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Thermal inactivation of foodborne pathogens and the USDA pathogen modeling program

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Abstract The use of heat to inactivate foodborne pathogens is a critical control point and the most common means for assuring the microbiological safety of processed foods. A key to optimization of the heating step is defining the target pathogens' heat resistance. Sufficient evidence exists to document that insufficient cooking, reheating, and/or subsequent cooling are often contributing factors in food-poisoning outbreaks. Accordingly, the objective of thermal processing is to design sufficient heating regiments to achieve a specific lethality for foodborne pathogens in foods. The effects and interactions of temperature, pH, sodium chloride content, sodium pyrophosphate, and sodium lactate concentration are among the variables that were considered when attempting to assess the heat inactivation kinetics of *Escherichia coli* O157:H7, *Listeria monocytogenes*, *Salmonella* spp., and spores of non-proteolytic *Clostridium botulinum*. Incorporation of these multiple barriers usually increases the sensitivity of pathogens to heat, thereby reducing heat requirements and ensuring the safety of ready-to-eat food products. Complex multifactorial experiments and analysis to quantify the effects and interactions of additional intrinsic and extrinsic factors and development of "enhanced" predictive models are underway to ensure the microbiological safety of thermally processed foods. Predictive inactivation kinetics (thermal death) models for foodborne pathogens have been converted into an easy-to-use computer program that is available on the USDA–Eastern Regional Research Center website. These models should aid in evaluating the safety of cooked products and are being used as building blocks for microbial risk assessment.

Keywords Pathogens · Heat inactivation · Modeling · Thermal inactivation

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Introduction

The contamination of the food supply with spoilage and pathogenic microorganisms continues to be a global problem despite the wide range of preservation methods employed. In addition, the growth or survival of potentially life-threatening pathogens in foods and in food environments is a significant food safety hazard. Consumers continue to demand fresher, more convenient, minimally or mildly processed, ready-to-eat (RTE) foods. In response to consumers' demands, the food industry continues to be innovative in the development and production of food. Since product composition, processing and preparation operations, supply chain of the modern food industry, consumers' life-style, and eating habits have changed dramatically, differences in the levels and kinds of pathogens encountered can be expected. The ability of low numbers of human pathogens to survive or proliferate in foods even when stored under refrigeration or in reduced oxygen atmospheres is a risk for potential public health hazard. The risk of foodborne disease is a combination of likelihood of exposure to the pathogen, the likelihood of infection resulting in illness, and the severity of the illness. This leads to increased pressure on the food producers and the regulatory agencies to obtain better understanding about the pathogens likely to be present on food products and the changes in microbial populations as a result of food environments and processing.

Accordingly, food preservation is designed to enhance or protect food safety while maintaining the organoleptic attributes of food. Inactivating or inhibiting the growth of undesirable microorganisms is very important for the successful and acceptable preservation of food. While a large number of preservation processes are at the disposition of food processors, the use of adequate heat treatment to destroy pathogenic and spoilage microorganisms is one of the most effective food preservation methods in use today. Therefore, heat treatment designed to achieve a specific lethality for foodborne pathogens is a critical control point in food processing and is fundamentally important to assure the shelf-life and microbiological safety of thermally processed foods. A key to optimization of the heating step is defining the target pathogen's heat resistance. While over-estimating the heat resistance negatively impacts the product quality by altering the organoleptic attributes and nutritional qualities of a food, under-estimating increases the likelihood that the contaminating pathogen persists after heat treatment or cooking. Inadequate heat treatment or undercooking is an important contributing factor in causing food-poisoning outbreaks.

Inactivation kinetics parameters

Primary models—linear thermal inactivation kinetics

The higher the initial microbial population in a food, the longer the processing/heating time at a given temperature is required to achieve a specific lethality of microorganisms. Accordingly, the thermal process is designed to base on the expected microbial load in the raw product. For most foodborne pathogens and spoilage microorganisms (vegetative cells or spores), the inactivation usually follows the first-order kinetics (Eqs. 1 and 2). As such, the heat resistance of bacteria is described by two parameters: *D*- and *z*-values. The *D*-value is the time at a particular temperature necessary to destroy 90% of the viable cells or spores of a specific organism, which is the inverse of the slope in Fig. 1. It is a measure of the death rate or the heat sensitivity of the organism. The *z*-value, an indicator of temperature sensitivity, is the change in heating temperature needed to change the *D*-value by 90% (1 log cycle) and is obtained by plotting log *D*-values on the *x*-axis and temperature on the *y*-axis (thermal death time curve; Fig. 2). D_{ref} is the *D*-value at a reference temperature (T_{ref}). *D*- and *z*-values are used for designing heat processing requirements for desirable destruction of microorganisms in a particular food.

