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Global epidemiology, seasonality and climatic drivers of the four human parainfluenza virus types

Yi Song [a,b], Yu-Nong Gong [c,d,e,f], Kuan-Fu Chen [c,g,h], David K. Smith [a,b], Hassan Zaraket [i], Seweryn Bialasiewicz [j], Sarah Tozer [k], Paul KS Chan [l], Evelyn SC Koay [m,n], Hong Kai Lee [n], Kok Keng Tee [o], Pieter LA Fraaij [p,q], Lance Jennings [r], Matti Waris [s,t], Hidekazu Nishimura [u], Aripuana Watanabe [v], Theo Sloots [w,x], Jen Kok [y,z], Dominic E. Dwyer [z,aa], Marion PG Koopmans [q], David W. Smith [ab,ac], Julian W. Tang [ad,ae], Tommy TY Lam [a,b,af,ag,ah,*], on behalf of the INSPIRE consortium [1]

[a] *State Key Laboratory of Emerging Infectious Diseases, School of Public Health, The University of Hong Kong, Hong Kong SAR, China*
[b] *Laboratory of Data Discovery for Health, Hong Kong SAR, China*
[c] *Research Center of Emerging Viral Infections, College of Medicine, Chang Gung University, Taoyuan, Taiwan*
[d] *International Master Degree Program for Molecular Medicine in Emerging Viral Infections, College of Medicine, Chang Gung University, Taoyuan, Taiwan*
[e] *Department of Laboratory Medicine, Linkou Chang Gung Memorial Hospital, Taoyuan, Taiwan*
[f] *National Institute of Infectious Diseases and Vaccinology, National Health Research Institutes, Zhunan, Taiwan*
[g] *College of Intelligent Computing, Chang Gung University, Taoyuan, Taiwan*
[h] *Department of Emergency Medicine, Chang Gung Memorial Hospital, Keelung, Taiwan*
[i] *Department of Experimental Pathology, Immunology and Microbiology, Faculty of Medicine, American University of Beirut, Beirut, Lebanon*
[j] *Australian Centre for Ecogenomics, School of Chemistry and Molecular Biosciences, The University of Queensland, Brisbane, Queensland, Australia*
[k] *Patient Safety and Quality, Clinical Excellence Queensland, Department of Health, Queensland Health, Australia*
[l] *Department of Microbiology, The Chinese University of Hong Kong, Hong Kong SAR, China*
[m] *Department of Pathology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore*
[n] *Molecular Diagnosis Centre, National University Hospital, Singapore*
[o] *Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia*
[p] *Department of Pediatrics, Erasmus Medical Centre, Rotterdam, the Netherlands*
[q] *ViroScience, Erasmus Medical Centre, Rotterdam, the Netherlands*
[r] *Canterbury Health Laboratories and Department of Pathology & Biomedical Sciences, University of Otago, Christchurch, New Zealand*
[s] *Institute of Biomedicine, University of Turku, Finland*
[t] *Clinical Microbiology, Turku University Hospital, Turku, Finland*
[u] *Virus Research Center, Sendai Medical Center, Sendai, Japan*
[v] *Department of Parasitology, Microbiology and Immunology, Federal University of Juiz de Fora, Juiz de Fora, Minas Gerais, Brazil*
[w] *Child Health Research Centre, The University of Queensland, Brisbane, Australia*
[x] *Children's Health Queensland, Brisbane, Australia*
[y] *Centre for Infectious Disease and Microbiology Laboratory Services, NSWHP-Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales, Australia*
[z] *Centre for Infectious Diseases-Public Health, Westmead Hospital, Westmead, New South Wales, Australia*
[aa] *Pathology and Infectious Diseases, University of Sydney, Sydney, Australia*
[ab] *PathWest Laboratory Medicine, Perth, Western Australia, Australia*
[ac] *School of Medicine, University of Western Australia, Perth, Western Australia, Australia*
[ad] *Department of Respiratory Sciences, University of Leicester, Leicester, UK*
[ae] *Clinical Microbiology, University Hospitals of Leicester NHS Trust, Leicester, UK*
[af] *Centre for Immunology & Infection, Hong Kong SAR, China*
[ag] *HKU-Pasteur Research Pole, Hong Kong SAR, China*
[ah] *The Hong Kong Jockey Club Global Health Institute, Hong Kong SAR, China*

[*] Correspondence to: 5/F, Laboratory Block, Li Ka Shing Faculty of Medicine, 21 Sassoon Road, Pokfulam, Hong Kong SAR, China.
E-mail address: ttylam@hku.hk (T.T. Lam).
[1] See Appendix A for **Contributors of INSPIRE consortium**.

