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Quantitative risk assessment for *Escherichia coli* O157:H7 in ground beef hamburgers

Michael H. Cassin^a, Anna M. Lammerding^{b,*}, Ewen C.D. Todd^c, William Ross^d,
R. Stephen McColl^e

^aDecisionalysis Risk Consulting, Ottawa, ON, Canada

^bHealth of Animals Laboratory, Health Canada, Guelph, ON, Canada N1G 3W4

^cBureau of Microbial Hazards, Health Canada, Ottawa, ON, Canada

^dBureau of Biostatistics and Computer Applications, Health Canada, Ottawa, ON, Canada

^eDepartment of Health Studies and Gerontology, University of Waterloo, Waterloo, ON, Canada

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Abstract

Quantitative Risk Assessment (QRA) is a methodology used to organize and analyze scientific information to estimate the probability and severity of an adverse event. Applied to microbial food safety, the methodology can also help to identify those stages in the manufacture, distribution, handling, and consumption of foods that contribute to an increased risk of foodborne illness, and help focus resources and efforts to most effectively reduce the risk of foodborne pathogens. The term Process Risk Model (PRM) is introduced in this paper to describe the integration and application of QRA methodology with scenario analysis and predictive microbiology to provide an objective assessment of the hygienic characteristics of a manufacturing process. The methodology was applied to model the human health risk associated with *Escherichia coli* O157:H7 in ground beef hamburgers. The PRM incorporated two mathematical submodels; the first was intended to describe the behaviour of the pathogen from the production of the food through processing, handling, and consumption to predict human exposure. The exposure estimate was then used as input to a dose–response model to estimate the health risk associated with consuming food from the process. Monte Carlo simulation was used to assess the effect of the uncertainty and variability in the model parameters on the predicted human health risk. The model predicted a probability of Hemolytic Uremic Syndrome of 3.7×10^{-6} and a probability of mortality of 1.9×10^{-7} per meal for the very young. These estimates are likely high for all hamburger meals, but may be reasonable for the home-prepared hamburgers described by this model. The efficacy of three risk mitigation strategies were evaluated by modifying the values of the predictive factors and comparing the new predicted risk. The average probability of illness was predicted to be reduced by 80% under a hypothetical mitigation strategy directed at reducing microbial growth during retail storage through a reduction in storage temperature. This strategy was predicted to be more effective than a hypothetical intervention which estimated a plausible reduction in the concentration of *E. coli* O157:H7 in the feces of cattle shedding the pathogen and one aimed at convincing consumers to cook hamburgers more thoroughly. The conclusions of this approach are only accurate to the extent that the model accurately represents the process. Currently, uncertainty and ignorance about the hygienic effects of the individual operations during production, processing, and handling limit the applicability of a PRM to specify HACCP criteria in a quantitative manner. However, with continuous improvement through stimulated research, a PRM should encompass all

*Corresponding author. Tel.: +519 8223300; fax: +519 8222280; e-mail: Anna_Lammerding@hc-sc.gc.ca

available information about the process, food, and pathogen and should be the most appropriate decision-support tool since it represents current knowledge. © 1998 Elsevier Science B.V.

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1. Introduction

Governments and industry have begun to focus attention on the production of foodstuffs as a source of risk to public health. The cost of foodborne disease is estimated to exceed \$5 billion per year in the United States (Foegeding et al., 1994), and \$1.3 billion annually in Canada (Todd, 1989). In recent years, *Escherichia coli* O157:H7 has emerged as a primary food safety concern. The annual cost to the U.S. economy of the estimated 10 000–20 000 *E. coli* O157:H7-related illnesses is between \$216–580 million dollars (Mark and Roberts, 1993). Several outbreaks, most notably a recent large outbreak in Washington State, have identified undercooked hamburgers as a significant vehicle for *E. coli* O157:H7 related disorders (AGA, 1995; Griffin and Tauxe, 1991; Bryant et al., 1989).

Growing public concern over the microbiological safety of foods and the shortcomings of both Good Manufacturing Practices (GMP) and end-product testing have prompted industry and regulators to accept hazard analysis critical control point (HACCP) as the system to ensure food safety (van Schothorst and Jongeneel, 1993). The U.S. Department of Agriculture has proposed that all meat and poultry establishments be required to adopt HACCP systems for their processes, as a means to assure the safety of their products (USDA, 1996). HACCP principles can be found in a number of EC directives for meat, poultry, and fish (van Schothorst and Jongeneel, 1993). However, subjective assessments of the hygienic conditions of raw product are impeding the development of effective HACCP systems in primary processing (Gill, 1995). There is a lack of knowledge about the points that are critical to controlling microbiological contamination in meat production (USDA, 1994a). Objective identification of the hygienic characteristics of a meat plant process is a necessary first step towards developing a HACCP system (Gill et al., 1996a).

The prominence of *E. coli* O157:H7 warrants the conduct of a detailed Quantitative Risk Assessment (QRA) to support risk management actions, in both regulatory and HACCP programs. QRA will also identify appropriate future risk management strategies, and where in the food production pathway it would be most appropriate to implement control actions, or focus research.

This paper uses the term Process Risk Model (PRM) to describe the integration and application of QRA methodology with scenario analysis and predictive microbiology to provide an objective assessment of the hygienic characteristics of a manufacturing process. Although the PRM outcome is given in terms of the human health risk presented by the product, ground beef in home-prepared hamburger patties, the emphasis of the PRM is to apply QRA as a tool that can be used to identify intervention procedures that might mitigate the risk experienced and perceived by the public.

1.1. Risk assessment framework

The PRM described is consistent with the risk assessment framework described in the report 'Application of Risk Analysis to Food Standards Issues', a document prepared by the FAO/WHO Expert Consultation to provide the Food and Agriculture Organization (FAO), the World Health Organization (WHO), the Codex Alimentarius Commission (CAC) and member countries with advice on approaches for the application of risk analysis, with a focus on risk assessment, to food standards issues (WHO, 1995).

It is acknowledged that risk assessment terminology for microbial food safety is not yet definitive, and differences currently exist among various regulatory/international agencies/organizations. Nevertheless, the key elements required for an accurate risk assessment are the same, regardless of semantics. In particular, the term 'dose–response assessment' used

in this assessment is in essence consistent with the 'hazard characterization' step defined in the FAO/WHO report (WHO, 1995).

The Risk Assessment definitions used in this document are similar to that of Potter (1996):

Hazard: A biological, chemical, or physical agent in, or property of food with the potential to cause an adverse effect.

Hazard identification: Identification of known or potential health effects associated with a particular agent in food.

Exposure assessment: The evaluation of the degree of intake likely to occur.

Dose–Response assessment: Determination of the relationship between the magnitude of exposure and the magnitude and/or frequency of adverse effects.

Risk Characterization: The estimation of the adverse effects likely to occur in a given population, and a summary of assumptions and sources of uncertainty

In addition, Importance and Sensitivity Analysis, the identification of factors which most significantly contribute to risk, was included in this assessment.

1.2. Process risk modelling

The model developed differed from a conventional QRA which solely attempts to obtain an estimate of risk (Rodricks, 1994). The PRM incorporated two mathematical submodels; the first was intended to describe the behaviour of the pathogen from the production of the food through processing, handling, and consumption to predict human exposure. This may be considered as a measure of the hygienic quality of the system. The exposure estimate was then used as an input to a dose–response model to estimate the health risk associated with consuming food from the process. The outcome combining exposure and dose–response yielded an estimate of health risk, rather than hygiene. Risk to human health was regarded as the measure of the quality of the system on the premise that it is the parameter of interest.

By quantifying the risks associated with the practices of food production from 'farm-to-fork', a model should be able to accurately describe the process by which contamination occurs and the impact to the endpoint of interest: human health. Currently, uncertainty and ignorance about the hygienic effects of the individual operations during

production, processing, and handling limit the applicability of a PRM to specify HACCP criteria in a quantitative manner. The usefulness of the PRM is expected to be more significant with continuous improvement from collaboration and stimulated research aimed at reducing uncertainty.

2. Materials and methods

Information and data for the development of the model were obtained from literature and expert opinion. The risk model was developed to facilitate Monte Carlo simulation, for a discussion of which the reader should refer to Vose (1996). This procedure entails generating hypothetical scenarios in terms of the values attributed to the identified factors in the exposure and dose–response assessments. The simulation represents the inherent variability in the process of food production and consumption and the uncertainty in the mathematical model of the process. The outcome is a statistical distribution of risk experienced by the diverse members of the population.

A Monte Carlo simulation of the model was performed using the uncertain factors described by probability distributions. Twenty-five thousand iterations were performed for each simulation, using Latin Hypercube sampling, with the @RISK™ software package version 3.5e [Palisade, Newfield, NY] and Microsoft Excel™ [Microsoft Corp., CA], running on a Intel Pentium 166 MHz based PC. The number of iterations provided adequate convergence of the simulation statistics ($\pm 2.5\%$) (Morgan and Henrion, 1990).

After the model was developed and results obtained, analysis and experimentation with the model were performed. We break from the traditional format for reporting on pure science experiments, by reporting the methodology and some interpretation of the analysis in the Results and Analysis section.

3. Risk assessment

The PRM described ground beef produced by a particular hypothetical abattoir. The commercial plant modelled produces beef trimmings from cattle

destined for retail sale as ground beef. The retailers grind this beef on site as required to stock the display cabinet. Fig. 1 shows the conceptual model upon which the mathematical model was based.

