Comparative Risk Assessment for Intact (Non-Tenderized) and Non-Intact (Tenderized) Beef: Technical Report

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FSIS

1. INTRODUCTION, HISTORY OF THE PROBLEM, AND BACKGROUND INFORMATION ON STEAK AND ROAST PRODUCTION

E. coli O157:H7, a Shiga toxin-producing *E. coli*, is a recognized human pathogen. A draft risk assessment of the public health impact from *E. coli* O157:H7 in ground beef (ECRA, 2001) is currently under review by the public and the National Academies of Sciences. That assessment quantifies the risk to public health from *E. coli* O157:H7 in ground beef.

Since 1994, FSIS has deemed ground beef contaminated with *E. coli* O157:H7 to be adulterated. Yet, the National Advisory Committee on Microbiologic Criteria for Foods (NACMCF) determined in 1997 that "due to the low probability of pathogenic organisms being present in or migrating from the external surface to the interior of beef muscle, cuts of intact muscle (steaks) should be safe if the external surfaces are exposed to temperatures sufficient to effect a cooked color change." Therefore, intact steaks and roasts contaminated with *E. coli* O157:H7 have not been deemed adulterated.

On January 19, 1999, FSIS published a Federal Register notice explaining that, in addition to ground beef, raw non-intact beef products (e.g., beef that has been mechanically tenderized by needling or cubing) that are found to be contaminated with *E. coli O157:H7* must be processed into ready-to-eat product, or they would be deemed adulterated.

The physical process of tenderization is an important reason for FSIS concern. Mechanical tenderization translocates dangerous pathogens from the surface of intact beef cuts to beneath the surface thereby potentially shielding those pathogens from the lethal effects of heat during cooking.

1.1. Process flow for Steak Production

After cattle are slaughtered, their carcasses are processed and usually chilled. Eventually, the carcasses undergo a fabrication step wherein primal and sub-primal cuts of meat are generated. Fabrication is a process that involves the use of knives to trim carcasses into smaller subunits of muscle tissue. The first division of the carcass is into primal cuts. These cuts are further subdivided into subprimal cuts (Figure TR 1 and Figure TR 2). Fat, defects, and most bones are removed during trimming. Byproducts of fabrication are beef trimmings that are used to make ground beef. A single serving of ground beef is a mixture of trim from many carcasses. In contrast, a single serving of steak or roast is from a single carcass.

Following fabrication, subprimal cuts can be mechanically tenderized. Tenderization involves repeated penetration of the muscle surface to disrupt muscle fibers and render the muscle more tender. The effect of these physical tenderization processes is not visible to the naked eye. Following tenderization, the subprimal piece is usually further cut into steaks or roasts.

Tenderization can move pathogens on the surfaces of subprimal cuts to beneath the surfaces. Cutting steaks from a subprimal can also move pathogens from the surface to the freshly cut surface. If we consider a tenderized steak that is a cross-section of a subprimal piece, then the edges of the steak may be contaminated from the original contamination on the surface of the subprimal, the steak's surface may be contaminated from the knife cutting to create the steak, and the interior may be contaminated from the tenderization process.

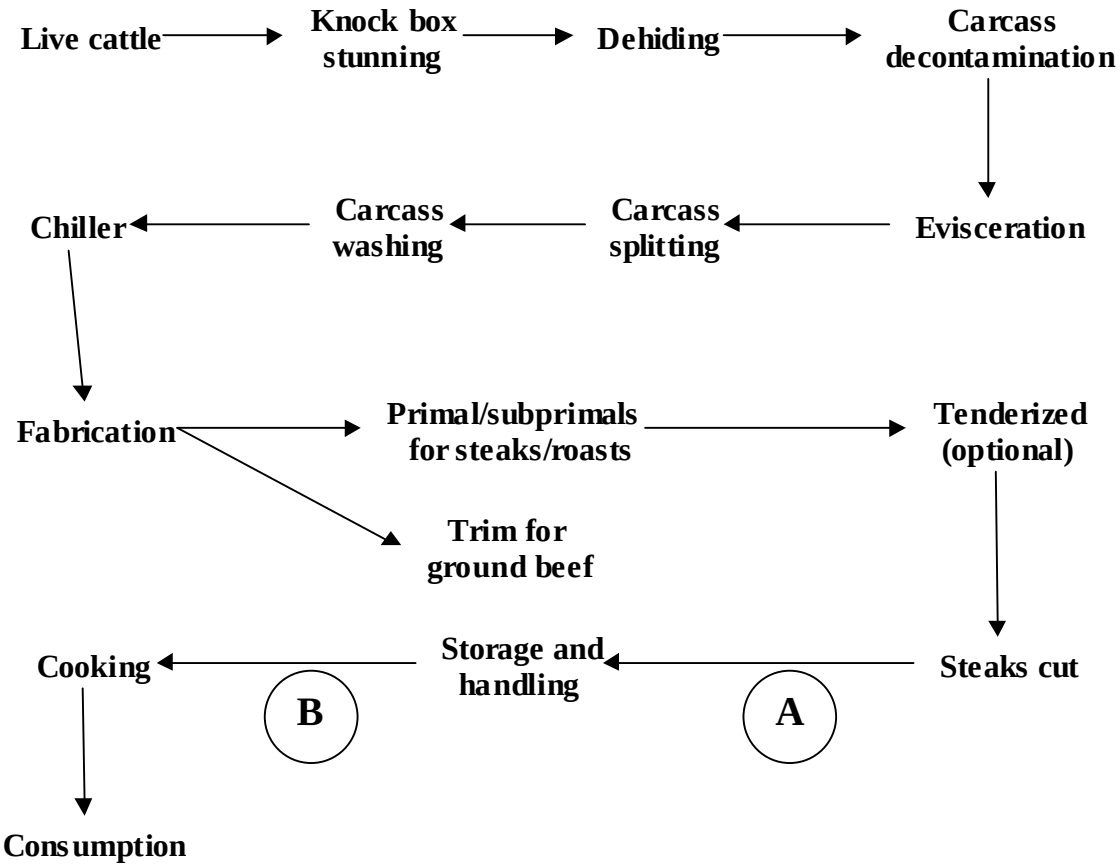

Figure TR 1. Flow diagram of steaks and roasts from slaughter to consumption.

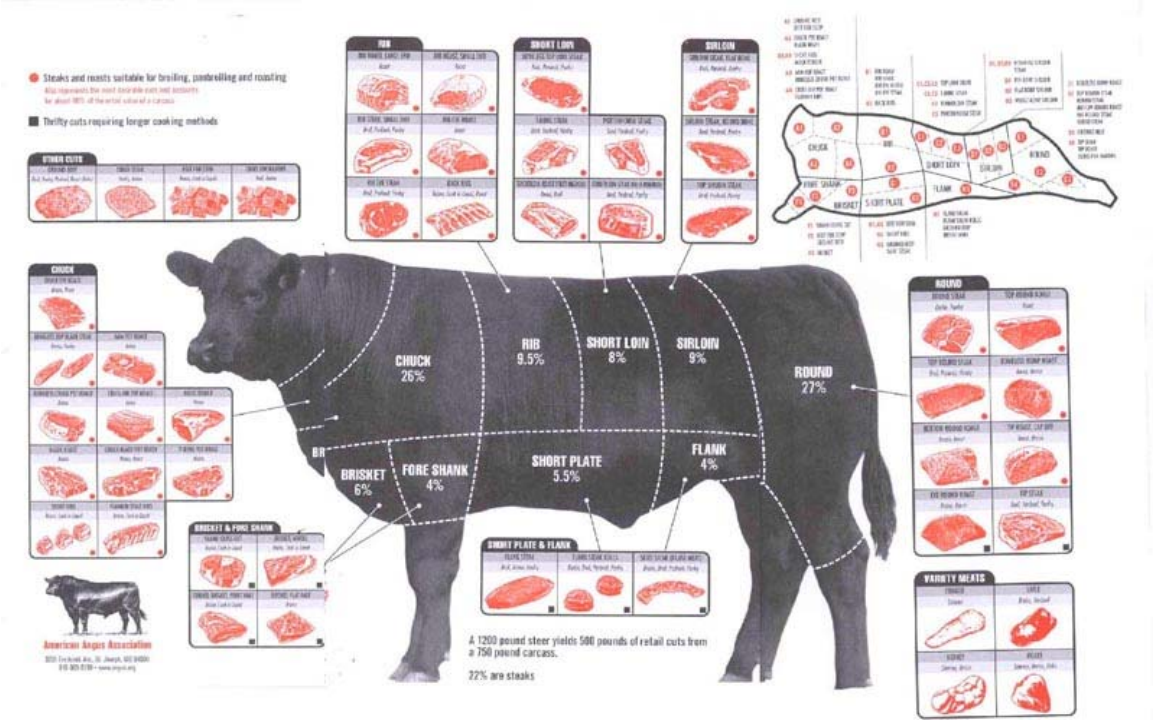

Figure TR 2. American Angus Association's description of beef cuts.

1.2. Mechanical Tenderization

Mechanical tenderization is performed using stainless steel blades or needles. The needles or blades are capable of penetrating the meat by cutting through muscle tissues and fibers, rather than tearing the tissue or punching holes. Blade tenderization refers to a series of sharp stainless steel, double-edged blades, 3 mm in width, that pierce the meat. The blades are oriented at right angles with each other in a pattern of 5 blades per square centimeter (Ross Industries, 1998, used in Sporing, 1999). This results in a distance of about 5 mm between parallel blades.

One type of needle tenderization uses 3mm diameter stainless-steel needles to pierce a subprimal in a similar manner to the blades. The distance between needles is 5 mm. Two heads of needles alternately pierce a subprimal piece of beef. The heads are mounted at a 45° angle to either side of a plumb line. A conveyor belt advances the subprimal 38mm (1.5 inches) between each stroke of the tenderizer. Each needle head contains 268 needles on a surface that is 76mm by 280mm (3 by 11 inches). Therefore, 1.26 needles puncture each cm^2 of meat surface each time the needle head strikes. It is estimated that each piece of meat is pierced about 3 times.

1.3. The Mechanism of Cross-contamination

While blade or needle tenderization can internally contaminate a subprimal whose surface is contaminated, those processes may also cross-contaminate subprimals that are not contaminated. As a blade or needle passes through a contaminated surface, *E. coli* O157:H7 is carried deeper into the muscle tissue. Presumably, there is some initial transfer of surface *E. coli* O157:H7 onto the blade followed by a transfer from the blade to the internal muscle tissues. In work done by Sporing (1999), blade tenderization carried *E. coli* O157:H7 from the top surface to a depth of 6 cm. Yet, the amount of *E. coli* O157:H7 translocated decreased with increasing depth of penetration. For each cm of penetration from the inoculated surface, there was about a 0.5 log decrease in the amount of *E. coli* O157:H7 detected. If organisms are transferred from the blade to muscle from a frictional mechanism that is constant throughout the muscle tissue, then the lower numbers of organisms at greater depths are best explained as a result of fewer organisms available on the blade (i.e., with increasing depth there are fewer organisms to transfer). If true, this mechanism suggests there are few organisms remaining on a blade after it is used on a contaminated subprimal. Furthermore, when that blade encounters the next subprimal, the same mechanism suggests that most organisms will be deposited near the surface of that subprimal. Consequently, cross-contamination may be a less hazardous outcome from tenderization than the direct effect of translocating *E. coli* O157:H7 beneath the surface of a contaminated cut of meat.

1.4. Consumption

Given that most steaks and roasts consumed in the U.S. originate from steer/heifer carcasses, we can get a rough estimate of the total number of such servings annually consumed. Assume that roughly 27 million steers and heifers are slaughtered annually. A typical carcass is 750 lbs. but 30% of that weight is fat, bone, and shrinkage. So, there are about 500 lbs. available for retail cuts of beef. Roughly one-third of retail cuts are in the form of ground beef and stew meat. The remaining two-thirds are equally divided between steaks and roasts. Therefore, about 330 lbs. of steaks and roasts are harvested from such a typical carcass. If we assume that a typical serving size is about a quarter pound across the entire population of consumers, then there are about 36 billion servings of steak and roast consumed annually (i.e., 27 million carcasses X 330 lbs./carcass X 4 servings/pound).

