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Behavior of *Listeria monocytogenes* in the presence or not of intentionally-added lactic acid bacteria during ripening of artisanal Minas semi-hard cheese

Ursula Gonzales-Barron[a,1], Fernanda B. Campagnollo[b,1], Donald W. Schaffner[c], Anderson S. Sant'Ana[b,*], Vasco A.P. Cadavez[a]

[a] CIMO Mountain Research Centre, School of Agriculture, Polytechnic Institute of Bragança, Bragança, Portugal
[b] Department of Food Science, Faculty of Food Engineering, University of Campinas, Campinas, SP, Brazil
[c] Department of Food Science, School of Environmental and Biological Sciences, Rutgers – The State University of New Jersey, New Brunswick, NJ, USA

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ABSTRACT

The fate of *Listeria monocytogenes* during ripening of artisanal Minas semi-hard cheese, as influenced by cheese intrinsic properties and by autochthonous (naturally present) or intentionally-added anti-listerial lactic acid bacteria (LAB) was modeled. Selected LAB strains with anti-listerial capacity were added or not to raw or pasteurized milk to prepare 4 cheese treatments. Counts of LAB and *L. monocytogenes*, pH, temperature and water activity were determined throughout cheese ripening (22 days, 22 ± 1°C). Different approaches were adopted to model the effect of LAB on *L. monocytogenes*: an independent approach using the Huang primary model to describe LAB growth and the linear decay model to describe pathogen inactivation; the Huang-Cardinal [pH] model using the effect of pH variation in a dynamic tertiary approach; and the Jameson-effect with $N_{max\ tot}$ model which simultaneously describes *L. monocytogenes* and LAB fate. *L. monocytogenes* inactivation occurred in both treatments with added LAB and inactivation was faster in raw milk cheese ($-0.0260\ h^{-1}$) vs. pasteurized milk cheese ($-0.0182\ h^{-1}$), as estimated by the linear decay model. Better goodness-of-fit was achieved for the cheeses without added LAB when the Huang primary model was used. A faster and great pH decline was detected for cheeses with added LAB, and the Huang-Cardinal [pH] model predicted higher pathogen growth rate in cheese produced with raw milk, but greater *L. monocytogenes* final concentration in pasteurized milk cheese. The Jameson-effect model with $N_{max\ tot}$ predicted that LAB suppressed pathogen growth in all treatments, except in the treatment with pasteurized milk and no LAB addition. The Huang-Cardinal [pH] model was more accurate in modeling *L. monocytogenes* kinetics as a function of pH changes than was the Jameson-effect model with $N_{max\ tot}$ as a function of LAB inhibitory effect based on the goodness-of-fit measures. The Jameson-effect model may however be a better competition model since it can more easily represent *L. monocytogenes* growth and death. This study presents crucial kinetic data on *L. monocytogenes* behavior in the presence of competing microbiota in Minas semi-hard cheese under dynamic conditions.

1. Introduction

Minas artisanal cheeses are traditionally made on family farms located in Minas Gerais state, Brazil, using commercial rennet and raw cow's milk and autochthonous lactic acid bacteria (LAB), commonly known as "pingo", derived from the whey recovered from a previous batch of cheese) (Kamimura et al., 2019a). These cheeses are highly important from the social and economic aspects, as these cheeses are made by more than 30,000 small producers with an annual production of 50,000 tons (MilkPoint, 2017). Minas artisanal cheeses are classically ripened for 14–22 days (Borelli et al., 2006; Kamimura et al., 2019a), with differences in the making procedures being described within farmers located in the same production region as well as amongst the 7 producing regions catalogued (Kamimura et al., 2019a). These differences are mainly related to the coagulation time, starter cultures, pressing conditions, as well as salt and moisture content in the final product (Borelli et al., 2006; Kamimura et al., 2019a). These differences in making conditions are known to impact on the microbiota present in

* Corresponding author. Rua Monteiro Lobato, 80, Cidade Universitária, CEP 13083-862, Campinas, SP, Brazil.
E-mail address: and@unicamp.br (A.S. Sant'Ana).
[1] These authors contributed equally to this study.

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the artisanal Minas cheeses (Kamimura et al., 2019b; Sant'Anna et al., 2019), while the safety aspects frequently demand the assessment of the fate of foodborne pathogens in the product.

Cheeses represent a category of foods in which *Listeria monocytogenes* is a concern. Because of that, cheeses have been submitted to inspection by food safety agencies throughout the world. For instance, 0.9% of semi-soft and soft cheeses made with low-heat-treated or raw milk were contaminated with *L. monocytogenes* according to the European Rapid Alert System for Food and Feed (RASFF) (EFSA and ECDC – European Food Safety Authority and European Centre for Disease Prevention and Control, 2018). Even though data on *L. monocytogenes* occurrence in semi-hard artisanal Minas cheese are scarce, this bacterium has been found in soft and semi-soft cheeses made in Brazil with prevalence ranging from 1.4 to 6.0% (Raimundo, 2013; Silva et al., 1998; Souza, 2006). Although listeriosis outbreaks have been more commonly linked with fresh and soft cheeses (Martínez-Rios and Dalgaard, 2018), a quantitative microbial risk assessment predicted 0.6 and 26 annual mean illnesses arising per 10,000 individuals by healthy and vulnerable (immunocompromised patients, pregnant women, newborns and elderly) populations, respectively, as a result of artisanal semi-hard Minas cheese consumption (Campagnollo et al., 2018a). Besides, the study revealed that the addition of LAB with anti-listerial properties reduced the risks of listeriosis due to the consumption of the cheeses (Campagnollo et al., 2018a). Therefore, these findings suggest that assessing the behavior of *L. monocytogenes* throughout cheese manufacturing steps and ripening/storage (Gérard et al., 2018) comprise key information for risk management. This characterization is necessary considering that the changes in cheese intrinsic parameters during processing and ripening/storage will likely impact on the fate of *L. monocytogenes*. Among these factors, microbial competition seems to be highly relevant in foods that contain indigenous microbiota with known antimicrobial activity (Orihuel et al., 2018; Cavicchioli et al., 2019; Miranda et al., 2018). Therefore, the use of these microorganisms with antimicrobial properties as strategy to improve the safety of cheeses seems feasible and promising. For instance, the addition of a cocktail of LAB strains with anti-listerial activity to semi-hard Minas cheese resulted in a significant decline of *L. monocytogenes* populations during the cheese ripening. On the other hand, *L. monocytogenes* still survived in semi-hard Minas cheese in which the cocktail of LAB was not added (Campagnollo et al., 2018b). Besides, the effectiveness of the cocktail of LAB in inhibiting *L. monocytogenes* varied with the type of cheese studied. While more than 4 log-reduction of this bacterium was observed in semi-hard Minas cheeses, in fresh Minas cheese a bacteriostatic effect was observed (Campagnollo et al., 2018b). Therefore, for the use of findings towards refining risk assessment studies, predictive models must be generated that incorporate *L. monocytogenes* dynamics throughout the process as affected also by microbial competition.