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SUMMARY

Objectives: Human parainfluenza viruses (hPIV) are a common cause of acute respiratory infections, especially in children under five years and the elderly. hPIV can be subclassified as types 1–4: these showed various seasonality patterns worldwide, and it is unclear how climatic factors might consistently explain their global epidemiology.
Methods: This study collected time-series incidence data from the literature and hPIV surveillance programs worldwide (47 locations). Wavelet analysis and circular statistics were used to detect the seasonality and the months of peak incidence for each hPIV type. Relationships between climatic drivers and incidence peaks were assessed using a generalized estimating equation.
Results: The average positive rate of hPIV among patients with respiratory symptoms was 5.6% and ranged between 0.69–3.48% for different types. In the northern temperate region, the median peak incidence months for hPIV1, hPIV2, and hPIV4 were from September to October, while for hPIV3, it was in late May. Seasonal peaks of hPIV3 were associated with higher monthly temperatures and lower diurnal temperatures range throughout the year; hPIV4 peaks appeared to correlate with lower monthly temperatures and higher precipitation throughout the year. Different hPIV types exhibit different patterns of global epidemiology and transmission.
Conclusions: Climate drivers may play a role in hPIV transmission. More comprehensive and coherent surveillance of hPIV types would enable more in-depth analyses and inform the timing of preventive measures.

Introduction

Human parainfluenza viruses (hPIV) are enveloped, single-stranded RNA viruses of four types (1–4) in the *Paramyxoviridae* family. hPIV1 and hPIV3 are from the genus *Respirovirus*, while hPIV2 and hPIV4 are from the genus *Rubulavirus*.[1] hPIV is a leading cause of respiratory infections, especially among children, the elderly, and immunocompromised patients,[2–5] and is sometimes found as part of co-infections.[6] hPIV1–4 account for 2% to 18% of acute respiratory infections, with hPIV3 the most often detected type.[3,7–11] Clinical manifestations of hPIV are similar to other common respiratory viruses, and include croup, fever, bronchiolitis, pneumonia, and tracheobronchitis.[2] It is possible to be reinfected with hPIV in different epidemic seasons, as immunity after a previous infection is usually incomplete or short-lasting.[1] No licensed vaccine to prevent hPIV disease is currently available, but several phase I and II trials are ongoing. A global systematic review assessed the burden of hPIV in children under five years as causing 13% of acute lower respiratory infection (ALRI) cases, 4–14% of infant ALRI hospitalizations, and 4% of ALRI mortality.[12]

The World Health Organization (WHO) initiated the 'Battle against Respiratory Viruses (BRaVe)' in 2013 to promote further research and understanding of the environmental factors and transmission mechanisms of respiratory viruses.[13] Global and local surveillance of influenza and other viruses has provided valuable information about the seasonality and circulating strains of influenza, RSV, and SARS-CoV-2 to guide vaccination strategies,[14] but less is known about the seasonality of the different hPIV types.

Previous work showed a longer duration of hPIV epidemics and diverse patterns of seasonality and periodicity,[7,15] with the peak incidence of hPIV3 occurring in late spring to summer, and those for other types from autumn to winter in the United States of America (USA), China, the United Kingdom (UK), and Brazil.[16–19] hPIV1 and hPIV2 can have biennial (24-month) and annual patterns,[16,18–20] and hPIV3 has shown annual and semi-annual (6-month) cycles.[15–19,21] hPIV4 has circulated annually in the UK, but is less studied because of its low incidence rate and inadequate testing in diagnostic laboratories.[18]

Several single-center studies have examined the relationship between the seasonality of hPIV1–4 and climatic drivers. A study in Edinburgh found that hPIV3 incidence was negatively associated with humidity.[22] hPIV peaks were associated with temperature, atmospheric pressure, vapor pressure, precipitation, wind speed, and hours of sunlight in China.[23] Local seasonal patterns of hPIV have usually been reported from sites located in the temperate region of the northern hemisphere, and in many places the diagnostics did not differentiate between the four hPIV types. The limited amount of epidemiological surveillance data has largely restricted our understanding of the global seasonality and periodicity of the four different hPIV types.

This study aims to provide a global overview of the epidemiology of hPIV1–4 based on the incidence rates analyzed from worldwide laboratory surveillance data. We identified seasonal patterns of the four hPIV types from incidence time series across geographic regions in America, Europe, Asia, and Oceania, and investigated the relationships between climatic drivers and disease incidence. This work can lead to an improved understanding of the populations at risk, the temporal and geographical distribution of hPIV, and the climatic predictors of virus transmission, which will facilitate the prediction and, hopefully, prevention of future hPIV epidemics.

Methods

Data collection

Case data from cross-sectional studies

Papers published before December 2019 were searched via PubMed using the keywords 'parainfluenza virus' and 'parainfluenza viruses' in the title or abstract. Case data were collected from cross-sectional studies that reported hPIV test-positive rates from at least one hundred samples. Cross-sectional studies sampling from influenza-like illness (ILI), acute respiratory infections (ARI), severe acute respiratory infections (SARI), or pneumonia cases were included, while those from immunocompromised patients were excluded. If the study did not clearly report the symptom type (ILI, ARI, SARI, or pneumonia), then this was determined using the WHO definition.[24] Surveillance programs sampling from asymptomatic populations were also included. Studies were classified as from children if all participants were under 18 years of age, and as from adults if samples were from all age groups or adults only.