1.5. Roasts versus steaks

Roasts and steaks are cut from the same primal cuts of beef. Roasts are multiple serving cuts, while steaks are usually fabricated to be single servings. Roasts are usually thicker than steaks when cooked. Thickness can influence the rate at which cooking kills *E. coli* O157:H7 inside muscle tissue. Yet, because of their thickness, roasts are usually cooked for a longer time than steaks. A conclusion from a cooking study (Sporing 1999) was that thicker cuts of steak (3.2 cm vs. 1.3 cm) resulted in more effective log reductions when cooked to the same target internal temperature. The time needed to achieve a target internal temperature necessarily increased with thickness. Longer cooking times subjected the more superficial layers of the roast to higher temperatures for longer periods. Greater log reductions in contamination resulted.

Three cooking methods were evaluated by Sporing (1999): broiling, grilling, and skillet frying. These are typical methods for cooking steaks. Nevertheless, roasts are usually cooked by braising or roasting. Of the three methods evaluated by Sporing (1999), broiling seems to be the best surrogate for braising or roasting. As will be discussed later, both skillet frying and grilling were shown to be less effective cooking methods for eliminating *E. coli* O157:H7 bacteria than broiling. Therefore, we will focus on the hazard of tenderization for steaks but assume that this hazard is similar, or possibly less, for roasts.

1.6. Purpose

The purpose of the analysis to be described below is to compare the risk to human health from *E. coli* O157:H7 in blade-tenderized steaks to that from *E. coli* O157:H7 in steaks that have not been tenderized. We do this by calculating the difference in the frequency of human illness caused by tenderized and non tenderized steaks. We assume that the risk from tenderized roasts is similar to, or less than, the risk from steaks. We also assume that blade tenderization is a valid surrogate for any mechanical tenderizer.

Non-tenderized steaks are considered 'safe' if cooked to effect a color change. Therefore, if such steaks are contaminated with *E. coli* O157:H7, they are not considered adulterated. The concern with tenderized steaks is that they may not be safe in comparison to non-tenderized steaks. This analysis should be useful in policy-making and serve as a tool for measuring the potential public health benefits of regulatory options regarding mechanical tenderization (including the option not to regulate).

2. ASSUMPTIONS AND METHODS

The model to be described below is based on the following modeling assumptions:

a) Meat is normally sterile beneath exposed surfaces, but some surface contamination with pathogens can occur. Therefore, intact beef (i.e., non-tenderized) can be contaminated on the exposed surfaces with *E. coli* O157:H7, but such contamination does not occur beneath the surface. During storage and handling of meat, *E. coli* O157:H7 levels can increase. Growth of *E. coli* O157:H7 on the surface or beneath the surface of steaks is similar. Assuming no differential growth rates allows growth of *E. coli* O157:H7 to be modeled as independent of the tenderization step.

b) Lethality of cooking is a function of the cooking temperature, method of cooking, time of cooking, and thickness of the meat. Lethality can be measured in log reductions of *E. coli* O157:H7 achieved. Increased cooking temperature always increases lethality if all other factors remain unchanged. Furthermore, there exists a temperature at which an infinite log reduction occurs (i.e. at which all bacteria are killed).

c) One *E. coli* O157:H7 organism contains all that is necessary to result in infection and illness if consumed. The likelihood of infection and illness is dose-dependent.

d) The results of blade tenderization experiments by Sporing (1999) reasonably apply to other methods of mechanical tenderization.

e) The practice of cooking roasts is reasonably similar to cooking of steaks.

f) The level of translocation noted by Sporing (1999) (i.e., ~4% of surface *E. coli* O157:H7), is reasonably indicative of the results of commercial tenderization.

g) The effect of cooking on specific thicknesses of steaks is similar to the effect of cooking on similar thicknesses. Thus, thicknesses of 1.3 cm, 1.9 cm, and 3.2 cm represent all steak thicknesses.

h) Not-tenderized and tenderized steaks are all stored, prepared, and cooked in identical conditions. This assumption allows focusing on the specific differences in risk between not-tenderized and tenderized steaks.

The following sections describe, using illustrative examples and minimal mathematical notation, the 9 steps involved in the analytical framework. These steps involve discussions on:

1. The issue described
2. The computation process
3. Bacteria in servings before cooking.
4. The attenuation factor
5. Bacteria in servings after cooking
6. Uncertainty about variability
7. Cooking attenuation diagrams
8. Cooking input/output relationship
9. The Dose response relationship

2.1. The issue described

The majority of steaks and roasts destined for hotel, restaurant, and institutional use in the United States may be subjected to "mechanical tenderization." This is a process in which large pieces of meat are penetrated, usually in several directions, by sets of needles, or double-edged blades, and then cut into steaks and roasts. The purpose of the process is to break up the meat's structure and make it more tender. Sometimes the needles used are hollow, so that through the holes the meat can be injected with solution containing flavorings and/or digestive agents such as papain. Sometimes the injected solution is recycled. There are three items of concern in connection with this process.

1. If there are bacteria (especially *E. coli* O157:H7) on the surface of the meat, the tenderizing process may transfer some of these organisms to the interior of the meat where they may be shielded to some extent from the heat of the cooking process. Thus some of these may survive a cooking process that would kill all bacteria on the surface.

2. Unless the needles are sanitized after each piece of meat they could carry bacteria from a contaminated piece to subsequent piece that were previously uncontaminated.

3. If the injected fluid is recycled, this constitutes another mechanism by which bacteria from a contaminated piece could be transferred to previously uncontaminated piece.

If we envision a series of meat pieces coming down a conveyor to the tenderizer then some fraction of those pieces would be contaminated and some not. The effect of items (2) and (3) then would be that in the series of pieces downstream of the tenderizer the fraction contaminated would be somewhat increased. Most worrisome, however, is item (1) because it suggests that some of the bacteria in the final product may survive the cooking process, and may thus cause illness in the consumer.

The purpose of this report is to compare the risk of *E. coli* O157:H7 illness from steak servings that have been prepared from tenderized product to the risk from product that has not been tenderized. For this purpose we adopt the following computational process:

Degree of contamination before cooking.

This is expressed by a variability histogram of BPS_{BC} (bacteria per serving before cooking) (Figure TR 3)

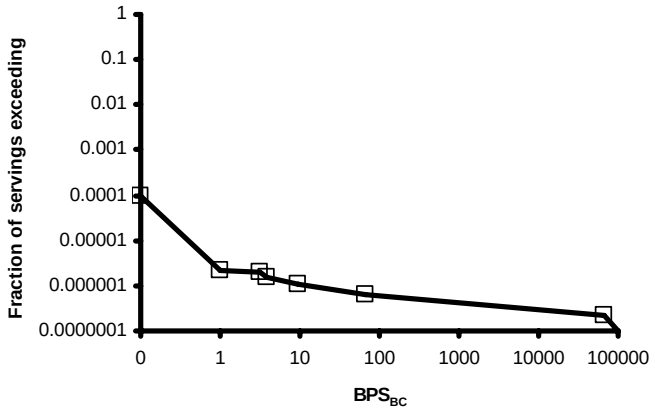

Figure TR 3. Diagram of the variability in *E. coli* O157:H7 organisms per serving before cooking (BPS_{BC}). This is an exceedance diagram and shows the fraction of servings exceeding the BPS_{BC}.

2.1.1. Degree of attenuation during cooking

This is expressed by Attenuation Factor diagrams, with and without tenderizing (Figure TR 4).

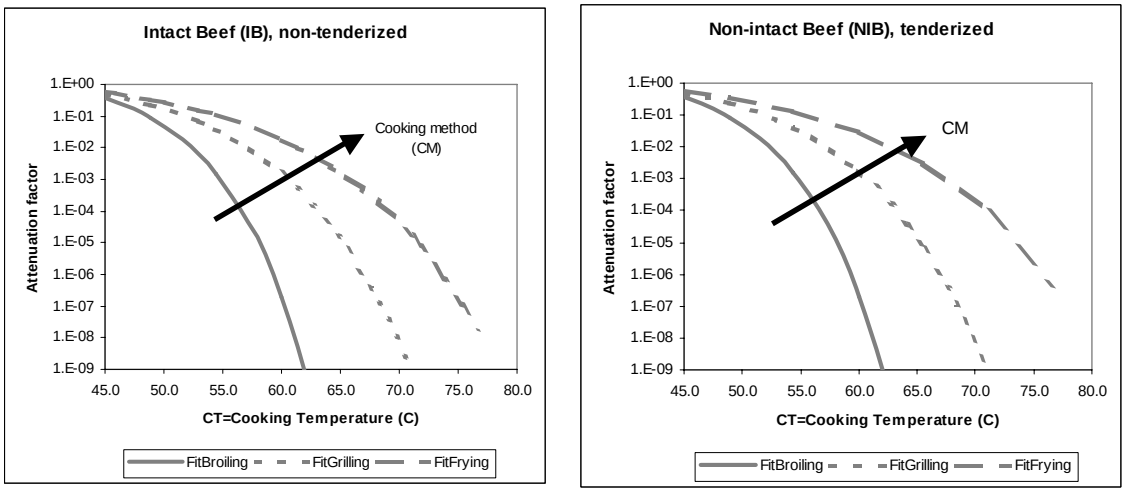

Figure TR 4. Example attenuation factor diagrams for beef that is not-tenderized or tenderized, and cooked by broiling, grilling, or frying

2.1.2. Degree of contamination after cooking

Contamination after cooking is calculated by multiplication

eqn (1) (bacteria per serving after cooking) = BPS_{AC}= BPS_{BC} x AF

where AF (attenuation factor) is a function of:

1. CT, cooking temperature
2. CM, cooking method
3. ST, steak thickness
4. T or NT, intact or non-intact

2.1.3. Illnesses resulting from that contamination.

BPS_{AC} can be converted to Illnesses per Serving (IPS) by passing BPS_{AC} through the Dose-Response Curve [DRC(dose)].

eqn (2) IPS = DRC(BPS_{BC} x AF(CM, CT, ST, T or NT))

The effect of tenderization is shown by changing the variable T to NT in this equation.

2.2. Computation process

For point estimate:

Choose BPS_{BC}, CM, CT, ST, T or NT

Find AF(CM, CT, ST, T or NT) from the attenuation curves.

Calculate $BPS_{AC} = BPS_{BC} \times AF$

Calculate $IPS = DRC(BPS_{AC})$

Our output quantity, IPS, is thus a function of five variables:

BPS_{BC} (input variable)

CM (parameter)

CT (parameter)

ST (parameter)

T or NT (parameter)

The cooking effect for T or NT is shown by comparing the two sets of curves in section 2.1.2.

2.3. Bacteria per serving before cooking (BPS_{BC})

The following conceptual model reflects the relevant stages in the farm-to-table process where *E. coli* O157:H7 is present (and could potentially grow) in tenderized and nontenderized beef products (Figure TR 5).

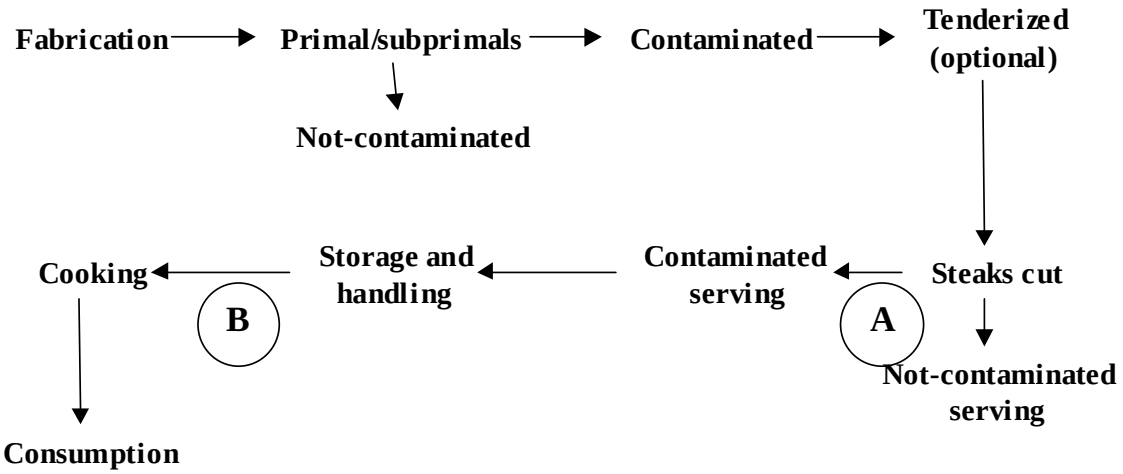

Figure TR 5. Conceptual model of *E. coli* O157:H7 contamination on steaks.

