Associations between ambient temperature and enteric infections by pathogen: a systematic review and meta-analysis

Paul L C Chua, Chris Fook Sheng Ng, Aurelio Tobias, Xerxes T Seposo, Masahiro Hashizume

Summary

Background Numerous studies have quantified the associations between ambient temperature and enteric infections, particularly all-cause enteric infections. However, the temperature sensitivity of enteric infections might be pathogen dependent. Here, we sought to identify pathogen-specific associations between ambient temperature and enteric infections.

Methods We did a systematic review and meta-analysis by searching PubMed, Web of Science, and Scopus for peer-reviewed research articles published from Jan 1, 2000, to Dec 31, 2019, and also hand searched reference lists of included articles and excluded reviews. We included studies that quantified the effects of ambient temperature increases on common pathogen-specific enteric infections in humans. We excluded studies that expressed ambient temperature as a categorical or diurnal range, or in a standardised format. Two authors screened the search results, one author extracted data from eligible studies, and four authors verified the data. We obtained the overall risks by pooling the relative risks of enteric infection by pathogen for each 1°C temperature rise using random-effects modelling and robust variance estimation for the correlated effect estimates. Between-study heterogeneity was measured using I^2, τ^2, and Q-statistic. Publication bias was determined using funnel plot asymmetry and the trim-and-fill method. Differences among pathogen-specific pooled estimates were determined using subgroup analysis of taxa-specific meta-analysis. The study protocol was not registered but followed the PRISMA guidelines.

Findings We identified 2981 articles via database searches and 57 articles from scanning reference lists of excluded reviews and included articles, of which 40 were eligible for pathogen-specific meta-analyses. The overall increased risks of incidence per 1°C temperature rise, expressed as relative risks, were 1·05 (95% CI 1·04–1·07; I^2 97%) for salmonellosis, 1·07 (1·04–1·10; I^2 99%) for shigellosis, 1·02 (1·01–1·04; I^2 98%) for campylobacteriosis, 1·05 (1·04–1·07; I^2 36%) for cholera, 1·04 (1·01–1·07; I^2 98%) for *Escherichia coli* enteritis, and 1·15 (1·07–1·24; I^2 0%) for typhoid. Reduced risks per 1°C temperature increase were 0·96 (95% CI 0·90–1·02; I^2 97%) for rotaviral enteritis and 0·89 (0·81–0·99; I^2 96%) for noroviral enteritis. There was evidence of between-pathogen differences in risk for bacterial infections but not for viral infections.

Interpretation Temperature sensitivity of enteric infections can vary according to the enteropathogen causing the infection, particularly for bacteria. Thus, we encourage a pathogen-specific health adaptation approach, such as vaccination, given the possibility of increasingly warm temperatures in the future.

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Department of Global Health Policy, Graduate School of Medicine, University of Tokyo, Bunkyo-ku, Tokyo, Japan (P L C Chua PhD, C F S Ng PhD, Prof M Hashizume PhD); **Department of Global Health, School of Tropical Medicine and Global Health, Nagasaki University, Nagasaki, Japan** (P L C Chua, C F S Ng, A Tobias PhD, X T Seposo PhD); **Institute of Environmental Assessment and Water Research, Spanish Council for Scientific Research, Barcelona, Spain** (A Tobias)

Correspondence to:
Mr Paul L C Chua, Department of Global Health Policy, Graduate School of Medicine, University of Tokyo, Bunkyo-ku, Tokyo 113–0033, Japan
paulchua@m.u-tokyo.ac.jp

Introduction

Enteric infections, which are caused by bacterial, viral, and protozoal enteropathogens, affect the regular functions of the intestinal tract, resulting in several gastrointestinal manifestations, such as nausea, vomiting, and diarrhoea.[1] These infections accounted for an estimated 1·7 million deaths (22 deaths per 100 000) and 4·5 billion episodes (0·61 episodes per person-year) globally in all ages in 2016.[2] Although the burden of enteric infections remains high, considerable reductions have been achieved, especially in mortality.[2,3] However, progress in reducing the number of future enteric infections could be affected by climate change, particularly by rising ambient temperatures. Such changes can seriously disrupt natural and human processes,[4] thereby affecting the disease ecologies of enteric infections.[5,6] These infectious intestinal diseases were thought to be indirectly affected by increasingly warm temperatures, which can increase pathogen loads in animal hosts and water systems,[7,8] increase drinking-water system contamination[8] and food spoilage,[9,10] and change water-consumption habits in people.[11]

Although the factors behind how ambient temperatures affect enteric infections are complex,[6] studies have reported associations between ambient temperature and enteric infections using statistical modelling techniques.[11] In the early 2000s, two prominent studies reported positive associations between temperature and cases of

Research in context

Evidence before this study

We searched PubMed, Web of Science, and Scopus on Dec 15, 2019, for systematic reviews and meta-analysis articles published in English between Jan 1, 2000, and Dec 1, 2019 that reported associations between temperature and enteric infections. The search included terms for outcome: "enteric", "diarrhoea", and "gastroenteritis"; for exposure: "temperature", "climate", "weather", and "meteorological"; and the term "review". We found seven systematic review articles, four of which did a meta-analysis. The first systematic review reviewed a substantial number of research articles that quantified associations between temperature and enteric infections published up to the year 2013. The majority of the included studies reported positive associations, but several included studies reported negative associations for viral infections. A companion meta-analysis article reported relative risks per 1°C temperature increase for all-cause infections (1·07 [95% CI 1·03–1·10]) and did taxa analysis for bacteria (1·07 [1·04–1·10]) and for virus (0·96 [0·82–1·11]). But no study had updated the systematic review and meta-analysis of enteric infections considering studies that analysed the specific enteropathogens causing the infections.

Added value of this study

Our study provided an updated systematic review and meta-analysis of pathogen-specific enteric infections and expanded the literature search to cover 13 common pathogens that cause such infections, whereas the previous review meta-analysed only by taxa, or by bacteria or virus. We did eight pathogen-specific meta-analyses, for non-typhoidal salmonellosis, shigellosis, campylobacteriosis, cholera, *Escherichia coli* enteritis, typhoid, rotaviral enteritis, and noroviral enteritis. Evidence of pathogen differences was observed in bacterial infections, although the unexplained heterogeneity remained substantial and the numbers of studies were unevenly distributed among subgroups.

Implications of all the available evidence

The pathogen-specific pooled estimates derived in this study can help to improve the modelling of the associations between ambient temperature and enteric infections by accounting for pathogen-specific temperature sensitivity. Increased understanding of pathogen-specific temperature sensitivity could help to inform how to approach pathogen-specific health-care adaptations, such as vaccinations, given the possibility of increased incidence of some enteric infections in the future owing to increasingly warm temperatures.

all-cause diarrhoea in Peru[12] and Fiji,[13] and became the basis of estimating the global risk of enteric infections caused by future temperature rises.[14,15] With the availability of further studies, the first systematic review and meta-analysis[11] of studies that reported associations between temperature and enteric infection showed that the majority of included studies found positive associations between ambient temperature and all-cause enteric infections. The meta-analysis quantified a 7% increase of incidence of all-cause infections per 1°C temperature rise,[16] which was similar to the estimate from the earlier Peru study.[12]

