

Applying a Path-Dependent Model for *Salmonella* Thermal Inactivation in Slow-Cooked Turkey and Beef Products

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ABSTRACT

Thermal process lethality is currently determined in industry using models that predict inactivation rate based solely on the present state of the food product. However, it is known that sub-lethal injury, which might occur during slow heating processes, can increase the thermal resistance of bacteria. The objective of this project was to estimate the parameters for a modified 1st-order-Arrhenius inactivation model, which accounts for the effect of prior sub-lethal heating on subsequent inactivation rates, and to test the model against *Salmonella* inactivation in meat products cooked in a moist-air convection oven.

Irradiated (>10 kGy) ground turkey breast and beef round were inoculated with an 8-serovar *Salmonella* cocktail, and 1-g samples were subjected to 45 different non-isothermal heating profiles in a thermocycler to target a series of lethalties between 6 and 15 log reductions according to the traditional state-dependent model (in triplicate). Each heating profile consisted of a randomly selected combination of a linear heating rate (1, 2, 3, 4, or 7 K/min), a variable-length sub-lethal holding period (40, 45, or 50°C), and a final holding temperature (55, 58, 61, or 64°C).

Survivors were enumerated on aerobic Petrifilm™. Sub-lethal injury (τ) was quantified as an integral function of the temperature profile between 38 and 52°C. This is the range where *Salmonella* appears to increase its thermal resistance, and thus what renders traditional path-dependent models less effective.

The three parameters of the new model were estimated using a random selection of 30 heating profiles ($n=3 \times 30$) via non-linear regression of computed vs. experimental survivor data. The resulting parameters were then tested against the remaining 15 heating profiles ($n=3 \times 15$) to verify the predicting ability of the new model.

Additionally, 25-g, inoculated samples ($n=9$) of the same products were cooked in a computer-controlled, laboratory-scale, moist-air convection oven, with variable cooking schedules (30-120 min) to a target 7.0 or 6.5 log reductions for turkey and beef, respectively.

The new model parameters were also applied to the oven-cooked samples. In addition, the sole parameter that accounts for τ was estimated from the 25-g data with the same computing methods as above to compare against the state-dependent model.

The error between the traditional, state-dependent model predictions and experimental lethality increased ($P<0.05$) with τ , with fail-dangerous deviations as high as 8 and 7 log CFU/g for turkey and beef, respectively, in the 1-g samples. However, the systematic error with τ was eliminated ($\alpha=0.05$) when the new model was applied. The root mean squared error in the products decreased from approximately 9.5 log to 1.15 log. For the oven-cooked product, the traditional model significantly ($P<0.05$) over-predicted *Salmonella* lethality, with errors as high as 3 log reductions increasing with τ . However, when the new parameter was applied to the same data, the root mean squared error was reduced from approximately 3.55 log to 1.72 log.

The results show that significant process lethality errors can occur in slow cooked meat products, which could jeopardize product safety; however, a new, path-dependent inactivation model eliminated the systematic error.