3.1. Hazard identification

The hazard associated with the consumption of hamburgers in this risk assessment was *Escherichia*

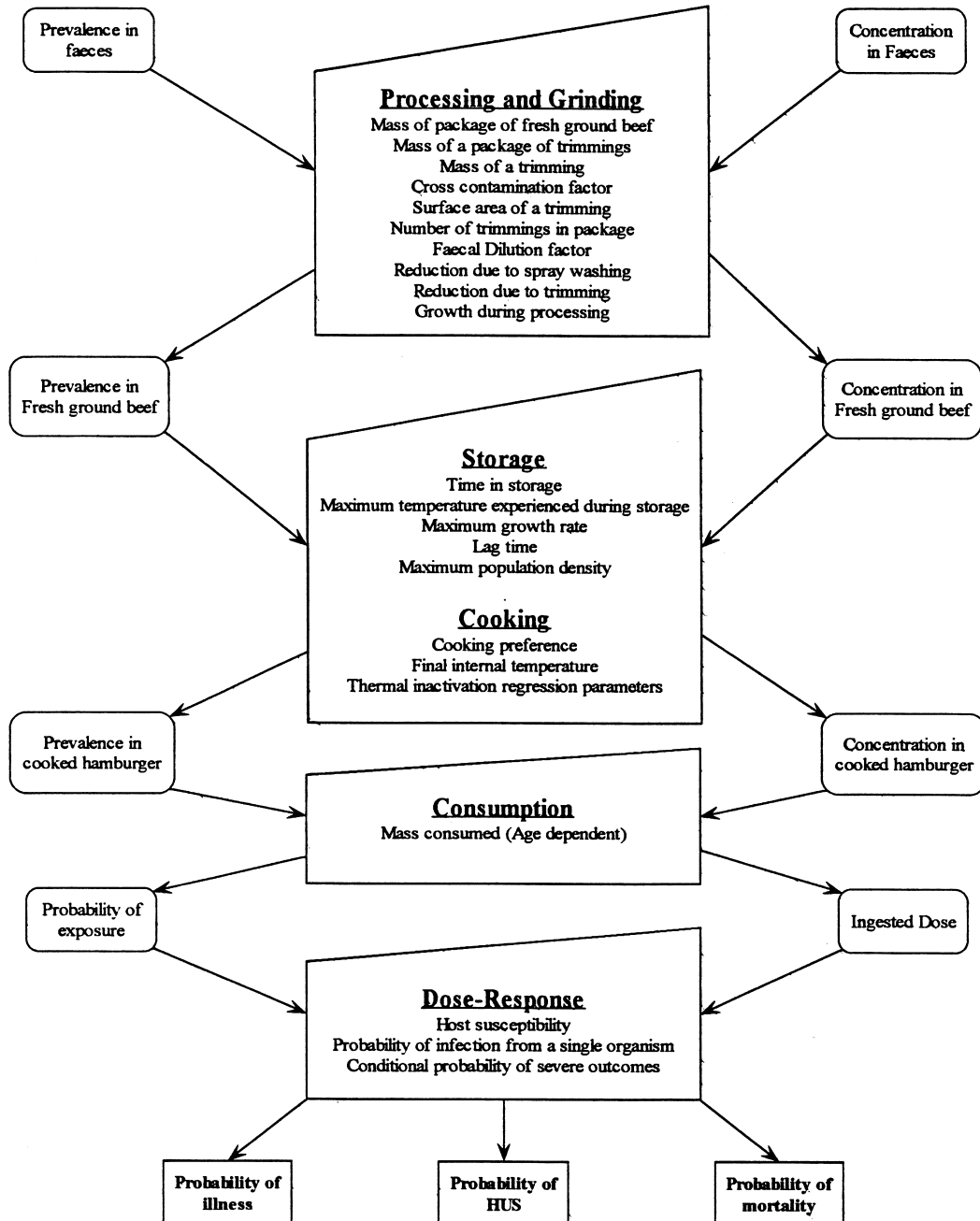


Fig. 1. Flow diagram of the mathematical model of exposure assessment and dose-response for *E. coli* O157:H7 in hamburgers.

coli O157:H7. *E. coli* is a species of gram-negative, facultatively anaerobic, rod-shaped bacteria commonly found in the lower part of the intestine of warm-blooded animals (USDA, 1994a). *E. coli* O157:H7 is a particular serotype of the group referred to as enterohemorrhagic *E. coli* (EHEC). This is the subgroup of verocytotoxigenic *E. coli* (VTEC) that have been shown to cause human illness. VTECs produce verotoxins, or shiga-like toxins, that are closely related to the toxin produced by *Shigella dysenteriae* (Tarr, 1995).

It has been shown that cattle may be a reservoir of *E. coli* O157:H7, and that contamination of carcasses during slaughter and processing may be the manner by which beef and beef products become contaminated and transmit the organism to humans (Chapman et al., 1993). Well-documented clinical findings link *E. coli* O157:H7 to human health effects (AGA, 1995; Tarr, 1995), and the probability that *E. coli* O157:H7 presents a hazard to humans is assumed to be 100%. Certainty regarding the hazard is expected to be common among microbial risk assessments, in contrast to the many carcinogen risk assessments which must consider the possibility that the agent is non-carcinogenic in humans.

E. coli O157:H7 infection results in moderate to severe disease, with most deaths in young children and aged persons (AGA, 1995; Tarr, 1995). The name of the disease commonly associated with *E. coli* O157:H7 infection is haemorrhagic colitis. Typically, cases develop diarrhea, often bloody, with acute abdominal cramps about 3 to 7 days after infection. Some cases may never show blood in their stools and, therefore, this observation is only diagnostic for the more severely ill patients. In about 10% of cases, usually in children, kidney damage occurs causing haemolytic uraemic syndrome (HUS) (AGA, 1995; Bell et al., 1994; Vogt, 1994; Ries et al., 1993). Further complications may result in thrombotic thrombocytopenia purpura (TTP), more typical of older patients. An illness may last from several days to many months, and result in death or permanent damage.

Beef, sausages (pork and beef), raw milk, apple cider, handling potatoes, lettuce, mayonnaise, handling of potatoes, and drinking and recreational water have been associated with *E. coli* O157:H7 infections (Armstrong et al., 1996). In Canada, between 1990 and 1995, 1014 to 1432 VTEC cases were

reported annually (LCDC, 1995; Khakhria et al., 1996, 1997). The majority of VTEC isolated were *E. coli* O157:H7. Undercooked or raw ground beef has been frequently implicated in foodborne outbreaks. The U.S. Centers for Disease Control and Prevention (CDC) outbreak data from 1982 to 1994 reveal that 1137/2334 cases were associated with ground beef consumption (Armstrong et al., 1996). Other vehicles, particularly recreational waters, appear to be emerging as significant sources of the organism in the U.S. (Anon., 1997).

3.2. Exposure assessment

In order to assess the risk to human health from *E. coli* O157:H7 associated with the consumption of ground beef, the potential exposure to the organism in a single-meal serving was estimated. The exposure was characterized by the probability that viable organisms were present in the meal at the time of consumption and the distribution of the ingested dose in terms of colony-forming units (CFU). Both the *probability of exposure* and the *dose* were outputs of a mathematical model describing the entire process of food production, processing, and consumption. The details of this mathematical model, including assumed probability distributions for parameters and the equations predicting the behaviour of the pathogen can be found in the appendix.

3.2.1. Production

To estimate the extent to which a carcass may become contaminated with fecal material containing *E. coli* O157:H7, the prevalence and concentration of the organism in feces were considered. There are a number of studies that suggest seasonal differences in the prevalence of the pathogen in cattle and disease in humans. Some research has shown that the magnitude, in CFU/g, and the duration of shedding for colonized animals differs between adult cattle and preweaned calves (Cray and Moon, 1995). Feeding practices have also been shown to effect the growth of the pathogen in the ruminal environment (Rasmussen et al., 1993). Seasonality, geographical effects, and feeding practices are acknowledged as possibly important parameters, but were not incorporated in this model.

Table 1
Concentration of *E. coli* O157:H7 in feces of shedding cattle (Zhao et al., 1995)

Concentration in feces [\log_{10} CFU/g]	Cumulative number of animals	Percentile
less than -1^a	0/31	0%
less than 2^b	15/31	48%
less than 3	17/31	54%
less than 4	28/31	90%
less than 5	31/31	100%

^a The minimum concentration of *E. coli* O157:H7 in contaminated feces was assumed to be 0.1 CFU/g based on positive isolation from a 10 g enriched sample.

^b 10^2 CFU/g limit of detection for plating method.

3.2.1.1. Concentration

Experimental data for the concentration of *E. coli* O157:H7 in cattle feces are shown in Table 1 (Zhao et al., 1995). In 31 animals detected positive for *E. coli* O157:H7 by enrichment methods, the microbial load of *E. coli* O157:H7 in the feces of shedding animals was found to range from undetectable by direct plating (i.e., $<2.0 \log_{10}$ CFU/g) to $5.0 \log_{10}$ CFU/g. The distribution of the concentration of *E. coli* O157:H7 in the feces of colonized animals was constructed from the histogram of these data.

