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The effect of temperature on food poisoning: a time-series analysis of salmonellosis in ten European countries

R. S. KOVATS[1]*, S. J. EDWARDS[1], S. HAJAT[1], B. G. ARMSTRONG[1], K. L. EBI[2,3], B. MENNE[2] AND The Collaborating Group†

[1] *Department of Public Health and Policy, London School of Hygiene and Tropical Medicine, London, UK*
[2] *Global Change and Health, WHO European Centre for Environment and Health, Rome, Italy*
[3] *Exponent Health Group, Alexandria, VA, USA*

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SUMMARY

We investigated the relationship between environmental temperature and reported *Salmonella* infections in 10 European populations. Poisson regression adapted for time-series data was used to estimate the percentage change in the number of cases associated with a 1 °C increase in average temperature above an identified threshold value. We found, on average, a linear association between temperature and the number of reported cases of salmonellosis above a threshold of 6 °C. The relationships were very similar in The Netherlands, England and Wales, Switzerland, Spain and the Czech Republic. The greatest effect was apparent for temperature 1 week before the onset of illness. The strongest associations were observed in adults in the 15–64 years age group and infection with *Salmonella* Enteritidis (a serotype of *Salmonella*). Our findings indicate that higher temperatures around the time of consumption are important and reinforce the need for further education on food-handling behaviour.

INTRODUCTION

Salmonella is one of the most important foodborne pathogens affecting European populations. For example, *Salmonella* infection causes more deaths annually than any other foodborne pathogen in England and Wales [1]. Within Europe, *Salmonella* sp. accounts for 71% of all laboratory-confirmed outbreaks of foodborne disease [2]. Outbreak investigations indicate that 'temperature misuse' was a contributory factor in 32%, and of these, inappropriate storage and preparation too far in advance were responsible for 25% [2]. Although the effect of temperature on the growth of salmonellas in food is well understood [3, 4], the links between ambient air temperature and the transmission of sporadic salmonellosis are yet to be fully elucidated.

There are over 2500 different serotypes of *Salmonella*, but the two most commonly reported, *S.* Typhimurium and *S.* Enteritidis, together account for at least 70% of reported human infections in Europe [2, 5]. *S.* Enteritidis is found almost exclusively in poultry and eggs. There are statutory or voluntary surveillance systems for salmonellosis in all European countries [2]. The worldwide reported incidence of salmonellas in humans increased steadily during the 1980s and early 1990s due to the emergence and rapid spread of *S.* Enteritidis which overtook *S.* Typhimurium as the dominant *Salmonella* serotype in many industrialized countries [6]. However, recently many countries (including the countries in this study) have observed marked declines in the reporting of salmonellosis coincident with falls in *S.* Enteritidis

* Author for correspondence: R. S. Kovats, Department of Public Health and Policy, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK.
† The members of the Collaborating Group are listed in the Appendix.

Table 1. *Description of surveillance and meteorological data (ordered by increasing mean summer temperature)*

Country	Unit of analysis	Time-period of data series	Cases associated with travel	Cases associated with outbreaks	Average annual total of cases	Stations used in national temperature series	Mean summer temperature (June to August) (°C)
Poland	2 weeks	2000–2002	Excluded	Included	21 159	Gdansk, Poznan, Warsaw, Krakow (4)	12·66
Scotland	Week	1990–1997	Excluded	Excluded	2108	Edinburgh, Paisley, Perth, Aberdeen (4)	14·24
Denmark	Week	1991–2001	Excluded	Included	2657	Odense, Alborg, Copenhagen (3)	15·94
England & Wales	Week	1989–1997	Excluded	Excluded	24 346	Central England Temperature (CET) (4)*	16·07
Estonia	Month	1990–2001	Included	Included	840	Tallin, Tartu, Paernu (3)	16·31
The Netherlands	Week	1984–2001	Excluded	Included	3285	De Bilt (1)	16·76
Czech Republic	Week	1993–2001	Included	Included	40 970	Prague, Cheb, Usti Nad Orlici (3)	17·48
Switzerland	Week	1990–2000	Included	Included	4632	Geneva, Bern, Zurich (3)	18·15
Slovak Republic	Month	1983–2000	Included	Included	11 816	Bratislava, Zilina, Poprad, Kosice (4)	18·34
Spain	Week	1994–2000	Included	Included	5238	Madrid (1)	24·24

* CET is weighted mean average of Squires Gate, Lancashire; Manchester Airport; Malvern, Worcestershire; and Rothamsted, Herts [17].

infection, following the introduction of various interventions to control the carriage of *S.* Enteritidis in poultry flocks.

