

Comparative transmissibility of SARS-CoV-2, influenza virus, and common human coronaviruses in the post-pandemic era: a community- and household-based prospective cohort study in China

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Article

Keywords:

Posted Date: April 2nd, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9045299/v1>

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Additional Declarations:

Yes there is potential Competing Interest. BJC consults for AstraZeneca, Fosun Pharma, GlaxoSmithKline, Moderna, Pfizer, Roche and Sanofi Pasteur. The other authors report no other potential conflicts of interest.

We confirm that this study was approved by the Institutional Review Board of the Chinese Academy of Medical Sciences & Peking Union Medical College (Approval No. CAMS&PUMC-IEC-2024-068). Written informed consent was obtained from all adult participants and from parents or legal guardians for minors prior to enrollment.

1 Comparative transmissibility of SARS-CoV-2, influenza virus, and common human
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Abstract

Despite continuous viral evolution, it remains unclear whether SARS-CoV-2 has transitioned to transmission dynamics resembling those of other endemic respiratory pathogens in the post-pandemic era. During 2023–2025, we compared post-pandemic SARS-CoV-2 transmissibility with influenza virus (IFV) and common human coronaviruses (HCoV) transmissibility and SARS-CoV-2 transmissibility during and after the pandemic. Among 7,399 participants in 1,764 households, there were 704 acute-respiratory-infection index cases and 2,684 household contacts. Healthcare-seeking was higher for SARS-CoV-2 (17.6%) and IFV (28.1%) than HCoVs (9.8%). Post-pandemic SARS-CoV-2 household secondary attack risk (HSAR) (7.9%) was comparable to IFV (7.0%) and higher than HCoV (4.4%; $P<0.05$). Older adults had highest SARS-CoV-2 and HCoV transmissibility; children had highest IFV transmissibility, correlated with peak viral loads. Post-pandemic SARS-CoV-2 HSAR was lower than pandemic-period Omicron HSAR (adjusted odds ratio:3.30;95%CI:1.82–5.98) and Delta-period HSAR (aOR:2.46;95%CI:1.27–4.79). Pre-symptomatic transmission was observed for all three pathogens, highest for SARS-CoV-2 (35.7%). Our findings indicate that SARS-CoV-2 maintains high household transmissibility and requires substantial medical resources in the post-pandemic era, and non-pharmaceutical interventions and vaccination strategies that account for age-specific transmissibility and susceptibility merit study and consideration.

The COVID-19 pandemic was the most severe public health emergency of the 21st century, causing millions of deaths worldwide. During the pandemic, millions of lives were saved through public health and social measures (PHSMs) and pharmaceutical interventions¹⁻³. Research on SARS-CoV-2 transmissibility and transmission dynamics informed the design and use of these interventions. During the pandemic, SARS-CoV-2 was shown to have greater transmissibility than influenza virus, common human coronaviruses, SARS-CoV, and MERS-CoV, as evidenced by higher secondary attack rates⁴. Successive SARS-CoV-2 variants of concern were associated with greater transmissibility than the ancestral strain⁵. Infectiousness typically peaks around symptom onset, with presymptomatic and asymptomatic transmission both contributing substantially to spread⁶⁻⁹. On 5 May 2023, the World Health Organization declared that COVID-19 was no longer a Public Health Emergency of International Concern (PHEIC)¹⁰. Since then, very few studies have examined transmissibility and transmission dynamics of circulating strains^{11,12}, despite the need for ongoing monitoring to guide intervention development and use.

Households are important settings for transmission of respiratory viruses during seasonal epidemics and pandemics, in which prolonged, close contact and repeated exposures facilitate transmission¹³. Household studies generally follow a defined group of exposed, initially susceptible individuals, enabling reconstruction of transmission chains and assessment of transmissibility and transmission dynamics¹⁴. We hypothesized that in the post-pandemic period, SARS-CoV-2 transmissibility may evolve to resemble that of endemic respiratory viruses—driven by accumulating population immunity following relaxation of PHSMs—while remaining higher than transmissibility of other common respiratory pathogens. To address this hypothesis, we conducted a prospective household cohort study in Shanghai, China, to evaluate and compare transmissibility of post-pandemic SARS-CoV-2 with influenza virus and common human coronaviruses, and with SARS-CoV-2 variants that circulated during the pandemic. Findings may inform recommendations for duration of self-isolation, timing of vaccine booster doses, and protection of high-risk populations.

Results

Basic Characteristics

During November 2023 to October 2025, 7,399 participants from 1,764 households experienced 10,384 ARI episodes. Of 6,839 participants tested for respiratory pathogens, 1,283 had laboratory-confirmed SARS-CoV-2, influenza virus (IFV), or common human coronaviruses (HCoV) (Figure 1). Seasonal patterns were observed: SARS-CoV-2 peaked annually in July-August, IFV in December-January, and HCoV in September (Supplementary Figures 1 and 2). Among 1,283 laboratory-confirmed cases, 893 met the index-case definition. After excluding 101 households with coinfections involving other respiratory pathogens and 88 households where one or more members developed an ARI on the same day or within 14 days before the index case's symptom onset, 704 index case-households were included in the final analysis (Figure 1). Median household size was 4 (interquartile range [IQR], 2–5); 255 (36.3%) of 704 households had ≤ 3 members, and 384 (54.5%) included at least one child aged <15 years (Supplementary Table 2).

Among 171 IFV-positive index cases, 88 had A(H1N1), 64 A(H3N2), and 19 influenza B. Among 244 HCoV index cases, 99 were of NL63, 78 were OC43, 39 were HKU1, and 28 were 229E. The proportion of child index cases (<15 years) was higher for IFV (19.3%) and HCoV (15.6%) compared to SARS-CoV-2 (10.0%) ($P < 0.05$). Fever at onset was more common with IFV (70.2%) than SARS-CoV-2 (48.1%) and HCoV (20.5%) ($P < 0.05$). The most frequent symptoms at onset were sore throat (73.7%) for SARS-CoV-2, cough (71.9%) and fever (70.2%) for IFV, and runny nose (73.8%) for HCoV. Index cases with IFV or SARS-CoV-2 reported more symptoms at onset than those with HCoV. Healthcare-seeking behaviour varied by pathogen: 28.1% of IFV and 17.6% of SARS-CoV-2 index cases sought medical services during illness, compared to 9.8% for HCoV cases (Table 1).