3.2.1.2. Prevalence

Several sources have reported the detection of *E. coli* O157:H7 shed in the feces of cattle. There is obvious between-herd variance in the detection data due to the wide variety of circumstances under which each survey was performed including variability in region, sample size, and the type and age of bovine animal, and detection method (Armstrong et al.,

1996). The data used to estimate the distribution for the prevalence of cattle shedding *E. coli* O157:H7 in their feces are shown in Table 2. The data set was limited to those studies involving classes of cattle likely to be destined for consumption as ground beef by excluding studies that focused primarily on prevalence in calves. The distribution parameters were estimated using the method of moments (Vose, 1996) assuming that the prevalence can be characterized with a beta distribution and that the outcome of a detection study was a binomial random variable.

3.2.2. Processing and grinding

For the purposes of this model, processing was defined as those operations which begin at the slaughter of the animal and end at the packaging of fresh ground beef. The hypothetical food system involved an abattoir that supplied 5 kg vacuum packs of carcass trimmings to a retail outlet that ground the trimmings on-site for sale as ground beef. The probability of *E. coli* O157:H7-contaminated packages of fresh ground beef and the concentration of the pathogen in these packages was the focus of this section of the PRM. The mathematical model of processing attempted to estimate the probability and concentration of contaminated packages as a function of the prevalence in cattle shedding the organism, the concentration of the organism in feces, and various factors of the processing operations that may affect the prevalence and populations of the organism on meat. This was achieved by quantitatively describing the behaviour of *E. coli* O157:H7 during the beef carcass dressing process and subsequent grinding of the meat. Thirty-six distinct processing operations have been identified (Gill et al., 1996b), which were

Table 2
Detection rates for *E. coli* O157:H7 in cattle

Reference	Location	Year	Sampling site	No. positive/No. tested (%)
Wells et al., 1991	WI, WA, &OR, USA	1986–87	Dairy farms	1/662 cows (0.2) 12/394 heifers (3.1)
Wilson et al., 1992	ON, Can.	1988	Dairy farms	0/1131 cows (0)
Hancock et al., 1994	WA, USA	1992	Dairy farms	10/3570 dairy cows & calves (0.3)
			Farm	10/1412 pasture beef cows (0.7)
			Feedlot	2/600 beef cattle (0.3)
Clarke et al., 1994	ON, Can.	1992–1993	Abattoir	3/200 beef animals (1.5) 1/200 cull dairy cows (0.5)
Wilson et al., 1995	ON, Can.	1992–93	Dairy farms	4/1268 cows (0.3)
Hancock et al., 1997	13 states, USA	1994	Feedlot	188/11 881 samples (1.6)

divided into three main sub-operations: skinning, evisceration, and trimming.

E. coli are not naturally present on or in red meat, but they are present as the direct result of feces deposited on the carcass at one or several points between slaughter and packaging. The fecal contamination of a beef carcass during these operations is considered largely unavoidable. The model does not suggest that the presence of fecal material is a definitive indicator for the presence of *E. coli* O157:H7. However, for conceptual purposes, the original source of this enteric pathogen was considered to be the feces of an animal shedding the pathogen. Therefore, the approach asserted that the presence of *E. coli* O157:H7 was a perfect indicator for fecal contamination of the product, but not vice-versa. The medium in or on which the pathogen is carried, be it feces, soil, saliva, air, water, or meat becomes increasingly obscured through the farm-to-fork continuum.

The model incorporated the assumption that the microbial profile of a production lot of beef trimmings is independent of previous lots processed at the abattoir, i.e. that the plant environment is completely sterilized between runs. This simplifying assumption may be false. It is thought that at the end of the production run, pathogenic bacteria may be undetectable in the plant environment due to dilute concentration, but overnight growth may cause it to appear in the product the next day. However, the incremental risk associated with lot-to-lot contamination may be negligible, possibly reducing the importance of this assumption.

The prevalence of *E. coli* O157:H7 on carcasses was assumed to be proportional to the prevalence of animals shedding the pathogen. The prevalence in packages of trimmings was expected to be much higher because meat from several carcasses were expected to be in each package. The retail grinder was assumed to randomly mix the pathogen throughout the 5 kg lot of ground beef.

The production model provided the assumed prevalence of *E. coli* O157:H7-shedding animals. The ratio of carcasses contaminated with the feces of *E. coli* O157:H7-shedding animals to those free of such contamination was assumed to be two to three times the ratio of *E. coli* O157:H7-shedding animals to non-shedders. This increase in the prevalence was based on the possibility of cross-contamination be-

fore and during the dressing procedure. There is no information to support the assumed rate of cross-contamination. The soil on a hide is likely comprised of feces associated with several members of the production lot and in fact it may be that many or all of the animals in the production lot have come into contact with feces from the other animals being slaughtered. Also, the assumption does not address the difference between the pathogen concentrations in filth on an animal shedding the pathogen and filth which has come in contact with the pathogen.

Immediately following the removal of the hide, the concentration of *E. coli* O157:H7 on carcasses is assumed to be proportional to the concentration in feces. During skinning, fecal material residing on the hide of the carcass may come in contact with the newly exposed meat. USDA (1994b) baseline data, shown in Table 3 indicate that between 0 and 5 log₁₀ CFU/cm² *E. coli* (Biotype 1) are on the carcass. A dilution factor between the concentration in the feces and the concentration on the newly-exposed meat was proposed. The dilution factor was estimated as the number of grams of feces deposited per square centimetre of carcass surface area. This was achieved by simulating the difference between the log concentration in feces and the observed data for log concentration on carcasses. The log concentration of *E. coli* (Biotype 1) in bovine feces was assumed to have a Normal distribution. Fitting the parameters of this distribution to over 500 Australian data samples (Commonwealth Scientific and Industrial Research Organisation, unpublished data), the mean was estimated at 6.1 log₁₀ CFU/g and the standard deviation at 0.9 log₁₀ CFU/g. The simulation data for the log of the dilution factor was well represented by a Normal distribution with a mean of -5.1 log₁₀

Table 3
E. coli (Biotype 1) on beef carcass surface samples (USDA, 1994b)

log ₁₀ CFU/cm ²	Number of samples	Percentage of samples
<0	1917	91.8%
0–1	86	4.1%
1–2	50	2.4%
2–3	21	1.0%
3–4	10	0.5%
4–5	4	0.2%
5–6	1	0.0%

g/cm² and with a standard deviation of 0.9 log₁₀ g/cm². In light of the ambiguous nature of the medium carrying the pathogen, the use of the dilution factor is identified as a model simplification, representing a significant uncertainty in the model. Additionally, the feces and therefore the *E. coli* O157:H7 are spread non-homogeneously over the surface of the carcass. This effect was not modelled.

There is some disagreement as to the effect of various decontamination treatments, such as the trimming of visible defects, spray washing, and steam vacuuming. Some investigators suggest that there is a physical removal of bacteria on the carcass, whereas others suggest that the organisms are for the most part simply redistributed. Decontamination treatments may reduce *E. coli* counts by 2.6–4.3 log₁₀ CFU/cm² depending on the pressure and temperature and various wash/vacuum/steam combinations (Dorsa et al., 1996). Without some sort of thermal inactivation of the organisms during the procedure, as little as half of the organisms may be physically removed (Gill, personal communication).

Trimming of visible defects has not been shown to be effective in reducing microbial counts in the plant environment (Gill, personal communication). The physical removal of visible feces must have some effect in reducing the number of pathogens on the meat if the visible feces contains any *E. coli* O157:H7. The absence of a demonstrable effect is likely because the effect is statistically undetectable due to the wide variance of counts found in meat.

The log reduction in counts of *E. coli* O157:H7 on the carcass due to decontamination treatments was aggregated into a single parameter. Hand trimming followed by spray washing with plain water has been observed to reduce counts by 1.4–2.5 log₁₀ CFU/cm² (Gorman et al., 1995). A subsequent experiment considering the effect of the different times of exposure to fecal material reported 0.94–2.58 log₁₀ CFU/cm² reductions in counts of *E. coli* from spray-washing after 4 h of exposure to a fecal paste. The decontamination treatment in the hypothetical abattoir was assumed to be similar to that used in these experiments. The aggregate effect of all decontamination treatments was therefore assumed to be a 1 to 2.5 log reduction in counts.

There is a possibility of significant contamination occurring if the gut is nicked during the evisceration process, although no data are available to quantify

this risk. In fact, the microbial load on the carcass after evisceration is found to be not significantly different than just after skinning (Schnell et al., 1995). The process model will assume that this risk is negligible, until some data become available to quantify the probability of contamination from this operation. The excessive handling during evisceration and trimming processes is likely to spread the filth more evenly over the carcass surface. This effect was not modelled.

The chilling of the carcasses is not wholly effective in preventing microbial growth. *E. coli* proliferation of up to five generations have been observed on pig carcasses during cooling after passing through a freezing air blast tunnel (Gill, 1996). However, a decrease of about 2 log CFU/cm² may be observed during chilling of beef carcasses (Gill, personal communication). During chilling, the maximum possible growth at the warmest point on the surface of a beef carcass is between two and 14 generations (Gill, personal communication). The aggregate proliferation on the surface of a beef carcass was described by an assumed triangular distribution with a minimum of –2, a mode of 0, and a maximum of five generations.