In the laboratory, the rate of multiplication of *Salmonella* sp. is directly related to temperature within the range 7·5–37 °C [7]. Thus, in the absence of other controls, ambient (outdoor) temperature might be expected to influence the reproduction of salmonellas at various points along the food chain from farm to fork [8]. Cooking destroys salmonellas. Inadequate storage and the spread from contaminated to non-contaminated food are risk factors for transmission in sporadic cases [9–11]. Outdoor temperatures might also affect the exposure of individuals to salmonellas through seasonal changes in eating patterns (e.g. consumption of foods from buffets, barbecued foods, and salads, etc.) and behaviour (e.g. outdoor recreational activities such as swimming or hiking that increase contact with sources of *Salmonella* in the environment).

Few studies have looked at environmental temperature and *Salmonella* or foodborne infections generally [12, 13]. Year to year variability in summer temperatures might explain some of the variability in annual incidence of *Salmonella* infection in the United Kingdom (1962–1989) [14]. Monthly variation in food-poisoning notifications in England and Wales (clinical diagnoses that include a range of infectious and non-infectious diseases) was found to be positively related to outdoor temperatures in the previous month, but only at temperatures above 7·5 °C [12, 15]. Further, these relationships were used to estimate future additional cases of food poisoning in a warmer England due to global climate change [16].

We aim to determine how much of the variation in weekly *Salmonella* cases is explained by environmental temperatures using laboratory-confirmed cases of salmonellosis from passive surveillance in 10 European populations.

METHODS

Surveillance data

Data on laboratory-confirmed cases of *Salmonella* infection were obtained from national surveillance centres in the Czech Republic, Denmark, England and Wales, Estonia, The Netherlands, Scotland, Slovak Republic, Poland, Switzerland, and Spain (Table 1) [2]. We analysed weekly counts, except where these were not available for Poland

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Table 2. *Estimated average time difference between illness onset and date provided with surveillance data* (*estimated by data providers*)

Country	Date supplied in data-set	Estimated average delay in reporting system from illness to reported date
Poland*	Date specimen enters system after typing	3 days
Scotland	Date specimen enters system after typing	16 days
Denmark	'date specimen arrives in laboratory'	8 days
England & Wales	'date specimen arrives in laboratory'	5 days
Estonia†	Date sample entered recording system	5 days
The Netherlands	Laboratory test confirmed	12–16 days
Czech Republic	Date of onset of illness	0
Switzerland	Date sample entered recording system	10 days
Slovak Republic†	Date of onset of illness	0
Spain	Laboratory test confirmed	30 days

* Data supplied at the bi-weekly level.
† Data supplied at the monthly level.

(bi-weekly) and for Estonia and the Slovak Republic (monthly). Travel-associated cases were excluded where possible (5/10 countries), as infection acquired abroad is not likely to be associated with local temperatures. Cases linked to outbreaks were also excluded where possible (2/10 countries), as the relationship with temperature may be different for sporadic cases and for those linked to outbreaks. The ascertainment of travel- and outbreak-associated cases is likely to be incomplete even in those countries that identify such cases.

Changes in infection control measures in the UK poultry industry caused a significant decline in the number of reported cases in Scotland and England & Wales after 1997. For this reason, the period after January 1998 was dropped in these series. Our collaborators reported no significant changes in disease epidemiology or control during the data period with the exception of The Netherlands where control measures were put in place during 1997 and 1999 (W. van Pelt, personal communication). Abrupt changes in disease, however, were not apparent in the data and therefore the whole time-period was used. No countries reported significant changes in reporting practice.

The following age groups were also modelled where data were available: young children (0–5 years), children (6–14 years); adults (15–64 years); and the elderly (65+ years). For comparability, age-group series were analysed using the same thresholds as identified for the all-ages series for that country. The serotypes of *Salmonella* sp. were also modelled separately for Denmark, The Netherlands, England and Wales, and Scotland, as they were identifiable in the data-sets and had sufficient numbers. Threshold values for the different serotypes were estimated.