Case data from longitudinal studies

Incidence rates were calculated from surveillance programs that reported weekly or monthly data for at least one hPIV type in the literature. When these studies reported weekly or monthly detection

rates or counts, the raw data, if provided, were recorded or extracted from the manuscript figures using the software WebPlotDigitizer-4.2.[25] The location, time, and duration of sampling, the detection rate for hPIV1–4, sample size, sample population age, symptoms, sampling method, and detection method were recorded for each study, if available. Another data source was the INSPIRE (International Network for the Sequencing of Respiratory Viruses) network, from which 15 study sites (Turku, Leicester, Cambridge, Rotterdam, Vancouver, Ulaanbaatar, Halifax, Sendai, Hong Kong, Singapore, Brisbane, Canterbury, Sydney, Kuala Lumpur, and Taipei) reported monthly incidence rates of at least one of the hPIV types from January 2010 to December 2015.[15]

Climatic data

For sites with incidence rate time series, the monthly average temperature (°C), vapor pressure (hPa), precipitation (mm), and diurnal temperature range (°C) for each site during the study period were obtained from the Climatic Research Unit gridded Time Series (CRU TS) v4.03.[26] Absolute humidity (AH) and relative humidity (RH) were calculated by average temperature and vapor pressure.[27]

Data analysis

Seasonality detection and weighted seasonal peaks

The positivity of hPIV detection from infection cases showing different symptom types were summarized by patient age and virus type. Positive rates of subgroups were compared using Kruskal-Wallis tests, followed by Dunn's post hoc test. For data collected from longitudinal studies, incidence rate time series were analyzed to discover the periodicity and peak months of each hPIV type. Morlet wavelet analysis,[28] which has been extensively applied to modeling the seasonality of infectious diseases,[29–32] is particularly suited for this purpose due to its ability to handle non-stationary time series data and capture both frequency and temporal information simultaneously. This method was used to compute the wavelet power spectrum of incidence rates using the R package 'WaveletComp'.[33] P-values were calculated by comparing the wavelet power of the original time series, with mean power spectra randomly simulated from an autoregression process with the same auto-correlation as the original series.

Incidence time series were standardized by log-transformed, subtracted means, and divided by the standard deviation. When a significant ($p < 0.05$) high-power signal was seen at 6, 12, or 24 months in the power spectrum, seasonality was defined as semi-annual, annual, or biennial. A location would be considered seasonal for a type if at least one of its time series showed a high-power signal. If the hPIV types showed significant seasonality by wavelet analyses, the peak months of the cycles would be computed by the center of gravity, with months weighted by their average incidence rates.[32] Circular statistics were calculated using the R package 'circular'.[34] The circular mean month and its 95% confidence interval (CI) were estimated by 1000 bootstrap samples.

Impact of climatic factors on the seasonal peaks

A generalized estimating equation (GEE) was an extension of the generalized linear model to longitudinal data. GEE with a log link Poisson distribution family was used to estimate the impacts of climatic factors on monthly case numbers.[35] For each type of hPIV, only the places where seasonality was detected were included in the analysis. Spearman's correlation coefficients were calculated among the climatic variables. Coefficients between temperature, vapor pressure, and AH were > 0.9, indicating strong multi-collinearity, therefore, temperature (°C), precipitation (mm), diurnal temperature range (°C), and RH (%) were used as the independent variables in the model with autoregressive correlation structure of order 1. All predictors were standardized by subtracting from the mean and dividing by the standard deviation. Quasi-likelihood under Independence Model Criterion (QIC) was used to select the best set of variables for the best-fit regression model. The model was fitted using the 'geepack' package in R (version 4.0.4).[36]

Role of the funding source

The funding sources had no role in the study design, data collection, data analysis, data interpretation, or writing for this report, nor in the decision to submit it for publication.

Results

Positive rates of hPIV1-4 among different populations showing respiratory symptoms

The geographic overview of all samples included in this study is shown in Fig. 1 (and detailed in Supplementary Table 1). Among these, hPIV detection rates from 421 cross-sectional studies from 77 countries/regions are summarized in Table 1. The median positive hPIV detection rate among symptomatic patients was 5.46% (IQR: 3.14–8.69%), and the positive rates for types 1–4 were 1.42%, 0.69%, 3.48%, and 1.19%, respectively (Table 1). The overall positive rate of hPIV in children (under 18 years) was 6.45%, which was significantly higher than that in adults (4.34%, $p < 0.001$). No significant difference was found between the positive rates in the age groups 0–6 and 7–18 years (Supplementary Figure 1). Differences in positive rates between children and adults were only significant with hPIV1