$$\log\left(\frac{C}{C_0}\right) = -\frac{t}{D} \tag{1}$$

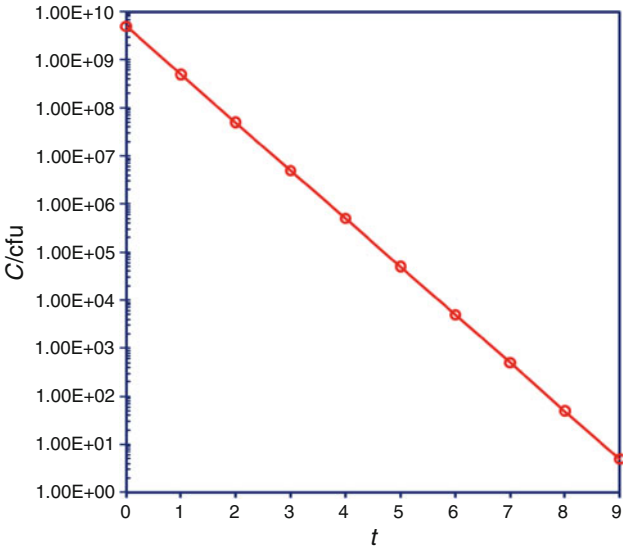

Fig. 1 The first-order kinetics of bacterial inactivation and the log-linear thermal inactivation curve (*C* is the bacterial concentration in colony-forming units (cfu), and *t* is the heating time under a constant temperature)

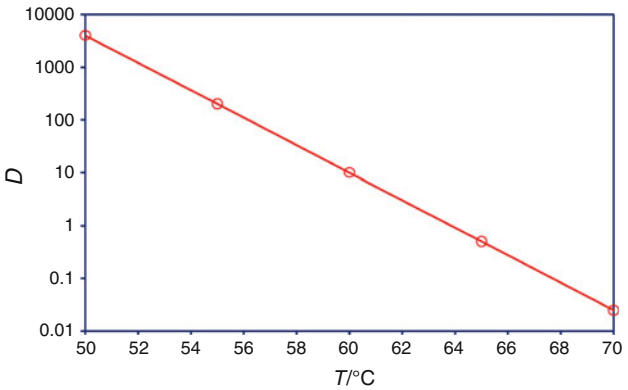

Fig. 2 Thermal death time curve (*D* is the thermal death time and *T* is temperature)

$$\log\left(\frac{D}{D_{ref}}\right) = -\frac{T - T_{ref}}{z} \tag{2}$$

This traditional first-order kinetic model of thermal inactivation [9] forms the basis of calculations used in thermal processing and has served the food industry and regulatory agencies for decades.

To use the first-order kinetics in designing thermal process, which is usually not held under constant temperature conditions, both Eqs. 1 and 2 must be integrated to calculate the surviving bacterial counts (Eq. 3).

$$\log\left(\frac{C}{C_0}\right) = -\frac{\int_0^t 10^{\frac{T-T_{ref}}{z}} \mathrm{dt}}{D_{ref}} \tag{3}$$

Deriving from Eq. 3, the concept of equivalent thermal process value, or F_0 value, is used in thermal process design. The F_0 value is defined as the ratio of the equivalent heating time at the reference temperature T_{ref} and D_{ref}, which in fact is the multiple of D_{ref} values. To make use of the F_0 value, the heat effect (or lethality) at different time and temperature conditions is converted to the equivalent heating time at the reference temperature. The final F_0 value is the cumulative equivalent heating time at reference temperature (T_{ref}) divided by the D-value (D_{ref}).