Ideally, time-series analysis would use dates of the onset of illness but this information is not routinely available in surveillance. The onset of an illness in sporadic cases is self-reported. Information was obtained from questionnaires completed by all data providers regarding the definition of date and estimated delay between illness onset and the date recorded in the data-sets (Table 2). The relationship between date of onset of illness ('onset date') and date specimen arrives at the laboratory ('specimen date') was also investigated using data from England and Wales, where both dates were recorded in a subset of records (11·96%). In these data, we found a mean delay between onset date and specimen date of 5·53 days (95% CI 5·45–5·61) for *Salmonella* sp. (all types) with over 78% of the specimen dates within 7 days of the onset date. We concluded that reporting date was a reasonable indicator of onset date, with an approximate 1-week delay. For other populations the average delay was estimated by our collaborators (Table 2).

Meteorological data

All countries in this study, with the exception of Spain, have a relatively homogenous climate and a single national temperature series was assumed to represent temporal variability for all the population.

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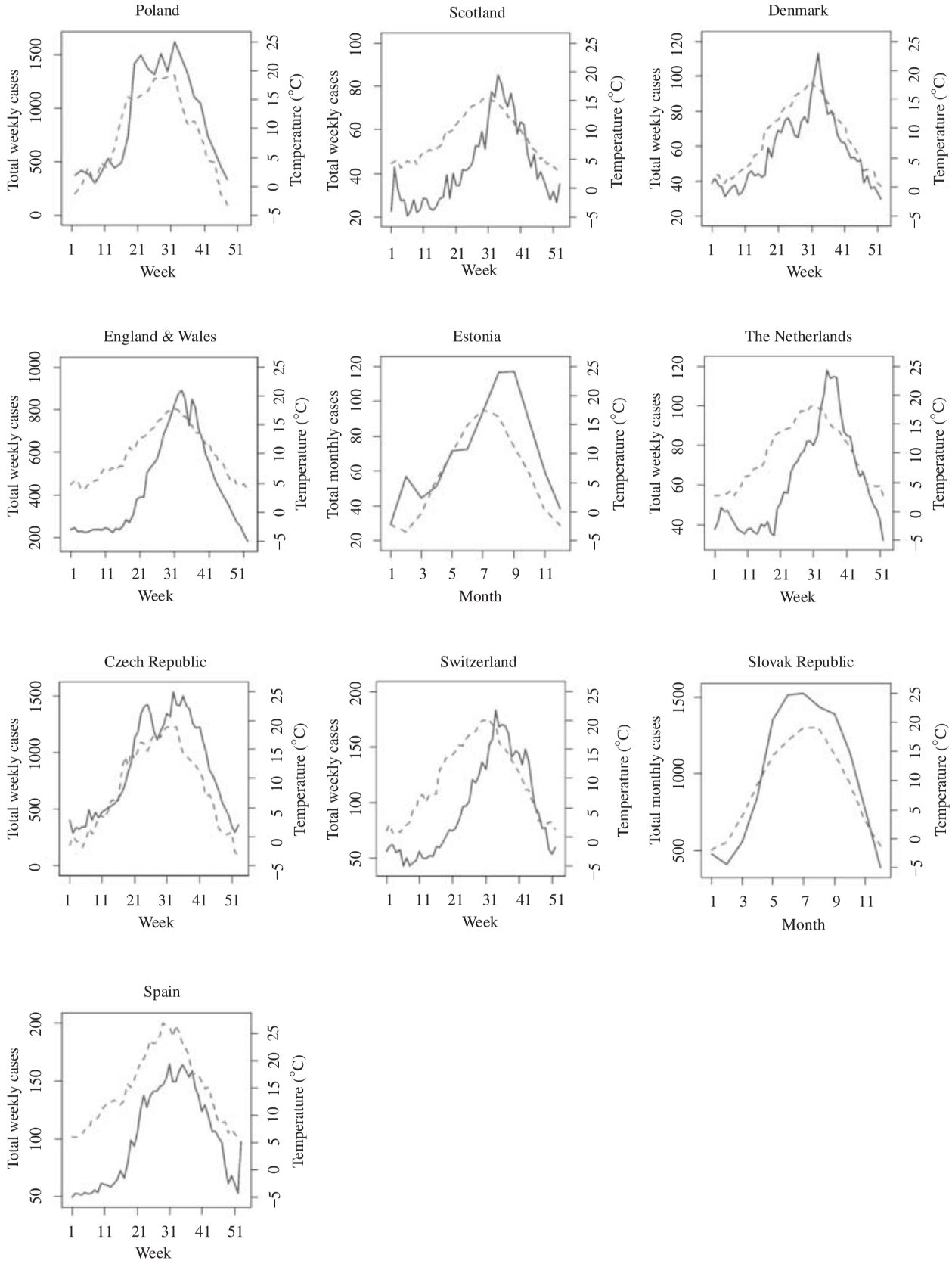

Fig. 1. Seasonal patterns of reported cases of infection with *Salmonella*, in order of country by mean summer temperature. - - - -, average temperature (by week or month). ——, no. of cases (by week or month).