There were 1,118 household contacts in SARS-CoV-2 households, 612 in IFV households, and 954 in HCoV households. Median contacts per index case ranged from 3 to 4 across pathogens. The proportion of child (<15 years) contacts was similar across groups, ranging from 14.4% to 15.3%.

Transmissibility

The number of secondary ARI cases per index case ranged from 0 to 3 across pathogens. The distribution of secondary ARI infections per index case was well characterised by a negative binomial distribution, with 11.3% to 16.7% of index cases responsible for 80% of household transmissions, indicating substantial heterogeneity in transmission dynamics (Supplementary Table 3, Supplementary Figure 3).

Crude HSARs were highest for SARS-CoV-2 (7.9%; 95% CI, 6.3–9.5), followed by IFV (7.0%; 95% CI, 5.1–9.2) and HCoV (4.4%; 95% CI, 3.1–5.8). Stratified by index case age, crude HSARs were highest among contacts of older index cases (≥ 60 years) for SARS-CoV-2 (11.1%; 95% CI, 7.8–14.9) and HCoV (7.1%; 95% CI, 4.3–10.2). In contrast, IFV had the highest crude HSAR associated with child index cases (10.9%; 95% CI, 5.0–17.8) (Figure 2, Supplementary Table 4, Supplementary Figure 4). Stratified by contact age, crude HSARs were highest among adult contacts for SARS-CoV-2 and IFV, whereas child contacts had the highest HSAR for HCoV (Figure 2, Supplementary Table 4, Supplementary Figure 4).

Results from the primary multivariable GLMM-logit model showed higher HSARs for SARS-CoV-2 and IFV than for HCoV (aOR 3.24, 95% CI 1.30–8.05; and 3.31, 1.21–9.07). Age-specific transmissibility varied by pathogen: older index cases (≥ 60 years) had higher transmissibility than adult index cases for SARS-CoV-2 (aOR 2.14, 1.01–4.52) and HCoV (aOR 3.44, 1.31–9.05), whereas IFV transmission was highest from child index cases (aOR 1.61, 1.02–2.50) (Figure 2). In susceptibility analyses, older household contacts had consistently lower susceptibility across pathogens. Findings were consistent in sensitivity analyses using a 7-day attribution window

(Supplementary Figure 5) and in pathogen-specific GLMMs (Supplementary Tables 5–7). Estimates from individual hazard models were consistent with observed age-specific patterns of transmissibility and susceptibility (Supplementary Figure 6). Compared with the post-pandemic period (this study), overall SARS-CoV-2 transmissibility was higher during Delta (aOR 2.46, 1.27–4.79) and pandemic-period Omicron (aOR 3.30, 1.82–5.98), but not significantly different from the ancestral strain (aOR 0.66, 0.40–1.10) (Figure 3A; Supplementary Table 8). Age-specific SARS-CoV-2 transmissibility did not vary significantly between pandemic and post-pandemic periods (Figure 3B). However, age-specific susceptibility profiles differed: older contacts, who had higher HSARs than adults during the pandemic, had reduced susceptibility in the post-pandemic era (aOR = 0.48; 95% CI: 0.27–0.86) (Figure 3C; Supplementary Table 8).

Viral Shedding and Infectivity

Temporal modelling of Ct values after illness onset showed that viral shedding for all three pathogens declined monotonically after symptom onset, with peak viral loads (lowest Ct value) occurring near the day of illness onset (Supplementary Figure 7). In multivariable analyses, age-specific viral burden largely mirrored HSAR patterns: older index cases had the highest viral loads at onset for all pathogens (Figure 4A–C), and for IFV, children had higher viral loads at onset than adults (Figure 4B). Index cases with ≥ 3 symptoms at onset tended to have lower Ct values (indicating higher viral loads) than those with < 3 symptoms (Figure 4D–F). This association was statistically significant for IFV, with a mean difference in Ct values at onset of -1.91 (95% CI: -3.55 to -0.27).

Key Epidemiological Parameters

We estimated latent-period distributions by fitting log-normal models. Median latent periods were 2.9 days (95% CI, 2.7–3.1) for SARS-CoV-2, 1.2 days (95% CI, 1.1–1.4) for IFV, and 2.8 days (95% CI, 2.6–3.0) for HCoV (Figure 5A, Supplementary Table 9). Latent periods were shorter than incubation periods by 0.7 days for SARS-

CoV-2, 0.2 days for IFV, and 0.4 days for HCoV, indicating possible presymptomatic transmission (Supplementary Table 9, Supplementary Figure 8).

Using fitted log-normal distributions, estimated serial interval distributions were similar across pathogens, with median and 95th percentile values of approximately 3 and 13 days, respectively (Figure 5B, Supplementary Table 9). Median generation time estimates were also similar across pathogens. However, the 95th percentile of the generation time was shorter for SARS-CoV-2 (7.9 days; 95% CI, 5.9–10.1) than for IFV (8.9 days; 95% CI, 7.3–11.3) and HCoV (10.8 days; 95% CI, 8.5–14.8) (Figure 5C, Supplementary Table 9).

By fitting Gamma distributions, we found that infectiousness profiles for all pathogens peaked around symptom onset: 0.2 days (95% CI, -0.1–0.5) before onset for SARS-CoV-2, 0.5 days (95% CI, 0.0–0.9) after onset for IFV, and 0.8 days (95% CI, 0.4–1.3) after onset for HCoV. The estimated proportion of presymptomatic transmission was highest for SARS-CoV-2 (35.7%; 95% CI, 26.7–45.2), followed by IFV (20.1%; 95% CI, 13.9–26.8) and HCoV (17.7%; 95% CI, 10.1–26.6). Approximately 54.0% (95% CI, 47.1–61.8) of SARS-CoV-2 transmission occurred within 3 days after symptom onset, compared with 49.0% (95% CI, 41.3–57.2) for IFV and 58.1% (95% CI, 51.5–65.5) for HCoV (Figure 5D, Supplementary Table 10).

