be triggered as an adaptation mechanism that potentially contributes to the persistence of this food pathogen in FFPE, although persister formation alone does not constitute persistence.

Persistent and pervasive *Listeria monocytogenes*

Three bacterial food-related pathogens are considered the most relevant in terms of persistence in the FFPE by EFSA,[16] namely *L. monocytogenes*, *Salmonella enterica*, and *Cronobacter sakazakii*. Persistent *L. monocytogenes* strains are defined in the literature as those repeatedly isolated from the same source or environment at different times, exhibiting identical subtypes as determined by phenotypic or genotypic tools.[30,31] In contrast, "non-persistent" or sporadic strains are occasionally isolated from the same source and display distinctive subtypes.[32] Finn et al. [33] proposed a shortened and standardised definition of persistence, describing it as strains of the same subtype isolated on two or more occasions over 6 months. Based on their conclusions, a well-standardised definition of persistence is vital for reliably comparing studies, requiring both high-discriminatory subtyping methods and accurate isolation methodologies. Similar concerns regarding the definition of persistent *L. monocytogenes* had already been raised earlier.[30] The expression "pervasive *L. monocytogenes*" was introduced to define persistence, referring to genetically similar strains, but isolated from different food processing facilities and environments.[34] The differences between the three types of strains, namely sporadic, persistent, and pervasive, are illustrated in Fig. 2.

Considering this, we believe both terminologies, "persistent" and "pervasive" strains, should be progressively taken into consideration in future research on *L. monocytogenes* persistence, to differentiate between strains confined to a single facility and strains with genetic similarity detected across different locations. Since *L. monocytogenes* is known to be widely present in the environment and can also be asymptomatically carried by both humans and animals,[35] pervasive strains may be more common and widespread in the food chain than is currently recognized.

Listeria monocytogenes adaptive mechanisms

L. monocytogenes exhibits a remarkable ability to resist and induce cellular alterations, allowing it to persist under stressful conditions. Several mechanisms are directly linked to the adaptive capacity of

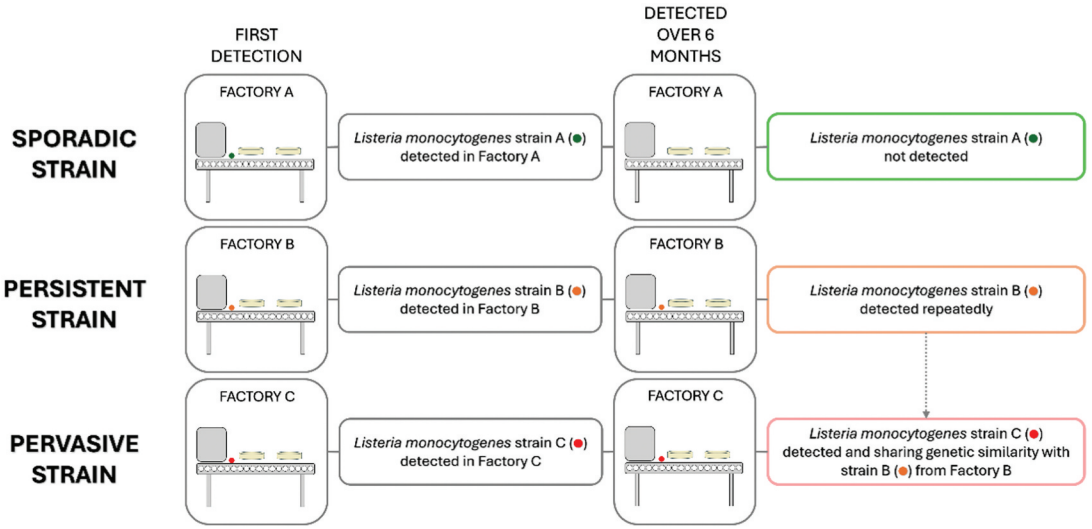

Figure 2. Representation of the occurrence of *L. monocytogenes* in three different food and feed processing factories (A, B and C). Factory a represents a sporadic strain, characterized by infrequent isolation of a specific strain. Factory B represents a persistent strain, corresponding to a repeated isolation of a specific strain over a continuous period of time. Factory C represents a pervasive strain, defined by widespread and recurrent detection of genetically similar strains (strain B and C) but isolated from multiple factories.

this microorganism. For example, upregulation of sigma factor B (σ^B), the presence of specific plasmids (e.g., pLM58) and stress associated proteins (e.g., ClpL), thiol-disulfide oxidoreductases, and efflux pumps contribute to its tolerance to low pH, high temperature, oxidative stress, and antimicrobial agents.[36–39] Additionally, certain proteins involved in gene regulation, such as cold shock proteins (CSPs), play a crucial role in *L. monocytogenes* survival by modulating molecular and biological response mechanisms.[40–42]

