PEER REVIEW HISTORY

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ARTICLE DETAILS

TITLE (PROVISIONAL)	A modelling study to estimate the health burden of foodborne disease: cases, general practice consultations and hospitalisations in the UK, 2009
AUTHORS	O'Brien, Sarah; Larose, Tricia; Adak, Goutam; Evans, Meirion; Tam, Clarence

VERSION 1 - REVIEW

REVIEWER	Robert Scharff The Ohio State University, USA
REVIEW RETURNED	05-Feb-2016

GENERAL COMMENTS	This is a potentially important paper that seeks to provide estimates for the burden of foodborne illness in the UK. The paper builds on the Tam et. al. (2012) study that estimated incidence of infectious disease in the UK by expanding the number of pathogens covered, assessing the portion of that burden attributable to foodborne illness, and estimating hospitalization rates. Though the paper does make a contribution, there are a number of issues that need to be handled/clarified, before publication would be recommended. 1. p. 3 line 27. The material in the text does not convince me that this is true. Can you make the argument for this to me? 2. p. 3. line 48. I'm not sure this is the conclusion of your paper. The conclusion should be related to the aims of the paper. 3. p. 5. line 27, p. 6-7. The description of the literature review on 6-7 is not really that useful. I'd much rather actually see a real literature review that examines this study in the context of the existing literature. I have no idea how the literature that was reviewed was used to inform this study. Also, reference 4 (Mead) was made obsolete by references 2 and 3 (Scallan et. all, 2011), which are believed to use better data and methods. 4. I'm concerned about the limitation of outbreak data to 2001-2008. This gives you very small numbers of illnesses/hospitalizations upon which to build your estimates. Also, if you were to choose any period, why wouldn't you choose data from 2009-2015 to get the most up too date estimates? I'm not sure what the reporting changes were after 2008, but the crucial question is whether they are likely to affect the proportion of illnesses assessed as foodborne or the proportion hospitalized. If there is no reason to expect a big change in either of these numbers, the benefits from increased sample size would likely outweigh any concern about different reporting procedures.

	5. p.6 line 8. It's not clear how this data is used in your analysis. I wonder if this data has death rates associated with it (like the NNDSS does in the US). If it does, this may be a way of including deaths in your analysis, which would make it a more complete and valuable paper. 6. p8. lines 19-29 + Appendix. First, what is 'appendix 3'? Next, there are some very small numbers in table 1. The way you deal with this in the appendix (p. 29, lines 15-28; p.31, lines 22-28) is arbitrary. 7. p.9. lines 26-44. The assumption here is that studies with the high and low estimates have equal weight and all other studies have no weight. If you are going to assume that all studies are equally valid, this does not make much sense. It would make more sense to use a generalized distribution giving equal weight to each study used. That said, each study used does not have equal weight. As mentioned above, Mead (1999) has essentially been replaced by Scallan (2011) so Mead should be given little weight. Is there anyone on this team who has the experience/expertise to determine which studies are most valid? If so, use it. If not, I'd recommend bringing in an expert. 8. p.10 & appendix. The hospitalization models are not particularly well explained. As a result it's hard for me to assess the validity of the models. What are the parameter distributions based on in model 1? Model 2 and 3 supposedly use data from the lit review, but the data from these studies is nowhere to be found. How is this data integrated? This section needs to be made clearer. 9. p.13.lines 54-58. ...or not everyone made ill due to a source identified in an outbreak reports their illness. Scallan et al. (2011) dealt with this by using different multipliers for incidence, doctors visits, and hospitalizations. Could you use the approach in Scallan as a guide? Generally I like this effort and believe it could be very important! I would like to see some more work done before I actually believe the numbers you estimate, however.

REVIEWER	Elaine Scallan Colorado School of Public Health, USA
REVIEW RETURNED	06-Feb-2016

GENERAL COMMENTS	This rigorous study estimates the human health burden of foodborne disease in the UK caused by specific pathogens using existing data from prospective cohort studies, outbreak surveillance, and a systematic review of the literature. Different modeling approaches are used and compared. The use of prospective cohort study data was a strength allowing the authors to overcome the limitations of surveillance data, including various reporting biases. While the results of these cohort studies have been published they did not previously attempt to estimate the number of illnesses attributable to contaminated food which is of interest to many. Some additional information on the cohort studies in the methods may be useful, e.g., document sample sizes.