Trimmings collected from the deboning process, destined for the manufacture of ground beef, are commonly between 100 g and 500 g in size, and each trimming is likely associated with a different carcass due to sorting according to fat content. Since the concentration of the pathogen on the carcass meat was modelled per unit area, the surface area of the contaminated trimmings was required to estimate the total number of pathogens on the trimming. Surface area refers to the portion of the area which was exposed on the skinned carcass and does not include subcutaneous tissue newly exposed during deboning. The surface area is correlated with the mass of the trim. As a conservative assumption, the surface area was considered to be 0.25–1 cm²/g of trim.

The concentration of *E. coli* O157:H7 in 5 kg packages of trimmings was mathematically modelled using the assumptions discussed. The concentration of the organism was assumed to be reduced by spray washing and the trimming of visible filth. The concentration was assumed to have increased due to microbial growth during processing. The concentration was considered to be diluted in the 5 kg

package since many of the trimmings in a package will be free of the pathogen. The probability of *E. coli* O157:H7-contaminated packages of trimmings was modelled as the probability that a package contains at least one *E. coli* O157:H7-contaminated trimming.

The packages of trimmings were assumed to be ground by the retailer and set out for display in packages containing from 300 g to 1000 g of fresh ground beef. The occurrence of the organism in packages of fresh ground beef was modelled by a Poisson process, assuming that the ground beef packages were effectively sampled from the 5 kg package of trimmings. The probability of *E. coli* O157:H7 in ground beef was modelled as the probability that the package contains one or more *E. coli* O157:H7. The average probability of *E. coli* O157:H7 in fresh ground beef could be interpreted as the model's estimate of the prevalence in retail ground beef.

3.2.3. Post-processing

This section of the PRM was concerned with changes in the concentration of *E. coli* O157:H7 in ground beef between the time it is ground at retail and the time it is consumed. During this time, microbial growth and subsequent cooking were considered in estimating the concentration of viable *E. coli* O157:H7 in the consumed product. The log concentration in the cooked product was estimated as the log concentration in fresh ground beef as estimated by the Processing and Grinding submodel, plus an increase in log concentration due to microbial growth during retail display, minus log inactivation due to cooking.

The probability of some concentration of *E. coli* O157:H7 was the product of the prevalence of *E. coli* O157:H7-contaminated packages of fresh ground beef and the probability that viable organisms survived the growth/inactivation stages of post-processing.

3.2.3.1. Microbial growth

Growth of microorganisms may occur between the time of production and the time of consumption, and is influenced by the nature of the matrix (meat), temperature, length of storage time, and the behaviour of the specific organism under those conditions. Once the product is on display, loss of

control of the temperature of the meat and resultant microbial growth may occur. The amount of bacterial growth is dependent on many conditions of the food matrix, of which pH, percent NaCl, water activity (a_w), and temperature are most dominant.

The log increase in concentration was estimated using the modified Gompertz equation (Gibson et al., 1988), a commonly used mathematical model to predict the growth of microorganisms at constant temperature. The growth curve is sigmoidal with two shoulders and a period of rapid growth. Three parameters control the shape of the curve: C , B and M .

The parameter C is the difference between the maximum log population density and the lower asymptote of the growth curve. The lower asymptote is the initial concentration before the lag period, not simply any point defined by the model to be time zero. This initial concentration was assumed to be that in the freshly ground beef. The relative maximum growth rate, B , is related to the slope of the growth curve at the inflection point of the sigmoidal curve. It is affected by the intrinsic and extrinsic conditions of the matrix including pH, % NaCl and temperature. The parameter M is the length of time until maximum exponential growth occurs. Like relative maximum growth rate, it is affected by many conditions of the matrix.

Other than the previously discussed microbial proliferation during processing, the process was assumed to be under control until delivery to the retail outlet. The pH, % NaCl and a_w of ground beef were assumed to be constant. Temperature was presumed to be the determining factor in the magnitude of microbial growth. The temperature experienced by packages of ground beef on display is highly variable, both spatially and temporally. Average meat surface temperatures in the range of -1.7°C to 10°C have been observed in retail display cabinets (Greer et al., 1994).

Some experimental data are available for growth at fluctuating temperatures. Rajkowski and Marmar (1995) have fit the parameters of the Gompertz equation under several fluctuating temperature ranges. These investigators discovered that observed growth was in better agreement with estimates based on the maximum temperature experienced than estimates based on the average or mid-point of the temperature history. The distribution of maximum

temperature was assumed to have a minimum of 4°C, a mode of 10°C, and a maximum of 15°C_{TM}.

Predictions from Food MicroModelTM [Food MicroModel Ltd, Surrey, UK] were used to model the growth parameters. The parameter *C* was assumed to be between 7 and 9 log₁₀ CFU/g with a most likely value of 8.4. The parameters *B* and *M* were modelled as functions of maximum specific growth rate (μ_g) and lag time (t_1) which were fit as functions of temperature using linear regression on data from the Food MicroModel. The predictions of μ_g and t_1 from Food MicroModel were obtained using temperatures between 10 and 15°C, pH values between 5.1 and 6.1, and water activity values between 0.99 and 1.00. The linear regressions between temperature and μ_g and between temperature and t_1 predicted that no growth of *E. coli* O157:H7 would occur below 7.7°C.

3.2.3.2. Thermal inactivation

Cooking is likely the most effective barrier against exposure to *E. coli* O157:H7. This section of the post-processing model focused on the log reduction in concentration due to cooking. This parameter was modelled as a function of the final internal temperature of the hamburger. Thermal inactivation of *E. coli* O157:H7 was estimated using a linear model provided by Juneja et al. (1997). These investigators performed a regression analysis on data obtained from cooking experiments measuring log survivors of *E. coli* O157:H7 in hamburgers cooked to various internal temperatures (56.1–74.4°C). When fitting the model to the 20 collected internal temperature–log₂ survivor data points (including non-detects), the R^2 was 0.9139.

The internal temperature achieved by cooking a hamburger was assumed to be partially determined by the cooking preference of the consumer, and was thus based on consumer survey data. In a study of Texan consumers, McIntosh et al. (1994) reported the proportion of consumers who preferred hamburgers cooked rare, medium rare, medium, medium-well, and well-done (Table 4). A mean internal temperature was associated with each cooking preference (Jackson et al., 1996). For a given 'doneness', some variability of internal temperature is expected and therefore the internal temperature was modelled with a normal distribution with a standard deviation of 2°C.

Table 4

Internal temperature achieved by cooking hamburgers according to various 'doneness' preferences

Cooking preference	Percent of population ^a (McIntosh et al., 1994)	Internal temperature (Jackson et al., 1996)
Rare	3.0%	54.4°C
Medium rare	16.1%	58.6°C ^b
Medium	17.9%	62.7°C
Medium well	23.4%	65.6°C ^b
Well	39.6%	68.3°C

^a Normalized to sum to 100%.

^b Interpolated.

3.2.4. Consumption

The ingested dose is a function of the concentration of the organism in the beef at the time of consumption (an output of the *post-processing* sub-model), and the mass of hamburger ingested. The probability of exposure is the same as the prevalence of *E. coli* O157:H7 in the consumed product at the time of consumption.

The amount of hamburger ingested in a single adult meal was assumed to be distributed lognormally with a mean of 83 g and a standard deviation of 48 g. For children, the mean and standard deviation were assumed to be 42 and 27 g, respectively (Lin, personal communication).

3.3. Dose–response assessment

The dose response model estimated the probability of illness resulting from a certain level of exposure. The model used was based on the Beta-Poisson (BP) model for infection (Haas, 1983). The BP model predicts the percentage of the population which responds to a particular dose. This model uses parameters α and β which describe the distribution of susceptibility to the pathogen to characterize the variability between members of the population. The model assumes a non-threshold level of illness, i.e., that one cell is capable of causing illness, and that each cell is equally infective. The parameters α and β are fit to observed data for the response of interest, usually infection or frank disease.

A modified model, called the Beta-Binomial model, was developed which reflects the same assumptions used in the original BP model. However, the Beta-Binomial yields variability for probability of illness from a particular dose, in contrast to

the original model which only specifies a mean population risk.

The illness model was parameterized with the assumption that the virulence of the pathogen is similar to *Shigella dysenteriae*. The choice of parameter values for α and β are based on data from three published human feeding studies of two species of *Shigella* (*S. dysenteriae* and *S. flexneri*) reproduced in Crockett et al. (1995). Ross (1995) performed an analysis of the data using a hierarchical model for the synthesis of dose response datasets. Accordingly, variability between studies is used as a proxy for the uncertainty in the parameters α and β for the healthy adult population, and is included directly in the maximum likelihood estimation procedure. The result is a random coefficient model with predictive properties consistent with the data. Average parameter values obtained from this approach differ from individual parameter estimates determined by other methods, such as data pooling (Crockett et al., 1995). A discussion of estimation procedures for random coefficient models is given in Burnett et al.

(1995). Fig. 2 shows the dose response curve for the adult population based on the fit parameters. The figure shows considerable uncertainty in the probability of illness for a particular dose. The second inflection point depicted in the dose–response curve is believed to be inaccurate and an artifact of either the Monte Carlo sampling of rare events or the machine epsilon (a source of error – the smallest number larger than zero that a particular computer can use for calculations). Because the error is in the extreme tail of the dose–response curve, it is not believed to have any effect on the model results which predict low dose exposure.

