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Impact of chlorinated stress on thermal characteristics of *Listeria monocytogenes*

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Abstract The results showed that the D-value significantly decreased with increasing temperature, which due to heat damaged cell. The increased temperature range resulted in *L. monocytogenes* being heat sensitive. NaOCl developed in the increased mortality of *L. monocytogenes* as the temperature increased, the D-value, Z-value and k-value of *L. monocytogenes* was markedly increased when compared to those of normal *L. monocytogenes* in both microbiological media and model food.

Keywords: *Listeria monocytogenes*, Thermal characteristics, Chorinated stress

Introduction

Listeria monocytogenes are Gram-positive, facultative, and rod-shaped bacteria found in various environmental. This pathogen caused listeriosis, with an especially high mortality rate and was considered for one in four deaths due to infections. The estimated listeriosis cases of 1600 annually were reported (Ramaswamy *et al*., 2007). Listeriosis causes severe problems in pregnant women and immunocompromised persons such as elderly, newborn and those receiving special therapies (Rahimi *et al*., 2012; Wonderling *et al*., 2004). *L. monocytogene*s infects vulnerable persons through contaminated food (Derra *et al*., 2013). Subsequently *L. monocytogenes* flourished in high salt and cold conditions, it is normally related with refrigerated ready-to-eat food products (Gandhi and Chikindas, 2007). As environmental conditions exerted a great impact on bacterial cell, it was notified that the exposure to different stresses might directly affect the response characteristic of *L. monocytogenes* (Roche *et al*., 2005). *L. monocytogenes* was previsly reported the adaptive responses to physiological stresses such as acid, heat, or oxidative that these cells were likely to be contaminated in the food processing environments (Bolton and Frank, 1999; Lou and Yousef, 1997; O'Driscoll *et al.*, 1996). Hence, *L.*

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monocytogenes cells might become stress and resist to environmental conditions compared to non-adapted cells. Therefore, in current situation there has been a rising concern about the frequently exposure to sub-lethal concentrations of disinfectants might contribute to stress adaptation to the other treatment which was known as cross protection phenomena and resulting in food safety risk (Tezel an Pavlostathis, 2015; To *et al*., 2002). *L. monocytogenes* cells are frequently contacted to oxidative stress in food processing environments in according to the utilization of cleaning and sanitization compounds, especially NaOCl (Finnegan *et al*., 2010; Le *et al*., 2015). For this reason, it might result in alterations in response to other stimuli such as hermal treatment. Therefore, the understanding about the impact of sublethal concentrations of oxidative on the survival of *L. monocytogenes* in lethal oxidative stress was essential. The objective was to investigate the impact of sodium hypochlorite stress on thermal characteristic of *L. monocytogenes* in both microbiological media and model food.

Materials and methods

B*acterial strain and preparation of inoculum*

L. monocytogenes strain DMST17303 obtained from the Department of Medical Sciences, Ministry of Public Health, Thailand was used as tested organism. Briefly, the culture was activated by 50 mL of Tryptic Soy broth (BD Difco, Sparks, MD, USA) + 0.6% Yeast extract (BD Difco, Sparks, MD, USA) (TSB-YE) at 37 ℃ for 18 hrs. The obtained inoculum was centrifuged with Refrigerated Centrifuge (New Brunswich inniva 4000, U.K) at 4000×g at 4 ℃ for 15 min followed by washing with phosphate buffer solution 2 times. The initial population was around 9.0 Log_{10} CFU/mL

Preparation of chlorinated stressed L. monocytogenes

The activated *L. monocytogenes* was transferred to sodium hypochlorite solution at the concentration of 0.5 ppm for 30 min at 27 ℃. Cell suspension was withdrawn, and the population was emumerated on both TSA-YE and Oxford agar. Stress cell was calculated from the difference in the number of colonies on those two microbiological media.

Thermal inactivation

The heat resistance in the term of D-value and Z-values of both normal *L. monocytogenes* and chlorinated stress *L. monocytogenes* were determined. About 1.0 mL of tested *L. monocytogenes* was loaded into 9.0 mL of Meat extract broth (BD Difco, Sparks, MD, USA), which was pre-

heated in shaking water bath. The thermal treatments for both *L. monocytogenes* were at 40, 45, 50, 55, 60, 65, 70, 75, and 80. The treatment temperatures were selected based on the common Sous-vide temperature range. The come-up-time (CUT) was previously measured by T-type thermocouples at the 8 randoms geometrical point of water bath. The time at CUT was set to 0 min. For each isothermal treatment, sample was taken out at time intervial from 0-120 min, then immediately chilled in an ice-water bath for 2 min to stop further inactivation. Each thermal inactivation was repeated three times independently.