Discussion

SARS-CoV-2 transmissibility in the post-pandemic period was lower than during the pandemic period for Delta and Omicron variants, yet remained higher than for HCoV and was comparable to that of IFV. SARS-CoV-2 and HCoV had broadly similar age-specific transmission patterns, with index cases aged 60 years or older having the highest transmissibility. In contrast, children (<15 years) had the highest IFV transmissibility. SARS-CoV-2 was characterized by a higher proportion of pre-symptomatic transmission and faster within-household transmission dynamics

compared to IFV and HCoV. Index cases infected with IFV or SARS-CoV-2 were more likely to seek healthcare services during illness than index cases infected with HCoV.

Direct comparisons of transmission features across respiratory viruses are inherently challenging due to differences in case definitions, demographics, testing intensity, use of PHSMs, and community immunity levels ¹⁵. A previous systematic review reported higher pooled household secondary attack rate (SAR) for pandemic SARS-CoV-2 than for influenza, SARS, MERS, and HCoV ^{4,5}, but estimates were derived from heterogeneous settings across countries, where viral, host, and environmental factors likely influenced transmissibility ¹⁵. Moreover, early SARS-CoV-2 SAR estimates were influenced by lower population immunity and likely by PHSM implementation, variant lineage, and changing testing practices. Here, we compared multiple respiratory viruses in parallel within the same temporal and spatial setting using a case-ascertained household design ¹⁶, providing a consistent framework for cross-pathogen comparison. We observed marked overdispersion: 11–17% of index cases generated 80% of household transmission, consistent with prior evidence for SARS-CoV-2 and influenza and suggesting targeted interventions could yield outsized benefits even in high-immunity settings. Our estimates further indicated that post-pandemic SARS-CoV-2 HSAR (7.9%) was substantially lower than pandemic estimates (15.5%–42.7%), yet remained higher than HCoV and similar to IFV. Ascertainment based on symptomatic secondary illness may miss asymptomatic or mild infections, whereas reliance on PCR alone can also underestimate HSAR because of imperfect sensitivity ¹⁵. Notably, despite immune-evasive and less severe properties of JN.1 ¹⁷—the dominant strain in our study—household transmissibility was lower than during Delta and early Omicron periods, suggesting accumulated immunity now overrides intrinsic variant advantages with periodic population-level immunity now established ¹⁵.

SARS-CoV-2 transmission dynamics increasingly resemble those of HCoV: older index cases had the highest transmissibility, whereas children were the main drivers of IFV household spread. The age-related pattern of SARS-CoV-2 infectiousness observed in our study resembles patterns in studies conducted during the pandemic that reported similar or higher transmissibility in older index cases compared with children^{4,18-22}, although one study found higher transmission among school-aged children²³. For IFV, children are generally considered key amplifiers of household transmission²⁴, although evidence from household studies remains mixed¹⁴. Data on HCoV are limited, but one cohort study reported higher household transmission risks in households with older adults²⁵. These findings aligned with our viral load analyses, which showed that age-specific viral peaks (at onset) mirrored age-specific transmissibility patterns for each pathogen. Furthermore, we observed reduced susceptibility to SARS-CoV-2 among older contacts in the post-pandemic period, contrasting with pandemic-era findings of higher susceptibility in adults and older individuals than in children^{19,21,26,27}. Differences in age-specific transmissibility and susceptibility may reflect pre-existing immunity, antibody response kinetics, or antibody levels at the time of infection²⁸⁻³¹. For example, previous influenza studies have suggested that over one-third of individuals with serologic evidence of infection do not shed detectable virus in respiratory secretions²⁸.

Infectiousness for SARS-CoV-2, IFV, and HCoV peaked around symptom onset, with substantial presymptomatic transmission; SARS-CoV-2 had the highest proportion (35.7%). Pre-symptomatic transmission in all three pathogens highlights the limited effectiveness of symptom-based interventions, such as isolating symptomatic individuals or quarantining their contacts. Early SARS-CoV-2 studies suggested that over half of transmission events occurred before symptom onset, and that a combination of rapid case isolation, contact tracing, and population-level interventions such as mask use and restrictions on gatherings was necessary to achieve control^{6,32}. Although such measures are impractical in the post-pandemic context, our findings indicate transmission risk is highest on the day of symptom

onset and most household transmission occurs within three days thereafter, underscoring the importance of prompt symptom recognition and immediate self-isolation³³. Previous household studies have also found that timely interventions such as hand hygiene and mask use implemented soon after symptom onset can reduce secondary infections^{13,34,35}. Future randomized trials could investigate how timing and implementation of these interventions influence household transmission of respiratory viruses.

Our study has limitations. First, many secondary ARI cases were not laboratory confirmed. Asymptomatic or mild infections may therefore have been missed, leading to underestimation of household SARs. Pathogen-specific SAR estimates based on combined case-ascertainment approaches, including symptoms, PCR, and serology, should be further explored. Second, although our multivariable analyses adjusted for several demographic and clinical covariates, residual confounding due to unmeasured factors—such as pre-existing immunity, antiviral use, prophylactic measures, or household-level behaviors during illness (e.g., mask wearing, isolation practices, ventilation)—may have influenced the observed associations. Third, we used viral load data from index cases as a proxy for viral kinetics and transmissibility. However, index cases identified through reporting ARI in this study may experience more severe illness than the general population³⁶, potentially limiting generalizability. Fourth, we excluded households with secondary ARI onset on the same day or within 14 days before the index case’s symptom onset. While this strengthens temporal inference, it excludes negative serial intervals and may slightly bias estimated serial intervals upward.

In conclusion, SARS-CoV-2 transmissibility was substantially lower in the post-pandemic era than during the pandemic. Nevertheless, it remains more transmissible—and is associated with greater healthcare demand than HCoV and retains substantial presymptomatic transmission, indicating that it has not fully converted to a ‘common cold’-like respiratory virus. Further research on age-targeted

vaccination strategies—particularly strategies balancing differences in infectiousness (e.g., elevated in older individuals) and susceptibility (e.g., reduced in older adults)—is needed to optimize vaccination impact. Our study highlights the importance of integrated multi-virus surveillance systems and supports public health messaging focused on rapid testing and early isolation during the first few days of illness, when transmission risk is highest.