These associations between temperature and all-cause enteric infections provide a general overview of temperature-related risks for enteric infections but omit the specificity of particular risks, such as mode of transmission (eg, contaminated drinking water, food spoilage, or poor handwashing) and different enteropathogens. The advantage of quantifying cause-specific risks is that doing so can support and facilitate the implementation of increasingly tailored approaches to reduce temperature-related enteric infections, such as improved drinking water infrastructure and vaccination programmes. However, analysing temperature–enteric infection associations by specific modes of transmission remains a challenge, because current measuring tools for enteropathogens in the environment are imperfect, and the causal relationship between ambient temperature and enteric infections in humans is not yet fully established.[17] As a result, some studies have applied mathematical modelling tools to estimate risks of enteric infection caused by temperature changes. However, these studies are based on insufficient understanding of the dynamics of both enteropathogens in the environment and infection transmission.[18–20]

Advancements in diagnostics regarding the measurement of enteropathogens in humans has resulted in the increased availability of data about pathogen-specific enteric infections, and several studies[21–23] have analysed pathogen-specific temperature–enteric infection associations. The first systematic review and meta-analysis[16] of studies about such associations reported pooled estimates of temperature associations for taxa-specific infections, rather than pathogen-specific infections, because not enough studies were available. The systematic review found that the majority of included studies reported positive associations between temperature and bacterial infections, and negative associations between temperature and viral infections.[16]

This systematic review was intended to update the previous systematic review and meta-analysis of enteric infections[16] in terms of evidence used. In our study, we focused on common pathogens that cause enteric infections, as identified by the Global Enteric Multicentre Study group and the Institute of Health Metrics and Evaluation.[2,24] Analysing the temperature sensitivities of

enteropathogens can help to identify which pathogen could be infecting humans predominantly in a future in which increasingly warm ambient temperatures are a possibility. Such information could be valuable to support health-care adaptation measures, such as vaccination programmes, to deal with temperature-sensitive enteropathogens. Furthermore, determining pathogen-specific pooled estimates could help to model temperature-related enteric infections on a large geographical scale, given that all previous WHO global projections[14,15,25] were for all-cause enteric infections.

Methods

Study design and participants

For this systematic review and meta-analysis, we adopted a modified version of the US Office of Health Assessment and Translation (OHAT)[26] framework intended for use with environmental health topics.[27] The modified OHAT framework steps were: problem formulation, literature search, data extraction, risk-of-bias assessment, and evidence synthesis.[26] The protocol was not registered but followed the PRISMA guidelines for the reporting of systematic reviews (appendix p 1).

We formulated the research question following the population, exposure, comparator, outcomes framework (PECO). In the assessment of the associations between exposures and outcomes in environmental health, PECO is an increasingly accepted structure to direct the objectives of a systematic review.[28] Our PECO question was: among the general population (P), what is the effect of ambient temperature (E), per 1°C increase (C), on the risk of reported cases, hospital admissions or visits, or mortality caused by pathogen-specific enteric infections (O)?

We defined ambient temperature as the surface air temperature measured by weather stations or estimated by satellites or models,[29] as expressed in quantitative and continuous formats. For comparability, we chose time series regression or case-crossover studies that quantified pathogen-specific associations between ambient temperature and enteric infections and reported the relative risks (RRs) or percentage change per 1°C temperature rise.[16] We defined enteric infections as the intestinal infectious diseases listed in the International Classification of Diseases 10 Clinical Modification A00–A09 codes.[30,31]

Search strategy and selection criteria

We searched Web of Science, PubMed, and Scopus on Jan 2, 2020 for full-text original peer-reviewed time series regression or case-crossover studies, written in English and published from Jan 1, 2000, to Dec 31, 2019, using the search terms "diarrhoea" OR "gastroenteritis" AND "shigella" OR "campylobacter" OR "cholera" OR "escherichia coli" OR "clostridium" OR "aeromonas" OR "salmonella" OR "typhoid" OR "rotavirus" OR "norovirus" OR "adenovirus" OR "cryptosporidium" OR "entamoeba" AND "temperature" OR "ambient temperature" OR "climate" OR "weather" OR "meteorological". For the complete search syntax used for each database search, see appendix (p 2). We used pathogen-specific search terms for the most common enteric infections identified by the GBD Diarrhoeal Diseases Collaborators.[32] We also searched the reference lists of excluded review articles and included articles to identify additional literature for further screening. We restricted the literature search to articles published from Jan 1, 2000, onwards, to be consistent with the earliest reference used in WHO projections of temperature-related enteric infections.[14,15,25] We included articles that used continuous measurement of ambient temperature as the exposure criterion and that estimated the effect of associations between ambient temperature and mortality, hospital admissions, surveillance reports, or outpatient visits for enteric infections. We excluded studies that expressed ambient temperature as a categorical or diurnal range, or in a standardised format, and studies with non-human outcomes (eg, studies about animal diseases or water sample tests).

Two authors (PLCC and MH) each separately screened the search result titles and abstracts and then reviewed eligible full-text articles for definitions and details of exposure, health outcomes, and study design. At each review stage, the two authors discussed and reviewed their screening results to reach a consensus about inclusion.

See **Online** for appendix

Data extraction

One author (PLCC) extracted from included articles the publication year, study location, study population, ambient temperature data sources and characteristics (ie, type and temperature range), health outcome sources and characteristics (ie, aetiology), study design characteristics (ie, the years studied, temporal resolution, exposure lags, modelling approaches, and model specifications), and all reported effect estimates and CIs or SEs for the change in the amount of enteric infections per 1°C increase in ambient temperature. For the effect estimates and CIs shown in figures, we used WebPlotDigitizer (version 4.2) for data extraction. The XY plot from the software was calibrated using the available X and Y information from each article's figures, and was validated by the reported values, if available, from the articles. We extracted RRs or converted the reported percentage changes or β coefficients into RRs (appendix p 2).

For more on **WebPlotDigitizer** see https://automeris.io/WebPlotDigitizer/index.html

For studies with location-specific estimates, we added coordinates from each location's approximate centroids using Google maps and used the coordinates to identify the main climate categories (ie, temperate, snow, equatorial, and arid) of each study using an updated Köppen–Geiger climate classification map.[33] We also categorised the studies according to income for each country, using the World Bank classification of economies.[34] One author (PLCC) extracted data from each article and four authors (MH, CFSN, AT, and XTS) reviewed the data. Each category's descriptive statistics were calculated and shown.

Risk of bias assessment

We evaluated the internal validity of the study designs and the results of the selected articles using risk-of-bias assessment. Our experts (MH, CFSN, AT, and XTS) developed a risk-of-bias tool on the basis of publications by Luben and colleagues[35] and Carlton and colleagues,[16] and OHAT risk-of-bias tools[26] (appendix pp 3–4). The final risk-of-bias tool was used to examine separately exposure assessment, outcome assessment, confounding bias, selection bias, selective reporting, and other biases domains. Each domain was scored separately using four categories (ie, low, probably low, probably high, and high). In the exposure assessment, low and probably low scores favoured finer temporal resolution (ie, daily), appropriateness of the spatial coverage of the weather station or grid resolution, and no missing data. In the outcome assessment, low or probably low scores were given to studies that had a clear indication of a laboratory confirmatory test or clinical diagnosis, an assumed time of illness onset, and an absence of missing data. Under confounding bias, studies that statistically accounted for major potential confounders (particularly time-varying confounders) received low scores. For selection bias, low and probably low scores were based on data sources that closely represented the population within the study site (ie, studies using national surveillance systems had lower scores than single-hospital studies had). Selective reporting was scored according to whether the stated objectives were clearly answered in the results. A category called other biases was included in the assessment to allow the possible presence of biases not covered in the other domains. Two authors (PLCC and MH) each separately assessed the risk of bias for each included study, discussed their assessment results, and reached a consensus about any discrepancies.

