Risk Analysis, Vol. 35, No. 1, 2015 DOI: 10.1111/risa.12248

Risk Assessment of *Escherichia coli* O157 Illness from Consumption of Hamburgers in the United States Made from Australian Manufacturing Beef

Andreas Kiermeier,[1,*] Ian Jenson,[2] and John Sumner[2]

We analyze the risk of contracting illness due to the consumption in the United States of hamburgers contaminated with enterohemorrhagic *Escherichia coli* (EHEC) of serogroup O157 produced from manufacturing beef imported from Australia. We have used a novel approach for estimating risk by using the prevalence and concentration estimates of *E. coli* O157 in lots of beef that were withdrawn from the export chain following detection of the pathogen. For the purpose of the present assessment an assumption was that no product is removed from the supply chain following testing. This, together with a number of additional conservative assumptions, leads to an overestimation of *E. coli* O157-associated illness attributable to the consumption of ground beef patties manufactured only from Australian beef. We predict 49.6 illnesses (95%: 0.0–148.6) from the 2.46 billion hamburgers made from 155,000 t of Australian manufacturing beef exported to the United States in 2012. All these illness were due to undercooking in the home and less than one illness is predicted from consumption of hamburgers cooked to a temperature of 68 °C in quick-service restaurants.

KEY WORDS: Consumption; cooking; *E. coli* O157 concentration; *E. coli* O157 prevalence; lot contamination; quick-service restaurants

1. INTRODUCTION

The most recent estimate of foodborne illness in the United States[(1)] is that, of 9.4 million cases caused by 31 major pathogens, almost 56,000 result in hospitalization and 1,351 in death. An important foodborne pathogen continues to be enterohemorrhagic *Escherichia coli* (EHEC) of serotype O157, due both to its incidence, estimated to be around 63,000 foodborne cases/annum,[(1)] and to the severity of illness (2,138 hospitalizations and 20 deaths/annum),[(1)] especially among children.

[1]Statistical Process Improvement Consulting and Training Pty Ltd., Adelaide, Australia.
[2]Meat & Livestock Australia, Sydney, Australia.
*Address correspondence to Andreas Kiermeier, Statistical Process Improvement Consulting and Training Pty Ltd., Adelaide, Australia; tel: +61423028565; andreas.kiermeier@gmail.com.

The first documented outbreak of EHEC illness involving hamburgers occurred in Oregon and Michigan in 1982, with the report citing "*a rare E. coli serotype O157:H7*."[(2)] In 1992–1993, outbreaks involving more than 500 people in the western United States were linked to the consumption of undercooked hamburgers contaminated with *E. coli* O157.[(3)] Between 1982 and 2002, 350 outbreaks were attributed to *E. coli* O157, from which there were 8,598 illnesses (defined as isolation of *E. coli* O157 from stools, or bloody diarrhea or hemolytic uremic syndrome—HUS), 1,493 hospitalizations, 354 cases of HUS, and 40 deaths.[(4)] Of these, transmission routes were determined for 276 (79%), with food implicated in 183 (52%), person–person 50 (14%), recreational water 21 (6%), animal contact 11 (3%), drinking water 10 (3%), and laboratory-related 1 (<1%). Of foods implicated, a source was

determined in 141 (77%) outbreaks. Ground beef or other forms of beef were the major source of both outbreaks (47%) and cases (44%) with most (71%) occurring over a four-month period (May–August); 68% of cases involved hamburgers and 13% a meat-based sauce. Outbreaks involving ground beef occurred most frequently at the community level (48%), picnics/camps (15%), residences (11%), restaurants (9%), and schools (5%). Of the seven outbreaks associated with restaurants, five occurred in quick-service restaurants (QSR)—two in 1982, one in 1992–1993, one in 1995, and one in 1999. Of the 11 outbreaks involving "other beef," five were associated with roast beef, three with steak or sirloin tips, and one with salami; two outbreaks were identified only as "beef" or "raw roast beef."[4]

In the ensuing two decades since the large outbreak in the western United States, the red meat industry has been required to respond to a series of regulations aimed at preventing EHEC illness, particularly due to serotype O157 and more recently serotypes O26, O45, O103, O111, O121, and O145.[5] Over the same period, risk assessments to estimate the likelihood of illness from consuming hamburgers (and sometimes other ground meat products) contaminated with EHEC have been carried out in Canada,[6,7] the United States,[8] Ireland,[9] France,[10] Argentina,[11] and the United Kingdom.[12] Perhaps unsurprisingly, given different conditions of livestock-raising, meat-processing, and consumption patterns, estimates of illness vary widely from 1/10,000,000 servings[8] to 800/10,000,000 servings.[12] Approaches to these risk assessments were based on through-chain, farm-to-fork models, with the exception of the Irish assessment where a survey of retail ground beef was the starting point.[9]

For several decades Australia has been a major supplier to the United States of manufacturing beef intended for grinding, with exports in 2012 155,000 t, equating to around 2.46 billion hamburger patties. A major reason why manufacturing beef is imported from Australia is because of its low fat content (80–95% chemical lean), which enables blending with local U.S. meat from feedlot cattle with a higher fat content (around 50% chemical lean).While the microbiological status of Australian beef used for grinding in the United States has been well documented,[13–15] there has been no assessment of the extent to which Australian beef might contribute to *E. coli* O157 illness in the United States from consumption of hamburgers. Recently, the opportunity arose to estimate this risk, and also to estimate it using a novel approach based on the prevalence and concentration of *E. coli* O157 in lots of beef that had been withdrawn from the export chain following the detection of the pathogen by the N-60 testing regime used in testing of all export lots destined for grinding in the United States.[16]

The present article reports the results of a risk assessment of *E. coli* O157 from the consumption of beef burgers made only from lots of Australian manufacturing beef. Starting with the prevalence and concentration of *E. coli* O157 in lots of Australian manufacturing beef, contamination was modeled from grinding and patty forming through to cooking and consumption. For retail storage, transport home, home storage, and dose-response model this assessment followed a similar approach to the draft U.S. risk assessment.[8] The aim of this risk assessment was to estimate the risk of *E. coli* O157 illness from lots of Australian beef if purely Australian beef burgers were consumed in the United States, and particularly, how many illnesses might be expected per lot.

2. MATERIALS AND METHODS

2.1. Model Overview

Australian manufacturing beef (trim) is supplied frozen in 60 lb (27.2 kg) cartons, arranged in container lots of up to 700 cartons. The majority of Australian manufacturing beef is lean (chemical lean 80–95%) and is often mixed in the United States with meat of higher fat content during grinding. However, to assess the food safety risk associated with Australian beef, for the purposes of the present assessment it was assumed that Australian beef is made into "Australian" patties, with no comingling. Two product streams were modeled: a retail stream in which chilled patties/ground beef is purchased, transported to the home where it is refrigerated before being cooked and consumed, and a QSR stream where frozen patties are cooked to a fixed temperature rather than to the preference of the consumer. The process model is summarized in Fig. 1.