Dynamic predictive models considering the influence of temperature, pH and water activity (a_w) have been used to describe the behavior of *L. monocytogenes* in semi-hard cheese (Schvartzman et al., 2011; Tiwari et al., 2014). On the other hand, the inhibitory effect of cheese microbiota on *L. monocytogenes* growth has been assessed on wooden shelves during ripening of a smear cheese, and over refrigerated storage of cottage cheese by means of microbial competition models (Guillier et al., 2008; Østergaard et al., 2014). Recently, a dynamic approach has been employed to quantify the impact of pH, temperature, a_w and microbial interaction towards the growth of *L. monocytogenes* in Minas soft fresh cheese (Cadavez et al., 2019). In that study, likely due to the high moisture of Minas soft fresh cheese, *L. monocytogenes* was able to grow (Cadavez et al., 2019). The present study comprises a continued effort toward understanding the behavior of *L. monocytogenes* in Minas cheeses, specifically in artisanal Minas semi-hard cheeses, in which both growth and inactivation of this bacterium were observed. The objectives of the present study were (i) to mathematically determine the impact of type of milk (raw versus pasteurized) and type of LAB (indigenous versus intentionally-added) on the evolution of *L. monocytogenes* and LAB during ripening of semi-hard Minas artisanal cheeses; and (ii) to contrast the capabilities of a dynamic model (Huang-cardinal) and a competition model (Jameson effect) to describe such kinetics.

2. Material and methods

2.1. LAB and L. monocytogenes strains and cell suspensions preparation

A total of six strains of LAB (four *Enterococcus faecalis* and one *Lactobacillus brevis* and one *Lactobacillus plantarum*) isolated by (Campagnollo et al., 2018b) from Minas artisanal cheese and that presented anti-listerial activity as a pool were used in this study. Before cheese formulation, preparation of LAB strains cell suspensions included cultivation in MRS broth (de Man, Rogosa and Sharpe – Acumedia, Neogen Corporation, Lansing/MI) at 30ºC for 24 h for two successive inoculations. Then, next inoculation was performed in 90 mL of MRS broth with cell concentration determined in a McFarland turbidimeter (MS Tecnopon, Piracicaba/SP/Brazil) with a 1 reading correlated to 3×10^8 CFU/mL.

The two L. *monocytogenes* strains used in this study have been obtained from the Oswaldo Cruz Foundation (Rio de Janeiro/RJ/Brazil). Isolation sources included cheese for *L. monocytogenes* strain 3968 (serotype 1/2 b) and raw milk for *L. monocytogenes* strain 3973 (serotype 4 b). The preparation of cell suspensions (10^8 CFU/mL) of each strain was carried out separately as previously detailed (Sant'Ana et al., 2012) using TSBYE (Tryptic soy broth – Merck, Darmstadt, Germany, supplemented with 0.6% Yeast Extract – Acumedia, Neogen Corporation, Lansing/MI).

2.2. Semi-hard Minas artisanal cheese production

The laboratory-scale preparation of semi-hard Minas artisanal cheese was conducted as detailed in Campagnollo et al. (2018b). A total of 4 treatments were carried out in duplicate, on distinct days. Cheeses were produced using pasteurized or raw milk, with addition or not of anti-listerial LAB (10^{6-7} CFU/mL of milk) co-inoculated with *L. monocytogenes* (10^{5-6} CFU/mL of milk).

Cheese production started by heating 10 L of milk to 34 ± 1 ˚C, followed by the addition of 5 mL of $CaCl_2$ (saturated solution), rennet (9 mL, 85% bovine pepsin +15% bovine chymosin – Estrella, Chr. Hansen, Valinhos/SP/Brazil) and the prepared suspensions of *L. monocytogenes* and/or LAB, depending on the treatment. The curds coagulated for 40 min, after which they were cut (about 1 cm^2 squares), slightly agitated and rest for 30 min at room temperature (22 ± 1 ˚C). The curd was spread into holed sterilized cylinder-shaped forms (7 cm diameter × 10 cm height) after the draining off the whey. The whey was drained through the existing perforations in the form, with a light pressure using weights of about 100 g. After 1 h at room temperature, cheeses were rotated and pressed for an extra 1 h. Cheeses were surface salted, placed onto woody racks to ripen for 22 days at a controlled room temperature of 22 ± 1 ˚C. During the ripening period, the cheeses were revolved every day (Campagnollo et al., 2018b).

2.3. Physicochemical and microbial analysis throughout semi-hard Minas artisanal cheese ripening

LAB were enumerated according to Njongmeta et al. (2015) on MRS agar (Acumedia, Neogen Corporation, Lansing/MI) overlaid with 1.5% bacteriological agar and incubation at 30 ºC for 48 h. On the other hand, *L. monocytogenes* cells were enumerated following the methodology of Ryser and Donnelly (2015) on *Listeria* Selective agar (Oxford Formulation – Oxoid, Basingstoke, UK) and aerobically incubated at 37 ºC for 24 h. All analyses were performed in duplicate. The counts of LAB and *L. monocytogenes* were performed on days 0 (just after cheesemaking and then at 4, 8, 12, 16 and 20 h), 2, 3, 4, 8, 12, 16, 19 and 22

Table 1
Kinetic parameters (initial and maximum microbial concentration, Y_0, Y_{max} in ln CFU/g, specific growth rate, μ_{opt}, in h^{-1}, and inactivation rate, k, in h^{-1}) of *L. monocytogenes* and lactic acid bacteria (LAB) in semi-hard artisanal Minas cheese made from raw or pasteurized milk and with or without the addition anti-listerial LAB (LAB_{LM}), as estimated by the Huang model or the linear decay model, along with goodness-of-fit measures (residuals, σ^2, root mean square error, RMSE, and mean absolute error, MAE).

Treatments	Parameters	*L. monocytogenes*		LAB	
		Mean (SE)	Pr > |t|	Mean (SE)	Pr > |t|
Pasteurized milk + No LAB_{LM}	Y_0	14.58 (0.373)	< .0001	9.102 (0.872)	< .0001
	μ_{max}	0.178 (0.039)	< .0001	0.303 (0.073)	0.002
	Y_{max}	18.26 (0.017)	< .0001	18.00 (0.425)	< .0001
	Fit quality				
	σ^2	0.1990		1.2189	
	RMSE	0.4298		1.0638	
	MAE	0.3669		0.8877	
Pasteurized milk + LAB_{LM}	Y_0	13.27 (0.304)	< .0001	16.91 (0.355)	< .0001
	μ_{max}/k	−0.0182 (0.001)	< .0001	0.229 (0.030)	< .0001
	Y_{max}	–	–	23.84 (0.172)	< .0001
	Fit quality				
	σ^2	0.6980		0.2006	
	RMSE	0.8051		0.4316	
	MAE	0.5607		0.3028	
Raw milk + No LAB_{LM}	Y_0	14.30 (0.381)	< .0001	13.19 (0.489)	< .0001
	μ_{max}	0.253 (0.118)	< .0001	0.331 (0.053)	< .0001
	Y_{max}	16.01 (0.134)	< .0001	19.34 (0.220)	< .0001
	Fit quality				
	σ^2	0.1408		0.3353	
	RMSE	0.3616		0.5580	
	MAE	0.2312		0.4964	
Raw milk + LAB_{LM}	Y_0	12.38 (0.339)	< .0001	18.15 (0.275)	< .0001
	μ_{max}/k	−0.0260 (0.001)	< .0001	0.063 (0.008)	< .0001
	Y_{max}	–	–	24.18 (0.251)	< .0001
	Fit quality				
	σ^2	0.8662		0.2729	
	RMSE	0.8968		0.5034	
	MAE	0.7722		0.4195	

(once a day).