Baterial enumeration

The heat-treated *L. monocytogenes* was 10-fold serially diluted using PBS, pH 7.4, and 100 µl of an appropriate dilution were plated on TSA-YE plates in duplicate followed by incubation at 37±2 ℃ for 24 hrs. and counted for microbial populations which were converted to Log_{10} CFU/mL. The detection limit was 1.0 Log_{10} CFU/mL.

Calculation of D-value and Z-value

The survival curves of *L. monocytogenes* were obtained by correlation plotting the Log_{10} counts against the heat treatment times at each temperature. The best fit line for survivor plots were determined by linear regression with Microsoft Excel (Microsoft Corp., Redmond, WA) and the negative reciprocals of the slopes were calculated as the decimal reduction times (D-values). The logarithms of D were also plotted against temperature to determine the Z-value of the bacteria by calculating the inverse of the slope.

The specific death rate and Arrhenius equation

The inactivation of both *L. monocytogenes* in the view of kinetic model was presented in equation (1). N_0 was the initial population, N was the number of target organisms at the desired time (t), t was the time, and k-value represented the specific dead rate. The inactivation was identical to that in aqueous liquids, in which case the temperature dependence of k-value obeyed the Arrhenius law (Stanbury *et al*., 2016) as shown in equation (2)

$$\boldsymbol{N = N_0 e^{-kt}} \quad (1)$$

$$\boldsymbol{k = k_0 e^{\frac{-E_a}{RT}}} \quad (2)$$

Challenge test in fresh beef

Fresh beef was purchased from a local market near Thammasat University with the agreement of post-mortem inspection. Beef was cut in to 10 g per each and individually vacuum packed, one bag each. The prepared beef samples were frozen at -18 ℃. Avoiding the natural contamination, the prepared beef was irradiated at 25 kG using gamm-radiation. The inoculation procedure was devided into inside and outside contamination. For inside contamination, cell of L. monocytogned was injected in beef sample using sterile syringe at the volume of 1.0 mL. For outside contamination, the cell suspension wer inoculated on the surface of samples. The studies in chlorinated stressed *L. monocytogenes* cells were done the same as described above.

Statistical analysis

Experiments were designed as a complete randomized experiment. The data will be subjected to analysis of variance using the SPSS and treatment mean comparison by the Duncan's New Multiple Range Test at a significance level of $p \leq 0.05$ using the SPSS software package.

Results

Thermal inactivation of normal L. monocytogenes in meat extract broth

Population of normal *L. monocytogenes* cells in meat extract broth heated at 40, 45, 50, 55, 60, 65, 70, 75 and 80 ℃ for a specified time, it was found that at these temperatures promised for survival which showed the downward trend of *L. monocytogenes* (Figure 1). There was not changed in the population of *L. monocytogenes* at 40 ℃. At 45 and 50 ℃, the population of *L. monocytogenes* decreased by 1.0 Log_{10} CFU mL^{-1} after 120 min, and decreased to 2.0 Log_{10} CFU mL^{-1} after 35 min at 55 ℃.

At 60 and 65 ℃, there was rapid reduction of microorganism without change in heat resistance to 3.0 and 2.0 Log_{10} CFU mL^{-1} at 10 and 4 min, respectively. *L. monocytogenes* were not resisted to 70, 75 and 80 ℃, the populations were not detected within 1 min. The D-value of *L. monocytogenes* in normal cellular conditions decreased with increasing temperature. These phenomena resulted in damaging cells which did not present a regenerative nature. Since heat degrades proteins and enzymes, which were key components of many mechanisms in microbial cells. *L. monocytogenes* was unable to exist, and the measurable heat resistance was reduced. Higher temperatures rapidly increased inhibition, making D-values difficult to determine. The effect of temperature on *L. monocytogenes* inhibition is shown in Figure 2. Temperature ranged from 40

℃ to 80 ℃, the Z-value of *L. monocytogenes* in normal cell conditions, the slope was calculated at 8.58 ℃.

