

Title: Human mobility impacts the transmission of common respiratory viruses: A modeling study of the Seattle metropolitan area

Short title: Mobility and citywide respiratory virus dynamics

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Abstract

Many studies have used mobile device location data to model SARS-CoV-2 dynamics, yet relationships between mobility behavior and endemic respiratory pathogens are less understood. We studied the impacts of human mobility on the transmission of SARS-CoV-2 and 16 endemic viruses in Seattle over a 4-year period, 2018-2022. Before 2020, school-related foot traffic and large-scale population movements preceded seasonal outbreaks of endemic viruses. Pathogen circulation dropped substantially after the initiation of stay-at-home orders in March 2020. During this period, mobility was a positive, leading indicator of transmission of all endemic viruses and lagged SARS-CoV-2 activity. Mobility was briefly predictive of SARS-CoV-2 transmission when restrictions relaxed in summer 2020 but associations weakened in subsequent waves. The rebound of endemic viruses was heterogeneously timed but exhibited stronger relationships with mobility than SARS-CoV-2. Mobility is most predictive of respiratory virus transmission during periods of dramatic behavioral change, and, to a lesser extent, at the beginning of epidemic waves.

Teaser: Human mobility patterns predict the transmission dynamics of common respiratory viruses in pre- and post-pandemic years.

37 **Introduction**

38 In early 2020, there was widespread adoption of public health measures to slow the spread of severe acute
39 respiratory syndrome coronavirus 2 (SARS-CoV-2). A variety of non-pharmaceutical interventions (NPIs) were
40 implemented in most countries to reduce contacts between infected and susceptible individuals, including shelter-in-
41 place or stay-at-home orders, gathering restrictions, school and business closures, and travel bans. These
42 interventions were effective at reducing not only SARS-CoV-2 transmission but also the spread of other directly
43 transmitted respiratory pathogens (1-9). Many endemic respiratory viruses did not return to widespread circulation
44 until the end of 2020 or 2021 (3, 8-10), coinciding with the gradual lifting of social distancing measures and mask
45 mandates.

46 During the COVID-19 pandemic, aggregated location data from mobile phones became an important source of
47 information on changes in population-level movements and were used to model SARS-CoV-2 dynamics and assess
48 the effectiveness of NPIs on SARS-CoV-2 transmission (11-13). However, few studies have explored relationships
49 between human mobility and the dynamics of endemic respiratory pathogens during the pandemic. Here we define
50 "endemic" pathogens as those that have regular periodic cycles and stable rates of infection in outbreak periods. Due
51 to lack of circulation of endemic respiratory viruses in the first years of the pandemic, population susceptibility to
52 these pathogens is expected to have increased, leading to earlier, larger, or more severe epidemics a few months later
53 (14, 15). Understanding the influence of mobility patterns on the dynamics of endemic pathogens is important for
54 predictive purposes, especially as perturbed circulation can lead to overlapping epidemics of different pathogens and
55 in turn put extreme pressure on the healthcare system (e.g., the US "triple-demic" during winter 2022-2023)(16).

56 Here, we leverage fine-grained respiratory surveillance data and mobile device location data to explore links
57 between population behavior and the transmission of 17 common respiratory viruses in the greater Seattle,
58 Washington region over a 4-year period, 2018 – 2022. These viruses include SARS-CoV-2, influenza viruses
59 (A/H3N2, A/H1N1, and B), respiratory syncytial viruses (RSV A and B), seasonal coronaviruses (hCoV 229E,
60 OC43, HKU1, and NL63), human metapneumovirus (hMPV), human parainfluenza viruses (hPIV 1, 2, 3, and 4),
61 human rhinovirus (hRV), and adenovirus (AdV).

62 **Results**

63 **Study overview**

64 We use detailed individual-level surveillance data from the Seattle Flu Study (SFS), which launched in the Fall of
65 2018 to improve detection and control of epidemics and pandemics (17). SFS carried out intensive hospital and
66 community-based surveillance with systematic molecular testing of nasal swabs for up to 26 respiratory pathogens
67 (17) (Table S1). Our study spans November 19, 2018, to June 30, 2022, during which respiratory specimens were
68 collected from individuals with and without respiratory illness across a variety of sites throughout the Seattle
69 metropolitan region, as previously described (17-23). In total, 138,060 respiratory specimens were screened for the
70 presence of 24 or 26 pathogens (Table S1), and we retained 80,846 specimens after limiting our analysis to
71 symptomatic individuals and discarding samples with missing metadata or from multiple testing (Table 1, Table S2).
72 25.3% (N = 20,640) of samples were collected in hospitals, and 74.5% (N = 60,206) were collected through
73 community-based testing, including outpatient clinics, kiosks stationed in high foot traffic areas (17), swab-and-send
74 at-home testing programs (20, 22), and King County COVID-19 drive through testing sites (Table 1, Table S2). The
75 majority of hospital residuals were collected from younger age groups, while most community-based samples were
76 collected from adults (Table 1, Figure S1, Table S2).

77 **Table 1. Participant characteristics.** Home residence, sex, and age distributions for individuals contributing
 78 respiratory specimens to different Seattle Flu Study (SFS) surveillance arms, including hospitals, SFS community
 79 testing (e.g., kiosks, swab-and-send at-home testing, outpatient clinics), and Public Health – Seattle & King County
 80 (PHSKC) COVID-19 drive through testing sites.

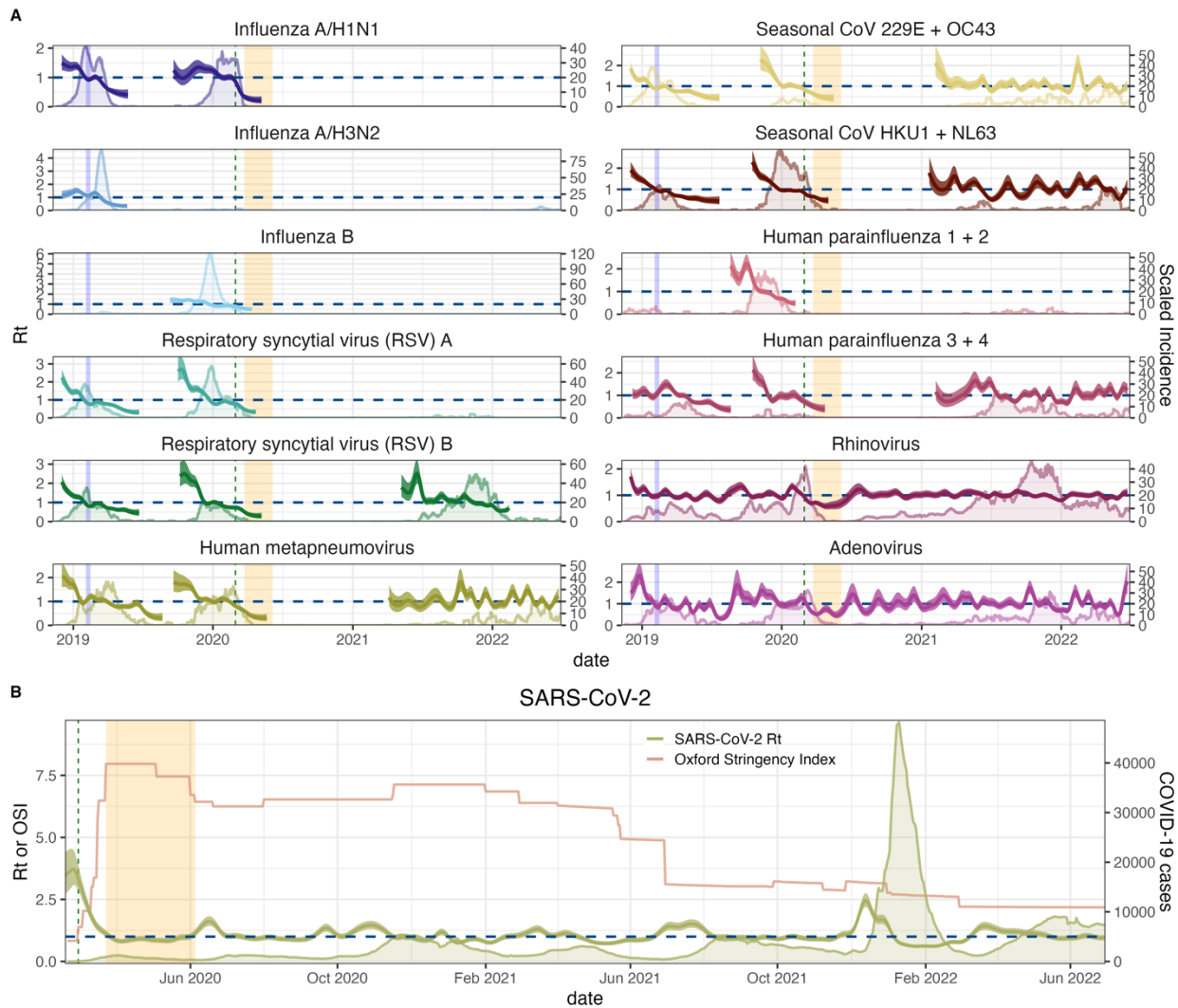
Variable	Overall, N = 80,846 ¹	Site Category		
		Hospital residuals, N = 20,640 ¹	SFS community surveillance, N = 52,272 ¹	PHSKC COVID-19 drive thru sites, N = 7,934 ¹
Home residence				
North King County	47,385 (59%)	10,456 (51%)	31,338 (60%)	5,591 (70%)
South King County	17,955 (22%)	4,430 (21%)	11,846 (23%)	1,679 (21%)
Puget Sound, non- King County ²	15,506 (19%)	5,754 (28%)	9,088 (17%)	664 (8.4%)
Sex				
Female	44,670 (56%)	9,399 (46%)	30,887 (59%)	4,384 (56%)
Male	35,771 (44%)	11,238 (54%)	21,071 (41%)	3,462 (44%)
Other (Missing)	44 (<0.1%) 361	0 (0%) 3	44 (<0.1%) 270	0 (0%) 88
Mean age	31 (21)	15 (20)	35 (18)	47 (18)
Age group				
<1	3,455 (4.3%)	2,986 (14%)	469 (0.9%)	0 (0%)
1-4	9,834 (12%)	6,363 (31%)	3,341 (6.4%)	130 (1.6%)
5-17	10,690 (13%)	5,823 (28%)	4,585 (8.8%)	282 (3.6%)
18-49	40,297 (50%)	3,307 (16%)	33,019 (63%)	3,971 (50%)
50-64	10,897 (13%)	1,209 (5.9%)	7,515 (14%)	2,173 (27%)
≥65	5,673 (7.0%)	952 (4.6%)	3,343 (6.4%)	1,378 (17%)
Broad age group				
<5	13,289 (16%)	9,349 (45%)	3,810 (7.3%)	130 (1.6%)
≥5	67,557 (84%)	11,291 (55%)	48,462 (93%)	7,804 (98%)
¹ n (%)				
² Pierce, Snohomish, Kitsap, San Juan, Whatcom, Skagit, Island, Clallam, Jefferson, Mason, and Thurston counties.				

81 Over the course of the four-year study, 40.6% (N = 32,841) of specimens tested positive for at least one respiratory
 82 pathogen (including SARS-CoV-2), 32.4% (N = 26,182) were positive for at least one endemic respiratory
 83 pathogen, and 9.1% (N = 7,374) were positive for more than one pathogen. Prior to the start of Washington’s
 84 COVID-19 restrictions in March 2020, the most prevalent pathogens among positive samples were influenza
 85 A/H1N1 virus (17.9%), followed by hRV (15.4%), influenza A/H3N2 virus (13.9%), influenza B virus (12.2%), and
 86 RSV A (9.7%) (Figure S2). After March 2020, the most prevalent pathogens were SARS-CoV-2 (39.5%), hRV
 87 (35.4%), and AdV (5.1%) (Figure S2).