Non-*L. monocytogenes* inoculated cheeses were subjected to physicochemical analysis including pH and a_w, moreover temperatures were recorded. For a_w determination, Aqualab a_w meter (model 4 TE, Decagon Devices Inc., São José dos Campos/SP/Brazil) was used, while pH and temperature were recorded using a transportable pH meter combined with a knife electrode and a temperature device (AK103 pH meter, SC18 electrode, Akso Electronic Products Ltda., São Leopoldo/RS/Brazil), respectively.

2.4. Modeling of *L. monocytogenes* inhibition during semi-hard Minas artisanal cheese ripening

2.4.1. Independent growth/decay of LAB and *L. monocytogenes*

Each of the experimental growth curves of *L. monocytogenes* and LAB were modeled by adjusting the integrated form of the Huang primary model (Eq. (1)) proposed for a constant temperature storage condition (Huang, 2008):

$$Y(t) = Y_0 + Y_{max} - ln\{\exp(Y_0) + (\exp(Y_{max}) - \exp(Y_0)) \times \exp(-\mu_{max} B(t))\}$$
$$B(t) = t + \frac{1}{\alpha} ln \frac{1+\exp(-\alpha(t-\lambda))}{1+\exp(\alpha\lambda)} \quad (1)$$

where: Y_0, Y_{max} and Y comprise the natural logarithms of microbial concentrations at initial time (=0), maximum population and actual time t; μ_{max} accounts for maximum specific rate of growth (h^{-1}); while λ is the delay interval (lag time) of a curve depicting microbial behavior through time (h); α is the coefficient that accounts for the lag phase shift (set to 4, as Huang (2013) recommended); and t is the time interval at a steady temperature (22 ± 1 °C). Since none of the curves showed a lag, λ value was set to zero.

For the treatments in which a decline in *L. monocytogenes* population was observed (i.e., cheeses with starter culture), the experimental curves were modeled by a simple log-linear decay model (Eq. (2)):

$$Y(t) = Y_0 - kt \quad (2)$$

where k was the specific inactivation rate (h^{-1}) of *L. monocytogenes*.

2.4.2. Growth of *L. monocytogenes* during semi-hard Minas artisanal cheese ripening

Since cheese pH changes during ripening at 22 °C, dynamic kinetic analysis has been employed to find out *L. monocytogenes* kinetic parameters in cheese. The differential form of the Huang primary growth model (Eq. (1)) with the cardinal parameter model of Rosso et al. (1995) has been concurrently fitted as a secondary model of the specific growth rate according to cheese pH. The dynamic tertiary model shown in Eq. (3) was adjusted to the experimental data in which *L. monocytogenes* population increased in time (i.e., treatments without addition of starter cultures).

$$\frac{dY}{dt} = \frac{\mu_{max}}{1+e^{-\alpha(t-\lambda)}}(1 - e^{Y-Y_{max}})$$
$$\mu_{max} = \mu_{opt}\left\{\frac{(pH-pH_{min})(pH-pH_{max})}{(pH-pH_{min})(pH-pH_{max})-(pH-pH_{opt})^2}\right\} \quad (3)$$

where μ_{opt} stands for optimum growth rate (h^{-1}).

Equation (3) was not adjusted for the treatments with addition of starter cultures, because it cannot mathematically represent decline in microbial population. In this work, this tertiary model will be called the Huang-Cardinal [pH] model (Cadavez et al., 2019). The parameters pH_{min} and pH_{max} represent limits of pHs in which no growth will be observed, while in the pH_{opt} the optimal μ_{max} is recorded. The parameter μ_{opt} can be inferred as the optimal *L. monocytogenes* growth rate in semi-hard Minas artisanal cheese at the pH_{opt}. *L. monocytogenes* cardinal parameters (pH_{min}, pH_{opt} and pH_{max}) are not estimated from the data (since the observed pH values of the Minas semi-hard cheese

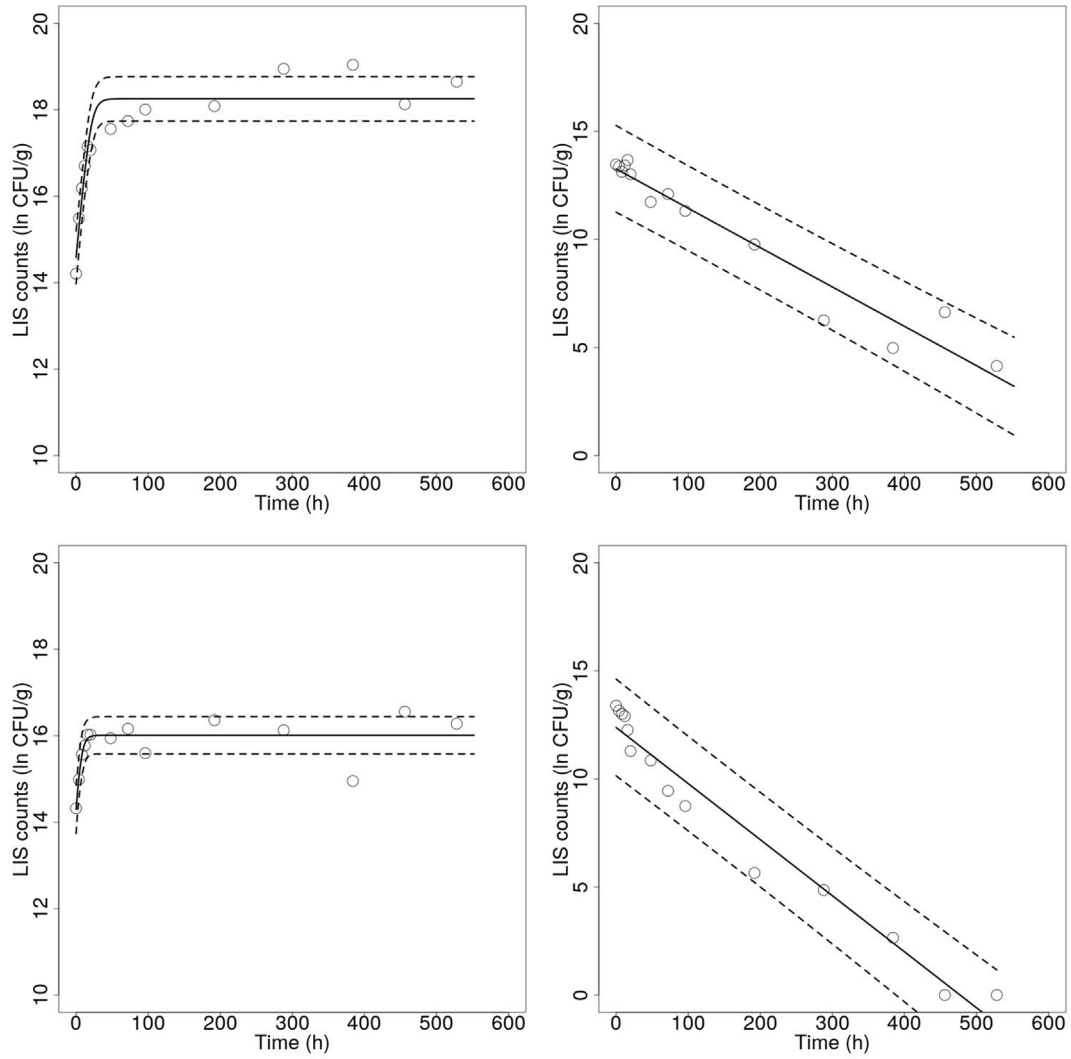

Fig. 1. Fate of L. *monocytogenes* (LIS) in semi-hard artisanal Minas cheese made from pasteurized milk without addition of anti-listerial lactic acid bacteria (LAB_{LM}, top left), pasteurized milk with LAB_{LM} (top right), raw milk without LAB_{LM} (bottom left) and raw milk with LAB_{LM} (bottom right), as depicted by the Huang or the linear decay models, with 95% confidence bands.

represent a limited and suboptimal range) but were set to the meta-analytical estimates by Nunes Silva et al. (2019) in liquid culture medium: pH_{min} = 4.298; pH_{opt} = 7.090; and pH_{max} = 9.510.

