

How Many, Not How Often: Why Reducing Contamination Level Beats Reducing Flock Prevalence in *Campylobacter* Control

Dr. T Rajendran

Assistant Professor Tamil Nadu Agricultural University, Department of Agricultural Economics

VOC Agricultural College and Research Institute

Killikulam -628 252

rajendran.t@tnau.ac.in

Abstract

Background. *Campylobacter* is the commonest bacterial cause of foodborne gastroenteritis in many countries and poultry is its principal vehicle. Control programmes are frequently organised around flock prevalence — the proportion of flocks colonised at slaughter — because it is measurable, auditable and attributable to a farm. Whether prevalence is the right target depends on the shape of the dose–response relationship, which is steeply non-linear at low dose.

Objective. To compare, within a single farm-to-fork risk model, the illness averted by reducing flock prevalence, by reducing carcass concentration at processing, and by improving consumer handling, and to determine which exposure pathway dominates.

Methods. A quantitative microbial risk assessment was constructed over 800,000 simulated servings. Sixty per cent of flocks were colonised; positive carcasses carried a log-normal load of mean 4.2 log₁₀ CFU with a log standard deviation of 0.9. Two exposure pathways were modelled: consumption of an inadequately cooked serving, occurring in 1.0 % of servings with a 3-log residual reduction, and transfer of organisms to a ready-to-eat food during preparation, occurring in 10 % of preparations. Illness probability followed a Beta-Poisson dose–response with $\alpha = 0.145$ and $\beta = 7.59$ and an illness-given-infection probability of 0.33. The model was calibrated to a plausible true incidence rather than to a reported case count.

Results. Baseline modelled incidence was 2,298 cases per 100,000 per year, of which 62.8 % arose from cross-contamination to ready-to-eat food and 36.4 % from undercooked servings. Risk was highly concentrated: the most contaminated tenth of carcasses carried 76.3 % of total risk and the worst 1 % carried 23.5 %. Reducing flock prevalence from 60 % to 20 % — a very demanding biosecurity achievement — reduced illness by 67.5 %. A 1-log reduction in carcass concentration reduced it by 83.5 % and a 2-log reduction by 97.9 %, while freezing modelled as 1.5 logs achieved 94.0 %. Halving cross-contamination reduced illness by 26.0 % and reducing undercooking from 1.0 % to 0.3 % of servings by 19.0 %.

Conclusions. Because the dose–response curve is steepest at low dose, moving every carcass down the concentration distribution outperforms removing a fraction of carcasses from it. Control programmes built on flock prevalence are targeting the measurable quantity rather than the influential one, and a process step delivering one or two logs of reduction dominates any plausible farm-level prevalence gain.

Keywords: Campylobacter; quantitative microbial risk assessment; poultry; food safety; cross-contamination; dose–response.

1. Introduction

Campylobacteriosis is the most frequently reported bacterial foodborne infection in many high- and middle-income countries, and broiler meat is its dominant vehicle. Infection is usually self-limiting, but it is not trivial: a minority of cases develop reactive arthritis, and a small proportion develop Guillain–Barré syndrome, which makes the aggregate burden substantial even at a low case fatality.

Control policy across the poultry chain has largely organised itself around flock prevalence. The proportion of flocks testing positive at slaughter is measurable, can be audited, is attributable to an identifiable producer, and has the political advantage of assigning responsibility at the farm. Targets are set in those terms, and programmes are judged by whether the proportion of positive flocks falls.

There is a reason to doubt that this is the right quantity. Illness depends on the dose a person ingests, and the relationship between dose and risk is strongly concave: the probability of infection rises steeply from the first few organisms and then flattens. A carcass carrying ten thousand organisms and one carrying a hundred are both positive and contribute very differently to disease. A programme that counts positives is treating those two as the same.

This study builds a farm-to-fork model to compare the two framings on a common outcome. Three questions are addressed: which exposure pathway from a contaminated carcass actually produces the illness; how concentrated risk is across the carcass population; and how a given effort spent on flock prevalence compares with the same objective pursued through concentration reduction at processing or through consumer handling.