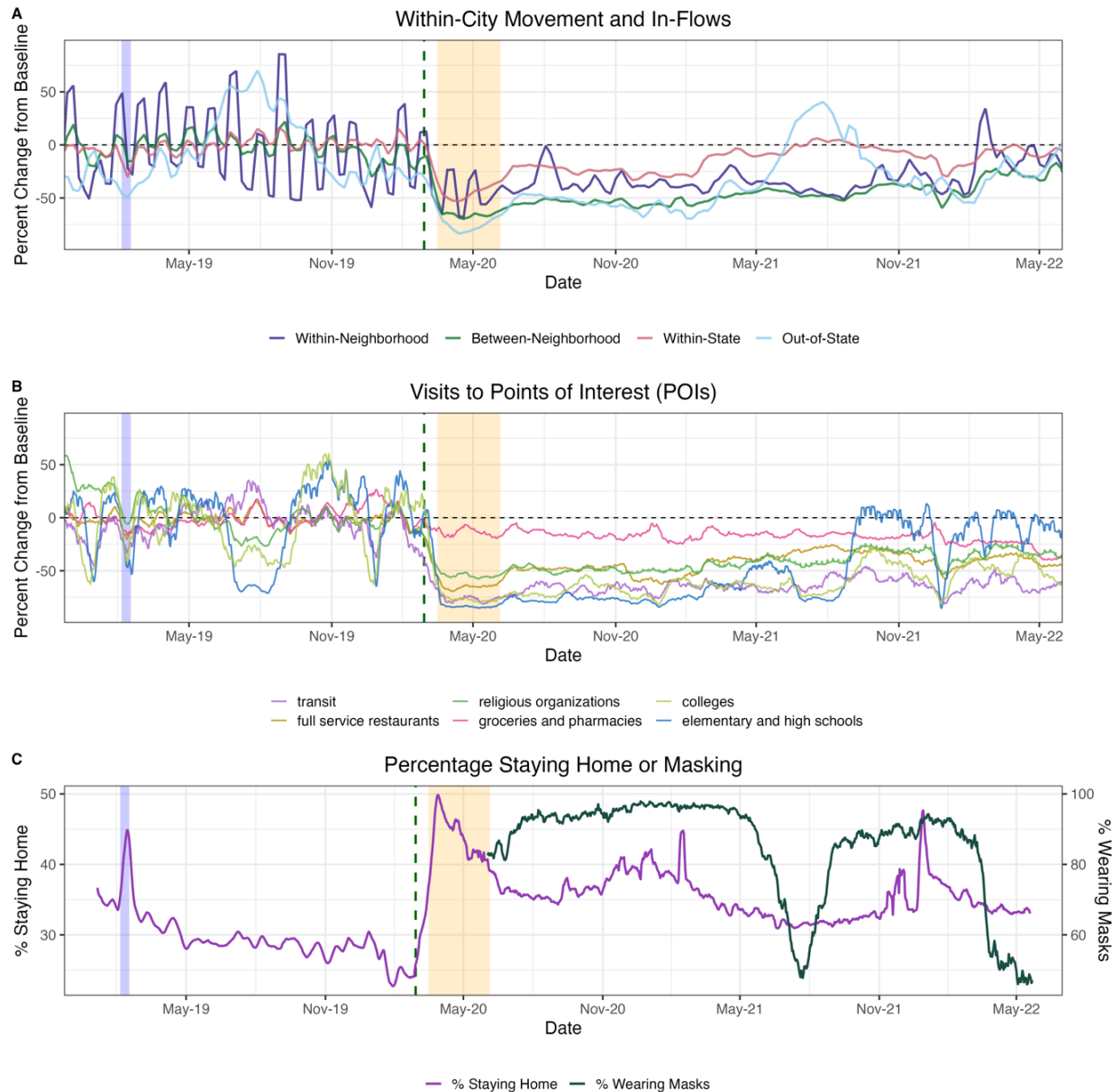
88 We reconstructed daily incidences for SARS-CoV-2 and each endemic pathogen, adjusting for testing volume over
 89 time, age, clinical setting, and local syndromic respiratory illness rates (Figure S3). Although SFS tested respiratory
 90 specimens for up to 26 pathogens, we limited our analysis to 17 viruses with sufficient sampling (≥ 400 positive
 91 samples during 2018-2022), including SARS-CoV-2, influenza A/H1N1, A/H3N2, and B viruses, RSV A and B,
 92 hCoV 229E, OC43, HKU1, and NL63, hPIV 1, 2, 3, and 4, hMPV, hRV, and AdV (Figure 1, Figure S3). Due to
 93 laboratory assay limitations (Table S1), we grouped epidemiologically distinct strains into one incidence time series
 94 each for hCoV 229E and hCoV OC43 (hereon hCoV 229E + OC43), hCoV HKU1 and hCoV NL63 (hCoV HKU1 +
 95 NL63), hPIV 1 and hPIV 2 (hPIV 1 + 2), hPIV 3 and hPIV 4 (hPIV 3 + 4), hRV, and AdV. hPIV 3 likely comprises
 96 most of hPIV 3 + 4 incidence because hPIV 4 infections are detected infrequently and tend to be mild or
 97 asymptomatic (24).

98 Based on reconstructed incidences, we used semi-mechanistic epidemiological models to measure the time-varying
 99 intensity of transmission via the daily effective reproduction number (R_t)(25, 26) (Figure 1). To generate R_t based
 100 on dates of infection, we convolved over uncertain incubation periods and reporting delay distributions (i.e., delays
 101 from symptom onset to testing), wherein delays were informed by our individual-level surveillance data. We used
 102 aggregated mobile device location data from SafeGraph and Meta Data for Good to assess the effects of population-
 103 level movements on citywide respiratory virus dynamics in pre- and post-pandemic years (Figures 2-3). During the
 104 pandemic period, we also considered the effects of non-mobility behavioral indicators, including the stringency of

105 Washington’s government response to COVID-19, measured by the Oxford Stringency Index (27) (Figure 1), and
 106 the proportion of individuals masking in public (28) (Figure 2).

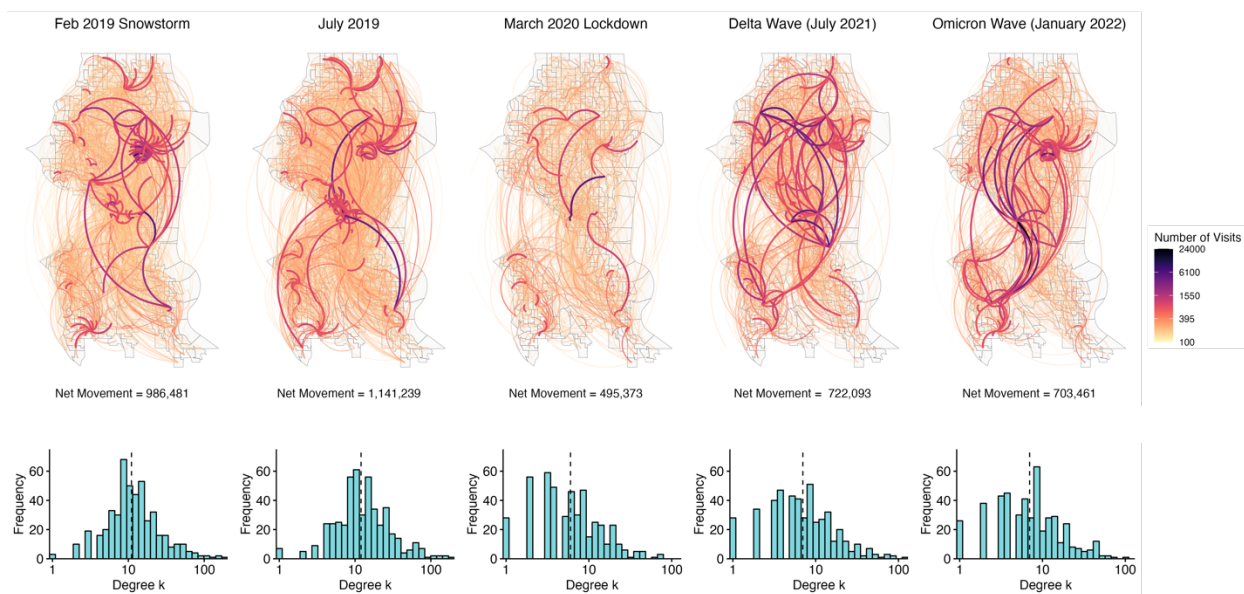


107
 108 **Figure 1. Daily incidence and transmissibility of endemic respiratory viruses and SARS-CoV-2 in the greater**
 109 **Seattle region. A.** Daily time-varying effective reproduction numbers (R_t , thick lines, left y-axis) and reconstructed
 110 incidences of endemic respiratory viruses (thin lines, right y-axis) during November 2018 – June 2022. The vertical
 111 blue shaded panel indicates the timing of a major snowstorm in Seattle (February 3-15, 2019), the vertical dashed
 112 line indicates the date of Washington’s State of Emergency declaration (February 29, 2020), and the vertical orange
 113 shaded panel indicates Seattle’s stay-at-home period (March 23 – June 5, 2020). **B.** Daily time-varying effective
 114 reproduction numbers of SARS-CoV-2 (R_t , thick green line, left y-axis), King County COVID-19 case counts (thin
 115 green line, right y-axis), and the stringency of non-pharmaceutical interventions in Washington, measured by the
 116 Oxford Stringency Index (thin orange line, left y-axis), during January 2020 – June 2022.



117

118 **Figure 2. Mobility and behavior trends in the greater Seattle region based on aggregated mobile device**
 119 **location data, November 2018 – June 2022.** In each panel, the vertical blue shaded panel indicates the timing of a
 120 major snowstorm in Seattle (February 3 – 15, 2019), the vertical dashed line indicates the date of the Washington’s
 121 State of Emergency declaration (February 29, 2020), and the vertical orange shaded panel indicates Seattle’s stay-at-
 122 home period (March 23 – June 5, 2020). **A.** The percent change from baseline for large-scale population
 123 movements: within-neighborhood movement of King County residents (purple), between-neighborhood movement
 124 of King County residents (dark green), inflow of visitors from other WA counties (red), and inflow of out-of-state
 125 visitors (light blue). **B.** The percent change from baseline in foot traffic to different categories of points of interest
 126 (POIs): transit stations (purple), religious organizations (dark green), colleges and universities (light green), full-
 127 service restaurants (dark yellow), groceries and pharmacies (pink), and elementary and high schools (blue). **C.** The
 128 percentage of devices staying completely at home (purple, left y-axis) and the percentage of individuals masking in
 129 public (dark green, right y-axis) in King County.



130

131 **Figure 3. Undirected network of mobile device movement between neighborhoods (census block groups,**
 132 **CBGs) in Seattle, Washington, during key epidemiological time points.** Time points include the week during a
 133 major snowstorm in February 2019, a week in July 2019 to show baseline movement, the beginning of stay-at-home
 134 orders in March 2020, a week during the Delta wave in July 2021, and a week during the Omicron BA.1 wave in
 135 January 2022. **Top:** Weekly visitors to points of interest (POI) are aggregated by visitor home CBG and POI CBG.
 136 Network edges (lines) are shaded according to the number of unique visitors between each pair of CBGs within a
 137 particular week, with thicker, darker edges indicating a greater number of visitors. **Bottom:** Histograms showing the
 138 frequency of degree k values for Seattle neighborhoods (i.e., the integer number of other neighborhoods each
 139 individual neighborhood is connected to) at each time point. The vertical dashed line overlaying each histogram
 140 indicates the median degree among Seattle neighborhoods.