2.4.3. Simultaneous modeling of LAB and L. monocytogenes

The Jameson-effect model fitted to the concomitant growth data of LAB and *L. monocytogenes* was an adaptation of Cornu (2001) as described by Cadavez et al. (2019). The Jameson-effect model fitted considered instead a total maximum cell density $N_{max\ tot}$, modifying the logistic deceleration function (Eq. (4)).

$$\frac{1}{LM}\frac{dLM}{dt} = \mu_{LM}\left(1 - \frac{LM + \gamma LAB}{N_{max\,tot}}\right)$$
$$\frac{1}{LAB}\frac{dLAB}{dt} = \mu_{LAB}\left(1 - \frac{LAB}{N_{max\,tot}}\right) \quad (4)$$

where LAB and *L. monocytogenes* counts through time are described as LAB and LM (CFU/g), and the LAB and *L. monocytogenes* maximum specific growth rates are described by μ_{LAB} and μ_{LM} (h^{-1}), respectively. The parameter γ accounts for interaction and it enables the counts of *L. monocytogenes* to raise ($\gamma < 1$) or drop ($\gamma > 1$) subsequently to a maximum density of LAB has been reached. When LAB promptly inhibits *L. monocytogenes* and causes its inactivation (i.e., a negative μ_{LM}), $\gamma > 1$ implies that, once LAB reached their maximum concentration, the remaining pathogen population becomes less sensitive to the inhibitory effect of LAB. Equation (4) is named as the Jameson-effect model with $N_{max\ tot}$, and was fitted to the experimental curves produced by each of the four treatments. Even though the Jameson-effect model was originally created to quantify the behavior of two populations in co-culture based on their growth parameters determined in mono-culture, here this model was not used in that way (Cadavez et al., 2019). Instead an inverse analysis of the Jameson-effect model was performed and the kinetic parameters of LAB and *L. monocytogenes* populations were estimated from the trial growth curves gathered in co-culture (Cadavez et al., 2019).

2.4.4. Estimation of parameters

Numerical methods can be used to determine ordinary differential equations (ODE) such as Equations (3) and (4). Numerical optimization searches for the model parameters resulting in least residual sum of squares (RSS) of the errors. Herein, the approach of Huang (2012) has been followed that used 4th order Runge-Kutta method to resolve ODE, where the unknown kinetic parameters were determined by least-square optimization, employing the 'deSolve' and 'FME' libraries from the R software. In addition, the R 'nlme' library has been employed to estimate the kinetic parameters of Equations (1) and (2). The mean absolute error (MAE) and the root mean square error (RMSE) were also

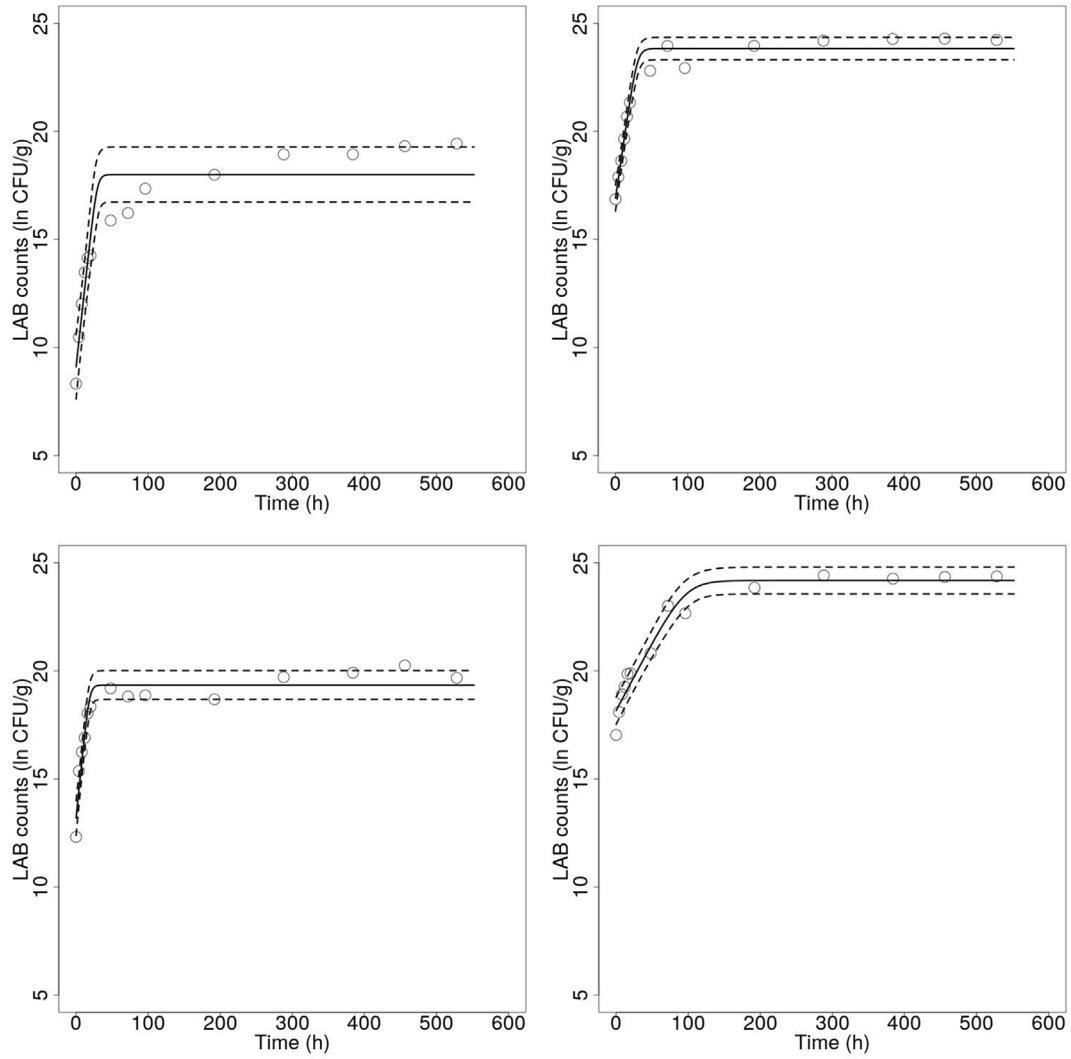

Fig. 2. Fate of lactic acid bacteria (LAB) in semi-hard artisanal Minas cheese made from pasteurized milk without addition of anti-listerial LAB (LAB_{LM}, top left), pasteurized milk with LAB_{LM} (top right), raw milk without LAB_{LM} (bottom left) and raw milk with LAB_{LM} (bottom right), as depicted by the Huang primary model, with 95% confidence bands.

computed to assess the fitting capacities of the Jameson–effect and Huang-Cardinal [pH] models:

$$MAE = \frac{\sum |Y_{obs\ i} - Y_{fit\ i}|}{n}$$
$$RMSE = \sqrt{\frac{\sum (Y_{obs\ i} - Y_{fit\ i})^2}{df}} \quad (5)$$

where $Y_{fit\ i}$ and $Y_{obs\ i}$ designate for each of the *i*-th *L. monocytogenes* concentrations adjusted by the dynamic/competition model and its corresponding observation, respectively. The degrees of freedom (df) is determined as 'n-np', where n is the amount of data points of a trial growth curve and np is the quantity of parameters of the adjusted model. When concurrent modeling of LAB and *L. monocytogenes* are performed (as through the Jameson-effect model), the residuals from the counts of LAB were not deemed to be comparable to the residuals of the model Huang-Cardinal [pH], because in this case only *L. monocytogenes* counts were considered.