Methods

Study Design and Participants

In November 2023, we initiated a prospective, community- and household-based cohort study in Pudong New Area District, Shanghai, China, to evaluate community burden and transmission dynamics of influenza virus, respiratory syncytial virus (RSV), SARS-CoV-2, and other respiratory pathogens (including viruses, bacteria, and fungi) ¹⁶. Eligible households had to include more than two individuals six months or older who resided in the area for at least six months. Recruitment was conducted by family physicians in 47 community health service centers; baseline participant and household data were obtained with questionnaires at enrolment. Throughout the follow-up period, participants were monitored for acute respiratory infections (ARI) with weekly phone surveys by study staff and online self-reporting. Nasopharyngeal and oropharyngeal swabs were obtained voluntarily from participants with ARI symptoms within five days of symptom onset. Samples were tested using a multiplex real-time quantitative PCR assay for respiratory pathogens, including SARS-CoV-2; influenza virus (subtypes A[H1N1], A[H3N2], and B); common human coronaviruses (229E, NL63, OC43, and HKU1); and other respiratory viruses. Nucleic acid was extracted using the Roche (China) Holding kit (catalog number: 6369750), and pathogen detection was conducted with the Thermo Fisher Scientific RespiFinder® panel (catalog number: 4398986).

Index cases were the first laboratory-confirmed, multiplex-PCR-positive ARI case for a given pathogen (SARS-CoV-2, influenza virus [IFV], or common human coronaviruses [HCoV]) in each household during follow-up. Households could contribute multiple index episodes for different pathogens over time and were eligible to re-enter analyses for subsequent episodes. Index cases co-infected with two or more pathogens were excluded³⁷. For index cases, we recorded demographic characteristics, underlying conditions, clinical features, medical service-seeking behaviour, and viral RNA levels as proxied by PCR cycle threshold (*Ct*) values and sampling time. For household contacts of index cases, demographic characteristics, underlying conditions, and household sizes were extracted from baseline data. To reduce misclassification of co-primary cases and prior within-household transmission, we excluded households if any member reported respiratory symptoms on the same day as, or within 14 days before the index case's illness onset. Secondary ARI cases were household contacts who developed an ARI within 14 days of the index case's symptom onset, with either laboratory confirmation of the same pathogen as the index case or no testing performed. For secondary ARI cases, we recorded dates of illness onset and symptom onset. The definitions of household contacts and ARI are in the Supplementary Materials.

Statistical Analysis

Crude household secondary attack risks (HSARs) were estimated for each pathogen as the proportion of household contacts who developed an ARI within 14 days of the index case's illness onset. We calculated 95% confidence intervals (CIs) for overall and age-specific HSAR using a cluster bootstrap method with 1,000 resamples, resampling with replacement to account for within-household correlation. To quantify transmission heterogeneity, the number of secondary infections per index case was modelled using a negative binomial distribution (Supplementary Materials).

We compared pathogen-specific transmissibility by combining pathogen-specific index–contact datasets. A mixed-effects multiple logistic regression model (GLMM)

with binomial distribution and logit link was used to estimate factors associated with secondary ARI, with a random intercept for household to account for within-household correlation. We included interaction terms between the pathogen and the age groups of index cases and contacts to evaluate age-specific transmissibility and susceptibility across pathogens. Potential confounders (listed in Supplementary Table 1) were then added sequentially to the model based on biological plausibility and prior evidence. At each step, we assessed model fit using Akaike's Information Criterion (AIC), and variables were retained only if they improved fit ($\Delta AIC \geq 2$). The final model included interaction terms between pathogen and ages of index cases and contacts, household size, and index-case characteristics (date of onset, total symptom count, and presence of cough).

We conducted three sensitivity analyses. First, we used a shorter attribution window (secondary ARI within 7 days of index case symptom onset instead of 14 days) to assess robustness to window choice and potential community-acquired or tertiary infections. Second, to verify whether age-pathogen interaction effects were influenced by outliers, we fitted separate GLMMs for each pathogen to assess factors influencing transmissibility and susceptibility. Third, to address potential for community-acquired infections and tertiary transmission^{14,15}, we fitted an individual-level hazard model to evaluate probabilities of community-acquired infection and within-household transmission, and to assess associated factors (Supplementary Materials).

We obtained individual-level data from household transmission studies conducted during the COVID-19 pandemic on the ancestral strain³⁸, Delta variant⁸, and Omicron variants⁷. We used these data to compare SARS-CoV-2 transmissibility and transmission characteristics between the post-pandemic and pandemic periods using a GLMM-logit model that accounted for household size and the interactions between SARS-CoV-2 strain and the ages of index cases and contacts.

We analysed viral shedding kinetics to explore factors influencing viral burden, with the minimum cycle threshold (Ct) value serving as a proxy for peak viral load. We first plotted Ct values from symptomatic index cases against days since symptom onset and fitted generalised additive models (GAMs) to describe temporal trends in Ct values for each pathogen. We then fitted pathogen-specific generalized linear models (GLMs) to find factors associated with viral burden. We used AIC values to assess goodness-of-fit for different GLM models (Supplementary Materials).

We constructed transmission chains between index cases (infectors) and secondary household cases (infectees) to estimate key epidemiological parameters. Bayesian statistical models and Hamiltonian Monte Carlo methods were used to estimate the latent period, serial interval, generation time, and infectiousness profile (Supplementary Materials). The models combined individual-level household data with published incubation period distributions^{7,39}, incorporated as fixed priors to anchor infection time estimation. To estimate the latent period distribution, a viral shedding window was established for each infector, during which detectable virus shedding was estimated to begin. The lower bound was the infection time, and the upper bound was the sampling date of the first positive PCR result. Latent period distributions were estimated separately for each pathogen in the Bayesian statistical model based on the infection time and interval-censored viral shedding windows. Serial interval parameters were estimated from onset times in infector–infectee pairs. Generation time distributions were derived from infection times of infectors and infectees, and infectiousness profiles were estimated using the infector’s onset time relative to the infectee’s infection time. Serial interval, generation time, and infectiousness profiles were jointly estimated using the transmission chains between infectors and infectees in the model (Supplementary Materials).

All analyses were performed using R software, version 4.4.1 (R Foundation for Statistical Computing).

417 ***Role of the funding source***

418 The funding bodies had no role in the design of the study, the collection, analysis, and
419 interpretation of data, or writing of the manuscript.

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Contributors

All authors meet the ICMJE criteria for authorship. This study was conceived by LH, ZL, and WY. HX, CY, and JY collected and analyzed the data. HX wrote the first draft of the manuscript. All authors provided critical review and revision of the text and approved the final version.

