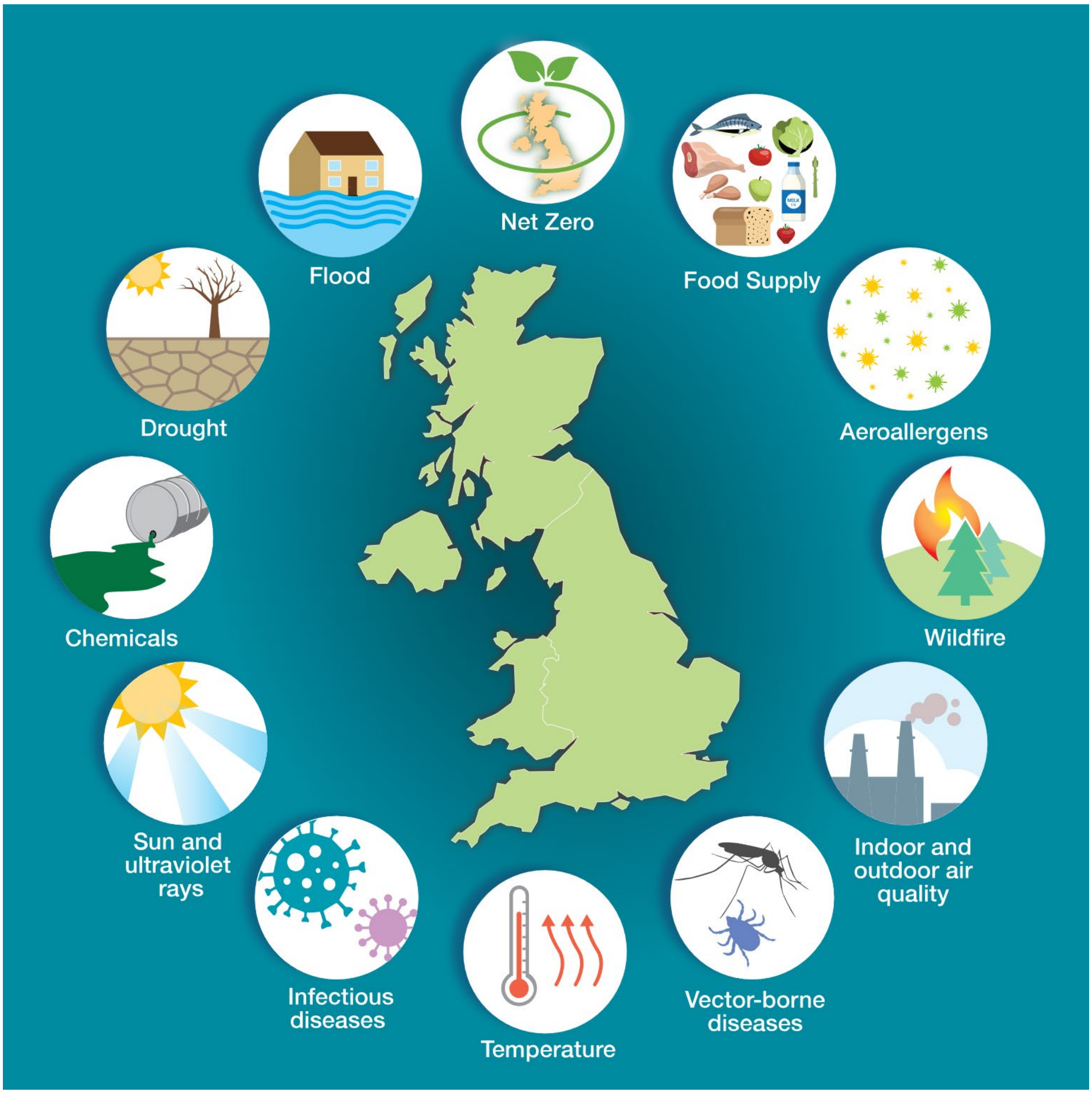
UK Health Security Agency

Health Effects of Climate Change (HECC) in the UK: 2023 report

Chapter 7. Effect of climate change on infectious diseases in the UK



Summary

Many infectious diseases are climate sensitive. Chapter 7 presents a scoping literature review of evidence on the impacts of weather and climate on foodborne, waterborne and respiratory infectious pathogens of public health importance in the UK. Vector-borne diseases are considered separately in Chapter 8. This chapter was jointly led by scientists at the UK Health Security Agency (UKHSA), University of East Anglia, University of Surrey and University of Exeter. Authors complement UK-based studies with relevant evidence from other countries where UK-specific evidence is limited.

The effect of climatic factors on infectious disease is complex with multiple interactions. Climate affects pathogen abundance, survival and virulence and it also impacts human behaviour and host susceptibility. Warmer temperatures can alter geographical distribution of infectious diseases and extend the transmission periods of some diseases.

The specific impacts that climate change will have on infectious diseases are uncertain and dependent on sociodemographic factors, human behaviour, the potential of emerging and re-emerging diseases, antimicrobial resistance and the adoptions and impacts from mitigation and adaptation measures. The authors find that the relationships between weather and incidence of disease are well-established for *Salmonella*, *Campylobacter*, and *Vibrio* spp., a group of foodborne and waterborne bacteria that lead to acute gastrointestinal illness, and there is evidence that risk of disease from these pathogens could increase in future. In contrast, and despite influenza incidence demonstrating seasonality, the impact of climate change is expected to be relatively minor as factors such as human behaviour and population immunity are more dominant drivers of incidence. Finally, there is insufficient evidence to assess UK climate impacts for a number of pathogens, including astroviruses, sapoviruses and noroviruses.

Although there is strong evidence that climate change can affect the risk of infectious diseases, better evidence is needed to quantify the magnitude and impacts of these changes. The chapter highlights 4 key insights for public health. First, as many emerging infectious diseases are zoonotic (meaning that they are transmitted between animals and people), increased attention should be given to the ways in which people interact with wild and domestic animals and animal products, and how this can affect changing disease risks. Strong collaborations are needed with other national and international public health agencies, cross-governmental agencies and those delivering care (such as the NHS), as well as farmers, food manufacturers and the public. Second, early detection is important in responding to infectious disease outbreaks, integrating a range of surveillance mechanisms such as sentinel and routine surveillance, epidemic intelligence and genomic data in a One Health approach. Global surveillance also needs to be strengthened so that emerging diseases can be detected rapidly and controlled where possible at source. Third, climate change is an important context within which we will need to protect ourselves from infectious disease threats. Changing climatic conditions will need to be taken into consideration when undertaking risk assessments and developing public health policy and

guidelines to prevent and control infectious diseases. Finally, the impact of climate change on future pathogen risk should be considered when prioritising development of new vaccines for the UK population.

This chapter highlights several research gaps and priorities, including the need to:

- improve our understanding of the magnitude and impacts of climate change on infectious diseases including advancing evidence attributing the burden of infectious diseases to specific weather and climate factors
- improve data analytics and platforms to integrate infectious disease and weather data at appropriate resolutions
- improve integration of human case data with that from other organisations, such as linking human cases to outbreaks in animals, detection of pathogens in food systems and environmental health to strengthen the One Health approach
- characterise thresholds determining the relationships between infectious disease and weather variables, including the interactions and combination of multiple weather variables
- assess the relative individual and compounding effects of climate change and weather variability on the emergence, establishment and spread of infectious diseases, including land-use, demographic, and socio-economic drivers
- quantify baseline burdens of infection to understand, predict, track and prevent the future impact of climate change
- project future infectious disease risk where feasible using a combination of different warming and adaptation scenarios

UKHSA is working with stakeholders and academic partners as part of the NIHR Health Protection Research Unit in Environmental Change and Health to improve the evidence base on the connection between weather and infectious diseases in England and Wales.

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Chapter 7. Effect of climate change on infectious diseases in the UK

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1. Introduction

The recognition that infectious diseases can potentially increase with changes in the climate dates back to the 1990s (1, 2). Climate change is and will continue to affect infectious diseases through diverse and often overlapping ecological, biological and social mechanisms. The effect of climatic factors on infectious disease is complex. Climate influences ecological factors, such as the natural environment in which infectious agents live, grow and multiply. Climatic and ecological factors will affect organisms' biology (for example, survival, proliferation, serotype fitness), which may lead to altered pathogen geographical ranges and distributions or the seasonality of individual pathogens. For example, there may be longer transmission periods associated with warmer conditions, although colder conditions drive people indoors, which can also increase the transmission of some respiratory infections during winter. In addition, the emergence of novel species may be influenced. Climate change may also affect transmission through impacts on key vectors, such as rodents, changes of impacts on farming and food production practices or important pathogen transport mechanisms, such as rainfall and wind contributing to the dispersal of soil organisms and pathogens. Finally, climate change may affect human behaviour and other social determinants of infectious disease risk: for example, warmer weather resulting in changed food preparation and consumption patterns, or increasing countryside visits, in turn increasing the potential exposure to infectious disease hazards (3). In England, the economic burden from infectious diseases (including costs to the health service, labour market and to individuals) is estimated at £30 billion annually, dominated by respiratory and gastrointestinal infections (4). Hence any changes to disease burdens will not only have public health implications, but also economic impacts.

Changing human behaviour and policy will play an important role in infectious disease risk, both by mitigating risk or creating new risks. For example, climate change-induced increases in foodborne disease outbreaks may lead to the development of new regulations that may in turn help to minimise health burden. Alternatively, efforts to decarbonise will have important implications for land use and agriculture (see Chapter 14), which could even increase health risks if new vector habitats (see Chapter 8 for vector-borne diseases) or transmission pathways are created (5). In future, changes to health associated with climate change in the UK will occur concurrently with 2 important trends which may influence disease risk. First, the UK population is ageing: by 2066, there are likely to be an additional 8.6 million residents aged 65 years and over, comprising 26% of the total population, with the over 85 years age group showing the fastest increase (6). The rising UK elderly population will make the population more susceptible to infections, due to lowered immunity or more frequent interactions with healthcare or living in communal settings, where for example, the potential for person-to-person transmission is elevated. Second, antimicrobial resistance (AMR) poses a growing threat to human, animal and environmental health, implying that future impacts may be greater, with 10 million deaths globally predicted to occur annually by 2050 due to AMR (7). There is emerging evidence suggesting that AMR may be influenced by climate change: warming temperatures may accelerate bacterial growth, increase bacterial infection rates, increase the frequency of infections in healthcare settings and expand geographical distributions (8 to 12). These processes increase the likelihood of horizontal gene transfer and thus the emergence of drug-resistant infections. However, the incidence of respiratory infections is higher during winter, resulting in greater antimicrobial use for treatment, and increasing the risk of drug-resistant infections developing as a result. In addition, wastewater is a well-known reservoir for antibiotic-resistance genes, as bacteria can encounter antimicrobial effluent and develop antimicrobial resistance genes (13, 14). More frequent and intense precipitation and flooding events in future will likely lead to increased agricultural runoff and pollutants in water, causing bacterial blooms and further opportunities for antibiotic resistant gene transfer (14, 15). Though details on AMR and effects on specific pathogenic species are out of the scope of this chapter, they are important to note as an emerging climate related health risk.

Infectious diseases have been considered in the ‘Health Effects of Climate Change in the UK (HECC)’ reports since 2002 (16), when infectious disease was mostly covered within the ‘Food poisoning and climate change’ chapter. This chapter highlighted parts of food chains where weather may affect the risk of food poisoning (an umbrella term which includes different foodborne illnesses). In another chapter, the potential for changing ultraviolet light (UV-B) levels to affect immune suppression and virus activation were discussed, and the report briefly considered adaptation, highlighting the largely unknown capacity of the population to adapt to a changing climate. Indirect influences of climate on infectious disease, such as dietary choice and human behaviour, were briefly mentioned. Integrated assessment models were highlighted as suitable for modelling the impacts of climate change on infectious diseases. In the 2008 report, ‘food poisoning’ was separated into constituent illnesses, predominantly *Salmonella* and *Campylobacter* (17). The flooding chapter included infectious disease risks, and UK consequences of climate change impacts on global food prices was also discussed.

By 2012, a wider range of infectious diseases and transmission routes were considered (18). The water and food chapters were merged, and there was greater emphasis on food as a global commodity. The impact of the built environment upon the airborne transmission of infectious diseases was explicitly addressed for the first time, and concerns about UV-B levels affecting immune suppression remained. The scarcity of data on how climate change may influence human behaviour, and hence impact disease incidence, was noted. Adaptation and resilience of the food sector to changing diseases were explicitly considered. The importance of food as a contributor to greenhouse gas (GHG) emissions was included, alongside the imperative of ensuring that food associated GHG mitigation measures do not adversely affect food safety. It highlighted the importance of dietary choice for GHG emissions and noted win-wins from dietary changes (for instance, lower animal product consumption reduces both GHG emissions and saturated fat intake). The report recommended that all climate change mitigation policies be subject to health impact assessment.

1.1 Chapter scope and methodology

This chapter focuses on health impacts affecting the UK population (excluding vector-borne diseases, which are covered in Chapter 8), focusing specifically upon infectious diseases acquired within the UK. This chapter focuses on the direct impacts of climate change, such as the influence of increasing temperatures upon *Salmonella* infections, as opposed to indirect consequences. It should be noted that some potentially important consequences of climate change, for instance infections derived from the influence of climate change upon economic growth, changes to diseases acquired abroad, or infections from migrating populations are not considered within this chapter. There are likely to be significant impacts of climate change upon non-UK populations which will have spill-over effects on the UK population, but these are outside of the scope of this chapter. The impact of climate change on pandemics has also not been considered in the chapter: whilst there is historical evidence of climate influences on major epidemics (19), systematic assessments of pandemic risks associated with climate change are lacking.

This chapter was informed by an extensive scoping review of publications on climate change (78,690 PubMed papers screened to the end of 2022). From this, 6,874 references focussed on infectious diseases were identified, and those publications relevant to the specific individual pathogens included within this chapter were reviewed. Focus was given to studies conducted in the UK; findings from research conducted in other high-income countries (such as those in Western Europe, as well as the USA, Canada and Australia) are also described in the absence of UK studies. Analytical studies and mathematical models relevant to climate and infection were the focus, but descriptive studies were also examined. Pathogen-specific searches were also conducted to account for inconsistency in vocabulary and indexing, with many relevant papers not using the term ‘climate change’. Snowballing of references was also carried out on relevant articles.

Table 1 comprises of a range of climate-sensitive infectious diseases assessed to be currently or likely to become of public health concern in the UK and are the focus of this chapter. The assessment was informed by previous HECC reports, a recent EEA report on climate change and infectious diseases (20), the ‘Sixth Assessment Report from the Intergovernmental Panel on Climate Change (IPCC)’ (21), the ‘Third UK Climate Risk Independent Assessment (CCRA3)’ (22), the most up-to-date study on infectious intestinal diseases in the community in the UK (23) and expert opinion in Europe (24, 25). In Table 1, infectious diseases are classified into those that are viral, bacterial or parasitic. The table also presents the main symptoms, the modes of transmission, as well as an estimate of the economic costs of each infectious disease to the UK. Studies of COVID-19 were not included in this chapter. Although there have been a number of papers published since 2020 investigating the impact between weather and COVID-19, the evidence remains nascent regarding the impact of climate change on COVID-19, and COVID-19 transmission patterns have not sufficiently stabilised post-pandemic to allow robust assessment. Organisms with similar pathways to other waterborne infections that pose a public health threat and are not covered elsewhere in the report, including for example biotoxins from harmful algal blooms are also included here. As the COVID-19 pandemic impacted on the ascertainment of some infections in the UK, pre-pandemic data detailing infection rates are reported in the chapter.

Table 1. Transmission mode, total cost and symptoms and severity of infectious diseases in the UK (excluding vector-borne diseases) considered in this chapter. Definitions of terms used in column 3 are given below the table.