3. Results

The parameter estimates of the Huang primary model or the log-linear decay model describing the independent growth/decay of *L. monocytogenes* and LAB during semi-hard Minas artisanal cheese ripening are showed in Table 1. Inactivation of L. *monocytogenes* occurred in both cheeses in which the LAB strains with anti-listerial properties were added, however in the cheeses produced with raw milk, *L. monocytogenes* presented a faster inactivation rate ($-0.0260\ h^{-1}$) than that observed in the cheeses produced with pasteurized milk ($-0.0182\ h^{-1}$). The cheeses made with raw milk and containing anti-listerial LAB presented *L. monocytogenes* counts below the enumeration limit of the method (100 CFU/g) after 456 h (19 days) of ripening (Fig. 1, bottom right). The growth of *L. monocytogenes* was higher in the cheese made with pasteurized milk and with no addition of the anti-listerial LAB than in the cheese made with raw milk and without anti-listerial LAB. For instance, the maximum population of the pathogen was 18.26 ln CFU/g vs. 16.22 ln CFU/g in pasteurized and raw milk, respectively, as shown in Fig. 1 (upper and lower left). Better goodness-of-fit quality was achieved for the cheeses without added LAB modeled by the Huang primary model (Table 1).

LAB counts during cheese ripening were higher when anti-listerial LAB was added for either milk type (Table 1), but LAB growth rates were less compared to treatments without LAB. The specific growth rate for LAB in raw milk cheese added of LAB was very low compared to other treatments (0.063 ln CFU/g h^{-1} vs. 0.229–0.331 ln CFU/g h^{-1}, respectively). There could be two reasons for this: because of the high initial LAB population (18.2 ln CFU/g vs. 9.1–16.9 ln CFU/g,

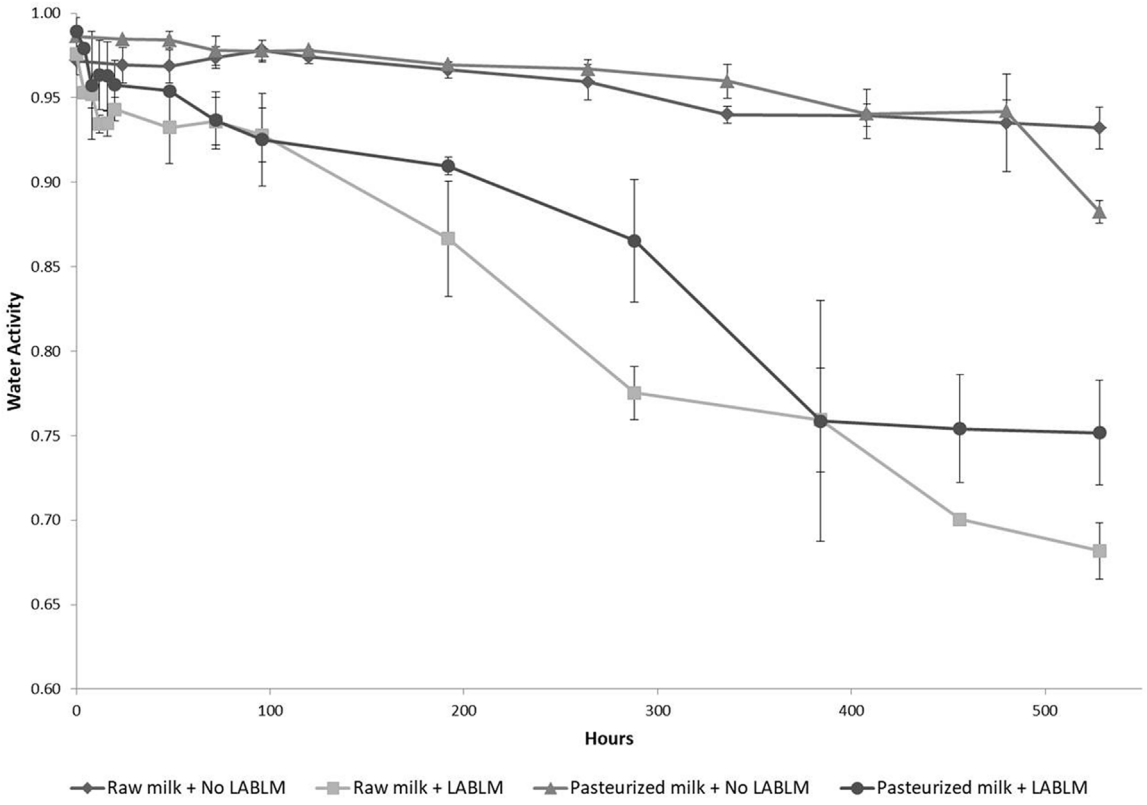

Fig. 3. Change in water activity during ripening of semi-hard artisanal Minas cheese. Treatments included the production of cheese with raw or pasteurized milk, and with addition or without anti-listerial lactic acid bacteria (LAB_{LM}).

respectively), due to the native LAB naturally existing in raw milk and added anti-listerial LAB, probably causing intra-species competition; and, most likely, because of the faster decrease in a_w observed in raw milk cheeses with added LAB. Notice in Fig. 3 that samples from this treatment showed the fastest drop in a_w among all treatments, and remained the lowest throughout the experiment. Growth of LAB would then become increasingly suppressed as the minimum water activity for growth is reached, which has been estimated to be around 0.928 for *Lactobacillus curvatus* (Mejlholm and Dalgaard, 2007) and 0.930 for *Lactococcus lactis* (Troller and Stinson, 1981). All models presented visually a good adjustment (Fig. 2), but to a lesser degree for the treatment made with pasteurized milk and no added LAB (Fig. 2, upper left), suggesting that low levels of autochthonous LAB (reduced due to pasteurization) as well as other unmeasured organisms was associated with greater dispersion (see values of σ^2 in Table 1).

The a_w changes of semi-hard Minas artisanal cheese (non-inoculated with *L. monocytogenes*) during ripening are represented in Fig. 3. The a_w was lower in treatments with added LAB (0.68 and 0.75 for raw milk cheese and pasteurized milk cheese, respectively) compared to treatments with no added LAB (0.93 and 0.88, respectively). The lowest a_w values found in treatments with added LAB is correlated with higher *L. monocytogenes* inactivation in these cheeses.

Change in pH throughout ripening of semi-hard Minas artisanal cheese is shown in Fig. 4. The pH in cheeses with anti-listerial LAB addition dropped faster (from 6.4 to 6.5 to 4.9–5.1) than in cheeses without added LAB (from 6.5 to 6.8 to 5.5). The kinetic parameters for *L. monocytogenes* increase during ripening determined dynamically by fitting the Huang primary growth model are described in Table 2. *L. monocytogenes* optimum growth rate in cheese produced with raw milk (0.346 h^{-1}) was higher than that of cheese produced with pasteurized milk (0.198 h^{-1}); however, *L. monocytogenes* final concentration was greater in pasteurized milk cheese.

