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Impact of extreme weather events on the occurrence of infectious diseases in Belgium from 2011 to 2021

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Abstract

The role of meteorological factors, such as rainfall or temperature, as key players in the transmission and survival of infectious agents is poorly understood. The aim of this study was to compare meteorological surveillance data with epidemiological surveillance data in Belgium and to investigate the association between intense weather events and the occurrence of infectious diseases. Meteorological data were aggregated per Belgian province to obtain weekly average temperatures and rainfall per province and categorized according to the distribution of the variables. Epidemiological data included weekly cases of reported pathogens responsible for gastroenteritis, respiratory, vector-borne and invasive infections normalized per 100 000 population. The association between extreme weather events and infectious events was determined by comparing the mean weekly incidence of the considered infectious diseases after each weather event that occurred after a given number of weeks. Very low temperatures were associated with higher incidences of influenza and parainfluenza viruses, *Mycoplasma pneumoniae*, rotavirus and invasive *Streptococcus pneumoniae* and *Streptococcus pyogenes* infections, whereas very high temperatures were associated with higher incidences of *Escherichia coli*, *Salmonella* spp., *Shigella* spp., parasitic gastroenteritis and *Borrelia burgdorferi* infections. Very heavy rainfall was associated with a higher incidence of respiratory syncytial virus, whereas very low rainfall was associated with a lower incidence of adenovirus gastroenteritis. This work highlights not only the relationship between temperature or rainfall and infectious diseases but also the most extreme weather events that have an individual influence on their incidence. These findings could be used to develop adaptation and mitigation strategies.

DATA SUMMARY

The authors certify that all supporting data, code and protocols are provided in the article or in data files S1, which are available in the online version of this article.

INTRODUCTION

Seasonality is a well-known factor in many infectious diseases [1–3]. Respiratory viruses tend to be more common in winter [4], whereas enterovirus central nervous infectious are more common in summer [5]. Weather plays an important role in this seasonality [6–8], either directly, by increasing individual susceptibility to infection or favouring the presence of the infectious agent in the environment, or indirectly, through the social implications of weather, such as gatherings and bathing in summer or confinement indoors in winter [9].

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Abbreviation: RSV, respiratory syncytial virus.

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A data sheet is available with the online version of this article.

The role of meteorological factors, such as precipitation or temperature, as key players in the transmission and survival of infectious agents is poorly understood behind the marked seasonality in temperate regions. Meteorological and epidemiological surveillance data are increasingly available and more frequently compared [10, 11]. Previous studies have shown that ambient temperature is associated with the annual influenza peak [12, 13], probably by influencing virus transmission and stability [14–16]. In addition, during seasonal influenza outbreaks, the weekly decrease in mean temperature (compared with the previous week) was strongly associated with the incidence of influenza in the following week. Others have shown that low temperature is associated with the weekly incidence of respiratory syncytial virus (RSV), metapneumovirus, bocavirus and adenovirus [17]. However, the incidence of human rhinovirus and enterovirus was independent of temperature. On the contrary, no association was found with relative humidity.

In addition to respiratory infections, climate change also plays a role in the incidence of vector-borne and waterborne infections. Global warming, and also other associated climate change such as changes in precipitation, either with increased flooding in some areas and drought in others, will affect pathogens, vectors and hosts, and thus vector-borne diseases. Malaria, dengue and Lyme disease are expected to spread beyond their current ranges [18]. The spread of the Asian tiger mosquito, *Aedes albopictus*, increases the risk of transmission of dengue, chikungunya and Zika [19]. Similarly, *Ixodes ricinus*, the primary vector for both Lyme borreliosis and tick-borne encephalitis, has expanded its range to higher altitudes and northern latitudes [20]. Extreme weather events have been reported to cause outbreaks of *Vibrio* spp. and *Leptospira* spp. [21]. In the Netherlands, an outbreak of Legionnaires' disease was reported to have been likely influenced by an increase in temperature in the 4 weeks prior to the outbreak, combined with rainfall in the previous 2 weeks [22].

Beyond seasonal variations in temperature and precipitation, global warming has increased the number of extreme weather events worldwide, such as episodes of extreme heat or cold and heavy rainfall or drought [23]. Guzman Herrador already showed the association between heavy precipitation events and waterborne outbreaks in Nordic countries [24], and Cherrie studied the pathogen seasonality and links with weather in England and Wales [25].

Despite their increasing frequency, the infectious consequences of extreme climatic events, whether heat waves or periods of rainfall leading to flooding or sewage overflows, are poorly understood. Although most countries have an epidemiological surveillance system based on weekly monitoring of the number of infections reported by a network of alert laboratories or by general practitioners [26, 27], to our knowledge, there is no system that compares epidemiological and meteorological data in monitoring the consequences of extreme weather events. However, such knowledge is essential if we are to implement the necessary prevention and control measures in the face of these increasingly frequent meteorological events.

The aim of this study was to compare meteorological surveillance data with epidemiological surveillance data in Belgium and to investigate the association between intense weather events and the occurrence of infectious diseases.

METHODS

Curation of meteorological data

Meteorological data provided by the Royal Belgian Meteorological Institute from more than 200 weather stations across the country were collected from January 2011 to December 2021. The data were aggregated per Belgian province to obtain weekly average temperatures (measured in °C) and rainfall (measured in millimetres) per province. As the province of Brussels is enclaved in the province of Flemish Brabant, these were considered as a single geographical unit. Weekly average temperatures and precipitation were categorized using percentile of the distribution of the variables between: 'very low', 'low', 'average', 'high' or 'heavy' and 'very high' or 'very heavy' (Table 1). The World Meteorological Organization defines extreme weather as 'an extreme weather event is rare at a particular place and time of year, with unusual characteristics in terms of magnitude, location, timing, or extent' [28]. Due to the relatively small size of Belgium and the absence of major geographical or climatic differences between regions, extreme weather events were defined as falling below the 5th percentile (i.e. representing very low temperatures or precipitation) or above the 95th percentile (i.e. representing very high temperatures or very heavy precipitation) as proposed in previous studies [29, 30].