Type of infection	Disease or pathogen	Transmission mode	Total cost (median £million, 95% CIs) in 2018 (26)	Symptoms and severity
Virus	Adenovirus	Contact, bathing, airborne, surface	£48.7 (£12.0 to £138.2)	Causes fever, upper respiratory infections, conjunctivitis, swollen glands and sore throat; species F causes gastroenteritis
Virus	Astrovirus	Contact, food, surface, water	£10 (£2.2 to £31.6)	Associated with acute diarrhoea in young children. Symptoms include diarrhoea, vomiting, and stomach cramps
Virus	Influenza	Airborne, surface	Not included in (26)	Sudden onset of fever, headache, cough, muscle and joint pain, severe unwell feeling, sore throat and runny nose. Most people recover within 7 days, but severe illness or death occurs in high-risk groups
Virus	Norovirus	Contact, water, food, airborne, surface	£1,678.2 (£238.6 to £1,943.6)	Highly infectious. Rapid onset of nausea, vomiting, diarrhoea, fever, headache, dehydration and aches. Most people recover within 1 to 3 days
Virus	Respiratory syncytical virus (RSV)	Airborne, surface	Not included in (26)	Nasal congestion, sneezing, cough and sometimes fever. Small numbers develop more severe illness
Virus	Rotavirus	Contact, water, food, surface	£8.5 (£2.1 to £23.5)	Highly infectious in children, with symptoms include vomiting, watery diarrhoea, and fever which last 1 to 2 days. Incidence has reduced since introduction of vaccination in 2013
Bacteria	Sapovirus	Contact	£169.5 (£112.4 to £251.7)	Similar to norovirus

Type of infection	**Disease or pathogen**	**Transmission mode**	**Total cost (median £million, 95% CIs) in 2018 (26)**	**Symptoms and severity**
Bacteria	*Campylobacter*	Zoonotic, food, bathing, water	£712.6 (£298.4 to £1,355.8)	Diarrhoea (sometimes bloody), abdominal pain, fever, headache, nausea and vomiting. Most cases are sporadic and self-limiting. One in 1,000 may develop Guillain-Barré syndrome
Bacteria	*Clostridium perfringens*	Contact, food	£101.5 (£25.3 to £385.0)	Watery diarrhoea (sometimes bloody), stomach cramps, dehydration, fever and loss of appetite and weight loss
Bacteria	*Clostridium difficile*	Contact, surface	Not included in (26)	Diarrhoea (sometimes bloody), abdominal pain, dehydration, fever
Bacteria	*Legionella*	Airborne, water	Not included in (26)	Fever, chills, headache, muscle pain, dry cough, diarrhoea and later pneumonia. High hospitalisation and fatality rate (5% to 15%)
Bacteria	Leptospirosis	Zoonotic, water, bathing	Not included in (26)	Flu-like symptoms including fever, chills, severe headache, muscle aches, vomiting and diarrhoea. In severe cases kidney, liver failure or meningitis may occur. High fatality rate of 6% to 17% (increasing to 40% when there is hepatic involvement)
Bacteria	*Listeria*	Food	£37.4 (£34.4 to £40.8)	Usually mild causing flu-like symptoms or gastroenteritis. Listeriosis is dangerous to pregnant women, the elderly and people with weakened immune systems. Mortality can be as high as 30% in vulnerable populations
Bacteria	Salmonellosis	Zoonotic, food, water, environment	£212.0 (£45.6 to £954.8)	Fever, stomach cramps, diarrhoea, vomiting and nausea and may last 4 to 7 days. Hospitalisation may be required for the young and old or those with weakened immune systems

Type of infection	**Disease or pathogen**	**Transmission mode**	**Total cost (median £million, 95% CIs) in 2018 (26)**	**Symptoms and severity**
Bacteria	Shigellosis	Food, water, contact	£12.3 (£0.8 to £38.3)	Diarrhoea (sometimes bloody), fever, nausea and stomach cramps which may last for around 7 days. Severity depends on serotype
Bacteria	STEC/VTEC	Food, water, zoonotic, surface, contact	£3.9 (£3.0 to £4.6)	Mild to severe diarrhoea (often bloody) stomach cramps and vomiting. In England 5% to 14% of O157 cases develop haemolytic uremic syndrome with kidney damage
Bacteria	Vibriosis	Food, bathing	Not included in (26)	Non-cholera vibrios cause a self-limiting diarrhoea. Eye, ear, and wound infection and blood poisoning are also possible
Bacteria	*Yersinia enterocolitica*	Food, water	Not included in (26)	Symptoms vary with age and include fever, abdominal pain, and diarrhoea are common. Symptoms may last 1 to 3 weeks but complications are rare
Parasite	*Cyclospora*	Food, water	Not included in (26)	Diarrhoea, loss of appetite and weight loss, stomach cramps and muscle aches. Self-limiting in individuals with healthy immune systems
Parasite	Cryptosporidiosis	Food, water, zoonotic, bathing	£2.1 (£0.3 to £15.3)	Diarrhoea, abdominal pain and less frequently fever, nausea and vomiting. Self-limiting but more severe illness occurs in individuals with impaired immunity
Parasite	Giardiasis	Food, water, zoonotic, contact, bathing, surface	£75.0 (£11.6 to £406.0)	Diarrhoea with bloating, pain or stomach cramps

Type of infection	Disease or pathogen	Transmission mode	Total cost (median £million, 95% CIs) in 2018 (26)	Symptoms and severity
Harmful algal bloom	Dinoflagellates and cyanobacteria	Food, water, bathing	Not included in (26)	Neurological and other toxic symptoms with diarrhoea. May be sporadic or an outbreak

Definitions

Airborne: via inhalation of respiratory droplets from an infected individual or aerosolised particles.

Bathing: water activities in coastal or inland water or swimming pools.

Contact: via direct contact from one infected human to another.

Environment: environmental contamination

Food: via consumption of contaminated food and drink.

Surface: via contact with contaminated surfaces

Water: via consumption of drinking water or inhalation of water droplets.

Zoonotic: via direct contact with infected animals and their faeces.

2. Transmission pathways

2.1 Water-associated infections

Water-related diseases include those that are waterborne, water-based, water-washed, diseases attributed to wastewater and diseases related to damp (27). Waterborne infections are acquired through drinking water, recreational water, inhalation, contact, contamination of wounds, growth in equipment and water systems, growth in soil or water, growth in coastal waters, food contamination with water or soil, water contamination of food, through near-drowning and through injection of non-sterile water. Biotoxins can be transmitted or ingested through inhalation, seafood, drinking, dialysis or through recreational exposure. Water-based diseases are where parasite lifecycles require water and are transmitted by water or food. Water-washed diseases (where there is poor access to water) can be hygiene-related or result from flooding or drought. Climate change has the potential to impact on most of these diseases (28, 29). Infectious diseases can be strongly influenced by flooding, drought and disaster, and the impact that climate can have on water, sanitation and hygiene (WASH) pathogens has been previously reviewed (27).

2.2 Foodborne infections

The climatic conditions and surrounding ecosystems in which food is produced potentially influence food safety. Methods of animal husbandry may influence the potential for animal-to-animal and animal-to-human transmission of zoonotic pathogens, and the surrounding environment may alter the propensity for transmission from the environment (for example, flooding). In addition to agriculture, pathogens can be introduced through cross-contamination across the food system, from manufacturing to human consumption. Environmental conditions such as temperature can also influence food risks through more rapid bacterial replication at warmer ambient temperatures. Sub-optimal food preparation, for example during cooking, may lead to further cross contamination or incomplete pathogen die off.

2.3 Airborne infections

Whilst a lot of attention has been paid to understanding the links between climate change and vector-borne, foodborne and waterborne infections, there has been less focus on airborne infections. There are a number of airborne infections with distinct seasonal cycles which are impacted by weather, but the effect of climate change on these pathogens remains unclear. One reason for this is that there are routine vaccination programmes for many airborne infections. In addition, as seen with the COVID-19 pandemic, immunity and the behaviour of people may have an overwhelming influence on the transmission of infections. Similarly, seasonality of transmission may reflect seasonal shifts in human behaviour (such as more time spent indoors in the winter) rather than ecological mechanisms, with indirect and unclear implications for how climate change might influence transmission. Two of the most important airborne infections in terms of number of people infected per year in the UK are influenza and respiratory syncytial virus and are reviewed below.

3. Viruses

3.1 Adenovirus (waterborne)

Adenoviruses are usually spread from close contact with an infected person, exposure to contaminated aerosol and droplets, contact with contaminated objects, via the faecal route and through contaminated water, such as swimming pools. Infection with waterborne adenoviruses occurs via the mouth, nasopharynx and the ocular conjunctiva (30). It is thought that adenoviruses are second only to rotaviruses as causative agents of gastroenteritis in young children (30). Adenoviruses were detected in respiratory samples throughout the year in Scotland, with a peak in infection detected during March and April (31). Similarly, detection rates of adenovirus in children in Japan were highest between December and March (32). Adenoviruses can be transmitted through person-to-person transmission routes, and outbreaks have been associated with swimming pools and natural water bodies (30).

Adenovirus levels in natural waters in Taiwan (which has a temperate climate) were affected by rainfall (33), although no significant relationship between adenoviruses or temperature and humidity was found in samples from Turkey (34). In Scotland, temperature and dew point were significantly lower on days when adenoviruses were present in respiratory samples (31). There is little evidence that human disease in the UK will be significantly increased by climate change. Swimming in UK inland waters may pose additional risks to health, but these are particularly associated with water contamination following heavy rains. Whilst there may be future health risks associated with new adenoviruses originating from wild animal sources (35), the emergence risks are difficult to predict, and emergence risk in the UK because of climate change appears low.

3.2 Astrovirus

Astroviruses are gastrointestinal agents largely associated with young children. Infection prevalence can vary: in most areas the reported prevalence is about 10% (range in temperate climates 2% to 9%), but higher rates of up to 30% have been reported in some low- and middle-income countries (see 36). Astrovirus outbreaks are combined with other enteric virus outbreaks in surveillance reports in England (for example, (37)), so UK seasonality is unclear, although they are considered to only make up a small proportion of the overall number reported. A study of astrovirus cases in children in Japan during 2009 to 2015 found that 72.5% of infections were detected from March to May, with no cases detected in winter months (38), with similar results also reported in USA (39). However, according to other studies in temperate climates, infection rates are higher in winter months, although cases have been reported in the spring and summer seasons as well (36).

There is very little research or evidence on the effect of weather on astroviruses. One study examined associations between 8 hydrometeorological variables and enteric viruses including astrovirus (40), but as the study compared tropical and sub-tropical locations, it is not comparable to the temperate UK climate. As such, the effect that climate change may have on UK cases of astroviruses is currently unclear and requires further research.

3.3 Influenza

Worldwide, annual epidemics of seasonal influenza are estimated to result in 3 to 5 million cases of severe illness, and up to 650,000 deaths per year (41). Seasonal influenza outbreaks most often occur during winter in temperate climates, such as the UK. Surveillance from across the UK suggests that influenza cases start to increase around October and November, with the peak in cases usually between December and January (42).

Several experimental and observational studies suggest that influenza transmission in temperate climates increases when humidity (particularly absolute humidity) is low (for example, (43 to 49)). Low temperature has been identified as an important driver of outbreaks (46, 50, 51), but it is likely that such associations will be driven by the strong correlation between temperature and absolute humidity (48). Analysis of influenza season in the USA suggests that fewer people are infected with influenza during warm winters, which leads to high numbers of susceptible people the following winter, resulting in early and severe outbreaks (52). There is therefore an interaction between weather and population immunity, with seasonal epidemics in winter months being directly and indirectly impacted by weather parameters. It is likely, however, that factors such as human behaviour (for example, crowding indoors during the winter), uptake of annual vaccination campaigns and population immunity (specifically the number of susceptible individuals) will be a greater driver of influenza burden in the UK in future, with climate change playing a relatively minor comparative role.

3.4 Norovirus

Human noroviruses are responsible for around a fifth of all acute gastroenteritis worldwide (53). Transmission is predominantly person-to-person, with foodborne, waterborne and surface contamination also possible. Noroviruses are important pathogens that trigger food safety incidents (particularly seafood and cross-contamination from infected food-handlers), waterborne outbreaks (particularly non-chlorinated drinking water supplies and bathing waters) and disruption in health and social care sectors. In England, mean laboratory-confirmed infections are at their peak between November and March, with the lowest number of infections confirmed during the summer period (37). Most norovirus infections are short-lived and resolve before medical attention or laboratory-confirmed diagnosis are necessary. As a result, surveillance catches only a small percentage of cases and focuses on outbreaks, particularly in hospital settings. In the UK, this under-ascertainment has been estimated as 288 unreported community cases (95% confidence intervals (CI): 239 to 346) for each norovirus case reported to national surveillance (53).

Norovirus incidence was higher when temperatures were colder in England and Wales (54) and Taiwan (55), and there were higher hospitalisations in children under 5 due to norovirus when temperatures were colder in Hong Kong (56). In England and Wales, norovirus cases were lower when relative humidity over the previous 5 weeks was high (54), yet hospitalizations in children under 5 in Hong Kong were positively correlated with higher relative humidity (56). It should be noted, however, that Hong Kong has a humid, subtropical climate, which might explain the contrasting results between the 2 studies. Rainfall has also been shown to affect norovirus cases. There was an increased hospitalization risk due to norovirus infection during extreme precipitation in Hong Kong (56), whilst positive correlations between rainfall and norovirus incidence were reported in Australia, with a 3-month lag between peak average rainfall and the incidence of norovirus outbreaks (57).

As norovirus incidence appears to be higher when temperatures are colder (54 to 56), it might be assumed that with warmer conditions in the UK as a result of climate change could lead to fewer cases, which could be a beneficial impact of climate change. It is unlikely, however, that weather factors alone are likely to drive infection seasonality. Norovirus is highly infectious; increasing population densities in future could result in increased norovirus reports irrespective of the climate. The role that climate change will play in changing norovirus risk remains unclear, but it is likely to be mixed and complex given the other drivers of transmission.

3.5 Respiratory syncytial virus (RSV)

Respiratory syncytial virus (RSV) is one of the most important viral respiratory infections and is transmitted by large droplets and secretions from an infected person. By 2 years of age, most children will have been infected by the virus. During 2019, it is thought that there were 33 million (uncertainty range: 25.4 million to 44.6 million) RSV-associated infection episodes occurring globally in children aged 0 to 60 months, with almost 1.7 million infection episodes (UR: 943,000 to 2.9 million) occurring in high income countries (58). Epidemics in the UK demonstrate distinct seasonal cycles: transmission usually begins in October and last for between 4 and 5 months, with the peak of infection generally occurring during November and December (31, 59).

A study of RSV climate drivers in the USA and Mexico identified precipitation and humidity to be important, similar to influenza studies: increased humidity driven by higher temperatures led to reduced transmission (60). Detection rates of RSV in respiratory samples from Scotland were lower when temperature and dew points were higher, with a 1°C mean temperature increase reducing the odds of detecting RSV by 17.3% (31). Analysis of laboratory isolations of RSV and emergency department admissions related to RSV in England and Wales showed that the RSV season ended approximately 3.1 weeks earlier per 1°C increase in mean daily temperature, although there was no effect of temperature on the start of the season (61). As a result, it was suggested that under warmer temperatures, there could be a shortening of the RSV season in future (61), but whether climate change will affect the burden of disease remains unclear, and weather correlations remain an imperfect proxy for climate impacts on transmission over longer periods of time.

3.6 Rotavirus

Rotaviruses are a leading cause of diarrhoeal diseases in children globally. Rotaviruses are shed in large quantities in faeces; transmission is through close person-to-person contact, primarily through the faecal-oral route (62). Infants and young children are at particular risk as they can easily become severely dehydrated and require hospitalisation (63). Adults can also be infected with rotavirus, but it is generally less severe than during childhood (63). Global deaths from rotavirus in children under the age of 5 have reduced in multiple countries following the introduction of a vaccine (64). The vaccine was added to the national vaccination schedule for babies in the UK in 2013, and following the addition, in England and Wales there was a 77% decline in laboratory-confirmed rotavirus infections and 26% decline in all-cause acute gastroenteritis-associated hospitalisations during 2013 and 2014, resulting in approximately 10,884 laboratory-confirmed infections and 50,427 hospitalisations averted (65). In England and Wales, laboratory-confirmed cases of rotavirus start to increase at the beginning of the year; since the introduction of the vaccine, the annual peak in reporting has dropped from over 1,000 cases per week to around 150 (66). In the USA, introduction of the vaccine resulted in a change in the peak of rotavirus cases from occurring annually to biennially (67).

In Spain, rotavirus incidence was associated with cold temperatures (68). Similarly in Australia, rotavirus hospital admissions of children under 5 years of age peaked in winter and spring, and were lowest in summer; higher temperature and relative humidity the previous week resulted in reduced hospital admissions (69). A systematic review of the effect of rainfall on diarrheal illness found that overall, incidence of bacterial and parasitic diarrhoeal infections was more common during rainy seasons, but the opposite was found for rotavirus: there was increased incidence during dry seasons (70). Similarly, a systematic review found a negative relationship between ambient temperature and incidence of diarrhoea (the majority of pathogens were rotavirus) (29). In the USA, a strong El Niño followed by a La Niña event had no impact on the seasonality of rotavirus infections (71), suggesting that climate may not be a significant driver of rotavirus. Similarly, another study found that only a low proportion (less than 10%) of the variability of rotavirus cases could be explained by hydrometeorological variables (72). It has been suggested instead that births of susceptible babies drive the annual cycle of rotavirus infections (73). The evidence for the role that weather conditions may have on rotavirus cases remains mixed; further analysis of the relationship between the climate and cases in the UK following the introduction of vaccination is required to determine how incidence may be affected in future.

3.7 Sapovirus

Sapovirus has been increasingly reported as the etiologic agent in outbreaks and sporadic acute gastroenteritis cases, which may in part be due to increased testing in recent years. The reporting of sapovirus infection is greatest in children under 5 years of age, although all ages can be affected (74). A systematic review of published studies in high-income countries that used molecular diagnostics found sapovirus prevalence was 3.4%, with high prevalence among children under 5 years of age (75). Sapovirus outbreaks are combined with other enteric virus outbreaks in surveillance reports in England (for example, (37)), so the seasonality in the UK is unclear, although they are considered to only make up a small proportion of the overall number reported. In studies conducted in Spain and Japan, the peak of infection in children and adults occurred during November to March (76, 77). There is very little research into the effect of weather on sapoviruses. As such, the effect that climate change may have on UK cases of sapoviruses is currently unclear and requires further research.

4. Bacteria

4.1 Campylobacteriosis

Campylobacter species are prevalent in food, animals (poultry, cattle, pigs, sheep and ostriches) and also in pets. There is a consensus that foodborne infection is the main route of transmission; in 2020, and similarly to previous years, the most common food vehicles for 11 campylobacteriosis outbreaks reported to the European Food Safety Authority (EFSA) with strong evidence were broiler meat and raw milk (78). Consuming contaminated water (or ice) and contact with contaminated water during recreational activities is an additional mode of transmission (79). From 2010 to 2019, the highest annual number of *Campylobacter* cases laboratory diagnosed in England was 61,146 (114.31 per 100,000 population) in 2012, and the lowest number of annual cases was 51,817 (94.56 per 100,000 population) in 2015 (80). Cases in the UK are considered underestimated; for every case of campylobacteriosis reported to national surveillance there were an estimated 9.3 cases (95% confidence intervals (CI): 6.0 to 14.4) in the community (53).