The results from fitting the Jameson-effect model with $N_{max\ tot}$ for the simultaneous growth of LAB and *L. monocytogenes* is presented in Table 2. The presence of LAB inhibited *L. monocytogenes* growth in all cheeses, except for cheese made with pasteurized milk and containing no anti-listerial LAB. In the cheese made with pasteurized milk containing no anti-listerial LAB, the interaction parameter γ, was < 1 and not statistically significant ($P = 0.589$), which means that LAB had no significant inhibitory effect on pathogen growth, as both populations reached their respective maximum (Fig. 5, top left). Cheeses produced with added LAB had the same magnitude of *L. monocytogenes* inhibition and the values found for pathogen specific growth rate (μ_{LM}, -0.046 h^{-1} and -0.048 h^{-1} for pasteurized milk cheese and raw milk cheese, respectively) and for the interaction parameters (ln γ, 0.632 and 0.661, respectively) were also similar. The lowermost goodness-of-fit quality values were found for the cheeses produced with pasteurized milk and added LAB, and the other treatments presented comparable residuals values (σ^2), mean absolute error (MAE) and root mean square error (RMSE) as seen in Table 2.

Looking at the goodness-of-fit quality values (Table 2) and at the ease of parameter optimization; the Huang-Cardinal [pH] model was more precise to model *L. monocytogenes* kinetics as impacted by changing pH than did the Jameson-effect model with $N_{max\ tot}$ as affected by the LAB inhibition influence. Since the decrease in fitting quality attained by the Jameson-effect model was minimal, the Jameson-effect model may be more preferred in some situations since it can be capable of modeling both the growth and death of *L. monocytogenes* in these cheeses which the Huang-Cardinal [pH] model cannot do.

4. Discussion

It is known that the most relevant *L. monocytogenes* growth inhibiting factors in cheeses are temperature, a_w, pH and lactic acid concentration (Wemmenhove et al., 2018). While there is a need for encompassing all these factors in growth models, it is also of primordial

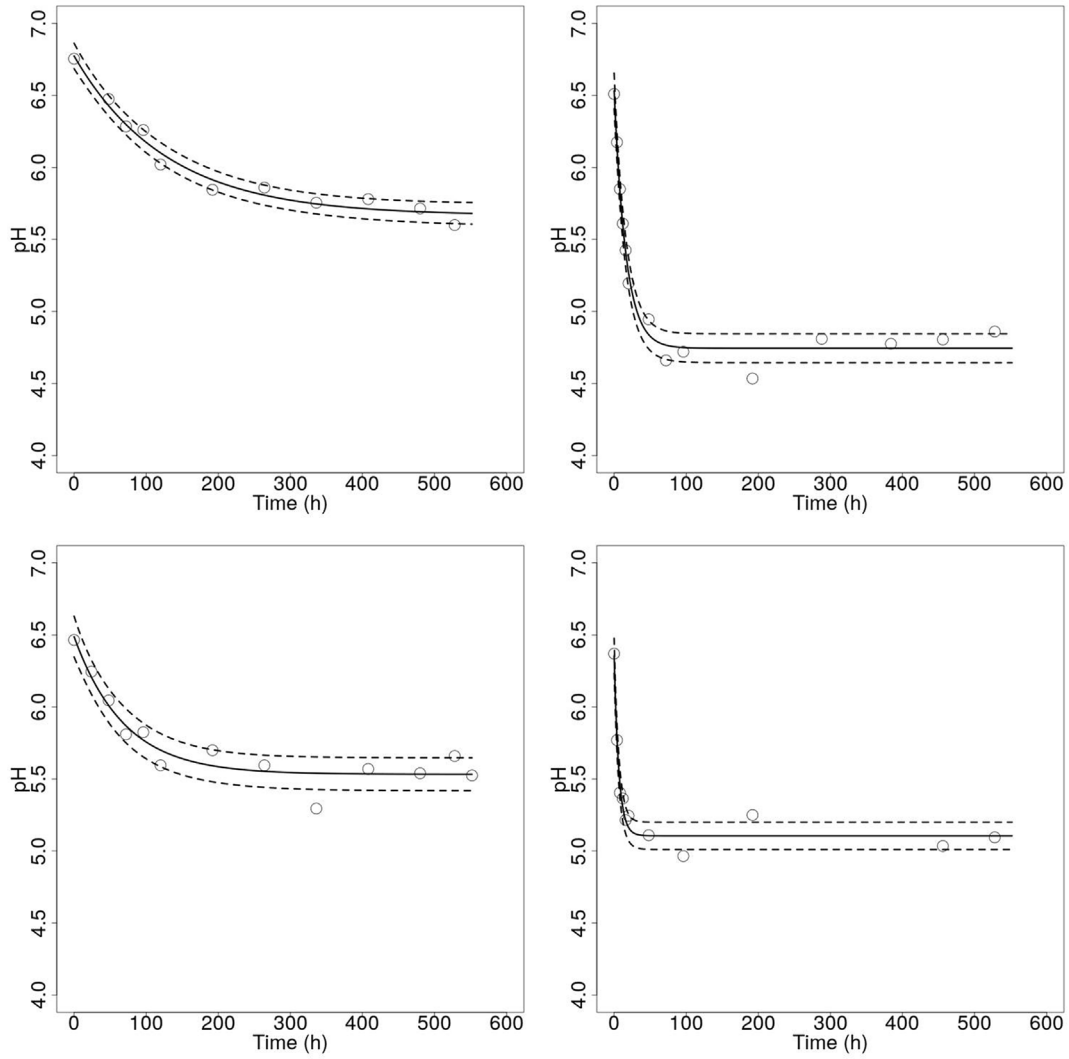

Fig. 4. Change in pH of semi-hard artisanal Minas cheese made from pasteurized milk without addition of anti-listerial lactic acid bacteria (LAB_{LM}, top left), pasteurized milk with LAB_{LM} (top right), raw milk without LAB_{LM} (bottom left) and raw milk with LAB_{LM} (bottom right). A two-parameter empirical decay function [$pH_t = pH_0.exp(kt)$], with 95% confidence bands, was added only for trend visualization.

importance to extend the existing predictive models for *L. monocytogenes* growth/decay on semi-hard cheeses since the variability of intrinsic parameters makes the pathogen response variable (Kapetanakou et al., 2017). Therefore, this study contributes to the field by reporting on the dynamic influence of cheese intrinsic parameters (Campagnollo et al., 2018b; Pinto et al., 2009) and expanding a previous application of the Huang-Cardinal [pH] model (Cadavez et al., 2019) also for semi-hard Minas artisanal cheese. Few studies have proposed dynamic tertiary models for other cheeses. Schvartzman et al. (2011) obtained an acceptable fit quality when they analyzed the behavior of *L. monocytogenes* in pasteurized and raw milk smeared cheese using a logistic primary growth model with a secondary cardinal growth model using parameters of temperature, pH, a_w and undissociated lactic acid. Rosshaug et al. (2012) developed a tertiary model for soft blue-white cheese that analyzed the interaction of multiple factors (temperature, pH, NaCl, and lactic acid) on the maximum specific growth rate using a combined cardinal parameter model. Lobacz et al. (2013) used a Baranyi primary model and a polynomial secondary model to predict the impact of temperature on growth of *L. monocytogenes* in Camembert and bleu cheeses. Tirloni et al. (2019) successfully developed and validated two different cardinal parameter models for *L. monocytogenes* growth in ricotta considering temperature alone, and temperature and pH with interaction. These authors also recalibrated an existing growth model which includes the effect of organic acids for use in ricotta cheese. Only two other studies have also determined the influence of LAB on *L. monocytogenes* behavior in cheeses. Guillier et al. (2008) reported that *L. monocytogenes* growth ceased at the same time that the microflora of a natural biofilm formed on wooden shelves used in the ripening of a soft and smear cheese entered in the stationary phase, i.e. the "Jameson-effect" (Jameson, 1962). Østergaard et al. (2014) predicted simultaneous growth of LAB and *L. monocytogenes* in cottage cheese considering the Jameson-effect as well as temperature, pH, NaCl, lactic- and sorbic acids and their interactions.