This conceptual model describes the movement of product from the fabrication step in slaughter through the generation of primal and subprimal cuts of beef, tenderization, creation of steak servings, storage and handling, cooking, and consumption. Ideally, we could describe input/output relationships at each of the steps shown here, but currently, there is no sampling

data on the prevalence and levels of *E. coli* O157:H7 on, or in, steaks and roasts. Therefore, we have chosen to begin our consideration of the bacteria on servings at point A in this figure.

Assuming we know the number of bacteria on steaks at Point A, then Point B describes the output from a series of steps wherein steaks are transported, stored, and prepared prior to cooking. Each step of storage and handling (e.g., storage prior to shipment, storage at retail, etc.) can be characterized by the relationship between the BPS at the input to the step and the BPS at the output. If storage and handling of steaks is properly done, the BPS_{out} equals the BPS_{in} (i.e., there is no growth of *E. coli* O157:H7). If time and temperature conditions are such as to enable *E. coli* O157:H7 to grow, then $BPS_{out} > BPS_{in}$. The growth factor, G, between points A and B, is defined as;

eqn (3) $$G = \frac{\text{BPS at point B}}{\text{BPS at point A}} = \frac{BPS_{out}}{BPS_{in}}$$

where G is a function of storage time, storage temperature, and growth parameters specific to *E. coli* O157:H7. If we collapse all the steps comprising point B into a single node, we can generate a diagram for this input/output stage (Figure TR 6).

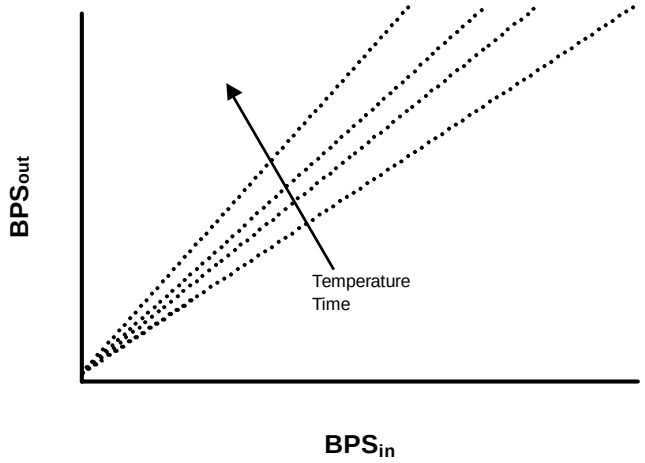

Figure TR 6. Example of input-output diagram for bacteria per serving at a single node.

Because storage time and temperature are variable among the population, then so also the growth ratio G is similarly variable. We describe this variability with a "Discrete Frequency Distribution" (Kaplan 1981, Kaplan 1987) for the growth parameter, G

eqn (4) $G = \{<FGi; Gi>\}$.

Here FGi is the fraction of servings that experience growth factor Gi.

Now define:

BPS_{in} = the number of bacteria on steaks at point A,
BPS_{out} = the number of bacteria on steaks at point B, just before cooking (i.e., this is also BPS_{BC}).

Since the quantity BPS_{in} A is variable we describe that variability with a discrete frequency distribution:

eqn (5) $BPSin = \{< FBPS_{in,\,k}; BPS_{in,\,k} >\}$

Therefore, from eqn (4) eqn (5) ,

eqn (6) $BPS_{BC} = \{<FBPS_{BCk,i}, BPS_{BCk,i} >\} = \{< FBPS_{in,k} \text{ x } FG_i ; BPS_{in,k} \text{ x} G_i >\}$

Figure TR 3 shows the curve for BPS_{BC} calculated according to eqn (6) .

2.4. Attenuation factor, AF

The factor AF defines the I/O relationship for cooking. That is, it relates BPS_{BC} to BPS_{AC} according to the relationship:.

eqn (7) $BPS_{AC} = BPS_{BC} \text{ x } AF$

AF is a function of four parameters: the cooking temperature, CT, cooking method, CM, Steak thickness, T, and tenderized or not, T/NT.

eqn (8) AF = AF(CT, CM, T, T/NT).

So, given values for each of the four parameters we can calculate an AF and using this in eqn (7) we can calculate a point estimate because,

eqn (9) $BPS_{BC} \rightarrow BPS_{AC} \rightarrow$ DRC $\rightarrow$ Illnesses per serving (IPS)

Thus, given an input BPS_{BC}, we can calculate IPS as a function of CT.

2.5. Uncertainty about a variability distribution

As an example of a variability distribution in the model we consider BPS_{BC}. This variability in the in the level of contamination in the incoming steak population (i.e. before cooking) was expressed as a curve (Figure TR 3). Call this curve V. To express our uncertainty about this variability curve, V, we use a discrete probability density format.

eqn (10) $V = \{ <V_i, P_i>\}$

i.e., put forth a set , $\{V_i\}$, of possible V curves and assign each a probability, P_i , such that the sum of all P_i is one.

For simplicity we select three possible curves for V. We choose V_2 as the "most likely" curve that represents our best guess about the true variability of bacteria per serving in the population of steaks under consideration. We chooseV_1 as a "lower-bound" curve that is shifted leftward from V_2, expressing a general reduction of bacteria per serving in the population. Similarly we choose V_3 as an "upper bound" curve that suggests a higher frequency of larger numbers of bacteria per serving. We next assign probability values to these curves, for example $P_1 = P_3 = 0.1$ and $P_2 = 0.8$ (for a more detailed treatment, if desired, we could include more curves for V that lie between V_1 and V_3, but the three curves described are a simple approach that communicates the degree of uncertainty).

Now we can calculate a new IPS with each of these curves as inputs for BPS_{AC}. The range of results so calculated describes our uncertainty in IPS given our uncertainty about V. This analysis is conditioned on the other model inputs being set to their most likely values.

We can perform similar analyses for other uncertain inputs to the model. For example, the curves for AF(CM, ST, CT) can be represented as sets of curves to represent our uncertainty in the parameter AF.

2.6. Cooking attenuation

Thus far, we have generated diagrams such as in Figure TR 4 to express the effect of cooking.

We have generated such diagrams for each combination of steak thickness and tenderized or non-tenderized. The attenuation factor, AF, is defined as;

eqn (11) $$AF = \frac{BPS_{AC}}{BPS_{BC}}$$

If we know the frequency of the various cooking methods (CM) and cooking temperatures (CT), then we can condense the attenuation diagrams to a single variability curve for AF.

Denote:

eqn (12) FCM = {<FCMF, frying>, <FCMG, grilling>, <FCMB, broiling>}

where:

FCMF is the fraction of steaks that are fried,
FCMG is the fraction of steaks that are grilled,
FCMB is the fraction of steaks that are broiled,
and FCMF + FCMG + FCMB = 1.0

Denote:

eqn (13) $FCT = \{<FCT_i, CT_i>\}$

where:

FCT_i is the fraction of steaks cooked at temperature CT_i, and $\sum_i FCT_i = 1.0$

Then we can calculate $AF(CM_j, CT_i)$ occurs with frequency FCM_j x FCT_i because the pair (CM_j, CT_i) defines a cooking condition and, through the attenuation diagram, an attenuation factor, AF_{ji}.

Thus, AF reduces to the discrete frequency distribution for AF as;

eqn (14) $AF = \{<FAF_{j,i}, AF_{j,i}>\}$

where $FAF_{j,i} = FCM_j$ x FCT_i and $AF_{j,i}$ is the attenuation factor for cooking by method j at temperature CT_i.

This discrete frequency distribution expresses the variability of the attenuation factor AF. If we combine (multiply) this with a variability curve for BPS_{BC}, we will get a variability curve for BPS_{AC}.

2.7. Input/output relationship for cooking

We now have a variability distribution for the contamination level before cooking, eqn (6) . We can combine this discrete frequency distribution with that for AF to get a distribution for contamination level after cooking, eqn (7) . Thus:

eqn (15) $BPS_{AC} = \{<FBPS_{ACn}; BPS_{ACn}>\}$

where $FBPS_{ACn} = FBPS_{BCk}$ x FAF_{ij} and $BPS_{ACn} = BPS_{BCk}$ x AF_{ji}

Therefore, we have a method for converting a variability curve for BPS_{BC} to a variability curve for BPS_{AC}. This curve includes the variability of cooking temperature and cooking method. We can extend this method to also include the variability of steak thickness.

2.8. Putting the BPS_{AC} curve through a dose response relationship

eqn (15) gives us a discrete frequency distribution for bacteria per serving after cooking. If we pass each value of BPS_{ACn} through a dose response curve (Figure TR 7), we get a corresponding illness per serving, IPS_n. So, passing a whole discrete frequency distribution, BPS_{AC}, though the dose-response curve, we get a discrete frequency distribution for IPS.

eqn (16) $IPS = \{< FBPS_{ACn} ; IPS(BPS_{ACn})>\}$.

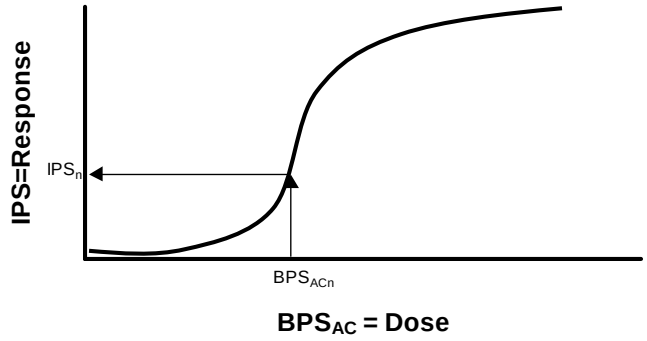

Figure TR 7. Dose response relationship

The IPS curve can be used as to summarize the results of this study. Thus, we can calculate the expected value of this curve to determine the expected illnesses per serving (EIPS) for steaks that are tenderized or not tenderized.

eqn (17) $IPS_{EV} = EV\{DRC(BPS_{AC})\} =$

$$\sum_{n} DRC\left(BPS_{ACn}\right) \times FBPS_{ACn}$$

3. Model Inputs

3.1. Organisms per serving before cooking, BPS_{BC}

Currently, there is no sampling data on the prevalence and levels of *E. coli* O157:H7 on, or in, steaks and roasts. To motivate an estimate, therefore, consider the conceptual model shown in Figure TR 5. This conceptual model describes the movement of product from the fabrication step in slaughter through the generation of primal and subprimal cuts of beef, tenderization, creation of steak servings, storage and handling, cooking, and consumption. Ideally, we could describe input/output relationships at each of the steps shown here, but we lack the data. Therefore, as noted before, we have chosen to begin our consideration of the organisms on servings at point A in this figure. We call this variable BPS_{in}, eqn (5) .

3.1.1. Initial organisms on serving before storage and handling, BPS_{in}

As a surrogate for the number of organisms on a steak just after it is produced (BPS_{in}), we modify the prediction from a risk assessment of *E. coli* O157:H7 in ground beef (ECRA 2001). Those predictions must be modified because ground beef is far from an ideal surrogate for steaks or roasts. Ground beef is generated from beef trim that typically comes from the surfaces of multiple carcasses. Given that *E. coli* O157:H7 resides on the surface of carcasses, it is likely that ground beef would be more contaminated than the primal/subprimal cuts of intact beef generated during fabrication. For example, a survey of raw ground beef found the average

density of generic *E. coli* (Biotype I) was 1.73 logs per gram (FSIS, 1996). Jericho (2000) enumerated generic *E. coli* counts on various cuts of beef just before, and soon after, commercial packaging in a large slaughter plant in Canada (Table TR 1). The average density of generic *E. coli* found on these cuts was 0.53 logs per cm^2. This comparison suggests that levels of bacteria on steaks and roasts are generally less than levels in ground beef.

Table TR 1. Generic *E. coli* results from sampling subprimal cuts of beef in a Canadian slaughter plant (Jericho 2000).