Campylobacteriosis incidence exhibits pronounced seasonality with infection peaks occurring in mid- to late summer (weeks 29 to 32) for Nordic countries, but earlier in the year for other European countries (81): in England, infections peak between May and August (80, 82). This remains unexplained, however, since the organism is unable to replicate outside the intestines of warm-blooded animals and the pathogen is thus presumed to have minimal environmental sensitivity. Potential explanations for such patterns include (82): a combination of fluctuating infection rates in poultry (caused either by a direct impact of temperature on *Campylobacter* growth rates or indirect effects such as changes in contamination sources) and changes in exposure due to human behaviour, such as frequency of barbecues (83); and seasonality in the abundance of flies which might act as mechanical vectors of infection, although the evidence is still debated (84 to 86). Environmental measurements of *Campylobacter* in raw sewage display a seasonal pattern related to campylobacteriosis incidence, with a peak in occurrences during summer (24, 87).

Heatwaves are associated with reduced *Campylobacter* incidence (82, 88), and increasing drought frequency is expected to decrease exposure risk to rapidly inactivating *Campylobacter* (89). Increasing annual precipitation and heavy rainfall events lead to higher infection risks (89). In Europe, campylobacteriosis cases were positively associated with temperature and, to a lesser degree, precipitation (81). This is in line with a previously published study showing that the increase of campylobacteriosis in the late spring in England and Wales was significantly linked to temperature 2 weeks before (90). Following on from this, and using a 14-day lag, the daily incidence of campylobacteriosis in England and Wales was associated with maximum air temperature: it is constant for temperatures below 8°C, with an increase in 1 case per million for every 5°C temperature rise between 8°C and 15°C, but no further rise in cases at temperatures over 15°C (91), which are broadly in line with a previous study (92). Furthermore, the prevalence of campylobacteriosis cases increased when relative humidity was in the region of 75% to 80% and with a strong association with day-length, while rainfall and wind-speed associations were weaker (91). It is worth noting however, that in South Korea, campylobacteriosis was not significantly associated with any combination of climatic factors (93, 94). These conflicting findings might be explained by the biology of the organism responding to climatic factors differently depending on the mode of transmission and host, as well as different food production approaches between countries. Survival of *Campylobacter* in animal effluents, soils, and surface waters decreases at higher temperatures, whilst in contrast, the detection frequency of *Campylobacter* in poultry and oysters increases at higher temperatures (84). Detection of *Campylobacter* in surface water, however, show less clear patterns (84).

Despite these challenges, the impact of climate change on campylobacteriosis has been assessed. *Campylobacter* cases in Denmark, Finland, Norway and Sweden could increase by 25% by the end of the 2040s, and 196% by the end of the 2080s compared to the predicted baseline of 2000 to 2015 (82). The models also predict a change in case seasonality, with an extension of the high season until November (82). In contrast, climate change is considered to have little overall impact on the runoff of *Campylobacter* from land to surface waters when considering the risk of infection with *Campylobacter* during bathing downstream of sewage emissions.

Based on the findings of the aforementioned study (82), it is possible that *Campylobacter* cases in the UK will increase due to climate change, with the seasonality of cases possibly extending into the autumn. This assessment, however, requires some caution since the study is based on Scandinavian countries, where incidence exhibits a statistically significant association with precipitation and temperature (82), which is not the case in the UK. On the other hand, the expected higher frequencies of heatwaves might result in a decrease in *Campylobacter* incidence. Understanding the mechanism of transmission is crucial for a robust assessment of the impact of climate change on the epidemiology of campylobacteriosis.

4.2 *Clostridium perfringens*

Clostridial food poisoning is caused by the spore-forming bacteria *Clostridium perfringens,* which causes illness in humans via toxins produced through its growth within foods, particularly meat or poultry products which are cooked in large batches and kept at unsafe temperatures (95). It occurs naturally in the environment, soil, water and in the gut flora of humans and animals, which makes laboratory detection problematic because quantitative counts are required (96). There are approximately 90,000 cases (1.5 cases per 1,000 person-years) of *Cl. perfringens* in the UK each year, with around 15,000 cases (0.2 cases per 1,000 person-years) presenting to general practitioners and 0.001 cases per 1,000 person years reported to national surveillance (53).

There was a seasonal occurrence of food poisoning caused by *Cl. perfringens* in South Korea, with more outbreaks in summer months and fewer during the winter period (97). Specifically, cases were very high during June and August, and although infection rates peaked at high temperature and relative humidity, there was no significant correlation with either variable (97). Another study in South Korea found that increased insolation (defined as the incident solar radiation received on a surface) and sunny weather could result in *Cl. perfringens* cases increasing (98). A study of *Cl. perfringens* outbreaks in north-east England, however, found the highest number of outbreaks occurring during November, followed by October and then February, April, July and August (99). Further studies are required to understand how the incidence of *Cl. perfringens* may be affected by weather conditions in the UK.

4.3 *Clostridium difficile*

Clostridium difficile is a bacterium found in the intestines. In healthy people, it causes no symptoms, but when normal gut bacteria are disadvantaged, such as when someone is taking antibiotics, *Cl. difficile* can grow to unusually high levels (100). In England during 2018 and 2019, there were over 12,000 cases of *Cl. difficile*, equating to 21.9 cases per 100,000 population (101).

Clostridium difficile is present both in the natural environment and in healthcare environments and varies seasonally (102). According to a systematic review, *Cl. difficile* rates peaked during March and April and declined from June onwards in the Northern Hemisphere (5 European and 13 American studies) (103). The occurrence of *Cl. difficile* was linked to flooding, where emergency room and outpatient visits in USA were elevated during a period of 7 to 13 days following a flood (104). In South Korea, intensity of sunshine was positively associated with infection rates of *Cl. difficile* (97), whilst a study in Queensland, Australia found that the odds of *Cl. difficile* incidence was positively associated with monthly rainfall (105). However, seasonality of infection might be confounded by antibiotic use, which also follows a seasonal pattern, with increased use during winter months proposed to be associated with higher incidence of respiratory infections: in Europe, antibiotic use peaks during January and February, and patients remain at risk for up to 3 months following antibiotic use (see (103).

Climate change could impact on *Cl. difficile* indirectly, by affecting exposure to antibiotics following seasonal respiratory diseases. Further work to determine associations and drivers in the UK are therefore required.

4.4 Legionellosis

Legionnaires’ disease (termed ‘legionellosis’ from hereon) is a severe pneumonia, caused by the gram-negative bacteria *Legionella pneumophila*. The bacteria are found naturally in water bodies such as lakes and rivers, as well as in rainwater puddles and in natural soils (106). Under favourable conditions in human-made water systems operating between 20°C and 45°C such as showerheads or cooling towers (107, 108), the bacteria are able to multiply, and can be transmitted by aerosols: infection occurs following inhalation of aerosols containing *Legionella* (109).

In temperate regions, the majority of legionellosis cases occur during the summer: in England and Wales, the highest number of cases are reported during June to October (110). A similar trend has also been reported across EU/EEA, the USA and Japan (111 to 113). A study of 800 legionellosis cases in the Netherlands during 2008 to 2011 found that 4-week mean temperature, 2-week rainfall duration and 2-week rainfall intensity were the best predictors of high case incidence (114). Warm temperatures were suggested to promote *Legionella* growth and the wet weather assisted with spreading aerosols (114). A study of cases in Philadelphia, USA found that whilst legionellosis cases occurred predominantly during summer months, rainy and humid periods during the summer were a better predictor of cases than temperature (112). Increased rainfall was also associated with increased infection risk in Spain (115) and in patients from 5 states in the USA (116). Analysis of legionellosis cases from 77 regions across 4 European countries (Denmark, Germany, Italy and the Netherlands) with weather variables found that simultaneous increases in rainfall and temperature were associated with a higher risk for cases, although temperatures above 20°C were not associated with a higher infection risk (117).

It is possible that with climate change, warmer temperatures could lead to more legionellosis cases being reported in many temperate countries during the spring and the autumn (118). In the UK, longer and hotter summers could result in extended use of evaporative cooling systems, such as cooling towers, which are known to be a source of large legionellosis outbreaks (108, 118). Control of microbial growth in surface waters and human-made water systems may be required to mitigate potential increased risk of legionellosis under warming conditions in the UK (118).

4.5 Leptospirosis

Leptospirosis is an important bacterial zoonosis and is considered the most geographically widespread zoonosis in the world (119). The brown rat (*Rattus norvegicus*) is the most important transmission source of human infections, although other wild and domestic animals act as reservoir hosts. Infection results from direct exposure to the urine of infected reservoir host animals or, more commonly, via indirect contact with water or soil contaminated with leptospires. It is usually associated with occupational (such as military personnel, mine workers, sewer workers, slaughterhouse workers) or recreational activities including swimming in freshwater bodies (119, 120).

Although 73% of the world's leptospirosis cases and deaths occur in tropical regions, the estimated annual leptospirosis morbidity and mortality in Western Europe are 3.90 (95% CI: 1.45 to 6.49) and 0.18 (0.07 to 0.29) per 100,000 population, respectively, resulting in 800 (300 to 1,200) deaths (121). In the EU/EEA, cases are most common during July to October (peak in cases during August and September (112)). Leptospirosis is rare in the UK: there were 55 confirmed and 115 probable cases in England and Wales during 2022, compared with 91 confirmed cases in 2019 and 72 in 2018, with many linked to travel overseas (123, 124).

Whilst traditionally considered a disease of tropical climates, leptospirosis has emerged as a health threat in temperate settings due to the influence of globalization and climate change (121). Extreme weather conditions, such as heavy rainfall, flooding, hurricanes, and typhoons (as well as disasters such as tornadoes, tsunamis and earthquakes) can result in large epidemics (119, 125 to 129). Above-normal temperature conditions may also result in greater risk of infection. For example, an increase in leptospirosis cases in the Netherlands during 2014 was linked to mild conditions during winter of 2013 and 2014, followed by the warmest year in 30 years (130). It is thought that warmer-than-average conditions could have improved survival of rodents and excreted virus particles, coupled with increased outdoor recreational activities earlier in the year might have led to more exposure and an earlier rise in cases (130).

The specific impact that climate change will have on leptospirosis cases in the UK is unclear. Survival of leptospires in the environment is likely to increase at higher temperatures, and as was suggested in the Netherlands (130), increases in recreational activities in response to higher temperatures may increase exposure to infection. Importantly, climate change might result in an expansion of the habitats of reservoir hosts into higher elevations and latitudes (131). More frequent and severe flooding events due to climate change are also expected to increase the transmission risk of leptospirosis (132).

4.6 Listeriosis

Human listeriosis is caused by the bacterium *Listeria monocytogenes* which is widely distributed in the environment, and is detected globally (24). Listeriosis is primarily associated with ready-to-eat foods, milk products, cheeses, as well as meat, poultry and seafood (84). Routine surveillance is for cases of invasive disease, which is relatively rare so results in lower numbers of reported cases compared with other foodborne pathogens, but it is a public health concern due to severity of infections and high case fatality ratio (26, 133). During 2011 to 2019, there were between 133 and 180 confirmed cases of listeriosis reported in England and Wales, with an incidence rate per 100,00 of between 0.23 and 0.31 (133). Seasonality of confirmed cases in the EU suggests slightly greater reporting in the second half of the year (78), but data for England and Wales during 2018 and 2019 suggests a rise in cases during April and May, but no clear pattern for the later part of the year (133).

A previous review found a weak association between meteorological variables and *Listeria* spp. (24), which was likely driven by a lack of scientific literature, which in turn has not significantly increased since the review was published. As *Listeria* bacteria grow in a wide temperature range, it is unlikely that climate change will directly impact listeriosis incidence in the UK (24), although further research on extreme weather events such as floods or droughts is needed to adequately assess the risk.

4.7 Salmonellosis

Non-typhoidal *Salmonella* spp. are one of the most widely distributed foodborne agents of infectious intestinal disease in humans, causing significant morbidity and mortality worldwide (134). In the EU, non-typhoidal *Salmonella* is the most common bacterial agent in foodborne outbreaks and, after *Campylobacter,* the second most common by number of affected people. In the EU during 2019, there were 926 foodborne outbreaks and 87,923 human cases of salmonellosis reported, corresponding to a notification rate of 20.0 per 100,000 population (135).

Salmonellosis occurrence has been associated with ambient air temperature (see (3). Infection risk of salmonellosis was positively associated with relatively high temperatures in Auckland and Christchurch, New Zealand; however the same study found no significant association between temperature and salmonellosis risk in Wellington (136). Because of this localised health impact, the authors advocate the need for region-specific preventative measures (136). A systematic review on *Salmonella* and the environment focusing on New York, USA found mixed results or no associations between occurrence of *Salmonella* and climatic variables (precipitation and humidity) other than ambient temperature (3). A study in Maryland, USA, showed that there was a 4.1% increase in salmonellosis risk per one unit increase in extreme temperature events, and a 5.6% increase in risk per one unit increase in extreme precipitation events, with the risks greater in coastal versus non-coastal communities (137). In contrast, there were strong positive correlations between high temperature and *Salmonella* infections in Mississippi, USA, with a 1°F rise in temperature leading to an increase of 4 *Salmonella* cases, but there was no correlation between monthly mean precipitation and infections (138).

The inconsistencies of findings, especially for the relationship between *Salmonella* and precipitation, is not surprising considering that the direct impact of climate on the biology of *Salmonella* is not trivial. High temperatures affect the survival and proliferation of salmonellosis in the environment and in food (139). However, survival of *Salmonella* in animal effluents, soils, and surface waters decreases at higher temperatures and in fluctuating ambient temperature or freeze-thaw events (see (84). In contrast, detection of *Salmonella* in poultry and oysters increases at higher temperatures (84). The survival of *Salmonella* in soil increases in moist conditions, and rainfall promotes its dispersal. Rainfall is also thought to increase the ability of lettuce to internalise bacteria through stoma in the leaves, and to allow bacteria to enter seeds (84, 140). Zoonotic spillover of *Salmonella* from rodents could increase with rainfall due to an increased population of animal hosts (141). Detection of *Salmonella* in surface water shows less clear patterns (84). Drought might enhance *Salmonella* internalization in lettuce (84), and wind and dust might increase airborne dispersal of *Salmonella*, although the strength of evidence is weak (84). Furthermore, some studies suggest that certain phage types and serovars are more sensitive to the effects of temperature which might explain the differences in patterns and lag effects found across a variety of studies (142). This is particularly important as certain serovars are associated with more severe disease (3) and potentially more exposed to environmental factors according to the main route of transmission (environment to human rather than foodborne). There may also be variations in *Salmonella* prevention between countries, which could also explain differences in patterns. For instance, most chickens in the UK are vaccinated for *Salmonella*, whilst there currently are not similar vaccination programmes in the USA.

Predicting how patterns of salmonellosis will change in response to climate change is difficult because of the intrinsic complexity and uncertainty of how climate change will affect the host–agent–environment components and their interactions (3, 143, 144). Furthermore, technical methodological challenges, such as highly correlated climatic variables, makes the task of identifying the true explanatory variables, and thus predicting risk, more difficult (145). Nevertheless, climatic changes could increase the future incidence of salmonellosis in selected areas (24, 137, 138). Based on the arguments above about the response of different serovars and the mode of transmission to ambient temperature, future predictions on the impact of climate change on salmonellosis, should distinguish between the specific serovars and mechanism of transmission involved.

Despite the differences highlighted above, the studies indicate that it is reasonable to expect that increased ambient temperature will result in an increase of salmonellosis in the UK, although England and Wales are successfully reducing the pathogen levels in major food groups and improving food hygiene at the domestic and institutional level thus adapting to the threat of increased foodborne illnesses posed by climate change (146). Currently, there is not enough compelling evidence to assess the impact of precipitation, drought and wind. Other climatic variables, like vapour pressure, soil temperature, global radiation, temperature threshold, UV light, and so on, might have an effect but the potential impact of these variables has not been extensively studied (24, 145). Human behaviour and land use is also expected to influence the cases reported (for instance, increased outdoor food preparation, and reduced food safety measures in hot days) (147). Similar to the US, extreme weather events might alter the risk of Salmonellosis in the UK, with some communities disproportionately affected than others (for example, coastal versus non-coastal as described above). It is also possible that there will be populations that will be more at risk than others, such as older age groups; more research in the UK is required to determine risk for different populations.

4.8 Shigellosis

Shigellosis is a diarrhoeal disease caused by *Shigella bacillus* and there are 4 serogroups: subgroup A, *Shigella dysenteriae*; subgroup B, *Shigella flexneri*; subgroup C, *Shigella boydii*; and subgroup D, *Shigella sonnei*. Whilst globally subgroups A, B and C cause more severe dysentery (148, 149), subgroup D causes epidemic diarrhoeal disease in school children (150) and other care institutions (151). Infection can be spread via the faecal-oral route, or from ingesting contaminated food or water (152). Whilst children are usually the most affected, travellers and men who have sex with men are the main risk groups in high-income settings (152). In the EU/EEA, cases of *Shigella* peak during August and September (152).
In Taiwan, the number of shigellosis cases started to increase when temperatures reached 21°C and relative humidity was between 70% and 74% (153). A meta-analysis found that the

incidence of enteric infection increased by 2.3% per 1°C temperature increase for shigellosis (154); all included studies investigated infections in persons from China and Iran, so interpretation of findings should be done with caution as they may not be directly comparable to the UK. An outbreak of *S. Sonnei* in homeless persons in Oregon, USA during July 2015 to June 2016 was attributed to the wettest rainy season on record whereby increasing precipitation resulted in increased cases amongst homeless persons but not amongst housed persons (155).

Warmer temperatures could facilitate the growth and survival of *Shigella*, and climate change is expected to increase temperature-related excess deaths due to *Shigella* globally (156). Whilst the impact of global temperature increases on shigellosis incidence in some countries has previously been modelled (156), to our knowledge, the impact of climate change on cases in the UK has not yet been assessed.