Declaration of interests

BJC consults for AstraZeneca, Fosun Pharma, GlaxoSmithKline, Moderna, Pfizer, Roche and Sanofi Pasteur. The other authors report no other potential conflicts of interest.

Data sharing statement

De-identified individual participant-level data will be available on reasonable request, upon written request to the corresponding author following publication.

Acknowledgements

This study was approved by Chinese Academy of Medical Sciences & Peking Union Medical College's Institutional Review Board (no. CAMS&PUMC-IEC-2024-068). This study was supported by the National Key Research and Development Program of China (2023YFC2308701), Chinese Academy of Medical Sciences Innovation Fund for Medical Sciences (2023-I2M-3-011), Non-profit Central Research Institute Fund of Chinese Academy of Medical Sciences (2022-ZHCH330-01), and Beijing Natural Science Foundation (L242053). We thank the staff of the Shanghai Pudong New Area Center for Disease Control and Prevention and the community health workers in Shanghai Pudong New Area for their essential contributions to field investigations, household enrollment, and data collection.

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557 **Table 1: Demographic and clinical characteristics of index cases and household**
558 **members with secondary ARI infections, overall and by pathogen**

Characteristic	Overall	SARS-CoV-2	IFV	HCoV
Index cases				
No. of cases	704	289	171	244
Age, years (Median, IQR)	41.0 (29.0-62.0)	44.0 (33.0-64.0)	40.0 (25.5-60.5)	39.0 (27.8-62.0)
<15	100 (14.2)	29 (10.0)	33 (19.3)	38 (15.6)
15-59	400 (56.8)	171 (59.2)	94 (55.0)	135 (55.3)
≥60	204 (29.0)	89 (30.8)	44 (25.7)	71 (29.1)
Male sex	301 (42.8)	112 (38.8)	84 (49.1)	105 (43.0)
With underlying conditions ^a	188 (26.7)	76 (26.3)	53 (31.0)	59 (24.2)
Clinical features at onset				
(% of index cases)				
Fever	309 (43.9)	139 (48.1)	120 (70.2)	50 (20.5)
Cough	422 (59.9)	167 (57.8)	123 (71.9)	132 (54.1)
Runny nose	423 (60.1)	153 (52.9)	90 (52.6)	180 (73.8)
Sore throat	473 (67.2)	213 (73.7)	101 (59.1)	159 (65.2)
Headache	222 (31.5)	86 (29.8)	72 (42.1)	64 (26.2)
Phlegm	209 (29.7)	84 (29.1)	71 (41.5)	54 (22.1)
Fatigue	285 (40.5)	120 (41.5)	84 (49.1)	81 (33.2)
No. of symptoms at onset (median, IQR)	3.0 (2.0-4.0)	3.0 (2.0-4.0)	4.0 (3.0-5.0)	3.0 (2.0-4.0)
<3	234 (33.2)	92 (31.8)	33 (19.3)	109 (44.7)
≥3	470 (66.8)	197 (68.2)	138 (80.7)	135 (55.3)
Sought healthcare services	123 (17.5)	51 (17.6)	48 (28.1)	24 (9.8)
Household secondary ARI cases				
No. of secondary ARI cases	173	88	43	42
Age, years (median, IQR)	54.0 (33.0-69.0)	56.0 (33.0-69.0)	52.0 (32.0-68.0)	52.0 (32.0-69.0)
<15	30 (17.3)	15 (17.0)	7 (16.3)	8 (19.0)
15-59	84 (48.6)	38 (43.2)	27 (62.8)	19 (45.2)
≥60	59 (34.1)	35 (39.8)	9 (20.9)	15 (35.7)
Male sex	81 (46.8)	44 (50.0)	19 (44.2)	18 (42.9)
Clinical features at onset				
(% of secondary ARI cases)				
Fever	55 (31.8)	28 (31.8)	21 (48.8)	6 (14.3)
Cough	102 (59.0)	43 (48.9)	33 (76.7)	26 (61.9)
Runny nose	99 (57.2)	51 (58.0)	22 (51.2)	26 (61.9)
Sore throat	101 (58.4)	56 (63.6)	19 (44.2)	26 (61.9)
Headache	36 (20.8)	18 (20.5)	11 (25.6)	7 (16.7)
Phlegm	45 (26.0)	16 (18.2)	19 (44.2)	10 (23.8)
Fatigue	53 (30.6)	20 (22.7)	20 (46.5)	13 (31.0)
No. of symptoms at onset (median, IQR)	3.0 (2.0-4.0)	2.0 (2.0-3.0)	3.0 (2.0-4.0)	2.0 (2.0-4.0)

<3	83 (48.0)	46 (52.3)	15 (34.9)	22 (52.4)
≥ 3	90 (52.0)	42 (47.7)	28 (65.1)	20 (47.6)
Sought healthcare services	26 (15.0)	14 (15.9)	10 (23.3)	2 (4.8)
No. of lab-confirmed cases	62 (35.8)	40 (45.5)	18 (41.9)	4 (9.5)

559 **Abbreviations:** SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; IFV,
560 influenza virus; HCoV, common human coronaviruses (229E, NL63, OC43, HKU1); IQR,
561 interquartile range; ARI, acute respiratory illness.

562 ^a Underlying conditions included diabetes mellitus, hypertension, heart disease, asthma, chronic
563 bronchitis, chronic obstructive pulmonary disease, chronic kidney disease, myocardial
564 infarction, stroke, cancers, and others.