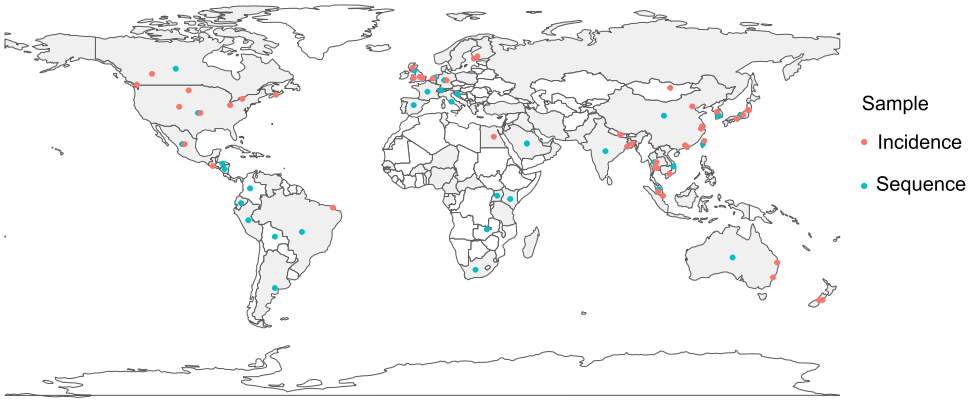

Fig. 1. Map of study sites with incidence and sequence data of human parainfluenza viruses. Geographic regions with incidence data are filled in grey. Study sites with incidence and sequence sample sites are indicated by red and blue dots, respectively.

Table 1
Positive rates of hPIV1–4 in adults and children showing different symptom severities as extracted from the literature review.

	hPIV Median (IQR, %)[a]	hPIV1 Median (IQR, %)	hPIV2 Median (IQR, %)	hPIV3 Median (IQR, %)	hPIV4 Median (IQR, %)	P-value
All studies (n=421)	5.46 (3.14–8.69)	1.42 (0.67–2.66)	0.69 (0.28–1.66)	3.48 (1.82–5.67)	1.19 (0.63–2.48)	**< 0.001**[b]
Age						
Child (n=247)	6.45 (3.82–9.90)	1.54 (0.76–3.05)	0.72 (0.27–1.95)	4.14 (2.49–6.65)	1.35 (0.88–2.43)	**< 0.001**[c]
Adult (n=174)	4.34 (2.58–6.65)	1.14 (0.54–1.95)	0.63 (0.29–1.28)	2.44 (0.94–4.33)	0.88 (0.52–2.53)	**< 0.001**[d]
P-value	**< 0.001**	**0.0086**	0.74	**< 0.001**	0.067	
Symptom type						
ILI (n=73)	5.14 (3.04–8.08)	1.28 (0.53–2.58)	0.84 (0.36–1.27)	2.92 (1.07–5.13)	1.40 (0.64–3.26)	**< 0.001**[e]
ARI (n=274)	5.59 (3.49–9.31)	1.46 (0.75–2.90)	0.63 (0.29–2.00)	3.56 (2.16–6.07)	1.24 (0.63–2.48)	**< 0.001**[c]
SARI (n=74)	5.00 (2.75–7.69)	1.00 (0.63–1.51)	0.60 (0.21–1.09)	3.52 (2.15–5.48)	1.20 (0.67–2.06)	**< 0.001**[d]
P-value	**0.029**	0.074	0.32	**0.0026**	0.81	
Detection method						
Immunoassay (n=73)	4.80 (3.16–6.77)	1.01 (0.62–2.41)	0.50 (0.20–2.22)	3.20 (1.86–4.94)	0.37 (0.37–0.37)	**< 0.001**[e]
PCR-based (n=324)	5.71 (3.27–9.28)	1.43 (0.73–2.70)	0.76 (0.30–1.44)	3.57 (1.82–5.99)	1.23 (0.66–2.64)	**< 0.001**[b]
Culture (n=20)	4.37 (2.36–7.69)	1.85 (1.01–2.39)	0.55 (0.10–1.16)	2.73 (1.47–3.65)	0.54 (0.31–0.77)	0.09
P-value	0.10	0.51	0.57	0.22	0.22	
Number of total sample size	9,793,630	814,933	784,473	821,971	150,755	

ILI: influenza-like illness, ARI: acute respiratory infections, SARI: severe acute respiratory infections (including pneumonia).
Statistical significance ($p < 0.05$) is indicated in bold.
[a] Including studies that did and did not distinguish hPIV types in their data.
[b] Dunn test adjusted by Bonferroni method indicated a significant difference ($p < 0.05$) between hPIV1 and hPIV2, hPIV1 and hPIV3, hPIV2 and hPIV3, hPIV2 and hPIV4, hPIV3 and hPIV4.
[c] Dunn test adjusted by Bonferroni method indicated a significant difference ($p < 0.05$) between hPIV1 and hPIV2, hPIV1 and hPIV3, hPIV2 and hPIV3, hPIV3 and hPIV4.
[d] Dunn test adjusted by Bonferroni method indicated a significant difference ($p < 0.05$) between hPIV1 and hPIV3, hPIV2 and hPIV3, hPIV3 and hPIV4.
[e] Dunn test adjusted by Bonferroni method indicated a significant difference ($p < 0.05$) between hPIV1 and hPIV3, hPIV2 and hPIV3.