4.9 STEC/VTEC

Shiga toxin-producing *Escherichia coli* (STEC, also called Verotoxigenic *E. coli* or VTEC) are a group of bacteria which cause gastrointestinal illness in humans. In the UK, the most commonly identified type of STEC is STEC serogroup O157 (157). Ruminants, particularly cattle, have been identified as important sources of STEC (158, 159).

Infection with STEC O157 can occur through a number of routes, including eating contaminated food (such as raw or undercooked meat, raw milk, fruit and vegetables contaminated with animal faeces); drinking from untreated water supplies; swimming in contaminated water; contact with infected animals (at petting farms); or contact with infected people (157). A review of sporadic STEC cases found that infection was most commonly linked to association with animals or their habitat (70.4% of studies), with consumption of raw or undercooked meat also a significant risk factor (62.5% of studies) (160). Another review looking at global STEC outbreaks found that in European cases which could be attributed to a source, 31% of outbreaks were due to beef consumption, 30% were due to fresh produce (vegetables) consumption and 16% due to dairy (161).

In the EU/EEA region (including the UK) during 2015 to 2019, the number of confirmed STEC cases began to increase generally during April and May, and the highest number of cases were reported during June to September (162). Similar results from a systematic review looking at incidence across multiple temperate countries have also been reported (163). Focusing on England only during 2015 to 2019, there was some variation, with cases increasing from April until July (2016 only) or August (all other years) and then decreasing (164). During 2019, the highest number of cases per 100,000 population in the EU/EEA was seen in 0- to 4-year-olds, who accounted for more than a quarter of all confirmed cases, whilst the lowest number of cases were in the 45 year olds to 64 year olds (162).

STEC have an optimum growth temperature of 37°C but can grow in temperatures ranging from 7°C to 50°C (165). A study of STEC infections in children in Italy showed an increase in cases during heatwaves: the duration, magnitude, amplitude, number and frequency of heatwaves was positively correlated with detected STEC infections (166). With the increased likelihood of more frequent heatwave periods, it is likely that there will be higher case numbers of STEC infections in future (166). A probabilistic modelling study estimated that temperature increases in France could affect the microbial growth rate in raw milk (167). The model predicted that warmer temperatures coupled with the time taken to pass along the supply chain resulted in higher estimated concentrations of *E. coli* in milk, which could increase the infection risk following raw milk consumption in future (167). In addition to raw milk, climate change will likely have both positive and negative impacts on the survival of STEC O157 in manure, soil and water, which has implications for leafy green vegetable contamination (see (140).

There are risks that STEC cases will increase in the UK under climate change, particularly in the context of warmer and drier summers and in relation to the integrity of water supplies used in salad production.

4.10 Vibriosis

There are at least 12 *Vibrio* spp. That are pathogenic for humans (168); the most significant are *Vibrio vulnificius*, *Vibrio parahaemolyticus* and *Vibrio cholerae*. Cholera epidemics are caused by *V. cholerae* (serogroups O1 and O139) and have been eliminated in high-income countries due to the availability of sanitation and treated and safe drinking water (169). However, cholera outbreaks can occasionally occur in parts of Europe (for example, (170)). There are also nontoxigenic *V. cholerae* (nonO1 and nonO139) which are associated with sporadic cases of infection. Human vibriosis infections have occurred following consumption of raw or undercooked seafood or fish, exposure to contaminated water or exposure of skin lesions, and can result in gastroenteritis, septicaemia, ear infections, wound infections and cholera (171). In the USA, vibriosis cases have a marked seasonal distribution, with most occurring during summer and early autumn (172). There have not been large foodborne *Vibrio* outbreaks in the UK in recent years, although there is evidence of the bacteria in the environment (173). The only vibriosis cases reported for England, Wales and Northern Ireland are linked to overseas travel (174).

Vibrio spp. Are commonly found in marine and estuarine waters around the world (175), and infections were traditionally observed in tropical and sub-tropical locations (176), but this is now changing. *Vibrio* presence and growth in shellfish is dependent on seawater temperature, and warmer temperatures are predicted to increase *Vibrio* growth, resulting in more disease outbreaks. Increasing sea surface temperatures are also thought to be driving the emergence of *Vibrio* infections in areas historically considered too cold for survival (177). For example, Galicia in north-western Spain is considered a hotspot region for *V. parahaemolyticus* and increases in sea surface temperature are associated with the emergence of cases and changes in the epidemiology of the pathogen (176, 178). Similarly, increases in UK sea surface temperatures have been associated with the presence of *V. parahaemolyticus* in shellfish samples collected from locations along the UK coastline, with high concentrations of *V. parahaemolyticus*, *V. vulnificus* and *V. cholerae* detected during periods of higher water temperatures and lower salinity (173, 179).

Coastal waters are becoming more suitable for non-cholera *Vibrio* (180): the area of suitable coastline has increased from 47.5% to 86.3% in the Baltic and from 30.0% to 57.1% in the north-eastern USA coastline (180). Compared with 1982 to 1989, an extra 4.3% of coastal waters in northern latitudes had temperatures suitable for *Vibrio* from 2014 to 2021, increasing the suitability of brackish waters for *Vibrio* transmission (180). Extreme heat events during summer months resulting in the warming of sea surface temperatures have been linked to the emergence of *Vibrio* infections in the Baltic region (177, 181, 182). In Denmark, human *Vibrio* infections were correlated with high summer coastal water temperatures and low salinity (182). A study focusing on the Swedish coastline found a statistically significant increased risk of *Vibrio* infections when sea surface temperatures exceeded 16°C (181). The authors looked at projection data for sea surface temperatures under the RCP4.5 and RCP8.5 scenarios and found that the area suitable for *Vibrio* growth was projected to expand, an increase in relative risk of infection was predicted beyond the year 2039 for both scenarios, and the number of months where transmission could take place increased (181).

As temperatures increase, UK coastal waters will become more favourable for the growth of *Vibrio* and the risk of vibriosis outbreaks (175). Predicted sea-level rise is projected to result in the flooding of low-lying coastal areas, expanding estuarine and brackish environments (24, 181). Elderly people have been recognised as vulnerable to vibriosis infection (182), and as the elderly population continues to grow in future, there will be a greater proportion of the population at risk of infection. While the occurrence of widespread cholera is unlikely, because of UK sanitation and safe potable water supplies, there could be increases in *Vibrio* infections related to bathing and to contaminated seafood.

4.11 *Yersinia enterocolitica*

In humans, infection with *Yersinia pestis* causes plague, whilst yersiniosis is predominantly caused by *Yersinia enterocolitica*, and on rare occasions by *Yersinia pseudotuberculosis*. Pigs are the most common reservoir of *Y. enterocolitica* and infections can occur following consumption of under-cooked pork, or cross-contamination following handling of raw pork, whilst outbreaks of Y. *pseudotuberculosis* have been associated with contaminated vegetable consumption (183). Fatalities are usually rare, but there is increased risk for patients with chronic liver disease. In addition, complications such as reactive arthritis and endocarditis can occur (84). During 2017 to 2019, incidence rates in the UK were approximately 0.2 to 0.3 per 100,000 population, which was lower than the overall European 2020 rate of 1.9 cases per 100,000 population (183).

To date, there has been very little research examining the impact of weather on *Y. enterocolitica* infections. In South Korea, infections of *Y. enterocolitica* were not significantly associated with any of the 5 climatic factors investigated: temperature, relative humidity, rainfall, insolation, and cloudiness (98). To the best of our knowledge, the impact of weather on cases within the UK has not been examined but warrants further investigation.

5. Parasitic diseases

5.1 Cyclosporiasis

Cyclosporiasis is caused by the parasite *Cyclospora cayetanensis*, with recent evidence suggesting that there are 2 other species that may also infect humans: *Cyclospora ashfordi* and *Cyclospora henanensis* (184). Infections are not passed from person-to-person but are readily passed in contaminated water or food (185, 186), and infected food can be imported from endemic countries into countries where *Cyclospora* is otherwise absent (187, 188). In the UK, infections are linked to travel abroad (189, 190), or through ready-to-eat foods such as salads, herbs and soft fruit (191).

Infections are seasonal in many countries (190, 192 to 195). However, there is little evidence to date of the impact that weather may have on *Cyclospora* cases. As UK cases are linked to travel abroad, rises in infections in other countries as a result of climate change could increase the risk of travel-related infections (for instance, (196)).

5.2 Cryptosporidiosis

Human cryptosporidiosis is caused by *Cryptosporidium* spp. That are both zoonotic (*Cryptosporidium parvum*) and predominantly associated with humans as the host species (*Cryptosporidium hominis*). *Cryptosporidium* spp. Are transmitted by environmentally robust oocysts, which also resist chlorine and some other disinfectants. Water samples from various sources are commonly contaminated (197). As a result, these organisms are commonly a cause of outbreaks caused by contaminated drinking water, swimming pools, food and contact with agricultural animals. The outbreaks can range from small and local – often linked to swimming pools – to large local outbreaks linked to drinking water, as well as national or international outbreaks (198) linked to contaminated food or water, outbreaks linked to foreign travel, or sporadic disease linked to continuing source exposure. During 2012 to 2017, there was a small peak in reported cases in England and Wales around mid-April until the end of May, with a much larger peak seen during late August until the end of November (199). Focusing solely on outbreaks in England and Wales during 2009 to 2017, the largest peak occurred during March and April, with the majority attributed to contact with animals; there were smaller peaks during August and September that were mostly associated with recreational water (200).

Heavy rainfall episodes are thought to be associated with cryptosporidiosis outbreaks, as wash-off may lead to infected faeces contaminating surface drinking water sources (201). In Canada, the odds of identifying *Cryptosporidium* oocysts in fresh surface water was 2.61 (95% CI: 1.63 to 4.21) times higher during and after extreme precipitation events compared to baseline conditions (202). In north-western England, increased rates of cryptosporidiosis were associated with increased levels of precipitation (203). Similarly, a study of cryptosporidiosis in Australia found there was increased risk of infection in years with higher rainfall, with an estimated 1.8% increase in cryptosporidiosis risk associated with a 78mm increase in annual rainfall (204). Several studies have also found a link between infections and temperature. In Massachusetts USA, *Cryptosporidium* infections peaked approximately one month after the peak in temperatures (205). Similar findings were reported from Australia, where cryptosporidiosis infections were significantly associated with monthly maximum temperature, with the model estimating that there could be an increase in 50 cases per year per 1°C increase in maximum temperature (206).

The risks of an increase in cryptosporidiosis in the UK associated with climate change are strongly linked to the main sources that have been shown to be responsible for outbreaks. Waterborne disease due to potable waters is the responsibility of Industry and Regulators, and additional measures may be required to upgrade catchments, storage, and treatment to cope with weather changes projected in UKCP18, with lower summer rainfalls and more extreme weather in winter months.

5.3 Giardiasis

Giardiasis is an infection caused by the protozoan parasite *Giardia lamblia* (synonyms: *Giardia duodenalis*, *Giardia intestinalis*). Giardiasis has a global distribution, and is the most reported foodborne and waterborne parasitic disease in the EU/EEA (207). In 2019, there were 18,004 confirmed cases of giardiasis reported across the EU/EEA, with the highest notification rates reported in Belgium (18.0 per 100,000 population), compared with a notification rate in the UK during 2019 of 7.7 per 100,000 (207). Giardiasis infections can be asymptomatic, acute or chronic, with the most frequent symptoms being diarrhoea, stomach cramps and bloating. Cysts are shed in faeces from an infected person or animal, and they can survive in water, food or on surfaces for prolonged periods of time (208). The most common route of infection is ingestion of cysts through contaminated water or food, and occasionally through person-to-person transmission (207).

A systematic review found that in the UK, there is a large summer peak of giardiasis cases during July to September (209). The peak in infections during the summer could result from higher temperatures, as positive associations between giardiasis and temperature have been previously reported (205, 210), but this may also be affected by increased travel during the summer months. Higher temperatures may increase foodborne disease transmission, or it may be driven by increased recreational water use during periods of high temperature to cool down, leading to greater exposure to untreated water sources.

In New Zealand, there were positive associations between giardiasis incidence and rainfall (210). Similarly, increased concentrations of *Giardia* cysts in the Delaware River, USA was associated with rainfall, thought to be driven by increased particulate matter in the water column following surface run off (211). In contrast, however, a study of *Giardia* cyst concentrations in the Grand River, Canada found significantly higher concentrations in the year with the lowest levels of average precipitation (212). The authors posed that the higher observed concentrations could be a result of low water levels in rivers due to the lack of precipitation, reducing the dilution of cysts (212). In addition, sewage effluents being washed into source waters can also result in high concentrations of cysts (212) which, if combined with low river levels or high temperatures, could have important public health implications.

It is possible that with predicted warmer summers, the peak of giardiasis cases during the summer could last longer. It is also possible that predicted increased rainfall could increase concentrations of cysts in rivers, as described above. Clearly, modelling using climate projections is required to understand future UK risk.

6. Harmful algal blooms

Algal blooms in water bodies are a natural phenomenon, yet there are a range of micro- and macro-algae that can bloom in marine and freshwater bodies and impact upon human, animal and ecosystem health (213). Harmful algal blooms (HABs) can occur due to several factors, including changes in water temperature, salinity, turbidity, and nutrients. Exposure to HABs occurs through ingestion of contaminated seafood products, ingestion of contaminated water, inhalation of aerosolised toxins or direct skin contact with toxin-contaminated water (214). Ingestion of contaminated seafood can cause a range of poisonings, including amnesic shellfish poisoning (SP), azaspirazid SP, diarrhetic SP, neurotoxic SP, paralytic SP and ciguatera fish poisoning (see (214). As well as direct impacts on human health, toxic HAB outbreaks can have important socio-economic consequences to the shellfish industry (215).

There is evidence to suggest that climate change has already affected the frequency and severity of HABs (214, 216 to 219). Possible responses of HABs to changing climatic conditions are likely to be highly species-specific, due to the diverse ecology, physiology and toxicity of HAB organisms (214). A study in the USA that used climate change projections from 5 global circulation models has predicted that the number of days with harmful cyanobacterial blooms will increase from 7 days per year per waterbody under current conditions to between 18 and 39 days in 2090 (220). It is likely that similar increases in HABs will be seen in Europe, including the UK in future, which will have public health implications.

A scoping review on the impact of HABs on human health found that there was a low number of studies describing outcomes in Europe (221), and only 3 relevant UK studies were detected (222 to 224). It is possible that the impact of HABs on health in the UK is currently unknown or underestimated; understanding the current burden is vital to predict how incidence may change in future (217, 219, 221, 225).

7. Discussion

Climate and weather can affect pathogen prevalence, survival in the environment and in the food chain, transmissibility and host susceptibility to infection as well as human behaviour (226) and other extrinsic and ecological factors, resulting in complex impacts on infectious diseases, notably seasonality and spatial distribution. This chapter has reviewed the information currently available in the literature detailing the seasonality, links between weather and disease incidence for pathogens which are currently or likely to become a public health concern in the UK, as well as impacts that the UK may face in future. A summary of the conclusions, the confidence in likely impact for the UK and estimated likely capacity to adapt to the changing risks from the literature review are presented in Table 2. For some pathogens there is existing robust modelling data on the relationships between weather variables and pathogen incidence, such as *Salmonella*, *Campylobacter*, *Vibrio* spp. which demonstrates well-documented sensitivity to climate variability, and there is some evidence suggesting that UK disease risk could increase in future. There are also infections such as influenza, where the impact of climate change will likely be relatively minor and factors such as human behaviour and population immunity will be greater drivers of incidence levels. For other pathogens (for instance, astroviruses, sapoviruses and noroviruses), the review conducted here has identified that not only is the interaction with weather variables unclear, but that this modelling data does not currently exist for the UK. It is currently unclear how disease incidence in the UK may change because of climate change, so analyses using climate projections, such as UKCP18 are vital to understand and prepare for how infectious diseases are likely to be affected by the UK's changing climate in future.

The chapter has focused on foodborne, waterborne and respiratory infections currently acquired within the UK. However, climate change is likely to increase human population movement in future, with mass displacement arising in response to sea-level rise, food insecurity or extreme weather events. Migrating populations may be exposed to endemic diseases for which they have limited resistance, or they may bring infections which can easily spread within host populations (227, 228). In addition, there may be an increased risk of multi-drug resistant infections, such as antimicrobial-resistant tuberculosis, which can spread in crowded living conditions and transmission can be exacerbated by lack of access to medical care and diagnostics (15). Climate change-induced migration is thought to be a major driver of emerging and re-emerging diseases. There has been a rise in emerging infectious diseases over the last few decades (229), with recent outbreaks including severe acute respiratory syndrome (SARS) coronavirus (2003), swine flu (2009), and the COVID-19 pandemic (2019). Although these have not been covered in the chapter, such outbreaks are likely to increase in future, and although they are likely to emerge overseas initially, there are likely to be spill-over effects on the UK population, as was seen during the COVID-19 pandemic.