	Outbreak data was used to estimate the percent of IID attributable to foodborne transmission as well as percent resulting in hospitalization. I share the authors' concern about the potential for over or underestimation of these measures due to the possibility of over-reporting of foodborne outbreaks relative to non-foodborne outbreaks, and to the case finding associated with outbreak investigations that may detect milder secondary cases. However, the authors did a nice job of discussing these potential limitations. I would like some further explanation of why only outbreak data from 2001-2008 was used. The authors note in the methods that due to changes in reporting it was unclear is data were comparable between the two time periods. Why choose 2001-2008 instead of 2008-14? More recent data may be more relevant. Did the authors compare the two time periods and find significantly differences? These outbreak data are extremely valuable. Some discussion of why 6 years of data were not included is warranted. The systematic review of existing studies is an important component of this analysis and would be of interest to others. However, I found it difficult to determine exactly which 35 studies were included. I recommend that the authors provide a summary of the papers and the findings for the systematic review in an appendix. The evaluation of viral pathogens is a strength. However, as the authors note, the low number of norovirus infections relative to bacterial infections is not consistent with prior studies. The authors should comment why this might be, and if the differences in study results may be due to different sampling or measurement methods. The figures are too small to interpret or review. The conclusion is rather brief, and it would be nice to consider the relevance of the findings to public health prevention policies in a little more detail.

REVIEWER	Fiona Barker Turning Point, Monash University Australia
REVIEW RETURNED	08-Feb-2016

GENERAL COMMENTS	It was unclear if ethical approval was required and/or sought for this study. References for the technical appendix were missing. Overall, this manuscript was very well written and the authors appear to have applied a thorough and rigorous approach to the work. It presents a novel method to estimate the burden of foodborne disease in the UK. **Specific manuscript comments:** Page 3 Line 42 consider spelling out Clostridium Page 3 Line 44 space required after hyphen Page 4 Line 8-16 full stops at end of sentences Page 4 Line 14 change to plural "applied these" Page 6 Line 19 remove hyphen after colon Page 6 Line 42 remove comma after prisons Page 6 Line 39-42 The first part of the text in brackets is

	confusing – maybe change to "with the exception of patients with crypto or listeria".
Page 7 Line 49	May need to spell out Clostridium if first use.
Page 8 Line 35-36	reword to "..no cases were found in IID2 so we applied…"
Page 8 Line 50-51	this final sentence – why did you single out rv and sv? Hasn't the first sentence of this paragraph indicated that data was taken from IID2?
Page 10 Line 36	is it worth citing others who have used this method? Or explaining why you used this method?
Page 10	Did you need ethics approval for this project?
Page 12 Line 23	STEC –are you using this interchangeably with E coli O157 or is there new/different data available in Model 2 compared to Model 1?
Page 12 Line 3-37	this whole paragraph gets a bit confusing because you include comparison with model 1
Page 12 Line 24	Suggest you start a new paragraph/section with "In general, …"
Page 13 Line 17	indicate which model these results come from
Page 13 Line 44	change to "areas where data are sparse"
Page 14 Line 4	change to ", where estimates for hospitalisations were …"
Page 14 Line 8	change to "a GP"
Page 14 Line 47-52	this paragraph does not flow. Second sentence doesn't appear to have anything to do with first sentence. Please clarify.
Technical Appendix	
Page 29 Line 40	be consistent in how you format numbers – with/without comma
Page 31 Line 5	change to "that were hospitalised to get an estimate of…"

Tables

Table 1 & Table 2

- Heading needs more information – what year(s)? where?
- Is it worth including a footer citing the references for the data

Table 3

- Do you need to add UK 2009?

Table A1

- Worth defining reporting ratio again
- Why is there no E for source?
- It would make it much easier to follow the table if the symbols were defined at the bottom of the tables

Table A2 and Table A3

- Where are the references for source?