Table 1. Categorization of weather events by rainfall and temperature in Belgium from 2011 to 2021

Category	Very low	Low	Average	High/heavy	Very high/heavy
Percentile	<5th	5th to <25th	25th–75th	>75th–95th	>95th
Rainfall (mm)	<0.10	0.10 to <4.20	4.20–23.50	>23.50–44.60	>44.60
Temperature (°C)	<1.70	1.70 to <6.20	6.20–16.00	>16.00–19.90	>19.90

Curation of epidemiological data

Epidemiological data were provided by Sciensano (Belgian National Institute of Public Health). They included cases per week and their postal code as reported by a surveillance network of laboratories in Belgium [26] from January 2011 to December 2021. The reported pathogens were those responsible for respiratory infections (*Bordetella pertussis*, *Legionella pneumophila*, *Mycoplasma pneumoniae*, RSV and influenza viruses), gastroenteritis (*Campylobacter* spp., enteroinvasive and enterohaemorrhagic *Escherichia coli*, *Salmonella* spp., *Shigella* spp., *Cryptosporidium* spp., *Giardia* spp., adenovirus, norovirus and rotavirus), invasive infections defined as septicaemia or meningitis (*Haemophilus influenzae*, *Streptococcus pneumoniae*, *Streptococcus pyogenes* and enterovirus) and vector-borne infections (*Bartonella* spp. and *Borrelia burgdorferi*). Weekly incidence has been normalized per 100 000 population to take account of population differences between provinces.

Determination of the time lag between weather and infection

Due to the incubation period (up to several weeks for *B. burgdorferi* [31]) and the time to diagnosis of infectious diseases, a time lag must be determined between a weather event and its impact on a given infectious disease. To determine the lag to be taken into account between weather events and subsequent infections, the weekly incidence per 100 000 population was determined after incremental lags of 1 to 8 weeks between an extreme weather event (very low or very high/heavy temperature or rainfall) and subsequent infections in the same province. The lag with the highest weekly incidence was chosen as the reference for determining the association between extreme weather events and subsequent infections.

Association between extreme weather events and infections

In order to determine the association between extreme weather events and infectious events, we compared the mean weekly incidence occurring after the previously determined time after a weather event in the same province between the different categories of temperature and precipitation using the Hsu multiple comparison with the best [31]. The reference 'best' group was either the group with the highest mean weekly incidence (weather event as risk factor) or the group with the lowest (weather event as protective factor). The association between extreme weather events and infections was considered significant if an extreme event (very low or very heavy/high rainfall/temperature) had a significantly higher (or lower) incidence than all other categories (p-value < 0.05 using Hsu multiple comparison with the best).

Statistical analysis

Statistical analyses were performed using Analyse-it for Microsoft Excel v5.30.4 (Analyse-it Software, Leeds, UK).

RESULTS

Weekly incidence of infections 1 to 8 weeks after an extreme weather event

Very low rainfall was associated with a higher incidence of *B. pertussis*, *Salmonella* spp., *Yersinia enterocolitica*, *Cryptosporidium* spp., rotavirus and invasive *S. pneumoniae* infections occurring 2 to 8 weeks later. Conversely, very heavy rainfall was associated with a higher incidence of respiratory infections (except *B. pertussis*), *Campylobacter* spp., *E. coli*, *Shigella* spp., adenovirus, norovirus gastroenteritis, invasive *H. influenzae*, *S. pyogenes*, enterovirus and vector-borne infections (Table 2).

Very low temperature was associated with a higher incidence of respiratory infections, *Y. enterocolitica* gastroenteritis, viral gastroenteritis and bacterial invasive infections occurring 1 to 8 weeks later. Conversely, very high temperature was associated with a higher incidence of parasitic and bacterial gastroenteritis (except *Y. enterocolitica*), enterovirus invasive infections and vector-borne infections occurring 1 to 8 weeks later (Table 3).

Respiratory infections

Very heavy rainfall was associated with a significantly higher incidence of RSV 1 week later (1.68 per week per 100 000 population, p-value ≤ 0.013 compared to all other categories). Influenza viruses and *M. pneumoniae* were also more frequent 8 weeks after very heavy rainfall, but not significantly different from after heavy rainfall. Very low temperature was significantly associated with a significantly higher incidence of *M. pneumoniae* (1.20 per week per 100 000 population, p-value < 0.0001) and influenza viruses (5.06 per week per 100 000 population, p-value<0.0001) 2 weeks later and parainfluenza viruses 8 weeks later (0.34 per week per 100 000 population, p-value<0.0001). *B. pertussis* had a higher incidence 4 weeks after very high temperature (0.11 per week per 100 000 population), but not significantly different from after high temperature (p-value=0.41). RSV had a significantly higher incidence 1 week after low temperature (2.53 per week per 100 000 population, p-value<0.0001) than after very low temperature or warmer temperature (Fig. 1).