141 **Declines in mobility correlate with reduced respiratory virus circulation during a major snowstorm in**
 142 **February 2019**

143 Most endemic viruses in our study, including influenza A viruses, RSV, hCoV, hPIV 3+4, hMPV, hRV, and AdV,
 144 circulated during the 2018-2019 winter season. This season was atypical in that Seattle experienced unusually high
 145 snowfall during February 2019, prompting widespread school and workplace closures and reduced regional travel
 146 from February 3 to February 15, 2019. The mobility categories most impacted by the snowstorm included foot
 147 traffic to elementary and high schools, colleges, and transit stations (> 75% declines below baseline), the inflow of
 148 out-of-state visitors (> 50% declines below baseline), and within- and between-neighborhood movement (29% and
 149 15% declines below baseline) (Figure 2, Figure S4). As previously described (21), this city-wide shutdown led to a
 150 conspicuous dip in incidence for several pathogens (Figure S5).

151 To measure the overall impact of the snowstorm on virus circulation, we compared pathogen specific R_t values
 152 during the two weeks before and after the start of heavy snowfall on February 3, 2019 (Table 2). RSV and AdV
 153 were the pathogens most affected by weather-related disruptions (37-40% declines), followed by influenza viruses
 154 and hCoV (10-20% declines, Table 2). Influenza A/H3N2 virus, hPIV 3 + 4, hMPV, hRV, and AdV rebounded after
 155 schools and workplaces reopened, and their epidemics subsequently peaked from mid-March to early April 2019
 156 (Figure S5).

157 During February 2019, reductions in mobility preceded or coincided with declines in pathogen transmission, though
 158 the strength of correlations varied across pathogens (Figure S6-S7). Among pathogens with the most substantial
 159 declines, drops in RSV R_t coincided most closely with reductions in within-city connectivity and foot traffic to
 160 schools, child daycare centers, and religious services (February 2019 mean cross-correlation coefficients, $r > 0.86$;

161 all reported correlations are statistically significant), while AdV Rt was moderate-to-strongly correlated with most
 162 mobility indicators, in particular visitor inflow, within-neighborhood movement, and visits to schools, child
 163 daycares, colleges, and religious services ($r > 0.94$) (Figure S6-S7). For pathogens that did not experience declines
 164 in transmission (hPIV 3 + 4, hMPV, hRV), Rt had negative or non-significant associations with mobility during the
 165 snowstorm (Figure S6-S7).

166 **Table 2. Changes in transmissibility (time-varying effective reproduction numbers, Rt) during the two weeks**
 167 **before and after two events: a major snowstorm in February 2019 and the initiation of COVID-19 social**
 168 **distancing measures in March 2020.** We compared Rt values before and after each event using t-tests for the ratio
 169 of two means. Fieller’s theorem was used to calculate the 95% confidence intervals of changes in Rt.

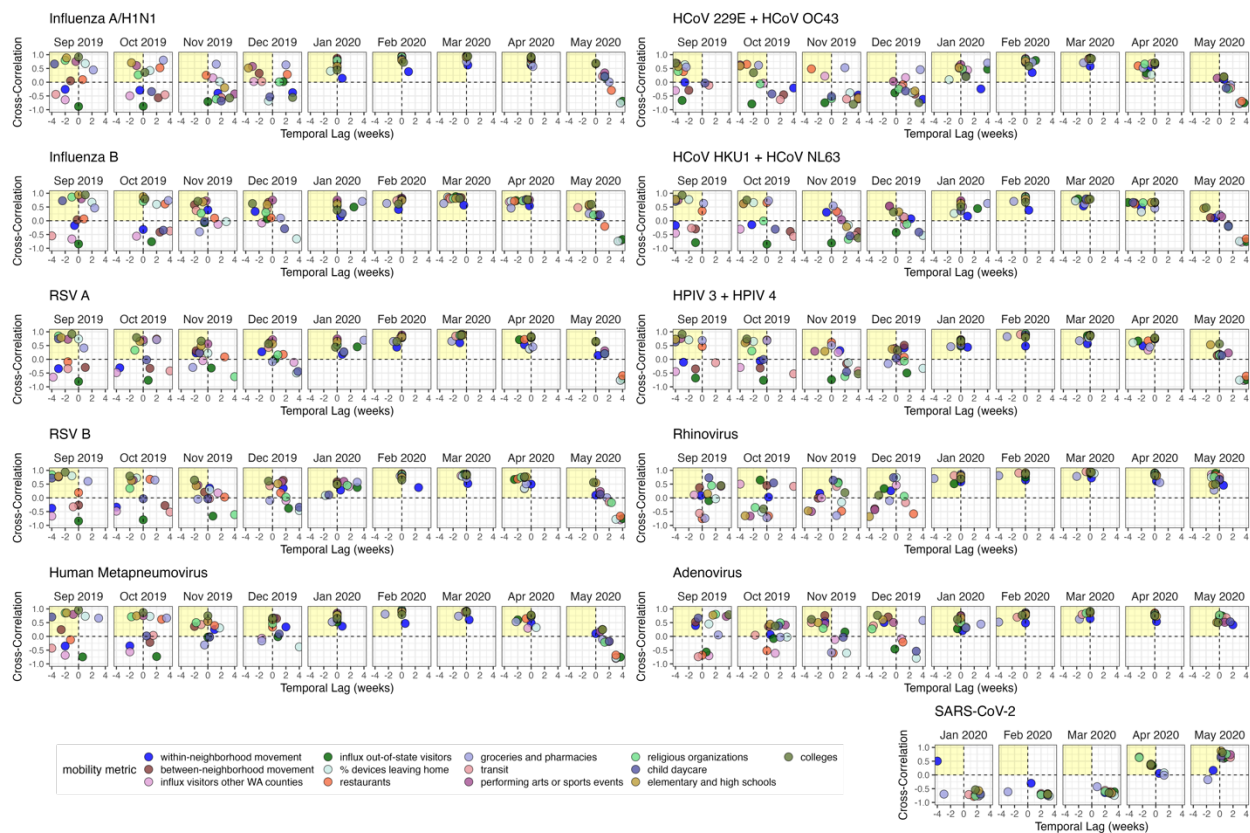
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Pathogen	Major snowstorm, February 2019		Early COVID-19 restrictions, March 2020	
	Change in Rt	P-value	Change in Rt	P-value
Influenza A/H3N2	-12 [-17, -8] %	0.00002	Not circulating	
Influenza A/H1N1	-19 [-25, -14] %	0.00001	-42 [-59, -29] %	$< 2 \times 10^{-16}$
Influenza B	Not circulating		-14 [-19, -10] %	$< 2 \times 10^{-16}$
RSV A	-40 [-50, -30] %	$< 2 \times 10^{-16}$	-9 [-14, -4] %	0.0006
RSV B	-37 [-45, -28] %	$< 2 \times 10^{-16}$	-4 [-5, -2] %	0.0003
hMPV	20 [16, 23] %	$< 2 \times 10^{-16}$	-20 [-26, -15] %	$< 2 \times 10^{-16}$
hPIV 1 + 2	Not circulating		Not circulating	
hPIV 3 + 4	14 [9, 18] %	0.00004	-22 [-28, -16] %	$< 2 \times 10^{-16}$
hCoV 229E + OC43	-10 [-15, -5] %	0.001	-20 [-25, -16] %	$< 2 \times 10^{-16}$
hCoV HKU1 + NL63	-18 [-22, -14] %	$< 2 \times 10^{-16}$	-25 [-31, -20] %	$< 2 \times 10^{-16}$
hRV	2 [-0.03, 3] %	> 0.05	-29 [-37, -22] %	$< 2 \times 10^{-16}$
AdV	-39 [-47, -31] %	$< 2 \times 10^{-16}$	-33 [-45, -22] %	$< 2 \times 10^{-16}$
SARS-CoV-2	Not circulating		-37 [-51, -25] %	$< 2 \times 10^{-16}$

171 **Relationships between mobility and pathogen transmission during the 2019-2020 winter season (pre-**
 172 **pandemic)**

173 The 2019-2020 virus respiratory season was a relatively typical season in Seattle with heightened activity of many
 174 common respiratory viruses (Figure 1). During Fall 2019, visits to child daycares, schools, colleges, and religious
 175 organizations preceded or coincided with initial increases in influenza viruses, RSV, hMPV, hCoV, and hPIV 3 + 4
 176 (moving window cross-correlation coefficients, r range: 0.53 – 0.97; all reported correlations are statistically
 177 significant; Figure 4, Figure S8). The transmission rates of RSV, hCoV, and hPIV 3 + 4 were also positively
 178 correlated with the percentage of devices leaving home (r : 0.68 – 0.81) (Figure 4, Figure S8). Increases in hPIV 1 +
 179 2 and AdV Rt coincided with visits to child daycares (both viruses, r : 0.63 – 0.88), between-neighborhood
 180 movement (AdV, r : 0.6 – 0.8), or out-of-state inflow (hPIV 1 + 2, r : 0.7 – 0.77), while hRV dynamics were not
 181 strongly tied to population mobility (Figure 4, Figure S8). For most pathogens, the strongest relationships between
 182 transmission and mobility occurred at the beginning of the season in early autumn (Figure 4, Figure S8).

183 We used multivariable generalized additive models (GAMs) to measure non-linear relationships between mobility
 184 and Rt and model selection of GAMs to assess the relative importance of different indicators in predicting Rt during
 185 the 2019-2020 season, prior to the start of the COVID-19 pandemic (September 2019 – January 2020). For each
 186 pathogen, we allowed candidate models to include a smoothed temporal trend and up to two smoothed mobility
 187 terms. Across all pathogens, minimal models included a school-related behavioral indicator (foot traffic to schools
 188 or colleges) or the percentage of devices leaving home and a covariate related to large-scale population movement
 189 (between-neighborhood movement or out-of-state inflow), with the partial effects of most mobility covariates
 190 monotonically increasing with Rt (Figure S9).



191

192 **Figure 4. Time series cross-correlations and optimal lags between respiratory virus transmissibility (time-**
 193 **varying effective reproduction numbers, R_t) and cell phone mobility in the greater Seattle region, September**
 194 **2019 – May 2020.** Points are individual mobility indicators derived from aggregated mobile device location data.
 195 Correlation coefficients are shown on the y-axis, and temporal lags (in weeks) between R_t and mobility are shown
 196 on the x-axis. Negative lags indicate behavior leads R_t , and positive lags indicate R_t leads behavior. The yellow
 197 shaded panel in each facet includes mobility indicators that have a leading, positive relationship with transmission,
 198 and hence would be considered predictive of transmission.

199 **Initial effects of COVID-19 restrictions on mobility and respiratory virus circulation**

200 The first SARS-CoV-2 infections in Washington state arose from a single introduction in late January or early
 201 February 2020, and at least one clade was circulating in the Seattle area for 3-6 weeks prior to February 28, when
 202 the first community acquired case was reported (18). To slow the spread of SARS-CoV-2, Washington declared a
 203 State of Emergency on February 29, closed schools in King, Pierce, and Snohomish counties on March 12, and
 204 enacted statewide stay-at-home (SAH) orders on March 23. In the interim, King County recommended that
 205 workplaces allow employees to work from home on March 4 and closed indoor dining and many other businesses on
 206 March 16.