Cut of meat	**Average Log(E. coli)/cm^2**	**Standard error (16 *df*)**
BCB – beef chuck boneless	0.36	0.11
SCC – short cut clod	0.07	0.11
BBB – beef brisket boneless	0.80	0.18
SRT – short ribs full cut	0.03	0.18
OSB – outside skirts boneless	0.11	0.09
RBE – round boneless-eye	0.33	0.09
SB – sirloin boneless	1.62	0.16
TB – tenderloin boneless	0.53	0.16
CBP – chuck boneless pectoral	0.16	0.16
RBC – round boneless center bone out	1.42	0.16
RBB – ribs boneless blade	0.02	0.09
RBP – round boneless peeled	0.96	0.09

Table TR 1 implies that levels of generic *E. coli* are relatively low on subprimal cuts of beef. Nevertheless, these data do not specifically address *E. coli* O157:H7. Therefore, we need an alternative approach to estimate BPS_{in}. Our best estimate of the levels of *E. coli* O157:H7 in ground beef comes from the draft risk assessment of *E. coli* O157:H7 (ECRA 2001). That risk assessment predicts:

- between 0.2% and 0.5% of ground beef servings are contaminated with one or more *E. coli* O157:H7 (Table TR 2),
- 75% of a steer/heifer carcasses' original surface area becomes beef trim used to make ground beef,
- 25% of the original surface area's *E. coli* O157:H7 remain to contaminate primal cuts of beef,
- the remaining surface *E. coli* O157:H7 on primal cuts are distributed across about 82% of the weight of the original carcass (i.e., 500 lbs. of beef per carcass and about 18% is trim).

Therefore, to estimate the fraction of contaminated steak/roast servings, we multiply the fraction of contaminated ground beef servings by a steak and roast adjustment factor.

eqn (18)

$$\text{steak \& roast adjustment factor} = \text{SRAF} = \frac{\text{bacteria per steak serving}}{\text{bacteria per ground beef serving}} = \frac{\frac{.25}{.82}}{\frac{.75}{.18}} = .07$$

Table TR 2. *E. coli* O157:H7 levels predicted in ground beef servings before storage and handling from a risk assessment model (ECRA 2001).

Bacteria per serving	**Fraction of servings**	
	Jun-Sep	**Oct-May**
0	99.5%	99.8%
1	0.46%	0.19%
3	0.038%	0.011%
10	0.0035%	0.00040%
31	0.0000027%	0.0000000002%
100	0%	0%

To adjust the ground beef predictions to model steaks and roasts we first determine the fraction of servings that would have no bacteria. We average the seasonal results to determine the annual fraction of servings containing no *E. coli* O157:H7:

eqn (19)

$$F\begin{pmatrix}\text{Ground beef}\\ \text{servings with}\\ \text{0 bacteria}\end{pmatrix} = 0.995 \times \frac{4}{12} + 0.998 \times \frac{8}{12} = 0.997$$

The complement of eqn (19) is the fraction of contaminated servings. This is multiplied by SRAF, eqn (18) to determine the fraction of steaks and roasts that are contaminated. The complement of this is the fraction of steaks and roasts with 0 bacteria.

eqn (20) F(BPSin=0) =1 - (1 – 0.997) x SRAF = 1 - (1 - 0.997) x 0.07 = 0.9998.

Thus, 99.98% of steaks/roasts contain no *E. coli* O157:H7 (i.e., BPS_{in}=0) and 0.02% of steaks/roasts contain one or more *E. coli* O157:H7 (i.e., BPS_{in}>0). For contaminated servings (BPS_{in}>0), the predictions in Table TR 2 show that contamination levels might vary from 1 to 31 bacteria per serving. Yet, servings with 1 bacteria occur >10 times more frequently than servings containing 3 or 10 or 31 bacteria. To simplify the model, we assume BPS_{in} can take only two values; 0 (for 99.98% of servings) or 1 (for 0.02% of servings).

3.1.2. Growth multiplier from storage and handling, G

If environmental conditions (e.g., storage temperatures) support growth of *E. coli* O157:H7, then bacteria in or on steaks/roasts may multiply between the time the steaks/roasts are produced/packaged and the time they are cooked. We have assumed the growth predictions

from the *E. coli* O157:H7 in ground beef model (ECRA 2001) approximately apply to steaks and roasts (Table TR 3). These predictions suggest the following:

- about 50% of servings have no change in the level of *E. coli* O157:H7 per serving,
- about 49% of servings have a reduction in levels of *E. coli* O157:H7 resulting from frozen storage,
- about 1% of servings are predicted to have some growth of *E. coli* O157:H7 contamination during storage and handling.

To simplify the model, we assume freezing eliminates any contamination on a steak or roast.

Table TR 3. Logs of *E. coli* O157:H7 growth in ground beef as predicted by ECRA (2001).

Logs of *E. coli* O157:H7 growth	Fraction of servings
-3.0	0.0136
-2.5	0.0136
-2.0	0.0815
-1.5	0.2852
-1.0	0.0951
0.0	0.4997
0.5	0.0030
1.0	0.0029
1.5	0.0015
2.0	0.0009
2.5	0.0006
3.0	0.0004
3.5	0.0003
4.0	0.0003
4.5	0.0002
5.0	0.0002
5.5	0.0002
6.0	0.0002
6.5	0.0002
7.0	0.0002
7.5	0.0001
8.0	0.0001
8.5	0.0001
9.0	0.0000

For contaminated servings in which *E. coli* O157:H7 grows (~1%), the predictions in Table TR 3 are separated into quintiles and the midpoints of each quintile (i.e., 10^{th}, 30^{th}, 50^{th}, 70^{th}, and 90^{th} percentile) are used to represent the logs of growth (Table TR 4).

Table TR 4. Midpoints of quintiles for logs of growth of *E. coli* O157:H7 in contaminated servings of steaks and roasts during storage and handling (i.e., for G>1, or Log(G)>0).

Percentile	Log(G)
10^{th}	0.500
30^{th}	0.578
50^{th}	0.972
70^{th}	1.827
90^{th}	4.819

3.1.3. Curve for BPS_{BC}

The number of bacteria per serving before growth (BPS_{in}) is multiplied by the amount of growth to determine the bacteria per serving before cooking (BPS_{BC}).

eqn (21) $BPS_{BC} = BPS_{in} \times G$,

The result of eqn (21) was shown in Figure TR 3.

3.1.4. Uncertainty in BPS_{BC}

We are uncertain about our assumptions concerning the derivation of the BPS_{BC}. Specifically, ground beef contamination may not be a reasonable surrogate for steak/roasts, and handling/storage of steaks/roasts may differ from handling/storage of ground beef. Consequently, we model uncertainty about BPS_{BC} using lower and upper bound distributions. To derive these distributions we assume:

BPS_{in}=0 for 99.998% of servings in the lower bound,

BPS_{in}=0 for 99.8% of servings in the upper bound,

the fraction of servings frozen is 80% in the lower bound or 20% in the upper bound,

different quintile values for BPS_{in} and G (Table TR 5) derived from uncertainty in the ground beef model's predictions.

Table TR 5. Assumed uncertainty bounds for quintiles of distributions when BPS_{in}>0 and Log(G)>0.

Percentile	BPS_{in}		Log(G)	
	Lower	Upper	Lower	Upper
10^{th}	1	1	0.250	0.500
30^{th}	1	3	0.285	0.620
50^{th}	1	10	0.479	1.097
70^{th}	1	32	0.883	2.226
90^{th}	1	100	2.242	5.637

3.2. Attenuation factor, AF

3.2.1. Available data

Sporing (1999) reports that the process of blade tenderization translocates 3-4% of the surface bacteria to the interior. She also provides data on cooking effectiveness for non-tenderized and tenderized beef steaks using three cooking methods (skillet frying, grilling, and broiling) and three steak thicknesses (1.3 cm, 1.9 cm, and 3.2 cm). Summaries of the results for each of the three cooking methods appear in Table TR 6.

Table TR 6. Summary of log reductions of *E. coli* O157:H7 for three different cooking methods and three different temperatures for non-tenderized and tenderized steak, Sporing (1999)

Steak Thickness (cm)	Cooking Temperature (C)	Cooking method					
		Broiling (average of 3 replicates)		Grilling (average of 6 replicates)		Skillet frying (average of 3 replicates)	
		Non-tend	Tend	Non-tend	Tend	Non-tend	Tend
1.3	48.9	4.3	2.3				
	54.4	2.7	4.4	3.4	2.4	2.6	3.1
	60.0	5.1	5.5	2.6	3.5	1.2	1.8
	65.6	5.9	6.1	4.2	4.5	3.5	2.7
	71.1	6.0	5.9	5.0	5.6	4.0	3.8
	76.7	6.4	6.1	5.6	4.8	3.7	5.9
1.9	48.9	5.0	3.9				
	54.4	4.7	5.1	3.3	3.5	1.9	0.5
	60.0	5.8	6.5	5.4	4.2	2.0	0.9
	65.6	6.6	6.3	5.6	5.2	2.1	2.3
	71.1	6.6	6.5	5.5	4.5	4.1	4.0
	76.7	6.6	6.5	6.2	5.9	5.0	2.8
3.2	48.9	4.9	2.9				
	54.4	5.1	6.3	5.1	3.8	2.6	3.1
	60.0	6.3	6.3	6.0	4.9	4.4	1.6
	65.6	6.4	6.3	6.0	5.4	5.0	3.7
	71.1	6.4	6.3	5.9	5.1	3.8	2.9
	76.7	6.4	6.3	6.1	5.3	4.0	4.3

Examination of the results suggest that broiling is more effective at killing *E. coli* O157:H7 than grilling which in turn is more effective than skillet frying. Further, there seems to be a suggestion that tenderized steaks are likely to have more *E. coli* O157:H7 remaining after cooking than non-tenderized steaks.

Figure TR 8, Figure TR 9, and Figure TR 10 display the results in Table TR 6. Separate charts are displayed for each thickness of steak and for tenderized and non-tenderized steaks. Within each chart results are separated by cooking method. These charts display the results as

attenuation factors rather than log reductions. A log reduction of 2 is equal to an attenuation factor of 10^{-2} or 0.01. This attenuation factor can then be multiplied by a starting number of organisms to give the number of expected surviving organisms. A smaller attenuation factor is thus associated with a higher log reduction (i.e., more effective cooking).

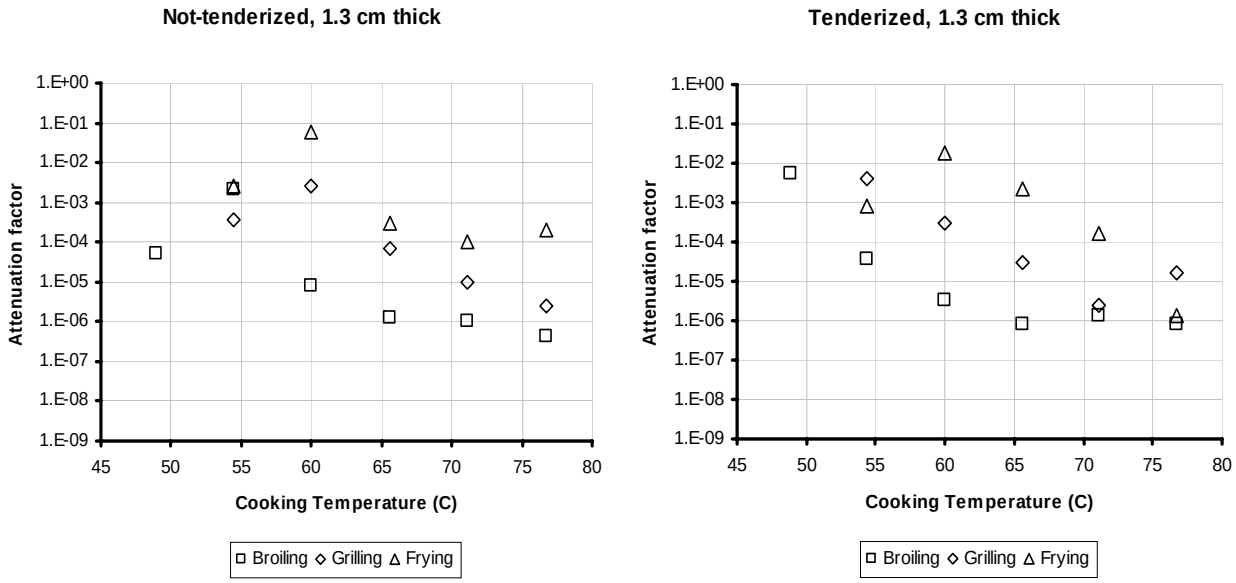

Figure TR 8. Summary of Sporing (1999) data for not-tenderized and tenderized steaks 1.3 cm thick.