Table 2. Weekly incidence (per 100 000 inhabitants) 1 to 8 weeks after an extreme rainfall event

Event	Very low rainfall								Very heavy rainfall							
Lag (week)	1	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
B. pertussis	0.078	0.082	0.057	**0.085**	0.082	0.080	0.079	0.077	0.069	0.071	0.070	0.065	0.078	0.075	0.070	0.078
L. pneumophila	0.068	0.051	0.051	0.042	0.056	0.044	0.050	0.061	0.097	0.102	0.098	0.102	0.085	0.094	0.088	**0.103**
M. pneumoniae	0.465	0.376	0.429	0.416	0.404	0.397	0.396	0.373	0.751	0.682	0.737	0.730	0.754	0.700	0.744	**0.757**
Influenza viruses	0.382	0.333	0.270	0.224	0.210	0.110	0.071	0.068	0.963	1.046	1.076	1.073	1.171	1.478	1.484	**1.618**
Parainfluenza viruses	0.124	0.129	0.101	0.135	0.106	0.097	0.109	0.106	0.177	0.172	0.158	0.165	0.183	0.155	0.186	**0.189**
RSV	0.317	0.306	0.327	0.356	0.363	0.293	0.286	0.364	**1.685**	1.546	1.468	1.354	1.338	1.284	1.345	1.311
Campylobacter spp.	0.932	0.879	0.918	0.929	0.992	0.989	0.957	1.024	0.927	1.009	1.027	**1.031**	0.915	0.938	0.937	0.893
E. coli	0.028	0.027	0.027	0.044	0.039	0.038	0.042	0.039	0.058	0.060	0.055	0.064	**0.065**	0.058	0.051	0.054
Salmonella spp.	0.046	0.058	0.065	**0.076**	0.066	0.075	0.071	0.070	0.055	0.055	0.057	0.048	0.057	0.060	0.060	0.064
Shigella spp.	0.026	0.019	0.022	0.029	0.030	0.031	0.022	0.019	**0.035**	0.028	0.028	0.028	0.027	0.034	0.035	0.032
Y. enterocolitica	0.035	**0.054**	0.045	0.047	0.040	0.042	0.049	0.040	0.031	0.034	0.036	0.037	0.030	0.038	0.033	0.036
Cryptosporidium spp.	0.065	0.050	0.058	0.061	0.057	**0.091**	0.073	0.072	0.065	0.074	0.067	0.058	0.054	0.066	0.062	0.069
Giardia spp.	0.147	0.171	0.170	0.159	0.169	0.166	0.178	**0.184**	0.170	0.165	0.162	0.176	0.160	0.148	0.155	0.165
Adenovirus	0.201	0.191	0.198	0.195	0.192	0.157	0.187	0.150	0.298	0.301	**0.303**	0.284	0.285	0.272	0.292	0.266
Norovirus	0.071	0.076	0.069	0.068	0.079	0.067	0.061	0.066	0.088	0.084	0.083	0.101	0.089	0.112	**0.114**	0.107
Rotavirus	0.290	0.275	0.323	**0.330**	0.289	0.236	0.224	0.218	0.153	0.143	0.169	0.187	0.194	0.191	0.196	0.227
H. influenzae	0.021	0.017	0.014	0.013	0.014	0.009	0.011	0.021	0.028	0.028	**0.030**	0.024	0.024	0.018	0.026	0.026
S. pneumoniae	0.141	0.151	0.153	0.157	0.145	0.146	0.170	**0.179**	0.097	0.126	0.113	0.115	0.111	0.104	0.119	0.115
S. pyogenes	0.074	0.058	0.059	0.054	0.049	0.056	0.050	0.051	0.086	0.074	0.076	0.084	0.087	**0.088**	0.075	0.086
Enterovirus	0.067	0.057	0.052	0.059	0.051	0.044	0.051	0.044	**0.093**	0.090	0.081	0.084	0.071	0.063	0.060	0.058
Bartonella spp.	0.016	0.021	0.024	0.028	0.020	0.023	0.015	0.026	0.022	**0.031**	0.026	0.024	0.026	0.027	0.028	0.026
B. burgdorferi	0.451	0.482	0.514	0.418	0.505	0.528	0.483	0.544	0.513	**0.544**	0.497	0.496	0.471	0.501	0.500	0.494
Percentile:	1	50	**99**													

RSV, Respiratory Syncitial Virus.

Gastroenteritis

Rainfall was not associated with a significantly higher or lower incidence of bacterial gastroenteritis 1 to 8 weeks later. *Y. enterocolitica* was more frequent 2 weeks after very low rainfall (0.05 per week per 100 000 population), but not significantly different from after average rainfall (p-value = 0.12). Very high temperature was significantly associated with a higher incidence of bacterial gastroenteritis caused by *E. coli* 8 weeks later (0.12 per week per 100 000 population, p-value ≤ 0.0014), *Salmonella* spp. 6 weeks later (0.13 per week per 100 000 population, p-value ≤ 0.033) and *Shigella* spp. 8 weeks later (0.06 per week per 100 000 population, p-value≤0.0052). *Campylobacter spp* gastroenteritis were more frequent 1 week after very high temperature (1.27 per week per 100 000 population), but not significantly higher than after high temperature (p-value=0.07, Fig. 2).

Very low rainfall was associated with a significantly lower incidence of adenovirus gastroenteritis 3 weeks later (0.20 per week per 100 000 population, p-value≤0.027). Norovirus was also less frequent 6 weeks after very low rainfall (0.006 per week per 100 000 population), but not significantly (p-value>0.05 compared to other categories except very high rainfall). Very high temperature was associated with a higher incidence of parasitic gastroenteritis caused by *Cryptosporidium* spp. 6 weeks later (0.16 per week per 100 000 population, p-value<0.0001) and *Giardia* spp. 8 weeks later (0.25 per week per 100 000 population, p-value≤0.013), whereas very low temperature was associated with a significantly higher incidence of rotavirus gastroenteritis 8 weeks later (0.81 per week per 100 000 population, p-value<0.0001, Fig. 3).