The Huang (2008) primary model was able to accurately model *L. monocytogenes* and LAB growth in semi-hard Minas artisanal cheese (MAE: 0.3424–0.3547 for the Huang-cardinal model versus 0.2364–0.2837 for the Jameson model). *L. monocytogenes* could slightly grow in the cheeses in which the anti-listerial LAB were not added, and this growth was greater in pasteurized milk cheese. In fact, it is known that cheeses made with pasteurized milk can favor the survival of *L. monocytogenes*, since these cheeses have lower counts of background microbiota, more specifically of LAB (Gérard et al., 2018). The specific growth rate for LAB in raw milk cheese added of LAB was very low compared to other treatments (0.063 h^{-1} vs. 0.229–0.331 h^{-1}, respectively), probably because of the high initial LAB population (18.2 ln

Table 2
Kinetic parameters (initial and maximum microbial concentrations LAB_0, LM_0, LAB_{max} and LM_{max} [ln CFU/g], specific growth rates of LAB and *L. monocytogenes*, μ_{LAB} and μ_{LM}, and optimum growth rate of LM, μ_{opt} [h^{-1}] in semi-hard artisanal Minas cheese made from raw or pasteurized milk and with or without the addition of anti-listerial LAB (LAB_{LM}), as estimated by the Jameson-effect model with $N_{max\ tot}$ and the dynamic Huang-Cardinal [pH] model. Goodness-of-fit measures (residuals σ^2, root mean square error RMSE and mean absolute error MAE) were computed for *L. monocytogenes* counts only to allow comparison.

Treatments	Jameson effect with $N_{max\ tot}$			Huang-Cardinal[pH]		
	Parameters	Mean (SE)	Pr > |t|	Parameters	Mean (SE)	Pr > |t|
Pasteurized milk + No LAB_{LM}	LAB_0	9.105 (0.674)	< .0001	LM_0	14.54 (0.347)	< .0001
	LM_0	14.53 (0.744)	< .0001	μ_{opt}	0.198 (0.042)	0.002
	N_{max}	17.98 (0.057)	< .0001	LM_{max}	18.03 (0.197)	< .0001
	μ_{LAB}	0.303 (0.058)	< .0001			
	μ_{LM}	0.194 (0.094)	0.050			
	$\ln(\gamma)$	−0.332 (0.607)	0.589			
	Fit quality			Fit quality		
	σ^2	0.1917		σ^2	0.1625	
	RMSE	0.4221		RMSE	0.3780	
	MAE	0.3547		MAE	0.2837	
Pasteurized milk + LAB_{LM}	LAB_0	16.95 (0.509)	< .0001			
	LM_0	13.73 (0.422)	< .0001			
	N_{max}	23.85 (0.248)	< .0001		ND	
	μ_{LAB}	0.223 (0.043)	< .0001			
	μ_{LM}	−0.046 (0.021)	0.041			
	$\ln(\gamma)$	0.632 (0.188)	0.003			
	Fit quality					
	σ^2	0.6292				
	RMSE	0.7644				
	MAE	0.5555				
Raw milk + No LAB_{LM}	LAB_0	13.44 (0.750)	< .0001	LM_0	14.28 (0.386)	< .0001
	LM_0	14.01 (0.698)	< .0001	μ_{opt}	0.346 (0.152)	0.044
	N_{max}	18.31 (0.310)	< .0001	LM_{max}	16.02 (0.137)	< .0001
	μ_{LAB}	0.391 (0.134)	0.008			
	μ_{LM}	0.140 (0.077)	0.084			
	$\ln(\gamma)$	0.877 (0.065)	< .0001			
	Fit quality			Fit quality		
	σ^2	0.2174		σ^2	0.1625	
	RMSE	0.4556		RMSE	0.3622	
	MAE	0.3424		MAE	0.2364	
Raw milk + LAB_{LM}	LAB_0	18.93 (0.260)	< .0001			
	LM_0	13.18 (0.327)	< .0001			
	N_{max}	24.48 (0.347)	< .0001		ND	
	μ_{LAB}	0.035 (0.002)	< .0001			
	μ_{LM}	−0.048 (0.006)	< .0001			
	$\ln(\gamma)$	0.661 (0.087)	< .0001			
	Fit quality					
	σ^2	0.1759				
	RMSE	0.4043				
	MAE	0.3143				

ND: Treatments not modeled since the tertiary Huang-Cardinal[pH] model is not meaningful for microbial decay.

CFU/g vs. 9.1–16.9 ln CFU/g, respectively), due to the native LAB naturally existing in raw milk and added anti-listerial LAB. For instance, *L. monocytogenes* survived during a 90 day-ripening period, with a maximum recovery of 6.8 log CFU/g cheese on day 30 when inoculated in semi-soft Trappist cheese manufactured from pasteurized milk (5.4 log CFU/ml milk) (Kovincic et al., 1990). Furthermore, most of cheese contaminants are not only associated to milk quality but to a sanitation deficiency during the post-pasteurization or post-processing steps (Kousta et al., 2010).

In fact, a recent study revealed that the major growth-limiting factors for *L. monocytogenes* in cheese can be the presence of undissociated lactic acid produced by LAB, the decreased a_w due to salt diffusion and water evaporation, and the limitation of or competition for nutrients (Kapetanakou et al., 2017). The a_w of cheeses made without anti-listerial LAB was higher (0.88–0.93) compared to the other treatments and close to the reported a_w limit of for *L. monocytogenes* growth (Melo et al., 2015). The a_w of cheeses made with anti-listerial LAB was lower likely due to the proteolysis caused by the enzymes released by viable LAB during ripening/storage. The enzymes break the casein networks present in cheese curd leading to an increased release of moisture from the cheese matrix, with clear impacts on cheese a_w. Besides, this phenomenon can be related to the low pH (~4.9) of the cheeses added with anti-listerial LAB as it is known that under these conditions casein networks can absorb but not hold moisture (Sousa et al., 2001; Pastorino et al., 2003) at ripening temperatures employed during the ripening of artisanal Minas semi-hard cheese. The a_w values achieved in ripening (0.68–0.75) favor pathogen inactivation, as it has been observed in other studies (Schvartzman et al., 2011).

The simplicity and a reasonably good fit were the main reasons why the log-linear decay model was chosen to describe *L. monocytogenes* inactivation (Peleg and Corradini, 2011). Many authors have used the log-linear model to estimate pathogen inactivation and survival data (Aguirre et al., 2015; Alvarenga et al., 2018; Coşansu, 2018; McKellar et al., 2014; Smith et al., 2016). Otherwise, Cadavez et al. (2019) explained that there is a critical difference between the growth rates obtained in the tertiary (e.g. the Huang-Cardinal [pH] model) vs. competition (e.g., the Jameson-effect model with $N_{max\ tot}$) models. While L. *monocytogenes* growth rate is a rate constant in the competition model and not linked to environmental factors, it is an optimum pathogen growth rate in the tertiary model and is based on an optimum pH. Any comparison between these models, their resulting growth rates and potential application for use should be done with care.