In the simulation model, lots of 700 cartons (27.2 kg) of beef trimmings were considered as the unit of interest since this is typically the mass exported to the United States in one shipping container. Consequently, each lot yields patties of varying size of which 50 g and 100 g patties have been used for the current assessment. Hence, for each lot a total of 380,800 × 50 g or 190,400 × 100 g patties are simulated. After patty production the

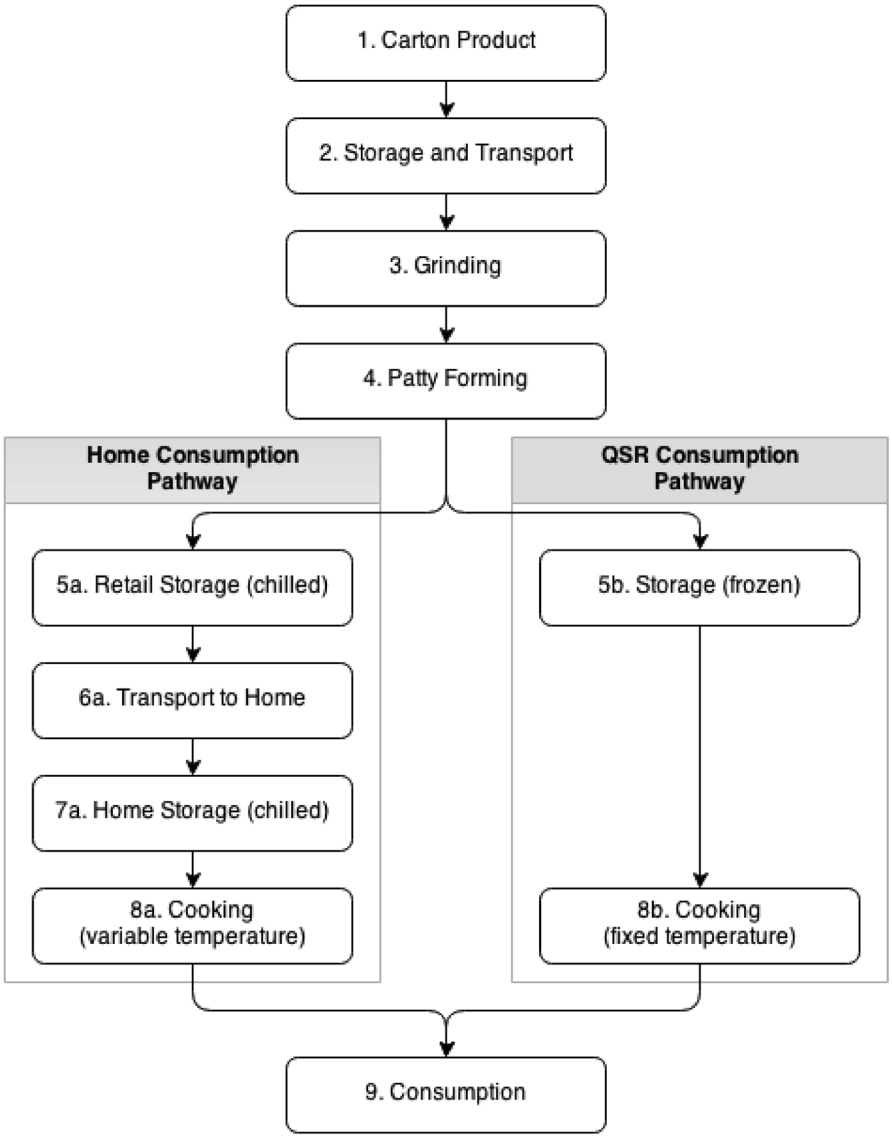

Fig. 1. Process model for consumption of hamburgers in the United States based on meat ground from Australian manufacturing beef: Step 2. "Storage and Transport" is not explicitly modeled and Steps 3 and 4 are modeled as one.

process from retail storage to consumption is modeled independently for each patty, that is, using independent random draws from the appropriate distributions. Finally, for each lot the illnesses from all patties are summarized. A total of 10,000 contaminated lots were simulated per scenario, with the exception of the QSR scenarios, which required 100,000 lots to be simulated to yield adequate illness estimates.

2.2. Exposure Assessment

In Australia, export establishments test for the presence of *E. coli* O157 in beef destined for grinding in the United States under the supervision of the Department of Agriculture using laboratories accredited to ISO 17025 by the National Association of Testing Authorities (NATA). Testing is performed on 375 g of surface meat, which is made up of five samples from each of 12 randomly selected cartons.[17] The sampling protocol requires that the 60 subsamples are enriched and tested for the presence of *E. coli* O157, so-called N-60 testing. Normally, and as part of Australia's regulatory obligations, all lots are tested and released into the marketplace only if *E. coli* O157 is not detected from the 375 g sample. However, for the purpose of this

risk assessment it was assumed that contaminated lots were not removed from the export supply chain due to N-60 testing at the processing plant and were shipped to the United States for grinding into patties. In addition, it was assumed that the contaminated lots that were detected during routine export certification testing[16] are representative of all other contaminated lots.

Previous work indicated that only a small fraction (0.5%) of Australian lots are likely contaminated with *E. coli* O157.[16]Hence, the efficiency of simulation could be improved by only simulating contaminated lots. This was done because uncontaminated lots do not add to the risk (constant risk that equals zero); however, the fraction of contaminated lots is taken into account for the calculation of final risk estimates.

The model was implemented in the open-source software R version 2.15.1,[18] utilizing functions from packages mc2d version 0.1–13[19] and fitdistrplus version 0.3–4.[20] The model was run for 10,000 contaminated lots of 700 cartons each, which were ground into 100 g hamburger patties. Since whole lots were modeled it was not possible to utilize the two-dimensional structure of the mc2d package to separate uncertainty and variability. A patty size of 50 g was also assessed as a scenario. Details of the model are described below and a summary of the variables, parameters, distributions, and associated references are provided in Table I.

2.2.1. Proportion of Contaminated Cartons in Contaminated Lots

The distribution and concentration (cfu/cm^2) of *E. coli* O157 in five lots of manufacturing beef that had failed to meet Australian requirements for export to the United States have previously been estimated.[16] There were four lots in which one carton was contaminated and one lot that had two contaminated cartons out of the 12 cartons that were sampled. However, due to the small number of cartons sampled per lot the subsequent carton prevalence estimates are uncertain.

Assuming that the extent of contamination in contaminated lots occurs at the above frequencies, that is, 4/5 and 1/5, the proportion of contaminated cartons (p_c) in a lot was modeled by a Beta distribution, that is, $p_c \sim \text{Beta}(X_{12}+1, 12+1)$, with $X_{12} = 1$ with probability 4/5 and $X_{12} = 2$ with probability 1/5.