Primary models—non-linear thermal inactivation kinetics

Although the linear kinetics is the predominant model used in food microbiology and in thermal process design, deviations from the classical semi-logarithmic linear declines in the log numbers with time have been frequently observed, even when precise attention is paid to methodology [1, 8, 10]. Such deviations are of two forms: (a) a shoulder or a lag period, i.e., time periods when the bacterial populations remain at the inoculation level (Fig. 3); (b) a tailing, i.e., a subpopulation of more resistant bacteria that decline at a slower rate (Fig. 4).

The shoulder effect (Fig. 3) under constant temperature conditions can be modeled by using a delay time to represent the shoulder portion, as expressed in Eq. 4. This model should be used with care, as the shoulder time may be misrepresented by the thickness of a sample. For solid foods, heat is conducted by conduction. Longer heating time may be needed to conduct the heat to the center for a thick sample. In order to eliminate the thickness effect, it is necessary to ensure that the thickness of the sample is thin enough such that the heat transfer time is negligible when compared with heating time. To eliminate the thickness

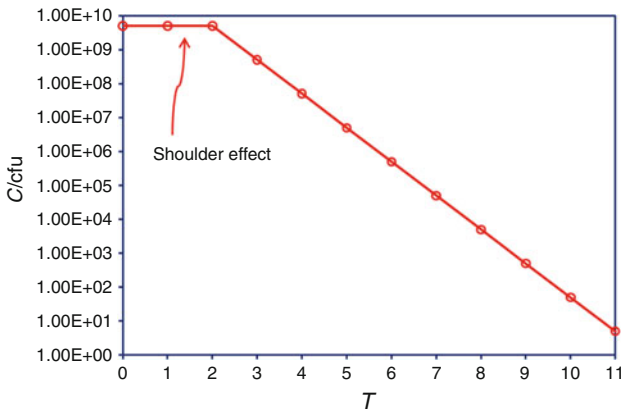

Fig. 3 Illustration of shoulder effect on thermal inactivation curve (C is the bacterial concentration in colony-forming units (cfu), and t is the heating time under a constant temperature)

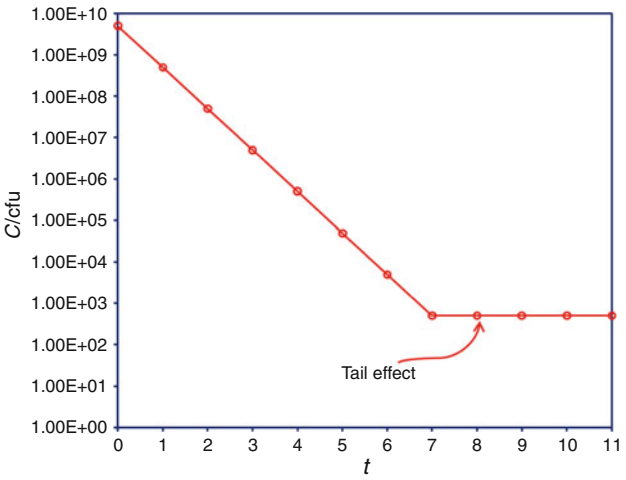

Fig. 4 Illustration of the trailed effect of a thermal inactivation curve (C is the bacterial concentration in colony-forming units (cfu), and t is the heating time under a constant temperature)

effect, it may be also necessary to deduct the initial heat time—the time needed to increase the temperature at center to the heating temperature from the total heating time. Although it is possible to correct the initial heating time, it is still necessary to ensure that a thin solid sample is used during kinetic studies.

$$\begin{cases} \log\left(\frac{C}{C_0}\right) = 0, & at\ t \le t_0 \\ \log\left(\frac{C}{C_0}\right) = -\frac{t}{D}, & at\ t > t_0 \end{cases} \quad (4)$$

The tail-effect can be modeled by the mixed culture model. Assuming that a culture consist of two different strains of bacteria with different capacities to resist heat, as represented by D_H (higher D-value, i.e., more heat-resistant) and D_L (lower D-value, i.e., less heat resistant). The fraction of the higher D-value strain in the culture is f, and the rest (1−f) is the fraction for the lower D-value strain. During heat treatment, the fraction of lower D-value will be preferentially inactivated by heat, leaving the strain with higher D-value to survive for a longer time. The model for the mixed culture is represented by Eq. 5. The tail-effect is more evident with larger f-values (Fig. 5).

$$\log\left(\frac{C}{C_0}\right) = \log\left[fe^{-\frac{2.303}{D_H}t} + (1-f)e^{-\frac{2.303}{D_L}t}\right] \quad (5)$$

A more general model [2] can be represented by introducing a power index to the linear model (Eq. 6). Depending on the shape of the curve, the power index (b) can be either >1, 1, or <1. With $b = 1$, the model is reduced to the traditional linear model (Fig. 6). With $b < 1$, the survival curve is convex, or upwardly concaved. Some of the tail-effect can be represented by a curve with $b < 1$. With $b > 1$, the survival curve is concave, or downwardly concaved. Some the shoulder effect can be represented with $b > 1$.