Cellular rearrangements in response to environmental conditions also occur, promoting oxidative-stress defences, fatty acid biosynthesis, and other adaptations in the *Listeria* cell wall.[40,43,44] Other cellular components, such as surface-associated internalins (Inls), which are not directly linked to persistence,[45,46] may possibly provide certain resilience advantages, including enhanced adhesion during biofilm formation.[47,48] Alongside these functions, *L. monocytogenes* can develop biofilms protecting cells within clusters from environmental stresses. Treatments targeting biofilms can also induce a viable but non culturable (VBNC) state, further contributing to persistence in food processing environments.[49] Recent studies have shown that *L. monocytogenes* resistance to disinfectants such as benzalkonium chloride (BAC) can be enhanced by the presence of other microorganisms, such as *Pseudomonas* spp., within their biofilms,[50–52] reinforcing the importance of controlling biofilm formation in FFPE.

Considering these factors, five main mechanisms contributing to *L. monocytogenes* persistence were selected for further assessment on this review (Fig. 3).

Cross-protection (CP)

Repeated exposure of *L. monocytogenes* to antimicrobial agents (e.g. peroxyacetic acid) is known to increase its tolerance, requiring higher concentrations to achieve the same antimicrobial effect.[53] Such exposure promotes adaptive responses that enhance survival under stress and may also confer cross-protection against other adverse conditions. These stress tolerance and cross-protection mechanisms contribute to the resilience of *L. monocytogenes* under continuous or repeated stress.[54]

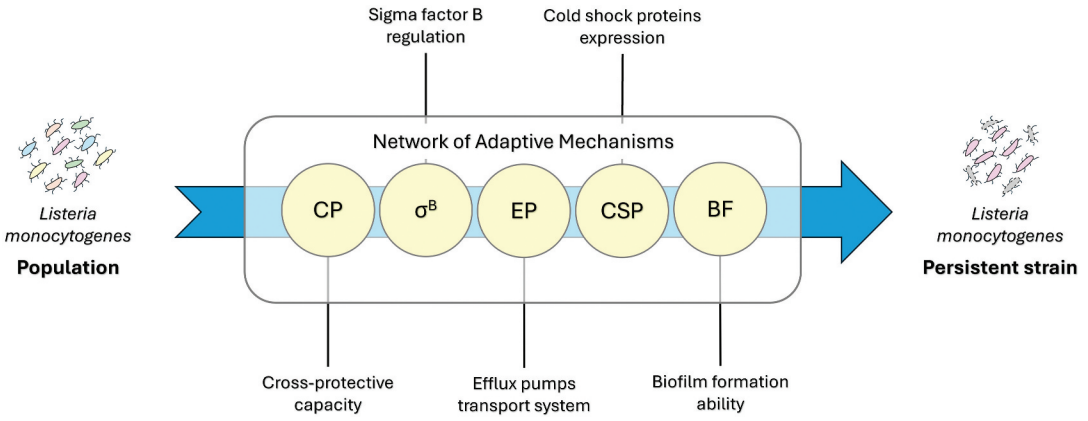

Figure 3. Network of adaptive mechanisms, namely cross-protection (CP), sigma factor B (σ^B), efflux pumps (Ep), cold shock proteins (Csp) and biofilm formation (Bf), contributing for the development of *L. monocytogenes* persistent strains in FFPE.

Lundén et al.[55] reported that when two persistent and two non-persistent *L. monocytogenes* strains were exposed to sublethal levels of different disinfectants, they developed cross-adaptive responses among themselves. The authors observed that one of the persistent strains showed higher MIC values than non-persistent strains. Interestingly, potassium persulphate was the only compound that did not induce cross-adaptation, even though exposure to it triggered cross-resistance against other disinfectants assessed. A similar pattern was reported by Yu et al.[56] who found that exposure of *L. monocytogenes* strains to increasing doses of BAC, a common disinfectant, not only increased resistance to BAC but also to other antibacterial agents such as cefotaxime and cephalothin. Likewise, Bland et al.[57] observed cross-protection ability, with reduced sensitivity to seven different antibiotics, after exposing this foodborne pathogen to sublethal concentrations of a commercial disinfectant derived from QACs.