Subsequently, for each carton i ($i = 1 \ldots 700$) in the contaminated lot the carton was either not contaminated ($Z_i = 0$) or was contaminated ($Z_i = 1$), where Z_i is the outcome from a Bernoulli trial, that is, $Z_i \sim \text{Bern}(p_c)$.

2.2.2. Concentration of E. coli O157 in Contaminated Lots

Based on the six contaminated cartons identified, the average concentration estimates, determined by the most probable number (MPN) technique, were <0.0013 (in one carton from each of three lots from which *E. coli* O157 could not be isolated again), and 0.0014, 0.019, and 0.093 cfu/cm^2 of external carcass surface.[16] A log-normal distribution (natural log) was fitted to these MPNs to model the distribution of concentration in a carton $\Lambda \sim \text{lnN}(-6.63, 2.91^2)$—the distribution and parameter estimates (mean = −6.63 and standard deviation = 2.91) were determined using the fitdistrplus package, which aids in fitting distributions to data.[20] For each contaminated carton, the concentration Λ (cfu/cm^2) was then multiplied by the external carcass surface area in the carton, which was sampled from the empirical distribution of carcass surface area in a carton (S_c),[16] to give the concentration per carton ($\lambda_c = \Lambda \times S_c$). This λ_c was then used as the parameter in a zero-truncated Poisson distribution $\text{Po}_z(\lambda_c)$ to give realizations of the number of organisms in a carton (Y_c)—the zero truncation was used as these cartons had already been determined to be contaminated and hence a realization of zero organisms, that is, $Y_c = 0$, is not possible.

2.2.3. Effect of Frozen Storage

The above-mentioned calculations of *E. coli* O157 concentrations[16] were based on cartons of frozen manufacturing beef and an assumption was made that no further reduction occurred during further frozen storage (in carton or patty form). This is probably conservative as varying degrees (0.5 and 2 $\log_{10}$ cfu) of sublethal injury during frozen storage of *E. coli* O157 for 12 weeks have been observed.[21]

2.2.4. Effect of Grinding into Patties

It is acknowledged that practices and equipment differ between grinding establishments. For example, it is industry practice to mix relatively fatty and lean lots of beef into a grinder to produce a patty to a particular fat specification. However, without information about how contamination is dispersed during

Table I. Summary of Model Inputs

Parameter	Description	Distribution	Reference
N_c	Number of 27.2 kg cartons per lot	Fixed: 700	
W_b	Weight of a hamburger patty	Fixed: 0.05 or 0.1 kg	
S_c	External carcass surface area in a carton	Empirical	Kiermeier *et al.*[16]
X_{12}	Number of contaminated cartons in a sample of 12	Empirical (values = (1,2), prob = (0.8,0.2)	Kiermeier *et al.*[16]
p_c	Proportion of contaminated cartons in a lot	Beta(X_{12}+1, 12+1)	Kiermeier *et al.*[16]
Z_i	Indicates if carton in the lot is contaminated ($Z_i = 1$) or not contaminated ($Z_i = 0$) for $I = 1 \ldots 700$ cartons in each lot	Bern ($p = p_c$)	Kiermeier *et al.*[16]
$\boldsymbol{\alpha}$	Comminution parameter	Fixed: $\alpha_i = 1$	
Λ	Concentration (cfu/cm^2)	log normal(-6.63, 2.91^2)	Kiermeier *et al.*[16]
λ_c	Concentration per carton (cfu/carton)	$\Lambda \times S_c$	
Y_c	Number of *E. coli* O157 organisms in a contaminated carton	Po$_z$(λ_c)	
$\boldsymbol{p_p}$	Vector of the expected proportion of organisms from a carton that end up in a hamburger patty	Dirichlet ($\boldsymbol{\alpha}$)	Pouillot (personal communication, 2009)
$Y_{p,c}$	The number of organisms in each of the hamburger patties produced from a single carton	Multinomial ($Y_c, \boldsymbol{p_p}$)	Nauta[22] Pouillot (personal communication, 2009)
D_{ret}	Duration of hamburger patty storage at retail	Empirical	EcoSure[25]
T_{ret}	Temperature of hamburger patty storage at retail	Empirical	EcoSure[25]
D_{trans}	Duration of hamburger patty storage during transport to home	Joint empirical	EcoSure[25]
T_{trans}	Temperature of hamburger patty storage during transport to home		EcoSure[25]
D_{home}	Duration of hamburger patty storage at home	Empirical	EcoSure[25]
T_{home}	Temperature of hamburger patty storage at home	Empirical	EcoSure[25]
T_{cook}	Temperature at the center of a hamburger patty after cooking	Empirical	EcoSure[25]
α_{DR}	Dose-response curve: susceptibility parameter	Fixed: 0.267	Cassin *et al.*[6]
β_{DR}	Dose-response curve: susceptibility parameter	Lognormal (5.435, 2.47^2)	Cassin *et al.*[6]

large-scale grinding, the assumption was made that each carton was ground independently of any other carton and that there was no cross-contamination between cartons. In addition, we assumed that Australian beef was not mixed with product of different origin to confine this assessment to the risk of *E. coli* O157 from Australian beef.

Modeling the dispersion of organisms in the carton over all the 544 × 50 g or 272 × 100 g patties in a carton was done via a two-step approach.[22] For each contaminated carton, the first step was to model the (average) proportion of organisms ($\boldsymbol{p_p}$ a vector of probabilities) to go into each patty using a Dirichlet distribution (R. Pouillot, 2009, personal communication) with a "comminution" parameter vector $\boldsymbol{\alpha} = (\alpha_1, \alpha_2, \ldots)$. In the present context all α_is are the same, as patty sizes are constant, with each patty having an equal chance of being contaminated as a result of thorough grinding and mixing. The size of α_i affects the variability in the probability of an organism ending up in patty *i*. For example, if α_i is large (e.g., 1,000), then each patty has basically an equal chance, while if α_i is small (e.g., 0.1), then most patties will have close to zero probability of containing organisms, and a few will have high probability. Studies on grinding[23,24] showed that *E. coli* added to meat during grinding tended to leave the grinder with little tailing, prompting the assumption that $\alpha_i = 1$ for a baseline scenario. Alternative scenarios using $\alpha_i = 0.5$ or 5 were also investigated in the sensitivity analysis.

For the second step the total number of organisms in a contaminated carton (Y_c) were distributed to individual hamburger patties ($Y_{p,c}$) using a multinomial distribution with probabilities $\boldsymbol{p_p}$.

2.2.5. *Growth During Retail Storage*

For hamburger patties sold through QSRs, it was assumed that they are stored at −20 °C (Fig. 1) until they are cooked and hence growth was not modeled for this pathway.