Invasive infections

Rainfall was not associated with a significantly higher or lower incidence of invasive infections 1 to 8 weeks later. Enterovirus invasive infections were more frequent 1 week after very heavy rainfall (0.09 per week per 100 000 population), but not significantly different from after low rainfall (p-value=0.07) and after very low rainfall (p-value=0.09). Very low temperature was associated with a higher incidence of invasive *S. pneumoniae* 3 weeks later (0.27 per week per 100 000 population, p-value<0.0001) and *S. pyogenes* 5 weeks later (0.14 per week per 100 000 population, p-value<0.0050). The incidence of invasive *H. influenzae* 3 weeks after very low temperature (0.04 per week per 100 000 population) was not significantly higher than after low temperature (p-value=0.10). Enterovirus had a higher incidence 3 weeks after high temperature (0.10 per week per 100 000 population), but not significantly different from after very high temperature (p-value=0.71, Fig. 4).

Table 3. Weekly incidence (per 100 000 inhabitants) 1 to 8 weeks after an extreme temperature event

Event	Very low temperature								Very high temperature							
Lag (week)	1	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
B. pertussis	0.078	0.082	0.057	**0.085**	0.082	0.080	0.079	0.077	0.069	0.071	0.070	0.065	0.078	0.075	0.070	0.078
L. pneumophila	0.155	**0.157**	0.146	0.137	0.141	0.127	0.135	0.120	0.129	0.129	0.127	0.137	0.129	0.141	0.135	0.140
M. pneumoniae	1.190	**1.199**	1.094	1.115	1.025	1.035	1.055	1.003	0.349	0.339	0.345	0.342	0.376	0.365	0.391	0.404
Influenza viruses	4.901	**5.064**	4.664	4.032	3.355	2.572	2.117	1.642	0.022	0.021	0.020	0.016	0.032	0.026	0.035	0.041
Parainfluenza viruses	0.249	0.248	0.252	0.269	0.276	0.308	0.307	**0.343**	0.110	0.119	0.089	0.087	0.090	0.093	0.114	0.118
RSV	**1.702**	1.490	1.276	1.116	0.969	0.881	0.779	0.631	0.054	0.057	0.053	0.071	0.084	0.117	0.172	0.198
Campylobacter spp.	0.736	0.700	0.702	0.726	0.764	0.754	0.781	0.761	**1.273**	1.197	1.198	1.143	1.176	1.148	1.076	1.039
E. coli	0.039	0.041	0.042	0.043	0.036	0.034	0.040	0.040	0.087	0.095	0.102	0.115	0.112	0.118	0.116	**0.124**
Salmonella spp.	0.041	0.045	0.047	0.054	0.049	0.048	0.053	0.048	0.100	0.118	0.109	0.113	0.129	**0.131**	0.122	0.122
Shigella spp.	0.034	0.033	0.040	0.034	0.037	0.034	0.036	0.032	0.043	0.048	0.042	0.047	0.048	0.045	0.054	**0.058**
Y. enterocolitica	0.036	0.037	0.039	0.046	0.041	0.044	**0.048**	0.047	0.036	0.037	0.033	0.033	0.027	0.031	0.032	0.040
Cryptosporidium spp.	0.036	0.042	0.042	0.035	0.036	0.034	0.030	0.034	0.121	0.137	0.150	0.143	0.156	**0.164**	0.157	0.157
Giardia spp.	0.137	0.137	0.134	0.138	0.144	0.145	0.131	0.126	0.195	0.222	0.234	0.233	0.233	0.234	0.239	**0.247**
Adenovirus	0.368	0.390	**0.415**	0.399	0.397	0.376	0.362	0.367	0.144	0.147	0.134	0.129	0.126	0.124	0.138	0.163
Norovirus	**0.124**	0.111	0.107	0.091	0.094	0.096	0.091	0.079	0.049	0.045	0.054	0.052	0.053	0.058	0.064	0.069
Rotavirus	0.427	0.505	0.594	0.649	0.733	0.765	0.805	**0.813**	0.065	0.064	0.062	0.060	0.057	0.059	0.052	0.054
H. influenzae	0.038	0.035	**0.039**	0.032	0.026	0.024	0.035	0.032	0.018	0.018	0.020	0.020	0.018	0.020	0.018	0.013
S. pneumoniae	0.249	0.269	**0.270**	0.259	0.261	0.214	0.216	0.209	0.063	0.059	0.058	0.067	0.073	0.082	0.091	0.092
S. pyogenes	0.124	0.139	0.119	0.131	**0.143**	0.137	0.135	0.128	0.080	0.072	0.059	0.059	0.051	0.052	0.057	0.062
Enterovirus	0.042	0.036	0.036	0.033	0.038	0.039	0.040	0.051	**0.103**	0.088	0.077	0.066	0.061	0.075	0.068	0.072
Bartonella spp.	0.034	0.028	0.026	0.031	0.026	0.022	0.029	0.032	0.039	0.047	**0.050**	0.045	0.048	0.042	0.045	0.046
B. burgdorferi	0.408	0.417	0.436	0.414	0.388	0.402	0.397	0.431	**0.780**	0.761	0.750	0.779	0.752	0.768	0.725	0.720
Percentile:	1	50	99													

RSV, Respiratory Syncitial Virus.

Vector-borne infections

Rainfall was not associated with a significantly higher or lower incidence of vector-borne infections 1 to 8 weeks later. *B. burgdorferi* was significantly more frequent 1 week after very high temperature (0.78 per week per 100 000 population, p-value<0.047), while *Bartonella* spp. was more frequent 3 weeks after very high temperature (0.049 per week per 100 000 population), but not significantly higher than after high temperature (p-value=0.09, Fig. 4).