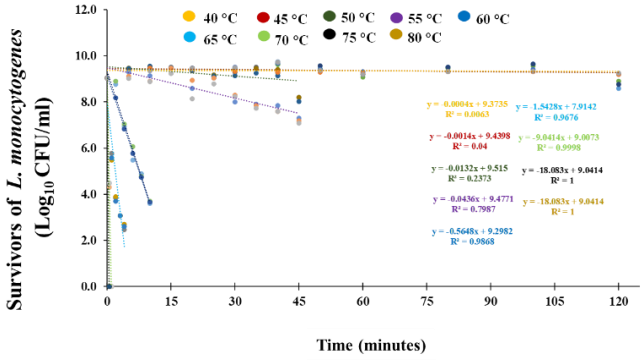

Figure 1. Survival curves and linear regression of normal *L. monocytogenes* in Meat extract broth at different temperature

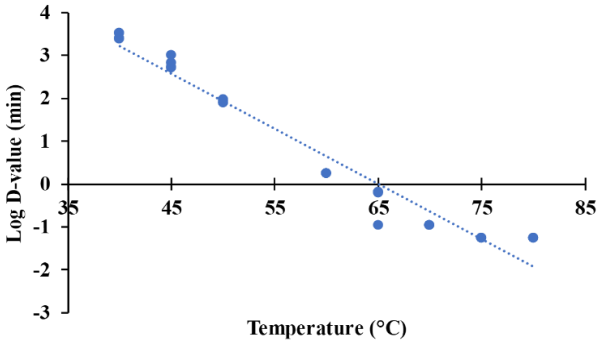

Figure 2. Thermal death time curve of normal *L. monocytogenes* in meat extract broth

Thermal inactivation of chlorinated stressed L. monocytogenes in meat extract broth

L. monocytogenes was subjected to stress from 0.5 ppm sodium hypochlorite solution for 30 min before heating (Figure 3). It was found that the *L. monocytogenes* population decreased by only 1.0 Log_{10} CFU mL^{-1} after 70 min at 40 and 45 ℃, while at 50 and 55 ℃ *L. monocytogenes* was decreased to 6.0 and 4.0 Log_{10} CFU mL^{-1} after 30 min, respectively. No survival was observed in the test bacteria injured by sodium hypochlorite solution when the media temperature reached to 60, 65, 70, 75 and 80 ℃. Comparison of the D-values of *L. monocytogenes* in the injured state showed that at the same temperature, the D-values of microorganisms injured by sodium hypochlorite solution were much lower due to the protein interaction with sodium hypochlorite solution in the cell

membrane of *L. monocytogenes* by penetrating into the protoplasmic membrane. The chlorine reacted with oxidation against the protoplasm of the cell. It precipitated proteins and reacted with the sulfhydryl's of the proteins to form irreversible products thereby disrupting the metabolism of *L. monocytogenes*. The increased destruction and inhibition were more rapidly than normal microorganisms when heated at the same temperature.

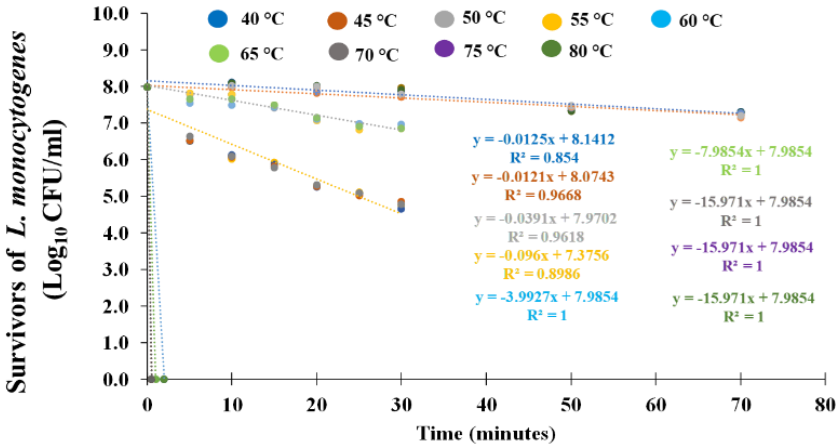

Figure 3. Thermal Death Time Curve of chlorinated stressed *L. monocytogenes* in Meat extract broth

The impact of temperature effects on *L. monocytogenes* inhibition in a temperature range from 40 ℃ to 80 ℃ was demonstrated in Figure 4. The Z-value of *L. monocytogenes* in the injured state was calculated from a slope of 10.54 ℃. This result indicated that sodium hypochlorite solution induces *L. monocytogenes*. It decreased heat sensitivity with temperature changes when compared to cells without treated with sodium hypochlorite solution. Such effects were caused by prolonged using sodium hypochlorite solution at low concentration causing stress to microbial cells and entering a viable but nonculturable state (VBNC) to respond to environmental stress.