Figures

All of the figures were too poor of quality to read (except for those in the technical appendix). Also, Figure heading were missing.

Figure A1

- Graph for astrovirus – check sig figs – is this intentional?
- Figure heading – indicate that fitted Beta distribution is the

	red line

REVIEWER	Elaine Nsoesie University of Washington, USA
REVIEW RETURNED	07-Apr-2016

GENERAL COMMENTS	The paper deals with an important public health issue – the burden of foodborne illness. The data used in the study is from 1994-2008. This fact should be highlighted in the title and abstract. The data sources section should indicate the number of cases from the different data sources or reference the table. Since there are no projections made in the analyses, the results should clearly state that inference can only be made for the period for which data is available. There is a gross underreporting of foodborne illness, however, the authors do not state how they account for under-ascertainment. Lastly, the authors should clearly state how studies used in informing the Bayesian uniform priors were selected.

REVIEWER	Adrian Barnett Queensland University of Technology Australia
REVIEW RETURNED	17-May-2016

GENERAL COMMENTS	This was a well conducted studied that was well explained (including a detailed appendix) and provides useful information for policy makers. The parameters (and their uncertainty) could be useful for future modeling studies. The figures need to be of much higher quality (increased dots per inch) and I could not see any legends. There was no research checklist. I can't see a good match in the EQUATOR list, but perhaps STROBE is best. Minor comments - Title, Rather than “using novel methods” the title could explain the motivation to account for the pathogen and transmission uncertainty - Abstract, other studies have used a systematic review to inform priors, so I wouldn’t call this “unique”, perhaps in this field? - At the top of page 8 where you talk about the use of the outbreak data to inform the parameters I would state that this assumes that the parameters are the same during outbreak and non-outbreak periods, and that this assumption will be discussed in the conclusion - Similarly on page 9 concerning the use of uniform distributions, it might be useful to flag that this choice was based on the small number of studies available and this will be discussed in the conclusion - Page 9, line 40, how often did it happen that the mean was outside the limits? - Page 10, line 10, for these sub-headings I would use the text description (e.g., proportion due to food) followed by the Greek letter in brackets - Page 13, line 10, for ease of reading it may be best to round these numbers to the nearest thousand (or hundred) - Page 13, line 36, guess why?

	- Page 15, line 53, better baseline estimates would also inform predictions of the likely increase in foodborne disease due to climate change (see BMJ Open e010204, with apologies for self-citation) - You could consider using tornado plots to find which parameter is the greatest cause of uncertainty in the final estimate. Such plots can help direct where better data (or future research) is most needed. - Table 3 needs location and time scale (UK 2009?)

VERSION 1 – AUTHOR RESPONSE

Reviewer: 1

This is a potentially important paper that seeks to provide estimates for the burden of foodborne illness in the UK. The paper builds on the Tam et. al. (2012) study that estimated incidence of infectious disease in the UK by expanding the number of pathogens covered, assessing the portion of that burden attributable to foodborne illness, and estimating hospitalization rates. Though the paper does make a contribution, there are a number of issues that need to be handled/clarified, before publication would be recommended.

1. p. 3 line 27. The material in the text does not convince me that this is true. Can you make the argument for this to me?

We have moderated the wording in this sentence to say that the three modelling methods produced similar estimates of disease burden and uncertainty.

2. p. 3. line 48. I'm not sure this is the conclusion of your paper. The conclusion should be related to the aims of the paper.

We have amended the conclusion slightly so that it reflects the aims of the paper as well as containing comment on the implications of the findings.

3. p. 5. line 27, p. 6-7. The description of the literature review on 6-7 is not really that useful. I'd much rather actually see a real literature review that examines this study in the context of the existing literature. I have no idea how the literature that was reviewed was used to inform this study. Also, reference 4 (Mead) was made obsolete by references 2 and 3 (Scallan et. all, 2011), which are believed to use better data and methods.