Meta-analysis

The RRs and 95% CIs per 1°C temperature rise, which refer to changes in the incidence of enteric infections per 1°C temperature increases, were obtained by pooling pathogen-specific enteric RRs from time-series regressions and case-crossover studies. The RRs were selected from the final models defined by the study authors or from models with the most important covariates, regardless of whether they were for mean, minimum, or maximum ambient temperature.[16] If a study showed estimates from multiple temperature indices, we selected the RRs for mean temperatures, or for both minimum and maximum temperatures. For the multisite studies, the RRs were selected from the pooled location-specific estimates if available, to reduce publication bias. For studies that did not select or favour a specific lag, we selected the RRs from a single or a cumulative lag with the highest mean RRs. If a study reported both cold and heat effects from non-linear models, we chose the RRs for heat effects, because the selected comparator was for each 1°C temperature rise. For studies that had overlapping outcome data, we selected those with wider geographical scales, all-age groupings, or longer study years. We excluded studies with non-time-series regressions or case-crossover designs, with RRs that could not be converted into per 1°C temperature rise, with no reported SEs or CIs, or with data duplicated from other studies.

We did random-effects meta-analysis with inverse-variance weighting and restricted maximum-likelihood method for heterogeneity variance to pool the effect estimates from multiple studies separately for each enteropathogen.[36,37] The between-study heterogeneity was described using: the Q-statistic, which estimates excess variance in our data; I^2 as the proportion of observed variability not caused by sampling error; and τ^2 to quantify the variance of true effect sizes underlying our data.[38,39] Heterogeneity is considered moderate if I^2 values are at least 30%, substantial if at least 50%, and considerable if at least 75%.[40] We explored the differences among pathogen-specific estimates by doing subgroup analyses per taxa, because it has been established that bacterial and viral infections have opposite temperature associations to each other.[16]

The sensitivity analyses involved running the meta-analysis by: first, removing the estimates from the studies with a high risk of bias score for exposure assessment, outcome assessment, and confounding bias to further reduce bias in our findings; second, switching the estimates from heat-related effects to cold-related effects for studies that showed both effects; and third, doing leave-one-out analysis, which assessed the influence of individual estimates. We did the subgroup analysis by climate, country-level income, temporal resolution (ie, daily, weekly, or monthly), and modelling approach (ie, linear *vs* non-linear). We fitted a correlated-effects model with robust variance estimation and small-sample adjustment to account for the dependency of effect sizes extracted from the same study.[41] We also explored the sources of heterogeneity by selecting estimates with similar temporalities and lags. We evaluated publication bias by assessing the degree of funnel plot asymmetry, which refers to asymmetry in the scatter of risk estimates from small studies that have more positive results than negative results, obtained by linear regression of the treatment effect weighted on its SE.[42] We accounted for publication bias by the trim-and-fill method, which estimates the number of studies that are missing owing to publication bias and adjusts for the overall estimates.[43,44]

We used R software (version 4.0.2) for statistical computing in analysing the extracted data and the meta (version 4.13-0), metaphor (version 2.4-0), and robumeta (version 2.0) packages for running the meta-analysis and generating the forest plots and funnel plots.[41,45,46]

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

2981 studies were identified by the database searches, of which 811 were duplicates and removed. We screened 2170 titles or abstracts, of which 1998 studies were excluded. We did a full-text review of 115 studies identified by the database searches, of which 38 were included in the systematic review, and an additional 57 articles from the reference lists of excluded review articles or included articles of which 42 were included in the systematic review. Altogether, 80 studies (appendix pp 5–8) were included in the systematic review and 40 pathogen-specific studies were eligible for meta-analysis (figure 1, table). For descriptions of the articles included in the systematic review, see the appendix (pp 9–10).

Of the 40 pathogen-specific studies, ten (26 estimates) were for non-typhoidal salmonellosis, ten (14 estimates) were for shigellosis, eight (ten estimates) were for rotaviral enteritis, six (seven estimates) were for campylobacteriosis, four (four estimates) were for cholera, three (four estimates) were for *E coli* enteritis, two (two estimates) were for typhoid, and two (two estimates) were for noroviral enteritis. Among the 40 pathogen-specific studies, 17 (43%) were from high-income countries, 16 (40%) were from middle-income countries, and three (8%) were from low-income countries. Four (9%) studies contained a mixture of high-income, middle-income, or low-income countries. 23 (58%) of the pathogen-specific studies were from locations with a temperate climate, eight (20%) with a snow climate, five (13%) with an equatorial climate, and three (8%) with an arid climate. All the pathogen-specific studies used time-series regression models for the temperature associations; 24 (60%) of the studies used linear models, 16 (40%) used non-linear models. The table shows the 40 meta-analysis eligible studies.

Most of the articles that reported pathogen-specific estimates were scored as having a low or probably low risk-of-bias (appendix pp 11–15). Only three studies had a high risk-of-bias score, each with a different domain of high risk. The first study[50] had a high risk of selective reporting bias, because it reported the temperature–shigellosis associations from only one of the study sites and did not provide any further explanation about why other study sites had been excluded from the analysis. The second study[51] had a high risk of confounding bias, because it did not consider important time-varying confounders, such as seasonality and long-term trend. The third study[78] had a high risk of both exposure assessment bias, because the ambient temperature was measured annually or in the long term, and for confounding bias, because it did not consider controlling for time-varying confounders like long-term trend.

Random effects meta-analysis of pathogen-specific RRs showed that the risk of enteric infection incidence increased per 1°C rise in temperature by 5·1% (95% CI 3·6–6·7%) for non-typhoidal salmonellosis, 7·0% (4·4–9·6%) for shigellosis, 2·3% (0·7–4·0%) for campylobacteriosis, 5·4% (4·2–6·6%) for cholera, 4·3% (1·2–7·4%) for *E coli* enteritis, and 15·1% (7·1–23·6%) for typhoid (figure 2). However, decreased risks per 1°C temperature rise were observed for rotaviral enteritis (–4·4% [95% CI –10·5 to 2·1%]) and noroviral enteritis (–10·6% [–19·3 to –0·9%]; figure 3). The highest risk was