The susceptible population was assumed to have a similar vulnerability to illness following ingestion of *E. coli* O157:H7, but an increased propensity for severe outcomes such as HUS. Thus, the same dose–response model was used to predict the probability of illness for susceptible groups. However, children under the age of five years and the elderly have an increased probability of severe outcomes such as HUS and mortality following infection (Griffin and

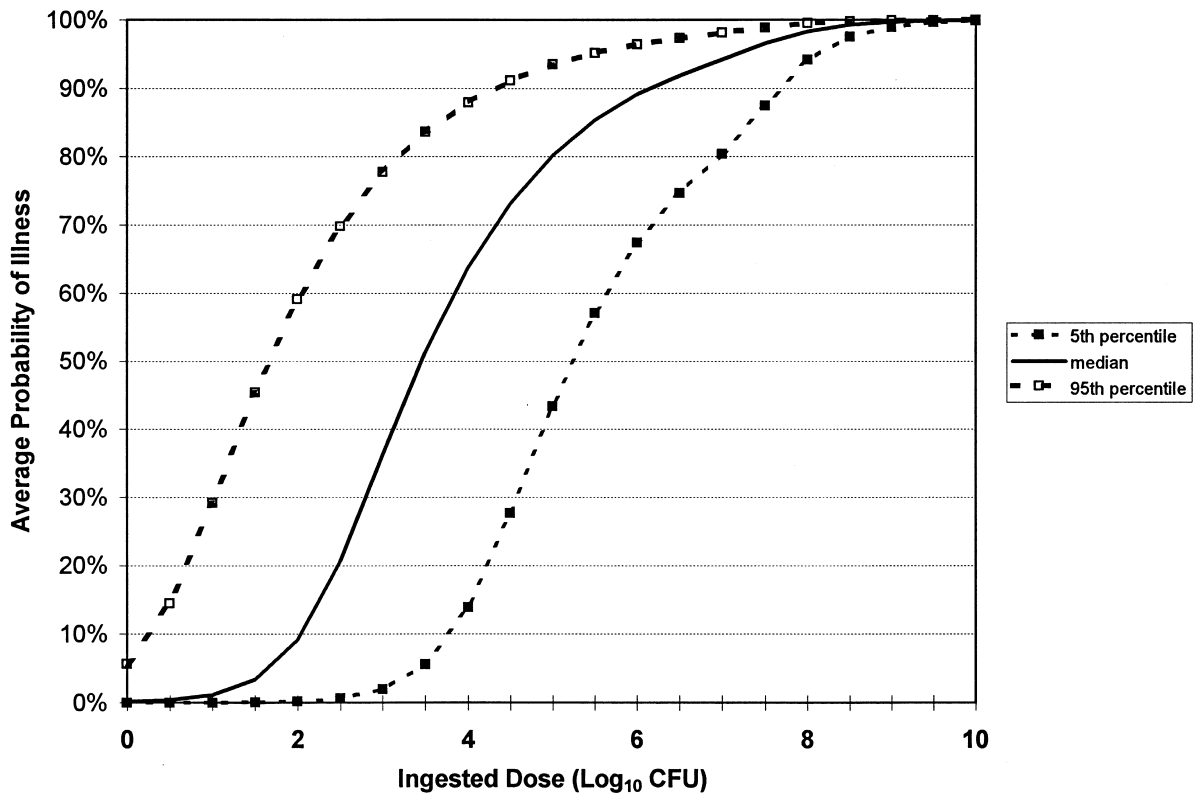


Fig. 2. Beta-Binomial dose–response model – Uncertainty in average probability of illness vs. ingested dose of *E. coli* O157:H7.

Table 5
Reported HUS and mortality case ratios following *E. coli* O157:H7 illness

Age	Ratio of HUS cases to illness cases ^a	Ratio of mortality to HUS cases ^a
Child	10% [37/371] (Bell et al., 1994) 10% (AGA, 1995)	5% (USDA, 1994a)
Adolescent		8% [3/37] (Bell et al., 1994)
Senior		12% [4/34] (Pavia et al., 1990)
All ages	9% [45/501] (Bell et al., 1994) 9% [56/611] (Vogt, 1994) 5% (Ries et al., 1993)	7% [3/45] (Bell et al., 1994) 7% [4/56] (Vogt, 1994) 8% [4/51] (Ryan et al., 1986)

^a Reported case ratios given in brackets if available.

Tauxe, 1991). The probability of severe outcomes such as HUS or mortality in the susceptible population was assumed to be some fraction of the probability of illness. In 1990, the U.S. Department of Commerce, Bureau of the Census, projected that 7.7% of the 1995 population would be under 5 years of age. Some data are available which suggest that the probability of HUS and mortality following HUS for this group are 10 and 5%, respectively (Table 5). For the elderly, the probability of HUS following infection is assumed to be the same as for the very young, but the probability of mortality following HUS was assumed to be 12% as the data in Table 5 suggest.

3.4. Risk calculation

The risk was estimated using predictions of the exposure assessment as inputs to the dose–response

model. The exposure assessment estimated the probability that there is a non-zero amount of *E. coli* O157:H7 in a meal. For non-zero exposure, a specific number of viable *E. coli* O157:H7 was predicted. The predicted exposure was the input for the dose–response model. Thus, the probability of illness was the product of the probability of non-zero exposure and the probability of illness from the output of the Beta-Binomial dose–response model.

4. Results and analysis

4.1. Simulation

An intermediate prediction of the model, the total number of *E. coli* O157:H7 in an *E. coli* O157:H7-contaminated package of fresh retail ground beef, is given in Fig. 3. The predicted distribution only

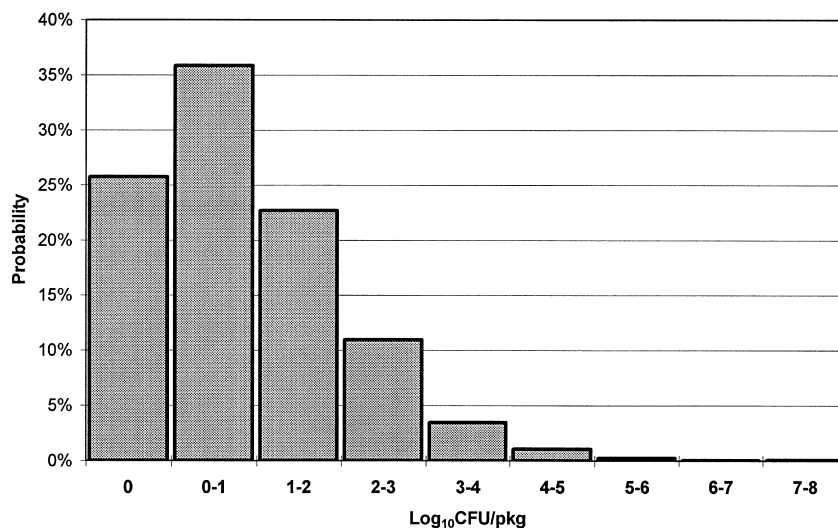


Fig. 3. Predicted total log₁₀ CFU of *E. coli* O157:H7 in a contaminated package of fresh retail ground beef.

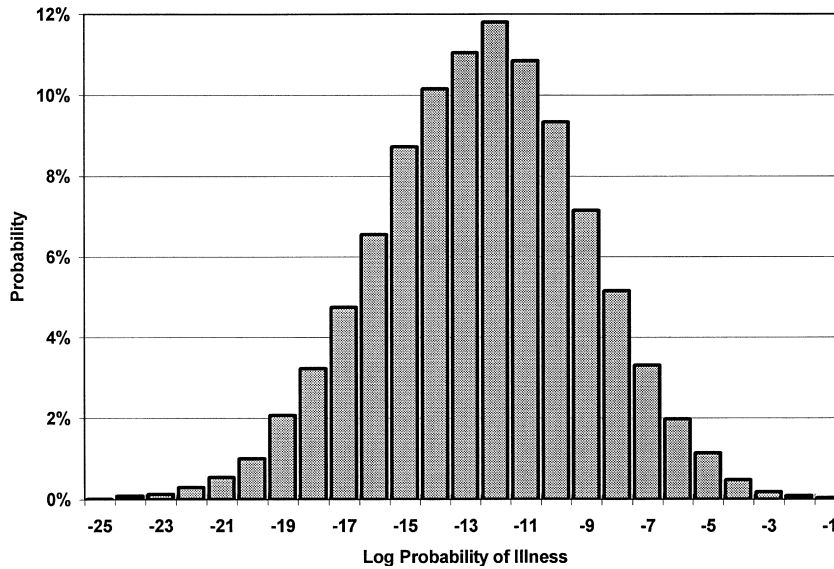


Fig. 4. Probability distribution for probability of illness from a single hamburger meal predicted by the *E. coli* O157:H7 Process Risk Model (PRM).

applies to those packages which contain *E. coli* O157:H7. The prevalence of such packages was predicted by the model to be 2.9%.

Fig. 4 shows the simulated distribution of probability of illness per meal. Each iteration predicted a probability of illness for a single hamburger meal. The range of this probability extended from 10^{-22} to 10^{-2} , including both the variability between meals and the uncertainty about the estimate. The distribution in Fig. 4 depicts the central tendency of the distribution at risk 10^{-12} . Log probability of illness was chosen as a convenient representation of the probability of risk, which is so concentrated near zero that it was not useful to display on a linear scale. This distribution does not have the same expected value as probability of illness, because the expected value is not constant across the log transformation.