(p=0.0086) and hPIV3 ($p < 0.001$). The positive rates of hPIV3 and overall hPIV among ARI patients were significantly higher than those among ILI patients. Various detection methods did not significantly affect the positive rates of hPIV1–4 they reported. For all age subgroups and severity of symptoms, hPIV3 was the most prevalent type, and hPIV2 was the least ($p < 0.001$) (Table 1).

Seasonality and peak months of hPIV1-4 by site

Incidence rate time series from 32 longitudinal studies and 15 INSPIRE longitudinal surveillance sites were included in the temporal analyses. The mean peak months, weighted by incidence rates, revealed distinct seasonal patterns of hPIV types (Fig. 2). hPIV1 was

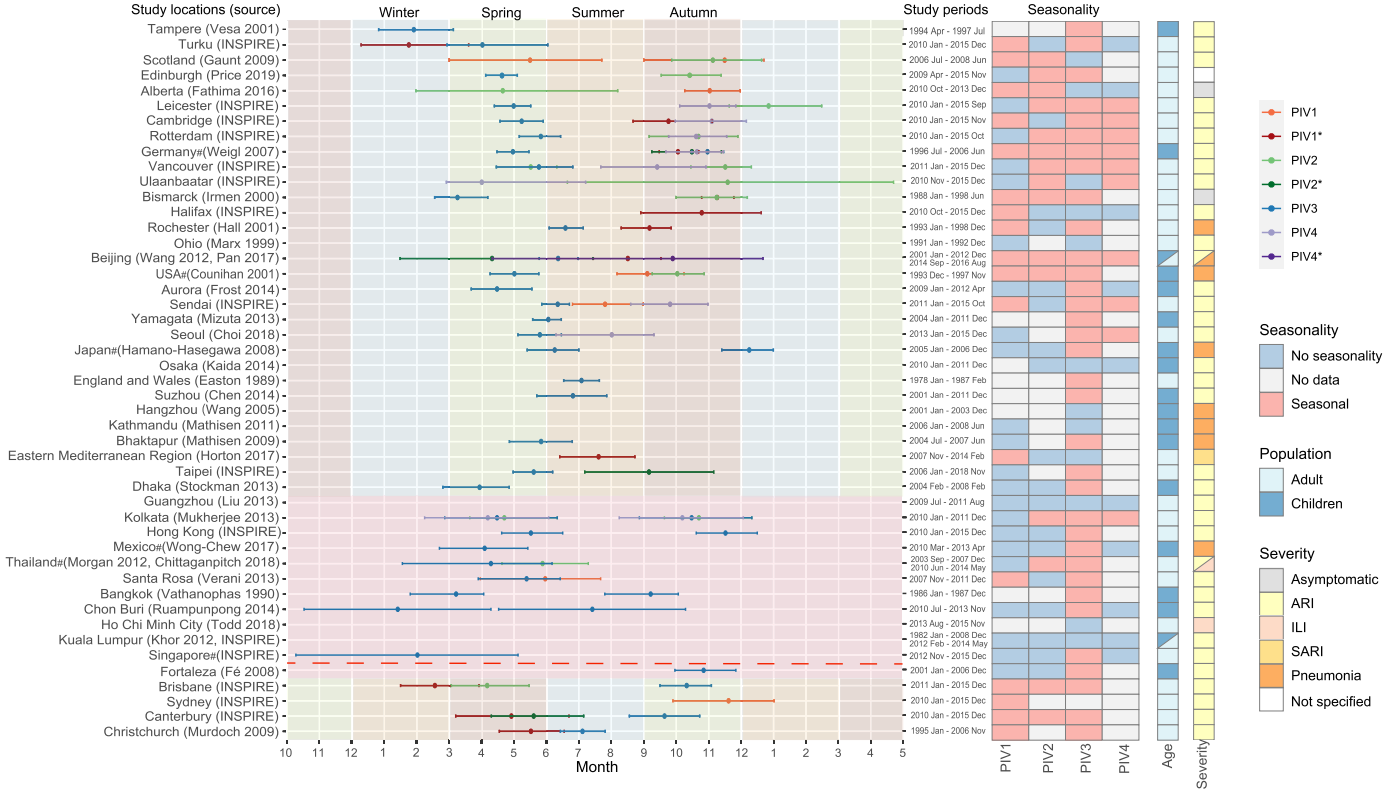

Fig. 2. Weighted mean peak months of hPIV1–4 listed by latitudinal order. The left axis shows the locations and source references. The right axis shows the duration of collection years and months. The equator is marked by the red dashed line. Seasons in temperate regions are shaded. * These represent the peak months of biennial (24-month) cycles. # These are inferred from overall national data where certain cities/states/provinces of these countries are also available, and are displayed in separate rows. ILI: influenza-like illness, ARI: acute respiratory infections, SARI: severe acute respiratory infections.