The spesific deat rate and Arrhenius equation

For the Arrhenius equation, the k-value of the thermal process changed with the temperature subjected to *L. monocytogenes* under normal (Figure 5) and cell injury conditions (Figure 6). The activation energy (E_a) was 3.96 and 2.97 kJ mol^{-1}, respectively, which showed that the injured microorganisms had calculated activation energy, which was much lower when compared to the activation energy of the normal cells.

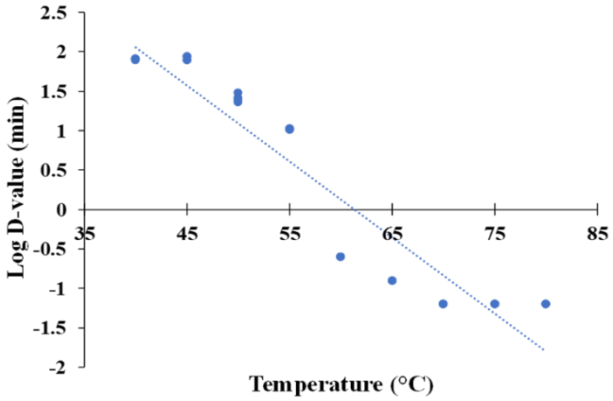

Figure 4. Thermal Death Time Curve of chlorinated stressed *L. monocytogenes* in Meat extract broth

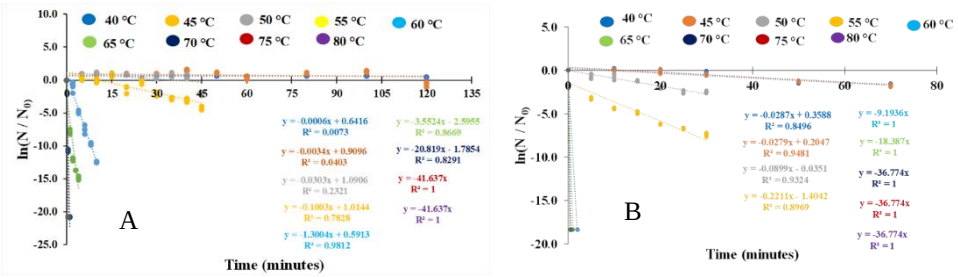

Figure 5. Specific death rate of normal *L. monocytogenes* (A) and chlorinated stress *L. monocytogenes* (B) in HM extract broth at different temperature

These observations indicated that at the same temperature, the sodium hypochlorite solution decreased the duration of *L. monocytogenes* destruction, consistent with the D-value of *L. monocytogenes*.

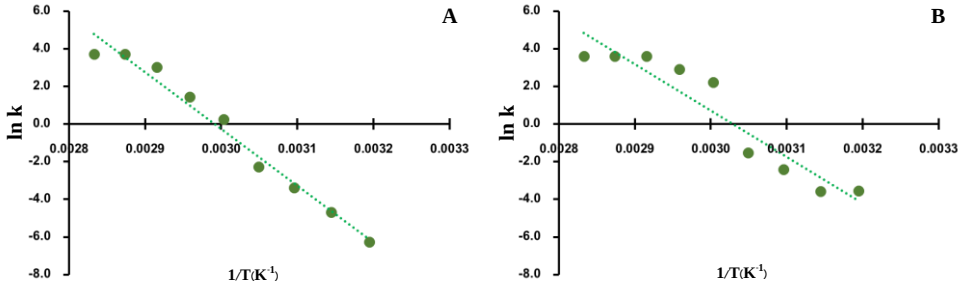

Figure 6. Arrhenius plot of nomal (A) and chlorinated stress (B) of *L. monocytogenes* in meat extract broth