DISCUSSION

The results of our study suggest the value of juxtaposing data from two different sources to find correlations between variations in observations (temperature and rainfall with infectious disease outbreaks) over a long period of time (from 2011 to 2021). Previous studies have focused on outbreaks following specific localized extreme weather events, such as hurricanes [32, 33] or floods [34, 35], but few have used the confrontation of localized and time-stamped epidemiological and meteorological data [24, 25]. In our work, we have shown that extreme weather events are associated with the incidence of infectious diseases. Indeed, we have shown that extreme temperatures are associated with higher incidences of infectious diseases, whether very low for influenza and parainfluenza viruses, *M. pneumoniae*, rotavirus and invasive *S. pneumoniae* and *S. pyogenes* infections or very high for *E. coli*, *Salmonella* spp., *Shigella* spp., parasitic gastroenteritis and *B. burgdorferi* infections. The effect of extreme rainfall is less clear. Very heavy rainfall was associated with a higher incidence of RSV 1 week later, which has been well described in tropical regions but less so in temperate regions [36, 37]. Conversely, very low rainfall was associated with a lower incidence of adenovirus gastroenteritis 3 weeks later. The association between rainfall and gastroenteritis has been reported previously [38]. However, to our knowledge, this is the first time that this association has been clearly demonstrated for adenovirus in temperate regions.

There are several limitations to our results. The first is the definition of extreme weather. In a review, Guzman Herrador noted that there is no consensus on the definition of extreme precipitation or temperature and that an association may be easier to find depending on the threshold used to classify extreme precipitation or temperature events [39]. We categorized weather events using percentiles of the distribution of weekly temperature and precipitation over the whole country during the whole observation period. This approach is possible because Belgium is a relatively small country with fairly homogeneous climatic conditions. Although simplistic, these empirical thresholds are useful for detecting trends and are easy to use and understand.

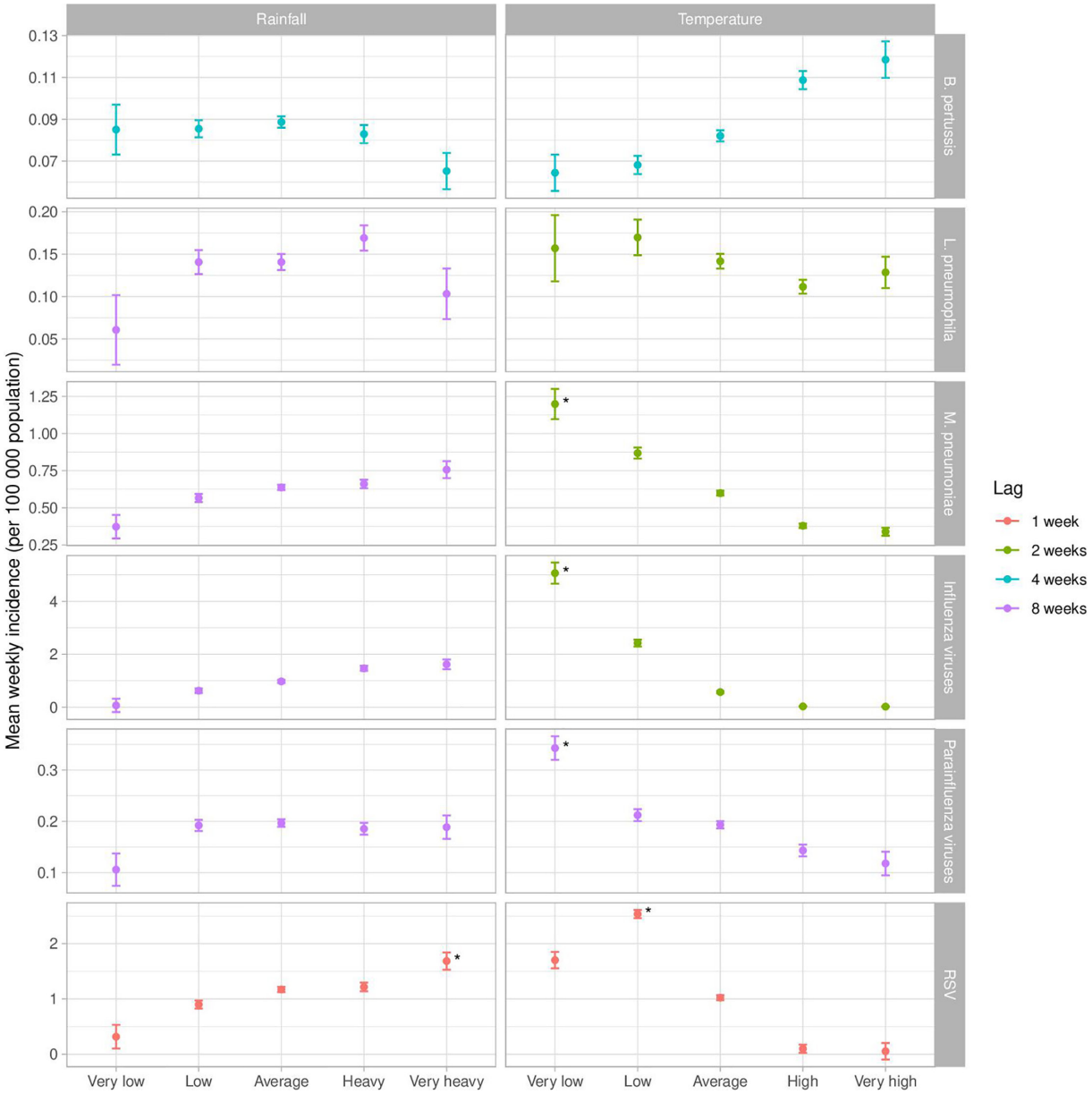

Fig. 1. Weekly incidence (per 100 000 population) of respiratory infections 1 to 8 weeks after a categorical weather event. *p-value < 0.05 compared to other categories (Hsu method).