2. Methods

The modelled farm-to-fork pathway

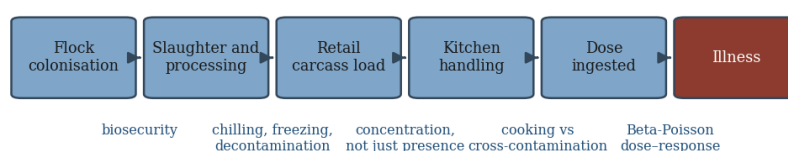


Figure 1. The modelled pathway from flock colonisation to human illness.

2.1 Exposure model

Eight hundred thousand servings were simulated. Each carcass was colonised with probability equal to the flock prevalence, and positive carcasses carried a load drawn from a log-normal distribution on the $\log_{10}$ scale. A serving was taken as 120 g from a 1,300 g carcass, so the organisms associated with a serving are the carcass load scaled by that fraction.

Two pathways lead from that carcass to an ingested dose.

Undercooked serving. In 1.0 % of servings cooking is inadequate and achieves only a 3-log reduction instead of the 6 logs assumed for adequate cooking. The dose is the serving load reduced accordingly.

Cross-contamination. In 10 % of preparations organisms are transferred from the raw carcass, via hands, surfaces or utensils, to a food eaten without a subsequent kill step. A fixed fraction of the carcass load, 10^{-5} , reaches that food. There is no cooking step on this pathway, which is why it matters despite the small transfer fraction.

The two doses act independently and the probability of illness from a serving is one minus the probability of escaping both.

2.2 Dose–response

Infection probability follows the Beta-Poisson relation conventionally applied to *Campylobacter jejuni*,

$$P_{\text{inf}}(d) = 1 - (1 + d/\beta)^{-\alpha} \quad (1)$$

with $\alpha = 0.145$ and $\beta = 7.59$, and illness follows infection with probability 0.33.

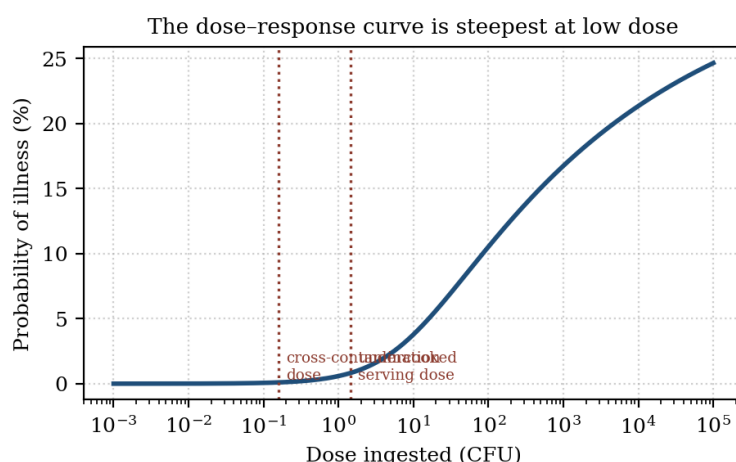


Figure 2. The Beta-Poisson dose–response curve with the two modelled doses marked. Horizontal axis is logarithmic.

Figure 2 contains the mechanism behind every result that follows. The curve rises steeply through the first few organisms and flattens thereafter, so a reduction of one log at a high dose changes risk far less than the same reduction at a low one. Both modelled doses sit on the steep portion, which is where concentration reductions do the most work.