4.2. Risk characterization

The distribution of risk to human health associated with *E. coli* O157:H7 and the consumption of hamburger is a complicated issue. In discussing the risk of illness, the probability of a particular health effect is commonly the outcome of interest. Usually, this probability is estimated without any knowledge of the particular set of circumstances which exist in

the consumption of one specific meal. In one circumstance, an individual may face a very high risk, say one in 100, by eating an undercooked hamburger prepared with meat from an abattoir that had recently slaughtered *E. coli* O157:H7-shedding cattle. The same individual may, on another occasion, could be subjected to a negligible risk such as one in 100 billion.

Thus, there is no one probability for illness resulting from the consumption of hamburgers, but rather there is a range of risk that is experienced by persons who eat rare hamburgers, by persons who are somehow susceptible to infection, or by any combination of scenarios. The conditional probability of illness given the particular production, handling, and consumption scenario described by an individual iteration of the simulation model is usually infinitesimal. As shown in Fig. 4, the risk for most scenarios is well less than one in 10 000 chance of illness. Most hamburger meals are predicted to present a very small risk to the consumer. However, if even a small percentage of scenarios present a non-negligible risk such as one in 10 000 or higher, the average risk of illness will be non-negligible. The average risk of illness is most significantly affected by the likelihood of those scenarios.

An informative risk indicator is the expected value of risk. This is the point estimate of the probability

of a particular health effect for a random individual eating a random hamburger using a random cooking method. The expected value of risk is often measured and compared to regulatory objectives to meet standards of acceptable risks. Although this practice is useful, information regarding the range of risk experienced by the population is lost. The extremes of the distribution of risk should not be forgotten, especially if those risks are focused on an identifiable high-risk segment of the population.

The average value of the probability of illness from a single meal for adult members of the population, obtained from simulation of the model, was estimated at 5.1×10^{-5} and for children the probability of illness was estimated to be 3.7×10^{-5} . Using the conditional probabilities of HUS and mortality following infection assumed for susceptible groups, the mean probability of HUS and mortality among children was estimated at 3.7×10^{-6} and 1.9×10^{-7} , respectively.

Assuming that half of the estimated 10 000–20 000 annual *E. coli* O157:H7 illnesses (Mark and Roberts, 1993) in the United States are related to hamburger meals, that 9% of people eat hamburgers on each day (Walls and Scott, 1997), and a population of 265 million persons in the United States, the probability of illness per hamburger meal would be estimated to be between 5.7×10^{-7} and 1.2×10^{-6} . Since this estimate of per meal chance of illness is based on Mark and Roberts' estimate of annual illness and the annual total number of hamburger meals, it is not strictly comparable to the estimate of the risk model which describes a very specific process. The PRM describes the consumption of retail ground beef as hamburger cooked in the home which might be expected to have a risk greater than, for example, that associated with the consumption of frozen patty hamburgers cooked in restaurants.

4.3. Importance analysis

Monte Carlo simulation can provide an importance analysis of the model to assist in the identification of critical points in the process that most significantly influence risk, i.e. those factors which are highly correlated with increased risk. An importance analysis was performed to provide a quantitative measure of determine the most important factors affecting the

risk to human health from *E. coli* O157:H7. This analysis was perhaps the most valuable outcome of the PRM. It has implications for HACCP strategies in the prioritizing of risk mitigation efforts and for improving the model by focusing research priorities. An importance analysis is quite difficult to perform analytically, but is readily available from a Monte Carlo simulation.

Importance analysis takes into account the sensitivity of the outcome to a factor and the uncertainty and variability of that factor. The outcome may be sensitive to a parameter, but the factor may not be important due to limited variability. For example, a particular individual's risk may be highly sensitive to the degree to which they cook their meat, but the cooking temperature may not be important because the individual always cooks their hamburger to the same degree of 'doneness'.

Because the importance of a factor considers both the uncertainty and the variability of the factor, it does not necessarily indicate that the factor can be used to mitigate risk. An uncertain factor may or may not contribute to risk depending on its unknown constant value. A variable factor may or may not contribute to risk depending on its changing value. Uncertainty may be reduced through research, whereas the magnitude of a factor due to variability may be reduced through process intervention.

The Spearman rank correlation coefficient (Morgan and Henrion, 1990) was used to measure importance. Rank correlation determines the degree to which large instances of a variable are associated with large instances of another variable. If the cases when the magnitude of the factor is large are strongly associated with those cases when the risk is high, then the capability of the factor to predict risk is strong and the factor is deemed important. The correlation coefficient lies between -1 (direct negative correlation) and $+1$ (direct positive correlation). Correlation values in the vicinity of zero indicate a weak predictive value of the variable. Fig. 5 shows a tornado chart identifying the fifteen predictive factors most highly correlated with risk. In this study, the risk was most sensitive to the concentration of *E. coli* O157:H7 in feces of animals shedding the pathogen. This highlights the risk mitigation strategy of screening the animals pre-slaughter in order to reduce the introduction of large numbers of the pathogen into the production environment. The pre-

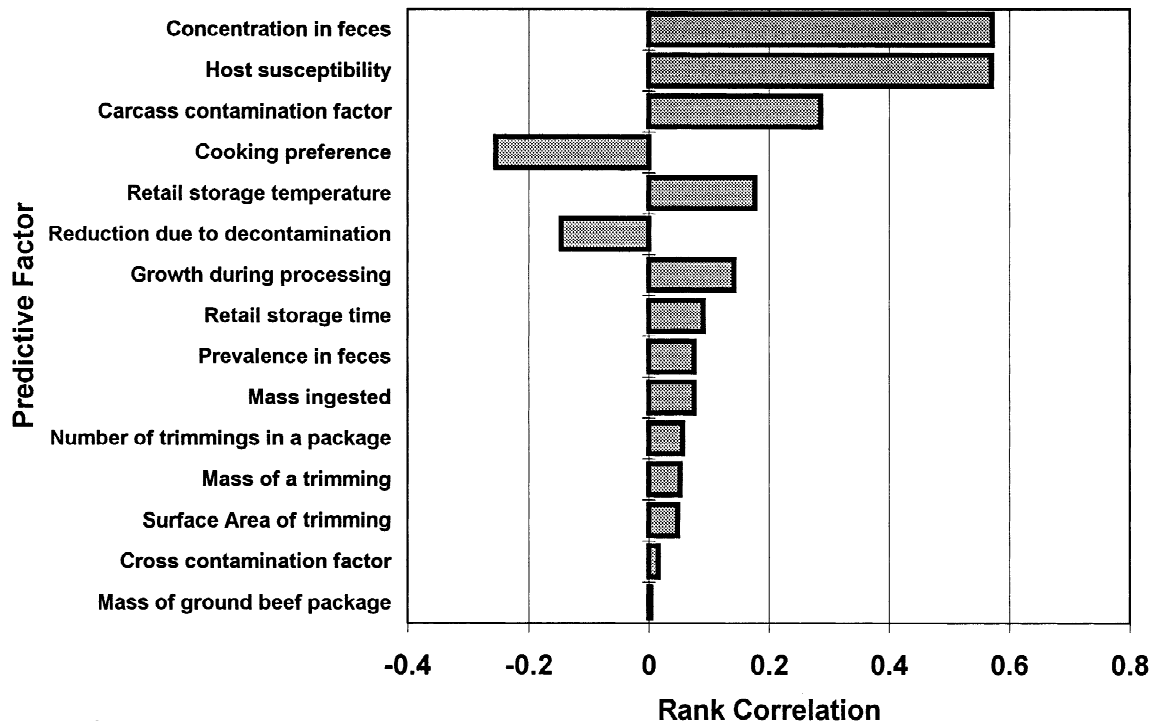


Fig. 5. Spearman rank correlation between the estimated probability of illness and the fifteen most important predictive factors of the Process Risk Model (PRM).

valence of cattle with the organism in their feces was not nearly as highly correlated with risk.

The probability of illness from a single organism, an indicator of host susceptibility, was also an important risk factor. The well-known risk associated with cooking preference was identified, and the probability of illness from a single organism, an indicator of host susceptibility, was also an important risk factor.

The risk was highly sensitive to the carcass contamination factor which is the amount of dilution between the concentration of the pathogen in the feces and the concentration on the carcass surface. Obviously, increasing the dilution factor, i.e. reducing the amount of feces which arrives on the meat, is desirable. This also suggested that a major uncertainty in the model may be the quantitative description of the process of carcass contamination, for which little information is available. As expected, storage time and temperature, affecting growth of the organism during retail display, were considered significant risk factors.

The importance of decontamination treatments indicated, subject to the validity of the model, that the observed reduction in counts does have a mitigating effect on risk. Additionally, microbial growth during processing was identified as a risk factor.

4.4. Risk mitigation strategies

The development of a PRM as a predictive risk tool provides an inexpensive technique to compare the efficacy of risk management options before they are implemented. By juxtaposing the relationships describing the hygienic effects of the sequential stages of food production, the effectiveness of unimplemented control strategies can be estimated. The efficacy of a risk mitigation strategy can be evaluated by modifying the values of the predictive factors and comparing the new predicted risk.