207 Mobility levels declined substantially after February 29, and, by the start of King County’s business closures on
 208 March 16, foot traffic to transit stations were > 90% below baseline and out-of-state inflow and within-city mixing
 209 were > 60% below baseline (Figure 2, Figure S4). After the enactment of SAH orders on March 23, foot traffic to
 210 POIs and large-scale movements declined to 70-90% below baseline (Figure 2, Figure S4), while the percentage of
 211 devices staying completely at home increased to > 50% (Figure 2). Notably, social distancing measures altered not
 212 only the volume of movement between Seattle neighborhoods but also the presence and absence of connections
 213 between neighborhoods. Specifically, neighborhoods with low degree centrality (i.e., fewer than 10 connections to
 214 other neighborhoods) became much more prevalent compared to weeks prior to March 2020 (Figure 3).

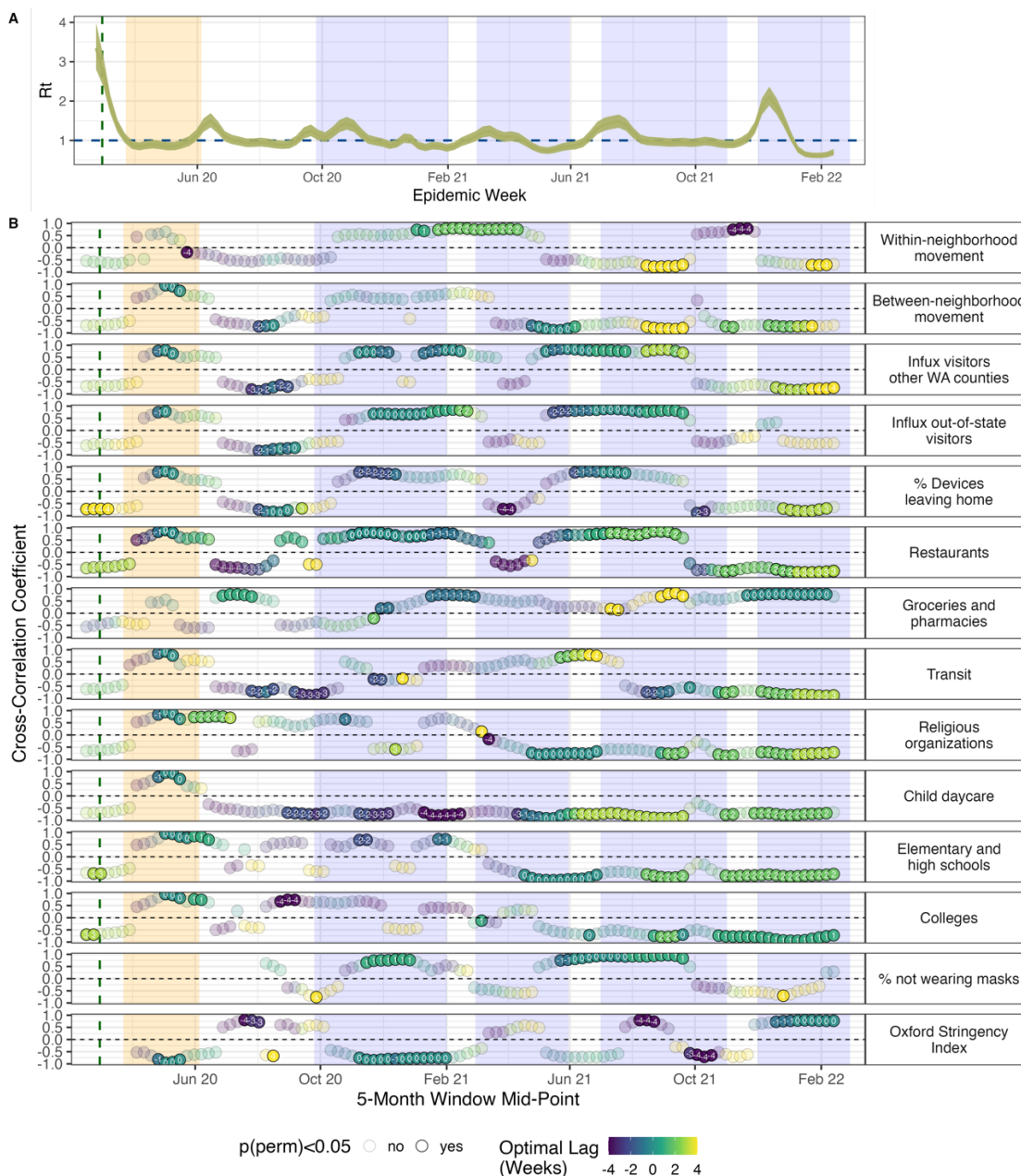
215 The transmission rates of all respiratory pathogens dropped substantively after the State of Emergency, though some
216 seasonal pathogens were already declining prior to February 29 (Figure 1, Figure S3). We measured the initial
217 impacts of COVID-19 NPIs on respiratory virus circulation by comparing R_t values during the 2 weeks before and
218 after the State of Emergency declaration on February 29 (Table 2). Early public health measures were effective at
219 lowering SARS-CoV-2 transmission rates by 37% [95% CI: 25, 51]. Among endemic pathogens, influenza A/H1N1
220 virus, AdV, and hRV were the most impacted by pandemic-related behavioral changes, experiencing 29 – 42%
221 reductions in transmissibility by March 16. hPIV 3 + 4, hMPV, and hCoV were also significantly affected,
222 experiencing 20 – 25% declines in R_t . Reductions in RSV and influenza B virus were more modest, given their
223 seasonal outbreaks had mostly concluded by late February. The hPIV 1 + 2 outbreak subsided in mid-February, and
224 thus was not impacted by COVID-19 NPIs.

225 We observed strong relationships between mobility and the transmission of respiratory pathogens in the Spring of
226 2020. All mobility metrics were positive, leading indicators of R_t across all endemic viruses (Figure 4, Figure S10).
227 In contrast, mobility lagged and was negatively correlated with SARS-CoV-2 transmission during this period
228 (Figure 4, Figure S10). COVID-19 restrictions began to relax on May 4, 2020, when King County entered Phase 1
229 of the state’s reopening plan, allowing outdoor dining, worship services, and fitness centers to reopen with limited
230 capacity. SARS-CoV-2 R_t values ranged from 0.8 to 0.9 throughout April and May and did not surpass 1 until late
231 May (Figure 1). During late April and May, SARS-CoV-2 R_t became positively correlated and synchronous with
232 most mobility indicators (r : 0.66 – 0.97) and inversely correlated with the stringency of NPIs (r : -0.97 – -0.81)
233 (Figure 4, Figure S10). Yet, these relationships did not persist after the virus’s initial rebound in early June 2020,
234 when King County reopened indoor dining and additional businesses (Figure S10). Due to the high degree of
235 collinearity and concavity among mobility metrics, we could not differentiate the effects of individual indicators on
236 R_t during Seattle’s SAH period.

237 Population mobility did not immediately recover after SAH orders lifted in June 2020 (Figures 2-3, Figure S4).
238 Visitor inflow from other WA counties and US states remained depressed at levels 50% below baseline until the
239 spring and summer months of 2021, and within-and-between-neighborhood movement had not returned to pre-
240 pandemic levels by the conclusion of our study in June 2022 (Figures 2-3, Figure S4). Further, SAH orders caused
241 long-lasting structural changes to the mobility network of Seattle, wherein neighborhoods with high degree
242 centrality (“hubs”) were most affected (Figure 3). After March 2020, neighborhoods with fewer than 10 connections
243 to other neighborhoods became much more prevalent in the network, causing an overall downshift in the mobility
244 network’s median degree for the remainder of the study period (Figure 3).

245 **Associations between SARS-CoV-2 transmission and behavior differ across COVID-19 waves**

246 We measured cross-correlations between SARS-CoV-2 transmission, mobility, masking, and NPI stringency during
247 subsequent pandemic waves in Seattle (Figure 5). After its first two COVID-19 outbreaks in Spring 2020 and
248 Summer 2020, Seattle experienced a large third wave during winter 2020-2021 despite high masking rates (Figure
249 5). During November-December 2020, daily R_t was strongly correlated with foot traffic to restaurants (r : 0.65 –
250 0.83), the percentage of devices leaving home (r : 0.7 – 0.83), the influx of visitors from other WA counties and US
251 states (r : 0.66 – 0.74), and NPI stringency (r : -0.85 – -0.76) (Figure 5). A smaller wave associated with the Alpha
252 variant spanned March to May 2021, during which R_t was not significantly associated with mobility (Figure 5).
253 COVID-19 cases and hospitalizations surged again in July 2021, due to the spread of the highly transmissible Delta
254 variant. From late June to early August 2021, the percentage of residents not masking in public, the percentage of
255 devices leaving home, and visitor inflow preceded or coincided with increases in R_t (r : 0.75 – 0.94) (Figure 5).
256 Transmission decoupled from behavior during the Omicron BA.1 wave in late 2021, wherein SARS-CoV-2 R_t was
257 negatively correlated and lagging most mobility metrics and positively correlated with masking and NPI stringency
258 (the inverse of expected relationships) (Figure 5). Minimal GAMs fit to the first three months of each wave retained
259 the percentage of devices leaving home, the percentage of residents not masking, foot traffic to restaurants, or out-
260 of-state inflow (Figure S11), but associations between behavior and R_t were often negative.



261

262 **Figure 5. Relationships between mobility, masking, and SARS-CoV-2 transmission in the greater Seattle**
 263 **region during the COVID-19 pandemic, January 2020 – March 2022. A.** Weekly effective reproduction number
 264 (Rt) of SARS-CoV-2, and **B.** Rolling cross-correlations between weekly Rt and mobility and behavioral indicators.
 265 Points represent the maximum coefficient values for 5-month rolling cross-correlations between weekly Rt and
 266 individual mobility metrics. Point color and the number within each point indicate the lag in weeks corresponding to
 267 the maximum cross-correlation coefficient value for each 5-month period (“optimal lag”). Negative values indicate
 268 that behavior leads Rt, and positive values indicate that Rt leads behavior. A lag of 0 indicates that the time series
 269 are in phase (i.e., synchronous). Point transparency indicates statistical significance of the cross-correlation
 270 coefficient (yes: solid, no: transparent). The vertical green dashed line indicates the date of Washington’s State of
 271 Emergency declaration (February 29, 2020), and the vertical orange shaded panel indicates Seattle’s stay-at-home
 272 period (March 23 – June 5, 2020). The blue shaded panels indicate the timing of four COVID-19 waves in Seattle:
 273 the winter 2020-2021 wave, the Alpha wave in Spring 2021, the Delta wave in Summer 2021, and the Omicron
 274 BA.1 wave during late 2021 to early 2022.

275 **Mobility predictors of endemic pathogen rebound during the COVID-19 pandemic**

276 We observed a remarkably fast rebound of hRV, and AdV to a lesser degree, when SAH restrictions relaxed in early
277 June 2020 (Figure 1, Figures S2-S3). Increases in hRV transmission were preceded by or synchronous with the
278 percentage of devices leaving home, foot traffic to restaurants, between-neighborhood movement, and visitor inflow
279 from June to early August 2020 ($r: 0.75 - 0.96$), inversely correlated with NPI stringency during September 2020 ($r:$
280 $-0.79 - -0.69$), and continuously correlated with foot traffic to religious organizations until late 2020 ($r: 0.64 - 0.91$)
281 (Figure S12). The rebound of AdV was preceded by or synchronous with slight increases in foot traffic to schools
282 and religious organizations from June to early August 2020 ($r: 0.7 - 0.87$) (Figure S12). For both viruses, minimal
283 GAMs fit to the first six months of rebound retained the percentage of devices leaving home and out-of-state inflow
284 (Figure S13).