2.3 Parameters and calibration

Table 1. Model parameters

Parameter	Value	Role
Flock prevalence	60 %	Proportion of carcasses positive; varied 10–80 %
Carcass load, positive carcasses	Log-normal, mean $4.2 \log_{10}$ CFU, log SD 0.9	Concentration; reduced by 0–3 logs in scenarios
Serving size / carcass weight	120 g / 1,300 g	Fraction of load in a serving
Inadequate cooking frequency	1.0 % of servings	Varied to 0.3 % in one scenario
Log reduction, inadequate cooking	3 logs	Against 6 logs for adequate cooking
Cross-contamination event frequency	10 % of preparations	Fixed
Transfer fraction to ready-to-eat food	10^{-5} of carcass load	Halved in the hygiene scenario

Parameter	Value	Role
Dose–response	$\alpha = 0.145, \beta = 7.59$	Beta-Poisson for <i>C. jejuni</i>
Illness given infection	0.33	Conventional value
Poultry servings per person per year	52	One per week
Servings simulated	800,000	Monte Carlo error negligible

Note. The transfer fraction and the undercooking parameters are not independently measurable at population scale and were set so that baseline modelled incidence falls in a range plausible for true rather than reported campylobacteriosis. This is a calibration, not an estimate, and it is stated because every absolute figure in this paper inherits it. The comparisons between control measures depend on the structure of the dose–response relationship and not on the calibration, as Section 4.3 sets out.

3. Results

3.1 Baseline and which pathway matters

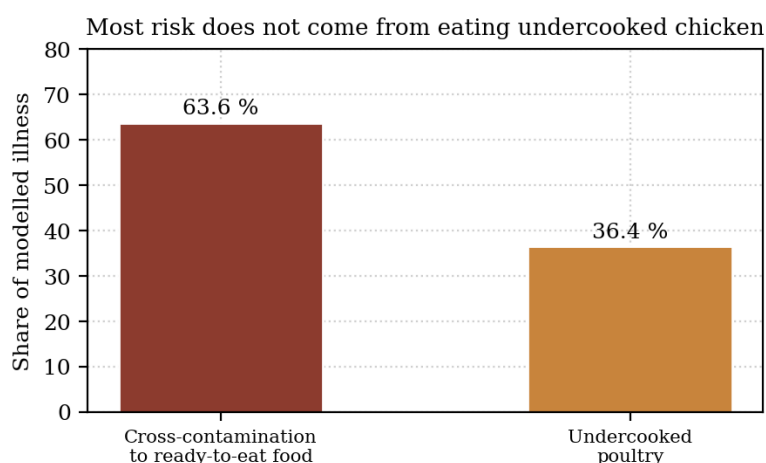


Figure 3. Share of modelled illness arising from each exposure pathway.

Baseline modelled incidence was 2,298 cases per 100,000 per year. Of that, 62.8 % arose from cross-contamination to ready-to-eat foods and 36.4 % from undercooked servings.

The result is driven by the absence of a kill step rather than by the size of the transfer. Only a hundred-thousandth of the carcass load reaches the ready-to-eat food, and it reaches it intact, whereas the far larger quantity on the serving itself passes through cooking that removes three to six logs. A small dose delivered reliably outweighs a large dose that is usually destroyed.

This matters for how consumer advice is framed. Messaging concentrated on cooking poultry thoroughly addresses the smaller of the two pathways in this model, while the larger one is governed by what happens to the raw carcass in the kitchen before it is cooked at all.

3.2 Risk is concentrated in a minority of carcasses

Table 2. Share of total risk carried by the most contaminated carcasses

Most contaminated carcasses	Share of total risk
Top 1 %	23.5 %
Top 5 %	58.6 %
Top 10 %	76.3 %
Top 25 %	94.3 %