Table 2. Summary of the monetary disease burden, likely impact of climate change (and confidence of the impact), as well as the capacity to adapt to climate change based on each of the infections reviewed in this chapter

Type of infection	Disease or pathogen	UK Monetary Disease Burden[1]	Likely climate change impact[2]	Confidence in likely impact[3]	Capacity to adapt to increased risk[4]
Virus	Adenovirus	Low	Low	Weak	Limited
Virus	Astrovirus	Low	Low	Weak	Limited
Virus	Influenza	High	Low	Strong	Good
Virus	Norovirus	High	Moderate	Weak	Limited#
Virus	RSV	Moderate	Low	Moderate	Limited#
Virus	Rotavirus	Low	Low	Moderate	Good
Virus	Sapovirus	High	Low	Weak	Limited
Bacteria	*Campylobacter*	High	Moderate	Moderate	Moderate
Bacteria	*Clostridium perfringes*	High	Low	Weak	Good
Bacteria	*Clostridium difficile*	High	Low	Weak	Good
Bacteria	*Legionella*	Moderate	Moderate	Moderate	Good
Bacteria	Leptospirosis	Low	Moderate	Weak	Good
Bacteria	*Listeria*	Low	Moderate	Moderate	Good
Bacteria	Salmonellosis	High	Moderate	Strong	Good
Bacteria	Shigellosis	Low	Moderate	Weak	Moderate
Bacteria	STEC/VTEC	Low	Moderate	Strong	Good
Bacteria	Vibriosis	Low*	Moderate	Moderate	Good
Bacteria	*Yersinia enterocolitica*	Low	Low	Weak	Good

Type of infection	Disease or pathogen	UK Monetary Disease Burden[1]	Likely climate change impact[2]	Confidence in likely impact[3]	Capacity to adapt to increased risk[4]
Parasite	Cyclospora	Low*	Moderate	Weak	Limited
Parasite	Cryptosporidiosis	Low	Moderate	Moderate	Good
Parasite	Giardiasis	Moderate	Moderate	Weak	Good
Harmful algal bloom	Dinoflagellates and cyanobacteria	Low*	Moderate	Weak	Moderate

Notes

[1] Most values are based upon information from (26), but for pathogens not included in the report, figures come from other sources (183, 230 to 234). An asterisk (*) indicates that no official figures could be found but values are estimated based on small case numbers and author judgement:

- Low: less than £50 million per annum (p/a) and shown in green
- Moderate: £50 million to £150 million p/a and shown in yellow
- High: more than £150 million p/a and shown in red

[2] Based on the findings from the literature review conducted in this chapter:

- Low: low evidence that climate change may affect incidence, or evidence that it may reduce incidence
- Moderate: moderate evidence that climate change may increase incidence
- High: high evidence that climate change may increase incidence

[3] Based on the findings from the literature review conducted in this chapter:

- Weak: low confidence of the impact described in the previous column due to low numbers of published studies, particulars of study design or conflicting findings
- Moderate: moderate confidence of the impact described in the previous column
- Strong: high confidence of the impact described in the previous column

[4] Based on academic judgement and routes of transmission highlighted within the document, the capacity to adapt should changes in disease incidence be apparent:

- Good: predominantly food and water transmission which are typically highly regulated. Or effective vaccine available.
- Moderate: mixture of food and water and human to human transmission.
- Limited: predominantly human-to-human or airborne transmission which is typically less regulated. A hashtag (#) indicates where current vaccine developments could improve response)

7.1 Climate mitigation impacts

Food is one important pathway for infectious disease, and the food system (farm to fork) accounts for around a third of global and UK GHG emissions (235, 236). Hence, a net zero strategy will involve changes to the food system to reduce GHG emissions and many companies have signed the Courtauld Commitment 2030, a voluntary agreement to deliver food chain reductions in GHG of 50% by 2030 (236). From an agricultural perspective, such reductions in GHG emissions will require "fundamental changes to how land is used" in the UK (237) and overseas where 23% of food system emissions originate. These changes have the potential to alter the range and type of pathogens in food in potentially unknown ways. Through their impacts on ecological and biological systems, they also have the potential to influence infectious disease risks. Furthermore, food waste (23%), refrigeration (2%) and packaging (3%) are other important components of total emissions, and changes to these components have the potential to influence infections disease agents in food. Dietary choice is also highlighted as important for climate change mitigation and several studies have suggested replacing red meat consumption with plant-based foods will help to reduce GHG emissions (238 to 240). The incorporation of sustainability into dietary guidelines can face stiff opposition from vested interests (241). Nonetheless, a potentially changing diet implies a shifting pattern of infectious diseases. For example, many recent outbreaks of STEC O157 have been associated with salad whereas *Campylobacter* is often associated with the consumption of poultry.

The management of water resources is responsible for around 5% of GHG emissions although there is much uncertainty (242). This chapter has highlighted the role that water plays in some infectious diseases, and changes to the sector to reduced GHG emissions have the potential to influence infectious disease risks associated with water transmission routes. Similarly, reductions in air exchange rates within buildings to save energy is another potentially important climate change mitigation measures (see Chapter 5) with the potential to influence viral transmission rates within buildings.

7.2 Adaptation to climate change

This chapter has presented evidence on how, within the UK, several infectious diseases are likely to be affected by climate change. The specific impacts are uncertain, and this is enhanced by wider issues such as the potential for the emergence of novel species, future levels of AMR and impacts from climate change mitigation. However, the overall impact of climate change will also depend upon the capability of society to adapt to changes in infectious disease. Understanding these capabilities and adaptation potentials are key to understanding not only the risks of climate change for infectious disease, but also how resilience to climate change may be enhanced (147).

Public health and preventative medicine are the disciplines with key responsibility for preventing infectious diseases, and this implies a commitment to address climate change (243). The tools and basic concepts of these disciplines provides a blueprint for responding and adapting to climate change, although these may need extending to meet future challenges (243). There are multiple frameworks classifying how health systems can be made more resilient to climate change (for example, (244)). Here we adapt the 10 World Health Organization (WHO) Europe Essential Public Health Operations (EPHOs) (245) and provide specific examples relevant to climate change and infectious disease. Other studies have used similar approaches (141, 143). The EPHOs are presented in Table 3, alongside examples pertinent to climate change.

Table 3. Ten Essential Public Health Operations (EPHOs) focusing on climate change and infectious diseases

Essential Public Health Operation (EPHO)	**Climate change and infectious disease examples**
1. Infectious disease surveillance	Sample sequencing data is becoming increasingly important for pathogen tracking (246) and also play a key role in identifying pathogen clusters (247). The UK has well-established surveillance systems for infectious diseases of concern in the environment, animals and humans. International surveillance conducted by the European Centre for Disease Prevention and Control (ECDC), EFSA and WHO informs public policy and prevention strategies. Laboratory services need strengthening to further understand climate impacts on disease; enhanced surveillance may be necessary for the climate-sensitive pathogens identified in this report or products originating in areas undergoing rapid environmental change (143).
2. Monitoring and responding to infectious disease outbreaks and threats	Monitoring for outbreaks and emerging threats potentially goes beyond surveillance. Collectively this effort is defined as epidemic intelligence, which combines surveillance information with event-based data such as the media, case reports and scientific publications (141) as well as investigation of potentially contaminated environments and materials. Strengthening such capacities is an important mechanism to prepare for climate threats to health.
3. Health protection	The field of health protection, including environmental, occupational, and food safety, is important to address the potential impacts of climate change. For example, within the UK, primary responsibility for food safety legally falls upon food business operators, who implement procedures based upon the Hazard Analysis and Critical Control Points principles (HACCP) for food protection and applies common hygiene requirements, with the policy and regulations being a responsibility of the Food Standards Agency (FSA). Within the water industry water safety plans perform a similar function. These standards and regulations provide capability within the food and water system but also need to be enhanced to cope with new emerging risks. Future approaches could include enhanced pathogen detection methods and One

Essential Public Health Operation (EPHO)	Climate change and infectious disease examples
	Health approaches to food safety (248). Other systems for detecting future threats include early warning systems which merge health, climate, veterinary and environmental data to indicate impending disease outbreaks (249). Horizon scanning is the identification of future hazards on the border of present thinking and planning (250).
4. Health Promotion including action to address social determinants and health inequity	Health promotion plays an important role in informing and empowering individuals to protect themselves from infectious diseases. However, it can be challenging to consistently affect human behaviour especially in relation to uncertain information (251). Challenges in health promotion arise when potential conflicts occur and these need to be carefully managed. Examples include the tension between food use by date (safety management) and wider objectives to reduce food waste, or balancing personalised choices and food safety, such as the consumption of uncooked seafood.
5. Infectious disease prevention, including early detection of illness	For effective disease prevention, surveillance is key, especially in situations undergoing rapid environmental changes. Early disease detection is vital and new rapid surveillance methods, including rapid antigen tests and calls to telehealth, potentially have a key role to play. Enhancements to surveillance are most useful if this data is integrated and rapidly disseminated. Schemes such as the FSA PATH-SAFE project, which aims to develop an enhanced UK surveillance system for tracking and monitoring foodborne disease and AMR in the environment and agri-food system and the FSA incident prevention strategies including root case analysis which aims to capture and communicate root causes of incidents including foodborne disease are welcome (252, 253). However, data sharing across international borders is key. These have changed following the UK's exit from the EU in 2020 (such as loss of full access to the EU Rapid Alert System for Food and Feed) that were replaced by the exchange of information with the International Food Safety Authority Network, (INFOSAN), and

Essential Public Health Operation (EPHO)	Climate change and infectious disease examples
	it is important that capacity is maintained. Possible examples include new data sharing agreements with ECDC (254) and EFSA, and enhanced use of INFOSAN.
6. Assuring governance for health and wellbeing	Good governance is essential to address current and future infectious disease threats. This requires efficient methods, processes and institutions which maintain accountability, quality and equity and have the capacity for early infectious disease prevention, detection and response. Internationally, such standards are mandated through the 2005 International Health Regulations (WHO 2005), which are legally binding for all WHO member states. Such structures are important to facilitate adaptation to changing infectious disease threats.
7. Assuring a sufficient and competent workforce	Human resource is essential, and through this chapter we have highlighted expertise such as sequencing, data science, risk assessment and early warning systems as being key. Interdisciplinary skills working within One Health are also important.
8. Sustainable organisational structures and financing	Minimising the impact of climate change upon infectious disease is potentially resource intensive. However, there is also the potential to increase efficient use of resources through prevention of diseases and associated human economic costs, food safety action and food safety product withdrawal and recall costs or reputational damage of food from environmental contamination. Appropriate and sustainable funding for these activities is key but challenging. Cost effectiveness is also important, and in many cases, public health funding is cost-effective in the short- and long-term (255).
9. Advocacy communication and social mobilisation for health	Reducing infectious disease burden is helped by effective communication across society between, for example individuals, businesses, UKHSA and the FSA. This is necessary to directly address infectious disease threats, but also to influence policy and sustain investment and multisectoral commitment in health protection. Climate change should be an important part of these discussions.

Essential Public Health Operation (EPHO)	Climate change and infectious disease examples
10. Advancing public health research to inform policy and practice	This chapter has detailed how climate change may affect infectious diseases and this plays a key role in helping to provide accessible information for practitioners and policy-makers. Public health research is key to the adaptation response, and this requires research across all sectors to support effective public health decision-making.

7.3 Conclusions

7.3.1 Research priorities

Most studies assessing the effect of weather on infectious diseases tend to focus on ambient air temperature, precipitation, relative humidity, and sunlight. For some infectious diseases, other meteorological factors may be more important, such as soil temperature, UV levels and dew point temperature. Evidence suggests that linking data on appropriately selected infectious disease incidence and weather parameters at a local level and determining a comparative conditional incidence across geographic boundaries can identify thresholds relating to disease occurrence and allow combinations of parameters to be compared together (for example, (256)). Further development of routine data linkage of surveillance data sets to all pathogens may allow the range of diseases impacted by climate change to be elucidated. As well as linking infectious disease to weather, methods need to be established for attributing the burden of infectious diseases due to climate change. Studies that quantify how much the burden of disease is ascribable to specific weather and climate factors are still uncommon, but it is important to quantify as climate change will have differential effects on different weather factors. To date, most research has focused on weather or meteorological variability as a proxy for longer-term climatic change, though weather remains an imperfect proxy and correlations with infectious disease incidence must be taken with caution when attempting to infer implications for how infectious diseases might respond to climate change, especially when inferring from studies based in different geographical locations.

This chapter has highlighted that for many infectious diseases, the exact transmission mechanism remains unclear, and this makes it challenging to estimate the likely impact of climate change. Even when transmission routes are relatively clear, holistic and multidisciplinary research incorporating all relevant biological and ecological influences are still uncommon. For instance, robust assessment of the impact of weather and climate on respiratory diseases would benefit from epidemiological studies integrated with research assessing the dynamics of aerosols and droplets emitted from individuals, in different meteorological settings. For foodborne diseases, the impact of food production or food chain factors and the interplay with human behavioural and societal influences requires further elucidation.

An additional priority is for more research to disentangle the individual, compounding and joint effects of climate (and climate change) on the multiple processes – including abiotic factors like land-use changes, demographic, and socio-economic factors – involved in the emergence, establishment, amplification and spread, of infectious diseases. Wildlife act as animal reservoir for many zoonotic diseases, and climate change-induced variations to the distribution and abundance of wildlife are also expected, which will have consequences for infectious disease burdens. For instance, changes in the distribution of rodent populations could lead to new populations being exposed to rodent-borne diseases. A second example is the association that has been identified between warming local temperatures, growing population densities and increasing antibiotic resistance in some common pathogens (*E. coli, Klebsiella pneumoniae* and *Staphylococcus aureus*), which would have serious consequences on global health (9).

As has been mentioned previously in this chapter, it is important to quantify baseline burdens of infection for climate-sensitive pathogens; this will help with understanding and predicting the impact that climate change could have in future. For instance, the symptoms of toxic exposure to HABs are similar to other infectious diseases, such as norovirus, and also mild and self-limiting, so it is possible that the impacts of HABs on human health are under-diagnosed (221). It is important, therefore, to improve estimations of the current burden of disease from HABs, and to assess the impact of climate on diseases to understand how incidence may change in future (217, 219, 225).

7.3.2 Implications for public health

As many emerging infectious diseases are zoonotic, there needs to be increased attention on the ways in which people interact with wild and domestic animals and animal products, and the impact this can have on disease risks. Strong collaborations are needed between other national and international public health agencies, and as some of the drivers for infectious diseases lie outside of public health, solutions will involve working with cross-governmental agencies, as well as farmers, food manufacturers and the public.

Early detection is important in responding to infectious disease, and a range of surveillance methods (such as sentinel surveillance, routine surveillance, epidemic intelligence and genomic data) all have roles to play. In analysing increasing large and complex surveillance data, artificial intelligence may play an important role. Global surveillance also needs to be strengthened so that emerging diseases can be detected and monitored domestically and globally.

The best defence against infectious disease emergence is dedicated reduction programmes against sporadic and outbreak disease. Risk assessments may need to be updated, however, (such as highlighting areas where droughts are increasingly likely), and policies (such as drinking and bathing water regulations) updated in light of future climatic change projections. Additionally, the availability of real time weather data enables dynamic risk assessment (such as on agricultural floodplains) to occur. However, climate change also implies increasing uncertainty and complexity of disease determinants, highlighting the importance not only of enhancing the speed of detection and response, but also techniques such as horizon scanning, which is the identification of hazards on the border of present thinking and planning. Response is equally key and requires efficient, rapid and multi-sector incident response procedures. Important recommendations and lessons with translatable insights for climate and health preparedness may emerge from the UK COVID-19 inquiry. In addition, responding to climate change impacts on infectious diseases requires a toolbox of interventions, many of which may not be the direct responsibility of public health agencies.

In designing interventions against climate change, the potential for co-benefits should be explored. For example, vaccination of animals such as livestock or poultry might not only reduce infectious disease risk, but also enhance animal welfare and have a positive influence on farm livelihoods. Second, vaccinations could be developed against a broad range of diseases that may be particularly sensitive to climate. Thus, active research is needed to target those climate-sensitive pathogens, as well as emerging pathogens that might be amenable to controls through vaccination.

Acronyms and abbreviations

Abbreviation	Meaning
AMR	anti-microbial resistance
ECDC	European Centre for Disease Prevention and Control
EFSA	European Food Safety Authority
EPHOs	Essential Public Health Operations
EU/EEA	European Union/European Economic Area
GHG	greenhouse gases
HABs	harmful algal blooms
RSV	respiratory syncytial virus
SP	shellfish poisoning
STEC	Shiga toxin-producing *Escherichia coli*

References

1. Shope R (1991). 'Global climate change and infectious diseases' Environmental Health Perspectives: volume 96, pages 171 to 174
2. McMichael AJ, Patz J, Kovats RS (1998). 'Impacts of global environmental change on future health and health care in tropical countries' British Medical Bulletin: volume 54, pages 475 to 488
3. Welch K, Shipp-Hilts A, Eidson M, Saha S, Zansky S (2019). '*Salmonella* and the changing environment: systematic review using New York State as a model' Journal of Water and Health: volume 17, pages 179 to 195
4. UK Chief Medical Officer (UKCMO) (2011). 'Infections and the rise of antimicrobial resistance' Annual Report of the Chief Medical Officer; Volume 2
5. Boxall ABA, Hardy A, Beulke S, Boucard T, Burgin L, Falloon PD, and others (2009). 'Impacts of climate change on indirect human exposure to pathogens and chemicals from agriculture' Environmental Health Perspectives: volume 117, pages 508 to 514
6. Office for National Statistics (ONS) (2018). 'Living longer: how our population is changing and why it matters'
7. O'Neill J (2016). 'Tackling drug-resistant infections globally: final report and recommendations'
8. Rodríguez-Verdugo A, Lozano-Huntelman N, Cruz-Loya M, Savage V, Yeh P (2020). 'Compounding effects of climate warming and antibiotic resistance' iScience: volume 23, page 101024
9. MacFadden DR, McGough SF, Fisman D, Santillana M, Brownstein JS (2018). 'Antibiotic resistance increases with local temperature' Nature Climate Change: volume 8, pages 510 to 514
10. Kaba HEJ, Kuhlmann E, Scheithauer S (2020). 'Thinking outside the box: association of antimicrobial resistance with climate warming in Europe – a 30-country observational study' International Journal of Hygiene and Environmental Health: volume 223, pages 151 to 158
11. Simmering JE, Cavanaugh JE, Polgreen LA, Polgreen PM (2018). 'Warmer weather as a risk factor for hospitalisations due to urinary tract infections' Epidemiology and Infection: volume 146, pages 386 to 393
12. Blot K, Hammami N, Blot S, Vogelaers D, Lambert ML (2022). 'Seasonal variation of hospital-acquired bloodstream infections: a national cohort study' Infection Control and Hospital Epidemiology: volume 43, pages 205 to 211
13. Mutuku C, Gazdag Z, Melegh S (2022). 'Occurrence of antibiotics and bacterial resistance genes in wastewater: resistance mechanisms and antimicrobial resistance control approaches' World Journal of Microbiology and Biotechnology: volume 38, page 152
14. Magnano San Lio R, Favara G, Maugeri A, Barchitta M, Agodi A (2023). 'How antimicrobial resistance is linked to climate change: an overview of 2 intertwined global challenges' International Journal of Environmental Research and Public Health: volume 20, page 1681