Table 2
Weighted mean peak months of hPIV1–4 in the tropical region, north temperate region, and south temperate region.

	hPIV1 Median (IQR)/ [Min–Max][a]	hPIV2 Median (IQR)/ [Min–Max][a]	hPIV3 Median (IQR)/ [Min–Max][a]	hPIV4 Median (IQR)/ [Min–Max][a]
North temperate region (> 23.5°N)	9.18 (7.81–10.79)	10.48 (9.16–11.25)	5.78 (4.89–6.35)	9.89 (9.41–10.64)
Tropical region (23.5°S–23.5°N)	[5.96–5.96]	5.88 (5.29–8.29)	5.39 (4.09–9.21)	[4.19–10.19]
South temperate region (> 23.5°S)	5.22 (4.33–7.04)	[4.17–5.60]	9.64 (8.37–9.98)	No data

[a] Minimum and maximum values are shown if there are fewer than three data sets in the category.

Table 3
The odds ratio of climatic factors on the incidence of hPIV1–4 by generalized estimating equation (GEE) model.

	Temperature (℃)	Diurnal temperature range (℃)	Precipitation (10 mm)	RH (%)
PIV1	0.838 (0.688–1.020, p=0.08)	1.020 (0.927–1.120, p=0.69)		
PIV2	0.880 (0.736–1.050, p=0.16)	0.918 (0.831–1.010, p=0.091)		
PIV3	**1.234 (1.003–1.518, p=0.047)**	**0.965 (0.935–0.996, p=0.027)**	0.982 (0.962–1.002, p=0.071)	0.997 (0.944–1.053, p=0.91)
PIV4	**0.665 (0.584–0.759, p < 0.001)**		**1.064 (1.009–1.121, p=0.022)**	

RH: relative humidity. P-values smaller than 0.05 are highlighted in bold.

mostly present in annual and biennial cycles, and a semi-annual cycle was only seen in Scotland. The median hPIV1 peak month was early September (IQR: 7.81–10.79) in the north temperate region, and early May (IQR: 4.33–7.04) in the south temperate region. hPIV1 peaked in late May in Santa Rosa in the tropical region (Table 2). hPIV2 circulated semi-annually in Kolkata, biennially in Germany, Beijing, Taipei, and Canterbury, and annually in the other sites. The median hPIV2 peak month was October (IQR: 9.16–11.25) in the north temperate region, and late May (IQR: 5.29–8.29) in the tropical region. hPIV2 showed April and May peaks in the south temperate region. hPIV3 was presented semi-annually in Germany, Japan, Hong Kong, Kolkata, Bangkok, and Chon Buri, and annually in the other sites. The median peak month for hPIV3 was late May (IQR: 4.89–6.35) in the north temperate region, May (IQR: 4.09–9.21) in the tropical region, and late September (IQR: 8.37–9.98) in the south temperate region. No hPIV4 data was reported from the south temperate region. hPIV4 circulated semi-annually in Kolkata, biennially in Beijing, and annually in other sites. The median hPIV4 peak month was late September (IQR: 9.41–10.64) in the north temperate region. The peak month in Kolkata in the tropical region was early April and October.

Effects of climatic factors on the incidence of hPIV

GEE-based Poisson regression (Table 3) showed that temperature, diurnal temperature range, and RH were related to the incidence peaks of hPIV1–4. Increases in hPIV3 incidence were associated with higher temperature (OR: 1.234, 95% CI: 1.003–1.518), and lower diurnal temperature range (OR: 0.965, 95%CI: 0.935–0.996), while incidences of hPIV1, hPIV2, and hPIV4 were negatively associated with temperature with OR of 0.838 (95%CI: 0.688–1.020), 0.880 (95%CI: 0.736–1.050), and 0.665 (95%CI: 0.584–0.759), respectively. The incidence of hPIV4 was also associated with higher precipitation (OR: 1.064, 95% CI: 1.009–1.121).

Discussion

We described the global epidemiology and seasonal patterns of the four hPIV types. They showed significant differences in incidence rates among age groups and by symptom severity, with hPIV3 being the most commonly detected. hPIV1, hPIV2, and hPIV4 showed annual or biennial cycles in their incidence peaks, while hPIV3 showed semi-annual or annual cycles, possibly supporting its higher overall incidence. The months of peak incidence for hPIV1, hPIV2, and hPIV4 were during autumn in the north and south temperate regions, while for hPIV3 this was during late spring to summer in temperate regions. The hPIV3 peaks in tropical regions were in early May. The positive correlation between temperature and hPIV3, and the negative correlation between temperature and hPIV1, hPIV2, and hPIV4 supported the observed seasonality. These epidemiological characteristics of the hPIV types could be used to guide resource planning for the times when hPIV-related pediatric healthcare burdens are likely to be higher.