Table II. Summary Temperature Data (°F) for Retail Storage, Home Transport, and Home Storage[25]

	Retail	Transport	Home
≤45	882	580	850
46–48	31	161	27
49–51	9	103	14
52–54	3	47	2
55–57	1	19	1
58–60	2	14	1
61–63	0	2	1
64–66	0	1	0
67–69	1	1	0
Total	929	928	896

Temperature profiles for ground beef have been determined in a survey[25] of ground beef at retail, during transport to the home, and during home storage; a summary of the EcoSure data is provided in Table II.

Growth was modeled independently for each patty, following the approach outlined in the draft USA risk assessment[8,26] for temperatures greater than 7.2 °C. The difference in the present risk assessment is that here the lag was not modeled. This approach is conservative and will overestimate the risk because of higher growth estimates than if a lag phase is modeled. The storage duration at retail (D_{ret}) was modeled as an exponential distribution with a parameter whose reciprocal is uniform on 0.5–1.5 days, that is, $D_{\text{ret}} \sim \text{Exp}(1/\text{U}(0.5, 1.5))$.[8]

Then, the log generation time at retail ($\ln(GT_{\text{ret}})$) was modeled from the storage temperature at retail T_{ret} as per FSIS (Ref. 26, p. 80, equation 3.28), that is, the temperature T_{ret} was randomly drawn from the empirical retail temperature distribution[25] and

$$\ln(GT_{\text{ret}}) \sim N(7.03 - 6.31 \times \ln(\ln(T_{\text{ret}})), 0.16^2)$$

where N denotes the normal distribution, and hence the growth is obtained as $G_{\text{ret}} = 2^{(D_{\text{ret}}/GT_{\text{ret}})}$.

The $\log_{10}$ maximum population density ($\log_{10}(MPD_{\text{ret}})$) was also modeled similar to FSIS (Ref. 26, p. 80, equation 3.30), that is:

$$\log_{10}(MPD_{\text{ret}}) \sim \text{N}(\text{Tri}(\min = 5, \text{mode} = U(5, 10), \max = 10) - 0.014 \times T_{\text{ret}}, 0.15^2),$$

where the minimum and maximum of the triangular distribution (Tri) denote the minimum and maximum of the theoretical maximum density and the mode is uniform on the interval [5,10].

Finally, the number of *E. coli* O157 was calculated after retail storage as:

$$Y'_{\text{p,c}} = \max\left\{Y_{\text{p,c}}, \min\left(G_{\text{ret}} \times Y_{\text{p,c}}, 10^{\log_{10}(MPD_{\text{ret}})}\right)\right\}.$$

It should be noted that the EcoSure data[25] contain an outlier retail temperature of 19.4 °C and it is unlikely that meat would remain saleable if stored at this temperature for even a few days. Consequently, excluding this outlier was evaluated as a separate scenario. In addition, Smith *et al.*[7] truncated the retail duration distribution at 10 days while limiting the home storage duration using the growth of *Pseudonomas* spp, following the approach of Signorini and Tarabla.[11] Consequently, two further scenarios were evaluated. In the first the total storage duration was limited to 10 days, similar to how storage duration might be limited by a labeled shelf-life. The second approach is similar to that of Smith *et al.*[7]but here it is modeled for both retail and home storage. The product shelf-life and temperature relationship was calculated by fitting an exponential model to the upper 99% two-sided confidence bounds on the mean estimated shelf-lives at 4.3, 8.1, and 15.5 °C given in table 3 of Limbo *et al.*[27] The resulting model:

$$\text{Shelf-life} = 19.9\text{e}^{0.136T_{\text{ret}}}$$

predicts a shelf-life of 10.0 days at 5 °C and it was used to limit (truncate) the storage duration distribution.

2.2.6. Growth During Transport to the Home

This step was only modeled for patties through the home consumption pathway (Fig. 1) using storage temperature profiles and durations of ground beef for transport from retail to home.[25] For each patty a random entry from the joint temperature and duration distribution was selected. The growth of *E. coli* O157 during home transport was modeled as for retail storage, using the number of organisms after retail as the starting point, along with transport storage temperature (T_{trans}) and duration (D_{trans}).

2.2.7. Storage of Patties Prior to Cooking

This step was also only modeled for patties through the home consumption pathway (Fig. 1) using home storage temperature profiles for ground beef.[25] Growth during home storage was modeled the same way that growth during retail storage was

modeled, but using the number of organisms after transport as the starting point, along with home refrigerator temperatures (T_{home}) and duration (D_{home}).

For the two scenarios that limited retail and home storage duration, the two storage periods were not modeled independently, but the "effects" of retail storage were taken into account when modeling home storage. That is, prolonged storage at retail was unlikely to also result in long storage at home, and was either limited by a label shelf-life of 10 days or spoilage of the product. For example, if at a given retail temperature the product was sold after 25% of the maximum shelf-life had expired, then only 75% of the shelf-life remained at the home storage temperature, hence reducing the right truncation point of the home storage duration distribution. Transport duration effects were assumed to not impact the maximum storage at home.

2.2.8. Cooking and Inactivation of E. coli O157

It is generally accepted that in the United States a proportion of hamburgers are consumed undercooked. McIntosh *et al.*[28] estimated 3% of consumers ate hamburgers cooked to an internal temperature no warmer than 54.4 °C while a more recent survey estimated that 9.3% of consumers cooked their ground beef meal to, at most, 54.4 °C.[25] It is assumed that these temperatures are also representative for hamburgers prepared in the home and hence for each hamburger patty the cooking temperature was randomly sampled from the empirical distribution of ground beef cooking temperatures collected by EcoSure.[25]

In contrast, for the QSR pathway it was assumed that patties are cooked to a fixed internal temperature of at 68 °C, based on information from one major QSR.[29] In addition, a scenario based on cooking QSR hamburger patties to just 1 °C higher, that is, 69 °C, was also assessed.

For inactivation of *E. coli* O157 during cooking, the inactivation model generated by Juneja *et al.*[30] was used to calculate the $\log_{10}$ reduction (cfu/g) in the survivors. For temperatures below 47 °C this reduction was capped at 0—variability in the inactivation was not included.

2.2.9. Consumption of Patties

It was assumed that only one patty is consumed in a single meal.

3. HAZARD CHARACTERIZATION

Hazard characterization measures the nature and severity of adverse health effects of *E. coli* O157 illness such as hemorrhagic colitis (HC), HUS, and thrombotic thrombocytopenic purpura (TTP). A key element of hazard characterization is the dose-response curve, which describes the likelihood of illness occurring at various ingested quanta of the pathogen. In the case of a dose-response curve for Shiga Toxic *E. coli* (STEC), an expert consultancy convened by FAO/WHO[31] considered the available models and concluded that the dose response was probably the weakest part of an STEC risk assessment. However, a consensus of the same meeting was that "*the random coefficients model developed by Ross (1995)*[32] *(and used by Cassin* et al.*(1998))*[6] *may be reasonable, at least as a first approximation."* Accordingly, the Beta-binomial dose-response model used by Cassin *et al.*,[6] which gives the probability of illness from consuming dose D, was used, that is,

$$P_{\mathrm{il}}(D) = 1 - (1 - P_{\mathrm{ill}}(1))^{D},$$

where $P_{\mathrm{ill}}(1)$ is the probability of illness from one organism, which is distributed according to a Beta distribution with parameters α and β (Table II).