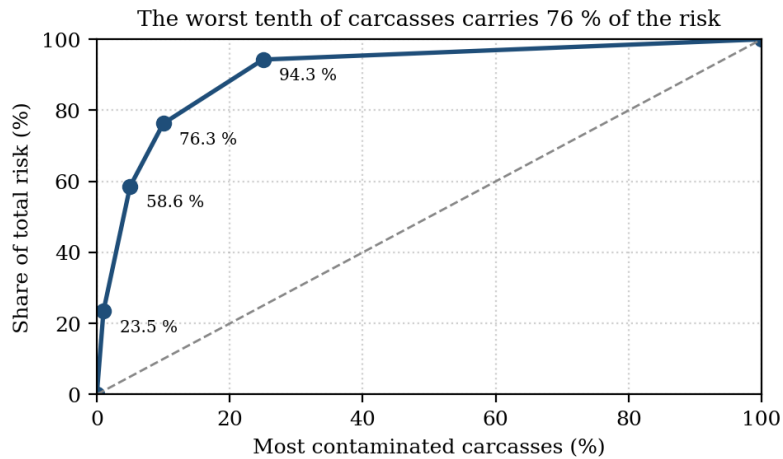


Figure 4. Cumulative share of risk against carcasses ranked by contamination.

The most contaminated tenth of carcasses carries 76.3 % of the modelled risk and the worst quarter carries 94.3 %. The remaining three quarters of positive and negative carcasses together contribute under 6 %.

This is the quantitative reason that counting positives misdescribes the problem. A carcass at the median of the positive distribution and one at the 99th percentile both register as one positive in a prevalence statistic and differ in their contribution to disease by more than an order of magnitude. A programme that reduced prevalence by removing randomly selected positive flocks would remove risk roughly in proportion to the flocks removed; one that acted on the upper tail of the concentration distribution would remove far more.

3.3 Comparing control measures

Table 3. Effect of each control measure

Control measure	Acts at	Cases per 100,000 per year	Reduction
Baseline	—	2,298	—
Flock prevalence 60 → 40 %	Farm	1,481	35.6 %
Flock prevalence 60 → 20 %	Farm	746	67.5 %
Undercooking 1.0 → 0.3 % of servings	Consumer	1,861	19.0 %
Cross-contamination halved	Consumer	1,700	26.0 %
Processing: 1 log reduction	Processing	379	83.5 %
Freezing: 1.5 log reduction	Processing	139	94.0 %
Processing: 2 log reduction	Processing	48	97.9 %
Processing 2 log + cross-contamination halved	Both	36	98.4 %

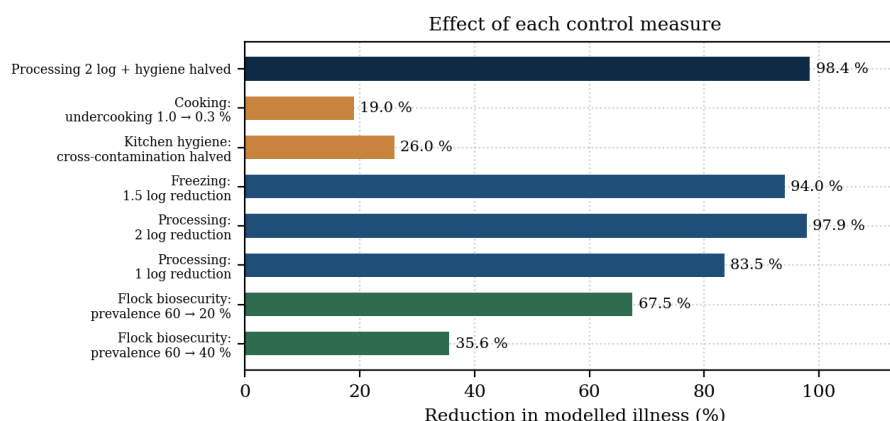


Figure 5. Reduction in modelled illness by control measure.

The ordering is unambiguous. A single log of concentration reduction at processing — achievable by chilling regimes, by scheduled slaughter of positive flocks at the end of the day, by physical decontamination or by freezing — reduced illness by 83.5 %. Reducing flock prevalence from 60 % to 20 %, which would represent a transformation of farm biosecurity across an entire industry, reduced it by 67.5 %.