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For England and Wales, the Central England Temperature (CET) series was used which is a weighted mean temperature indicator for central England [17]. For other countries, national series were constructed using daily temperature data from 3–4 weather climate stations obtained from the German meteorological office archive (Deustcher Wetterdienst) (Table 1). The new national series were validated against an independent national (monthly) data series [18].

Statistical methods

The analytical approach used Poisson regression models adapted for time-series data, originally developed for air-pollution studies [19]. These techniques allow us to assess any short-term effects of temperature on disease. Inter-annual variation was controlled for in all regression models by adding indicator variables for each year of the series. Fourier terms (up to the sixth harmonic) were added to each model to control for annually repeated patterns other than those related to temperature.

Indicator variables were used to control for the effect of public holidays (typically rates were low during holidays, and high following them). Previous analyses at the regional level in England (results not shown) indicated that relative humidity and other meteorological variables had no effect on *Salmonella* cases, so these were not included in the model.

We modelled the effect of temperature on the weekly count in two ways. First, to explore the shape of the relationship, we fitted and graphed a natural cubic spline of weekly temperature with 1 D.F. for every 5 °C of the temperature range. Secondly, in order to quantify the relationship, we fitted a 'hockey-stick' model under which it was assumed that there is no effect of temperature until a threshold value is reached, after which the relationship was assumed to be linear. The temperature threshold for each country was estimated by maximum likelihood from among thresholds across all integer values of the temperature measure. Likelihood-profile confidence intervals were calculated from these arrays of likelihood, scaled to allow for overdispersion, if present. The best single threshold common to countries was then also estimated by maximum likelihood. In the final model for most countries it was observed that the number of disease cases in any given week was strongly correlated to the levels of the preceding week. A first-order autoregressive term was therefore included in models to ensure statistical inference respected this feature of our data [20].

Exploratory analyses indicated that the delay between high temperature and increased case counts, where present, was not more than 9 weeks. Therefore, the temperature measure used in our standard model was an average value of lags 0–9 weeks; this provides the combined effect of temperature from the previous 2 months on disease. We also investigated the effect of individual lags of weekly temperature entered simultaneously into the model.

The population attributable fraction (PAF) of cases of salmonellosis due to temperatures above the identified threshold was calculated for each country [21]. All analyses were conducted in Stata 7.0 [22].

RESULTS

Figure 1 illustrates the seasonal patterns of infection with salmonellosis. Most countries in our study show a peak in the late summer months, after the peak in temperatures. The Czech Republic, Poland and the Slovak Republic also show an early summer peak in infections.

Figure 2 describe the fitted relationship between *Salmonella* cases (all types) and temperature (average of 0–9 weeks preceding case) for each country. The centre line is the estimated spline curve, and the upper and lower lines represent the 95% upper and lower confidence limits respectively. For most countries, the relationship is approximately linear above a threshold temperature, or simply linear. For the Slovak Republic and Denmark, however, there was no clear association of case occurrence and temperature.

Estimated thresholds and the per cent increase in cases for each °C above the threshold value are shown in Table 3. Thresholds vary substantially between countries, usually with wide confidence intervals. For Denmark and the Slovak Republic there is no evidence for a threshold. For other countries there was evidence for a threshold, although for four countries the confidence intervals indicated compatibility with a linear as well as a threshold model. Slopes of above-threshold relationships vary considerably, with some imprecisely estimated. Further, these slopes are very dependent on the threshold, and the consequent additional uncertainty is not reflected in the confidence interval. There is no relation between the observed thresholds and the mean summer temperature of each country.