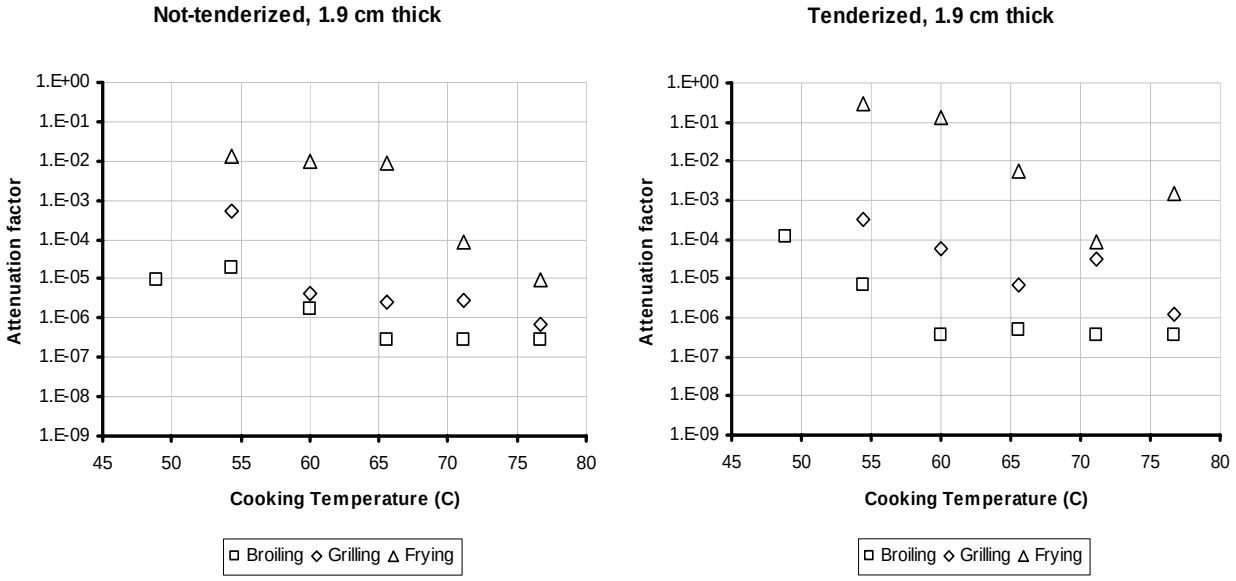

Figure TR 9. Summary of Sporing (1999) data for not-tenderized and tenderized steaks 1.9 cm thick.

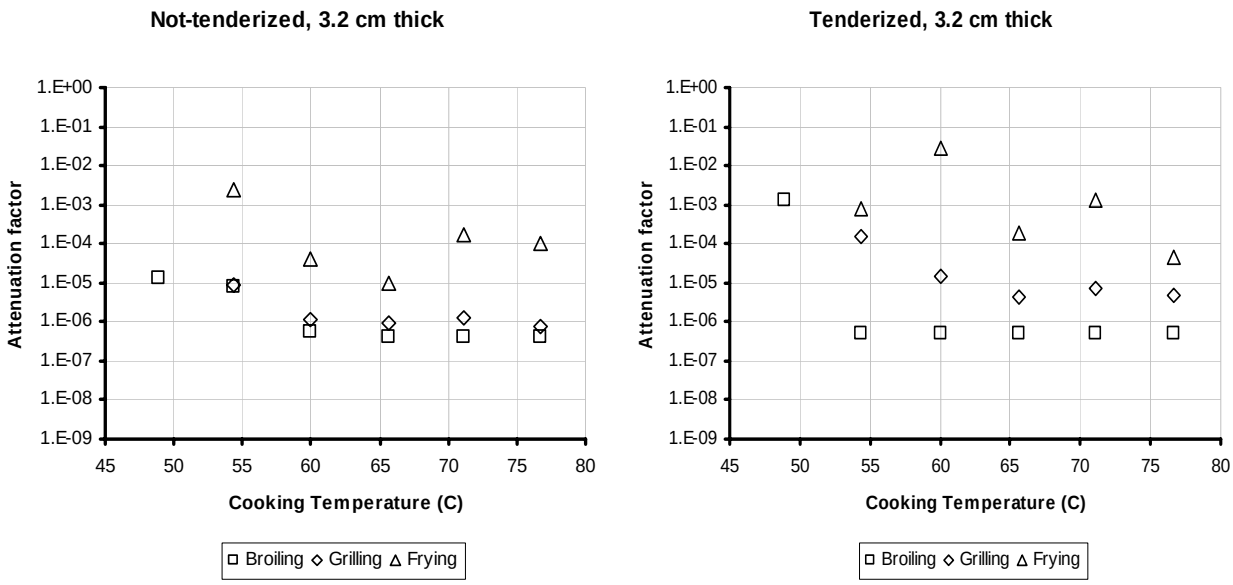

Figure TR 10. Summary of Sporing (1999) data for not-tenderized and tenderized steaks 3.2 cm thick.

3.2.2. Interpreting the data: The Plateau Effect

From the data in Figure TR 8, Figure TR 9, and Figure TR 10, it is clear that at the detailed level, there are many not fully controlled variables in this rather difficult experiment. Nevertheless, taken as a whole, or "gestalt," certain broad conclusions can be drawn. We will attempt to bring these out, in 3.2.3, in the course of fitting smooth curves to the data.

First, however, we note in Figure TR 8, Figure TR 9, and Figure TR 10, a flattening out or "plateauing" effect at higher cooking temperatures, most notably in the broiling curves. This is an artifact of the way the data was plotted and is not to be taken seriously. Indeed, as we shall show in 3.2.3. , rather than flattening out, these cooking attenuation curves should approach vertical asymptotes, expressing the fact that for each cooking method there is a cooking temperature at which all the bacteria are killed[1].

3.2.3. Fitting the Data to Smooth Curves

In this section we shall fit smooth curves to the data. The resulting curves will then constitute in effect a model of the effect of cooking. Our choice of model, then, is based on two fundamental physical/biological assumptions:

[1] Indeed, the fact that the three cooking methods seem to have different vertical asymptotes suggests the presence of another strong uncontrolled variable (perhaps cooking time?) that varies between the cooking methods.

Higher cooking temperatures kill more bacteria. Thus they should result in greater log reductions (lower attenuation factors). The empiric evidence in Table TR 6 often seems to contradict this assumption. For example broiling not-tenderized beef at 48.9 C resulted in a 4.3 log reduction while increasing the temperature to 54.4 C resulted in only a 2.7 log reduction. It seems implausible to suggest that bacteria that are killed at 48.9 C would survive temperatures above that. Thus, such a discrepancy is likely due to variations, errors, or uncontrolled variables within the study design or methods.

There is a temperature at which all bacteria are killed. Bacterial death is often thought of in probabilistic terms. In other words, a 2 log reduction results in an attenuation factor of 0.01 which means that 1% of the starting bacteria are likely to survive. Nevertheless, there must be a temperature at which the probability of survival is zero.

These two assumptions mean that when attenuation factor is plotted against cooking temperature, the resultant curve must have an increasingly negative slope as temperature increases, approaching a vertical asymptote as shown in Figure TR 11.

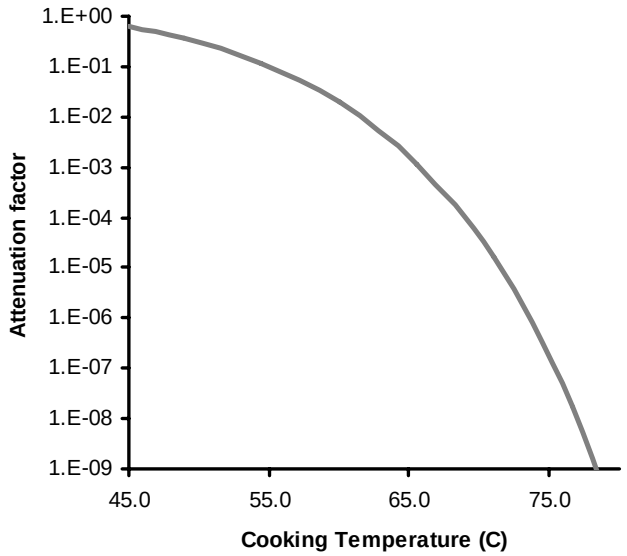

Figure TR 11. Sample attenuation curve

3.2.4. Modeling the attenuation curve

A function was developed to model the attenuation curves while satisfying the two modeling assumptions. Eqn (23) illustrates this function where AF is the attenuation factor, k is a constant, T is the internal temperature, and T_0 is the temperature below which no bacteria are killed**.**

eqn (22) $$Log(AF) = 1 - e^{(k \times (T - T_0))}$$

For all thicknesses and cooking methods T_0 was set at 40 C. We thus assumed that no attenuation would occur at a temperature of 40 C (104 F). Different "k"s were chosen for each thickness, cooking method, and for tenderized and not-tenderized steaks. Curves were selected on a visual judgment of the best fit to the data and discounting the plateau effect. Figure TR 12, Figure TR 13, and Figure TR 14 show the fitted curves for each thickness, cooking method, and tenderization combination.

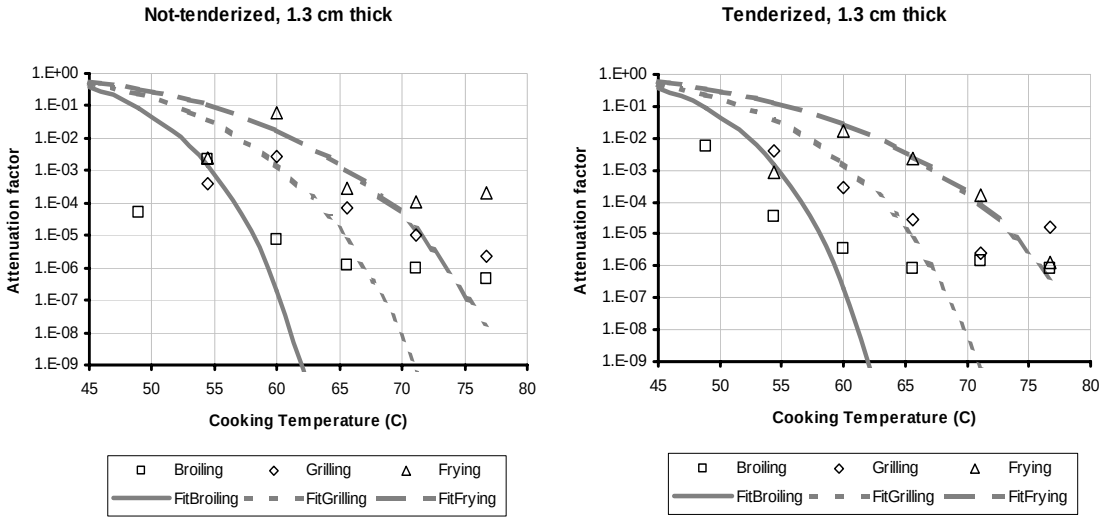

Figure TR 12. Best fit curves to Sporing (1999) data for broiling, grilling, and frying 1.3 cm thick not-tenderized and tenderized steaks.