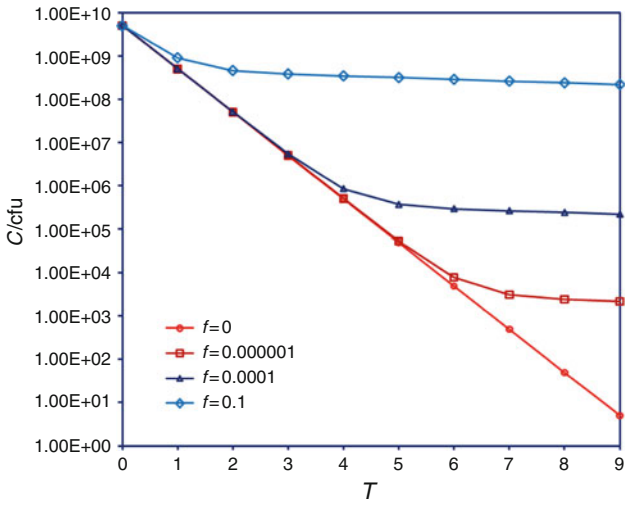

Fig. 5 Effect of mixed cultures on development of tail during thermal inactivation. Illustrated in this figure: $D_H = 25$, $D_L = 1$, and $f = 0$ to 0.1 (D is D-value and f is the fraction of the more heat-resistant strain in a two-strain mixed culture; C is the bacterial concentration in colony-forming units (cfu), and t is the heating time under a constant temperature)

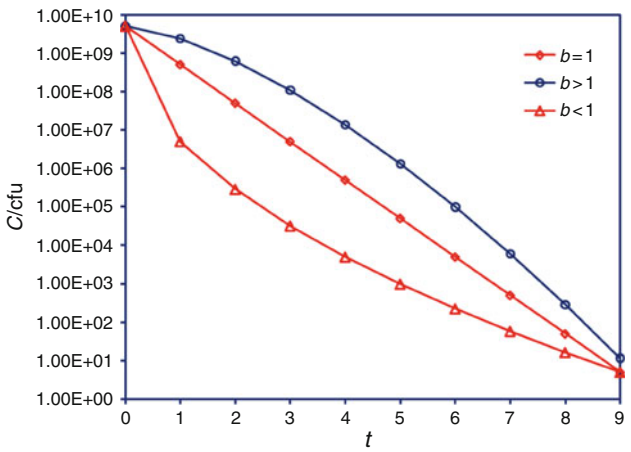

Fig. 6 A general thermal inactivation model (Eq. 6) for describing three different shapes of survival curves (b is the power index in Eq. 6; C is the bacterial concentration in colony-forming units (cfu), and t is the heating time under a constant temperature)

$$\log\left(\frac{C}{C_0}\right) = -at^b \tag{6}$$

A modified Gompertz model (Eq. 7) also can be used to describe non-linear thermal inactivation curves, and particularly curves with shoulders [3]. In Eq. 7, μ is the relative rate at $t = M$. This equation is totally an empirical model, but it can be used to represent the curves with shoulder effects (Fig. 7).