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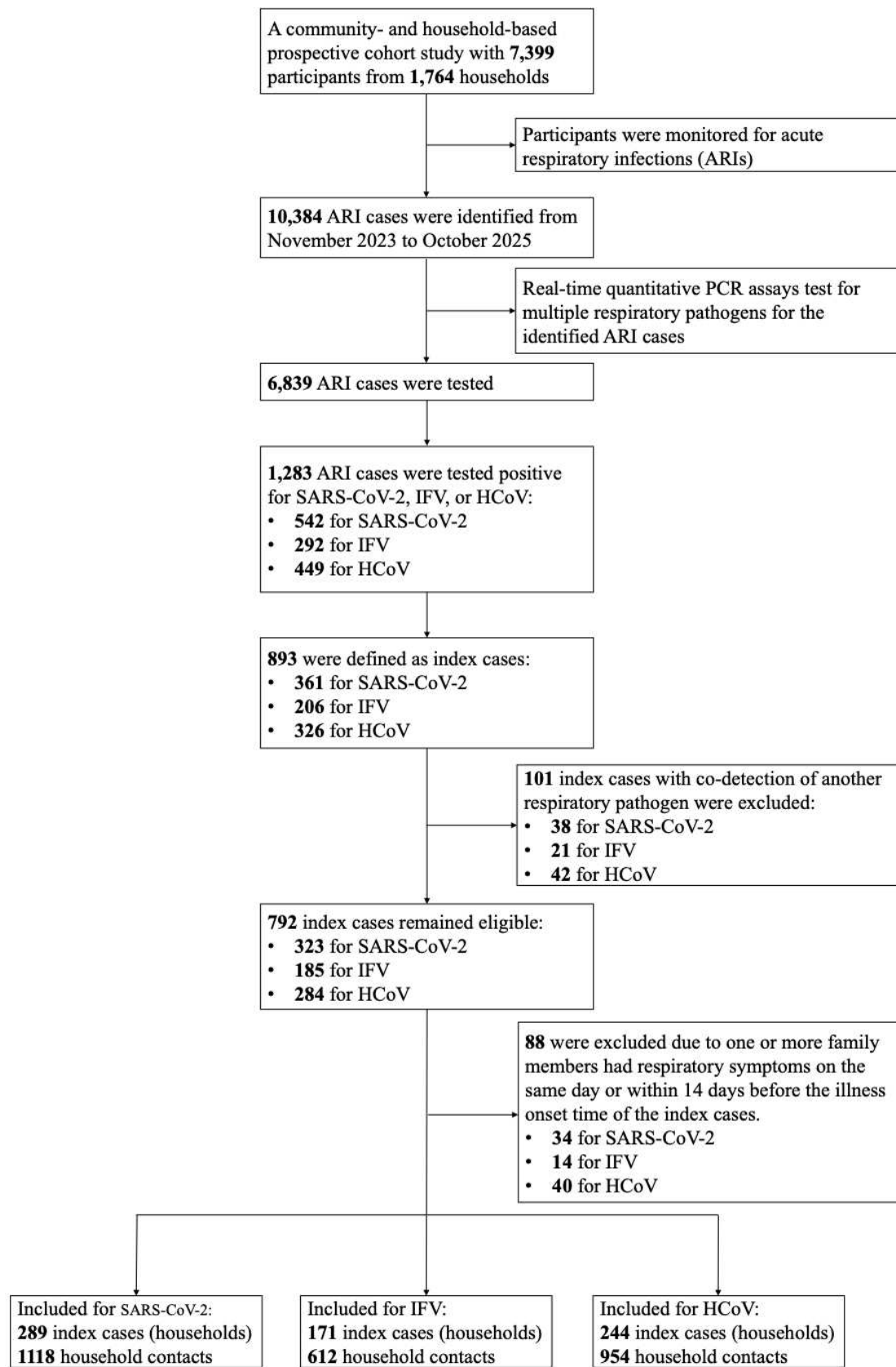