The number of organisms remaining in a patty (after cooking) was used in the dose-response model to yield a probability of illness (of any severity) for each patty. This in turn was used as the input probability for a Bernoulli trial to indicate whether the patty resulted in illness or not.

4. RISK CHARACTERIZATION

A summary of illness rates from contaminated lots under various simulation scenarios is provided in Table III. To calculate estimates for all Australian lots, the results from Table III need to be multiplied by 0.005, the estimated fraction of Australian lots of manufacturing meat that may be contaminated to some degree with *E. coli* O157.[16] This is a conservative approach as it ignores the removal of lots from the supply chain when *E. coli* O157 is detected as part of the mandatory N-60 testing program.

We were interested in assessing how contamination at the lot level would result in illnesses, particularly at how a potential loss of process control might manifest itself in a large proportion of contaminated cartons. Consequently, scatter plots of the lot contamination—the proportion of contaminated

Table III. Model Summaries (for Contaminated Lots Only)

Scenario	Cooking Temp.[a]	Patty Size (g)	α[b]	Retail Scenario[c]	Mean Number of Illnesses per Contaminated Lot[d]	Mean Illness Rate (per 100,000 Patties Consumed from Contaminated Lots)	97.5%-ile Illness Rate (per 100,000 Patties Consumed from Contaminated Lots)
1	Variable (home)	50	1	orig	13.5	3.55	10.8
2	Variable (home)	50	1	ex.outlier	13.4	3.53	10.8
3	Variable (home)	50	1	max10d	13.5	3.55	10.8
4	Variable (home)	50	1	temp.dep	13.8	3.62	11.0
5	Variable (home)	50	0.5	orig	12.3	3.22	9.7
6	Variable (home)	50	5	orig	14.9	3.92	11.8
7	Variable (home)	100	1	orig	10.8	5.65	16.8
8	Variable (home)	100	1	ex.outlier	10.8	5.67	16.8
9	Variable (home)	100	1	max10d	11.0	5.75	16.8
10	Variable (home)	100	1	temp.dep	10.7	5.63	16.8
11	Variable (home)	100	0.5	orig	9.8	5.15	15.2
12	Variable (home)	100	5	orig	11.8	6.22	18.4
13	Fixed at 68 °C (QSR)	50	1	NA	0.00131	0.00034	0.0
14	Fixed at 68 °C (QSR)	100	1	NA	0.00278	0.00146	0.0
15	Fixed at 69 °C (QSR)	50	1	NA	0.00119	0.00022	0.0
16	Fixed at 69 °C (QSR)	100	1	NA	0.00085	0.00063	0.0

[a]Home cooking scenarios used EcoSure (2008) cooking data to determine the inactivation of *E.coli* O157, while QSR cooking was assumed to result in a fixed internal endpoint patty temperature.
[b]Comminution parameter that determines the spread of contamination during grinding.
[c]Retail scenarios include: "orig"—include all Ecosure (2008) data; "ex.outlier"—as the "orig" scenario, but excluding a temperature outlier of 19.4 °C; "max10d"—the shelf-life at retail and in the home was limited to 10 days and the patty was assumed to be cooked and consumed by this time; "temp.dep"—the shelf-life was modeled to be temperature dependent so that combination of high temperature and long duration were not possible.
[d]Calculated as the sum of all illnesses from all lots divided by the number of lots (10,000 for scenarios 1–12 and 100,000 for scenarios 13–16).

cartons in a lot (panels) and the maximum number of organisms in any one carton in the lot (along the *X*-axis)—and number of illnesses per lot are shown in Fig. 2 for Scenario 1—each point in the graph represents one contaminated lot that was simulated. From this figure it can be seen that lots where the maximum number of *E. coli* O157 in any one carton are below 10^3 result in no, or very few, illnesses, especially when there are few contaminated cartons in a lot, that is, the proportion of contaminated cartons is small. However, when the proportion of contaminated cartons is large, there is also a greater chance of observing high numbers of *E. coli* O157 in a carton and hence the potential for a significant number of illnesses from a single lot is much higher. Nevertheless, it should be noted that the average number of illnesses over all contaminated lots was in the order of 10–14 (Table III), 97.5% of contaminated lots resulted in less than 41 and 32 illnesses per lot for 50 g and 100 g patties, respectively.

4.1. Home Consumption Pathway

More than 20% of hamburgers cooked at home in the United States reach an internal temperature no warmer than 60 °C.[25] In this supply chain (Fig. 1; Table III, Scenarios 1 & 7) contaminated lots (some of which would be removed from the supply chain through the testing program following usual practice) yield an average of 13.5 and 10.8 illnesses when consumed in 50 and 100 g burgers, respectively. The higher number of illness for 50 g burgers is due to the doubling of the number of burgers that are obtained, and consumed, from each lot when the patty size is halved. Despite the doubling in the number of burgers, the number of illnesses from a contaminated lot only increases by about 30% and hence the rate of illness is lower when considering the smaller patty size, namely, 1.78 and 2.83 illnesses per 10^7 burgers, when 50 and 100 g patties are consumed, respectively.

4.2. QSRs Consumption Pathway

In Table III are presented illness rates resulting from consumption of Australian manufacturing meat from contaminated lots (some of which would be removed from the supply chain through the testing program following usual practice) used for patties consumed in QSRs in the United States. Cooking