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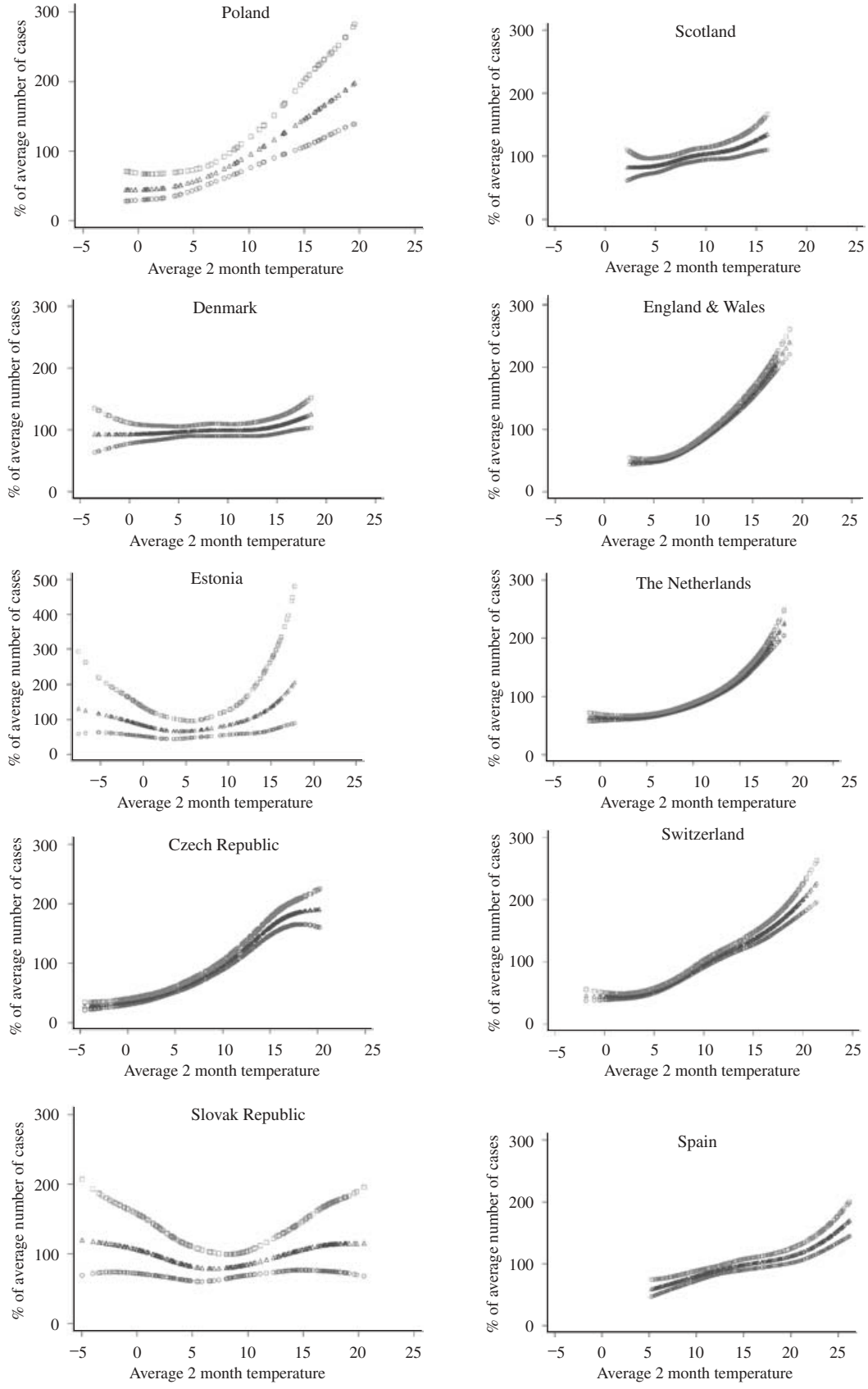

Fig. 2. For legend see opposite page.

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Table 3. *Thresholds and slopes estimated by country*

Country	Temp. range (°C) (9-week average)	Country-specific threshold (95% CI)* (°C)	% change per °C above country threshold (95% CI)	% change per °C above common overall threshold (6 °C) (95% CI)	Population attributable fraction (%) (95% CI)†
Poland	−1 to 18	6 (•–7)	8·7 (4·7–12·9)	8·7 (4·7–12·9)	33·8 (20·2–45·1)
Scotland	3 to 16	3 (•–12)	4·7 (2·1–7·3)	5·0 (2·2–7·9)	15·2 (7·06–22·58)
Denmark	−3 to 18	15 (•–•)	1·1 (−2·7–5·0)	0·3 (−1·1–1·8)	1·3 (•–6·5)
England & Wales	3 to 18	5 (5–6)	12·4 (11·6–13·3)	12·5 (11·6–13·4)	41·3 (38·6–42·7)
Estonia	−7 to 17	13 (3–14)	18·3 (3·6–35·1)	9·2 (−0·9–20·2)	27·4 (•–48·0)
The Netherlands	−1 to 19	7 (7–8)	9·3 (8·5–10·1)	8·8 (8·0–9·5)	32·6 (30·3–34·8)
Czech Republic	−7 to 20	−2 (−6 to −1)	9·5 (8·2–10·7)	9·2 (7·8–10·7)	29·1 (37·4–33·4)
Switzerland	−1 to 21	3 (•–3)	8·8 (7·6–9·9)	9·1 (7·9–10·4)	35·5 (31·7–39·1)
Slovak Republic	−4 to 20	6 (•–•)	2·5 (−2·6–7·8)	2·5 (−2·6–7·8)	11·5 (•–31·3)
Spain	6 to 25	6 (•–8)	4·9 (3·4–6·4)	4·9 (3·4–6·4)	35·1 (26·3–42·8)