Nevertheless, further statistical analysis is probably needed to refine these results, in particular to adjust the associations found for seasonality [7, 25]. Similarly, the limited number of reported cases for some infectious diseases led us to aggregate them per week, which limits the statistical power of this study. The second difficulty arises from the necessity of juxtaposing two different sets of national surveillance data in order to determine the time lag between weather events and their actual impact on infectious diseases. Indeed, there is a delay between the weather events that facilitate the occurrence of an infectious disease and its actual reporting due to incubation, spread, diagnosis and reporting time [26]. Here, we decided to use for each infectious disease the delay after an extreme weather event that led to the highest subsequent incidence. The results seemed quite consistent in temperatures for respiratory infections with lags of 1 to 4 weeks, except for parainfluenza viruses. Similarly, lags of more than 4 weeks are not surprising for parasitic diseases (*Cryptosporidium* spp. and *Giardia* spp.), which have longer incubation periods and can be more difficult to diagnose. Conversely, the rapid spread is expected for viral gastroenteritis, and delays seemed appropriate for adenovirus and norovirus, but not for rotavirus. However, other analytical methods – such as wavelet, Serfling or other time series methods – could be explored to evaluate the results of this study [40–43].

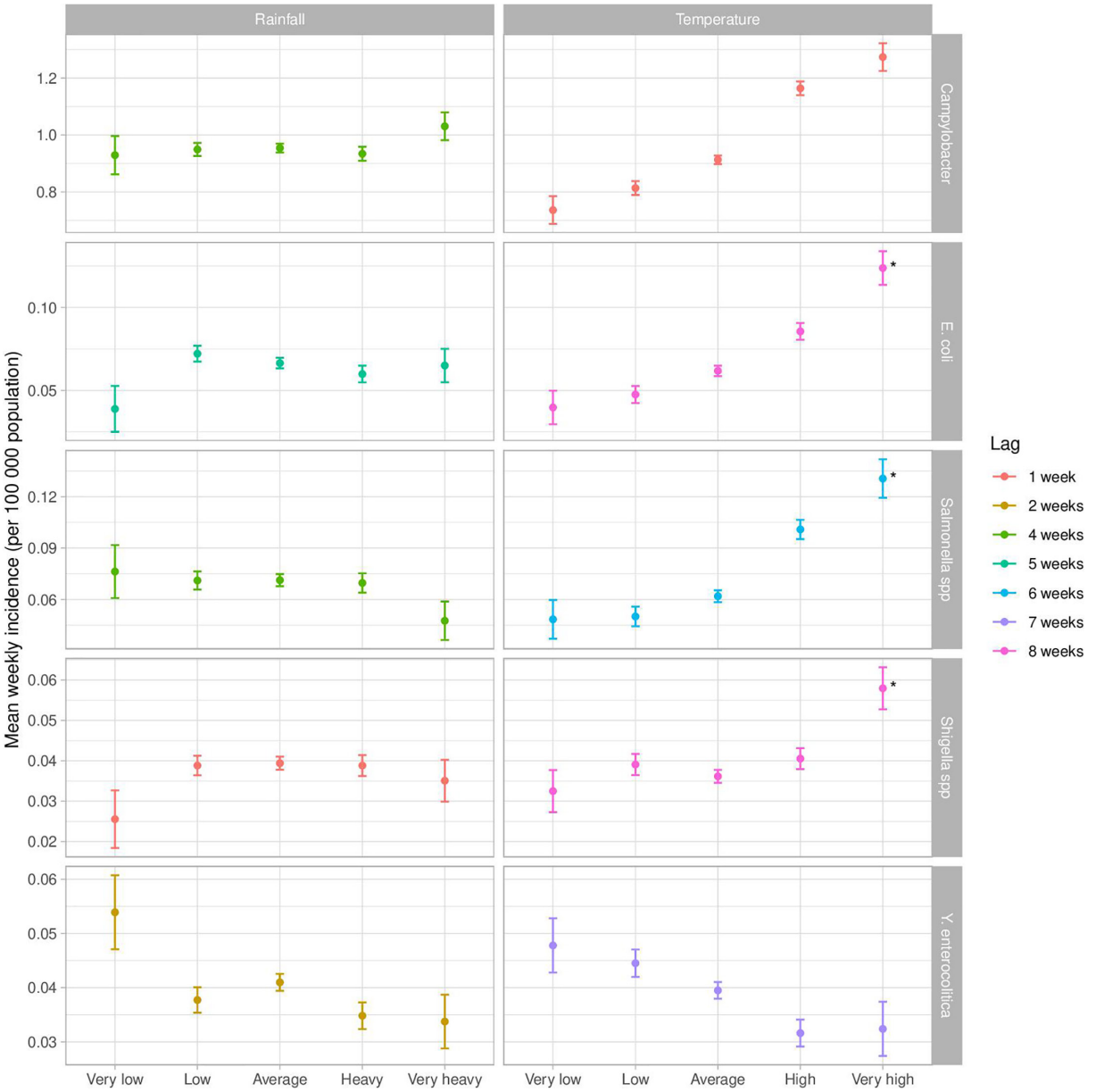

Fig. 2. Weekly incidence (per 100 000 population) of bacterial gastroenteritis 1 to 8 weeks after a categorical weather event. *p-value < 0.05 compared to other categories (Hsu method).

Second, we acknowledge that we were unable to assess the quality of reporting of epidemiological surveillance data, which is another potential source of bias. Wealthier and more urbanized regions are more likely to have greater diagnostic and reporting capacity than poorer and rural regions, leading to over- or underestimation of infectious disease activity during certain periods of the year. On the other hand, the sensitivity and representativeness of the coverage of the Belgian sentinel network of laboratories using test reimbursement data have been assessed to be stable over time and close to or greater than 50% for the clinical entities studied [44]. In our data, the average incidence per 100 000 population of certain diseases can be twice as high in urban provinces such as Antwerp as in rural provinces such as Luxembourg. In order to compare data on a larger scale than Belgium, diagnostic methods and indications should probably be normalized between regions, and the number of people tested should be taken into account in addition to the number of cases.