$$\log\left(\frac{C}{C_0}\right) = 1 - e^{-e^{-\mu(t-M)}} \tag{7}$$

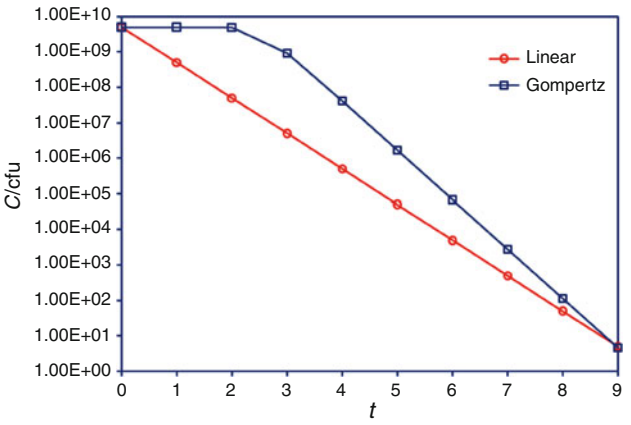

Fig. 7 A modified Gompertz equation (Eq. 7) used to describe shoulder effects (C is the bacterial concentration in colony-forming units (cfu), and t is the heating time under a constant temperature)

Factors affecting heat resistance

An appropriate heat treatment designed to achieve a specified lethality of microorganisms is influenced by many factors, some of these can be attributed to the inherent resistance of microorganisms, while others are due to environmental influences. Examples of inherent resistance include the differences among species and the different strains or isolates of bacteria (assessed individually or as a mixture), and the differences between spores and vegetative cells. Environmental factors include those affecting the microorganisms during growth and formation of cells or spores (e.g., stage of growth, growth temperature, growth medium, and previous exposure to stress, etc.) and those active during the heating of bacterial suspension, such as the composition of the heating menstruum (amount of carbohydrate, proteins, lipids, and solutes, etc.), water activity (a_w), pH, added preservatives, method of heating, and methodology used for recovery of survivors, etc.

Heat inactivation kinetics predictive models

The effectiveness of the individual effects of heat treatment, pH, and salt, etc., with regard to pathogen inactivation is maximized by conducting multiple factorial experiments in which the effects and interactions of these parameters in foods are assessed in lowering the heat resistance of foodborne pathogens. Subsequently, inactivation kinetics or thermal death models are developed which predict the target pathogen's survival within a specific range of food formulation variables. These models can help either to establish an appropriate heat treatment, or to

understand and determine the extent to which existing/traditional thermal processes could be modified for a variety of cooked foods. The models can contribute to more effective evaluation and assessment of the impact of changes in food formulations that could affect their microbiological safety or the heat lethality of pathogens. These predictive models enable food processors and regulatory agencies to ensure critical food safety margins by predicting the combined effects of multiple food formulation variables. The food processors are able to design appropriate processing times and temperatures for the production of safe food with extended shelf-life without substantially adversely affecting the sensory quality of the product. However, it is of critical importance that the *D*-values predicted by the models first be validated with heat resistance data obtained by actual experiments in specific foods before the predicted values can be used to design thermal processes for the production of a safe food.

It is universally known that heat is the most common method in use to destroy foodborne pathogens. Tunick et al. [10] reported that the denaturation transitions observed by "Differential Scanning Calorimetry" indicate potential sites of cellular injury. In another study [7], when bacterial pathogens were exposed to heat at 50 and 60 °C, DSC curves in the 5–99 °C range showed a series of endothermic transitions. Juneja et al. [4–6] employed a fractional factorial design to assess and quantify the effects and interactions of temperature, pH, salt, and phosphate levels and found that the thermal inactivation of non-proteolytic *C. botulinum* spores, *E. coli* O157:H7, and *L. monocytogenes* was dependent on all four factors. Thermal resistance of spores or vegetative cells can be lowered by combining these intrinsic factors. The following multiple regression equations, developed in these studies, predict *D*-values of non-proteolytic *C. botulinum* spores for any combinations of temperature (70–90 °C), salt (NaCl; 0.0–3.0%), sodium pyrophosphate (0.0–0.3%), and pH (5.0–6.5); *E. coli* O157:H7, for combinations of heating temperature (55–62.5 °C), salt (0.0–6.0%, w/v), sodium pyrophosphate (0.0.–0.3%, w/v), and pH (4.0–6.5); and *L. monocytogenes,* for combinations of heating temperature (55–6.5 °C), salt (0.0–6.0%, w/v), sodium pyrophosphate (0.0.–0.3%, w/v), and pH (4.0–8.0). The predicted *D*-values/the pathogens survival are for changes in the parameter values in the range tested from any combination of four environmental factors.