* A blank lower or upper confidence limit (denoted by •) indicates that no limit was found within the range of the data, which are thus compatible with a linear no-threshold relationship.

† A blank lower confidence limit (denoted by •) indicates that the relationship of *Salmonella* with disease was not significant ($P>0·05$), so a zero population attributable fraction is compatible with the data.

Because thresholds did not follow a clear pattern, and to avoid complexities in comparing relationships with both threshold and slope varying, we re-estimated slopes with a common threshold of 6 °C for all countries (as estimated by maximum likelihood) (Table 3). At this threshold, England and Wales has the steepest slope (12·5%), with several other countries with similar estimates. For Denmark, the Slovak Republic, and Estonia, the slope was not statistically significantly greater than zero ($P>0·05$). The slope for England and Wales using the whole data series (1989–1999) was reduced to 12·0% (95% CI 11·2–12·8). It was not possible to investigate effects for the years after 1997 separately due to insufficient data.

Figure 3 illustrates the per cent change in cases associated with temperature measured on each separate week before the onset of illness, up to a lag period of 9 weeks, in England and Wales (all lags included in the model simultaneously). The greatest effect of temperature is 1 week before the onset of illness, with diminishing but positive effects up to 5 weeks. Data from other countries using estimated date of onset show broadly similar patterns, but positive effects persisted longer, possibly reflecting imprecision in the estimated onset date.

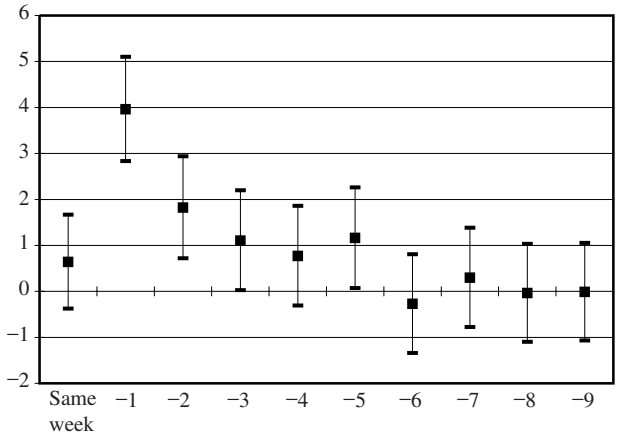

Fig. 3. The effect of temperature for each week lag between onset of illness and temperature exposure (% change), for temperatures above threshold (5 °C) in England and Wales.

Age-specific analyses were undertaken in England and Wales, Scotland, The Netherlands, Denmark, and Switzerland, assuming the country-specific thresholds. The adult age group (15–64 years) appears to be the most sensitive to temperature effects on the incidence of salmonellosis. The differences between the age groups are not statistically significant ($P>0·05$), except for England and Wales.

Fig. 2. Temperature–salmonellosis relationships by country (full model adjusted for season, trend and holidays), with temperature (°C) on the *x*-axis (0- to 9-week average), and salmonellosis cases on the *y*-axis as represented by percentage of the average number of cases. The centre line is the estimate. Upper and lower lines are the 95% confidence intervals.