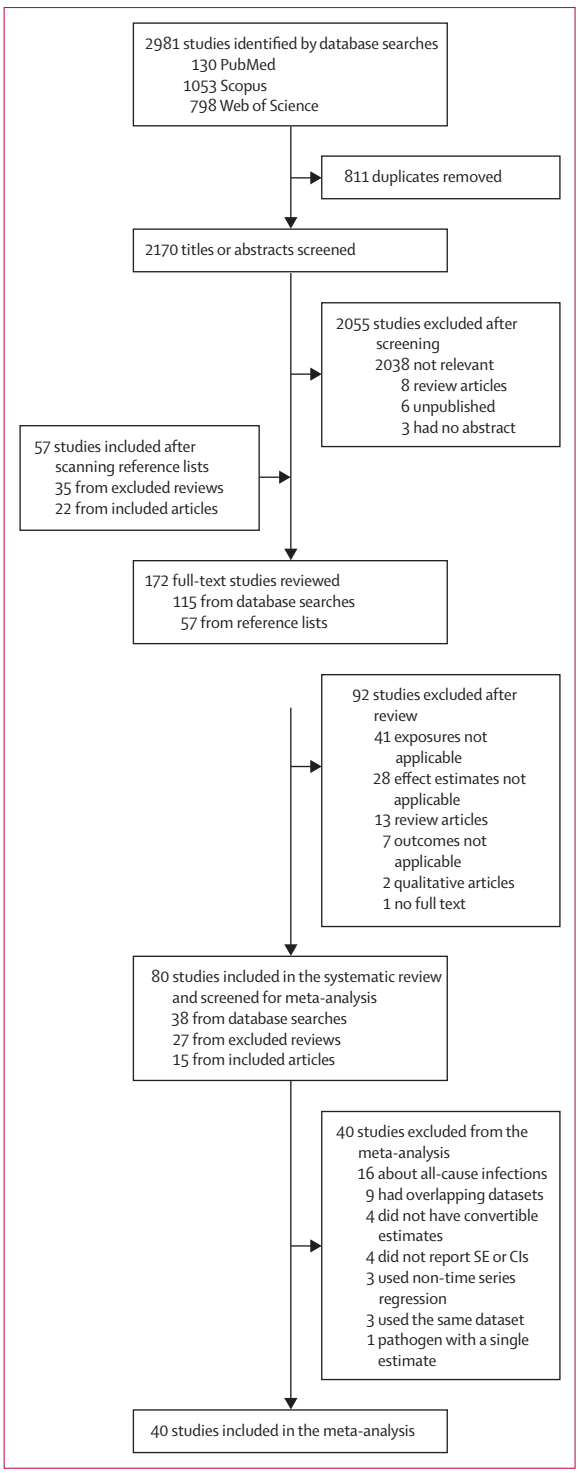

Figure 1: **PRISMA diagram of the systematic review and meta-analysis**

	Location	Years studied	Study population	Exposure	Outcome	Reporting frequency	Covariates	RR (95% CI) per 1°C ambient temperature increase
Shigella								
Aminharati et al (2018)[47]	Yazd province, Iran	2012–2015	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Humidity, rainfall, dust condition, seasons, and months	1·249 (1·077–1·450) at lag 0 months
Gao et al (2014)[48]	Changsha City, China	2004–10	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Humidity and atmospheric pressure	1·148 (1·050–1·246) at lag 1 month
Hao et al (2019)[49]	Anhui Province, China	2010–15	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Weekly	Seasonality, trend, and autocorrelation	1·034 (1·014–1·055) at lag 0–4 weeks for age <5 years; 1·069 (1·055–1·083) at lag 0–4 weeks for age >5 years
Lee et al (2017)[50]	Kom Tum, Vietnam	1999–13	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Humidity and calendar months	1·060 (1·040–1·090) at lag 0 months
Li et al (2013)[30]	Wuhan, China	2006–11	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Daily	Seasonality, trend, day-of-week, and holiday	1·009 (1·005–1·014) at lag 2 days*
Li et al (2014)[51]	Guangzhou, China	2006–12	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Relative humidity, wind velocity, rainfall, sunshine, and calendar year	1·036 (1·025–1·046) at lag 0 months
Li et al (2019)[52]	Xiangxi, China	2005–11	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Humidity, sunshine, rainfall, air pressure, seasonality, trend, and autocorrelation	1·027 (1·016–1·039) at lag 0 months
Liu et al (2019)[53]	Jinan, China	2005–13	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Daily	Humidity, rainfall, wind speed, sunshine, seasonality, and trend	1·100 (1·096–1·103) at lag 0–7 days
Yan et al (2017)[54]	Beijing, China	1970–2012	All ages	Mean temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Rainfall, seasonality, and autocorrelation	1·021 (1·005–1·037) at lag 7 months
Zhang et al (2007)[55]	Jinan, China; Baoan, China	1987–2000	All ages	Maximum and minimum temperatures from weather stations	Reported bacillary dysentery cases	Monthly	Seasonality and autocorrelation	1·114 (1·102–1·126) at lag 1 month for maximum temp in Jinan; 1·124 (1·110–1·138) at lag 1 month for minimum temp in Jinan; 1·157 (1·018–1·296) at lag 0 month for maximum temp in Baoan; 1·144 (1·009–1·279) at lag 0 month for minimum temp in Baoan
Salmonella								
Britton et al (2010)[56]	New Zealand	1965–2007	All ages	Mean temperatures from modelled weather stations records	Reported cases	Monthly	Outbreak indicator and autocorrelation	1·150 (1·071–1·242) at lag 0 months
D'Souza et al (2004)[57]	Perth, Australia; Melbourne, Australia; Sydney, Australia	1991–2001	All ages	Spatially interpolated mean temperatures from weather station records	Reported cases	Monthly	Seasonality and trend	1·041 (1·031–1·052) at lag 1 month in Perth; 1·056 (1·043–1·070) at lag 1 month in Melbourne; 1·051 (1·038–1·065) at lag 1 month in Sydney

(Table continues on next page)

	Location	Years studied	Study population	Exposure	Outcome	Reporting frequency	Covariates	RR (95% CI) per 1°C ambient temperature increase
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Fleury et al (2006)[58]	Alberta, Canada	1992–2000	All ages	Mean temperatures from weather stations	Reported cases	Weekly	Holiday, autocorrelation, region, seasonality, and trend	1·012 (1·009–1·015) at lag 1 week
Grjibovski et al (2013)[59]	Arkhangelsk City, Russia	1992–2008	All ages	Mean temperatures from weather stations	Reported cases	Monthly	Rainfall, seasonality, trend, and autocorrelation	1·020 (1·003–1·039) at lag 1 month
Grjibovski et al (2014)[60]	Astana; Almaty; north Kazakhstan; south Kazakhstan	2000–10	All ages	Mean temperatures from weather stations	Reported cases	Monthly	Rainfall, seasonality, trend, and autocorrelation	1·055 (1·022–1·088) at lag 0 months in Astana; 1·015 (0·973–1·058) at lag 0 months in Almaty; 1·000 (0·969–1·029) at lag 0 months in North Kazakhstan; 1·035 (0·979–1·091) at lag 0 months in South Kazakhstan
Kovats et al (2004)[61]	Poland, Scotland, Denmark, Estonia, the Netherlands, Czech Republic, Switzerland, Slovakia, Spain	2000–02 Poland; 1990–97 Scotland; 1991–2001 Denmark; 1990–2001 Estonia; 1984–2001 the Netherlands; 1993–2001 Czech Republic; 1990–2000 Switzerland; 1983–2000 Slovakia; 1994–2000 Spain	All ages	Mean temperatures from weather stations	Reported cases	Fortnightly (Poland); weekly (Scotland, Denmark, the Netherlands, Czech Republic, Spain); monthly (Estonia, Slovakia)	Seasonality, trend, and autocorrelation	1·087 (1·047–1·129) at lag 0–4 fortnights in Poland; 1·047 (1·021–1·073) at lag 0–9 weeks in Scotland; 1·011 (1·027–1·050) at lag 0–9 weeks in Denmark; 1·183 (1·036–1·351) at lag 0–2 months in Estonia; 1·093 (1·085–1·101) at lag 0–9 weeks in Netherlands; 1·095 (1·082–1·107) at lag 0–9 weeks in Czech Republic; 1·088 (1·076–1·099) at lag 0–9 weeks in Switzerland; 1·025 (0·974–1·076) at lag 0–2 months in Slovakia; 1·049 (1·034–1·064) at lag 0–9 weeks in Spain
Lake et al (2009)[62]	England and Wales	1981–2006	All ages	Mean temperatures from weather stations	Reported cases	Weekly	Seasonality, trend, and autocorrelation	1·054 (1·032–1·075) at lag 0–1 week
Milazzo et al (2016)[63]	Adelaide, Australia	1990–2012	All ages	Maximum temperatures from weather stations	Reported cases	Daily	Day-of-week, holidays, trend, and autocorrelation	1·013 (1·008–1·019) at lag 14 days in summer
Thindwa et al (2019)[64]	Blantyre, Malawi	2000–15	All ages	Mean temperatures from weather stations	Hospital admissions for febrile illness	Monthly	Rainfall, seasonality, and trend	0·933 (0·891–0·979 at lag 8 months†
Zhang et al (2010)[65]	Brisbane, Australia; Townsville, Australia	1990–2005	All ages	Maximum and minimum temperatures from weather stations	Reported cases	Weekly in Brisbane; monthly in Townsville	Rainfall, seasonality, trend, and autocorrelation	1·092 (1·079–1·105) at lag 2 weeks for maximum temp in Brisbane; 1·060 (1·051–1·069) at lag 2 weeks for minimum temp in Brisbane; 1·126 (1·091–1·162) at lag 0 months for maximum temp in Townsville; 1·060 (1·041–1·081) at lag 0 month for minimum temp in Townsville
Rotavirus								
Atchison et al (2010)[66]	England, Wales, Scotland, The Netherlands	1993–2007	Age <5 years	Mean temperatures from weather stations	Reported cases	Weekly	Humidity, rainfall, seasonality, trend, and public holidays	0·870 (0·850–0·890) at lag 0–4 weeks
Celik et al (2015)[67]	Sivas City, Turkey	2006–12	Age <5 years	Mean temperatures from weather stations	Hospital visits owing to acute gastroenteritis	Monthly	Humidity	0·947 (0·925–0·970) at lag 0 months
D'Souza et al (2008)[68]	Brisbane, Australia; Canberra, Australia; Melbourne, Australia	1993–2003	Age <5 years	Mean temperatures from weather stations	Hospital admissions owing to diarrhoea	Weekly	Seasonality, trend, autocorrelation, and population	0·951 (0·920–0·970) at lag 1 week in Brisbane; 0·970 (0·950–1·000) at lag 1 week in Canberra; 0·980 (0·960–1·000) at lag 1 week in Melbourne