Non-proteolytic *C. botulinum* spores

$$\text{Log}_e\ D\text{-value} = -9.9161 + 0.6159\ (\text{temp}) - 2.8600\ (\text{pH}) - 0.2190\ (\text{salt}) + 2.7424\ (\text{phos}) + 0.0240\ (\text{temp})(\text{pH}) - 0.0041\ (\text{temp})(\text{salt}) - 0.0611\ (\text{temp})(\text{phos}) + 0.0443\ (\text{pH})(\text{salt}) + 0.2937\ (\text{pH})(\text{phos}) - 0.2705\ (\text{salt})(\text{phos}) - 0.0053\ (\text{temp})^2 + 0.1074\ (\text{pH})^2 + 0.0564\ (\text{salt})^2 - 2.7678\ (\text{phos})^2$$

E. coli O157:H7

$$\text{Log}_e\ D\text{-value} = -43.0646 + 1.4868\ (\text{temp}) + 3.5737\ (\text{pH}) - 0.1341\ (\text{salt}) - 8.6391\ (\text{phos}) - 0.0419\ (\text{temp})(\text{pH}) + 0.0103\ (\text{temp})(\text{salt}) + 0.1512\ (\text{temp})(\text{phos}) - 0.0544\ (\text{pH})(\text{salt}) + 0.2253\ (\text{pH})(\text{phos}) - 0.2682\ (\text{salt})(\text{phos}) - 0.0137\ (\text{temp})^2 - 0.0799\ (\text{pH})^2 - 0.0101\ (\text{salt})^2 - 6.4356\ (\text{phos})^2$$

L. monocytogenes:

$$\text{Log}_e\ D\text{-value} = -61.4964 + 2.3019\ (\text{temp}) + 1.2236\ (\text{pH}) + 0.7728\ (\text{salt}) + 1.0477\ (\text{phos}) - 0.0102\ (\text{temp})(\text{pH}) - 0.0085\ (\text{temp})(\text{salt}) - 0.0566\ (\text{temp})(\text{phos}) - 0.0210\ (\text{pH})(\text{salt}) - 0.4160\ (\text{pH})(\text{phos}) + 0.1861\ (\text{salt})(\text{phos}) - 0.0217\ (\text{temp})^2 - 0.0273\ (\text{pH})^2 - 0.0213\ (\text{salt})^2 - 13.1605\ (\text{phos})^2$$

The authors developed confidence intervals (95%) to allow microbiologists to predict the variation in the heat resistance of the pathogens. Representative observed and predicted *D*-values of non-proteolytic *C. botulinum* in ground turkey, *E. coli* O157:H7 and *L. monocytogenes* are provided in Table 1. Predicted *D*-values from the model compared well with the observed thermal death values. Thus, the model provides a valid description of the data used to generate it.

Modeling of microbial pathogens in foods

The theory of predictive microbiology is based on the fact that the microbial growth, survival, and inactivation are affected by the environmental factors. It is also based on the assumption that the responses of microorganisms to these factors are reproducible and can be characterized and quantified. The microbial response to environmental factors can be described mathematically. In other words, microbiological modeling is an attempt to define the response of a microorganism to its environment in terms of mathematical equations.

Knowing the fate of foodborne pathogens in foods is important in determining the microbiological safety levels of food products. When the potential growth of a foodborne pathogen is likely in a food product, the food product then needs to be either reformulated or processed to eliminate the presence or growth potential of pathogens to ensure food safety. The growth, survival, or death of microorganisms in food are affected by many factors, both intrinsic and extrinsic. These include: intrinsic factors, such as pH, NaCl (salt), sugars, phosphates, nitrites, water activity, nutrient level, and extrinsic factors, such as temperature,

Table 1 Observed and predicted D-values at 70–90 °C of non-proteolytic *C. botulinum* in ground turkey, *E. coli* O157:H7 and *L. monocytogenes* in beef gravy

Temperature/°C	pH	% NaCl	% Phosphate	D-value observed/min	D-value predicted[a]/min
Non-proteolytic *C. botulinum*					
70	6.50	0.0	0.00	57.7	66.0
70	6.50	1.5	0.15	40.1	46.5
75	6.25	1.0	0.10	39.1	42.3
75	6.25	1.0	0.20	32.9	38.6
90	5.00	0.0	0.00	5.0	6.3
90	5.00	1.5	0.15	3.1	4.8
E. coli O157:H7					
55	4	0.0	0.0	2.8	4.1
55	4	0.0	0.30	1.9	2.7
60	4	3.0	0.15	2.1	2.2
60	6	3.0	0.30	1.8	2.1
L. monocytogenes					
55	4	0.0	0.0	5.35	9.03
55	4	6.0	0.0	12.49	15.74
57.5	5	4.5	0.10	6.92	8.05
57.5	5	4.5	0.20	10.61	8.45