285 Resurgence of other endemic viruses, including hCoV, hPIV, hMPV, and RSV B, was not observed until early-to-
286 mid 2021, and epidemic peaks for hCoV, hMPV, and RSV B occurred during the spring or summer outside of their
287 typical seasons (Figure 1, Figures S2-S3). We measured univariate associations between mobility, masking, NPI
288 stringency, and daily transmissibility and found fewer clear relationships compared to Seattle's SAH period and the
289 2019-2020 winter season. Multiple mobility indicators preceded or coincided with the rebound of these viruses,
290 though, for RSV B and hCoV, associations were briefer compared to those observed during seasonal outbreaks prior
291 to the pandemic (Figure S14). For example, at the beginning of RSV B's rebound in summer 2021, increases in R_t
292 were preceded by between-neighborhood movement and foot traffic to schools, child daycares, and religious
293 organizations ($r: 0.7 - 0.9$) and synchronous with the percentage of devices leaving home, the percentage of
294 residents not masking in public, and out-of-state inflow ($r: 0.7 - 0.91$) (Figure S14). These associations were more
295 transient compared to the pre-pandemic period, persisting for 1-2 months instead of 2-3 months (Figures S8 and
296 S14). Minimal GAMs fit to the first three months of each virus's rebound retained between-neighborhood
297 movement and out-of-state inflow, though relationships between mobility and R_t were nonlinear and not
298 consistently positive (Figure S15).

299 In late 2021, endemic virus circulation declined as Omicron cases surged, and reductions in endemic virus
300 transmission were universally preceded by or coincided with drops in mobility (Figure S12, Figure S14, Figure
301 S16). For example, reductions in most mobility indicators preceded declines in RSV B, hMPV, hCoV, and hPIV 3 +
302 4 circulation by 1 to 4 weeks, while reduced visits to child daycares, schools, colleges, and transit stations were
303 synchronous with declines in hRV transmission (Figures S12, Figure S14). The transmission rates of RSV B,
304 hMPV, and hCoV were also associated with the percentage of devices staying home, which spiked from 35% to
305 50% in December 2021 (Figure 2, Figure S16). During the Omicron wave (November 2021 – January 2022), the
306 best fit minimal GAMs for each virus retained a school-related behavioral indicator, the percentage of devices
307 leaving home, or out-of-state inflow, similar to results observed for the 2019-2020 season (Figure S17).

308 Although our study's aim was inferential rather than predictive, we built forecasting models of daily R_t for three
309 viruses that circulated continuously throughout the study period: hRV, AdV, and SARS-CoV-2. We used mobility
310 metrics, the co-circulation of other viruses, and activity of the target virus during the previous two weeks (14
311 autoregressive terms) as candidate predictors. For each virus, models with only mobility terms did not outperform
312 models with only autoregressive terms (Figures S18-S20) but still produced accurate forecasts over the entire study
313 period (Pearson's r with observed data, $r > 0.8$), and especially during Seattle's stay-at-home orders and the initial
314 lifting of restrictions ($r: 0.94 - 0.95$) (Table S3). Methodological details and results are provided in the
315 supplementary material.

316 **Discussion**

317 We investigated the impacts of human behavior on the transmission of respiratory viruses in the greater Seattle
318 region, during pre- and post-pandemic years, by modeling incidence derived from hospital and community-based
319 respiratory surveillance and human movements from high-resolution mobile device location data. From November
320 2018 to June 2022, we characterized the epidemiological dynamics of 16 endemic viruses and SARS-CoV-2 and
321 related changes in daily transmissibility (time-varying effective reproduction numbers, R_t) to trends in population
322 mobility, masking, and COVID-19 non-pharmaceutical interventions (NPIs). To our knowledge, this is the first
323 study to explore the effects of mobility and behavior on transmission across a large set of endemic pathogens;

324 interestingly, we saw notable heterogeneity in the timing and size of each endemic pathogen’s rebound during the
325 pandemic period.

326 Mobility was most predictive of transmission during periods of dramatic behavioral change, as observed during
327 Seattle’s stay-at-home (SAH) orders in March 2020. Smaller-scale changes in mobility also preceded or coincided
328 with increases in R_t at the beginning of outbreaks and declines in R_t during shorter interruptions to human
329 movement, as observed during a major snowstorm in February 2019 and the Omicron BA.1 wave in late 2021.
330 Across all endemic viruses, trends in daily R_t were repeatedly associated with the same set of mobility metrics,
331 including foot traffic to elementary and high schools, colleges, child daycare centers, restaurants, and religious
332 organizations, the percentage of devices leaving home, between-neighborhood movement, and the inflow of visitors
333 from other WA counties and US states. Outside the SAH period, SARS-CoV-2 transmission correlated with foot
334 traffic to restaurants and colleges, the percentage of devices leaving home, and visitor inflow. Foot traffic to specific
335 businesses and educational and religious activities may approximate close contacts or crowded conditions that
336 facilitate direct, aerosol, or droplet transmission, while the percentage of devices leaving home, within-city mixing,
337 and inflow from other regions may be indicative of human movements that promote viral introductions and
338 dispersal.

339 The age distribution of infections may explain slight differences in which categories of POIs correlated with
340 endemic virus transmission versus SARS-CoV-2 transmission. Recurrent associations between endemic virus R_t and
341 visits to schools and daycares are consistent with children experiencing the highest rates of (endemic) respiratory
342 infections and schools and daycares acting as a major source of transmission in the community (29-33). Unlike
343 endemic respiratory viruses, all age groups are susceptible to SARS-CoV-2 infection. Correlations between SARS-
344 CoV-2 R_t and foot traffic to colleges and restaurants, but not schools or daycares, could be attributed to greater rates
345 of symptomatic infection (and hence shedding propensity) in adults relative to younger age groups (34) or to the
346 greater relevance of adult networks in spreading SARS-CoV-2 compared to endemic viruses.

347 COVID-19 NPIs significantly perturbed the transmission of respiratory pathogens at a global level (1-8), causing the
348 complete disappearance, delayed return, or “off-season” outbreaks of endemic pathogens (6-9). In Seattle, all
349 endemic respiratory viruses experienced rapid declines at the beginning of Seattle’s SAH orders in March 2020, but,
350 as restrictions eased, their rebound was heterogeneous. Similar to trends observed in the US and other countries (4,
351 6, 7, 10, 35-39), the circulation of hRV and AdV resumed in early June 2020, immediately after nonessential
352 businesses and indoor dining reopened, while other respiratory viruses virtually disappeared during March 2020 and
353 did not recirculate until 2021. Further, the resurgence of RSV B, hCoV, and hMPV occurred outside of their typical
354 seasons, as reported in other locations (7-9). After the initial easing of COVID-19 restrictions, relationships between
355 endemic virus dynamics and mobility were less clear compared to Seattle’s SAH orders or the 2019-2020 winter
356 season, potentially due to continued social distancing and masking, a more refined understanding of “high risk”
357 activities, the delay of in-person instruction for school students until spring 2021, or structural changes to Seattle’s
358 mobility network. Nonetheless, associations between endemic virus R_t and population behavior were overall
359 stronger and longer-lasting than those observed for SARS-CoV-2.

360 It is remarkable that the two viruses that rebounded immediately after lockdown restrictions lifted, hRV and AdV,
361 are non-enveloped viruses, while the other viruses studied here are enveloped. The immediate rebound of non-
362 enveloped viruses could be attributed to viral stability and persistence. Non-enveloped viruses are less susceptible to
363 lipophilic disinfection and can persist on hands and fomites for longer periods of time than enveloped viruses (40,
364 41). In addition of the presence or absence of an envelope, several other factors, such as transmission mode,
365 seasonality, source/sink dynamics, and duration of infectious period and immunity, could have affected the timing of
366 rebound. While enveloped viruses disappeared in March 2020, non-enveloped viruses may have continued to spread
367 during SAH restrictions, due to their longer periods of viral shedding, high preexisting community prevalence, or
368 ability to persist on environmental surfaces (36, 40-42). We hypothesize that low levels of transmission or residual
369 viral particles on surfaces, combined with slight increases in movement, close contacts, and visitor inflow, were
370 sufficient to facilitate the rapid rebound and ongoing transmission of hRV and AdV after SAH orders lifted in June
371 2020. Further, surgical masks are less effective at filtering hRV compared to influenza viruses and seasonal
372 coronaviruses (43).

373 Our study period encompasses the two respiratory virus seasons prior to the start of the COVID-19 pandemic and
374 two pandemic years. The Seattle Flu Study (SFS) began collecting samples in November 2018, which precluded us

375 from evaluating potential leading indicators of transmission at the beginning of the 2018-2019 season. However, we
376 were able to detect strong links between mobility and transmission in February 2019 when a major snowstorm
377 forced work and school closures, consistent with previous SFS research that did not specifically examine cell phone
378 mobility patterns (21). SFS continued to collect respiratory samples throughout 2019, enabling us to test for leading
379 indicators of transmission during the 2019-2020 winter season. During Fall 2019, the transmission dynamics of
380 enveloped viruses were more strongly correlated with mobility than those of non-enveloped viruses. For enveloped
381 viruses, foot traffic to schools and colleges, between-neighborhood movement, and visitor inflow preceded or
382 coincided with increases in transmission, with associations between R_t and mobility weakening over the course of
383 the season, presumably due to accumulating immunity in the population. During this same period, non-enveloped
384 viruses had fewer positive relationships with mobility, potentially because hRV and AdV circulate year-round and
385 have less defined peaks and troughs.

386 SARS-CoV-2 began circulating in the greater Seattle region during January or February 2020 (18), with the first
387 community-acquired case confirmed on February 28, 2020. Mobility had a negative, lagging relationship with
388 SARS-CoV-2 R_t during the early months of 2020, suggesting that Seattle residents adjusted their behavior in
389 response to COVID-19 case counts or restrictions rather than the reverse. Mobility was briefly predictive of SARS-
390 CoV-2 transmission when social distancing restrictions first relaxed in summer 2020 and during the winter 2020-
391 2021 and Delta waves, before ultimately decoupling from R_t during the Omicron BA.1 wave in late 2021.
392 Compared to prior variants of concern (VOCs), Omicron BA.1 has a shorter serial interval, increased immune
393 evasion, and greater intrinsic transmissibility (44-46). A phylogeographic study linked inter-city travel to the spatial
394 spread of individual Omicron BA.1 lineages in the UK (47), suggesting that daily R_t estimated from all COVID-19
395 cases combined may lack the resolution to relate the rapid spread of Omicron BA.1 to mobility patterns (*see*
396 *methods for comments on VOC-specific R_t analyses*).

397 Climate affects the stability and seasonal dynamics of respiratory viruses (48-51) but was an unlikely driver of
398 endemic virus resurgence in Seattle. hRV and AdV have year-round circulation with peaks in the spring and early
399 autumn (52, 53), while influenza viruses, RSV, hCoV, hMPV, and hPIV have distinct seasonality with peaks during
400 the winter or spring (53-56). The lifting of SAH orders in June 2020 coincided with the typical timing of low
401 circulation for enveloped viruses and increasing activity for non-enveloped viruses. However, the intersection of
402 relaxing NPIs with warmer weather cannot account for the global differences observed between non-enveloped and
403 enveloped virus rebound. The prolonged absence of influenza and RSV circulation was also reported during the
404 Southern Hemisphere winter (2, 5, 9), and climatic factors cannot explain the rebound of hCoV, hMPV, and RSV B
405 outside of their typical seasons.