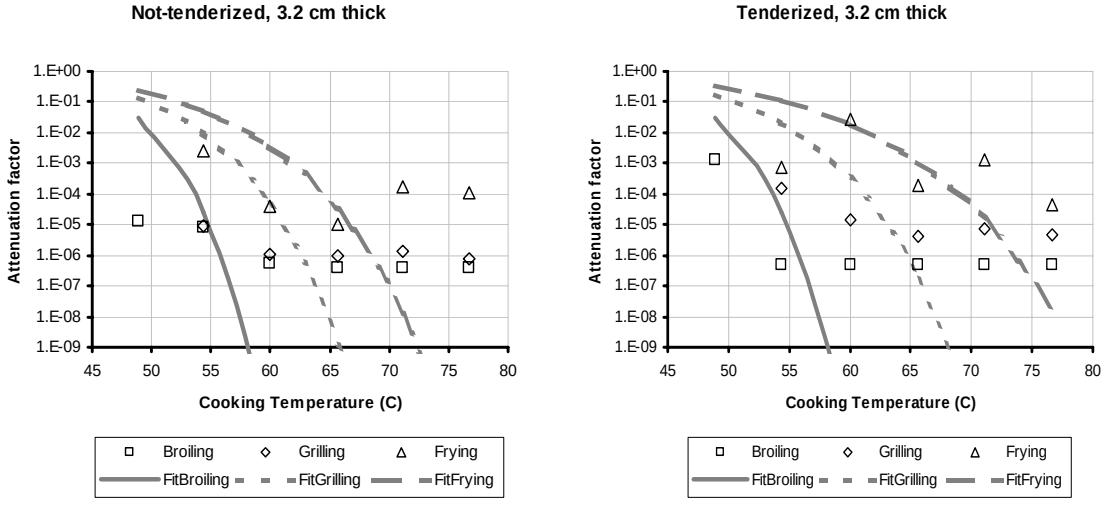

Figure TR 13. Best fit curves to Sporing (1999) data for broiling, grilling, and frying 1.9 cm thick not-tenderized and tenderized steaks.

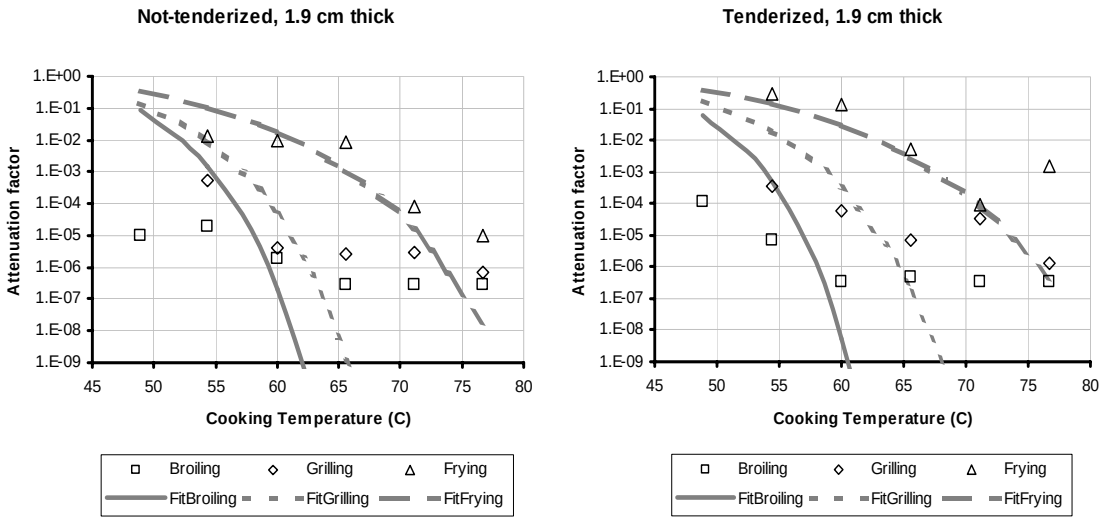

Figure TR 14. Best fit curves to Sporing (1999) data for broiling, grilling, and frying 3.2 cm thick not-tenderized and tenderized steaks.

3.2.5. Inferences From the Fitted Curves Regarding the Effect of Tenderization.

The fitted curves make it a bit easier to see the effect of tenderization after discounting the plateau effect and the other unwanted variabilities present in the raw data. So what is the message of these curves? The curves seem to suggest that tenderization causes a slight movement to the right, meaning less attenuation, and that this effect increases with steak thickness. This movement is consistent with Sporing's observation of 3-4% translocation, and with what one would expect physically. However we emphasize that this is a suggestion only, from the data, and that the variability does not permit a firm conclusion.

3.2.6. Expressing uncertainty in the cooking effect curves

The limited data available as well as the large apparent fluctuations in attenuation factors, for specific thickness and cooking method combinations, suggests that replicate studies could result in quite different results. Consequently, it is important to assign a wide band of uncertainty to the most-likely curves shown in Figure TR 12, Figure TR 13, and Figure TR 14.

The most-likely curves were determined in 3.2.3. by visually fitting curves to the full sets of data points. Upper and lower bounds were determined in this Section by visually fitting curves to envelop all the data points for each thickness and cooking method combination. This resulted in curves that were shifted either to the right (for a lower bound, or least effective [pessimistic] cooking curve) or to the left (for an upper bound, or most effective [optimistic] cooking curve)

of the most likely curve for each thickness and method. These uncertainty curves are depicted in Figure TR 15, Figure TR 16, and Figure TR 17.

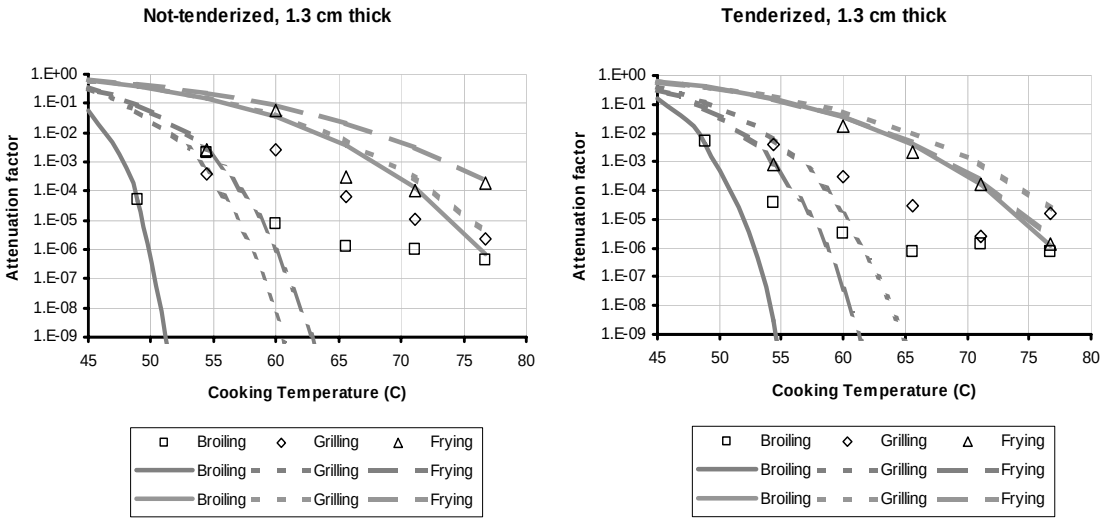

Figure TR 15. Upper and lower bounds for broiling, grilling, and frying for not-tenderized and tenderized steaks 1.3 cm thick.

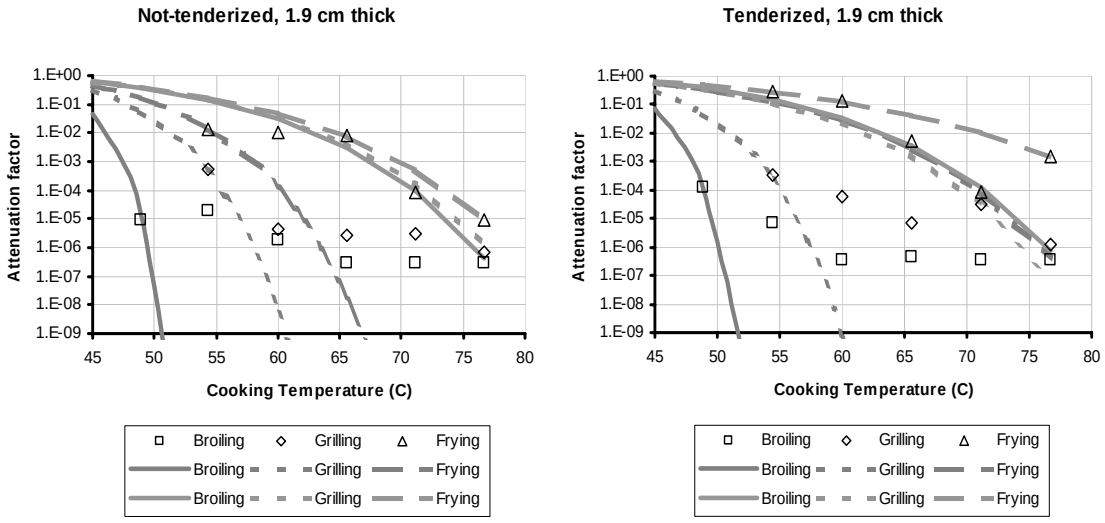

Figure TR 16. Upper and lower bounds for broiling, grilling, and frying for not-tenderized and tenderized steaks 1.9 cm thick.

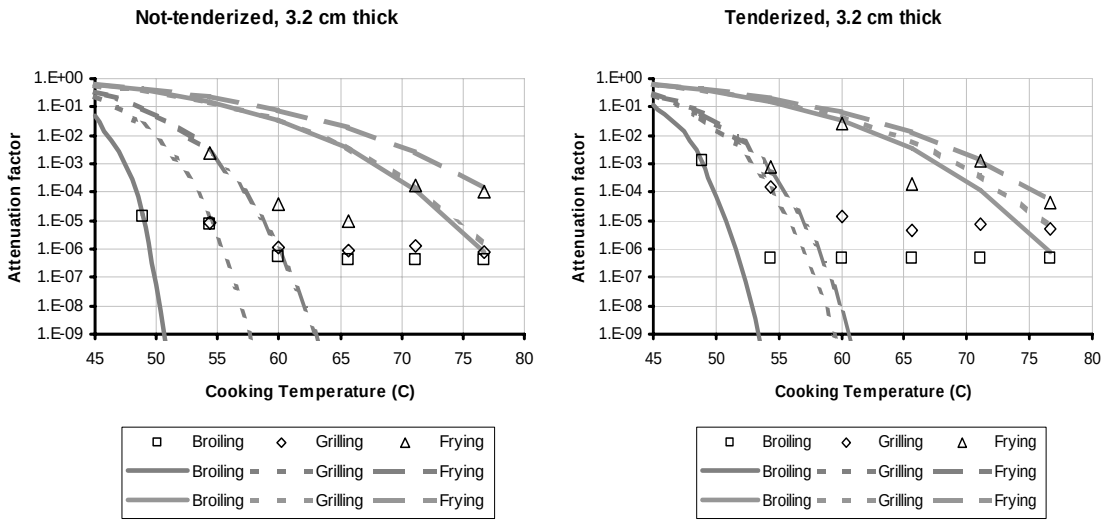

Figure TR 17. Upper and lower bounds for broiling, grilling, and frying for not-tenderized and tenderized steaks 3.2 cm thick.

3.3. Cooking temperature, T

3.3.1. Available data

Cooking temperatures for beef are included in the category of beef, pork, and lamb in the FDA Home Cooking Temperature Interactive Database (2000). The cumulative frequency of temperatures is shown in Figure TR 18.

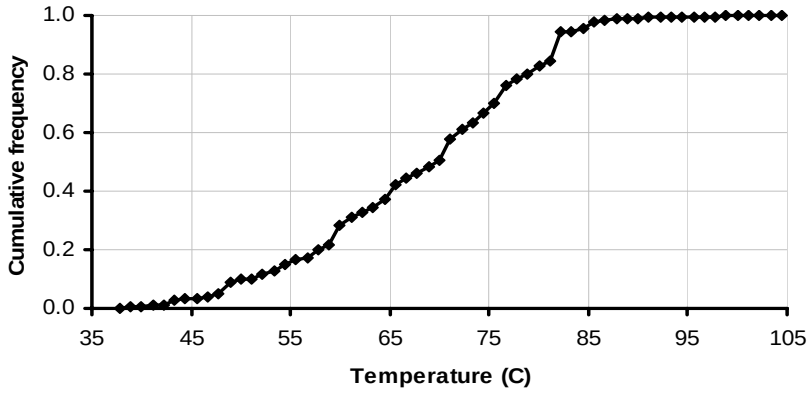

Figure TR 18. Internal cooking temperatures for the category of beef, pork, and lamb in the FDA Home Cooking Temperature Interactive Database (2000)

The minimum reported internal cooking temperature was 37.8 C. The maximum reported temperature was 104.4 C. The mean reported internal cooking temperature was 68.2 C. A summary of the data is given in Table TR 7.