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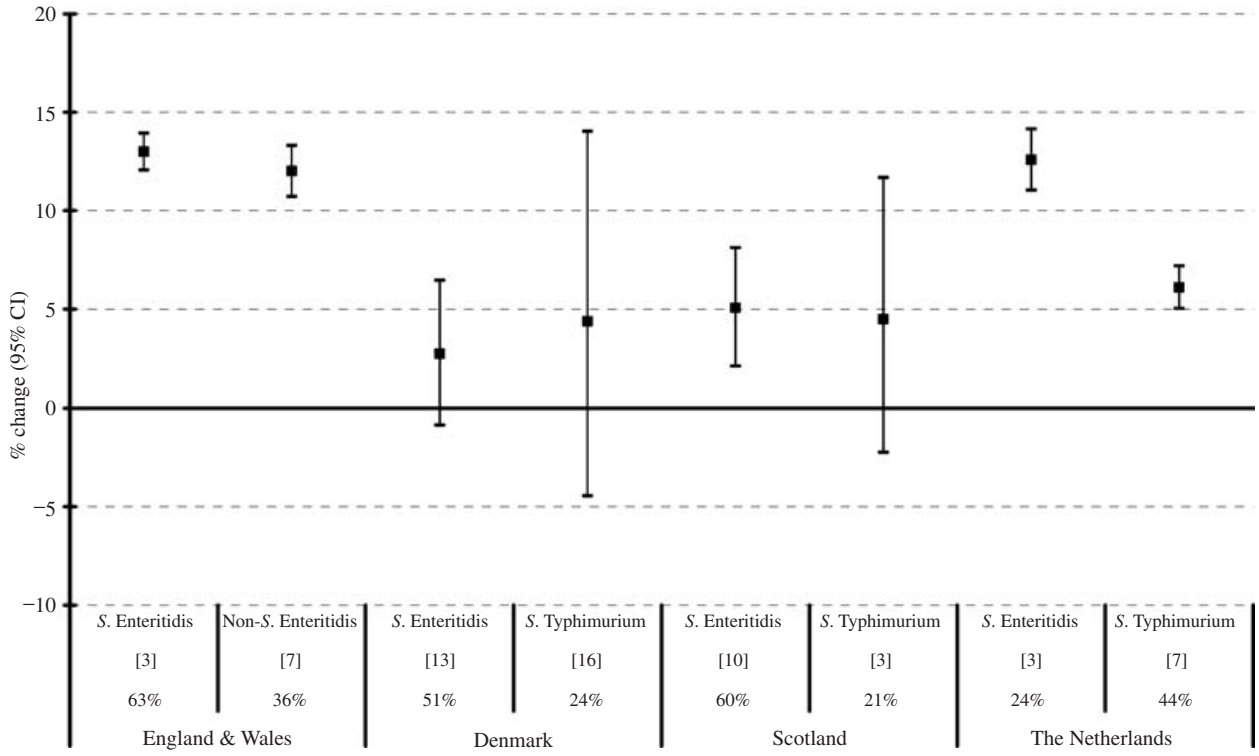

Fig. 4. The effect of temperature by serotype (% change), for temperatures above threshold [values within square brackets] for *Salmonella* in four populations. The proportion of each type in the total reported cases of salmonellosis in the data is shown as a percentage.

In countries with the information to distinguish *Salmonella* serotype, infection with *S.* Enteritidis appears to be more sensitive to the effects of environmental temperature than infection with *S.* Typhimurium (Fig. 4). In The Netherlands, the difference was statistically significant [increase in cases per °C increase in temperature: *S.* Enteritidis 12·6% (95% CI 11·1–14·2); *S.* Typhimurium 6·1% (95% CI 5·0–7·2)]. If the same threshold is assumed within each country, the estimates are largely unchanged. However, the difference in England and Wales becomes statistically significant [increase in cases per °C increase in temperature: *S.* Enteritidis 13·1% (95% CI 12·2–14·1); non-*S.* Enteritidis 10·6% (95% CI 9·4–11·8)].

DISCUSSION

This first international study of the association between environmental temperature and cases of *Salmonella* sp. infection shows clear relationships in many European countries. Details of relationships (threshold and slope) differ between countries and do not follow an obvious pattern, such as by latitude or mean summer temperature. There is no indication that a population's food hygiene behaviour is adapted to their climate in the sense that effects occur at higher threshold temperatures in warmer climates. For many countries, a threshold is not apparent and the relationship is approximately linear over the whole temperature range. Thresholds were not apparent for the effect of temperature on salmonellosis in five Australian cities, although similar slopes were estimated (5–10% per °C increase in temperature) [13].

The absence of a relationship in Denmark is remarkable. Although the seasonal pattern of cases is similar to other countries, an effect of temperature is not significant in the fully adjusted model. The distribution of serotypes in Denmark is similar to other countries. The main sources of salmonellosis are estimated in Denmark every year but this information is not available for most other countries. We have no reason to believe that certain major sources of infections are unique to Denmark. It has been observed by the Danish diagnostic laboratories that the incidence of salmonellosis increases during and after heat-waves (P. Gerner-Smit, personal communication).