Subinhibitory concentrations of biocides, such as peracetic acid or polyvinyl pyrrolidone iodine, also seem to affect the susceptibility of sessile (i.e. multicellular-life phase) *L. monocytogenes* cells to chloramphenicol and tetracycline, respectively.[58,59] Nonetheless, some of these adaptive responses and their cross-effects may be directly impacted by other factors, such as temperature. For example, Shen et al.[60] reported that acid-adapted *L. monocytogenes* cells grown at pH 5.0 for 1 h at 37°C retained this tolerance when incubated at 4°C, but not at 22°C or 37°C, after growing at pH 7.2 for 24 h. Notably, prior exposure to low temperature did not improve microbial tolerance to acid stress. This study also demonstrated that acid-adapted cells exhibited cross-protection against lethal doses of lauric arginate when immediately exposed after growing in acidic conditions; however, this resistance was entirely lost after 60 min at 4°C even though the acid-adapted state remained stable. The authors suggested that depending on the mechanism developed to increase cellular resilience and potential cross-protection, stability can differ. These findings are particularly relevant in FFPE, where *L. monocytogenes* is routinely subjected to fluctuating temperatures and diverse environmental stressors, with direct implications for the stability of adaptive responses and the efficacy of antimicrobial interventions.

Since it's known that cellular modifications such as alteration in membrane fatty acid composition can occur to support growth at lower temperatures,[61,62] these adjustments may interfere when *L. monocytogenes* is simultaneously exposed to common environmental stressors faced in food processing settings, including exposure to sanitizers, temperature fluctuations, osmotic pressure, or the presence of preservatives.[63] This represents an important limitation when assessing potential

cross-protection, as such effects may be detectable under lab-scale conditions but lost under variable environmental parameters, such as temperature or exposure duration.

Further investigation into the cross-protective ability of persistent versus non-persistent *L. monocytogenes* strains is needed to understand whether differences exist. Since exposure to sublethal levels of disinfectants may lead to higher tolerance not only to the same compound but also to other antimicrobial agents, future studies should carefully evaluate these variations, particularly considering the role of temperature in both the development and maintenance of such adaptive responses. At the molecular level, these adaptive responses are often orchestrated by transcriptional regulators, such as sigma factors (e.g. σ^B), as discussed in the following section.

Sigma factor B (σ^B)

Sigma factors are transcriptional regulators responsible for the activation of regulons and housekeeping genes, with a clear influence in *L. monocytogenes* virulence and stress response.[64] In this microorganism, five sigma factors have been identified, including σ^A, σ^B, σ^C, σ^H, and σ^L.[65] Among them, sigma factor B (σ^B) has been extensively studied, not only for its role in regulating *Listeria*'s robustness and general stress response,[66–70] but also for its contribution to the regulation of the positive regulatory factor A (PrfA), which directly impacts *L. monocytogenes* virulence.[71,72]

Becker et al.[73] demonstrated that a σ^B *L. monocytogenes* mutant strain exhibited a low capacity to accumulate betaine, an osmoprotectant, after exposure to 3% of NaCl, compared to the wild-type strain. The same σ^B dependence was observed for the bacterial growth under high-salt (1.75 M of NaCl) and acidic (pH 2.5) conditions.[74] Interestingly, the mutant strain demonstrated a better capacity to grow at 0.5 M of NaCl in comparison to the parent strain, which the authors attributed to possible alterations in key metabolic pathways under adverse conditions, as previously noted in other bacteria. Importantly, loss of σB was reported to induce hyper-resistance to hydrogen peroxide in *L. monocytogenes* under aerobic conditions, reinforcing the impact of σB modulation in potentially providing protective mechanisms, in this case by promoting oxidative stress defences.[75]

Wemekamp-Kamphuis et al.[76] further investigated the role of σ^B in acid stress and found that σ^B deletion markedly reduced survival at pH 2.5, resulting in a 5-log difference compared with the wild-type strain. Pre-exposure to a non-lethal pH of 4.5 slightly improved survival, suggesting the activation of σ^B independent protective mechanisms. Similarly, Ferreira et al. [77] observed that the σ^B mutant displayed lower resistance at pH 2.5, both with and without prior adaptation to pH 4.5, and also showed reduced survival in simulated gastric fluid. Overall, survival in this environment was dependent on the growth phase, being enhanced in stationary phase and partially dependent on σ^B.