The D-value of a normal cell after exposure to sodium hypochlorite solution decreased significantly when compared to the D-value of a normal cells at the same temperature. The relationship between the natural logarithm of N/N_0 and the linearization time was compared as shown in Figure 5. The slope was the specific death rate or k-value. These values were found in the incidence of *L. monocytogenes* death in normal and injured cells, at 40 and 45 ℃, no significantly decreased in microbial mortality was observed. At 50, 55, 60 and 65 ℃, *L. monocytogenes* in normal cell conditions showed a faster death rate. The mortality rate could not be determined at 70, 75 and 80 ℃, whereas the mortality rate of injured cells could not be determined at temperatures above 60 ℃. While *L. monocytogenes* injuried from sodium hypochlorite solution demonstrated the D-value and Z-value that could be used in the design of thermal inactivation system, that was analogous to the k-value and Arrhenius constant. However, using Z-value to predict microbial mortality at higher temperatures presented a higher specific mortality in readings than the Arrhenius equation. In addition, there was a survival mechanism by generating proteins called Heat Shock Proteins (HSPs) in response to heat stress. As these results, the mortality rate of microorganisms tended to decrease unsteadily. That inevitably suggested that the time required to thermal process at each temperature should be considered to reach a safe level for each.

Challenge test in fresh beef

Result showed the survivors plot of a population of *L. monocytogenes* contaminating outside and inside beef whiuch decreased in the survival trend of the test microorganisms (Figure 7). It was found that at 40 and 45 ℃ for 125 minutes, the reduction of *L. monocytogenes* contaminated outside beef was 1.0 Log_{10} CFU mL^{-1} as well as at 50 and 55 ℃ for 30 min. While at 60 and 65 ℃, it was destroyed *L. monocytogenes* after 10 and 1.5 min of elapsed times, respectively. Survival of *L. monocytogenes* was not observed in contaminated outside of the beef when the core temperature of 70, 75 and 80 ℃.

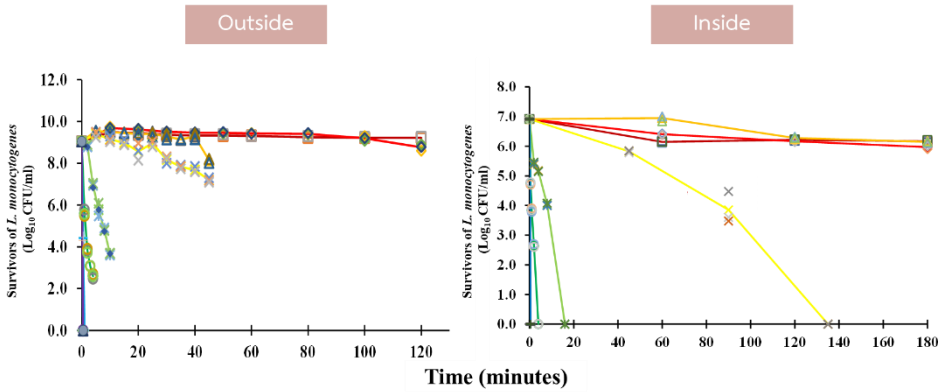

Figure 7. Number of normal *L. monocytogenes* contaminated inside and outside of fresh beef at different temperature

In addition, it was found that when heating for longer, the population of internal contamination *L. monocytogenes* was slightly decreased in survival at 40, 45 and 50 ℃ at 180 min. *L. monocytogenes* were destroyed at 135 min at 55 ℃, 8 and 2 min at 60 and 65 ℃, and survival of *L. monocytogenes* was not detected when the core temperature of the beef rose to 70, 75 and 80 ℃, as in the case with *L. monocytogenes* contaminated outside beef samples. Comparison of the decreased D-value of *L. monocytogenes* contaminated outside and inside the beef as the temperature rises. *L. monocytogenes* which contaminated the outside area was significantly decreased in D-value with increasing temperature, while the D-value of *L. monocytogenes* contaminated inside beef almost did not change when the temperature was increased from 40 to 45 and 50 ℃. The Z-value of the contaminated outside samples was 9.60 ℃, while the Z-value of *L. monocytogenes* contaminated inside was 9.07 ℃. It could be externally indicated that *L. monocytogenes* contaminated on the surface which showed greater resistance to temperature than *L. monocytogenes* contaminated inside the beef.