Recently reported studies of the circulation of hPIV1–4 in different locations that were published after our search in this analysis are generally consistent with these results. In Germany from 2015 to 2019,[37] seasonal patterns of hPIV1–4 were similar to our results (which included data from a German study from 1996 to 2006).[38] Surveillance in the USA from 2011 to 2019 found hPIV1 and hPIV2 circulated biennially, and hPIV3 and hPIV4 circulated annually.[39] Our analyses detected annual and biennial cycles for hPIV1, and annual cycles for hPIV2 in different regions of the USA. hPIV1 and 2 competed at the peaks in alternative years in the USA. In the non-advantage years, hPIV1 or 2 showed a lower peak in the populations. A cohort study in Western Australia found that hPIV1 peaked biennially in autumn, and hPIV3 peaked in spring.[40] They also reported that the hPIV3 peak was slightly decreased in the year when hPIV1 was in circulation. The alternative peaks by hPIV1 and hPIV2 and the weakening effect of hPIV1 circulation on hPIV3 peak were potentially attributable to cross-protective immunity and antigenic change.[41] A systematic analysis of the overall hPIV seasonality at 83 sites showed that hPIV epidemics occurred mostly in spring and early summer in the northern and southern hemispheres, and had a longer duration than other examined respiratory viruses.[42] As this analysis did not consider the individual types, its result largely reflected the most prevalent type, hPIV3, and is consistent with the findings here.

Climatic factors affect the efficiency of transmission and environmental survival of many respiratory viruses.[43] Unlike most respiratory viruses, where incidence is increased in winter, the peak months of hPIV3 circulation are in spring and summer when temperatures are higher. A ten-year surveillance in Wenzhou, China, also suggested temperature was positively correlated with hPIV3 activity.[44] Most respiratory viruses circulate less during summer, which allows the increasing incidences of hPIV3 under less competition. A contradictory relationship between temperature and hPIV1 infections was found in Singapore.[45] Small seasonal variations of temperature in tropical areas may weaken the association, as found in influenza that “cold-dry” peaks occurred in temperate regions, and “humid-rainy” peaks occurred in the tropical region.[46] hPIV1 and hPIV2 in the tropics did not show significant seasonality compared to temperate regions. hPIV3 presented more biannual peaks

and longer durations of peaks in the tropical region compared to temperate regions. Lack of clear seasonality or longer durations of peaks in the tropics were also found in influenza.[47] Price et al. suggested influenza A virus and respiratory syncytial virus seemed to prefer low humidity range,[22] which we did not include as a climatic factor due to the limitation of the CRU database. They also found hPIV3 peaks were negatively associated with RH, while no significant correlation was observed in our results.[22] hPIV3 appeared to be more stable at lower RH and higher temperatures than other enveloped RNA viruses.[22] In addition, the seasonal peak of hPIV4 is positively correlated with precipitation, unlike the other hPIV types. hPIV4 showed significant seasonality only when moderate or high precipitation in autumn was observed; the other types did not show any association with precipitation. The correlation between climatic factors and hPIV incidence could be further examined to see if meteorological factors might enhance prediction models for temporally and geographically forecasting hPIV trends.

There are several limitations to this study. Detection rates from surveillance programs do not represent the incidence amongst all members of any given population; not all diagnostic laboratories are able to screen for hPIV1–4; in places where the seasonal variation of climatic factors is small, the relationship between climatic factors and viral seasonality may not be significantly detected in the regression; studies included were from different time frames without considering the overall pattern shift through time; symptomatic screening for influenza-like illness may not capture all the hPIV incidence, and hPIV disease may be defined differently across age groups. Phylodynamic analyses have been useful to study virus epidemiology and seasonality.[48,49] Although it would be of epidemiological interest to conduct such analyses on hPIV, the relative scarcity of sequence availability prior to 2009 is a significant limitation. Following the 2009 influenza A (H1N1) pandemic, systematic surveillance on influenza and other respiratory viruses was enhanced via more widely available diagnostic and sequencing techniques, facilitated by local authorities and global collaborations such as the WHO Global Influenza Surveillance and Response System (GISRS).[50,51] Phylodynamic analyses of viral sequences from a climatically more diverse range of countries (both temperate and tropical) will improve our understanding of how the seasonal epidemiological patterns of the different hPIV types change across the world, which can inform health impact assessments. During the COVID-19 pandemic, the viral circulation of respiratory viruses, including hPIV, was largely reduced because of non-pharmaceutical interventions (NPIs).[52] After lifting the NPIs, the peak timing and epidemic duration of respiratory viruses seem to alter.[53] Recent studies show the shifts of seasonality hPIV1 and hPIV3 in China and hPIV in Canada during COVID-19.[54,55] Further surveillance following up for a sufficient period of time would be required to ascertain how the long-term epidemiology of different hPIV types are impacted by COVID-19. Furthermore, some climatic factors, such as relative humidity range, which are unavailable in the climate database used in this study, could be used for their associations with hPIV subtype epidemiology in the future.

In conclusion, this study summarized the worldwide epidemiology and seasonality of hPIV types 1–4 and explored the relationship of climatic drivers to their incidence peaks. hPIV3 was the most prevalent type and reached peak incidence within spring - around late May in the north temperate region, and late September in the south. hPIV1, hPIV2, and hPIV4 reached peaks in autumn. The increases in hPIV3 incidence correlated with higher temperature and lower diurnal temperature range, whereas hPIV4 was associated with lower temperature and higher precipitation. Identifying the seasonal patterns and climatic drivers affecting the incidence peaks enables further understanding of the virus circulation patterns and informs the timing for potential vaccination and healthcare management strategies.