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	Location	Years studied	Study population	Exposure	Outcome	Reporting frequency	Covariates	RR (95% CI) per 1°C ambient temperature increase
(Continued from previous page)								
Hashizume et al (2008)[69]	Dhaka, Bangladesh	1996–2001	All ages	Maximum temperatures from weather stations	Hospital visits owing to diarrhoea	Weekly	Humidity, river height, seasonality, trend, and holiday	1·402 (1·194–1·646) at lag 0–4 weeks
Hervás et al (2014)[70]	Mallorca, Spain	2000–10	Age <5 years	Mean temperatures from weather stations	Hospital admissions for gastroenteritis	Weekly	Solar radiation, wind speed, and atmospheric pressure	0·822 (0·790–0·855) at lag 1 week
Jagai et al (2012)[22]	Multicountry	1966–2010	All ages	Mean temperatures from weather stations	Cases from published articles	Monthly	Population and specific study	0·987 (0·985–0·989) at lag 0 months
Levy et al (2009)[21]	Multicountry	1988–2005	All ages	Mean temperatures from weather stations	Cases from published articles	Monthly	Autocorrelation and specific study	0·903 (0·867–0·941) at lag 0 months
Wang et al (2018)[71]	Hong Kong	2002–11	Age <5 years	Mean temperatures from weather stations	Hospital admissions	Daily	Humidity, rainfall, wind speed, solar radiation, seasonality, trend, day-of-week, holiday, and autocorrelation	0·956 (0·926–0·987) at lag 0–10 days
Campylobacter								
Allard et al (2011)[72]	Montreal, Canada	1990–2006	All ages	Mean temperatures from weather stations	Reported cases	Weekly	Seasonality, holidays, temperature, and autocorrelation	1·008 (1·003–1·013) at lag 1–6 weeks
Bi et al (2008)[73]	Brisbane, Australia	1990–2005	All ages	Maximum temperatures from weather stations	Reported cases	Weekly	Rainfall, season, and autocorrelation	1·009 (1·004–1·015) at lag 6 weeks in Brisbane
Fleury et al (2006)[58]	Alberta, Canada; and Newfoundland and Labrador, Canada	1992–2000	All ages	Mean temperatures from weather stations	Reported cases	Weekly	Seasonality, trend, holiday, autocorrelation, and region	1·022 (1·019–1·024) at lag 1 week in Alberta; 1·045 (1·033–1·058) at lag 1 week in Newfoundland and Labrador
Lake et al (2009)[62]	England and Wales	1989–2006	All ages	Mean temperatures from weather stations	Reported cases	Weekly	Seasonality, trend, and autocorrelation	1·053 (1·025–1·082) at lag 0–1 week
Milazzo et al (2017)[74]	Adelaide, Australia	1990–2012	All ages	Maximum temperatures from weather stations	Reported cases	Daily	Day-of-week, holidays, trend, and autocorrelation	0·995 (0·993–0·997) at lag 0 day in summer
White et al (2009)[75]	Philadelphia County, USA	1994–2007	All ages	Maximum temperatures from weather stations	Reported cases	Weekly	Humidity, river temperature, seasonality, and trend	1·049 (1·025–1·074) at lag 0 weeks
Vibrio cholerae								
Ali et al (2013)[76]	Matlab, Bangladesh	1988–2001	All ages	Minimum temperatures from weather stations	Reported cases	Monthly	Sea surface temperature, seasonality, and autocorrelation	1·064 (1·011–1·120) at lag 0 months
Luque Fernández et al (2009)[77]	Lusaka, Zambia	2003–06	All ages	Maximum temperatures from weather stations	Reported cases	Weekly	Rainfall, seasonality, and autocorrelation	1·052 (1·040–1·064) at 6 weeks

(Table continues on next page)

	Location	Years studied	Study population	Exposure	Outcome	Reporting frequency	Covariates	RR (95% CI) per 1°C ambient temperature increase
(Continued from previous page)								
Paz et al (2009)[78]	Sub-Saharan countries, Africa	1971–2006	All ages	Mean temperatures from weather stations	Reported cases	Annual	Sea surface temperature, temperature anomaly, and autocorrelation	1·107 (1·010–1·214) at lag 1 year
Trærup et al (2011)[79]	Tanzania	1998–2004	All ages	Maximum temperatures from weather stations	Reported cases	Monthly	Drought and trend	1·291 (1·039–1·605) at lag 0 months
Escherichia coli								
Bifolchi et al (2014)[80]	Alberta, Canada	2004–11	All ages	Mean temperatures from weather stations	Reported cases and hospital admissions	Monthly	Location and animal farming	1·034 (1·020–1·048) at lag 0 month for reported cases; 1·006 (1·003–1·010) at lag 0 months for hospital admissions
Fleury et al (2006)[58]	Alberta, Canada	1992–2000	All ages	Mean temperatures from weather stations	Reported cases	Weekly	Seasonality, trend, autocorrelation, holiday, and region	1·060 (1·050–1·069) at lag 1 week
Philipsborn et al (2016)[23]	Multicountry	1974–2004	All ages	Mean temperatures from weather stations	Incidence from published articles	Monthly	Rainfall, autocorrelation, and mortality	1·080 (1·050–1·110) at lag 0 months
Typhoidal *Salmonella*								
Dewan et al (2013)[81]	Dhaka, Bangladesh	2005–09	All ages	Mean temperatures from weather stations	Hospital admissions owing to typhoid fever	Weekly	River level, rainfall, seasonality, trend, and holiday	1·142 (1·044–1·250) at lag 0–4 weeks
Thindwa et al (2019)[64]	Blantyre, Malawi	2011–15	All ages	Mean temperatures from weather stations	Hospital admissions owing to febrile illness	Monthly	Rainfall, seasonality, and trend	1·166 (1·039–1·315) at lag 5 months†
Norovirus								
Lopman et al (2009)[82]	England and Wales	1993–2006	All ages	Mean temperatures from weather stations	Reported cases	Daily	Relative humidity, population immunity, epidemic seasons controlling for trend, improving diagnostics, seasonality, holidays, weekends, and autocorrelation	0·850 (0·830–0·860) at lag 49 days
Wang et al (2018)[71]	Hong Kong	2002–11	Age <5 years	Mean temperatures from weather stations	Hospital admissions	Daily	Humidity, rainfall, wind speed, solar radiation, seasonality, trend, day of week, holiday, and autocorrelation	0·944 (0·909–0·981) at lag 0–7 days

*Measured using WebPlotDigitizer. †Effect estimates for high temperature range.