406 Our findings suggest that in-person school instruction played a key role in the rebound of enveloped viruses in
407 Seattle. Prior research has shown that increased contact rates among older children during school terms influence the
408 timing of seasonal influenza and “common cold” virus outbreaks (29, 57, 58), and younger children and adults
409 acquire influenza and RSV infections from preschool or school-aged children living in the same household (59-61).
410 All King County public school districts began the 2020-2021 academic year remotely (62), with some districts
411 offering limited in-person instruction to special education students during Fall 2020. Foot traffic to schools was 75%
412 below baseline in Fall 2020, gradually increased to 50% below baseline during Spring 2021, and returned to baseline
413 levels in Fall 2021. We observed that the circulation of hCoV, hPIV, and hMPV increased after elementary school
414 students were offered in-person instruction in February and March 2021 and that the off-season RSV B wave in
415 summer 2021 began directly after hybrid learning became available to all grades in mid-April (62). These trends
416 suggest that a year of remote learning, and in turn reduced contacts among school-aged children, contributed to the
417 delayed return of enveloped virus circulation to Seattle.

418 The rebound of enveloped viruses also coincided with increasing rates of travel into Seattle. Annual influenza
419 epidemics in North America are seeded via air travel by strains originating in East and Southeast Asia (63, 64), and
420 the regional spread of influenza viruses correlates closely with work commutes (65, 66). We did not have data on
421 international air travel or commuting patterns, but cell phone data show that the inflow of visitors from other WA
422 counties and US states was 50% below baseline throughout 2020 and did not return to pre-pandemic levels until late
423 spring or summer 2021. Although the contribution of local persistence versus external seeding is less understood for
424 other seasonal respiratory viruses, increasing inflow into Seattle likely imported cases from other regions, seeding
425 new outbreaks (9).

426 Lastly, prolonged lack of exposure due to reduced viral circulation during 2020 and 2021 is expected to have
427 increased the cohort of children completely naïve to various respiratory viruses and the waning of immunity in
428 previously infected individuals (14, 67). This “immunity debt” may have provided enough susceptible individuals to
429 sustain spring and summer outbreaks of enveloped viruses. Although we expected these outbreaks to be larger or
430 more severe than those observed during pre-pandemic seasons, substantial influenza and RSV epidemics did not
431 occur until the Fall of 2022, potentially due to Seattle residents continuing to social distance and mask throughout
432 2021 or negative interference between Omicron BA.1 viruses and endemic viruses during the 2021-2022 winter
433 season. After the conclusion of our study, the 2022-2023 season saw atypically early outbreaks of influenza and
434 RSV and higher hospitalization rates in children and adolescents compared to pre-pandemic seasons (15, 68).

435 Our study has limitations related to the type of cell phone mobility data used and the underlying demographics of
436 mobile device data in general. Young children experience the highest rates of endemic respiratory infections, but
437 SafeGraph does not track individuals younger than 16 years of age. Nonetheless, we found that visits to schools and
438 daycares were positive, leading indicators of transmission, both prior to and during the pandemic, demonstrating that
439 mobile phone data derived from adults can approximate the movements or contacts of children. Second, although we
440 found statistically significant associations between foot traffic indicators and pathogen transmission, spatial co-
441 location data may better approximate the interpersonal contacts that underlie transmission and reduce statistical
442 noise. Longitudinal cross-sectional surveys on social interactions, such as the CoMix survey in England, can provide
443 more direct measures of epidemiologically relevant behavior and more representative samples of populations than
444 mobile device data (69). However, to our knowledge, similar data do not exist for the US.

445 Our findings are subject to several other limitations. First, due to the limited number of seasons in our study, we
446 could not determine if leading indicators of transmission are consistent across timespans longer than four years.
447 Although SFS continued to conduct respiratory surveillance into the 2022-2023 winter season, its community
448 surveillance approach changed substantially after July 2022, making it difficult to extend our study. Second,
449 variability in test volume over time caused SFS surveillance to sometimes miss less prevalent pathogens. For
450 example, SFS detected only a few cases during a small influenza A/H3N2 wave in winter 2021-2022. Third, our
451 multiplex PCR assay could not distinguish between types, strains, or serotypes of some pathogens (hCoV, hPIV,
452 AdV, hRV). Consequently, our Rt estimates may average over heterogeneities in transmission dynamics among
453 viruses belonging to the same species (19). Fourth, previous work has shown that SARS-CoV-2 transmission
454 dynamics differed between North and South King County (20, 70), potentially due to socioeconomic inequities (e.g.,
455 differences in income, household size, proportion of essential workers) and North King County maintaining a
456 greater reduction in mobility over time. However, we did not have sufficient surveillance data to explore geographic
457 differences in the transmission dynamics of endemic viruses. Fifth, population immunity may modulate relationships
458 between mobility and transmission; analysis of serologic data could shed light on this question. Additional research
459 is needed to delineate the contributions of an increasingly susceptible population and decreased social distancing to
460 the rebound of endemic viruses.

461 In summary, mobility patterns are most predictive of respiratory virus transmission during drastic changes in
462 contacts, and, to a lesser extent, at the beginning of epidemic waves. During the pandemic period, endemic
463 respiratory viruses exhibited stronger relationships with mobility than pandemic SARS-CoV-2. As SARS-CoV-2
464 transitions to endemicity, relationships with mobility could gradually start to operate similarly to those of other
465 enveloped viruses. Our study shows that mobile phone data can approximate transmission relevant contacts and has
466 the potential to support the surveillance of endemic respiratory viruses, with the caveat that relationships between
467 transmission and mobility vary depending on the pathogen, magnitude of mobility change, and phase of the
468 epidemic. Future research should consider other host factors, such as prior immunity, and more direct proxies of
469 interpersonal contacts to further disentangle relationships between population behavior and respiratory virus
470 dynamics.

471 **Methods**

472 **Virologic surveillance and laboratory methods**

473 This population-level study uses cross-sectional surveillance data collected through the Seattle Flu Study (SFS) from
474 November 2018 to June 2022. Initiated in November 2018, SFS was a multi-arm surveillance study of influenza and
475 other respiratory pathogens in the greater Seattle, WA region, that utilized community and hospital-based sampling

476 (17). In its first 1.5 years, SFS tracked the transmission of influenza and other respiratory pathogens in the Seattle
477 region by testing swabs collected at hospitals, community sites (e.g., kiosks in high foot traffic areas, outpatient
478 clinics, workplaces, college campuses), and through its swab-and-send at-home testing study (17, 22) (Table S2).
479 The SFS team launched the greater Seattle Coronavirus Assessment Network (SCAN) in March 2020 to detect and
480 understand the spread of SARS-CoV-2 (23). SCAN deployed self-administered at-home testing kits to monitor the
481 spread of both SARS-CoV-2 and endemic respiratory pathogens from March 2020 to July 2022. We describe each
482 surveillance arm in the supplementary methods.

483 The Institutional Review Board of the University of Washington gave ethical approval of this work (protocol
484 #00006181). All participants who contributed specimens to the Seattle Flu Study or Greater Seattle Coronavirus
485 Assessment Network provided informed consent at enrollment. Informed consent for residual sample and clinical
486 data collection was waived, as these samples were already collected as part of routine clinical care, and it was not
487 possible to re-contact these individuals. This study followed the Strengthening the Reporting of Observational
488 Studies in Epidemiology (STROBE) reporting guidelines for cross-sectional studies.

489 Each respiratory specimen was screened in duplicate for a panel of respiratory pathogens using a custom TaqMan
490 RT-PCR OpenArray panel (Thermo Fisher). Laboratory methods are described in detail elsewhere (20, 22).
491 Pathogen targets included adenovirus (AdV); human bocavirus (hBoV); human coronaviruses (hCoV) 229E, OC43,
492 HKU1, and NL63; human metapneumovirus (hMPV); human parainfluenza viruses (hPIV) 1, 2, 3, and 4; human
493 parainfluenza virus (hPeV); influenza A (IAV) H1N1 and H3N2; pan influenza A (IAV); pan influenza B (IBV); pan
494 influenza C (ICV); respiratory syncytial viruses (RSV) A and B; human rhinovirus (hRV); enterovirus D68
495 (EV.D68); pan enterovirus excluding D68 (EV); *Streptococcus pneumoniae* (Spn); *Mycoplasma pneumoniae* (Mpn);
496 *Chlamydia pneumoniae* (Cpn); and SARS-CoV-2. Due to assay limitations, epidemiologically distinct strains were
497 grouped into one assay each for hCoV 229E and hCoV OC43, hCoV HKU1 and hCoV NL63, hPIV 1 and hPIV 2,
498 hPIV 3 and hPIV 4, EV, hRV, and AdV.

499 A substantial number of specimens tested positive for SPn, a common commensal, with SPn detected in 27.3% of
500 positive samples prior to March 2020 and 18.7% of positive samples after March 2020 (Figure S2). We opted to not
501 include SPn in downstream analyses due to our inability to distinguish acute infections from chronic carriage.

502 **Syndromic surveillance data**

503 We obtained respiratory syndromic surveillance data for King County, WA from the Rapid Health Information
504 Network (RHINO) program at the Washington Department of Health (WA DOH) (Figure S21). Syndrome criteria
505 are defined by the Electronic Surveillance System for the Early Notification of Community-Based Epidemics
506 (ESSENCE). We received weekly counts of total emergency department (ED) visits and ED visits classified as
507 *influenza-like illness* (ILI) (mention OR diagnosis of influenza OR fever ($\geq 100^\circ\text{F}$) and cough OR fever ($\geq 100^\circ\text{F}$) and
508 sore throat), *COVID-like illness* (CLI) (mention OR diagnosis of coronavirus AND no diagnosis of influenza OR
509 fever OR chills AND cough OR shortness of breath OR difficulty breathing), and *broad respiratory illness* (acute
510 bronchitis OR chest congestion OR cough OR difficulty breathing OR hemoptysis OR laryngitis OR lower
511 respiratory infection OR nasal congestion OR otitis media OR pneumonia OR shortness of breath OR sore throat OR
512 upper respiratory infection OR wheezing OR acute respiratory distress). Weekly data were disaggregated by age
513 group (0-4, 5-24, 25-64, and ≥ 65). We collapsed age groups into two categories, < 5 and ≥ 5 years of age, and
514 calculated the weekly proportion of ED visits coded as ILI, CLI, or broad respiratory illness (Figure S21).

515 Respiratory syndromic surveillance data for Washington state were obtained from the U.S. Outpatient Influenza-like
516 Illness Surveillance Network (ILINet) via the CDC FluView Interactive dashboard (71). ILINet consists of
517 approximately 3,200 sentinel outpatient healthcare providers throughout the United States that report the total
518 number of consultations for any reason and the number of consultations for ILI every week. ILI is defined as fever
519 (temperature of 100°F [37.8°C] or greater) and a cough and/or a sore throat. ILINet provides the weekly proportion
520 of outpatient consultations for ILI and the number of ILI encounters by age group (0-4, 5-24, 25-64, and ≥ 65).

521

522 **Data on cell phone mobility, masking, and the stringency of non-pharmaceutical interventions**

523 We obtained mobile device location data from SafeGraph (<https://safegraph.com/>), a data company that aggregates
524 anonymized location data from 40 million devices, or approximately 10% of the United States population, to
525 measure foot traffic to over 6 million physical places (points of interest) in the US. We estimated foot traffic to
526 specific points of interests (POIs), movement within and between census block groups, and the in-flow of visitors
527 residing outside of King County from June 2018 to June 2022, using SafeGraph’s “Weekly Patterns” dataset, which
528 provides weekly counts of the total number of unique devices visiting a POI from a particular home location. POIs
529 are fixed locations, such as businesses or attractions. A “visit” indicates that a device entered the building or spatial
530 perimeter designated as a POI. A “home location” of a device is defined as its common nighttime (18:00-7:00)
531 census block group (CBG) for the past 6 consecutive weeks. We restricted our datasets to King County POIs that
532 had been recorded in SafeGraph’s dataset since January 2019.