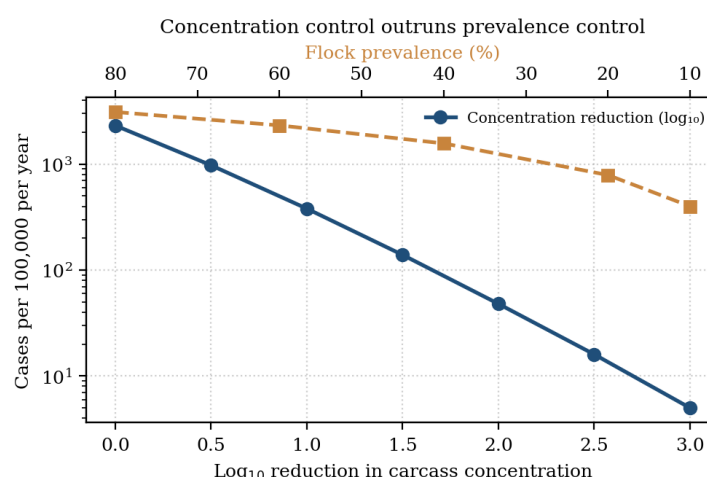


Figure 6. Cases against concentration reduction and against flock prevalence, with a logarithmic vertical axis.

Figure 6 places the two levers on the same axis and the difference in slope is the finding. Illness falls roughly log-linearly with concentration reduction — each log removing approximately six sevenths of what remains — while falling only in proportion to prevalence. Prevalence reduction is a linear lever pulled against a non-linear response; concentration reduction is matched to it.

The reason is Figure 2. Reducing prevalence removes some servings from the exposed group entirely while leaving the dose unchanged for the rest. Reducing concentration moves every serving down a curve that is steepest exactly where these doses sit. The second acts on the whole distribution and on the part of the dose–response where the derivative is largest.

3.4 Consumer measures

The two consumer scenarios are informative in opposite directions. Halving cross-contamination — an ambitious behavioural target — reduced illness by 26.0 %, which is a meaningful contribution and considerably more than improving cooking. Reducing inadequate cooking from 1.0 % to 0.3 % of servings, a threefold improvement in a behaviour that public messaging has emphasised for decades, reduced illness by 19.0 %.

Neither approaches what a processing intervention delivers, and this deserves care in interpretation rather than a conclusion that consumer behaviour does not matter. Consumer measures act on a dose that is already large relative to the steep part of the curve, and they act on only one pathway each. They are also, unlike a processing step, not verifiable, not uniform across households and not sustained without continuing investment.

The combined final row shows the practical position: with a 2-log processing reduction in place, halving cross-contamination adds only a further 0.5 percentage points, because the dose has already been moved onto the flat lower portion of the dose–response curve. Consumer measures are most valuable where the upstream chain has not been controlled, which is the reverse of how responsibility is usually assigned in food safety communication.

4. Discussion

4.1 Principal findings

Four results follow. Cross-contamination to ready-to-eat foods accounted for 62.8 % of modelled illness against 36.4 % from undercooked servings, because the smaller dose reaches the consumer without a kill step. Risk is highly concentrated, with the most contaminated tenth of carcasses carrying 76.3 %. A 1-log reduction in carcass concentration reduced illness by 83.5 % while reducing flock prevalence from 60 % to 20 % reduced it by 67.5 %. And with a 2-log processing reduction in place, further consumer improvement added almost nothing.

4.2 Implications for control programmes

Table 4. Implications

Finding	Implication	Recommendation
1 log at processing beat 40 points of prevalence	Concentration is the influential quantity	Set targets on carcass counts, not only on flock positivity
Top 10 % of carcasses carry 76 % of risk	Positives are not equivalent to one another	Monitor the upper tail of the concentration distribution
Cross-contamination dominated undercooking	The kill step is bypassed, not overwhelmed	Direct consumer messaging at raw-meat handling, not cooking alone
Freezing achieved 94 %	An existing, cheap, verifiable step is highly effective	Consider freezing or equivalent for high-load consignments
Consumer measures gave 19–26 %	Real but secondary, and unverifiable	Do not substitute consumer education for process control
Consumer gain collapses once processing is controlled	The measures are not additive	Sequence process control first, then assess residual need
Prevalence is linear, response is not	The measurable target is the wrong target	Report both prevalence and concentration in surveillance

The first and last rows together are the paper’s message and they concern what is counted. Flock prevalence is attractive because it is binary, cheap to determine and attributable, and those are properties of the measurement rather than of the risk. Enumeration of organisms on carcasses is more expensive and less politically convenient, and it is the quantity that determines how much disease occurs.