15. Burnham JP (2021). 'Climate change and antibiotic resistance: a deadly combination' Therapeutic Advances in Infectious Disease: volume 8, pages 1 to 7
16. Department of Health (DH) (2002). 'Health Effects of Climate Change in the UK' 2002
17. Health Protection Agency (HPA) (2008). 'Health Effect of Climate Change in the UK 2008' Kovats S, editor
18. HPA (2012). 'Health Effects of Climate Change in the UK 2012' Vardoulakis S, Heaviside C, editors
19. McMichael AJ (2012). 'Insights from past millennia into climatic impacts on human health and survival' Proceedings of the National Academy of Sciences: volume 109, page 4730 to 4737
20. European Economic Area (EEA) (2022). 'Climate change as a threat to health and well-being in Europe: focus on heat and infectious diseases'
21. Pörtner HO, Roberts DC, Adams H, Adelekan I, Adler C, Adrian R, and others (2022). 'Technical summary' Pörtner HO, Roberts DC, Tignor M, Poloczanska ES, Mintenbeck K, Alegría A, and others, editors. Climate Change 2022: Impacts, Adaptation and Vulnerability. Contribution of Working Group II to the Sixth Assessment Report of the Intergovernmental Panel on Climate Change
22. Kovats S, Brisley R (2021). 'Health, communities and the built environment' In: Betts RA, Haward AB, Pearson K V, editors. The Third Climate Change Risk Assessment Technical Report
23. Tam C, Viviani L, Adak B, Bolton E, Dodds J, Cowden J, and others (2012). 'The second study of infectious intestinal disease in the community (IID2 Study)
24. Semenza JC, Herbst S, Rechenburg A, Suk JE, Höser C, Schreiber C, and others (2012). 'Climate change impact assessment of food and waterborne diseases' Critical Reviews in Environmental Science and Technology: volume 42, pages 857 to 890
25. Semenza JC, Rocklöv J, Ebi KL (2022). 'Climate change and cascading risks from infectious disease' Infectious Diseases and Therapy: volume 11, pages 1371 to 1390
26. Daniel N, Casadevall N, Sun P, Sugden D, Aldin V (2020). 'The burden of foodborne disease in the UK 2018'
27. Nichols G, Lake I, Heaviside C (2018). 'Climate change and water-related infectious diseases' Atmosphere: volume 9, page 385
28. Levy K, Smith SM, Carlton EJ (2018). 'Climate change impacts on waterborne diseases: moving toward designing interventions' Current Environmental Health Reports: volume 5, pages 272 to 282
29 Levy K, Woster AP, Goldstein RS, Carlton EJ (2016). 'Untangling the impacts of climate change on waterborne diseases: a systematic review of relationships between diahheal diseases and temperature, rainfall, flooding and drought' Environmental Science and Technology: volume 50, pages 4905 to 4922
30. Mena KD, Gerba CP (2009). 'Waterborne adenovirus' In: Whitacre DM, editor. Reviews of Environmental Contamination and Toxicology
31. Price RHM, Graham C, Ramalingam S (2019). 'Association between viral seasonality and meteorological factors' Scientific Reports: volume 9, page 929
32. Dey SK, Hoq I, Okitsu S, Hayakawa S, Ushijima H (2013). 'Prevalence, seasonality, and

peak age of infection of enteric adenoviruses in Japan, 1995-2009' Epidemiology and Infection: volume 141, pages 958 to 960

33. Shih YJ, Chen JS, Chen YJ, Yang PY, Kuo YJ, Chen TH, and others (2021). 'Impact of heavy precipitation events on pathogen occurrence in estuarine areas of the Puzi River in Taiwan' PLOS ONE: volume 16, page e0256266
34. Celik C, Gozel MG, Turkay H, Bakici MZ, Güven AS, Elaldi N (2015). 'Rotavirus and adenovirus gastroenteritis: time series analysis' Pediatrics International: volume 57, pages 590 to 596
35. Ntumvi NF, Diffo JLD, Tamoufe U, Ndze VN, Takuo JM, Mouiche MMM, and others (2021). 'Evaluation of bat adenoviruses suggests co-evolution and host roosting behaviour as drivers for diversity' Microbial Genomics: volume 7, page 000561
36. De Benedictis P, Schultz-Cherry S, Burnham A, Cattoli G (2011). 'Astrovirus infections in humans and animals: molecular biology, genetic diversity, and interspecies transmissions' Infection, Genetics and Evolution: volume 11, pages 1529 to 1544
37. UK Health Security Agency (UKHSA) (2022). 'National norovirus and rotavirus report, week 6 report: data up to week 4 (29 January 2023)'
38. Yoneda M, Nakano M, Sugimoto D, Inada M, Fujitani M, Kitahori Y (2017). 'Epidemiological characteristics of sapovirus and human astrovirus detected among children in Nara Prefecture, Japan, during the 2009/2010 to 2014/2015 seasons' Japanese Journal of Infectious Diseases: volume 70, pages 87 to 91
39. Dennehy PH, Nelson SM, Spangenberger S, Noel JS, Monroe SS, Glass RI (2001). 'A prospective case-control study of the role of astrovirus in acute diarrhea among hospitalized young children' The Journal of Infectious Diseases: volume 184, pages 10 to 15
40. Colston J, Paredes Olortegui M, Zaitchik B, Peñataro Yori P, Kang G, Ahmed T, and others (2020). 'Pathogen-specific impacts of the 2011-2012 La Niña-associated floods on enteric infections in the MAL-ED Peru cohort: a comparative interrupted time series analysis' International Journal of Environmental Research and Public Health: volume 17, page 487
41. World Health Organization (WHO) (2018). 'Influenza (seasonal)'
42. UKHSA (2022). 'Surveillance of influenza and other seasonal respiratory viruses in winter 2021 to 2022'
43. Lowen AC, Mubareka S, Steel J, Palese P (2007). 'Influenza virus transmission is dependent on relative humidity and temperature' PLoS Pathogens: volume 3, pages 1470 to 1476
44. Lowen AC, Steel J (2014). 'Roles of humidity and temperature in shaping influenza seasonality' Journal of Virology: volume 88, pages 7692 to 7695
45. Shaman J, Kohn M (2009). 'Absolute humidity modulates influenza survival, transmission, and seasonality' Proceedings of the National Academy of Sciences: volume 106, pages 3243 to 3248
46. Chong KC, Lee TC, Bialasiewicz S, Chen J, Smith DW, Choy WSC, and others (2020). 'Association between meteorological variations and activities of influenza A and B across different climate zones: a multi-region modelling analysis across the globe' Journal of

Infection: volume 80, pages 84 to 98
47. Barreca AI, Shimshack JP (2012). 'Absolute humidity, temperature, and influenza mortality: 30 years of county-level evidence from the United States' American Journal of Epidemiology: volume 176, pages S114 to S122
48. Caini S, Spreeuwenberg P, Donker G, Korevaar J, Paget J (2018). 'Climatic factors and long-term trends of influenza-like illness rates in The Netherlands, 1970 to 2016' Environmental Research: volume 167, pages 307 to 313
49. Shaman J, Pitzer VE, Viboud C, Grenfell BT, Lipsitch M (2010). 'Absolute humidity and the seasonal onset of influenza in the continental United States' PLoS Biology: volume 8, page e1000316
45. Ianevski A, Zusinaite E, Shtaida N, Kallio-Kokko H, Valkonen M, Kantele A, and others (2019). 'Low temperature and low UV indexes correlated with peaks of influenza virus activity in northern Europe during 2010 to 2018' Viruses: volume 11, page 207
46. Ballester J, Rodó X, Robine JM, Herrmann FR (2016). 'European seasonal mortality and influenza incidence due to winter temperature variability' Nature Climate Change: volume 6, pages 927 to 930
47. Towers S, Chowell G, Hameed R, Jastrebski M, Khan M, Meeks J, and others (2013). 'Climate change and influenza: the likelihood of early and severe influenza seasons following warmer than average winters' PLoS Currents: volume 5, page ecurrents.flu.3679b56a3a5313dc7c043fb944c6f138
53. Tam CC, Rodrigues LC, Viviani L, Dodds JP, Evans MR, Hunter PR, and others (2012). 'Longitudinal study of infectious intestinal disease in the UK (IID2 study): incidence in the community and presenting to general practice' Gut: volume 61, pages 69 to 77
54. Lopman B, Armstrong B, Atchison C, Gray JJ (2009). 'Host, weather and virological factors drive norovirus epidemiology: time-series analysis of laboratory surveillance data in England and Wales' PLoS ONE: volume 4, page e6671
55. Chiu SC, Hu SC, Liao LM, Chen YH, Lin JH (2022). 'Norovirus genogroup II epidemics and the potential effect of climate change on norovirus transmission in Taiwan' Viruses: volume 14, page 641
56. Wang P, Goggins WB, Chan EYY (2018). 'A time-series study of the association of rainfall, relative humidity and ambient temperature with hospitalizations for rotavirus and norovirus infection among children in Hong Kong' Science of The Total Environment: volume 643, pages 414 to 422
57. Bruggink LD, Marshall JA (2010). 'The incidence of norovirus-associated gastroenteritis outbreaks in Victoria, Australia (2002 to 2007) and their relationship with rainfall' International Journal of Environmental Research and Public Health: volume 7, pages 2822 to 2827
58. Li Y, Wang X, Blau DM, Caballero MT, Feikin DR, Gill CJ, and others (2022). 'Global, regional, and national disease burden estimates of acute lower respiratory infections due to respiratory syncytial virus in children younger than 5 years in 2019: a systematic analysis' Lancet: volume 399, pages 2047 to 2064
59. UKHSA (2021). 'Respiratory syncytial virus (RSV): symptoms, transmission, prevention, treatment'

60. Baker RE, Mahmud AS, Wagner CE, Yang W, Pitzer VE, Viboud C, and others (2019). 'Epidemic dynamics of respiratory syncytial virus in current and future climates' Nature Communications: volume 10, page 5512
61. Donaldson GC (2006). 'Climate change and the end of the respiratory syncytial virus season' Clinical Infectious Diseases: volume 42, pages 677 to 679
62. Crawford SE, Ramani S, Tate JE, Parashar UD, Svensson L, Hagbom M, and others (2017). 'Rotavirus infection' Nature Reviews Disease Primers: volume 3, page 17083
63. Public Health England (PHE) (2019). 'Rotavirus: guidance, data and analysis'
64. Tate JE, Burton AH, Boschi-Pinto C, Parashar UD, World Health Organization–Coordinated Global Rotavirus Surveillance Network (2016). 'Global, regional, and national estimates of rotavirus mortality in children under 5 years of age, 2000 to 2013' Clinical Infectious Diseases: volume 62, pages S96 to S105
65. Atchison CJ, Stowe J, Andrews N, Collins S, Allen DJ, Nawaz S, and others (2016). 'Rapid declines in age group–specific rotavirus infection and acute gastroenteritis among vaccinated and unvaccinated individuals within one year of rotavirus vaccine introduction in England and Wales' The Journal of Infectious Diseases: volume 213, pages 243 to 249
66. PHE (2018). 'Rotavirus data 2007 to 2016'
67. Aliabadi N, Tate JE, Haynes AK, Parashar UD (2015). 'Sustained decrease in laboratory detection of rotavirus after implementation of routine vaccination: United States, 2000 to 2014' Morbidity and Mortality Weekly Report: volume 64, pages 337 to 342
68. Morral-Puigmal C, Martínez-Solanas È, Villanueva CM, Basagaña X (2018). 'Weather and gastrointestinal disease in Spain: a retrospective time series regression study' Environment International: volume 121, pages 649 to 657
69. D'Souza RM, Hall G, Becker NG (2008). 'Climatic factors associated with hospitalizations for rotavirus diarrhoea in children under 5 years of age' Epidemiology and Infection: volume 136, pages 56 to 64
70. Kraay ANM, Man O, Levy MC, Levy K, Ionides E, Eisenberg JNS (2020). 'Understanding the impact of rainfall on diarrhea: testing the concentration-dilution hypothesis using a systematic review and meta-analysis' Environmental Health Perspectives: volume 128, page 126001
71. Turcios RM, Curns AT, Holman RC, Pandya-Smith I, LaMonte A, Bresee JS, and others (2006). 'Temporal and geographic trends of rotavirus activity in the United States, 1997-2004' The Pediatric Infectious Disease Journal: volume 25, pages 451 to 454
72. Colston JM, Zaitchik B, Kang G, Peñataro Yori P, Ahmed T, Lima A, and others (2019). 'Use of earth observation-derived hydrometeorological variables to model and predict rotavirus infection (MAL-ED): a multisite cohort study' Lancet Planetary Health: volume 3, pages e248 to e258
73. Nichols GL, Lo Iacono G (2019). 'Examining the influence of weather on rotavirus infection' The Lancet Planetary Health: volume 3, pages e236 to e237
74. Becker-Dreps S, Bucardo F, Vinjé J (2019). 'Sapovirus: an important cause of acute gastroenteritis in children' The Lancet Child and Adolescent Health: volume 3, pages 758 to 759
75. Diez Valcarce M, Kambhampati AK, Calderwood LE, Hall AJ, Mirza SA, Vinjé J (2021).

'Global distribution of sporadic sapovirus infections: a systematic review and meta-analysis' PLoS ONE: volume 16, page e0255436
76. de Oliveira-Tozetto S, Santiso-Bellón C, Ferrer-Chirivella JM, Navarro-Lleó N, Vila-Vicent S, Rodríguez-Díaz J, and others (2021). 'Epidemiological and genetic characterization of sapovirus in patients with acute gastroenteritis in Valencia (Spain)' Viruses: volume 13, page 184
77. Dey SK, Phathammavong O, Nguyen TD, Thongprachum A, Chan-It W, Okitsu S, and others (2012). 'Seasonal pattern and genotype distribution of sapovirus infection in Japan, 2003 to 2009' Epidemiology and Infection: volume 140, pages 74 to 77
78. European Food Standards Agency (EFSA), European Centre for Disease Prevention and Control (ECDC) (2021). 'The European Union One Health 2020 zoonoses report' EFSA Journal: volume 19, page 6971
79. WHO (2020). '*Campylobacter*'
80. UKHSA (2023). 'Campylobacter data 2010 to 2019'
81. Lake IR, Colón-González FJ, Takkinen J, Rossi M, Sudre B, Dias JG, and others (2019). 'Exploring *Campylobacter* seasonality across Europe using The European Surveillance System (TESSy), 2008 to 2016' Eurosurveillance: volume 24, page 1800028
82. Kuhn KG, Nygård KM, Guzman-Herrador B, Sunde LS, Rimhanen-Finne R, Trönnberg L, and others (2020). '*Campylobacter* infections expected to increase due to climate change in Northern Europe' Scientific Reports: volume 10, page 13874
83. Rushton SP, Sanderson RA, Diggle PJ, Shirley MDF, Blain AP, Lake I, and others (2019). 'Climate, human behaviour or environment: individual-based modelling of *Campylobacter* seasonality and strategies to reduce disease burden'. Journal of Translational Medicine: volume 17, page 34
84. Hellberg RS, Chu E (2016). 'Effects of climate change on the persistence and dispersal of foodborne bacterial pathogens in the outdoor environment: a review' Critical Reviews in Microbiology: volume 42, pages 548 to 572
85. Cousins M, Sargeant JM, Fisman D, Greer AL (2019). 'Modelling the transmission dynamics of *Campylobacter* in Ontario, Canada, assuming house flies, *Musca domestica*, are a mechanical vector of disease transmission' Royal Society Open Science: volume 6, page 181394
86. Nichols GL (2005). 'Fly transmission of *Campylobacter*' Emerging Infectious Diseases: volume 11, pages 361 to 364
87. Jones K (2001). 'Campylobacters in water, sewage and the environment' Journal of Applied Microbiology: volume 90, pages 68S to 79S
88. Milazzo A, Giles LC, Zhang Y, Koehler AP, Hiller JE, Bi P (2017). 'The effects of ambient temperature and heatwaves on daily *Campylobacter* cases in Adelaide, Australia, 1990 to 2012' Epidemiology and Infection: volume 145, pages 2603 to 2610
89. Schijven J, Bouwknegt M, de Roda Husman AM, Rutjes S, Sudre B, Suk JE, and others (2013). 'A decision support tool to compare waterborne and foodborne infection and/or illness risks associated with climate change' Risk Analysis: volume 33, pages 2154 to 2167
90. Djennad A, Lo Iacono G, Sarran C, Lane C, Elson R, Höser C, and others (2019).