We have now amended this section to include an abbreviated description of the literature review, the full version of which is available at http://www.food.gov.uk/science/research/foodborneillness/b14programme/b14projlist/fs231043ext *and is referenced at the end of the manuscipt. In the Technical Appendix, we have provided a complete reference list of the included studies that were used to inform Bayesian models. Reference 4 (Mead) is a reference used as supporting evidence for statements in the introduction of the submitted manuscript (page 5, line 10). Mead* et al *it is not a study that was used for Bayesian modelling. We hope the new information in the appendix with the full list of the included studies will prevent further confusion.*

On page 8, lines 26-30, we clearly state the parameters, "in the Bayesian approach…were modelled as binomial quantities and given priors informed by published studies…"

On page 9, lines 29-43, we further explain how we obtained prior distributions for the Bayesian parameters.

4. I'm concerned about the limitation of outbreak data to 2001-2008. This gives you very small numbers of illnesses/hospitalizations upon which to build your estimates. Also, if you were to choose any period, why wouldn't you choose data from 2009-2015 to get the most up to date estimates? I'm not sure what the reporting changes were after 2008, but the crucial question is whether they are likely to affect the proportion of illnesses assessed as foodborne or the proportion hospitalized. If there is no reason to expect a big change in either of these numbers, the benefits from increased

sample size would likely outweigh any concern about different reporting procedures.

There were substantial changes to outbreak reporting in 2009. Prior to that date PHE (and its predecessor organisations) collected data on all gastrointestinal infection outbreaks no matter what the route of transmission i.e. foodborne, waterborne, person-to-person, environment-to-person and animal-to-person. In 2009 PHE limited the collection of outbreak data on "non-foodborne outbreaks" to "gastrointestinal outbreaks including illnesses associated with recreational water exposure, environmental exposure at outdoor events e.g. contact with mud, contact with animals or their faeces and outbreaks of Vero cytotoxin-producing Escherichia coli *(VTEC) mediated through person-to-person transmission. Thus the data collected up to December 2008 are substantially different to those collected after that point. In particular the change affects the proportion of illnesses assessed as foodborne because the denominators of outbreaks and cases in outbreaks are changed substantially by the changes in reporting definitions. This is particularly problematic for pathogens like norovirus and* Cryptosporidium*. We elected to use data up to the end of 2008 because the outbreak dataset also coincided with the data capture for the IID2 study, which we relied on in these analyses.*

5. p.6 line 8. It's not clear how this data is used in your analysis. I wonder if this data has death rates associated with it (like the NNDSS does in the US). If it does, this may be a way of including deaths in your analysis, which would make it a more complete and valuable paper.

We agree that to be able to estimate deaths would be very valuable. Unfortunately laboratory reports do not contain information about deaths. Although we looked at several different data sets, including hospital episode statistics and death registrations, it was impossible to determine from these datasets deaths from food-related disease because of the way that deaths from gastrointestinal infection are recorded and coded. In the absence of alternative data sources, we used laboratory report data as a measure of community incidence of listeriosis. This is because Listeria *was not detected in either the IID1 or the IID2 studies. This is explained in Table A1, so we have deleted this sentence from the manuscript to avoid confusion.*

6. p8. lines 19-29 + Appendix. First, what is 'appendix 3'? Next, there are some very small numbers in table 1. The way you deal with this in the appendix (p. 29, lines 15-28; p.31, lines 22-28) is arbitrary.

We have corrected the typographical error. Appendix 3 should have been "Technical Appendix."

7. p.9. lines 26-44. The assumption here is that studies with the high and low estimates have equal weight and all other studies have no weight. If you are going to assume that all studies are equally valid, this does not make much sense. It would make more sense to use a generalized distribution giving equal weight to each study used. That said, each study used does not have equal weight. As mentioned above, Mead (1999) has essentially been replaced by Scallan (2011) so Mead should be given little weight. Is there anyone on this team who has the experience/expertise to determine which studies are most valid? If so, use it. If not, I'd recommend bringing in an expert.