As the main objective of our work is to determine whether a relationship can be established between the incidence of infectious diseases and extreme climatic events, further studies are needed to confirm the results obtained on a larger spatial scale and to investigate the mechanisms explaining the observed associations. In this regard, the role of absolute humidity (the amount of

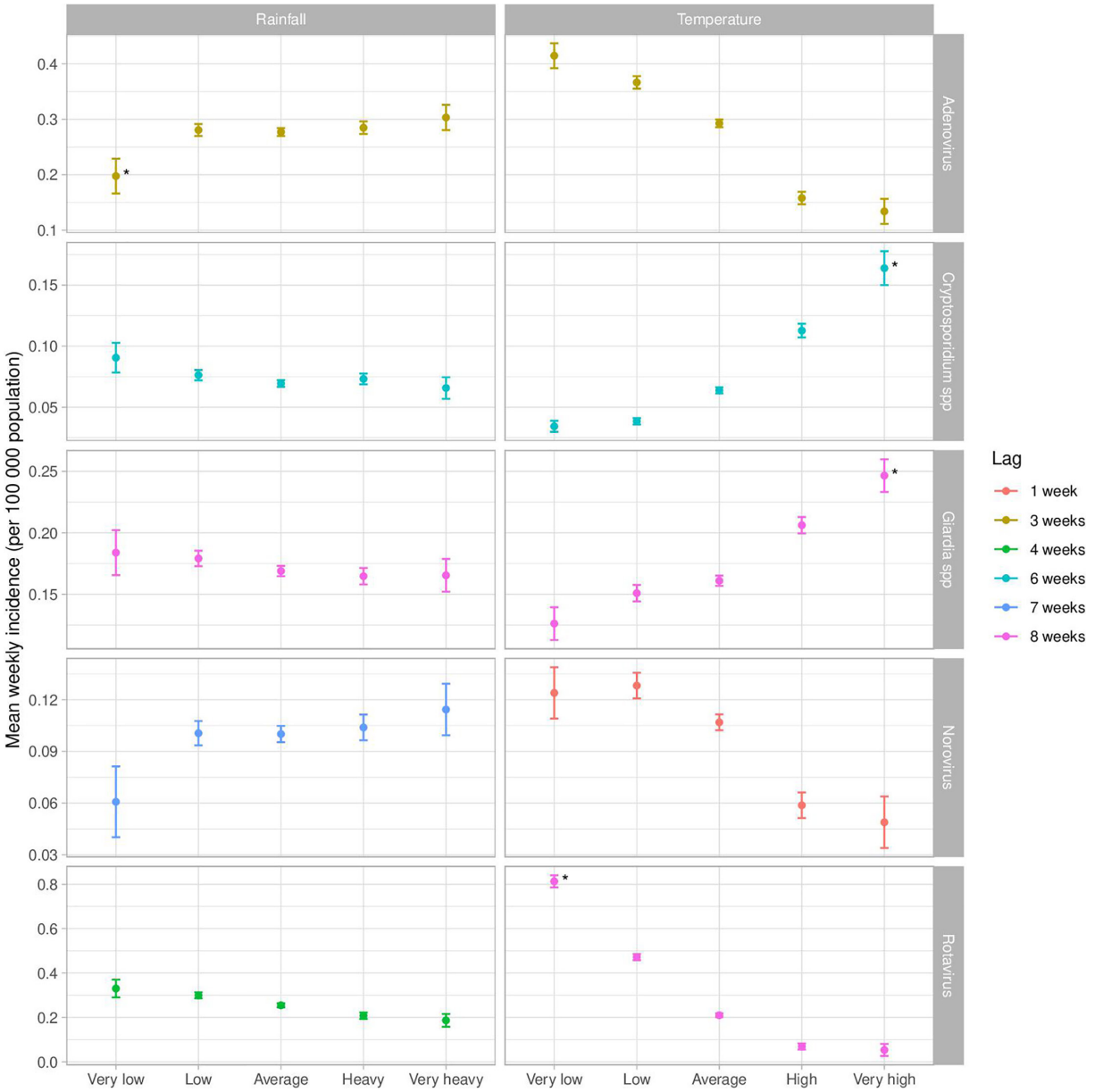

Fig. 3. Weekly incidence (per 100 000 population) of parasitic and viral gastroenteritis 1 to 8 weeks after a categorical weather event. *p-value < 0.05 compared to other categories (Hsu method).

water per unit volume of air (g/m^3)) and relative humidity should be considered in addition to precipitation and temperature [17]. Furthermore, at the local level, the utilization of an automated reporting system for notifiable cases by clinical laboratories to a central public health database, which includes the postcode of residence and/or is linked to local meteorological data from the previous days, could facilitate a more precise assessment of epidemiological links. In the case of campylobacteriosis, Oberheim [45] used gridded weather cells that overlapped with incidence reporting areas to link each reporting area with local weather data. At a larger regional scale, the use of aggregated data, such as those of the European Surveillance System [46], can be employed to validate the reproducibility of observations between countries and reporting systems within the same world region. Complex mathematical models can assist in identifying which weather factors are most significantly linked to a given infectious disease. This enables the investigation of how the seasonal burden of diseases will change under different climatic scenarios, as demonstrated by Lo Iacono in campylobacteriosis [47]. Artificial intelligence could also help to analyse large datasets linking notifiable case reporting with multiple meteorological data and their combination, as well as integrating population movement [48].