For example, the change in the predicted health effect endpoint, such as per meal risk, can be determined under different HACCP and non-HACCP strategies. In this way, the model acts as a predictive

tool for evaluating future scenarios, rather than presenting a static picture on the present risk to health. Simulation provides this important link between QRA and HACCP – one that has not been used in microbial risk assessment.

The tool can also be used to determine the required scope of an intervention strategy to achieve a regulatory objective. A sampling plan intended to monitor microbial contamination could be designed in a cost effective way. The cost of the sampling program, in terms of intervals and sensitivity, could be minimized subject to the constraint that the predicted health risk to the general population was reduced to fall within regulatory limits.

4.4.1. Hypothetical strategies

The PRM can evaluate the effect that a change in an assumption will have on the predicted risk to human health. This procedure was tested on the PRM for three hypothetical risk mitigation strategies. The effect of these strategies on predicted probability of illness per meal was examined to determine the most effective hypothetical control measure. The efficacy was measured as the percentage reduction of the per meal probability of illness predicted under the original assumptions for the parameter distributions.

Strategy 1: Storage temperature control

The original set of assumptions for the PRM stated that ground beef was subjected to a fluctuating temperature regime during storage and that the maximum temperature experienced was described by a random variable with a minimum of 4°C, a mode of 10°C, and a maximum of 15°C.

Suppose that compliance among retailers with some regulatory instruction is expected to reduce cases of temperature abuse. Suppose regulatory compliance estimates indicate that most retailers will store the product in such a way that the temperature will never exceed 8°C (i.e., the mode of maximum storage temperature), and that temperature abuse will be curbed such that in the worst case, the maximum temperature during storage is 13°C (i.e., the maximum of maximum storage temperature).

Strategy 2: Pre-slaughter screening

Suppose an alternative to strategy 1 is proposed. Perhaps, for the same price/effort, a pre-slaughter

Table 6

Risk mitigation strategy 3: Hypothetical effectiveness of a consumer information program directed at changing hamburger 'doneness' preferences among consumers

Cooking preference	Current preferences ^a (% of population) (McIntosh et al., 1994)	Hypothetical preferences after strategy 3 campaign
Rare	3.0%	2%
Medium rare	16.1%	10%
Medium	17.9%	18%
Medium well	23.4%	30%
Well	39.6%	40%

^aNormalized to sum to 100%

control could be put in place. For example, a certain feeding practice is expected to significantly reduce the number of animals shedding more than 4 log₁₀ CFU *E. coli* O157:H7 per g. The original assumption for the distribution of shedding levels was given in Table 1. For strategy 2, let us assume that 4 log₁₀ CFU/g is the 99th percentile rather than the 90th percentile of the distribution.

Strategy 3: Consumer information program

A third strategy might entail a consumer information program which attempts to convince people to cook ground beef more thoroughly. Limited compliance is expected, but the preferences in hamburger 'doneness' may shift according to Table 6.

4.4.2. Comparison of strategies

Table 7 reveals a comparison of the efficacy of the three strategies. The per meal probability of illness under the original model was considered the baseline. The efficacy of the various strategies was expressed as the percentage fewer illnesses predicted under the new set of assumptions.

The results are based upon the effectiveness of each of the management strategies as predicted by the PRM assuming that the hypothetical control strategies achieved the goals described. Given the compliance estimates used in the examples selected, the most effective control point would appear to be retail storage temperature. A control measure that achieves good compliance in reducing storage temperatures to the assumed distribution (minimum 4°C, mode 8°C, and maximum 13°C) was predicted to reduce the incidence of sporadic *E. coli* O157:H7 illnesses by 80%. Limited compliance with the

Table 7

Efficacy of hypothetical risk mitigation strategies: Percent reduction in predicted per meal illness from *E. coli* O157:H7 given assumed compliance (see text)

Strategy	Control variable	Predicted reduction in illness
1. Storage temperature control	Maximum storage temperature (T_s)	80%
2. Pre-slaughter screening	Concentration of <i>E. coli</i> O157:H7 in feces (C_F)	46%
3. Hamburger cooking. Consumer information program on cooking hamburgers	Cooking temperature (T_{CKG})	16%

hypothetical consumer information program to increase cooking temperatures was predicted to reduce the probability of illness by only 16%.

5. Discussion

The model described predicted the distribution of probability of illness attributable to *E. coli* O157:H7 in a particular ground beef hamburger manufacturing scenario. The model predicted risk by integrating predictive microbiology with techniques of quantitative risk assessment. An analysis of the important risk factors and a comparison of risk mitigation strategies was presented.

The predicted risk should be interpreted carefully. A particular meal may pose no risk or a very high risk to an individual considering the process by which it arrived on the plate of the consumer. Some individuals may always experience a higher or lower risk, due to their particular immunocompetence and their cooking habits. The expected value of risk is that risk experienced by an average person, consuming a hamburger meal processed in the described manner. The diversity of individual's immunocompetence and consumption patterns were averaged into the expected value of risk. No confidence limits or uncertainty bounds can be put on the expected value of risk because the distribution represents both uncertainty and variability. Treatment of variability and uncertainty separately would be necessary to estimate uncertainty bounds on the risk estimate, requiring more sophisticated simulation techniques.

The scope of a PRM is limited to a particular food production system. For this reason, a traditional QRA, and not this PRM, would be better suited to

estimating the annual number of illnesses. Because the estimation of risk was not the ultimate purpose of the PRM, analyses necessary for the estimation of annual risk were not included. The scope of a traditional QRA for *E. coli* O157:H7, describing all pathways of exposure, is not specific to a particular plant. All of these issues, among others, would need to be addressed to perform a risk estimate but are excluded from the PRM:

- An individual consumes retail ground beef from more than one production facility.
- The retailer may be supplied with beef from several domestic and international sources.
- The beef may be contaminated with sheep, pork, or poultry
- The retailer may also augment the product with their own trimmings from primal cuts having a different microbiological profile.
- Hamburger patties may be frozen prior to consumption.
- Hamburgers may be consumed from a restaurant.

The PRM was developed to provide a means to analyze the relationship between risk and factors which might be used to mitigate risk. A risk manager is likely to be more interested in the importance analysis and comparison of intervention strategies than the risk distribution presented in Fig. 4. Importance analysis of the model input parameters identified several factors which contribute significant uncertainty to the total uncertainty of the risk of illness prediction. Possible interventions can be deduced from controllable variables which have an important contribution to risk. Hypothetical control strategies were simulated, demonstrating the applica-

tion of the PRM approach for decision-making. Using this approach for comparison of possible management options de-emphasizes the importance of the actual risk estimate, and emphasizes the relative risk estimates under possible intervention options. This allows consideration and allocation of resources to potential risk reduction strategies that may be immediately feasible, while at the same time identifying priorities for focused and longer term research to better understand, and intervene, at critical stages of the process.

In this document, several parameters were modelled using data from a wide variety of processes, such as the log reduction in counts resulting from various types of spray washes and steam-vacuum sanitizers. The PRM for a specific plant might include the range of reduction expected for a particular technique. A decision-maker could estimate the value of switching from the current spray washing technique to a more effective or less costly pressure-temperature combination by altering the model parameters.

Obviously, the conclusions of this model are only accurate to the extent that the model accurately represents the process. Model validation is an important consideration. Other estimates of probability of illness may not be specifically comparable to the predictions based upon the particular scenario of the PRM. Therefore, validation is not necessarily conferred by agreement with other estimates of probability of illness, nor does disagreement invalidate. Another approach involves comparing intermediate predictions of the model to reported data. The predicted total *E. coli* O157:H7 in contaminated packages of retail ground beef appears sound. Considering the assumed package size of 300–1000 g, Fig. 3 illustrates that the majority of the 2.9% of packages predicted to be contaminated with *E. coli* O157:H7 were predicted to be contaminated at concentrations below the sensitivity of most *E. coli* O157:H7 detection methods. Eighty-seven percent of contaminated packages were predicted to contain fewer than ten *E. coli* O157:H7, which is less than 0.03 CFU/g in a 300 g package. Packages containing $10-10^4$ *E. coli* O157:H7 were predicted to be rare, accounting for the remaining 13% of the contaminated packages, or 0.3% of all packages. These predictions are quite similar to observed counts (Johnson et al., 1995; Wells et al., 1983; Todd et al., 1988).

Although the results seem reasonable, there are obvious areas of improvement for the model. There are steps in the production, processing, distribution, and consumption of hamburgers that are missing from the model. For example, the dilution factor used to estimate the concentration of the fecal material on the carcass is an unsatisfactory model of the process of contamination. A more accurate representation of the sources of cross-contamination is necessary. Some dressing processes include steam pasteurization of the carcass which significantly affects the bacterial counts. More complex and appropriate growth models, possibly using time-temperature history rather than maximum temperature, are needed. The homogenous distribution of feces on the carcass and the pathogen on beef and in ground beef is assumed throughout this paper; an assumption of some clustering may yield different results. The possibility that some of the *E. coli* O157:H7 that survive cooking may not be fully infective was not incorporated in the model. The dose-response relationship is likely to be inaccurate because it was based upon feeding studies, involving a different organism, and using healthy adults. The probability of HUS and mortality following infection may not be independent of dose and are likely variable rather than fixed, as more variation is observed in outbreaks than can be explained by simple binomial variability. Several assumptions were made due to an inability to obtain empirical data. Some data are likely available that were not included in this model and other statistical interpretations of the data may be equally valid or more appropriate. The results of the risk mitigation strategies were a function not only of the model, but also of the assumptions made for effectiveness of the particular hypothetical scenarios tested. The model did not demonstrate that retail temperature control per se is more effective than end-product cooking, but rather that the level of consumer compliance in end-product cooking would be less than the supposed level of compliance in retail storage temperature control, and hence the end result was a greater reduction in risk using the latter strategy.