533 To measure movement within and between CBGs (“neighborhoods”) in King County, we extracted the home CBG
534 of devices visiting points of interest (POIs) and limited the dataset to devices with home locations in the CBG of a
535 given POI (within-neighborhood movement) or with home locations in CBGs outside of a given POI’s CBG
536 (between-neighborhood movement). To measure the inflow of visitors from other counties in Washington state or
537 from out-of-state, we limited the dataset to devices visiting POIs in King County with home locations in other WA
538 counties or in other US states, respectively. For each POI in each week, we excluded home census block groups with
539 fewer than five visitors to that POI. To measure foot traffic to specific categories of POIs, we aggregated daily visits
540 to POIs by North American Industry Classification System (NAICS) category, without considering the home
541 locations of devices visiting these POIs. To adjust for variation in SafeGraph’s device panel size over time, we
542 divided Washington’s census population size by the number of devices in SafeGraph’s panel with home locations in
543 Washington state each month and multiplied the number of daily or weekly visitors by that value. For each mobility
544 indicator, we summed adjusted daily or weekly visits across POIs and measured the percent change in movement
545 over time relative to the average movement observed in all of 2019, excluding national holidays.

546 Daily data on the percentage of devices staying home in King County were obtained from SafeGraph’s Social
547 Distancing Metrics (<https://docs.safegraph.com/docs/social-distancing-metrics>) and Meta Data for Good’s
548 Movement Range Maps (<https://dataforgood.facebook.com/dfg/tools/movement-range-maps>) (72). SafeGraph social
549 distancing metrics were available from January 1, 2019, to April 16, 2021, and Meta Movement Range Maps were
550 available from March 1, 2020, to May 22, 2022. Trends in the percentage of devices staying home were almost
551 identical across the two data sources, though the percentage of devices staying home in the Meta dataset was lower
552 than that observed in the SafeGraph dataset. We added a scaling factor to the Meta indicator and joined the two time
553 series to create a single metric for our study period.

554 We obtained survey data on the daily percentage of King County residents wearing face masks in public from the
555 Carnegie Mellon University Delphi Group Covidcast API (28). Masking data were collected as part of the COVID-
556 19 Trends and Impact Survey conducted by the Delphi group, in collaboration with Meta and a consortium of
557 universities and public health officials (73). The survey ran continuously from April 6, 2020, to June 25, 2022, with
558 approximately 40,000 individuals in the United States participating every day. The survey included specific
559 questions about masking from September 8, 2020, to June 25, 2022. We supplemented the Covidcast King County
560 masking data with COVIDNearYou survey data for Washington state (74) to extend the time series to June 2, 2020.

561 We extracted data collected by the Oxford COVID-19 Government Response Tracker (OxCGRT)(27) to measure
562 variation in Washington state’s government policies related to COVID-19 from March 1, 2020 to June 30, 2022.
563 The OxCGRT database tracked publicly available information for policies related to closure and containment,
564 health, and economic policy in 180 countries, recording policy responses on ordinal or continuous scales for 19
565 policy areas. We obtained daily values for the stringency index, which combines all containment and closure (C)
566 indicators and the H1 indicator (public information campaigns).

567 **Reconstructing pathogen incidences**

568 While SFS sampling is robust enough to provide granular (daily) surveillance data on the circulation of multiple
569 pathogens, the diversity of SFS sampling schemes requires pre-processing to infer pathogen incidence. To properly

570 reconstruct pathogen incidences through time, we considered the different populations sampled by SFS, particularly
571 regarding age group, clinical setting, and the presence of respiratory symptoms.

572 We first excluded samples with missing age or home address information (as reported by individuals participating in
573 community surveillance or obtained through electronic hospital records), samples from individuals residing outside
574 the greater Seattle region (King, Pierce, Snohomish, Kitsap, San Juan, Whatcom, Skagit, Island, Clallam, Jefferson,
575 Mason, and Thurston counties), samples from individuals who were asymptomatic for respiratory illness, and
576 samples from multiple testing of individuals. If an individual was tested more than once in a 30-day period, we kept
577 one result per pathogen in that period. If test results for all pathogens were consistent across the testing instances in
578 the 30-day period, we kept the results from the first testing instance and discarded the subsequent instances. If an
579 individual tested negative and then positive, or tested positive then negative, we kept the result for the first positive
580 testing instance and discarded the instances prior to or after that result. We also excluded samples collected as part
581 of Public Health – Seattle & King County’s (PHSKC) contact tracing efforts or through collaborations with
582 community-based organizations.

583 Next, we used a three-step approach to control for sampling variation over time. In the first step, we disaggregated
584 daily pathogen presence and absence data derived from OpenArray testing by clinical setting (hospital or
585 community) and age (≥ 5 years or < 5 years). We then divided the number of positive samples for each pathogen by
586 the total number of specimens tested in each setting and age stratum. Daily proportion positive values were
587 multiplied by the expected age distribution of cases for each pathogen in each setting, which were obtained from the
588 U.S. Outpatient Influenza-like Illness Surveillance Network (ILINet) (71), the U.S. Influenza Hospitalization
589 Surveillance Network (FluSurv-NET)(75), the Washington State Department of Health (76), or published literature
590 (Table S4).

591 In the second step, we combined pathogen proportion positive information from SFS virologic surveillance with
592 citywide syndromic surveillance indicators for respiratory illnesses. A similar approach has been successfully used
593 to model influenza activity over multiple seasons (77). Specifically, we multiplied the age-adjusted proportion
594 positive data by a weekly indicator of the proportion of the King County population seeking care for respiratory
595 illness at emergency departments (ED). This adjustment was applied separately to community and hospital data to
596 generate community and hospital-based incidences for each pathogen. For hospital- and community-based
597 incidences of each pathogen, we multiplied daily proportion positive values for individuals < 5 or ≥ 5 years by the
598 weekly percentage of ED visits coded as general respiratory illness (all endemic viruses except influenza),
599 influenza-like illness, ILI (influenza), or COVID-like illness, CLI (SARS-CoV-2) for each age group.

600 In a third step, hospital- and community-based incidences were each rescaled to fall between 0 and 1 and summed to
601 provide an aggregate measure of incidences in the greater Seattle region. We used this approach to estimate daily
602 incidences from November 2018 to June 2022 for pathogens with sufficient sampling (≥ 400 positive specimens
603 during 2018 – 2022), including influenza A/H1N1, A/H3N2, and B viruses, RSV A and B, seasonal coronaviruses,
604 human parainfluenza viruses, human metapneumovirus, rhinovirus, and adenovirus.

605 We estimated daily SARS-CoV-2 incidence from publicly available COVID-19 case data for King County (76)
606 because SCAN did not test respiratory specimens for SARS-CoV-2 during May and June 2020 (Figure S22).

607 **Statistical analysis**

608 All analyses were performed using R version 4.3.0.

609 **Transmission modeling.** For each pathogen, we estimated time-varying (instantaneous) reproduction numbers, R_t ,
610 by date of infection using the Epidemia R package (26). The instantaneous reproduction number is the number of
611 secondary cases arising from a symptomatic individual at a particular time, assuming conditions remain identical
612 after that time. Epidemia implements a semi-mechanistic Bayesian model using the probabilistic programming
613 language Stan (78). Prior to R_t estimation, we computed proxies of daily case counts of endemic pathogens by
614 multiplying daily incidence rates by 1000 and rounding the resultant values to integers. We estimated reporting
615 delays (i.e., the delay from symptom onset to testing) using kiosk, swab-and-send, and SCAN questionnaire
616 metadata collected from symptomatic individuals who tested positive for endemic respiratory viruses (hRV, $N =$

617 4848 survey responses; influenza viruses, N = 830; RSV, N = 423; hPIV, N = 325; hCoV, N = 666; hMPV, N =
618 148; AdV, N = 443) or SARS-CoV-2 (N = 3566). We used Stan to fit a lognormal distribution to 100 subsampled
619 bootstraps (each with 250 samples drawn with replacement) of the available reporting delay data separately for all
620 endemic respiratory viruses combined and SARS-CoV-2, with a maximum allowed delay of 30 days (EpiNow2 R
621 package (79)). This resulted in a lognormal onset-to-testing delay distribution with mean 0.49 (1.02 SD) days for
622 endemic viruses and mean 0.65 (1.1 SD) days for SARS-CoV-2.

623 To estimate R_t , observed cases were modelled as a function of latent infections in the population, assuming a
624 negative binomial distribution. For each pathogen, we estimated the time distribution for infection-to-case-
625 observation by summing the lognormal-distributed incubation period and the lognormal-distributed reporting delay.
626 Pathogen-specific incubation periods and generation or serial intervals were obtained from published literature
627 (Table S5). Instead of using the renewal equation to propagate infections, we treated infections as latent parameters
628 in the model, because the additional variance around infections leads to a posterior distribution that is easier to
629 sample (26). To control for temporal autocorrelation, we modelled R_t as a daily random walk. Epidemic trajectories
630 were fit independently using Stan's Hamiltonian Monte Carlo sampler. For each model, we ran 4 chains, each for
631 30,000 iterations (including a burn-in period of 15,000 iterations that was discarded), producing a total posterior
632 sample size of 60,000. We verified convergence by confirming that all parameters had sufficiently low \hat{R} values
633 (all $\hat{R} < 1.1$) and sufficiently large effective sample sizes (>15% of the total sample size).

634 Following methods from (7), we evaluated changes in transmissibility during the two weeks before and after two
635 major events during our study period: a major snowstorm in February 2019 and the initiation of COVID-19 social
636 distancing measures in March 2020. We used t-tests for the ratio of two means to compare R_t values before and after
637 each event and Fieller's theorem to calculate the 95% confidence intervals of changes in R_t .

638 **Cross-correlations between human behavior and pathogen transmission.** From Fall 2019 to Summer 2022, we
639 computed Pearson cross-correlations between weekly pathogen specific R_t values and the weekly percent change
640 from baseline in mobility, in rolling five-month windows. During the 2018-2019 respiratory virus season, we
641 computed cross-correlations between daily R_t values and the daily percent change from baseline in mobility in
642 rolling one-month windows, due to limited data at the start of that season (SFS began collecting samples in
643 November 2018) and to better capture the effects of the 12-day snowstorm in February 2019. For time periods that
644 data were available, we estimated weekly cross-correlations and optimal lags between R_t and the proportion of
645 individuals masking in public (June 2020 to June 2022) and between R_t and the Oxford Stringency Index (March
646 2020 to June 2022). In all analyses, cross-correlations were weighted with an exponential decay such that
647 observations at the edges of each time window were weighted approximately 50% less than observations at the
648 window midpoint.

649 For each rolling window, we estimated weighted cross-correlations between mobility behavior and R_t at different
650 lags (up to 4 weeks for 5-month rolling windows and up to 14 days for one-month rolling windows) and extracted
651 the maximum (absolute) coefficient value and the lag (in weeks or days) at which this value occurred (the 'optimal
652 lag'). Negative lag values indicate behavior leads R_t , and positive lag values indicate R_t leads behavior. A lag of 0
653 indicates that two time series are in phase (i.e., synchronous). To generate monthly cross-correlations and lags, we
654 averaged the correlation coefficients and optimal lags of window midpoints that fell within a given calendar month.
655 As an example, for five-month rolling windows each month's statistics are an average of correlation coefficients and
656 lags for dates falling 10 weeks prior to and 10 weeks following each week in that month. To test the statistical
657 significance of cross-correlations for each rolling window, we used a block bootstrap approach to generate 1000
658 samples of each mobility time series in two week increments and recomputed weighted cross-correlations between
659 R_t and mobility for each replicate, yielding a null distribution of 1000 cross-correlations. Cross-correlations between
660 R_t and mobility indicators were considered statistically significant when observed coefficients were outside the
661 bounds of the null distribution's 90% interval.