***Table*: Eligible time series studies for meta-analysis by pathogen**

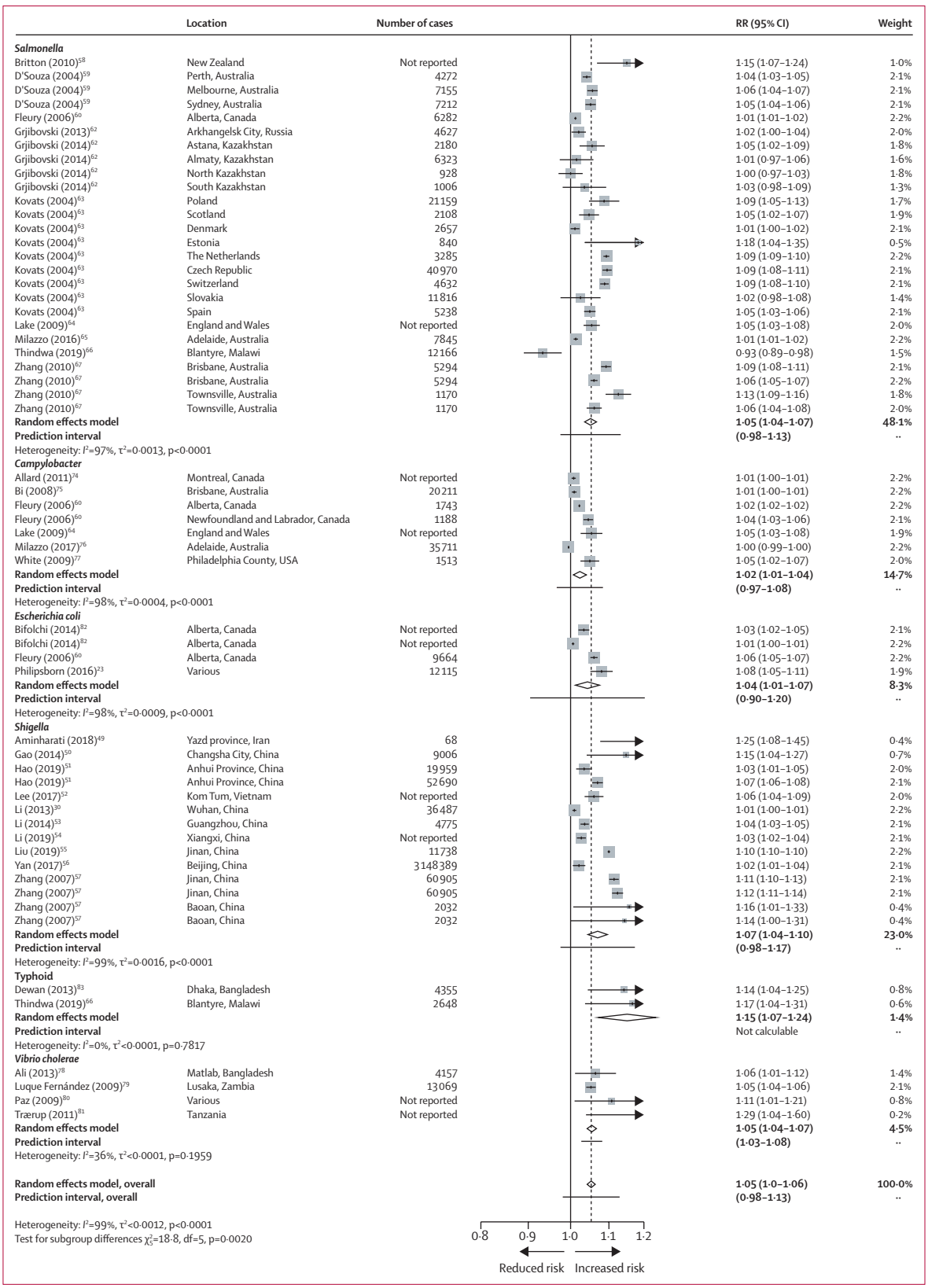

Figure 2: **Forest plot showing random effects meta-analysis of risk estimates for associations between temperature rise and bacterial enteric infections with subgroup analysis by pathogen**

RR of enteric infection incidence per 1°C temperature rise. df=degrees of freedom. RR=relative risk.

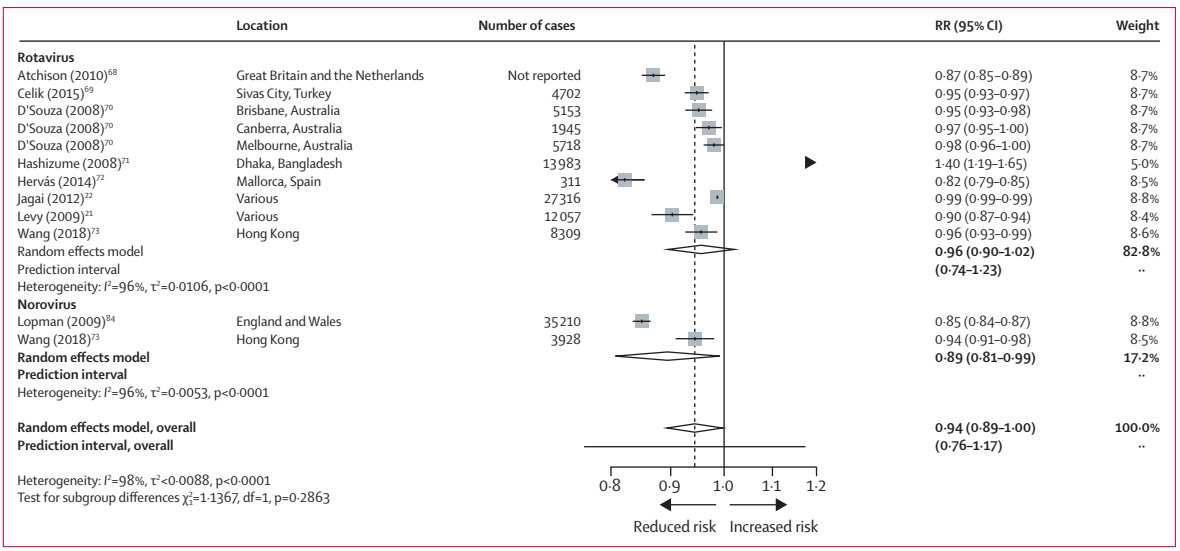

Figure 3: **Forest plot showing random effects meta-analysis of risk estimates for associations between temperature rise and viral enteric infections with subgroup analysis by pathogen**

RR of enteric infection incidence per 1°C temperature rise. df=degrees of freedom. RR=relative risk.

observed for typhoid and the lowest risk for noroviral enteritis. Large heterogeneity was present in all pathogens except for typhoid. All the estimates except for cholera had wide prediction intervals in which the value of 1·00 was included. The results of each pathogen-specific meta-analysis are summarised in the appendix (pp 17–18, 26, 31, 36, 38–40).