Figure 1: Flowchart of household recruitment and case ascertainment

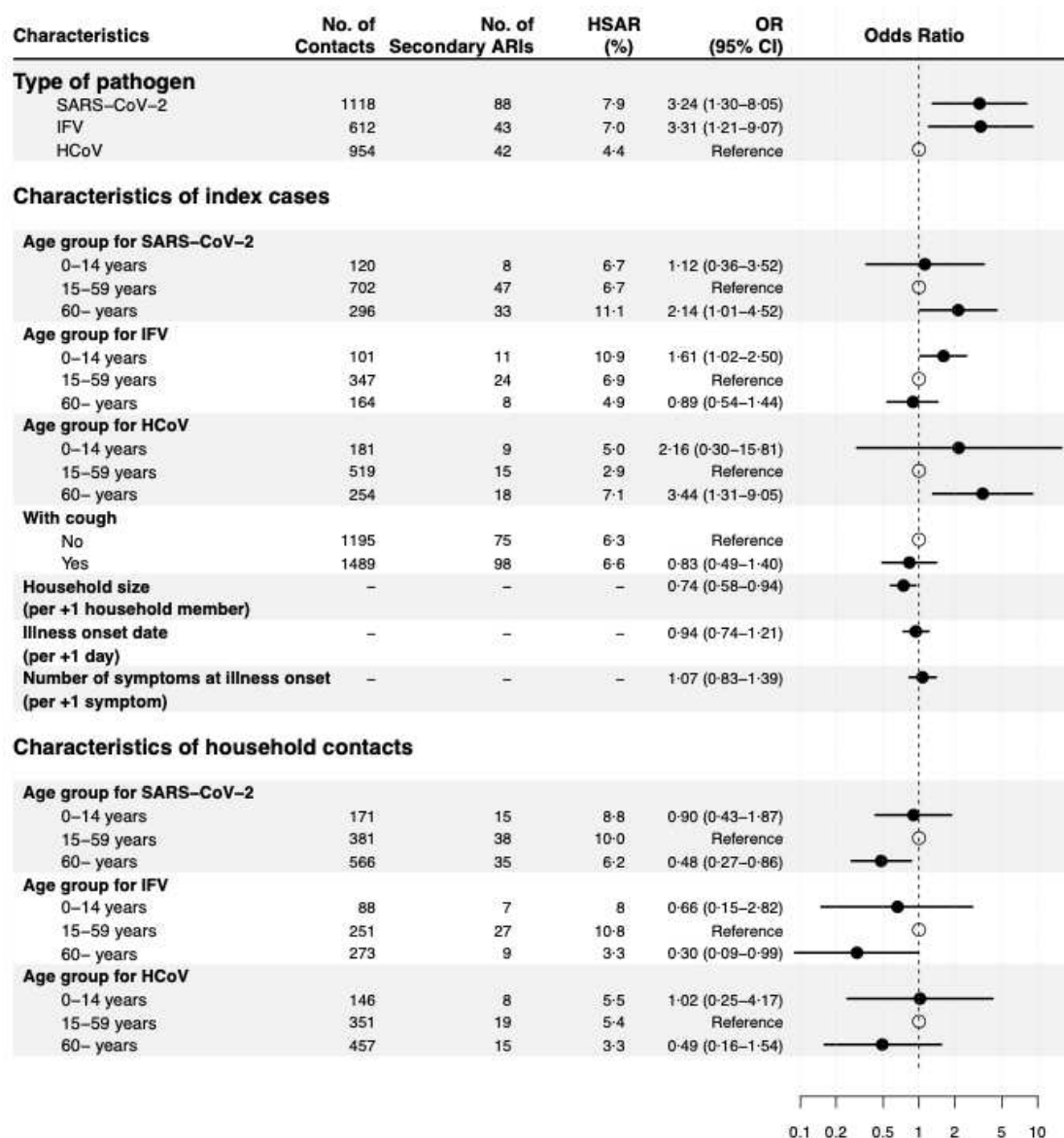


Figure 2: Adjusted odds ratios from mixed-effects logistic regression model of pathogen- and population-specific transmissibility and susceptibility based on household secondary attack risks (HSAR). Models include index-case and contact characteristics with a random intercept for household. Hollow points represent the reference group; black points represent adjusted ORs; horizontal lines represent 95% confidence intervals. HSAR = proportion of contacts with ARI within 14 days after index-case onset.

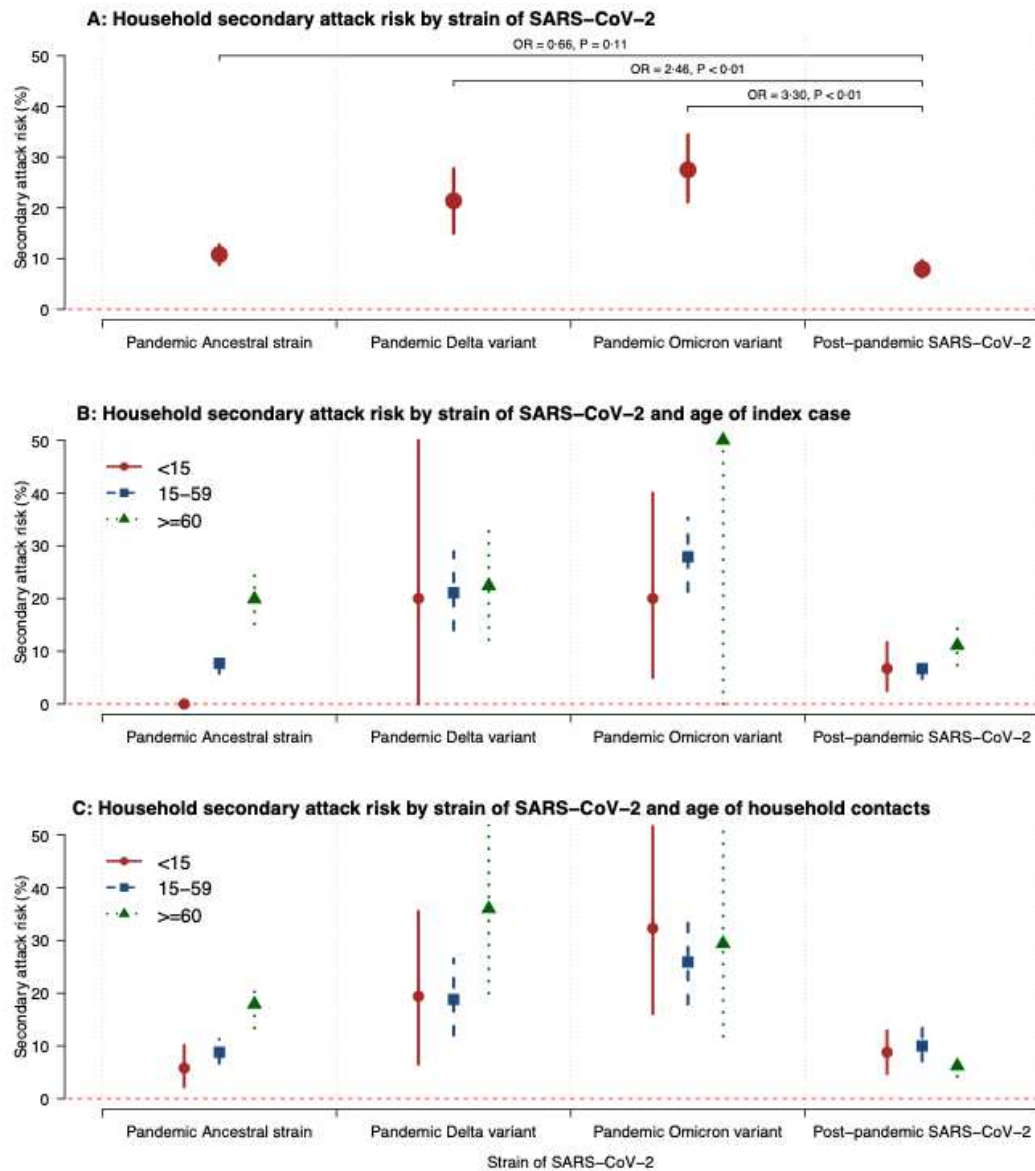


Figure 3: Household secondary attack risk (HSAR, %) by SARS-CoV-2 variant-period overall (A) and stratified by index-case age (B) and household contact age (C). Points show HSAR with 95% confidence intervals. Brackets in panel A indicate odds ratios from mixed-effects logistic regression comparing each pandemic-period strain of SARS-CoV-2 with post-pandemic SARS-CoV-2 (reference).