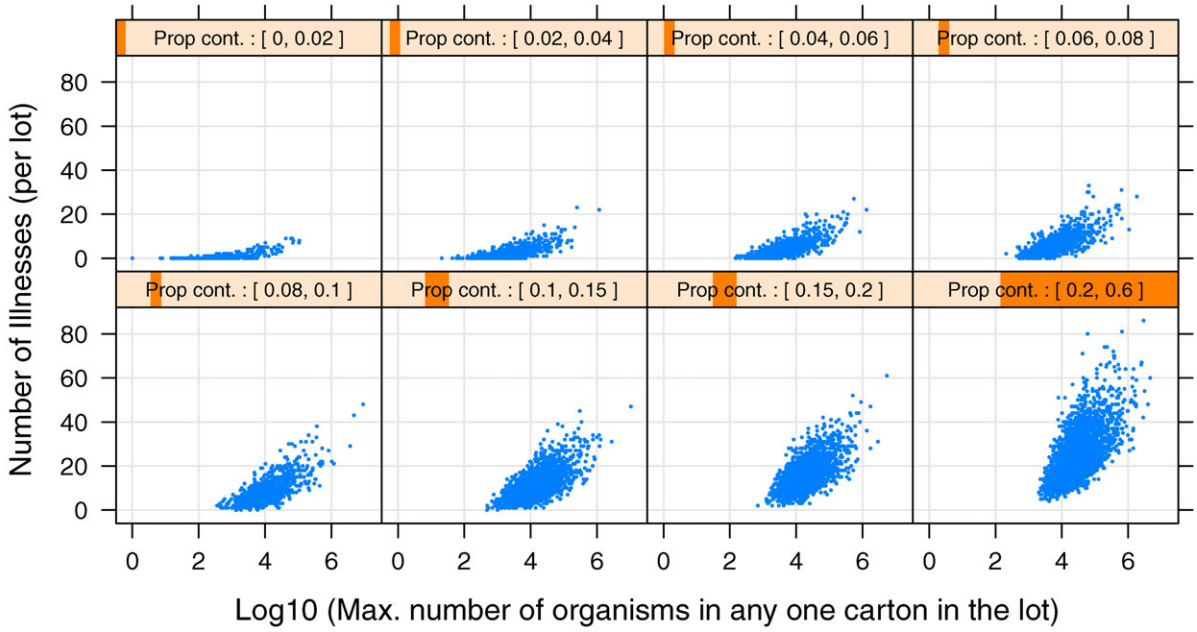

Fig. 2. Scatter plots of the number of illnesses from contaminated lots (Scenario 1) given the maximum number of *E. coli* O157 in any one carton (along the *X*-axis) for various proportion of contaminated cartons per lot (panels): each point represents a contaminated lot that was simulated.

patties to 68 °C (Scenarios 13 & 14) resulted in 1.72 and 7.30 illness per 10^{11} hamburgers from all Australian lots, when 50 and 100 g patties are consumed, respectively.

4.3. Expected Number of Illness from Consumption of "Purely" Australian Beef Burgers in the Home and QSRs

Typically, Australia exports 100,000–150,000 t/annum of manufacturing meat to the United States for grinding into patties, the volume varying according to market demand and currency exchange rates. In 2012, Australia exported 155,000 t of manufacturing meat for grinding in the United States, which industry sources suggest yields about 5% of the approximately 50 billion hamburgers consumed in the United States annually.[33] Of these, around 10%, or 15,500 t, were retailed for home consumption and 139,500 t were ground for consumption in QSRs.

In Table IV is presented an estimate of illnesses, based on 2012 export figures, likely to occur annually in the home and in QSRs if hamburgers were made only from Australian beef. Of the 49.59 illnesses predicted to occur each year from patties produced from only Australian manufacturing meat, all occur from hamburgers cooked and consumed in the home.

By contrast, the previous U.S. risk assessment estimated that consumption in 2001 of 18.2 billion servings of ground beef in the United States caused 19,000 cases (median estimate) of symptomatic *E. coli* O157:H7 illness.[8]

4.4. Sensitivity Analysis

The results for contaminated lots (only) for the various scenarios are shown in Table III for 50 and 100 g patties. The exclusion of the 19.4 °C retail storage outlier (Scenarios 2 & 8) had little impact on the mean number of illnesses per lot and the illness rate per 100,000 patties consumed compared to the baseline scenarios (1 & 7). Similarly, truncating the retail and home storage durations at 10 days (Scenarios 3 & 9) had little impact on the illnesses, nor did the spoilage-based maximum storage (Scenarios 4 & 10).

In contrast, reducing the comminution parameter α_i from 1 to 0.5, which results in carton contamination being spread over fewer patties, yielded about a 10% reduction in the illness rate (Scenarios 5 & 11). On the other hand, increasing α_i from 1 to 5, and hence spreading any carton contamination over more patties, resulted in about a 10% increase in the rate of illnesses.

If cooking in QSRs is undertaken to 69 °C instead of 68 °C, then for 50 and 100 g patties the rate

Table IV. Estimated Illnesses in the United States (Home and QSRs) from Consumption of Hamburger Patties Ground from 155,000 t of Australian Manufacturing Meat Exported to the United States in 2012

Location	Patty Size (g)	Proportion[a]	Weight (kg)[b]	Number of Patties	Illnesses/Number of Patties[c]	Illnesses Predicted
Home	50	0.5	7,750,000	155,000,000	1.78/10,000,000	27.59
	100	0.5	7,750,000	77,500,000	2.83/10,000,000	21.93
QSRs	50	0.6	83,700,000	1,674,000,000	1.72/100,000,000,000	0.03
	100	0.4	55,800,000	558,000,000	7.30/100,000,000,000	0.04
		Totals	155,000,000	2,464,500,000		49.59

[a]For each location, the estimated proportion of meat that is consumed as 50 g and 100 g burgers.
[b]Industry information indicates that of 155,000 t of Australian manufacturing beef exported to the United States in 2012, only 10% entered the retail stream.
[c]Illnesses were calculated from Table III (Scenarios 1, 7, 13, and 14) by multiplying the "mean illness rate (per 100,000 patties consumed from contaminated lots)" by the fraction of contaminated lots (0.005).

of illness reduces to 1.1 and 3.1 per 10^{11} patties from all Australian lots. However, irrespective of whether an internal temperature of 68 °C or 69 °C is reached, the rates of illness are considerably lower—by a factor of about 10,000—through QSRs than the home consumption pathway with variable cooking temperatures. While thorough cooking by the consumer can clearly not be relied on, these results do however given an indication of its effectiveness.

5. DISCUSSION

Estimates in the present assessment are markedly lower than any of the estimates quoted in other risk assessments, for example, 1/1,000,000 or 19,000 annual illnesses from ground beef consumption in the United States.[8]

This risk assessment takes advantage of having relatively good data for contamination reasonably close to the end of the supply chain. We have used estimates of between-lot prevalence, within-lot carton prevalence, and concentration of *E. coli* O157 based on routine sampling, together with testing of multiple lots of product in a regulated environment, analysis of known contaminated lots, and statistical inference.[16]

We have made our assessment based on no product being removed from the supply chain following mandatory sampling and testing program results. This is contrary to the regulated practice in which all lots are sampled and tested, and no product in which *E. coli* O157 is detected is allowed to enter commerce. The approach of using this "base case" allows for future investigation of the impact that various sampling strategies may have on public health outcomes.

We have modeled cooking and consumption through two pathways: the home consumption pathway, with variable burger temperature that takes into account consumer preference, and QSR consumption pathway, which was defined as burgers being cooked to a fixed temperature. Our QSR definition differs from that of Bogard *et al.*,[34] who found that 21% of quick service and fast food restaurants cooked burgers to a customer's preference—consequently, burgers consumed through these restaurants are likely to pose a similar risk to the home consumption pathway modeled here.