Subgroup analysis to synthesise the estimates of bacterial enteric infections resulted in significant between-subgroup differences among pathogens (p=0·0020; figure 2). Sensitivity analysis, done by omitting the estimates for typhoid, retained the significant between-subgroup difference among bacterial pathogens (p=0·0152; appendix p 41). Subgroup analysis for the estimates of viral pathogens did not show any between-subgroup difference (p=0·29; figure 3). Both subgroup analyses had large heterogeneity and unevenly distributed subgroups.

Leave-one-out analysis from each pathogen-specific meta-analysis (except for typhoid and noroviral enteritis, which had only two studies each) revealed that some studies had considerable influence, but generally did not change the summary estimates and the large heterogeneity (appendix pp 19, 26, 31, 36, 38, 39). For shigellosis and cholera, omitting the estimates from studies with a high risk of bias did not change the effect estimate and heterogeneity (appendix pp 26, 38). However, omitting an outlier study did significantly reduce the heterogeneity for cholera and the summary estimates for rotavirus (appendix pp 31, 38). Additional sensitivity analysis was done for non-typhoidal salmonellosis and typhoid for a study that reported heat and cold effects. Switching the RRs from heat effects to cold effects did not change the summary estimates and heterogeneity for non-typhoidal salmonellosis, but did change the effect estimate for typhoid (appendix pp 19, 40).

Subgroup analysis was done for the pooled estimates of non-typhoidal salmonellosis, shigellosis, campylobacteriosis, and rotaviral enteritis (appendix pp 19–24, 27–29, 32–34, 37). For climate, income, and temporality subgroup analysis, only non-typhoidal salmonellosis showed a between-subgroup difference, but heterogeneity remained large. The rest of the pathogens had unevenly distributed subgroups for climate, income, and temporality. For cumulative and single-lag subgroup analyses, no between-subgroup difference was found. For linear and non-linear model subgroup analyses, only shigellosis showed a between-subgroup difference, but the heterogeneity remained substantial.

Funnel plot asymmetry was done with the estimates of non-typhoidal salmonellosis, shigellosis, and rotaviral enteritis, which had at least 10 estimates (appendix pp 25, 30, 35). Estimates of non-typhoidal salmonellosis and rotaviral enteritis had funnel plot asymmetry based on Egger's test. The trim-and-fill method showed a reduced effect estimate for non-typhoidal salmonellosis, but a slightly increased one for rotaviral enteritis that was closer to the null association (appendix pp 25, 35).

Robust variance estimation was done for non-typhoidal salmonellosis, shigellosis, campylobacteriosis, and rotaviral enteritis because they had multiple estimates per study (appendix p 17). The pooled estimates derived from robust variance estimation were similar to the those derived from random effects meta-analysis, but resulted in wider 95% CIs, with the pooled estimates of campylobacteriosis and rotaviral enteritis including the value 1·00 or null association.

Discussion

We did pathogen-specific pooled analyses of estimates representing associations between temperature and

enteric infections. We found that incidence risk for non-typhoidal salmonellosis, shigellosis, campylobacteriosis, cholera, *E coli* enteritis, and typhoid increased per 1°C temperature rise on average. Conversely, incidence risk for rotaviral enteritis and noroviral enteritis decreased per 1°C temperature rise on average. These findings accord with previous findings of a taxa-specific meta-analysis[16] that showed increased risk of bacterial pathogens and decreased risk of viral pathogens per 1°C temperature rise. However, beyond taxa, subgroup analysis by pathogen revealed evidence of between-pathogen differences among the pooled estimates of bacterial pathogens, which suggested pathogen-specific temperature sensitivity. However, this sensitivity was not observed for the pooled estimates of viral pathogens.

It should be noted that the results of the pathogen-specific meta-analysis were uncertain because of the wide prediction intervals and large heterogeneity that remained. Even though we considered a few categories for subgroup analysis—and found significant between-subgroup differences among climate regions, country-level income, temporal resolution, and modelling approach—the heterogeneity remained substantial. Most of the subgroup analyses were based on unevenly distributed subgroups with different numbers of studies, possibly contributing to the uncertainty of the subgroup results. There were many possible sources of heterogeneity. First was the exposure measurement, because studies collected ambient temperatures from different sources with varying definitions. For example, ground-based weather stations and satellite data would contain different temperature values, ranges, and spatial characteristics to each other. Second was the outcome measurement, because sources range from hospitals to surveillance reports, which would mean varying degrees of infection severity and means of diagnosis. Diagnostics to accurately determine enteropathogens in humans are usually available in high-income countries but not in the low-income nations where most of the infections are harboured. Third was age groups of the enteric infections, because some studies involved children and most involved all age groups. Vulnerability and susceptibility to enteric infections vary by age and children might have higher risks compared with other age groups. Fourth was the modelling approach, because studies modelled temperature–enteric infection associations differently to each other and many applied varying model specifications. We did subgroup analysis between linear and non-linear models but this showed no substantial between-subgroup differences (appendix pp 23, 28, 33). Fifth was the definition of the estimate, which was change in risk per 1°C temperature rise. This definition could introduce heterogeneity, because the risk function might differ in a location or climate region with a different temperature range. Finally, each pathogen could also infect humans through varying modes of transmission, which could assume different risk functions. The substantial heterogeneity can be contained by having a consistent methodological approach, possibly by modelling a specific mode of transmission,[19,83] and setting similar definitions for ambient temperatures and enteric infection cases, similar to what has been done in the modelling of temperature effects on all-cause mortality.[84]

The pathogen-specific results were also uncertain because of the small number of studies available, especially for campylobacteriosis, cholera, *E coli* enteritis, typhoid, and norovirus. For protozoal infections, we found only a single estimate for cryptosporidiosis. Additional studies in the future could easily change the pathogen-specific effect estimates. For future research in modelling the associations between temperature and enteric infections, we highly encourage that pathogen-specific analysis be explored whenever possible and whenever better diagnostics becomes generally available. Such studies would provide additional evidence and update the pathogen-specific pooled estimates.

Based on the funnel plots, there was evidence of publication bias for salmonellosis, shigellosis, and rotavirus estimates. This concern is particularly true for the sets of estimates reported from the included studies, because they overwhelmingly reported significant associations and only one study reported estimates with null associations. Although extending the literature search to include unpublished studies would help, publication bias can be avoided by doing studies using a single methodological approach with similar sets of temperature and enteric infections data.

Significant differences among bacterial pathogens were detected despite the large heterogeneity that widened the SEs of each pathogen-specific pooled estimate. Even when the typhoid risk estimates (which had the highest positive associations among bacterial pathogens) between ambient temperature and infections) were removed, the significant between-pathogen difference was retained. However, any effects to be found using subgroup analysis of studies on bacterial pathogens are likely to be small. In that case, the non-significant difference between two viral pathogens might not truly reflect the absence of difference, because of the low statistical power of the subgroup analysis to detect small differences.[85] Doing further studies on viral pathogens (ie, beyond rotavirus and norovirus) and on bacterial pathogens, by following a single methodological approach using similar datasets from multiple locations, would help to detect any difference among pathogens in the risk estimates.