A comparable study observed the same importance of σ^B in the survival capacity of *L. monocytogenes* strains under acid conditions (pH 2.5), oxidative stress, and carbon starvation stresses though similar viabilities were observed between mutant and wild-type strains under heat (50 °C) or ethanol (16.5%, v/v) stresses.[78]

A large-scale genomic analysis of 46,921 *L. monocytogenes* genomes revealed two variants of σ^B, namely $\sigma^B{}_{T1}$ and $\sigma^B{}_{T2}$, associated with lineage I/III and lineage II, respectively. The authors suggested that $\sigma^B{}_{T1}$ may contribute to enhanced invasiveness, virulence and persistence in the environment observed in lineages I and III, potentially serving as a useful marker of epidemiological distribution of this pathogen.[79]

Overall, σ^B appears to be strongly linked to *L. monocytogenes* survival under adverse environmental conditions and thus may play a key role in its persistence. Nonetheless, further studies are needed to clarify how σ^B contributes to persistence specifically in FFPE and whether differences exist between persistent and non-persistent strains under such conditions. Since σ^B controls the general stress response and influences adaptive mechanisms (e.g., efflux systems), this factor activation may be a critical trait for *Listeria* persistence.[69,80]

Efflux pumps (EPs)

Bacterial efflux pumps (EPs) are transport proteins that remove various compounds (e.g. antibiotics) from the intracellular to the extracellular environment, thereby contributing to multidrug resistance.[81,82] Due to their importance, EPs inhibition has been proposed as a strategy to reduce drug resistance, virulence, and biofilm production.[81–84] In *L. monocytogenes*, inhibition of these systems not only reduces toxic compounds removal but also impacts flagellum formation, invasiveness, and the production of virulence-associated proteins.[85]

As reported in the literature on *L. monocytogenes*, MdrL and Lde are two well-known efflux pumps, which play a crucial role in resistance to disinfectants such as BAC and antibiotics such as ciprofloxacin, respectively.[86–89] Romanova et al. [88] evaluated eight *L. monocytogenes* strains (four Bac-resistant and four Bac-sensitive) and showed that exposure of sensitive strains to sub-lethal BAC concentrations led to the overexpression of the *mdrl* gene. This gene is associated with BAC resistance.[90,91] No differences were observed for the gene *lde*, despite its known involvement in fluoroquinolone resistance.[39,86,92] Interestingly, the addition of an efflux pump inhibitor (reserpine) reduced the MIC values of sensitive but not resistant strains, suggesting that resistant isolates may rely on additional survival mechanisms beyond *mdrL*-mediated efflux. These findings were consistent with those of a previous study,[93] which confirmed that efflux pumps are key for adaptation to BAC in sensitive strains but not in naturally resistant ones, supporting the existence of alternative tolerance mechanisms.

Another gene implicated in resistance to QAC-based biocides is *emrE*. Mutation of this gene in *L. monocytogenes* increased susceptibility to QAC sanitizers (BAC and E-San) in comparison to the parent strain, although no differences in the MIC values were observed for other antimicrobial agents tested, including erythromycin and triclosan.[94] More recently, Schulz et al. [95] demonstrated that *L. monocytogenes* EGD-e adapted to BAC and cetyltrimethylammonium bromide (CTAB), developed mutations in efflux pump-related genes. In BAC-tolerant strains, mutations in *fepR* led to upregulation of FepA efflux pump, conferring increased resistance not only to BAC but also to gentamycin, CTAB, and ciprofloxacin. CTAB tolerance, on the other hand, was associated with mutations in *sugR*, which regulates SugE1/E2 efflux pumps. Remarkably, even in the absence of FepA or SugE1/2), *L. monocytogenes* could still adapt through compensatory activation of alternative efflux systems. When both EPs were absent, mutations in *lmo1753*, encoding a diacylglycerol kinase, appeared to facilitate membrane remodelling, suggesting an export-independent adaptation mechanism.

Bechtel et al.[96] further reported that ethyl methanesulfonate (EMS) induced mutations in a BAC-susceptible *L. monocytogenes* strain resulted in two mutants with increased BAC tolerance. These mutants showed upregulation of *lmo2463*, encoding a MmpL family transporter protein, which was associated with increased resistance to both BAC and fluoroquinolones. Mutations affecting genes related to cell membrane organization, such as *lmo1753* (diacylglycerol kinase) or *lmo2768* (permease) may also enhance *L. monocytogenes* survival in food processing environments. In addition to these findings, both mutants exhibited significantly higher haemolytic activity compared to the parent strain.

Generally, most of these studies report tolerance development at disinfectant concentrations below those recommended by manufacturers. However, such sublethal concentrations can easily occur in practice, for example in certain areas, such as floors or drains, due to inadequate cleaning or dilution.[16] Spontaneous mutations induced by oxidative stress due to disinfectant exposure can lead to efflux pump overexpression further contributing to *L. monocytogenes* tolerance in FFPE.[87,95,97] To the best of our knowledge, no studies have specifically investigated whether persistent *L. monocytogenes* strains exhibit enhanced efflux activity compared with sporadic strains, representing an important knowledge gap in understanding their persistence mechanisms.