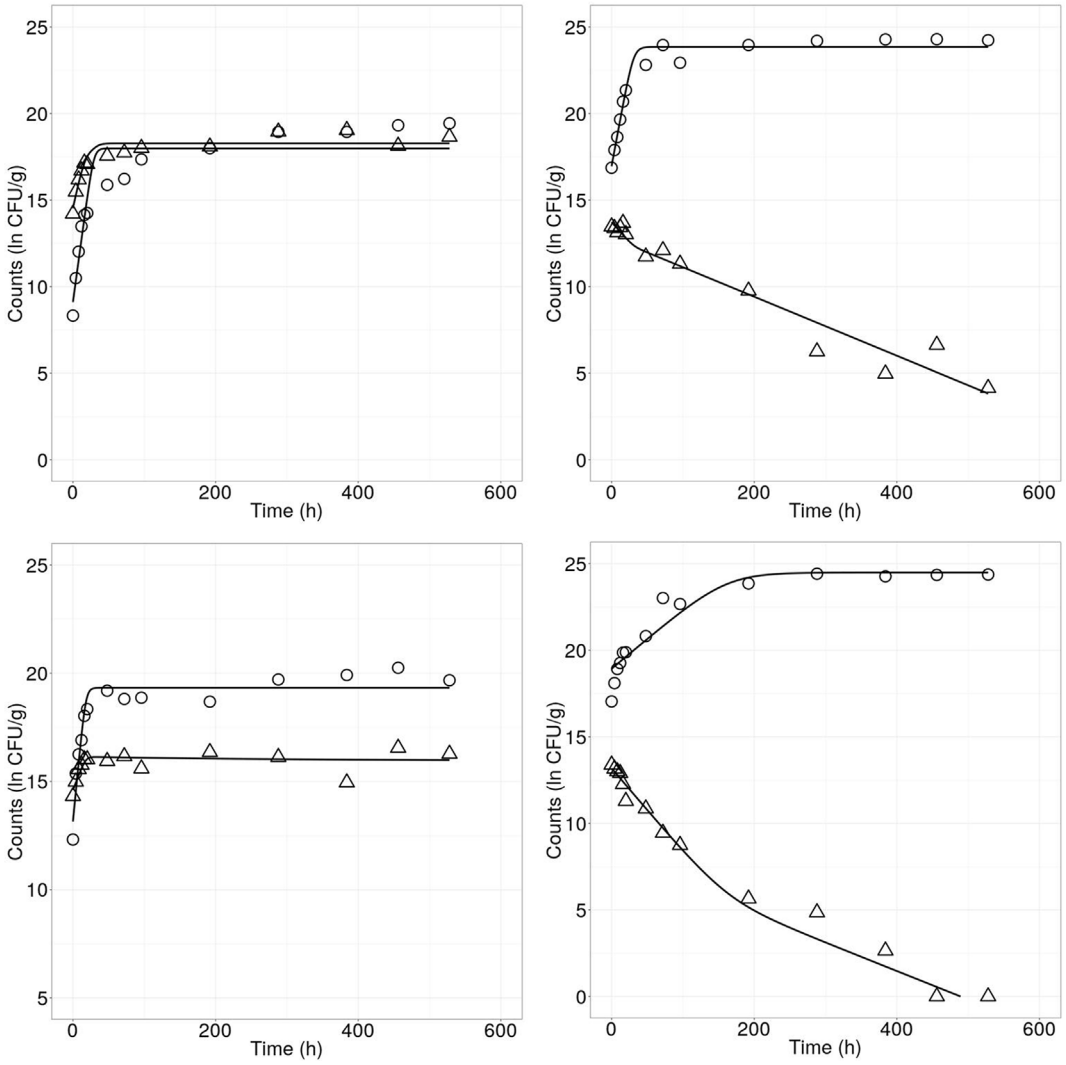

Fig. 5. Simultaneous growth of lactic acid bacteria (LAB) (-O-) and *L. monocytogenes* (-Δ-) in semi-hard artisanal Minas cheese elaborated from pasteurized milk without addition of anti-listerial LAB (LAB_{LM}, top left), pasteurized milk with LAB_{LM} (top right), raw milk without LAB_{LM} (bottom left) and raw milk with LAB_{LM} (bottom right), as depicted by the Jameson-effect model with $N_{max\ tot}$.

It is known that *L. monocytogenes* is able to grow in a wide pH range (4.6–9.5, Carpentier and Cerf, 2011), and the final pH values found in this study for semi-hard Minas cheese with added LAB (4.9–5.1) were close to the lower limit of this interval. In fact, this was expected because in this study the LAB added to cheese making were selected based on their ability to inhibit *L. monocytogenes* mainly considering their acidification power (Campagnollo et al., 2018b). Furthermore, the data suggest that not only the final pH is key in inhibiting the growth of *L. monocytogenes*, but also the rate at which the pH falls as it has been observed that an increase in LAB counts at the beginning of the ripening period associated with a reduction in *L. monocytogenes* counts in cheeses with added LAB. The data obtained here are support by the fact that the LAB added were chosen based on their capacity of inhibiting *L. monocytogenes* growth (Campagnollo et al., 2018b), the cheeses with no starter added had a much lower LAB concentration and by considering that the added LAB competed with *L. monocytogenes* for nutrients and produced metabolites such as lactic acid inhibiting their growth. LAB can grow rapidly in the first hours of ripening, producing organic acids which cause a 1.5–2.0 unit drop in pH (Gérard et al., 2018). Therefore, it becomes clear that LAB members with acidifying properties (and not only bacteriocinogenic strains) can play a key role in inhibiting *L. monocytogenes* during cheese ripening (Callon et al., 2011; Campagnollo et al., 2018b). These observations are consistent with a "hurdle strategy" combining biotic factors such as the microbial composition (milk endogenous bacteria and starter culture) and abiotic factors such as the environmental conditions during ripening (temperature and relative humidity) and cheese intrinsic parameters like pH, a_w, lactic acid and sodium chloride concentration.

5. Conclusion

In this work, three different approaches to describe the behavior of *L. monocytogenes* throughout ripening of semi-hard artisanal Minas cheese: (i) independent modeling of *L. monocytogenes* and LAB; (ii) dynamic modeling of *L. monocytogenes* using the Huang-Cardinal [pH] model; and (iii) simultaneous modeling of *L. monocytogenes* and LAB using a Jameson-effect model. The goodness-of-fit measures for the Huang-Cardinal [pH] model were better than the Jameson-effect model, but the Jameson-effect model could represent *L. monocytogenes* growth and death, while the Huang-Cardinal [pH] model could only model growth. The addition of anti-listerial LAB had a pronounced effect on pathogen growth, so the Jameson-effect model offers the advantage of quantitatively characterizing LAB inhibitory effect. All these models are valuable tools that can be used to help risk managers and cheese producers to predict *L. monocytogenes* concentration in semi-hard Minas artisanal cheese. Among other options, these predictive

models can be used in the refinement of quantitative risk assessment tools (particularly in exposure assessment), leading to more accurate outputs that can be used to safeguard public health.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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