We have provided a list of the studies that were included in the modelling in the Technical Appendix, which did not include Mead et al*. The reviewer suggests that we give importance weights to each study and have priors that give equal weight to estimated proportions from published studies. We disagree for a number of reasons. Firstly, it would mean that the priors give weight only to observed values, which would give rise to non-contiguous distribution and result in nonsensical posteriors. Statistically, observed values should be drawn from some underlying distribution, but filtered by chance and publication bias, so it makes sense to have more conservative priors, particularly as for most pathogens there were very few published studies. Another problem is that for some pathogens, like norovirus, variability in estimated proportions is likely to reflect, at least partly, real differences in epidemiology between settings, rather than merely chance or study quality. Ideally, one would want more informative distributions centred around the 'true' value, but this would only come from a multitude of studies that converge to the mean. In the absence of that evidence, we think that a conservative prior is more realistic. We also mention in the discussion that it is unreasonable to weight studies that have inherently different designs. Even within case-control studies, it is impossible most of the time to know exactly what risk factors they collected information on, or how much they focused on food-related versus other risk factors. There are various scoring systems for quality assessment of RCTs, case-control studies, cohort studies etc., but none of these suggest that one*

should compare the quality of RCTs with that of case-control studies using the same assessment tool, let alone expert elicitation or outbreak surveillance studies.

8. p.10 & appendix. The hospitalization models are not particularly well explained. As a result it's hard for me to assess the validity of the models. What are the parameter distributions based on in model 1? Model 2 and 3 supposedly use data from the lit review, but the data from these studies is nowhere to be found. How is this data integrated? This section needs to be made clearer.

We have amended the relevant text in the Technical appendix to clarify how we used the outbreak data to derive hospitalisation proportions for Model 1. Models 2 and 3 use data from the literature review to inform priors for the proportion of cases foodborne. The references for the studies used to derive priors for each pathogen are given in Table A2, but unfortunately our original file was missing the reference list at the end. We have now added this to the Technical Appendix.

9. p.13.lines 54-58. ...or not everyone made ill due to a source identified in an outbreak reports their illness. Scallan et al. (2011) dealt with this by using different multipliers for incidence, doctors visits, and hospitalizations. Could you use the approach in Scallan as a guide?

Since we had used as a basis for disease incidence the IID1 and IID2 population-based prospective cohort studies and prospective studies of presentation to primary care, in which we measured under-ascertainment very carefully, we did not need to estimate multipliers in the way that Scallan et al*. did. We know what the multipliers were because we had measured them directly and so they were accounted for in the analyses from the outset.*

Generally I like this effort and believe it could be very important! I would like to see some more work done before I actually believe the numbers you estimate, however.

Thank you for the encouragement.

Reviewer: 2

This rigorous study estimates the human health burden of foodborne disease in the UK caused by specific pathogens using existing data from prospective cohort studies, outbreak surveillance, and a systematic review of the literature. Different modeling approaches are used and compared.

Thank you for this supportive comment.

The use of prospective cohort study data was a strength allowing the authors to overcome the limitations of surveillance data, including various reporting biases. While the results of these cohort studies have been published they did not previously attempt to estimate the number of illnesses attributable to contaminated food which is of interest to many. Some additional information on the cohort studies in the methods may be useful, e.g., document sample sizes.

We have tabulated this information.

Outbreak data was used to estimate the percent of IID attributable to foodborne transmission as well as percent resulting in hospitalization. I share the authors' concern about the potential for over or underestimation of these measures due to the possibility of over-reporting of foodborne outbreaks relative to non-foodborne outbreaks, and to the case finding associated with outbreak investigations that may detect milder secondary cases. However, the authors did a nice job of discussing these potential limitations.

Thank you for this supportive comment.

I would like some further explanation of why only outbreak data from 2001-2008 was used. The authors note in the methods that due to changes in reporting it was unclear is data were comparable between the two time periods. Why choose 2001-2008 instead of 2008-14? More recent data may be more relevant. Did the authors compare the two time periods and find significantly differences? These outbreak data are extremely valuable. Some discussion of why 6 years of data were not included is warranted.