Cold shock proteins (CSPs)

Cold shock proteins (CSPs), are a group of highly conserved, relatively small proteins, typically consisting of 65 to 75 amino acids and weighing approximately 7.4 kDa, even though sizes can vary among *L. monocytogenes* strains.[98,99] This species possesses three CSPs, CSP-A, CSP-B, and CSP-D, which are widely known to be expressed as a defence mechanism against low temperature downshifts.[100–105] In addition to their role in cold stress tolerance, CSPs have also been reported to influence virulence-associated factors, such as motility or haemolytic activity.[106,107] CSP-A is mostly associated with cold stress adaptation, CSP-B is typically linked to virulence, and CSP-D contributes to both phenotypes.[40]

Under high salt concentrations, Duché et al. [108] observed an upregulation of GbuA (a glycine betaine transporter) as part of the salt-tolerance mechanism, along with two other general stress proteins, DnaK and Ctc. In total, twelve salt-induced proteins were identified, belonging to either the salt-shock or stress-acclimation protein groups. *L. monocytogenes* CspA, CspB, and CspD, provide dual protection against osmotic (NaCl) and low-temperature stresses, with CspA playing a more important role in cold tolerance and CspD contributing more significantly to salt resistance.[103]

Kragh et al.[41] evaluated the impact of CSPs in desiccation tolerance and biofilm development in 29 *L. monocytogenes* strains, 21 wild type and eight CSP-mutant. Their findings indicated that CSPs negatively affect desiccation tolerance (relative humidity of 43% at 15 °C). Interestingly, a correlation between a reduced motility and an increased desiccation tolerance, as well as a possible relation between deleted *csp-A* and a lower capacity to form biofilms was observed by the authors. Consistent with previous observations,[106] mutation of CSPs in *L. monocytogenes* EGDe led to attenuated virulency, reduced cellular aggregation, and impaired flagella biosynthesis and motility. Among the three CSPs genes, *csp-B* followed by *csp-D* was observed to have functional contributions to virulence, which may compromise *L. monocytogenes* host cell invasion when a mutation in these genes occurs.

Using the same *Listeria* wild-type strain and *csp* (A, B, and D) mutant strains, the effects on two virulence factors, hemolysis and listeriolysin O (LLO) production, were assessed.[107] The study showed that deletion of all three genes, or of exclusively *csp-B*, resulted in a significant reduction in LLO production. Intriguingly, when only *csp-B* was deleted, LLO levels remained relatively comparable to those of wild-type strain. On the other hand, only the double (*csp-AB* and *csp-AD*) or triple (*csp-ABD*) mutants had a reduced hemolytic activity, while single deletions did not affect this phenotype.

While CSPs seem to have an important role in various cellular processes linked to their infecting capacity, they have shown to be essential in ensuring *L. monocytogenes* survival upon exposure to refrigeration temperatures. Until now, no studies were done linking CSPs and persistence, although various studies point the importance of csp-A and csp-D proteins in high salt and refrigeration conditions.

Biofilm formation (BF)

Biofilms are complex multicellular structures in which microbial communities are enclosed within extracellular polymeric substances, providing an extraordinary protection against external stresses.[109,110] Briefly, biofilm formation typically occurs in three-steps: (I) aggregation and attachment – bacteria aggregate to each other or attach to biotic or abiotic surfaces; (II) growth and accumulation – cell growth and recruitment; and (III) disaggregation and detachment – cells leave the film as aggregates or singles cells.[111] Biofilm production by *Listeria* strains is an important survival mechanism, increasing its overall resistance against chemical or mechanical removal, contributing to their persistence in FFPE.[33]

A recent study has associated *L. monocytogenes* biofilm formation with high cell surface hydrophobicity, self-aggregation capacity and exopolysaccharide synthesis.[112] Strains with strong biofilm-forming ability, in comparison to their planktonic state, exhibited significantly higher adhesion and invasion of Caco-2 cells and reduced susceptibility to several antibiotics, including erythromycin, clindamycin, ciprofloxacin, and daptomycin. Upregulation of *agrA*, *flgE*, *fliD*, and *inlA* has been linked to biofilm development, whereas *motB* downregulation contrasts with previous studies showing the