'Seasonality and the effects of weather on *Campylobacter* infections' BMC Infectious Diseases: volume 19, page 255

91. Lo Iacono G, Cook A, Derks G, Fleming LE, French N, Gillingham EL, and others (2023). 'A mathematical, classical stratification modelling approach to disentangling the impact of weather on infectious diseases: a case study using spatio-temporally disaggregated Campylobacter surveillance data for England and Wales' PLoS Computational Biology (In press)
92. Patrick ME, Christiansen LE, Wainø M, Ethelberg S, Madsen H, Wegener HC (2004). 'Effects of climate on incidence of *Campylobacter* spp. in humans and prevalence in broiler flocks in Denmark' Applied and Environmental Microbiology: volume 70, pages 7474 to 7480
93. Lal A, Lill AWT, Mcintyre M, Hales S, Baker MG, French NP (2015). 'Environmental change and enteric zoonoses in New Zealand: a systematic review of the evidence' Australian and New Zealand Journal of Public Health: volume 39, pages 63 to 68
94. Park MS, Park KH, Bahk GJ (2018). 'Combined influence of multiple climatic factors on the incidence of bacterial foodborne diseases' Science of The Total Environment: volume 610 to 611, pages 10 to 16
95. Bennett SD, Walsh KA, Gould LH (2013). 'Foodborne disease outbreaks caused by *Bacillus cereus*, *Clostridium perfringens*, and *Staphylococcus aureus*: United States, 1998 to 2008' Clinical Infectious Diseases: volume 57, pages 425 to 433
96. Bhattacharya A, Shantikumar S, Beaufoy D, Allman A, Fenelon D, Reynolds K, and others (2020). 'Outbreak of *Clostridium perfringens* food poisoning linked to leeks in cheese sauce: an unusual source' Epidemiology and Infection: volume 148, page e43
97. Oh EJ, Kim JM, Kim JK (2021). 'Interrelationship between climatic factors and incidence of FBD caused by *Clostridioides difficile* toxin B, *Clostridium perfringens*, *Campylobacter* spp., and *Escherichia coli* O157:H7' Environmental Science and Pollution Research: volume 28, pages 44538 to 44546
98. Park MS, Park KH, Bahk GJ (2018). 'Interrelationships between multiple climatic factors and incidence of foodborne diseases' International Journal of Environmental Research and Public Health: volume 15, page 2482
99. Dolan GP, Foster K, Lawler J, Amar C, Swift C, Aird H, and others (2016). 'An epidemiological review of gastrointestinal outbreaks associated with *Clostridium perfringens*, North East of England, 2012 to 2014' Epidemiology and Infection: volume 144, pages 1386 to 1393
100. UKHSA (2022). '*Clostridioides difficile*: guidance, data and analysis'
101. Office for Health Improvement and Disparities (OHID) (2023). 'Fingertips: public health data'
102. Rodriguez C, Bouchafa L, Soumillion K, Ngyuvula E, Taminiau B, van Broeck J, and others (2019). 'Seasonality of *Clostridium difficile* in the natural environment' Transboundary and Emerging Diseases: volume 66, pages 2440 to 2449
103. Furuya-Kanamori L, McKenzie SJ, Yakob L, Clark J, Paterson DL, Riley T V, and others (2015). '*Clostridium difficile* infection seasonality: patterns across hemispheres and continents: a systematic review' PLoS ONE: volume 10, page e0120730

104. Lin CJ, Wade TJ, Hilborn ED (2015). 'Flooding and *Clostridium difficile* infection: a case-crossover analysis' International Journal of Environmental Research and Public Health: volume 12, pages 6948 to 6964
105. Furuya-Kanamori L, Robson J, Soares Magalhães RJ, Yakob L, McKenzie SJ, Paterson DL, and others (2014). 'A population-based spatio-temporal analysis of *Clostridium difficile* infection in Queensland, Australia over a 10-year period' Journal of Infection: volume 69, pages 447 to 455
106. van Heijnsbergen E, de Roda Husman AM, Lodder WJ, Bouwknegt M, Docters van Leeuwen AE, Bruin JP, and others (2014). 'Viable *Legionella pneumophila* bacteria in natural soil and rainwater puddles' Journal of Applied Microbiology: volume 117, pages 882 to 890
107. Collins S, Stevenson D, Bennett A, Walker J (2017). 'Occurrence of *Legionella* in UK household showers' International Journal of Hygiene and Environmental Health: volume 220, pages 401 to 406
108. Ishimatsu S, Miyamoto H, Hori H, Tanaka I, Yoshida S (2001). 'Sampling and detection of *Legionella pneumophila* aerosols generated from an industrial cooling tower' The Annals of Occupational Hygiene: volume 45, pages 421 to 427
109. Fields BS, Benson RF, Besser RE (2002). '*Legionella* and Legionnaires' disease: 25 years of investigation' Clinical Microbiology eviews: volume 15, pages 506 to 526
110. PHE (2020). 'Monthly *Legionella* report: December 2019'
111. ECDC (2022). 'Legionnaires' diseases - annual epidemiological report for 2020'
112. Fisman DN, Lim S, Wellenius GA, Johnson C, Britz P, Gaskins M, and others (2005). 'It's not the heat, it's the humidity: wet weather increases legionellosis risk in the greater Philadelphia metropolitan area' The Journal of Infectious Diseases: volume 192, pages 2066 to 2073
113. Fukushima S, Hagiya H, Otsuka Y, Koyama T, Otsuka F (2021). 'Trends in the incidence and mortality of legionellosis in Japan: a nationwide observational study, 1999-2017' Scientific Reports: volume 11, page 7246
114. Brandsema PS, Euser SM, Karagiannis I, Den Boer JW, Van Der Hoek W (2014). 'Summer increase of Legionnaires' disease 2010 in The Netherlands associated with weather conditions and implications for source finding' Epidemiology and Infection: volume 142, pages 2360 to 2371
115. Garcia-Vidal C, Labori M, Viasus D, Simonetti A, Garcia-Somoza D, Dorca J, and others (2013). 'Rainfall is a risk factor for sporadic cases of *Legionella pneumophila* pneumonia' PLoS One: volume 8, page e61036
116. Hicks LA, Rose Jr CE, Fields BS, Drees ML, Engel JP, Jenkins PR, and others (2007). 'Increased rainfall is associated with increased risk for legionellosis' Epidemiology and Infection: volume 135, pages 811 to 817
117. Beauté J, Sandin S, Uldum SA, Rota MC, Brandsema P, Giesecke J, and others (2016). 'Short-term effects of atmospheric pressure, temperature, and rainfall on notification rate of community-acquired Legionnaires' disease in 4 European countries' Epidemiology and Infection: volume 144, pages 3483 to 3493
118. Walker JT (2018). 'The influence of climate change on waterborne disease and

Legionella: a review' Perspectives in Public Health: volume 138, pages 282 to 286
119. Guerra MA (2013). 'Leptospirosis: public health perspectives' Biologicals: volume 41, pages 295 to 297
120. Haake DA, Levett PN (2015). 'Leptospirosis in humans' Current Topics in Microbiology and Immunology: volume 387, pages 65 to 97
121. Costa F, Hagan JE, Calcagno J, Kane M, Torgerson P, Martinez-Silveira MS, and others (2015). 'Global morbidity and mortality of Leptospirosis: a systematic review' PLoS Neglected Tropical Diseases: volume 9, page e0003898
122. ECDC (2022). 'Leptospirosis: annual epidemiological report for 2019'
123. UKHSA (2023). 'Common animal-associated infections (England and Wales): fourth quarter 2022'
124. PHE (2020). 'Common animal-associated infections (England and Wales): fourth quarter 2019'
125. Dechet AM, Parsons M, Rambaran M, Mohamed-Rambaran P, Florendo-Cumbermack A, Persaud S, and others (2012). 'Leptospirosis outbreak following severe flooding: a rapid assessment and mass prophylaxis campaign; Guyana, January to February 2005' PLoS ONE: volume 7, page e39672
126. Naing C, Reid SA, Aye SN, Htet NH, Ambu S (2019). 'Risk factors for human leptospirosis following flooding: a meta-analysis of observational studies' PLoS ONE: volume 14, page e0217643
127. Suk JE, Vaughan EC, Cook RG, Semenza JC (2020). 'Natural disasters and infectious disease in Europe: a literature review to identify cascading risk pathways' European Journal of Public Health: volume 30, pages 928 to 935
128. Kouadio IK, Aljunid S, Kamigaki T, Hammad K, Oshitani H (2012). 'Infectious diseases following natural disasters: prevention and control measures' Expert Review of Anti-Infective Therapy: volume 10, pages 95 to 104
129. Aoki T, Koizumi N, Watanabe H (2001). 'A case of leptospirosis probably caused by drinking contaminated well-water after an earthquake' Japanese Journal of Infectious Diseases: volume 54, pages 243 to 244
130. Pijnacker R, Goris MGA, te Wierik MJM, Broens EM, van der Giessen JWB, de Rosa M, and others (2016). 'Marked increase in leptospirosis infections in humans and dogs in the Netherlands, 2014' Eurosurveillance: volume 21, page 30211
131. Chen IC, Hill JK, Ohlemüller R, Roy DB, Thomas CD (2011). 'Rapid range shifts of species associated with high levels of climate warming' Science: volume 333, pages 1024 to 1026
132. Lau CL, Smythe LD, Craig SB, Weinstein P (2010). 'Climate change, flooding, urbanisation and leptospirosis: fuelling the fire?' Transactions of The Royal Society of Tropical Medicine and Hygiene: volume 104, pages 631 to 638
133. UKHSA (2022). 'Listeriosis in England and Wales: summary for 2020'
134. Havelaar AH, Kirk MD, Torgerson PR, Gibb HJ, Hald T, Lake RJ, and others (2015). 'World Health Organization global estimates and regional comparisons of the burden of foodborne disease in 2010' PLoS Medicine: volume 12, page e1001923
135. EFSA, ECDC (2021). 'The European Union One Health 2019 Zoonoses Report'

136. Lal A, Hales S, Kirk M, Baker MG, French NP (2016). 'Spatial and temporal variation in the association between temperature and salmonellosis in NZ' Australian and New Zealand Journal of Public Health: volume 40, pages 165 to 169
137. Jiang C, Shaw KS, Upperman CR, Blythe D, Mitchell C, Murtugudde R, and others (2015). 'Climate change, extreme events and increased risk of salmonellosis in Maryland, USA: evidence for coastal vulnerability' Environment International: volume 83, pages 58 to 62
138. Akil L, Ahmad HA, Reddy RS (2014). 'Effects of climate change on *Salmonella* infections' Foodborne Pathogens and Disease: volume 11, pages 974 to 980
139. Miraglia M, Marvin HJP, Kleter GA, Battilani P, Brera C, Coni E, and others (2009). 'Climate change and food safety: an emerging issue with special focus on Europe' Food and Chemical Toxicology: volume 47, pages 1009 to 1021
140. Liu C, Hofstra N, Franz E (2013). 'Impacts of climate change on the microbial safety of pre-harvest leafy green vegetables as indicated by *Escherichia coli* O157 and *Salmonella* spp.' International Journal of Food Microbiology: volume 163, pages 119 to 128
141. Semenza JC, Menne B (2009). 'Climate change and infectious diseases in Europe' The Lancet Infectious Diseases: volume 9, pages 365 to 375
142. Milazzo A, Giles LC, Zhang Y, Koehler AP, Hiller JE, Bi P (2016). 'The effect of temperature on different *Salmonella* serotypes during warm seasons in a Mediterranean climate city, Adelaide, Australia' Epidemiology and Infection: volume 144, pages 1231 to 1240
143. Lake IR, Barker GC (2018). 'Climate change, foodborne pathogens and illness in higher-income countries' Current Environmental Health Reports: volume 5, pages 187 to 196
144. Lake IR, Hooper L, Abdelhamid A, Bentham G, Boxall ABA, Draper A, and others (2012). 'Climate change and food security: health impacts in developed countries' Environmental Health Perspectives: volume 120, pages 1520 to 1526
145. Lo Iacono G, Armstrong B, Fleming LE, Elson R, Kovats S, Vardoulakis S, and others (2017). 'Challenges in developing methods for quantifying the effects of weather and climate on water-associated diseases: a systematic review' PLoS Neglected Tropical Diseases: volume 11, page e0005659
146. Lake IR, Gillespie IA, Bentham G, Nichols GL, Lane C, Adak GK, and others (2009). 'A re-evaluation of the impact of temperature and climate change on foodborne illness' Epidemiology and Infection: volume 137, pages 1538 to 1547
147. Lake IR (2017). 'Foodborne disease and climate change in the United Kingdom' Environmental Health: volume 16, page 117
148. Taylor CM (2008). 'Enterohaemorrhagic *Escherichia coli* and *Shigella dysenteriae* type 1-induced haemolytic uraemic syndrome' Pediatric Nephrology: volume 23, pages 1425 to 1431
149. Kernéis S, Guerin PJ, von Seidlein L, Legros D, Grais RF (2009). 'A look back at an ongoing problem: *Shigella dysenteriae* type 1 epidemics in refugee settings in Central Africa (1993 to 1995)' PLoS ONE: volume 4, page e4494
150. Mattison CP, Calderwood LE, Marsh ZA, Wikswo ME, Balachandran N, Kambhampati AK, and others (2022). 'Childcare and school acute gastroenteritis outbreaks: 2009 to 2020'

Pediatrics: volume 150, page e2021056002
151. McCall B, Stafford R, Cherian S, Heel K, Smith H, Corones N, and others (2000). 'An outbreak of multi-resistant *Shigella sonnei* in a long-stay geriatric nursing centre' Communicable Diseases Intelligence: volume 24, pages 272 to 275
152. ECDC (2022). 'Shigellosis: annual epidemiological report for 2019'
153. Chen CC, Lin CY, Chen KT (2019). 'Epidemiologic features of shigellosis and associated climatic factors in Taiwan' Medicine: volume 98, page e16928
154. Chua PLC, Ng CFS, Tobias A, Seposo XT, Hashizume M (2022). 'Associations between ambient temperature and enteric infections by pathogen: a systematic review and meta-analysis' The Lancet Planetary Health: volume 6, pages e202 to e218
155. Hines JZ, Jagger MA, Jeanne TL, West N, Winquist A, Robinson BF, and others (2018). 'Heavy precipitation as a risk factor for shigellosis among homeless persons during an outbreak - Oregon, 2015 to 2016' Journal of Infection: volume 76, pages 280 to 285
156. Chua PLC, Huber V, Ng CFS, Seposo XT, Madaniyazi L, Hales S, and others (2021). 'Global projections of temperature-attributable mortality due to enteric infections: a modelling study' The Lancet Planetary Health: volume 5, pages e436 to e445
157. PHE (2017). 'Shiga toxin-producing *Escherichia coli* (STEC): symptoms, how to avoid, how to treat'
158. Gyles CL (2007). 'Shiga toxin-producing *Escherichia coli*: an overview' Journal of Animal Science: volume 85, pages E45 to E62
159. Mughini-Gras L, van Pelt W, van der Voort M, Heck M, Friesema I, Franz E (2018). 'Attribution of human infections with Shiga toxin-producing *Escherichia coli* (STEC) to livestock sources and identification of source-specific risk factors, The Netherlands (2010 to 2014)' Zoonoses and Public Health: volume 65, pages e8 to e22
160. Kintz E, Brainard J, Hooper L, Hunter P (2017). 'Transmission pathways for sporadic Shiga-toxin producing *E. coli* infections: a systematic review and meta-analysis' International Journal of Hygiene and Environmental Health: volume 220, pages 57 to 67
161. Pires SM, Majowicz S, Gill A, Devleesschauwer B (2019). 'Global and regional source attribution of Shiga toxin-producing *Escherichia coli* infections using analysis of outbreak surveillance data' Epidemiology and Infection: volume 147, page e236
162. ECDC (2021). 'Shiga toxin-producing *Escherichia coli* (STEC) infection. Annual epidemiology report for 2019'
163. Philipsborn R, Ahmed SM, Brosi BJ, Levy K (2016). 'Climatic drivers of diarrheagenic *Escherichia coli* incidence: a systematic review and meta-analysis' The Journal of Infectious Diseases: volume 214, pages 6 to 15
164. UKHSA (2022). 'Shiga toxin-producing *Escherichia coli* (STEC) data: 2019'
165. WHO (2018). '*E. coli* factsheet'
166. Acquaotta F, Ardissino G, Fratianni S, Perrone M (2017). 'Role of climate in the spread of shiga toxin-producing *Escherichia coli* infection among children' International Journal of Biometeorology: volume 61, pages 1647 to 1655
167. Feliciano R, Boué G, Mohssin F, Hussaini MM, Membré JM (2021). 'Probabilistic modelling of *Escherichia coli* concentration in raw milk under hot weather conditions' Food Research International: volume 149, page 110679