As noted in our response to reviewer 1, there were substantial changes to outbreak reporting in 2009. Prior to that date PHE (and its predecessor organisations) collected data on all gastrointestinal infection outbreaks no matter what the route of transmission i.e. foodborne, waterborne, person-to-person, environment-to-person and animal-to-person. In 2009 PHE limited the outbreak data on "non-foodborne outbreaks" that it collected to "gastrointestinal outbreaks including illnesses associated with recreational water exposure, environmental exposure at outdoor events e.g. contact with mud, contact with animals or their faeces and outbreaks of Vero cytotoxin-producing Escherichia *coli (VTEC) mediated through person-to-person transmission." Thus the data collected up to December 2008 are substantially different to those which have been collected after that point. In particular the change affects the proportion of outbreaks and illnesses assessed as foodborne. This is particularly problematic for pathogens like norovirus and* Cryptosporidium*.*

The systematic review of existing studies is an important component of this analysis and would be of interest to others. However, I found it difficult to determine exactly which 35 studies were included. I recommend that the authors provide a summary of the papers and the findings for the systematic review in an appendix.

We now provided a complete list of the studies included in the online Technical Appendix. We also provide a summary of findings from those studies that were used to define uniform distributions for π_p based on the minimum and maximum estimates of the proportion of cases transmitted through food.

The evaluation of viral pathogens is a strength. However, as the authors note, the low number of norovirus infections relative to bacterial infections is not consistent with prior studies. The authors should comment why this might be, and if the differences in study results may be due to different sampling or measurement methods.

A likely reason for this discrepancy is the way that outbreak data are used in the various modelling studies. The outbreak datasets vary in the composition and definitions for outbreaks to be included e.g. some include only foodborne outbreaks whilst others like ours, at least until 2008, captured gastrointestinal infection outbreaks no matter what the mode of transmission. This means that the proportion of norovirus transmitted through food is likely to be overestimated in datasets that contain only outbreaks transmitted through food. We have added a comment to this effect.

The figures are too small to interpret or review.

We have re-drawn all the figures.

The conclusion is rather brief, and it would be nice to consider the relevance of the findings to public health prevention policies in a little more detail.

Given the length of the manuscript we have not expanded this section further.

Reviewer: 3

It was unclear if ethical approval was required and/or sought for this study.

An Ethics Committee favourable opinion was not required. These were secondary analyses of previously collected, publicly available data. All datasets were completely anonymous and so there was no risk of disclosure of personal data. We have added a statement to this effect in the manuscript.

References for the technical appendix were missing.

We have rectified this omission.

Reviewer: 4

The paper deals with an important public health issue – the burden of foodborne illness. The data used in the study is from 1994-2008. This fact should be highlighted in the title and abstract.

We have amended the title and abstract to make it clear that the estimate is for 2009.

The data sources section should indicate the number of cases from the different data sources or reference the table.

We have referenced table 1 in the data sources section as suggested.

Since there are no projections made in the analyses, the results should clearly state that inference can only be made for the period for which data is available.

We have not sought to claim that these estimates are projections.

There is a gross underreporting of foodborne illness, however, the authors do not state how they account for under-ascertainment.

Since we had used the IID1 and IID2 population-based prospective cohort studies and prospective studies of presentation to primary care as a basis for disease incidence in the population and presenting to primary healthcare services, in which we measured under-ascertainment very carefully, we did not need to estimate multipliers. We know what the multipliers were because we had measured them directly and, by using these studies, under-ascertainment was accounted for in the analyses from the outset. We describe this in the third paragraph of the discussion.

Lastly, the authors should clearly state how studies used in informing the Bayesian uniform priors were selected.

We have provided a list of the studies that were included in the modelling in the Technical Appendix and have provided the link in reference 16 of the main paper that fully describes the systematic literature review.

Reviewer: 5

This was a well conducted studied that was well explained (including a detailed appendix) and provides useful information for policy makers. The parameters (and their uncertainty) could be useful for future modeling studies.

Thank you for this encouraging comment.

The figures need to be of much higher quality (increased dots per inch) and I could not see any legends.

We have re-drawn all the figures.

There was no research checklist. I can't see a good match in the EQUATOR list, but perhaps STROBE is best.