importance of motility in surface attachment.[113,114] Liu et al. [115] demonstrated that strains carrying *clpL*, *mdrL*, and *lde* genes exhibited stronger biofilm formation and resistance to sublethal chlorine concentrations (250 mg/L). Interestingly, one of the strains, which possessed six biofilm-associated genes (*actA*, *prfA*, *lmo0673*, *recO*, *lmo2504*, and *luxS*) exhibited strong biofilm formation within the first 4 days; however, this ability declined significantly thereafter. In contrast, the two resistant strains showed a marked increase in biofilm production after day 4. Notably, only these two strains carried the *clpL* gene, associated with stress response, suggesting a potential role for *clpL* in enhancing or upregulating biofilm development under prolonged or stressful conditions. Environmental conditions can also influence biofilm formation and cell adherence. Low temperatures (12 °C) reduced biofilm formation among 19 *L. monocytogenes* strains isolated from a cold-smoked salmon plant, whereas temperature of 30–37°C favoured biofilm development. These isolates showed correlations between biofilm formation capacity and the presence of internalin genes, stress survival islet (SSI), and erythromycin resistance.[48] Strains origin may further influence biofilm behaviour. In a study of 57 food-related strains from Italy (2003–2014), all strains formed biofilms, but moderate to strong biofilm producers were more frequent among meat isolates (57%) than dairy isolates (28%). The presence of *SSI-1*, *arsD*, and truncated InlA protein was linked to higher levels of biofilm production, consistent with other studies reporting similar genetic associations.[116]

Even though persistent strains, as the name suggests, may indicate a stronger biofilm production than sporadic strains, the relationship between biofilm formation and persistence remains debated. Harvey et al. [117] examined the biofilm forming capacity of 138 *L. monocytogenes* strains using both short and long incubation assays. In the first assay, 92% of the strains were classified as weak biofilm formers, 6.5% as moderate and 1.5% as strong biofilm formers. Similar trends were observed between persistent and sporadic food-related strains. When extending the incubation period, many strains demonstrated a considerable improvement in biofilm formation. In this prolonged assay, strains of the less virulent serotype 1/2c, both sporadic and persistent food isolates, were the strongest biofilm formers, whereas the highly virulent serotype 4b failed to form biofilms. Contrary to these findings, among 80 *L. monocytogenes* isolates tested for biofilm formation, no significant differences between serotype and biofilm formation were observed, even though persistent strains from bulk milk samples showed increased biofilm formation.[118] Persistent strains isolated from a chicken-processing plant also tend to form thicker biofilms than sporadic ones. However, once again, no significant relationship between strains serotypes and biofilm formation was detected.[119]

As previously discussed,[33] a deeper understanding of how *L. monocytogenes* biofilms develop is urgently needed, along with the standardization of methods and the redesign of assays to better reflect real food-associated environments. Differences in the strains used, methods applied and environmental conditions tested can lead to inconsistencies among studies, hindering clear interpretation and comparison of results. For example, *L. monocytogenes* biofilm formation in persistent versus sporadic strains, as well as its possible relation with serotype, remains a matter of debate within the scientific community. Several studies have reported a clear relation between enhanced biofilm development and persistence,[118–121] while others do not identify this attribute as a key trait of persistent strains.[117,122–124]

Furthermore, factors such as multi-species biofilms (e.g. *L. monocytogenes* with *Pseudomonas fluorescens*), or biofilm formation under stress conditions have been shown to have an impact on *L. monocytogenes* biofilm development.[125,126] This mutual aid between *L. monocytogenes* and other microorganisms, such as the presence of *Pseudomonadaceae* or *Lactiplantibacillus plantarum* in multi-species biofilms, can enhance its resistance to disinfection agents, such as peracetic acid or BAC.[50,127] Even though these aspects are not considered when evaluating this protective attribute, they should be considered on future studies comparing biofilm formation between persistent and non-persistent strains.

Overall, biofilms clearly represent an important defensive mechanism for *L. monocytogenes, yet* their role as a major contributor for persistence remains uncertain.

Genomics-based methodologies for persistence

Genomic approaches offer promising complementary tools for investigating *L. monocytogenes* persistence. Genomic analysis has revolutionized the study of this pathogen, enabling detailed molecular typing, strain identification, and clustering through diverse methods such as polymerase chain reaction (PCR) amplification-based typing, pulsed-field gel electrophoresis (PFGE) and multi-locus sequence typing (MLST).[128,129] Since some of these subtyping methods (Table 1) can be considerably time consuming, as well as requiring complex methodologies and data analysis, new solutions are trying to significantly reduce the time and complexity of identifying food pathogen within FFPE, including capacity to recognize persistent *L. monocytogenes* strains.

Table 1. Methodologies commonly used for *L. monocytogenes* identification and subtyping, containing the key advantages and main disadvantages of each technique.