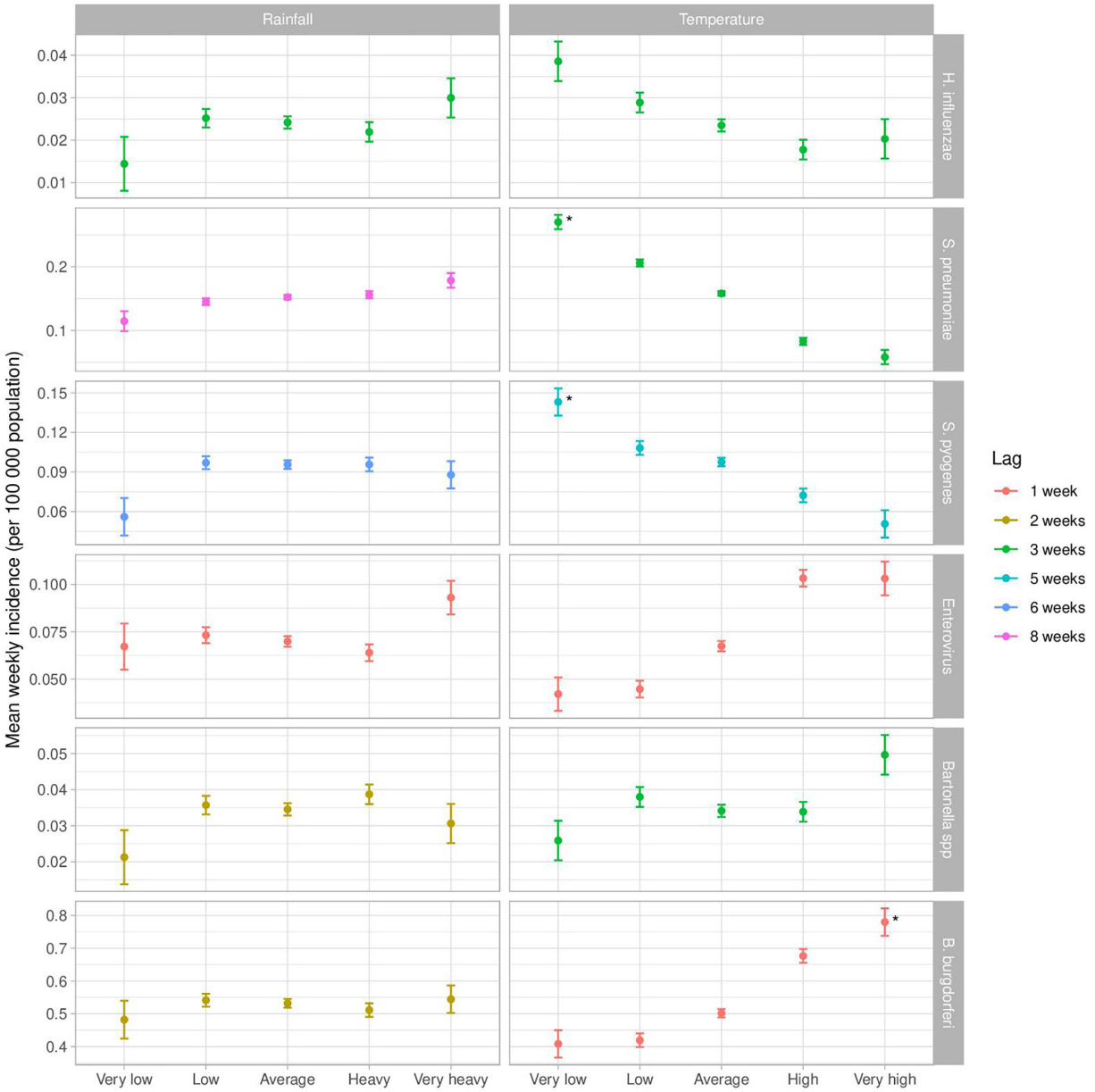

Fig. 4. Weekly incidence (per 100 000 population) of invasive and vector-borne infections 1 to 8 weeks after a categorical weather event. *p-value <0.05 compared to other categories (Hsu method).

In summary, the impact of extreme climate events on the incidence of infectious diseases is complex, and the occurrence of opposing effects makes general predictions difficult. Nevertheless, the consequences of these changes are already having an impact on public health, and health systems need to be prepared to mitigate these threats. Although our analysis of 11 years of high-quality laboratory-confirmed surveillance data for an entire country provides new and relevant information on when to anticipate and plan for increases in infectious diseases, the effects of climate change need to be studied at a very specific and local scale, taking into account the existence of confounding factors such as human behaviour or levels of disease reporting, in order to provide evidence-based public health recommendations that can be translated into health policy. There is also a need for a better understanding, from a multidisciplinary perspective, of the biological phenomena underlying the impact of climate on pathogens [49].

This work highlights not only the relationship between temperature or rainfall and infectious diseases but also the impact of the most extreme weather events on their incidence. Anticipating the epidemic risks associated with extreme weather events can help

public health authorities better plan public health responses and develop adaptation and mitigation strategies in places affected by these events, not least by disseminating appropriate communication messages to people affected by these events.

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Author contributions
The project was conceived and supervised by O.V. and M.H., within the framework of Z.F.'s Master of Science work and N.Y PhD thesis. N.Y., Z.F. and D.V.C. performed data curation. N.Y. and Z.F. performed formal data analysis. N.Y. drafted the original manuscript. All authors reviewed and edited the manuscript.

Conflicts of interest
The authors declare that there are no conflicts of interest.

Ethical statement
This study is a retrospective epidemiological observational study using unidentified agglomerated surveillance data; therefore, no ethical approval was required.

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