In general, the assessment approach used for the draft U.S. risk assessment, particularly in terms of retail storage, transport home, home storage, and dose-response, was used in the present assessment.[8] However, we also chose to simulate whole lots and estimated illnesses that may occur from each of these manufacturing lots if no blending with product of different origin and fat content were to occur.

Where possible, we have made conservative assumptions on what happens through the supply chain, including the following:

- The fraction of contaminated Australian lots does not take into account that all Australian lots are tested according to N-60 and that lots in which *E. coli* O157 is detected are removed from commerce.
- Previous work[16] established estimates of *E. coli* O157 contamination in manufacturing beef that had been stored frozen. Consequently, it was assumed that further frozen storage, before or after grinding and patty forming, did not result in additional reductions in *E. coli* O157 concentration.

- During chilled retail storage, and beyond, we did not model a lag period as was done in the draft U.S. risk assessment.[8]
- We assumed that there was no loss of product throughout the supply chain. For example, the retail temperature scenarios that limit the shelf-life (4 & 10, Table III) do not allow for actual product spoilage and subsequent removal of product.

However, we also believe that some assumptions may not be conservative, including:

- No cross-contamination during grinding: This assumption results in less spread of contamination to otherwise uncontaminated cartons of meat. However, the impact of this assumption is less than the impact of changing the comminution parameter α from 1 to 5 as the spread of organisms between cartons is less than that within a carton due to grinding.
- No mixing with other beef sources: This assumption also reduced the potential spread of contamination, though other sources of meat may contain *E. coli* O157, for which we did not have comparable data.
- Lot size is fixed: Because we assumed no cross-contamination between cartons, this assumption does not have an important impact on the results. While the number of illness per lot would be reduced proportionally to the size of the lot, the illness rate per 100,000 burgers consumed would remain unchanged.
- Variability in the inactivation model due to cooking[30] was not included in the model and we assumed that the inactivation was not different between frozen or chilled patties, but determined only by the endpoint cooking temperature of the patty.

We have estimated that Australian manufacturing beef consumed as burgers results in a very low number of *E. coli* O157 illnesses. Considerable progress has been made in relation to controlling *E. coli* O157 in beef since the draft U.S. risk assessment was published.[8] Scallan *et al*.[1] provide a more up-to-date estimate of foodborne illness, but do not attribute illnesses to different foods. Whichever figures are used, Australian beef would still be responsible for a small proportion of cases.

Support for the contention that Australian manufacturing meat is less likely to be contaminated with pathogenic bacteria is gained from a survey[35] of beef destined for grinding in the United States from Australia, New Zealand, Uruguay, and the United States. Analysis of indicator organisms and pathogens, including *Salmonella* and non-O157 STEC, led the researchers to conclude that Australian and New Zealand beef trim had lower levels of contamination than did U.S. and Uruguayan trim, and that differences were more likely to be associated with the processing environment and processes in use, rather than seasonal differences.

The results of this assessment are supported by the epidemiology of EHEC infections in Australia and an increased understanding of the virulence of Australian *E. coli* O157 strains. There is a tendency in Australia to cook ground beef products thoroughly and this is reflected in the complete lack of association between illness and ground beef.[36] Over the period 2000–2010 the rate of notified STEC illness was 0.4 cases/100,000 per annum (0.12 for *E. coli* O157). Of the 11 outbreaks recorded over the same period, none was shown to be associated with beef. The incidence of *E. coli* illness in Australia may also be affected by the virulence of *E. coli* O157 strains. *E. coli* strains isolated in Australia appear to be genetically divergent from those isolated in the United States, and Australian strains are less likely to carry the *stx2* gene, which is associated with more severe disease.[37] Consequently, the current dose-response relationship will likely overestimate the probability of illness for a given dose of *E. coli* O157 strains originating from Australia.

6. CONCLUSIONS

Based on a risk assessment model with a series of conservative assumptions (including absence of the compulsory testing program), consumption of "Australian" patties is estimated to result in 50 illnesses while supplying the equivalent amount of meat for around 5% of the approximately 50 billion patties consumed in the United States annually.

Given that Australian beef is mixed with domestic beef and with beef imported from other countries, it is unlikely that Americans consume an "Australian" hamburger *per se*. Nonetheless, the present assessment is useful because it provides an estimate of illness from one specific source of manufacturing in international trade destined for grinding in the United States.

ACKNOWLEDGMENTS

Meat & Livestock Australia Market Information Service for data on exports from Australia, and

consumption patterns within the United States. The majority of this work was undertaken while the first author was employed by the South Australian Research and Development Institute (SARDI). In addition, SARDI's Oceanography group is thanked for providing computing resources for the simulations. We also thank the reviewers for their comments.