Common identification and classification methodologies	Based on	Advantage(s)	Disadvantage(s)	References
MALDI-TOF MS Matrix-Assisted Desorption/Ionization-Time of Flight Mass Spectrometry	Spectroscopy	• Good discrimination power among different microorganisms • Relatively fast identification and classification • Requires limited sample preparation • High cost of acquisition but low cost to operate	• Impacted by the age of the culture and growth conditions • Affected by a limited number of mass spectra in the reference database • Limited discriminatory power for closely related strains and subtypes, making it less suitable for epidemiological investigations and outbreak source tracing	[130–132]
PFGE Pulsed-field Gel Electrophoresis	Restriction enzymes	• Strong discrimination power among different microorganisms • Commonly used as a standard method for food safety surveillance and outbreak investigations • Commonly used in *L. monocytogenes* subtyping studies	• Time consuming • Requires specialized personal and equipment • Lack of standardization rises challenges in inter-laboratory comparisons • Not applicable to non-cultivable microorganisms	[129,132–134]
MLST Multilocus Sequence Typing	Polymerase chain reaction	• Strong clusterization power • Used to study the population structure of *L. monocytogenes* • Categorization of *L. monocytogenes* groups into sequence types (ST) and clonal complexes (CCs)	• Costly and time consuming • Limited discriminatory capacity for serotype 4b	[132,135,136]
WGS Whole Genome Sequencing	Next-generation sequencing	• Considered the current "gold standard" method for surveillance of foodborne pathogens and outbreak investigations • Enables linkage of listeriosis cases over long periods of time The increasing widespread use of WGS is reducing sequencing costs • Provides higher discriminatory power for identifying persistent strains	• High initial investment costs • Requires specialized bioinformatics expertise and robust analytical pipelines • Large amounts of data require substantial storage and computational resources	[137–140]

For this, new approaches such as qPCR-based GENE-UP® TYPER (bioMérieux) can be easily used to subtype and cluster strains, identifying dominant strain recurrently isolated within a factory.[141] Among sequencing-based approaches, whole genome sequencing (WGS) has become the ultimate tool on persistence studies, allowing high-resolution comparisons between persistent and non-persistent strains.[31,140,142] Generally, WGS offers a substantially higher discriminatory power, better data accuracy and richer genomic information compared with traditional typing approaches, which improves its utility for outbreak investigation and source tracing of listeriosis.[143] In addition, novel methods, such as the one developed by bioMérieux, propose a faster and reliable way to cluster strains without needing specific and time-consuming assays, which should really be the main target in development of new methods and devices to assess *L. monocytogenes* persistence in FFPE.

Genomic methodologies usually generate a vast amount of complex data, which requires substantial computational power and specialized knowledge to translate this data into practical results. Due to the decreasing cost of these methods (e.g., WGS) along the time, the quantity "big data" produced is continuously increasing, mainly due to research done on epidemiological studies.[144] The use of computational tools with WGS data holds great potential for advancing persistence research. Currently, such tools are already used in predictive microbiology for food safety, image classification in clinical microbiology, and big data analysis generated by DNA and RNA sequencing.[145–147] Recent advances in this area have led to various studies exploring machine-based methods mainly targeting food safety, such as biofilm development and detection [148,149] or by studying well-known foodborne pathogens, such as *L. monocytogenes*,[150–152] *Clostridium* spp.[153] or *Salmonella* spp.[154,155]

Tanui et al.,[156] for example, successfully applied supervised machine learning (ML) to attribute listeriosis cases to specific food sources, demonstrating how ML can enhance source attribution when fed high-resolution WGS data. Gmeiner et al.[157] used this approach to analyse the virulence of *L. monocytogenes* at the sub-species level using six ML algorithms. The authors demonstrated the potential of these models to assess virulence and variation in this pathogen. ML predictors were developed using WGS and MIC values for QAC from 1649 isolates. The results showed a promising capacity to predict tolerance and MIC values, as well as potential to uncover new genes associated with disinfectant tolerance.[158] Based on this work, Gmeiner et al.[159] recently developed a ML tool, ListPred, designated for the analysis of virulence potential and disinfectant tolerance in *L. monocytogenes*. ML-based WGS sequence analysis can also be valuable for predicting stress response phenotypes, such as acid, cold, salt, and desiccation tolerance which, according to the authors, represent a step forward for food safety and risk assessment regarding this pathogen. Using *L. monocytogenes* as a model, Njage at al.[160] demonstrated that ML models can predict stress phenotype components in novel or previously uncharacterized strains based on WGS data, offering valuable insights for future persistence analysis. Considerable progress has been made using computation-driven solutions applied to *L. monocytogenes*, even though most studies have focused on the virulence and resistance of *L. monocytogenes* strains, failing to differentiate persistent and non-persistent strains.[161] It has also been emphasized that the use of small datasets may limit model accuracy and predictive performance, posing a notorious challenge on the development of such tools. Nevertheless, integrating sequencing data with bioinformatic and ML analysis represents a significant step toward improving the understanding of persistence mechanisms, ultimately supporting more effective food safety management strategies.