[a] Predicted D-values are the 95% upper confidence limits

atmosphere (i.e., aerobic, anaerobic, and modified atmosphere), and relative humidity. By manipulating one or more of these factors, it is possible to alter the behavior of microorganisms in foods. For examples, extending lag and generation time of microorganisms, increasing sensitivity of microbial cells or spores to heat or any other intervention technology, or preventing spore outgrowth and toxin production. By quantifying the effects and interactions of multiple food factors, predictive mathematical models can be developed to predict behavior of microorganisms in foods. Steps for development of predictive models include experimental design, data collection, model development, model validation, and development of an effective interface between the models and the end-user. An example of the interface is the USDA, Agricultural Research Service, Pathogen Modeling Program (PMP; http://ars.usda.gov/Services/docs.htm?docid=6786), a stand-alone software package of microbial models.

USDA–ARS pathogen modeling program (PMP)

PMP is a computer-based software program that constitutes a group of models that can be used to estimate the behavior of common foodborne pathogens in various environmental conditions. The models in PMP cover almost all the common foodborne pathogens in foods and environmental factors found in common types of food products. The PMP has become a premier international modeling tool and is downloaded more than 8,000 times each year in over 35 countries.

The food industry requires information on how the growth, survival, and inactivation of pathogenic bacteria are influenced by different intrinsic and extrinsic environmental conditions, several of those mentioned above, in foods to ensure the safety of their products. In response to the industry needs, PMP, has been available in different forms over the years. It consists of various models that describe the effect of environmental conditions on the behavior of common foodborne pathogenic microorganisms.

The scientists at the ERRC organized and conducted extensive microbiological experimentation with foodborne pathogens. Microbial growth, survival, or inactivation data were developed into primary mathematical models to describe microbial changes with time. Secondary mathematical models were then developed to describe the effects of environmental factors on the microbial growth, survival, or inactivation. These models can be used to predict the growth or inactivation of most common pathogens as affected by environmental conditions. Realizing that these models in mathematical equation would not be fully understood by users, the scientists transformed these predictive equations into an easy-to-use computer software, named the PMP. Through the user-friendly interface, users can select models of their interest and input environmental conditions to retrieve predictions on the growth, inactivation/lethality (thermal and non-thermal), and decline/survival of microorganisms. It is free for download from the

ARS website or can be accessed online. This program does not require any modeling and mathematical knowledge or calculations by the users. To further assist food processors in meeting regulatory requirements, references are provided for each model via direct Internet access to portable document format (PDF) files containing the research articles associated with the developed models. The PMP is a useful tool for obtaining microbial growth potential to be used in designing HACCP plans, in identifying critical control points and critical limits, and evaluating the consequences of process deviations as well as in determining safe corrective actions to be taken. Furthermore, the models enable food processors to assess the microbial risks of a particular food and estimate consequences of out-of-control process events, e.g., cooling deviation and refrigeration failure.

Before using the PMP, the user should become familiar with some basic knowledge regarding the microbial growth, principles, and the development of predictive models and the limitation of their uses. The latter is particularly important when applying information obtained from the models to real world use. The users should recognize that the predictions obtained from PMP models cannot be solely relied upon as a sole means of ensuring the microbiological safety of food products. The models cannot replace microbial validation or experimental challenge studies. Models in PMP were developed from studies that closely simulated the intrinsic and extrinsic environmental conditions which are relevant to the food products of interest. However, not all the conditions can be completely incorporated into the studies to represent the real world food systems. The predictions obtained from PMP, therefore, cannot be guaranteed to indicate the microbial behavior in food systems.