REFERENCES

1. Scallan E, Hoekstra RM, Angulo FJ, Tauxe RV, Widdowson M-A, Roy SL, Jones JL, Griffin PM. Foodborne illness aquired in the United States: Major pathogens. Emerging Infectious Diseases, 2011; 17(1):7–15.
2. Wells JG, Davis BR, Wachsmuth IK, Riley LW, Remis RS, Sokolow R, Morris GK. Laboratory investigation of hemorrhagic colitis outbreaks associated with a rare *Escherichia coli* serotype. Journal of Clinical Microbiology, 1983; 18(3):512–520.
3. Bell BP, Goldoft M, Griffin PM, Davis MA, Gordon DC, Tarr PI, Bartleson CA, Lewis JH, Barrett TJ, Wells JG, Baron R, Kobayashi J. A multistate outbreak of *Escherichia coli* O157:H7-associated bloody diarrhea and hemolytic uremic syndrome from hamburgers: The Washington experience. Journal of the American Medical Association, 1994; 272(17):1349–1353.
4. Rangel JM, Sparling PH, Crowe C, Griffin PM, Swerdlow DL. Epidemiology of *Escherichia coli* O157:H7 outbreaks, United States, 1982–2002. Emerging Infectious Diseases, 2005; 11(4):603–609.
5. Anonymous. Shiga toxin producing *Escherichia coli* in certain raw beef products. Federal Register, 2011; 76(182):58157–58165.
6. Cassin MH, Lammerding AM, Todd ECD, Ross W, McColl RS. Quantitative risk assessment for *Escherichia coli* O157:H7 in ground beef hamburgers. International Journal of Food Microbiology, 1998; 41:21–44.
7. Smith BA, Fazil A, Lammerding AM. A risk assessment model for *Escherichia coli* O157:H7 in ground beef and beef cuts in Canada: Evaluating the effects of interventions. Food Control [Internet] 2013; 29(2):364–381.
8. Ebel E, Schlosser W, Kause J, Orloski K, Roberts T, Narrod C, Malcolm S, Coleman M, Powell M. Draft risk assessment of the public health impact of *Escherichia coli* O157:H7 in ground beef. Journal of Food Protection, 2004; 67(9):1991–1999.
9. Duffy G, Butler F, Cummins E, O'Brien S, Nally P, Carney E, Henchion M, Mahon D, Cowan C. *E. coli* O157:H7 in Beefburgers Produced in the Republic of Ireland: A Quantitative Microbial Risk Assessment. Dublin: TEAGASC, 2006.
10. Delignette-Muller ML, Cornu M. The AFSSA STEC Study Group. Quantitative risk assessment for *Escherichia coli* O157:H7 in frozen ground beef patties consumed by young children in French households. International Journal of Food Microbiology, 2008; 128(1):158–164.
11. Signorini M, Tarabla H. Quantitative risk assessment for verocytotoxigenic *Escherichia coli* in ground beef hamburgers in Argentina. International Journal of Food Microbiology, 2009; 132(2–3):153–161.
12. Kosmider RD, Nally P, Simons RRL, Brouwer A, Cheung S, Snary EL, Wooldridge M. Attribution of human VTEC O157 infection from meat products: A quantitative risk assessment approach. Risk Analysis, 2010; 30(5):753–765.
13. Phillips D, Sumner J, Alexander J, Dutton K. Microbiological quality of Australian beef. Journal of Food Protection, 2001; 64:692–696.
14. Phillips D, Jordan D, Morris S, Jenson I, Sumner J. A national survey of the microbiological quality of beef carcasses and frozen boneless beef in Australia. Journal of Food Protection, 2006; 69(5):1113–1117.
15. Phillips D, Bridger K, Jenson I, Sumner J. An Australian national survey of the microbiological quality of frozen boneless beef and beef primal cuts. Journal of Food Protection. Journal of Food Protection, 2012; 75:1862–1866.
16. Kiermeier A, Mellor G, Barlow R, Jenson I. Assumptions of acceptance sampling and the implications for lot contamination: *Escherichia coli* O157 in lots of Australian manufacturing beef. Journal of Food Protection, 2011; 74(4):539–544.
17. Anonymous. AQIS Meat Notice Number 2008/05: *Escherichia coli* O157:H7 Testing of Raw Ground Beef Components Destined for Export to the US and US Territories. 2008. Available at: http://www.daff.gov.au/__data/assets/pdf_file/0020/753104/2008--05.pdf, Accessed April 13, 2014.
18. R Core Team. R: A Language and Environment for Statistical Computing [Internet]. Vienna, Austria: R Foundation for Statistical Computing, 2012. Available at: http://www.R-project.org.
19. Pouillot R, Delignette-Muller ML. Evaluating variability and uncertainty separately in microbial quantitative risk assessment using two R packages. International Journal of Food Microbiology, 2010; 142(3):330–340.
20. Delignette-Muller ML, Pouillot R, Denis J-B, Dutang C. fitdistrplus: Help to Fit of a Parametric Distribution to Non-Censored or Censored Data, 2010. Available at: http://riskassessment.r-forge.r-project.org/, Accessed June 18, 2014.
21. Dykes GA. The effect of freezing on the survival of *Escherichia coli* O157:H7 on beef trimmings. Food Research International [Internet], 2000; 33(5):387–392.
22. Nauta MJ. Microbiological risk assessment models for partitioning and mixing during food handling. International Journal of Food Microbiology, 2005; 100:311–322.
23. Flores RA, Tamplin ML. Distribution patterns of *Escherichia coli* O157:H7 in ground beef produced by a laboratory-scale grinder. Journal of Food Protection, 2002; 65(12):1894–1902.
24. Flores RA, Stewart TE. Empirical distribution models for *Escherichia coli* O157:H7 in ground beef produced by a mid-size commercial grinder. Journal of Food Science, 2004; 69(5):M121–M126.
25. EcoSure. 2007 U.S. Cold Temperature Evaluation: Design and Summary Pages, 2008. Available at: http://foodrisk.org/exclusives/EcoSure/, Accessed April 14, 2014.
26. FSIS. Risk Assessment of the Public Health Impact of *Escherichia coli* O157:H7 in Ground Beef. Food Safety and Inspection Service, 2001. Available at: http://www.fsis.usda.gov/OPPDE/rdad/FRPubs/00--023N/00--023NReport.pdf, Accessed April 14, 2014.
27. Limbo S, Torri L, Sinelli N, Franzetti L, Casiraghi E. Evaluation and predictive modeling of shelf life of minced beef stored in high-oxygen modified atmosphere packaging at different temperatures. Meat Science, 2010; 84(1):129–136.
28. McIntosh WA, Christensen LB, Acuff GR. Perceptions of risks of eating undercooked meat and willingness to change cooking practices. Appetite, 1994; 22:83–96.
29. Welse E. How McDonald's makes sure its burgers are safe [Internet]. USA Today, 2009. Available at: http://www.usatoday.com/money/industries/food/2009--12--29-mdonalds-burgers-food-safety˙N.htm, Accessed April 13, 2014.
30. Juneja VK, Snyder OP, Williams AC, Marmer BS. Thermal destruction of *Escherichia coli* O157:H7 in hamburger. Journal of Food Protection, 1997; 60(10):1163–1166.
31. FAO/WHO. Enterohaemorrhagic *Escherichia coli* in Raw Beef and Beef Products: Approaches for the Provision of Scientific Advice Meeting Report. Food and Agriculture Orga-

nization of the United Nations/World Health Organization, 2011. Available at: ftp://ftp.fao.org/ag/agn/jemra/MRA_18.pdf, Accessed June 18, 2014.
32. Ross W. Accounting for Uncertainty in the Synthesis of Dose-Response Data. Ottawa, Canada: Environmental Health Directorate, Health Canada, 1995.
33. Thomson JR. Hamburger trivia: Everything you ever needed to know. Huffington Post, 2013. Available at: http://www.huffingtonpost.com/2013/07/28/burger-facts-trivia_n_3654636.html, Accessed April 13, 2014.
34. Bogard AK, Fuller CC, Radke V, Selman CA, Smith KE. Ground beef handling and cooking practices in restaurants in eight states. Journal of Food Protection, 2013; 76(12):2132–2140.
35. Bosilevac JM, Guerini MN, Brichta-Harhay DM, Arthur TM, Koohmaraie M. Microbiological characterization of imported and domestic boneless beef trim used for ground beef. Journal of Food Protection, 2007; 70:440–449.
36. Vally H, Hall G, Dyda A, Raupach J, Knope K, Combs B, Desmarchelier P. Epidemiology of shiga toxin producing *Escherichia coli* in Australia, 2000–2010. BMC Public Health, 2012; 12(63):1–12.
37. Mellor GE, Besser TE, Davis MA, Beavis B, Jung W, Smith HV, Jennison AV, Doyle CJ, Chandry PS, Gobius KS, Fegan N. Multilocus genotype analysis of *Escherichia coli* O157 isolates from Australia and the United States provides evidence of geographic divergence. Applied and Environmental Microbiology, 2012; 79(16):5050–5058.