168. Baker-Austin C, Oliver JD, Alam M, Ali A, Waldor MK, Qadri F, and others (2018). '*Vibrio* spp. infections' Nature Reviews Disease Primers: volume 4, pages 1 to 19
169. Brumfield KD, Usmani M, Chen KM, Gangwar M, Jutla AS, Huq A, and others (2021). 'Environmental parameters associated with incidence and transmission of pathogenic *Vibrio* spp.' Environmental Microbiology: volume 23, pages 7314 to 7340
170. Maggi P, Carbonara S, Fico C, Santantonio T, Romanelli C, Sforza E, and others (1997). 'Epidemiological, clinical and therapeutic evaluation of the Italian cholera epidemic in 1994' European Journal of Epidemiology: volume 13, pages 95 to 97
171. Tantillo GM, Fontanarosa M, Di Pinto A, Musti M (2004). 'Updated perspectives on emerging vibrios associated with human infections' Letters in Applied Microbiology: volume 39, pages 117 to 126
165. Iwamoto M, Ayers T, Mahon BE, Swerdlow DL (2010). 'Epidemiology of seafood-associated infections in the United States' Clinical Microbiology Reviews: volume 23, pages 399 to 411
166. Ford CL, Powell A, Lau DYL, Turner AD, Dhanji-Rapkova M, Martinez-Urtaza J, and others (2020). 'Isolation and characterization of potentially pathogenic *Vibrio* species in a temperate, higher latitude hotspot' Environmental Microbiology Reports: volume 12, pages 424 to 434
174. UKHSA (2022). 'Travel-associated infections in England, Wales and Northern Ireland: 2020 and 2021'
175. Baker-Austin C, Trinanes J, Gonzalez-Escalona N, Martinez-Urtaza J (2017). 'Non-cholera vibrios: the microbial barometer of climate change' Trends in Microbiology: volume 25, pages 76 to 84
176. Martinez-Urtaza J, Simental L, Velasco D, DePaola A, Ishibashi M, Nakaguchi Y, and others (2005). 'Pandemic *Vibrio parahaemolyticus* O3:K6, Europe' Emerging Infectious Diseases: volume 11, page 1319
177. Baker-Austin C, Trinanes JA, Taylor NGH, Hartnell R, Siitonen A, Martinez-Urtaza J (2013). 'Emerging *Vibrio* risk at high latitudes in response to ocean warming' Nature Climate Change: volume 3, pages 73 to 77
178. Martinez-Urtaza J, Trinanes J, Abanto M, Lozano-Leon A, Llovo-Taboada J, Garcia-Campello M, and others (2018). 'Epidemic dynamics of *Vibrio parahaemolyticus* illness in a hotspot of disease emergence, Galicia, Spain' Emerging Infectious Diseases: volume 24, pages 852 to 859
179. Harrison J, Nelson K, Morcrette H, Morcrette C, Preston J, Helmer L, and others (2022). 'The increased prevalence of *Vibrio* species and the first reporting of *Vibrio jasicida* and *Vibrio rotiferianus* at UK shellfish sites' Water Research: volume 211, page 117942
180. Romanello M, Di Napoli C, Drummond P, Green C, Kennard H, Lampard P, and others (2022). 'The 2022 report of the Lancet Countdown on health and climate change: health at the mercy of fossil fuels' The Lancet: volume 400, pages 1619 to 1654
181. Semenza JC, Trinanes J, Lohr W, Sudre B, Löfdahl M, Martinez-Urtaza J, and others (2017). 'Environmental suitability of *Vibrio* infections in a warming climate: an early warning system' Environmental Health Perspectives: volume 125, page 107004
182. Gildas Hounmanou YM, Engberg J, Bjerre KD, Holt HM, Olesen B, Voldstedlund M, and

others (2023). 'Correlation of high seawater temperature with *Vibrio* and *Shewanella* infections, Denmark, 2010 to 2018' Emerging Infectious Diseases: volume 29, page 605

183. ECDC (2022). 'Yersiniosis: annual epidemiological report for 2021'
184. Barratt JLN, Shen J, Houghton K, Richins T, Sapp SGH, Cama V, and others (2022). '*Cyclospora cayetanensis* comprises at least 3 species that cause human cyclosporiasis' Parasitology: volume 150, pages 269 to 285
185. Sağlam T, Düşen S, Karaman Ü, Mete E (2022). 'The presence of protozoan parasites in drinking waters and environmental waters in Denizli city centre' Turkiye Parazitolojii Dergisi: volume 28, pages 271 to 275
186. Tram NT, Hoang LMN, Cam PD, Chung PT, Fyfe MW, Isaac-Renton JL, and others (2008). '*Cyclospora* spp. in herbs and water samples collected from markets and farms in Hanoi, Vietnam' Tropical Medicine and International Health: volume 13, pages 1415 to 1420
187. Hoang LMN, Fyfe M, Ong C, Harb J, Champagne S, Dixon B, and others (2005). 'Outbreak of cyclosporiasis in British Columbia associated with imported Thai basil' Epidemiology and Infection: volume 133, pages 23 to 27
188. Morton V, Meghnath K, Gheorghe M, Fitzgerald-Husek A, Hobbs J, Honish L, and others (2019). 'Use of a case-control study and control bank to investigate an outbreak of locally acquired cyclosporiasis in Canada, 2016' Canada Communicable Disease Report: volume 45, pages 225 to 229
189. Marques DFP, Alexander CL, Chalmers RM, Elson R, Freedman J, Hawkins G, and others (2017). 'Cyclosporiasis in travellers returning to the United Kingdom from Mexico in summer 2017: lessons from the recent past to inform the future' Eurosurveillance: volume 22, page 30592
190. Nichols GL, Freedman J, Pollock KG, Rumble C, Chalmers RM, Chiodini P, and others (2015). '*Cyclospora* infection linked to travel to Mexico, June to September 2015' Eurosurveillance: volume 20, page 30048
191. Hadjilouka A, Tsaltas D (2020). '*Cyclospora cayetanensis*: major outbreaks from ready to eat fresh fruits and vegetables' Foods: volume 9, page 1703
192. Chacin-Bonilla L, Sanchez Y (2022). 'Seasonal trends of *Cyclospora cayetanensis* infections in a community of Falcon State, north-western Venezuela' Transactions of The Royal Society of Tropical Medicine and Hygiene: volume 116, pages 673 to 675
193. Kaminsky RG, Lagos J, Raudales Santos G, Urrutia S (2016). 'Marked seasonality of *Cyclospora cayetanensis* infections: 10-year observation of hospital cases, Honduras' BMC Infectious Diseases: volume 16, page 66
194. Frickmann H, Alker J, Hansen J, Dib JC, Aristizabal A, Concha G, and others (2021). 'Seasonal differences in *Cyclospora cayetanensis* prevalence in Colombian indigenous people' Microorganisms: volume 9, page 627
195. Almeria S, Cinar HN, Dubey JP (2019). '*Cyclospora cayetanensis* and cyclosporiasis: an update' Microorganisms: volume 7, page 317
196. Ma DW, Lee MR, Ku B, Cho SH, Lee SE (2020). 'Outbreak of cyclosporiasis in Korean travelers returning from Nepal' The Korean Journal of Parasitology: volume 58, pages 589 to 592

197. Daraei H, Oliveri Conti G, Sahlabadi F, Thai VN, Gholipour S, Turki H, and others (2021). 'Prevalence of *Cryptosporidium* spp. in water: a global systematic review and meta-analysis' Environmental Science and Pollution Research: volume 28, pages 9498 to 9507
198. Fournet N, Deege MP, Urbanus AT, Nichols G, Rosner BM, Chalmers RM, and others (2013). 'Simultaneous increase of *Cryptosporidium* infections in the Netherlands, the United Kingdom and Germany in late summer season, 2012' Eurosurveillance: volume 18, page 20348
199. PHE (2019). '*Cryptosporidium* data 2008 to 2017'
200. Chalmers RM, Robinson G, Elwin K, Elson R (2019). 'Analysis of the *Cryptosporidium* spp. and gp60 subtypes linked to human outbreaks of cryptosporidiosis in England and Wales, 2009 to 2017' Parasites and Vectors: volume 12, page 95
201. Cann KF, Thomas DR, Salmon RL, Wyn-Jones AP, Kay D (2013). 'Extreme water-related weather events and waterborne disease' Epidemiology and Infection: volume 141, pages 671 to 686
202. Young I, Smith BA, Fazil A (2014). 'A systematic review and meta-analysis of the effects of extreme weather events and other weather-related variables on *Cryptosporidium* and *Giardia* in fresh surface waters' Journal of Water and Health: volume 13, pages 1 to 17
203. Naumova EN, Christodouleas J, Hunter PR, Syed Q (2005). 'Effect of precipitation on seasonal variability in cryptosporidiosis recorded by the North West England surveillance system in 1990 to 1999' Journal of Water and Health: volume 3, pages 185 to 196
204. Forbes O, Hosking R, Mokany K, Lal A (2021). 'Bayesian spatio-temporal modelling to assess the role of extreme weather, land use change and socio-economic trends on cryptosporidiosis in Australia, 2001 to 2018' Science of The Total Environment: volume 791, page 148243
205. Naumova EN, Jagai JS, Matyas B, DeMaria A, MacNeill IB, Griffiths JK (2007). 'Seasonality in 6 enterically transmitted diseases and ambient temperature' Epidemiology and Infection: volume 135, pages 281 to 292
206. Hu W, Tong S, Mengersen K, Connell D (2007). 'Weather variability and the incidence of cryptosporidiosis: comparison of time series Poisson regression and SARIMA models' Annals of Epidemiology: volume 17, pages 679 to 688
207. ECDC (2022). 'Giardiasis (lambliasis): annual epidemiological report for 2019'
208. Adam EA, Yoder JS, Gould LH, Hlavsa MC, Gargano JW (2016). 'Giardiasis outbreaks in the United States, 1971 to 2011' Epidemiology and Infection: volume 144, pages 2790 to 2801
209. Lal A, Hales S, French N, Baker MG (2012). 'Seasonality in human zoonotic enteric diseases: a systematic review' PLOS ONE: volume 7, page e31883
210. Britton E, Hales S, Venugopal K, Baker MG (2010). 'The impact of climate variability and change on cryptosporidiosis and giardiasis rates in New Zealand' Journal of Water and Health: volume 8, pages 561 to 571
211. Atherholt TB, LeChevallier MW, Norton WD, Rosen JS (1998). 'Effect of rainfall on *Giardia* and crypto' Journal AWWA: volume 90, pages 66 to 80
212. Swirski AL, Pearl DL, Peregrine AS, Thomas J, Pintar K (2018). 'Temporal trends in *Giardia* occurrence in the Grand River and surrounding tributaries, Waterloo, Ontario

(2005 to 2013), a retrospective analysis of surveillance data' Zoonoses and Public Health: volume 65, pages 291 to 303

213. Turner AD, Lewis AM, Bradley K, Maskrey BH (2021). 'Marine invertebrate interactions with harmful algal blooms: implications for One Health' Journal of Invertebrate Pathology: volume 186, page 107555
214. Berdalet E, Fleming LE, Gowen R, Davidson K, Hess P, Backer LC, and others (2016). 'Marine harmful algal blooms, human health and wellbeing: challenges and opportunities in the 21st century' Journal of the Marine Biological Association of the United Kingdom: volume 96, pages 61 to 91
215. Jin D, Thunberg E, Hoagland P (2008). 'Economic impact of the 2005 red tide event on commercial shellfish fisheries in New England' Ocean and Coastal Management: volume 51, pages 420 to 429
216. Moore SK, Mantua NJ, Hickey BM, Trainer VL (2009). 'Recent trends in paralytic shellfish toxins in Puget Sound, relationships to climate, and capacity for prediction of toxic events' Harmful Algae: volume 8, pages 463 to 477
217. Gobler CJ, Doherty OM, Hattenrath-Lehmann TK, Griffith AW, Kang Y, Litaker RW (2017). 'Ocean warming since 1982 has expanded the niche of toxic algal blooms in the North Atlantic and North Pacific oceans' Proceedings of the National Academy of Sciences: volume 114, pages 4975 to 4980
218. McCabe RM, Hickey BM, Kudela RM, Lefebvre KA, Adams NG, Bill BD, and others (2016). 'An unprecedented coastwide toxic algal bloom linked to anomalous ocean conditions' Geophysical Research Letters: volume 43, pages 10366 to 10376
219. Van Dolah FM (2000). 'Marine algal toxins: origins, health effects, and their increased occurrence' Environmental Health Perspectives: volume 108, pages 133 to 141
220. Chapra SC, Boehlert B, Fant C, Bierman VJJ, Henderson J, Mills D, and others (2017). 'Climate change impacts on harmful algal blooms in U.S. freshwaters: a screening-level assessment' Environmental Science and Technology: volume 51, pages 8933 to 8943
221. Young N, Sharpe RA, Barciela R, Nichols G, Davidson K, Berdalet E, and others (2020). 'Marine harmful algal blooms and human health: a systematic scoping review' Harmful Algae: volume 98, page 101901
222. Young N, Robin C, Kwiatkowska R, Beck C, Mellon D, Edwards P, and others (2019). 'Outbreak of diarrhetic shellfish poisoning associated with consumption of mussels, United Kingdom, May to June 2019' Eurosurveillance: volume 24, page 1900513
223. Scoging A, Bahl M (1998). 'Diarrhetic shellfish poisoning in the UK' The Lancet: volume 352, page 117
224. Whyte C, Swan S, Davidson K (2014). 'Changing wind patterns linked to unusually high *Dinophysis* blooms around the Shetland Islands, Scotland' Harmful Algae: volume 39, pages 365 to 373
225. Moore SK, Trainer VL, Mantua NJ, Parker MS, Laws EA, Backer LC, and others (2008). 'Impacts of climate variability and future climate change on harmful algal blooms and human health' Environmental Health: volume 7, page S4
226. Fisman DN (2007). 'Seasonality of infectious diseases' Annual Review of Public Health: volume 28, pages 127 to 143

227. McMichael C (2015). 'Climate change-related migration and infectious disease' Virulence: volume 6, pages 548 to 553
228. McMichael C, Barnett J, McMichael AJ (2012). 'An ill wind? Climate change, migration, and health'. Environmental Health Perspectives: volume 120, pages 646 to 654
229. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, and others (2008). 'Global trends in emerging infectious diseases' Nature: volume 451, pages 990 to 993
230. Romanelli RJ, Cabling M, Marciniak-Nuqui Z, Marjanovic S, Morris S, Dufresne E, and others (2023). 'The societal and indirect economic burden of seasonal influenza in the United Kingdom'
231. Fusco F, Hocking L, Stockwell S, Bonsu M, Marjanovic S, Morris S, and others (2022). 'The burden of respiratory syncytial virus: understanding impacts on the NHS, society and economy' Rand Health Quarterly: volume 10, page 2
232. Robertson C, Pan J, Kavanagh K, Ford I, McCowan C, Bennie M, and others (2020). 'Cost burden of *Clostridioides difficile* infection to the health service: a retrospective cohort study in Scotland' Journal of Hospital Infection: volume 106, pages 554 to 561
233. Cossali G, Ratcliffe MS, Routledge EJ, Karayiannis T, Fielder J (2013). 'The cost of Legionellosis and technical ways forward' In: CIBSE Technical Symposium
234. Forbes AE, Zochowski WJ, Dubrey SW, Sivaprakasam V (2012). 'Leptospirosis and Weil's disease in the UK' QJM: An International Journal of Medicine: volume 105, pages 1151 to 1162
235. Tubiello FN, Rosenzweig C, Conchedda G, Karl K, Gütschow J, Xueyao P, and others (2021). 'Greenhouse gas emissions from food systems: building the evidence base' Environmental Research Letters: volume 16, page 65007
236. Waste and Resources Action Programme (WRAP) (2021). 'Pathway 2030: delivering a 50% reduction in the GHG footprint of UK food and drink'
237. Climate Change Committee (CCC) (2018). 'Land use: reducing emissions and preparing for climate change'
238. Costa C, Wollenberg E, Benitez M, Newman R, Gardner N, Bellone F (2022). 'Roadmap for achieving net-zero emissions in global food systems by 2050' Scientific Reports: volume 12, page 15064
239. Marteau TM, Chater N, Garnett EE (2021). 'Changing behaviour for net zero 2050' BMJ: volume 375, page n2293
240. Hemler EC, Hu FB (2019). 'Plant-based diets for personal, population, and planetary health' Advances in Nutrition: volume 10, pages S275 to S283
241. Swinburn BA, Kraak VI, Allender S, Atkins VJ, Baker PI, Bogard JR, and others (2019). 'The global syndemic of obesity, undernutrition, and climate change: the Lancet Commission report' The Lancet: volume 393, pages 791 to 846
242. Howard G, Calow R, Macdonald A, Bartram J (2016). 'Climate change and water and sanitation: likely impacts and emerging trends for action' Annual Review of Environment and Resources: volume 41, pages 253 to 276
243. Frumkin H, McMichael AJ (2008). 'Climate change and public health: thinking, communicating, acting' American Journal of Preventive Medicine: volume 35, pages 403 to 410

244. Frumkin H, Hess J, Luber G, Malilay J, McGeehin M (2008). 'Climate change: the public health response' American Journal of Public Health: volume 98, pages 435 to 445
245. WHO Regional Office for Europe (2012). 'Strengthening public health services and capacity: an action plan for Europe: promoting health and well-being now and for future generations
246. Greig DR, Mikhail AFW, Dallman TJ, Jenkins C (2020). 'Analysis Shiga toxin-encoding bacteriophage in a rare strain of Shiga toxin-producing *Escherichia coli* O157:H7 stx2a/stx2c' Frontiers in Microbiology: volume 11, page 577658
247. Conrad AR, Tubach S, Cantu V, Webb LM, Stroika S, Moris S, and others (2023). '*Listeria monocytogenes* illness and deaths associated with ongoing contamination of a multiregional brand of ice cream products, United States, 2010–2015' Clinical Infectious Diseases: volume 76, pages 89 to 95
248. Wielinga PR, Schlundt J (2014). 'One Health and food safety' In: Yamada A, Kahn LH, Kaplan B, Monath TP, Woodall J, Conti L, editors. Confronting Emerging Zoonoses: The One Health Paradigm
249. National Research Council (NRC) (2001). 'Toward the development of disease early warning systems' In: Under the Weather: Climate, Ecosystems, and Infectious Disease
250. FAO (2014). 'Horizon scanning and foresight: an overview of approaches and possible applications in food safety'
251. Nauta MJ, Fischer ARH, Van Asselt ED, De Jong AEI, Frewer LJ, De Jonge R (2008). 'Food safety in the domestic environment: the effect of consumer risk information on human disease risks' Risk Analysis: volume 28, pages 179 to 192
252. Food Standards Agency (FSA) (2023). 'Pathogen surveillance in agriculture, food and environment programme'
253. FSA (2022). 'Summary of food/feed safety incident root cause analysis (RCA) information received by the Food Standards Agency'
254. ECDC (2021). 'ECDC signs memorandum of understanding with UK Health Security Agency'
255. WHO Regional Office for Europe (2014). 'The case for investing in public health: the strengthening public health services and capacity, a key pillar of the European regional health policy framework Health 2020: a public health summary report for EPHO 8'
256. Djennad A, Lo Iacono G, Sarran C, Kessel A, Haines A, Fleming LE, and others (2018). 'A comparison of weather variables linked to infectious disease patterns using laboratory addresses and patients' home addresses.' BMC Infectious Diseases: volume 18, page 198

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