Table TR 7. Summary of the FDA Home Cooking Temperature Interactive Database (2000) for beef, pork, and lamb (n=584)

Temperature range (C)	Observations	Frequency
37.8 to 51.1	59	0.101
51.1 to 64.4	158	0.271
64.4 to 77.8	242	0.414
77.8 to 91.1	121	0.207
91.1 to 104.4	4	0.007

3.3.2. Adjusting cooking temperature for beef

The data from the FDA Home Cooking Temperature Interactive Database do not specify whether each observation is for beef, pork, or lamb. Nevertheless, it is likely that beef is routinely cooked to temperatures lower than for pork. As an example, the FDA Model Food code recommends cooking beef and lamb to at least 63 C and pork to at least 68 C. Thus, it is likely that the lower temperatures in the database are more representative of beef than the higher temperatures. To account for this, the temperatures are shifted down 3 C.

3.3.3. Accounting for uncertainty in cooking temperature distribution

Several sources of uncertainty exist for the internal cooking temperature distribution. First, it is unknown to what extent beef is represented in these data. As has been noted earlier, the distribution has been shifted down to account for a possible bias if the higher temperatures are more representative of pork than beef. The amount of such bias is unknown. Second, it is unknown how representative these data are of United States cooking practices. Finally, the raw data appears to have some reporting bias. These data were originally recorded in degrees Fahrenheit. Of the 584 observations, 252 are for temperatures ending in “0”. The direction of this reporting bias is unknown. It is possible that some observers rounded up or down while recording temperatures. Thus, temperatures may be shifted several degrees from their true values. It is also possible that these recording errors essentially cancel each other over the range of the distributions and the given percentiles represent the distribution fairly accurately.

To account for these potential biases and uncertainty, the data values were increased and decreased by 6 C for lower and upper bound curves. This represents a difference between the upper and lower bounds of 12 C or 21.6 F. Note that because the lower bound is defined as the bound that would result in less likelihood of illness, it is represented by higher temperatures while the upper bound is represented by lower temperatures.

Figure TR 19 shows the resultant modeled cumulative distributions for internal cooking temperatures. Note that the lowest point of the curve for the upper bound, which represents 20% of all cooking occasions approaches 42 C or 108 F.

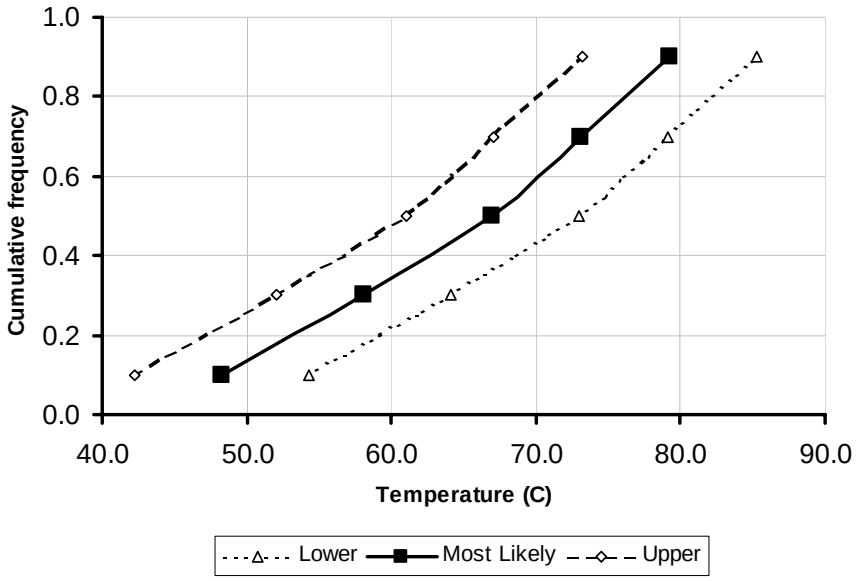

Figure TR 19. Cumulative frequency of modeled internal cooking temperatures

3.4. Path fractions

The model simulates three different cooking methods and three different steak thicknesses for a total of nine different paths. It is assumed that the fraction of product going into any one path is identical for not-tenderized and tenderized steaks and regardless of whether steaks are contaminated or if growth occurs.

3.4.1. Available data

Sporing (1999) studied three different cooking methods: broiling, grilling and skillet frying. Bogen (2001) reports that these three methods account for approximately 56% of ground beef servings. Within these three methods skillet frying accounts for approximately 40% of the servings while broiling and grilling each account for 30%.

Sporing (1999) studied steaks of three different methods: 1.3 cm, 1.9 cm, and 3.2 cm. No data is available on the proportion of steaks cooked at these three thicknesses. Thus, it is assumed that each of these thicknesses accounts for 1/3 of all steak servings.

3.4.2. Modeled path fractions

Table TR 8 shows the nine different paths modeled for not-tenderized and tenderized steaks and the fraction of servings for each path.

Table TR 8. Path fractions by steak thickness and cooking method

Steak Thickness	Cooking Method		
	Broil	**Grill**	**Fry**
1.3 cm	0.10	0.10	0.13
1.9 cm	0.10	0.10	0.13
3.2 cm	0.10	0.10	0.13

3.4.3. Uncertainty in path fractions

Uncertainty exists for both cooking method and steak thickness. Additionally, there are other cooking methods and steak thicknesses that have not been modeled. Thus, a great deal of uncertainty has been attached to these inputs in order to explore their effect. At the upper bound for each of the cooking methods it is assumed that the method accounts for approximately 90% of all steaks. At the lower bound it is assumed that the method accounts for only 1% of all steaks. The same upper and lower bounds are assumed for steak thicknesses.

3.5. Dose-response curve, DRC

3.5.1. Human outbreak data

There are three human outbreaks of *E. coli* O157:H7 illness from which pertinent data are available. These are:

An outbreak in school children and teachers in Morioka City, Japan, (Shingawa 1997; report is in Japanese) was discussed in the Netherlands risk assessment (Nauta 2001). This outbreak involved school-aged children and their adult teachers. The implicated food was salad and seafood sauce. The estimated percent ill and dose for children and adults was not substantially different (Table TR 9). The fraction of infected individuals who became ill was 55%. Infection status was determined using fecal culture of exposed individuals.

An outbreak from contaminated hamburgers in the Pacific northwest U.S. is discussed in Powell (2000) and elsewhere. Various estimates of the average dose consumed and the percent ill from this outbreak are available. We have used the estimate from FSIS (ECRA 2001) that increases the reported percent ill by a factor of 10 to account for unreported cases (Table TR 9).

Another outbreak occurred in the Chiba Prefecture, Japan. This outbreak involved very young children (ages 3-5 years) and was caused by consuming contaminated melons (Uchimura 1997:

as translated and interpreted by Dr. Fumiko Kasuga[2]). The average level of contamination in the implicated melon was 43 CFU/gram (by MPN method). It was estimated that each child consumed about 50 grams of melon. Therefore, the estimated dose ingested for this age group is 2150 organisms per serving. The reported percent of young children ill (77%) was larger than the infection fraction (54%). Because infection is a necessary precursor to illness, these results are counterintuitive. Explanations for this discrepancy include the possibility that some truly infected children were test-negative on fecal examine; or the possibility that some illnesses were incorrectly attributed to *E. coli* O157:H7. There were also four adults who were thought to have consumed the implicated melon. All four were culture-positive, but none were symptomatic. Information about the amount of melon consumed per adult was not provided, so we assume the dose is similar to that estimated for the young children.

Table TR 9. Human outbreak evidence used for development of DRC.

Outbreak	Age exposed	Number exposed	Number infected	Number ill	Percent ill	Dose ingested*
Morioka City, Japan	Adult	43	7	4*	9%	35
Morioka City, Japan	School children	828	208	114*	14%	31
Pacific northwest, U.S.	Various ages	52,600*	Not reported	3740*	7%	23
Chiba Prefecture, Japan	Young children, 3-5 yrs. old	44	24	34	77%	2150
Chiba Prefecture, Japan	Adults	4	4	0	0%	2150

*Estimated

3.5.2. Interpreting the data

The outbreak evidence provides limited illumination regarding the true dose-response curve, or curves, for *E. coli* O157:H7.

- Two of the outbreaks involve similarly small doses and, therefore, can only serve to inform a small part of the dose-response curve.

- The one outbreak reporting a larger dose involved very young children. If this age group is more susceptible to infection and/or disease than the general population, then the true dose-response curve for this age group must be distinguished from the general population's dose-response curve. Support for the notion of different dose-response curves for young children and adults is demonstrated by the failure of any of the presumably exposed adults, in the second Japanese outbreak, to become ill. While these four adults might not have consumed

[2] National Institute of Infectious Diseases, Japan

the same dose as the children, it seems difficult to argue that they consumed much less than 3-5 year-old children. Yet, this discrepancy in percent ill may be an artifact of an imperfect outbreak investigation (e.g., maybe exposed adults were not followed sufficiently over time to identify their illness).

- We are uncertain about the true percent ill and doses occurring in these reported outbreaks. For the two low dose outbreaks, the number ill was derived from a series of assumptions. For the larger dose outbreak, the percent ill is reportedly greater than the percent infected.

Obviously, the above outbreak data leaves much to be desired, but it is the best we have. In fact it is all we have. So we use it to draw a "best estimate" DRC and then put large uncertainty bounds on that curve.

3.5.3. Fitting the "best estimate" curve to the data

We are interested in describing the functional relationship between dose consumed and fraction of illness per serving for the general population.

Figure TR 20 shows the data points corresponding to the outbreaks. Overlaid on these points are the DRCs for ground beef (ECRA 2001), *Shigella dysenteriae*, and Enteropathogenic *E. coli* (EPEC) which are thought to be plausible upper and lower bounds for *E. coli* O157:H7.

The most likely curve in Figure TR 20 is not consistent with the outbreak data. Because three of the outbreak data-points relate to doses between 20 and 40 bacteria, and these data suggest similarity fractions ill, we move the most likely curve to the left to intersect these data points. Similarly, the boundary curves are shifted to envelop the outbreak data.

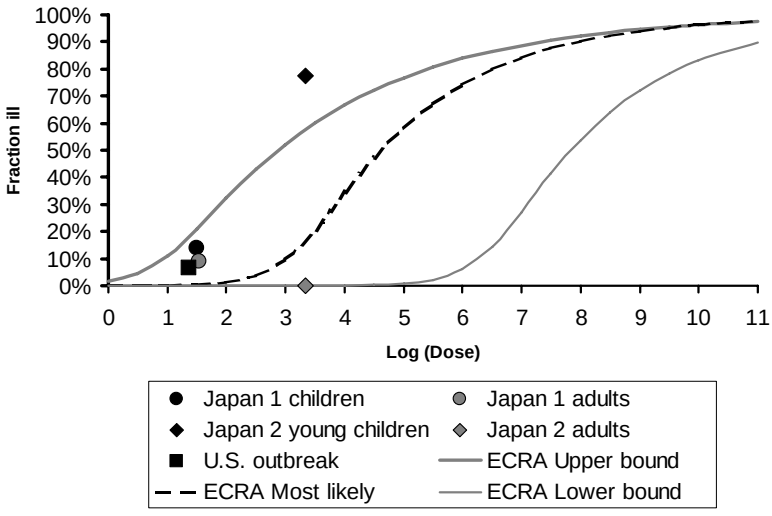

Figure TR 20. Plotted human *E. coli* O157:H7 outbreak data and the predicted dose-response curves from the ground beef risk assessment (ECRA 2001).

Figure TR 21 diagrams the adjusted dose-response curves. The most likely curve intersects the cluster of outbreak points at lower doses, but remains well to the right of the outbreak point for very young children. We would expect that a dose-response curve for young children (<5 years old) would be shifted to the left of this most likely curve to account for that data point. The boundary curves envelop the outbreak data and express our uncertainty regarding the true dose-response curve for the general population. Nevertheless, the upper bound curve may also reflect a more likely curve for susceptible sub-populations such as children <5 years old.