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Author contributions

YS and TTL conceived and designed the study. YNG, KFC, HZ, SB, ST, PKSC, ESCK, HKL, KKT, PLAF, LJ, MW, HN, AW, TS, JK, DED, MK, and JWT contributed to data collection. YS, DKS, and TTL conducted the data analysis. YS drafted the manuscript. DWS and all other authors critically reviewed the final manuscript and approved it for submission.

Data availability

Data supporting the findings of this study are available from the corresponding author on request.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Contributors of INSPIRE consortium

- **Steven J Drews**, Alberta Provincial Laboratory for Public Health, University of Alberta, Edmonton, AB, Canada
- **Todd Hachette**, Department of Pathology, Dalhousie University, Halifax, NS, Canada
- **Mel Krajden**, British Columbia Centre for Disease Prevention and Control, Vancouver, BC, Canada
- **André Corriveau**, Government of the Northwest Territories, Yellowknife, NT, Canada
- **Hamid Jalal**, Department of Pathology, Addenbrookes Hospital, Cambridge, UK
- **Pagbajabyn Nymadawa**, Laboratory of Virology, National Center for Communicable Diseases, Ulaanbaatar, Mongolia
- **Darmaa Badarch**, Laboratory of Virology, National Center for Communicable Diseases, Ulaanbaatar, Mongolia
- **Alice Kabanda**, National Reference Laboratory, Kigali, Rwanda
- **Yu-Nong Gong**, Research Center of Emerging Viral Infections, College of Medicine, Chang Gung University, Taoyuan, Taiwan
- **Kuan-Fu Chen**, College of Intelligent Computing, Chang Gung University, Taoyuan, Taiwan
- **Hassan Zaraket**, Department of Experimental Pathology, Immunology and Microbiology, Faculty of Medicine, American University of Beirut, Beirut, Lebanon
- **Seweryn Bialasiewicz**, Australian Centre for Ecogenomics, School of Chemistry and Molecular Biosciences, The University of Queensland, Brisbane, QLD, Australia
- **Sarah Tozer**, Patient Safety and Quality, Clinical Excellence Queensland, Department of Health, Queensland Health, Australia
- **Paul KS Chan**, Department of Microbiology, The Chinese University of Hong Kong SAR, Hong Kong SAR, China
- **Evelyn SC Koay**, Department of Pathology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore; Molecular Diagnosis Centre, National University Hospital, Singapore
- **Hong Kai Lee**, Molecular Diagnosis Centre, National University Hospital, Singapore

- **Kok Keng Tee**, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia
- **Pieter LA Fraaij**, Department of Pediatrics, Erasmus Medical Centre, Rotterdam, Netherlands; ViroScience, Erasmus Medical Centre, Rotterdam, Netherlands
- **Lance Jennings**, Canterbury Health Laboratories and Department of Pathology & Biomedical Sciences, University of Otago, Christchurch, New Zealand
- **Jean-Michel Heraud**, National Influenza Center, Virology Unit, Institut Pasteur de Madagascar, Antananarivo 101, Madagascar
- **Matti Waris**, Institute of Biomedicine, University of Turku, Finland; Clinical Microbiology, Turku University Hospital, Turku, Finland
- **Hidekazu Nishimura**, Virus Research Center, Sendai Medical Center, Sendai, Japan
- **Aripuana Watanabe**, Department of Parasitology, Microbiology and Immunology, Federal University of Juiz de Fora, Juiz de Fora, Minas Gerais, Brazil
- **Theo Sloots**, Child Health Research Centre, The University of Queensland, Brisbane, Australia; Children's Health Queensland, Brisbane, Australia
- **Jen Kok**, Centre for Infectious Disease and Microbiology Laboratory Services, NSWHP-Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales, Australia; Centre for Infectious Diseases-Public Health, Westmead Hospital, Westmead, New South Wales, Australia
- **Dominic E Dwyer**, Centre for Infectious Diseases-Public Health, Westmead Hospital, Westmead, New South Wales, Australia; Pathology and Infectious Diseases, University of Sydney, Sydney, Australia
- **Marion PG Koopmans**, ViroScience, Erasmus Medical Centre, Rotterdam, Netherlands
- **David W Smith**, PathWest Laboratory Medicine, Perth, Western Australia, Australia; School of Medicine, University of Western Australia, Perth, Western Australia, Australia
- **Julian W Tang**, Department of Respiratory Sciences, University of Leicester, Leicester, UK; Clinical Microbiology, University Hospitals of Leicester NHS Trust, Leicester, UK
- **Tommy TY Lam**, State Key Laboratory of Emerging Infectious Diseases, School of Public Health, The University of Hong Kong, Hong Kong SAR, China; HKU-Pasteur Research Pole, Hong Kong SAR, China; Laboratory of Data Discovery for Health, Hong Kong SAR, China; Centre for Immunology and Infection, Hong Kong SAR, China; The Hong Kong Jockey Club Global Health Institute, Hong Kong SAR, China

Appendix B. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jinf.2025.106451.

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