Nonetheless, the associations derived in this study can be explained by the direct or indirect effects of temperature on the growth and proliferation of different enteric pathogens, depending on the environment and animal hosts.[9,86] For mesophilic bacteria like *Salmonella* (non-typhoidal and typhoidal), *Shigella*, *Campylobacter*, and *E coli*, several in-vitro research studies found that these bacteria grow optimally in temperatures around 37°C, which is similar to human body temperature, although all

of these bacteria can survive temperatures higher than 40°C.[88–91] Studies on *Salmonella enterica* serovars found that growth rates in food were enhanced at room temperature (mostly defined as 25°C) in products like pork, fish, and eggs compared with at low temperatures (less than 25°C).[86,92,93] *S enterica* isolates also had a better growth rate and reached maximum expression of virulent genes in fish within 24 h at 45°C compared with at low temperatures.[92] Similarly, *Shigella* and *E coli* isolates were found to have increased growth at room temperature in various foods and even had better growth rates at 30°C or higher, compared with at 5°C for some foods and conditions; *Shigella* grew more at 37°C or 25°C than at 5°C after 72 h on cooked rice, mashed potato, milk, lentil soup, cooked fish and beef, and cucumber[94] and *E coli* grew more at 30°C than at 5°C after 24 h on beef salads with mayonnaise[95] and more at 25°C than at 5°C after 48 h on sliced peaches.[96] Farm animals such as pigs, cattle and chickens shed a considerably higher number of *Salmonella*, *E coli*, and *Campylobacter* during summer and early autumn than during the colder months in Greece,[86] Norway, Denmark, and Scotland,[97] the USA,[98] and the Netherlands.[99] A study in Norwegian farms producing chicken meat revealed that the proportion of chicken flocks that were positive for *Campylobacter* increased as the temperature increased from 0°C.[100] *Vibrio cholerae* is an autochthonous aquatic bacterium, and its detection in the waters of northern Chesapeake Bay, USA, is more frequent in summer than during colder months, and correlates with higher surface water temperatures and low salinity.[101]

However, the heat capabilities of such bacterial pathogens do not fully translate into their survival in the environment, because of a major role of other factors, such as pH, nutrients, moisture, and medium. For *Salmonella*, *Shigella*, *E coli*, and *Campylobacter*, numerous studies[86,97,102–104] reported increased die-off of isolates in surface water, soil, animal manure, and sewage at high temperatures compared with at low temperatures, although manure-amended soils improved survival of *Salmonella* at high temperatures compared with non-manure-amended soils.[105]

Unlike bacterial pathogens, rotavirus and norovirus generally survive in the environment and on various surfaces better at low than warm temperatures.[9,106] Using a model for human rotavirus, higher rotaviral infectivity was observed at 4°C than at 20°C after 30 days in both river and tap water.[107] This increase was also observed for soils, vegetables, porous, and non-porous inanimate surfaces, in which rotaviral infectivity was higher at refrigeration temperatures (4°C) than at room temperature (20–25°C) or warmer (37°C).[107–110] For norovirus, viral surrogate infectivity reduced significantly at room temperature compared with at 4°C on surface water, oysters, and peppers.[111,112] High temperatures on surface water made the noroviral surrogate undetectable after 7 days at 30°C and after 3 days at 65°C.[112] However, there were instances that rotavirus and norovirus infectivity was retained at warm temperatures. For liquid mediums such as some fruit juices or saline solutions with a low acidity, rotaviral infectivity could be kept after several h at room temperature or even warmer.[113,114] Human rotaviral particles in faeces have also been noted to remain infective at around 30°C for more than 2 months.[115] Noroviral surrogate infectivity at room temperature can be kept in manure and biosolids for up to 60 days,[116] or on non-porous surfaces for up to 28 days, as long as relative humidity is around 80%.[117]

These examples of heat susceptibility of enteropathogens suggest that warmer temperatures can increase the incidence of human enteric infections, particularly bacterial infections, mainly through pathways related to food or animals. However, there is uncertainty about these pathways because they are indirect and complex. For foodborne pathways, inadequate food storage provision or poor practices in handling and preparing food (which might occur more often in low-resource or marginalised settings than elsewhere) can result in contamination of food at ambient temperatures. Summertime gatherings could also be a source of transmission, because food is increasingly likely to be exposed to ambient temperatures. For animal reservoirs, the transmission of enteropathogens to humans takes a longer route than with foodborne pathways, because contaminated animal effluents need to be dispersed to agricultural fields or waterbodies. Therefore, other pathways than animal-borne and foodborne ones could be possible contributors to temperature-related enteric infections in humans. For example, ingestion of enteropathogens via contaminated water and surfaces can arise during the summer months because water shortages and high water demand could affect the quality and availability of safe water, sanitation, and hygiene.[118–120] Even though there might be fewer enteropathogens present in the environment during summertime than in other seasons with colder temperatures, temperature-related enteric infections could still occur, especially for pathogens with low infective doses (eg, *Shigella*[121]) or on a short time span or lag. Several included studies[30,58,62,75] did consider no lag or very short lags in modelling daily or weekly temperature–enteric infections association. These studies reported an increased risk of enteric infections (*Shigella, Campylobacter,* and *Salmonella*) per 1°C temperature rise at short lags, such as 2 days or during the same week.

Considering that enteric infections owing to bacterial enteropathogens could rise in the future with increased ambient temperatures, one potential adaptation is vaccines. Currently, mass vaccination for enteric infections is only available for rotavirus in young children and not all countries have introduced it. In 2016, about 28 000 deaths in young children were averted because of rotavirus vaccination,[122] and further mortality reductions should be expected in the far future if increasingly warm temperatures do reduce rotavirus incidence. Vaccines for typhoid and cholera are available but their roll-out,

particularly to low-income countries that need them, has remained stagnant.[123] There are vaccines in the pipeline for *Shigella*, enterotoxigenic *E coli*, and norovirus, but might take considerable time to be developed because enteric infections provide little incentive for manufacturers,[123] because occurrence is more common in low-income countries than in the high-income countries. Pathogen-specific risk estimates can be applied when simulating future temperature-related enteric infections to consider variations in temperature sensitivity. A process-based modelling study[18] in an Indian city combined an increased risk of diarrhoea in children from *E coli* and *Cryptosporidium* with a decreased risk of rotavirus in a future with increasingly warm temperatures, which resulted in an overall increased incidence of diarrhoea of 6·4% per 1°C temperature increase. Similarly, another study[124] applied our pathogen-specific summary estimates to the global projections of temperature-related enteric infections, which suggested considerable excess mortality from enteric infections in the future because of increasingly warm ambient temperatures. The authors of these global projections also found that lower excess mortality from temperature-related enteric infection was estimated when using pathogen-specific risk estimates than using taxa-specific and all-cause infection risk estimates.

In summary, our study found increased risks in bacterial enteric infections and decreased risks in viral enteric infections per 1°C temperature rise. We also found that the temperature sensitivity of bacterial enteric infections could differ for each enteropathogen. These pathogen-specific differences in temperature associations can help to improve modelling of the future temperature-related enteric infections and support the implementation of pathogen-specific adaptation measures like vaccinations. However, the small number of studies could limit the interpretation of our findings. Thus, we encourage future studies to investigate pathogen-specific temperature–enteric infection associations by using a similar methodological approach and datasets to determine any pathogen-specific differences.

Contributors

MH and PLCC conceptualised the study, screened the studies, and assessed the risk of bias. PLCC extracted the data, analysed it, and led the writing of the manuscript. MH, CFSN, XTS, and AT generated the risk of bias tool, verified the extracted data, and reviewed the data analysis. All authors wrote, reviewed, and approved the manuscript, had access to all the data, and were fully responsible for submitting this manuscript.

Declaration of interests

PLCC received a Japanese Government (Monbukagakusho) Scholarship from the Ministry of Education, Science, Sport, and Culture of Japan, outside of this study. MH received funding from the Japan Science and Technology Agency as part of SICORP (grant number JPMJSC20E4), outside of this study. AT received funding from the Japanese Society for the Promotion of Science Invitational Fellowships for Research in Japan (grant number S18149), outside of this study. All other authors declare no competing interests.

Data sharing

There are no original data to share because we used publicly available data summarised in the table and in the appendix.

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