Future prospects

Assessing and controlling *L. monocytogenes* persistence in FFPE remains challenging. As reviewed in Listeria monocytogenes persistence-4, persistence is not explained by a single trait but instead emerges from the interaction of multiple adaptive mechanisms (e.g., cross-protection, σ^B-mediated stress response, efflux activity, cold-stress adaptation, and biofilm-associated protection) with the specific environmental conditions encountered in industrial settings.

Several FFPE factors may favour the establishment and long-term maintenance of persistent populations. Ineffective sanitation and inadequate cleaning, particularly in hard-to-reach sites (e.g., drains, niches, and equipment interfaces), can create recurring exposure to sub-inhibitory biocide concentrations, which may select for increased tolerance and promote adaptation. In addition, some disinfectants have been reported to induce a VBNC state in *L. monocytogenes*, suggesting that persistence may sometimes involve reversible physiological states rather than stable genetic resistance.[162] Repeated population bottlenecks during disinfection may also contribute to genetic drift, reducing diversity and potentially enriching variants with traits that favour survival in the facility environment.[163] At the population level, lineage background and phylogeny may influence persistence likelihood, with lineage II strains reported to persist more frequently in FFPE than lineage I in some studies.[164] Microbial interactions are an additional, and still underexplored, determinant of persistence. Resident microbiota can either promote *L. monocytogenes* survival (e.g., cooperative associations within mixed-species communities) or inhibit growth through antagonism and competition.[165] Therefore, a more realistic understanding of persistence will require integrating *L. monocytogenes* physiology with FFPE ecology, including community composition, spatial organization, and the facility-specific selective pressures. Processing environment conditions may also drive genomic adaptation more strongly than host origin, reinforcing the need to study isolates in the context of their industrial niche.[166]

Future studies should therefore adopt a multifactorial framework when comparing persistent and sporadic strains. Rather than evaluating single traits in isolation, experimental designs should combine relevant stresses (e.g., temperature, pH, salinity, desiccation, and repeated biocide exposure) and include conditions reflecting the isolation environment (e.g., dairy-associated pH and salinity conditions in cheese production).[161,167]

In parallel, improved use of WGS data linked to detailed, standardized metadata (sampling site, time, sanitation regime, product/environment type, and co-occurring microbiota) will be essential to identify genomic determinants and to build robust, generalizable predictive models across facilities and countries. Studying mixed-species biofilms and microbial community dynamics in situ should be prioritized to clarify when microbiota promotes persistence versus exclusion.[168,169]

Ultimately, translating these insights into practice will require improved, facility-tailored control measures that minimize the formation of sublethal disinfectant zones and target persistent reservoirs, thereby reducing selection for adaptation and lowering the risk of contamination along the food chain.

Conclusions

Listeria monocytogenes persistence is best understood as a multifactorial phenotype shaped by the interaction between strain background and facility-specific selective pressures, rather than by a single defining trait. As summarized in this review, adaptive responses involving cross-protection, σ^B-associated stress regulation, efflux-mediated tolerance, cold-stress adaptation, and biofilm-associated protection can each contribute to survival and long-term maintenance in FFPE, although their relative importance likely depends on environmental context and the resident microbiota. A key priority for the field is to improve discrimination between persistent and sporadic strains by integrating standardized phenotypic assays with high-resolution genomics. Future research should couple WGS with robust, standardized metadata and realistic experimental designs that reproduce relevant industrial conditions (e.g., repeated sub-inhibitory sanitizer exposure, low temperature, matrix-specific pH/salinity and mixed-species communities). Integrating these datasets through reproducible bioinformatic pipelines and, where appropriate, machine-learning models, will support identification of genetic and ecological determinants underlying persistence, strengthen predictive capacity across facilities, and guide more effective, targeted control strategies to reduce contamination and improve food-chain safety.

Acknowledgments

We would also like to thank the scientific collaboration under the FCT project UID/50016/2025.

Author contributions

CRediT: **Pedro Sousa:** Conceptualization, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing; **Rui Magalhães:** Validation, Visualization, Writing – review & editing; **Paula Teixeira:** Funding acquisition, Project administration, Supervision, Validation, Writing – review & editing.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This work was supported by National Funds from FCT - Fundação para a Ciência e a Tecnologia through project GenoPhenoTraits4Persitence - Genomic and phenotypic traits contributing to persistence of Listeria monocytogenes in food processing environment (PTDC/BAA-AGR/4194/2021), https://doi.org/10.54499/PTDC/BAA-AGR/4194/2021.

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