We had looked very carefully at the EQUATOR Network list prior to the original submission but could not find a research checklist that seems to apply to the modelling study that we have undertaken. The STROBE and RECORD research checklists apply to observational research but not, apparently, to modelling studies. So whilst the STROBE checklist is clearly appropriate for the IID1 and IID2 studies themselves, and PRISMA for the systematic review, neither checklist really addresses a modelling study using multiple different data sets, which is what we have done here. In short, we could not find an applicable checklist. We have drawn this to the attention of the Journal Editor in the covering letter to our revision.

Minor comments

- Title, Rather than “using novel methods” the title could explain the motivation to account for the pathogen and transmission uncertainty

The title does not contain the phrase "using novel methods" but we have reworded the statement of objectives in the abstract.

- Abstract, other studies have used a systematic review to inform priors, so I wouldn't call this "unique", perhaps in this field?

We have amended this statement as suggested.

- At the top of page 8 where you talk about the use of the outbreak data to inform the parameters I would state that this assumes that the parameters are the same during outbreak and non-outbreak periods, and that this assumption will be discussed in the conclusion

In general, we have avoided including items of discussion in the methods section of the paper, which is why we discuss this assumption in the discussion section of the paper.

- Similarly on page 9 concerning the use of uniform distributions, it might be useful to flag that this choice was based on the small number of studies available and this will be discussed in the conclusion

In general, we have avoided including items of discussion in the methods section of the paper, which is why we discuss this assumption in the discussion section of the paper.

- Page 9, line 40, how often did it happen that the mean was outside the limits?

There were three pathogens for which the mean fell outside the original uniform priors derived from the literature review. These were C. perfringens, Cryptosporidium *and norovirus. In all instances, estimates of the proportion foodborne based on outbreak data were lower than those suggested by the literature.*

- Page 10, line 10, for these sub-headings I would use the text description (e.g., proportion due to food) followed by the Greek letter in brackets

We have made this amendment as suggested.

- Page 13, line 10, for ease of reading it may be best to round these numbers to the nearest thousand (or hundred)

Thank you for this comment. However, we have left these numbers unchanged because we wish to illustrate the uncertainty around the estimates.

- Page 13, line 36, guess why?

A likely reason for this discrepancy is the way that outbreak data are used in the various modelling studies. The outbreak datasets vary in the composition and definitions for outbreaks to be included e.g. some include only foodborne outbreaks whilst others like ours, at least until 2008, captured gastrointestinal infection outbreaks no matter what the mode of transmission. This means that the proportion of norovirus transmitted through food is likely to be overestimated in datasets that contain only outbreaks transmitted through food. We have added a comment to this effect.

- Page 15, line 53, better baseline estimates would also inform predictions of the likely increase in foodborne disease due to climate change (see BMJ Open e010204, with apologies for self-citation)

We have added a comment to this effect.

- You could consider using tornado plots to find which parameter is the greatest cause of uncertainty in the final estimate. Such plots can help direct where better data (or future research) is most needed.

We thank the reviewer for this helpful suggestion. We feel, however, that including such plots would lengthen the paper substantially, as these would have to be done for 3 separate outcomes and 13

different pathogens, since parameter uncertainty varies by pathogen. That said, it is clear that uncertainty around hospitalisation parameters, and the proportion of cases foodborne are major sources of uncertainty for which more data would be useful. There is considerable uncertainty around incidence estimates for some pathogens, but it is unlikely that very much can be done to improve on these, as these are taken from one of only a very few studies that estimates pathogen-specific incidence in the community.

- Table 3 needs location and time scale (UK 2009?)

We have made this amendment.

VERSION 2 – REVIEW

REVIEWER	Adrian Barnett Queensland University of Technology Australia
REVIEW RETURNED	26-Jun-2016

GENERAL COMMENTS	The authors have answered my few previous queries. Minor comments - Page 11, Line 46 comma before 'respectively' - Page 12, the thinning and discarding of burn-in for the simulations may not have been necessary given there was no likelihood to maximise - Page 12, 'clearance' rather than 'favourable opinion' for the ethics committee?