A review of the model indicates additional factors that could be considered in the process, and specific stages that might warrant more detailed examination to better understand the significant risk factors. For example, future considerations and refinements of

the model might include quantifying the effects of factors such as feed withdrawal and animal-transport conditions on levels of pathogen shedding (Armstrong et al., 1996). A significant factor, the concentration of the organism in the feces of shedding cattle, was based on limited data. The data used to describe this parameter were derived from a small sample size in a single study (Zhao et al., 1995). Future research should be directed at refining this distribution, especially in determining the probability of very high shedding levels, as increased confidence in this distribution would be of great value to the exposure assessment.

Future work will include a separation of uncertainty and variability in the model assumptions. The performance of a two-dimensional Monte-Carlo analysis, simulating variability in each of several simulations of uncertainty, will provide information to differentiate between uncertainty that can be reduced through research and variability which can be reduced through control.

Further refinements to the PRM are required to provide an accurate representation of the process of ground beef production, distribution, and consumption. There exist steps in the process and scenarios of contamination that were aggregated or not considered in the model. Detailed criticism, experimentation, and validation is necessary to make such improvements. The risk estimate should be validated as individual assumptions are challenged and revised.

With continuous improvement, the model should encompass available information about the process,

food, and pathogen. The model then should be the most appropriate decision-support tool, since it represents current knowledge. The likely possibility that the model under or overestimates the probability of illness from consumption of ground beef hamburger does not prohibit its use as a decision-making tool. The accuracy of the specification of the knowledge that is available is important. If the model is able to rank the efficacy of risk mitigation strategies with some accuracy, then the risk estimate can be used as a quantitative measure of process safety. Efforts for risk mitigation should be focused on reducing the risk estimate, even if this represents a relative, rather than absolute value.

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Appendix 1. Detailed model

Table A1. Production

Variable	Description	Units	Distribution/Model
C_F	Concentration of <i>E. coli</i> O157:H7 (H7) in contaminated feces	\log_{10} CFU/g	Custom distribution (Table 1)
P_F	Prevalence of <i>E. coli</i> O157:H7 in cattle feces		Beta (2.7, 250) Fit from data in Table 2

Table A2. Processing and grinding

Variable	Description	Units	Distribution/Model
F_{CC}	Factor for cross-contamination of carcasses	–	Uniform (2, 3) ^a
F_{DIL}	Log dilution factor between fecal and carcass surface concentration	$\log_{10} \text{ g/cm}^2$	Normal (–5.1, 0.9) (based on assumptions in text and data in Table 3) ^b
R_{DEC}	Log reduction in counts due to decontamination treatments	$\log_{10} \text{ CFU/cm}^2$	Uniform (1, 2.5)
G_{PRC}	Microbial growth during processing	generations	Triangular (–2, 0.5) ^c
m_{TRM}	Mass of a trimming destined for ground beef	g	Normal (300, 100), truncated to disallow mass below 50 g
A_{APG}	Surface area per gram of trimming	cm^2/g	Uniform (0.25, 1)
A_{TRM}	Average surface area of trimmings	cm^2	$m_{TRM} \times A_{APG}$
m_{PKG}	Mass of a vacuum pack of trimmings	g	5000
N_{TRM}	Number of trimmings in a package	–	Custom distribution simulated using m_{PKG} and m_{TRM}
P_{CTRM}	Prevalence of contaminated trimmings	–	$F_{CC} \times P_F / (1 - P_F + F_{CC} \times P_F)$
N_{CTRM}	Number of H7 ^d contaminated trimmings in a package	–	Binomial (N_{TRM}, P_{CTRM}) ^e
C_{CTRM}	Concentration of H7 on contaminated trimmings	$\log_{10} \text{ CFU/cm}^2$	$C_F + F_{DIL} - R_{DEC} + \log_{10} 2^{G_{PRC}}$
C_{PCT}	Concentration of H7 in a package of trimmings	$\log_{10} \text{ CFU/g}$	$\log_{10}(N_{CTRM} \times 10^{C_{CTRM}} \times A_{TRM} m_{PKG})$
P_{PCT}	Probability of H7 in packages of trimmings	–	$\Pr(N_{CTRM} > 0) = 1 - (1 - P_{CTRM})^{N_{TRM}}$
m_{FGB}	Mass of a retail package of fresh ground beef (FGB)	g	Triangular (300, 500, 1000)
C_{FGB}	Concentration of H7 in contaminated FGB	$\log_{10} \text{ CFU/g}$	$\log_{10}(N/m_{FGB})$ where $N \sim \text{Poisson}(m_{FGB} \times 10^{C_{PCT}})$ ^e
P_{FGB}	Probability of H7 in FGB	–	$P_{PCT} \times (1 - e^{-m_{FGB} \times 10^{C_{PCT}}})$ ^f

^a Uniform (min, max).^b Normal (μ, σ).^c Triangular (min, mode, max).^d H7: *E. coli* O157:H7.^e Only non-zero values simulated in each iteration.^f Accounts for truncation of zero-valued iterations.

Table A3. Post-processing

Variable	Description	Units	Distributional assumption
t_s	Time on retail display	h	Triangular (4, 48, 96)
T_s	Maximum retail storage temperature	°C	Triangular (4, 10, 15)
μ_g	maximum exponential growth rate	h^{-1}	$\mu_g = C_0 + C_1 \times T_s$
t_l	lag time	h	$t_l = C_2 + C_3 / \mu_g$

Table A3. (Continued)

Variable	Description	Units	Distributional assumption
C_0, C_1, C_2, C_3	Regression parameters, fitted to predictions of Food Micromodel TM	–	Regression method and Food Micromodel data are described in text.
C	Gompertz equation: maximum population density	–	Triangular (7, 8.4, 9) – C_{FGB}
M	Gompertz equation: time to maximum growth	h	$M = t_l + 1/B$
B	Gompertz equation: exponential growth rate	h^{-1}	$(\mu_g \times C)/e$
G_{RTL}	Growth during retail storage	–	$G_{\text{RTL}} = C \cdot e^{-e^{[-B(t_g - M)]}}$
K_0	Thermal inactivation model: regression coefficient	\log_{10} CFU/g	–10.165
K_1	Thermal inactivation model: regression coefficient	\log_{10} CFU/g °C	0.211
T_{CKG}	Internal temperature of cooked hamburger	°C	Custom distribution (Table 4)
I_{CKG}	Thermal inactivation from cooking	\log_{10} CFU/g	$I_{\text{CKG}} = K_0 + K_1 T_{\text{CKG}}$
C_{CKGB}	Concentration in cooked ground beef	\log_{10} CFU/g	$C_{\text{CKGB}} = C_{\text{FGB}} + G_{\text{RTL}} - I_{\text{CKG}}$

Table A4. Consumption

Variable	Description	Units	Distributional Assumption
D	Ingested dose of <i>E. coli</i> O157:H7	CFU	$D \sim \text{Poisson}(10^{C_{\text{CKGB}}} \times m_l)^a$
m_l	Mass of hamburger ingested	g	Adult: Lognormal (84, 48) ^b Child: Lognormal (42, 27) ^b
P_E	Probability of exposure to <i>E. coli</i> O157:H7	–	$P_E = P(D > 0) = P_{\text{FGB}} \times (1 - e^{-10^{C_{\text{CKGB}}} \times m_l})^c$

^a Only non-zero values simulated in each iteration.

^b Lognormal (μ, σ).

^c Accounts for truncation of zero-valued iterations.

Table A5. Dose–response assessment

Variable	Description	Distributional assumption
$P_i(D)$	Probability of illness from dose: Beta Binomial model	$P_i(D) = 1 - (1 - P_i(1))^D$
$P_i(1)$	Probability of illness from exposure to one organism	Beta (α, β)
α	Susceptibility parameter	0.267
β	Susceptibility parameter	$\ln \beta \sim \text{Normal}(5.435, 2.47)^a$

^a Normal ($\mu_{\ln \beta}, \sigma_{\ln \beta}$).

Table A6. Risk calculation

Variable	Description	Distributional assumption
P_I	Probability of illness	$P_I = P_E \times P_I(D)$
$P_{HUS I}$	Probability of HUS given illness	10%
$P_{MORT HUS}$	Probability of mortality given HUS	Children: 5% Elderly: 12%
P_{HUS}	Probability of HUS	$P_{HUS} = P_{HUS I} \times P_I(D)$
P_{MORT}	Probability of mortality	$P_{MORT} = P_{MORT HUS} \times P_{HUS}$

This appendix contains the details of the mathematical model used to describe a specific process of production, distribution, preparation, and consumption of ground beef as hamburgers. A table for each major section of the paper provides the distributional assumptions or function which models the variability and/or uncertainty in the model variables, along with variable names and units.

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