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Delay between high temperatures and onset of disease (lag) could only be studied directly in England and Wales, where we found it to peak at 1 week, but persist up to 5 weeks. A previous study of food-poisoning notification data in England and Wales found a longer lag effect (2–5 weeks) between the temperature exposure and the reported onset of disease [12]. However, the data-set used (GP notifications based on clinical diagnosis) would be expected to have included a range of pathogens other than *Salmonella* sp., most importantly campylobacter [23] that has a less clear relationship with short-term temperature variability.

There are limitations in the use of national passive surveillance data. First, not all cases in the community are represented in national surveillance data and the degree of under-reporting varies by country [24, 25] and by pathogen. However, except where changes to surveillance systems are described above, there is no reason to believe that the degree of under-reporting has varied over time. Secondly, cases reported to national surveillance are not necessarily representative of all cases [26]. However, our collaborators considered it unlikely that there were important differences in reporting during the year (e.g. cases of salmonellosis are not more likely to be reported and detected in a laboratory during hot weeks). Thus the undoubted, substantial under-ascertainment in these surveillance series would not have been expected to caused bias.

In half the countries it was not possible to exclude cases where infection was acquired abroad, however, these were, in general, expected to be only a small proportion of all cases. The adjustment for season in the model would remove the effect to the extent to which foreign travel is a regular seasonal occurrence. Further, analysis of temperature effects with and without travel cases in those countries providing these data did not substantially affect the results (not shown). It is not possible to identify cases where infection was associated with imported food, although this is thought to be an increasing source of salmonellosis for many countries [4].

The differences between the age groups, although statistically significant only for England and Wales, are largely consistent across countries. It can be assumed that adults prepare food for children (who have the highest incidence of salmonellosis) and possibly the elderly. We suggest that food hygiene behaviour or food vehicles relevant to the effects of temperature on disease may be different in adults that live alone. Similarly, evidence for greater sensitivity to temperature of infection with *S.* Enteriditis compared to *S.* Typhimurium is clear in The Netherlands and suggested in England and Wales. Infection with *S.* Typhimurium is more common in rural areas and can be obtained through non-food contact (in the environment). *S.* Enteritidis is more strictly related to transmission via food. This further supports the hypothesis that temperature effects are more strongly mediated through the activities related to food preparation (and particularly egg-handling behaviour) rather than other non-food sources.

These results suggest that temperature influences transmission of infection in about 35% of all cases of salmonellosis in England and Wales, Poland, The Netherlands, Czech Republic, Switzerland, and Spain (Table 3), assuming that the relationships described here with reported cases are applicable to all cases in the community. This has implications for programmes and strategies to reduce foodborne disease. The main mechanisms for this increased risk of *Salmonella* infections with higher outdoor temperature cannot be estimated using these methods without further information on cases from routine surveillance. It may be a hitherto unidentified direct mechanism or it may be an indirect mechanism caused by altered eating habits during hot weather, e.g. barbecuing and eating more dishes containing raw or insufficiently heat-treated food during the summer. Average temperatures are increasing due to global climate change, and more weeks with above-threshold temperatures will occur. It is likely that temperature–salmonellosis relationships may change in the future, particularly as the contribution of *S.* Enteritidis decreases due to active control measures. Although the underlying trend in *Salmonella* infections is decreasing, due to active control measures, strategies are needed to combat the proportion of salmonellosis attributable to climate.

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APPENDIX. The Collaborating Group

J. Cowden (Scottish Centre for Infection and Environmental Health, Scotland), P. Gerner-Smidt (Statens Serum Institut, Denmark), G. Hernández Pezzi (Instituto de Salud Carlos III, Spain), Z. Kristufkova (State Health Institute of Slovak Republic, Slovak Republic), B. Kriz (National Institute of Public Health, Czech Republic), K. Kutsar (Health Protection Inspectorate, Estonia), W. Magdzik (National Institute of Hygiene, Poland), S. J. O'Brien (PHLS Communicable Disease Surveillance Centre, UK), H. Schmid (Federal Office of Public Health, Switzerland), W. van Pelt (National Institute of Public Health and the Environment, The Netherlands).

The Collaborating Group prepared and provided the data, provided expert advice, and also commented on drafts of this paper.

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