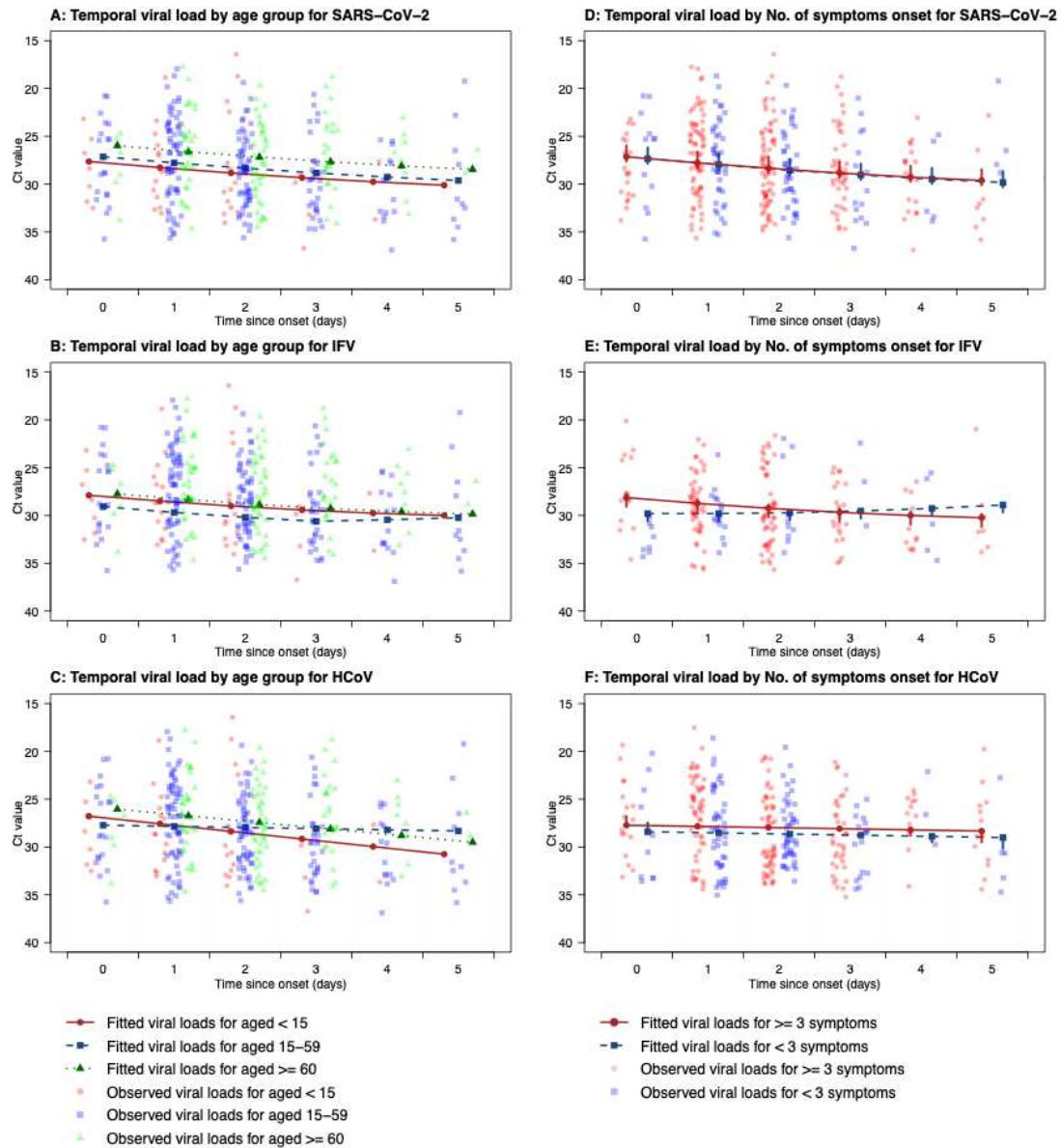


Figure 4: Observed and model-predicted cycle threshold (C_t) values by days since symptom onset (day 0), stratified by index-case age group (A–C) and symptom burden at illness onset (D–F). Points represent observed C_t values; solid lines represent generalized linear model fits with 95% confidence intervals each day since symptom onset (day 0). Lower C_t values indicate higher viral loads (higher viral RNA). C_t values are assay-dependent; comparisons are intended within pathogen rather than across pathogens.

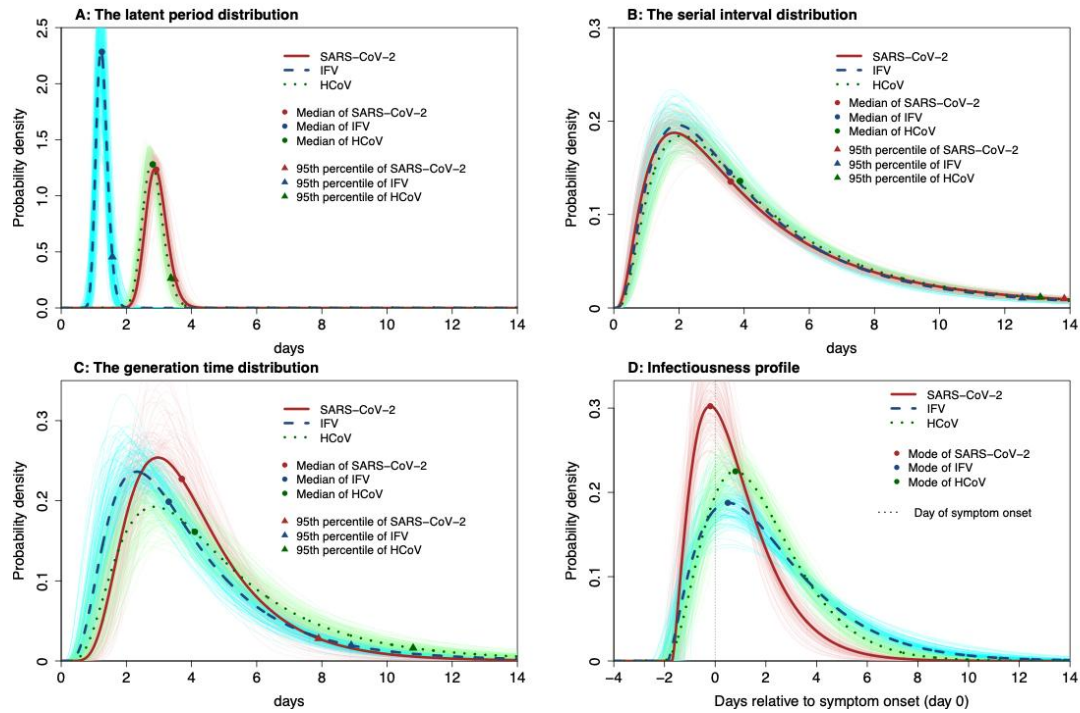


Figure 5: Probability density distributions of the latent period (A), serial interval (B), generation time (C), and infectiousness profile relative to symptom onset (D) for each pathogen. The infectiousness profile represents the probability of infection in contacts in relation to the symptom onset of index cases. Curves show posterior median estimates from Bayesian models; shaded areas indicate 95% credible intervals.

Supplementary Files

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