The program is updated from time to time to include newly developed models and to make it more user friendly by making improvements to the user interface. While most models included in the PMP are isothermal, it contains four models that are able to predict the growth of *C. botulinum* and *C. perfringens* under time-varying temperature conditions (cooling models). Version 7.0 of the PMP is the latest available version and contains the following types of models:

Growth models

The majority of PMP models are growth models. Model variables include atmosphere (aerobic, anaerobic), temperature, pH, water activity, and additives such as nitrite and phosphates. Aerobic growth models are for *Aeromonas hydrophila*, *Bacillus cereus*, *Clostridium perfringens*, *E. coli* O157:H7, *L. monocytogenes*, *Salmonella* spp., *Shigella flexneri*, *Staphylococcus aureus*, and *Yersinia enterocolitica*. Anaerobic growth models are for *A. hydrophila*, *B. cereus*, *C. perfringens*, *E. coli* O157:H7, *L. monocytogenes*, *S. flexneri*, and *S. aureus*

Survival (non-thermal inactivation) models

The survival models predict the inactivation of bacterial pathogens as a function of temperature, NaCl, pH, nitrite, and lactic acid. The publication for each model should be read to determine the acid that was used in the broth models to adjust broth pH. In general, survival models were developed using an organic acid (lactic acid) as the acidulant. Non-thermal inactivation/survival models are for *E. coli* O157:H7, *L. monocytogenes*, *Salmonella* spp., and *S. aureus.*

Thermal inactivation models

There are three thermal inactivation models. They are *C. botulinum*, *E. coli* O157:H7, and *L. monocytogenes*, with variables for temperature, pH, NaCl, and sodium pyrophosphate.

In their current form, PMP models are not suitable for determining process lethality calculations. To make these calculations, it is necessary to know the z-value and T_{ref} temperature, and to calculate F-values over a range of changing temperatures.

Cooling models

The cooling/growth models for *C. botulinum* and *C. perfringens* is one of the most used models by the food industry. The USDA Food Safety and Inspection Service "Compliance Guidelines for Cooling Heat-Treated Meat and Poultry Products" states that during cooling, the product's maximum internal temperature should not remain between 130 and 80 °F for more than 1.5 h nor between 80 and 40 °F for more than 5 h (http://www.fsis.usda.gov/OA/fr/95033F-b.htm). When cooling for a product deviates from the compliance guidelines, the cooling model can be used to estimate if the deviation would result in unacceptable growth of pathogens.

Conclusions and outlook to the future

The use of heat for the inactivation of microorganisms is the most common process in use in food preservation today. Heat treatment designed to achieve a specific lethality for foodborne pathogens is one of the fundamentally important strategies used to assure the microbiological safety of thermally processed foods. Heat resistance of microorganisms can vary depending on the species and strain of bacteria, food composition, physiological stage of

microbial cells or spores, and recovery conditions (type of media, temperature, atmosphere, and time of incubation) for the detection of survivors. Food characteristics leading to increased heat resistance of an organism include water activity and the presence of carbohydrates, lipids, proteins, and salt, etc.

Quantitative knowledge of the factors in food systems that interact and influence the inactivation kinetics are required to accurately estimate how a particular pathogen is likely to behave in a specific food. There is a need for better understanding of how the interaction among preservation variables can be used for predicting safety of minimally processed, ready-to-eat foods. The effects and interactions of temperature, pH, sodium chloride content, and sodium pyrophosphate concentration are among the variables that researchers have considered when attempting to assess the heat inactivation kinetics of foodborne pathogens. Incorporation of these multiple barriers increased the sensitivity of cells/spores to heat, thereby reducing heat requirements and ensuring the safety of ready-to-eat food products.

The future of thermal death determination of bacteria will likely rely on predictive thermal inactivation kinetics modeling. Complex multifactorial experiments and analysis to quantify the effects and interactions of additional intrinsic and extrinsic factors and development of "enhanced" predictive models are warranted to ensure the microbiological safety of thermally processed foods. In view of the continued interest that exists minimally processed foods, it would be logical to define a specific lethality at low temperatures. It would be useful to determine the possible effects of injury to vegetative cells and spores, that may result from mild heat treatments and factors in foods that influence the recovery of cells/spores heated at these low temperatures. In conclusion, the future research should focus on conducting dynamic pasteurization (low temperature-long-time cooking) studies to assess the integrated lethality of cooking, and develop integrated predictive models for pathogens for the thermal inactivation, injury, repair, and behavior in ready-to-eat meats.

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