It showed survivors curves of a population of *L. monocytogenes* injured by sodium hypochlorite solution and contaminated outside and inside beef with the tendency of microbial survival decreased by 1.0 Log_{10} CFU mL^{-1} at 40 and 45 ℃ after 175 min and decreased to 5.5 - 6.0 Log_{10} CFU mL^{-1} at 50 ℃ and 55°C after 120 min, with no change in microbial heat resistance (Figure 8). It was found that at 60 and 65°C, all microorganisms could be destroyed in only 10 and 1.5 minutes, respectively. The survival of *L. monocytogenes* was not observed in the externally contaminated when the core temperatures were 70, 75 and 80 ℃. When considering the injured *L. monocytogenes* contaminated inside beef, it was found that *L. monocytogenes* increased over time. There was 1.0 Log_{10} CFU mL^{-1} decreased in survival tendency when beef samples which heated for 180 min at 40 and 45 ℃. At 50 ℃ for 135 min, the population of *L. monocytogenes* was reduced to 4.0 – 5.0. Log_{10} CFU mL^{-1}. *L. monocytogenes* was also found in the injury state, all lesions were destroyed in less than 90, 16 and 1 min at 55, 60 and 65°C, and no survival of the microorganisms was detected at 70, 75 and 80, as in the case of *L. monocytogenes* in the state contaminated outside of the beef.

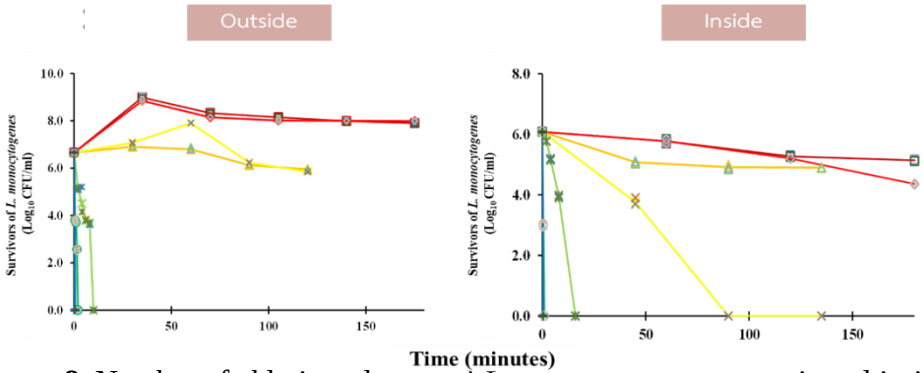

Figure 8. Number of chlorinated stressed *L. monocytogenes* contaminated inside and outside of fresh beef at different temperature

Comparison of the decreased in D-value of *L. monocytogenes* contaminated outside and inside the beef samples as the temperature increased, D-value of *L. monocytogenes* was significantly decreased with increasing temperature. The contamination inside the beef almost did not change as the temperature increased from 40 to 45 and 50°C. Considering the calculated Z-value of the contaminated outside samples was 8.58°C, while the Z-value of *L. monocytogenes* was 9.54°C for inside contaminated. It was indicated that at the same temperature caused to decrease 1.0 Log_{10} CFU mL^{-1} of *L. monocytogenes* which contaminated outside beef samples would be longer. However, when the temperature changed, externally contaminated *L. monocytogenes* were less resistant to thermal change than *L. monocytogenes* contaminated inside the beef.

Discussion

In this study, the stability of *L. monocytogenes* chlorinated stress adaptation against thermal inactivation was investigated. In the Meat extract broth assay, at all temperature conditions were tested considering the demonstrated that thermal adaptation in the term of D-value and Z-value was progressively reversed after incubation. The D-values of chlorinated stressed *L. monocytogenes* was lower decreased significantly as the temperature increased than normal *L. monocytogenes*, showing increased thermal resistance of stressed cell. Oxidative stress is one of the main physiological stresses that L. monocytogenes cells undergo during cleaning and sanitation in the food processing environments. Chlorinated stress is the part of oxidative stress and affected to the adaptation of *L. monocytogenes* under a range of sublethal oxidative stress. From the results showed that pre-exposure to 50 ppm for 30 min sodium hypochlorite readily triggered oxidative stress adaptation against *L. monocytogenes* to heat treatment. Expansion in oxidative stress confrontation in foodborne pathogens such as *L. monocytogenes*, following exposure to sublethal condition have been formerly reported (Cebrián *et al*., 2010; Christman *et al*., 1985; Lou and Yousef, 1997). Increased tolerance of oxidative stress in foodborne pathogen was directly associated with a higher activity of antioxidant enzymes (Scott *et al*., 1987). Presence of H_2O_2 forms reactive oxygen species such as peroxides and superoxide which could cause genetic and physiological mechanisms that were in charge for producing various antioxidant enzymes. H_2O_2 was accomplished of inducing the production of catalase through activating an *Oxy*R regulator in *Escherichia coli* (Aslund *et al*., 1999). In addition, reactive oxygen species correspondingly activated *Sox*R transcriptional activator through oxidizing the iron sulfur center and thus encourage the production of antioxidant enzymes such as endonuclease iv, glucose 6-phosphate dehydrogenase, Mn superoxide dismutase, and paraquat diaphoroase (Gaudu and Weiss, 1996; Greenberg *et al*., 1990; Hidalgo *et al*., 1997). Therefore, when exposed to sublethal oxidized agents, *L. monocytogenes* cells might produce more antioxidant enzymes that improved their survival during lethal oxidative stress compared to control cells. Mechanisms accountable for the