The fourth row is the most immediately actionable. Freezing is an established, inexpensive process with a well-characterised effect on *Campylobacter* viability, and applying it selectively to consignments from high-load flocks would act precisely on the upper tail identified in Section 3.2. It carries commercial consequences for a product sold as fresh, which is a real obstacle and a different kind of obstacle from a scientific one.

4.3 Limitations

The model is calibrated rather than fitted. The cross-contamination transfer fraction and the frequency and severity of undercooking are not measurable at population scale, and they were set so that baseline incidence falls in a plausible range for true infection. Different plausible combinations of those parameters would shift the balance between the two pathways, and the finding that cross-contamination dominates is therefore weaker than the finding that concentration reduction outperforms prevalence reduction, which follows from the concavity of the dose–response function rather than from the calibration.

The dose–response parameters are taken from human feeding studies in healthy adult volunteers and are applied to a whole population including children, older adults and the immunocompromised, in whom both susceptibility and consequence differ. Acquired immunity from repeated low-dose exposure is not modelled and would reduce risk in high-exposure populations, flattening the benefit of intervention where baseline exposure is highest.

The exposure model treats servings as independent and identically distributed, ignoring clustering of consumption within households, correlation between a household’s hygiene practices across occasions, and the distribution of poultry consumption across the population, which is far from uniform. Only broiler meat is considered, whereas *Campylobacter* is also acquired from other meats, raw milk, water, produce and direct animal contact, so the attributable fraction modelled here is a component of total incidence rather than all of it.

Finally, no cost is attached to any intervention. A 2-log processing reduction and a 40-point fall in flock prevalence are compared on effect alone, and the comparison that a regulator needs is effect per unit cost, including the commercial consequences of measures such as freezing. That analysis requires data this study does not contain and is the natural extension of it.

5. Conclusion

In this model a single log of reduction in *Campylobacter* concentration on broiler carcasses averted 83.5 % of illness, while reducing flock prevalence from 60 % to 20 % averted 67.5 %. The difference arises because the dose–response relationship is steeply concave at low dose: reducing prevalence removes some people from exposure and leaves the dose unchanged for everyone else, whereas reducing concentration moves every exposure down the part of the curve where the slope is greatest.

Two further results follow from the same structure. Risk is concentrated, with the most contaminated tenth of carcasses carrying 76.3 % of it, so positives are not equivalent and a prevalence count treats them as if they were. And cross-contamination to ready-to-eat food produced 62.8 % of modelled illness against 36.4 % from undercooked meat, because the smaller dose is the one that reaches the consumer without passing through a kill step.

The recommendation is therefore about measurement before it is about intervention. Flock prevalence is counted because it is binary, cheap and attributable to a producer; carcass concentration determines how much disease occurs. A control programme that reports only the first is monitoring the quantity it can measure conveniently rather than the one it is trying to change.

Declarations

Ethical approval. Not applicable. This is a computational risk assessment using simulated servings and published dose–response parameters; no human participants, animals, clinical isolates or surveillance records were involved.

Funding. [To be completed by the authors.]

Conflicts of interest. [To be completed by the authors. Any relationship with poultry producers, processors or retailers should be disclosed.]

Scope disclaimer. No country, producer, processor, product or national control programme is evaluated. Prevalence, contamination, transfer and behaviour parameters are representative assumptions and the model is calibrated, not fitted; absolute incidence figures must not be cited as estimates for any population.