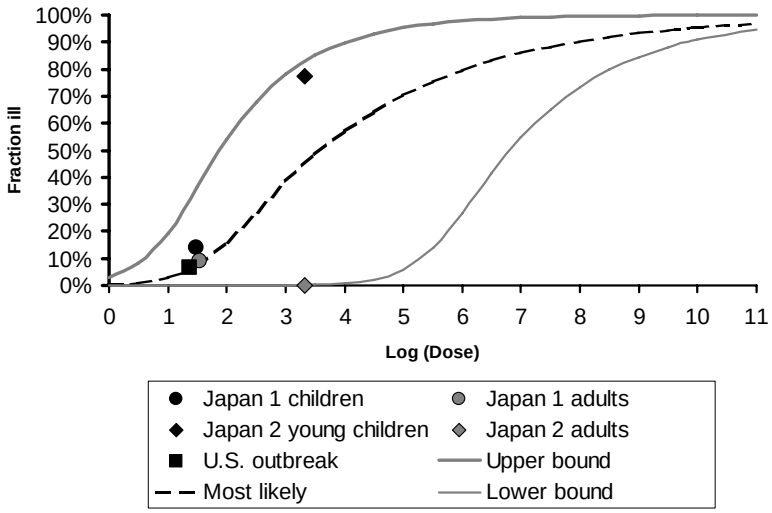

Figure TR 21. Dose-response curves used in non-intact beef model to represent the relationship between average dose consumed and the fraction of the general population who become ill. The upper bound curve, in this case, may be reflective of a dose-response curve for highly susceptible individuals.

4. Results

The probability of *E. coli* O157:H7 surviving typical cooking practices in either tenderized or not-tenderized steaks is minuscule. As can be seen in Figure TR 22, 0.000026 percent (i.e., 2.6 of every 10 million servings) of steaks that are not tenderized contain one or more bacteria. For tenderized steaks, 0.000037 percent (i.e., 3.7 of every 10 million servings) contain one or more bacteria.

As shown in Figure TR 22, illness seldom occurs at doses less than 10 bacteria per serving. At a dose of 100 bacteria, approximately 16 percent of those exposed will become ill. The fraction of not tenderized servings with doses of 100 or more bacteria is about 1.4 in 10 million, while the fraction of tenderized servings with doses $\geq$100 is about 1.5 in 10 million.

Differences in bacterial dose after cooking attributable to tenderized versus not-tenderized steaks are minimal at most. Figure TR 22 shows a barely discernable difference at dose levels

greater than 1 between tenderized and not-tenderized steaks. The expected illnesses per serving (IPS_{EV}) for tenderized steaks is 1 illness per 14.2 million servings (7.0×10^{-8}). For not-tenderized steaks the IPS_{EV} is 1 illness per 15.9 million servings (6.3×10^{-8}). What this means is that there will be seven additional illnesses due to tenderization for every billion steak servings($7.0 \times 10^{-8} - 6.3 \times 10^{-8}$).

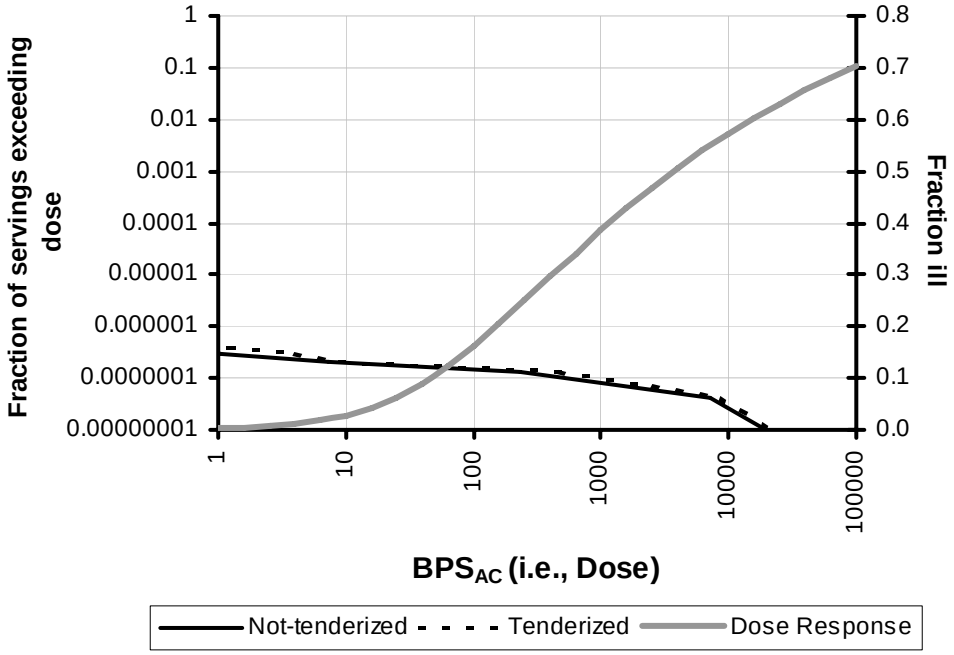

Figure TR 22. Model output showing predicted bacteria per serving after cooking (Dose) and corresponding frequency of illness (Dose Response).

4.1. Uncertainty in the Analysis

A systematic sensitivity analysis was conducted on the above results to measure the effects on the output, IPS_{EV}., of uncertainties in the input. A summary of that sensitivity analysis is presented in

Figure TR 23. Values reflecting upper and lower bounds for each identified input were compared one-by-one while using the most likely parameters for all other inputs. This analysis shows that IPS_{EV} is most sensitive to the inputs for initial contamination, growth effects, and health effects (dose response curve – DRC( )). For these inputs, the final results differ by almost 3 logs, based on whether the upper or lower bounds of uncertainty were used. The initial contamination and growth effects are used to calculate the bacteria per serving before cooking (BPS_{BC}). Other input values (internal cooking temperature, cooking effect or attenuation factor, and fractions of steaks cooked using different cooking methods or at different thicknesses) were found to be substantially less sensitive. Although the effect of cooking was initially hypothesized to be an important determinant of human illness, its influence was less than that for other inputs.

As to distinguishable differences between tenderized and not-tenderized steaks, the sensitivity analysis failed to isolate any. Upper and lower ranges of risk moved in tandem for both tenderized and not-tenderized steaks.

Note that the horizontal lines in Figure TR 23 reflect the risk associated with the combination of most likely input values. In this case tenderized steaks show marginally higher risk than not-tenderized steaks, but confidence in this conclusion is not high.

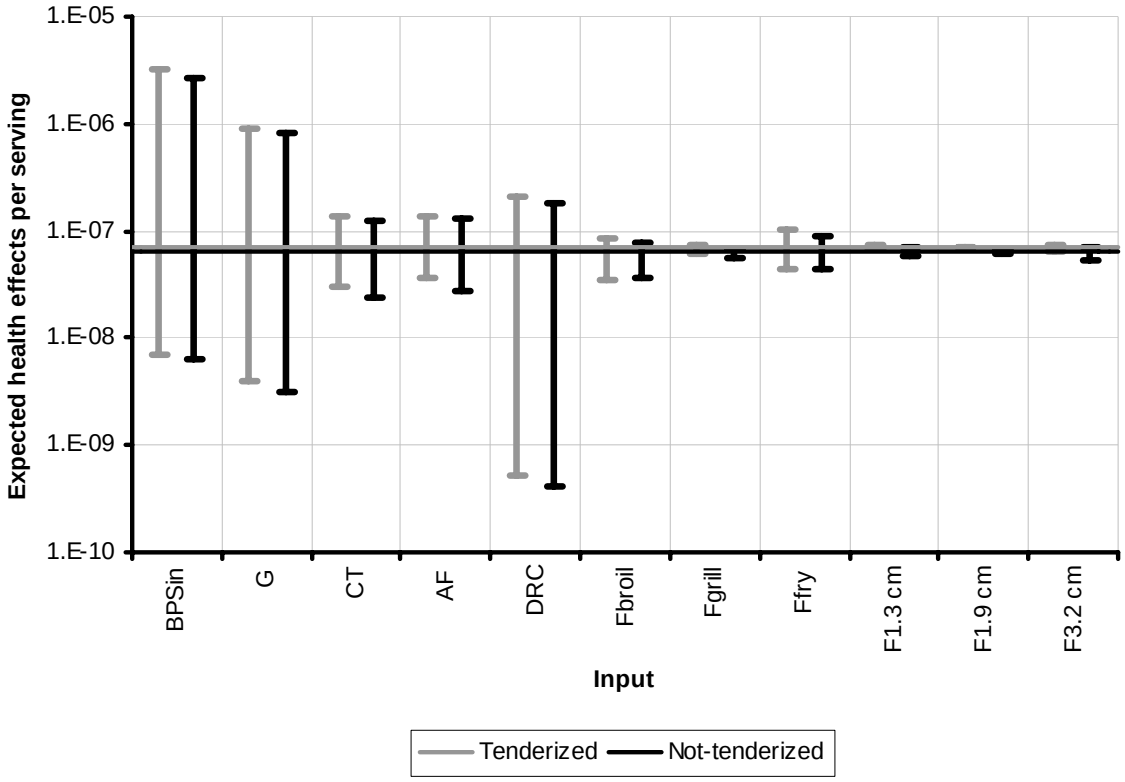

Figure TR 23. Sensitivity of expected health effects per serving, IPS_{EV}, due to various uncertain model parameters.

4.2. Uncertainty in IPS_{EV}

Given the input settings, the model calculates the expected value of the illnesses per serving (IPS_{EV}) distribution (eqn (17)). Because we are uncertain about model inputs (e.g., BPS_{in}, CT, AF), we calculate different IPS_{EV} values for different inputs. We use the discrete probability technique to propagate uncertainty in model inputs through the model. In this case, we've assigned probabilities of 0.1 to both the upper and lower bounds for each uncertain input. Consequently, we've assigned a probability of 0.8 to each uncertain input's best guess. The results of this calculation, for tenderized and not tenderized steaks, are shown in Figure 1.

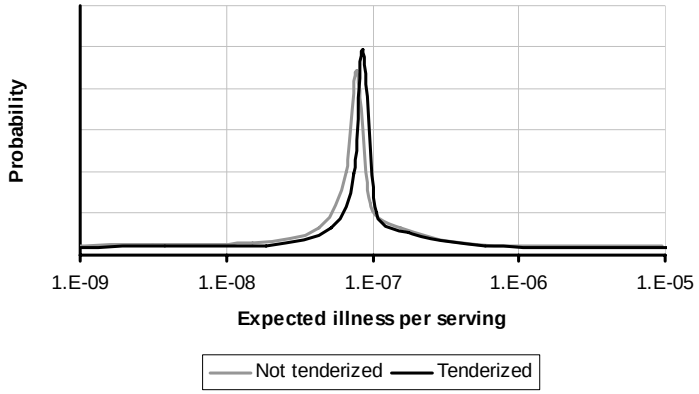

Figure TR 24. Probability curves of IPS_{EV} for not tenderized and tenderized steaks.

This figure shows that about 80% of our confidence lies between IPS_{EV} values of 4.5×10^{-8} and 1.0×10^{-7}. Further, it shows that the probability distribution for tenderized steaks is shifted slightly towards higher expected illnesses per serving relative to not tenderized steaks. To present this effect in another way we show, in Figure TR 25, the probability curve for the difference: IPS_{EV} (tenderized) – IPS_{EV} (npn tenderized).

This figure shows that we are about 80% confident that the difference in IPS_{EV} between tenderized and not tenderized steaks lies between 5.7×10^{-11} and 1.3×10^{-7}. Also the figure shows a 5% probability that this difference is negative (i.e., that IPS_{EV} for tenderized steaks is actually less than IPS_{EV} for not-tenderized).

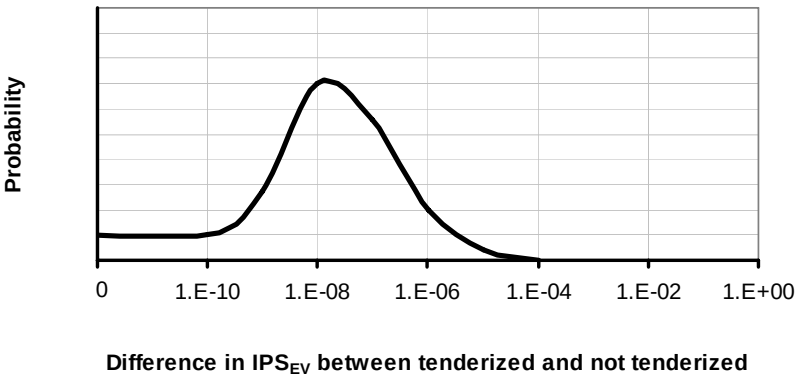

Figure TR 25. Probability distribution for the difference in IPS_{EV} between tenderized and not tenderized steaks.

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