increased resistance within the cholinated stressed were not well understood. Limited studies indicated that the observed greater heat resistance after sublethal injury condition of microorganism might be by reason of an upsurge in potassium influx by the *kdp* transporter (Gruzdev *et al*., 2012), expression of heat and cold shock proteins, Fe-S clusters, sigma factors (*rpo*E and *rpo*S) (Deng *et al*., 2012), osmoprotectant transport (*pro*PU and *osm*U) (Finn *et al*., 2013), trehalose synthesis (Li *et al*., 2012), and others, which together deliberate protection to heat treatments. At the phenotypic level, several studies reported that tolerance of *L. monocytogenes* decreased during log phase growth at optimal temperatures (30–37 ℃) (Datta and Benjamin, 1997; Davis *et al*., 1996; O'Driscoll *et al*., 1996). Moreover, altered fatty acid composition of cell membrane after adaptation in *L. monocytogenes* has been proposed in numerous studies (Giotis *et al*., 2007; Mastronicolis *et al*., 2010; van Schaik *et al*., 1999). Hence, the cross protection against thermal application might result from the modified fatty acid composition of cytoplasmic membrane contributing to the membrane stabilization. The effect of the food matrix on the inactivation kinetics of *L. monocytogenes* was studied by means of the effect of the contamination area including inside and outside contamination. For all inactivation temperatures, it was observed that the maximum specific death rate in the model food system was significantly higher than in the respective micobiological broth. The residual cell population related to the log-reduction caused by the thermal treatments. A protective effect of a food matrix against thermal inactivation was presented, although dependent on the inoculumn procedures and cell type and also the inactivation temperature. It has been reported that bacterial cells which are surrounded by food matrix might involvement alterations in cell development, membrane permeability, surface tension, osmotic pressure and metabolism, potentially affecting their thermal resistance (Dervakos and Webb, 1991; Meldrum *et al*., 2003; Noriega *et al*., 2013; Wilson *et al*., 2002). It was already previous repoted that the effect of the growth morphology of *L. monocytogenes* on thermal inactivation kinetics was not significant. Therefore, the higher heat resistance which has often been observed for submerged colonies in a food environment as compared to planktonic cells in broth systems (Murphy *et al*., 2000; Velliou *et al*., 2013), is probably also caused by a protective effect of the food environment and not by the differences in growth morphology. Verheyen *et al*. (2018) demonstrated that cells in a food matrix were subjected to more stressing conditions. These stressing conditions might lead to an increased resistance to a subsequent thermal treatment. The cells could develop more heat resistant after growth in food matrix as a result of a cross-protective effect triggered by tolerance response to several stress conditions (Noriega *et al*., 2013; Malakar *et al*., 2000; Walker *et al*., 1997; Wilson *et al*., 2002). Therefore, the protection against thermal treatment from both adaptation and food metrix should be wisely considered when chlorine compounds was used as a pre-lethal inactivation treatment in ready to eat food products. Since adaptation in *L. monocytogenes* could protect the cells from heat inactivation, investigation of the stability of

several adaptations of foodborne pathogens induced cross resistance could have more practical value in food industry.

The thermal characteristic as indicated by D-values of *L. monocytogenes* in both normal cell and chlorinated stress cell increased as the heat temperature decreased. Chlorinated stress condition was found to induce more heat resistant of *L. monocytogenes.* Reserch finding provided an important information to the food industry in develop the thermal processes for effectively control *L. monocytogenes* to be caused stress from the using chlorine compounds in the raw material preparation.

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