Model disclaimer. The comparison between control measures rests on the concavity of the dose–response function and is robust to the calibration; the split between exposure pathways is more sensitive to unmeasurable parameters, as set out in Section 4.3.

Data and code availability. No surveillance or laboratory data were used. All results were generated by the model described in Section 2; the code, run with a fixed random seed, reproduces every reported value and is available from the corresponding author.

Author contributions. [To be completed by the authors in accordance with ICMJE authorship criteria.]

References

1. European Food Safety Authority Panel on Biological Hazards. Scientific opinion on quantification of the risk posed by broiler meat to human campylobacteriosis in the EU. *EFSA J.* 2010;8(1):1437.
2. European Food Safety Authority. Update and review of control options for *Campylobacter* in broilers at primary production. *EFSA J.* 2020;18(4):6090.
3. Nauta MJ, Jacobs-Reitsma WF, Havelaar AH. A risk assessment model for *Campylobacter* in broiler meat. *Risk Anal.* 2007;27(4):845–861.
4. Nauta M, Hill A, Rosenquist H, et al. A comparison of risk assessments on *Campylobacter* in broiler meat. *Int J Food Microbiol.* 2009;129(2):107–123.
5. Rosenquist H, Nielsen NL, Sommer HM, Nørnung B, Christensen BB. Quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* species in chickens. *Int J Food Microbiol.* 2003;83(1):87–103.
6. Teunis PFM, Havelaar AH. The Beta Poisson dose-response model is not a single-hit model. *Risk Anal.* 2000;20(4):513–520.
7. Teunis P, Van den Brandhof W, Nauta M, Wagenaar J, Van den Kerkhof H, Van Pelt W. A reconsideration of the *Campylobacter* dose-response relation. *Epidemiol Infect.* 2005;133(4):583–592.
8. Black RE, Levine MM, Clements ML, Hughes TP, Blaser MJ. Experimental *Campylobacter jejuni* infection in humans. *J Infect Dis.* 1988;157(3):472–479.
9. Havelaar AH, Kirk MD, Torgerson PR, et al. World Health Organization global estimates and regional comparisons of the burden of foodborne disease in 2010. *PLoS Med.* 2015;12(12):e1001923.
10. Kaakoush NO, Castaño-Rodríguez N, Mitchell HM, Man SM. Global epidemiology of *Campylobacter* infection. *Clin Microbiol Rev.* 2015;28(3):687–720.
11. Nachamkin I, Allos BM, Ho T. *Campylobacter* species and Guillain-Barré syndrome. *Clin Microbiol Rev.* 1998;11(3):555–567.
12. Lubber P, Brynestad S, Topsch D, Scherer K, Bartelt E. Quantification of *Campylobacter* species cross-contamination during handling of contaminated fresh chicken parts in kitchens. *Appl Environ Microbiol.* 2006;72(1):66–70.
13. Kusumaningrum HD, Riboldi G, Hazeleger WC, Beumer RR. Survival of foodborne pathogens on stainless steel surfaces and cross-contamination to foods. *Int J Food Microbiol.* 2003;85(3):227–236.
14. Georgsson F, Thornorkelsson AE, Geirsdóttir M, Reiersen J, Stern NJ. The influence of freezing and duration of storage on *Campylobacter* and indicator bacteria in broiler carcasses. *Food Microbiol.* 2006;23(7):677–683.
15. Sandberg M, Sorensen LL, Steenberg B, Chowdhury S, Jespersen AM, Alban L. Risk-based approach to control *Campylobacter* in broiler flocks and meat. *Food Control.* 2015;58:2–8.

16. Codex Alimentarius Commission. Guidelines for the control of Campylobacter and Salmonella in chicken meat. CXG 78-2011. Rome: FAO/WHO.
17. Havelaar AH, Mangen MJJ, de Koeijer AA, et al. Effectiveness and efficiency of controlling Campylobacter on broiler chicken meat. Risk Anal. 2007;27(4):831–844.