662 As a sensitivity analysis, we estimated the daily transmissibility of the ancestral SARS-CoV-2 virus and each major
663 variant of concern (VOC), using generation intervals, incubation periods, and reporting delays specific to each
664 lineage, and computed rolling cross-correlations between VOC-specific R_t values and behavioral indicators. Most
665 VOC time series were too short to measure dynamic changes in correlations between R_t and behavior, likely
666 because we could not include the period immediately preceding increases in R_t in VOC-specific analyses.

667 ***Multivariable generalized additive regression models.*** We used multivariable generalized additive regression
668 models (GAMs) to measure non-linear relationships between mobility and R_t and assess the relative importance of
669 different indicators in predicting R_t for each pathogen during key epidemiological timepoints: the beginning of the
670 2019-2020 respiratory virus season (September 2019 – January 2020), the first three months of each of four
671 COVID-19 waves, the first six months of rebound of non-enveloped viruses (June – November 2020), the first three
672 months of rebound of each enveloped virus in 2021 (January – August 2021), and the decline of endemic viruses
673 during the Omicron wave in late 2021 (November 2021 – January 2022). We used the *mgcv* R package (80) to fit
674 each GAM with a Gamma error distribution and log link. Mobility covariates and time trends were modelled using
675 thin plate regression splines (the default smooth for s terms). We specified for the model to add an extra penalty to
676 each term so that it could be penalized to zero (`select = TRUE`), which enables the smoothing parameter estimation
677 to completely remove terms when fitting the model.

678 To further refine our set of predictors and reduce concavity, we used Akaike's Information Criteria corrected for
679 small sample sizes (AICc) to select the best fit "minimal" model for each pathogen, allowing candidate models to
680 include a smoothed weekly time trend and up to two smoothed population behavior terms. Candidate predictors
681 included within-neighborhood movement, between-neighborhood movement, inflow from other WA counties,
682 inflow from other US states, the percentage of devices leaving home, and foot traffic to different categories of POIs,
683 including restaurants, religious organizations, child daycare centers, elementary and high schools, and colleges. For
684 SARS-CoV-2, we also included the proportion of individuals masking in public and NPI stringency as candidate
685 predictors. After performing model selection of candidate models, parameter estimation of the final model was
686 performed by restricted maximum likelihood.

687

688 **References**

- 689 1. B. J. Cowling *et al.*, Impact assessment of non-pharmaceutical interventions against coronavirus disease
690 2019 and influenza in Hong Kong: an observational study. *Lancet Public Health* **5**, e279-e288 (2020).
- 691 2. S. Tempia *et al.*, Decline of influenza and respiratory syncytial virus detection in facility-based surveillance
692 during the COVID-19 pandemic, South Africa, January to October 2020. *Euro Surveill* **26**, (2021).
- 693 3. M. Bardsley *et al.*, Epidemiology of respiratory syncytial virus in children younger than 5 years in England
694 during the COVID-19 pandemic, measured by laboratory, clinical, and syndromic surveillance: a
695 retrospective observational study. *Lancet Infect Dis* **23**, 56-66 (2023).
- 696 4. L. Rodgers *et al.*, Changes in Seasonal Respiratory Illnesses in the United States During the Coronavirus
697 Disease 2019 (COVID-19) Pandemic. *Clin Infect Dis* **73**, S110-S117 (2021).
- 698 5. Q. S. Huang *et al.*, Impact of the COVID-19 nonpharmaceutical interventions on influenza and other
699 respiratory viral infections in New Zealand. *Nat Commun* **12**, 1001 (2021).
- 700 6. V. Dhanasekaran *et al.*, Human seasonal influenza under COVID-19 and the potential consequences of
701 influenza lineage elimination. *Nat Commun* **13**, 1721 (2022).
- 702 7. S. Park, I. C. Michelow, Y. J. Choe, Shifting Patterns of Respiratory Virus Activity Following Social
703 Distancing Measures for Coronavirus Disease 2019 in South Korea. *J Infect Dis* **224**, 1900-1906 (2021).
- 704 8. T. C. Williams, I. Sinha, I. G. Barr, M. Zambon, Transmission of paediatric respiratory syncytial virus and
705 influenza in the wake of the COVID-19 pandemic. *Euro Surveill* **26**, (2021).
- 706 9. J. S. Eden *et al.*, Off-season RSV epidemics in Australia after easing of COVID-19 restrictions. *Nat*
707 *Commun* **13**, 2884 (2022).
- 708 10. M. W. Fong, N. H. L. Leung, B. J. Cowling, P. Wu, Upper Respiratory Infections in Schools and Childcare
709 Centers Reopening after COVID-19 Dismissals, Hong Kong. *Emerg Infect Dis* **27**, 1525-1527 (2021).
- 710 11. S. Chang *et al.*, Mobility network models of COVID-19 explain inequities and inform reopening. *Nature*
711 **589**, 82-87 (2021).
- 712 12. M. U. G. Kraemer *et al.*, The effect of human mobility and control measures on the COVID-19 epidemic in
713 China. *Science* **368**, 493-497 (2020).
- 714 13. K. H. Grantz *et al.*, The use of mobile phone data to inform analysis of COVID-19 pandemic
715 epidemiology. *Nat Commun* **11**, 4961 (2020).
- 716 14. R. E. Baker *et al.*, The impact of COVID-19 nonpharmaceutical interventions on the future dynamics of
717 endemic infections. *Proc Natl Acad Sci U S A* **117**, 30547-30553 (2020).
- 718 15. E. B. White *et al.*, High Influenza Incidence and Disease Severity Among Children and Adolescents Aged
719 <18 Years - United States, 2022-23 Season. *MMWR Morb Mortal Wkly Rep* **72**, 1108-1114 (2023).
- 720 16. J. H. Tanne, US faces triple epidemic of flu, RSV, and covid. *BMJ* **379**, o2681 (2022).
- 721 17. H. Y. Chu *et al.*, The Seattle Flu Study: a multiarm community-based prospective study protocol for
722 assessing influenza prevalence, transmission and genomic epidemiology. *BMJ Open* **10**, e037295 (2020).
- 723 18. T. Bedford *et al.*, Cryptic transmission of SARS-CoV-2 in Washington state. *Science* **370**, 571-575 (2020).
- 724 19. R. Burstein *et al.*, Interactions among 17 respiratory pathogens: a cross-sectional study using clinical and
725 community surveillance data. *medRxiv*, (2022).
- 726 20. C. Hansen *et al.*, Trends in Risk Factors and Symptoms Associated With SARS-CoV-2 and Rhinovirus
727 Test Positivity in King County, Washington, June 2020 to July 2022. *JAMA Netw Open* **5**, e2245861
728 (2022).
- 729 21. M. L. Jackson *et al.*, Effects of weather-related social distancing on city-scale transmission of respiratory
730 viruses: a retrospective cohort study. *BMC Infect Dis* **21**, 335 (2021).
- 731 22. A. E. Kim *et al.*, Evaluating Specimen Quality and Results from a Community-Wide, Home-Based
732 Respiratory Surveillance Study. *J Clin Microbiol* **59**, (2021).
- 733 23. H. Y. Chu *et al.*, Early Detection of Covid-19 through a Citywide Pandemic Surveillance Platform. *New*
734 *England Journal of Medicine* **383**, 185-187 (2020).
- 735 24. T. C. Marcink, J. A. Englund, A. Moscona, in *Viral Infections of Humans*. (2022), chap. Chapter 25-1, pp.
736 1-50.
- 737 25. A. Cori, N. M. Ferguson, C. Fraser, S. Cauchemez, A new framework and software to estimate time-
738 varying reproduction numbers during epidemics. *Am J Epidemiol* **178**, 1505-1512 (2013).
- 739 26. J. A. Scott *et al.*, Epidemia: An R package for semi-mechanistic bayesian modelling of infectious diseases
740 using point processes. *arXiv preprint arXiv:2110.12461*, (2021).
- 741 27. T. Hale *et al.*, A global panel database of pandemic policies (Oxford COVID-19 Government Response
742 Tracker). *Nat Hum Behav* **5**, 529-538 (2021).

- 743 28. A. Reinhart *et al.*, An open repository of real-time COVID-19 indicators. *Proc Natl Acad Sci U S A* **118**,
744 (2021).
- 745 29. S. Cauchemez, A. J. Valleron, P. Y. Boelle, A. Flahault, N. M. Ferguson, Estimating the impact of school
746 closure on influenza transmission from Sentinel data. *Nature* **452**, 750-754 (2008).
- 747 30. V. Peltola *et al.*, Rhinovirus transmission within families with children: incidence of symptomatic and
748 asymptomatic infections. *J Infect Dis* **197**, 382-389 (2008).
- 749 31. C. B. Hall *et al.*, The Burden of Respiratory Syncytial Virus Infection in Young Children. *New England*
750 *Journal of Medicine* **360**, 588-598 (2009).
- 751 32. H. Y. Chu *et al.*, Molecular epidemiology of respiratory syncytial virus transmission in childcare. *J Clin*
752 *Virol* **57**, 343-350 (2013).
- 753 33. E. T. Martin *et al.*, Heterotypic Infection and Spread of Rhinovirus A, B, and C among Childcare
754 Attendees. *J Infect Dis* **218**, 848-855 (2018).
- 755 34. N. G. Davies *et al.*, Age-dependent effects in the transmission and control of COVID-19 epidemics. *Nat*
756 *Med* **26**, 1205-1211 (2020).
- 757 35. S. Poole, N. J. Brendish, A. R. Tanner, T. W. Clark, Physical distancing in schools for SARS-CoV-2 and
758 the resurgence of rhinovirus. *Lancet Respir Med* **8**, e92-e93 (2020).
- 759 36. S. Kitanovski *et al.*, Rhinovirus prevalence as indicator for efficacy of measures against SARS-CoV-2.
760 *BMC Public Health* **21**, 1178 (2021).
- 761 37. H. M. Kim *et al.*, Impact of coronavirus disease 2019 on respiratory surveillance and explanation of high
762 detection rate of human rhinovirus during the pandemic in the Republic of Korea. *Influenza Other Respir*
763 *Viruses* **15**, 721-731 (2021).
- 764 38. I. Kuitunen, M. Artama, M. Haapanen, M. Renko, Rhinovirus spread in children during the COVID-19
765 pandemic despite social restrictions-A nationwide register study in Finland. *J Med Virol* **93**, 6063-6067
766 (2021).
- 767 39. E. Takashita *et al.*, Increased risk of rhinovirus infection in children during the coronavirus disease-19
768 pandemic. *Influenza Other Respir Viruses* **15**, 488-494 (2021).
- 769 40. A. N. M. Kraay *et al.*, Fomite-mediated transmission as a sufficient pathway: a comparative analysis across
770 three viral pathogens. *BMC Infect Dis* **18**, 540 (2018).
- 771 41. J. S. Kutter, M. I. Spronken, P. L. Fraaij, R. A. Fouchier, S. Herfst, Transmission routes of respiratory
772 viruses among humans. *Curr Opin Virol* **28**, 142-151 (2018).
- 773 42. A. F. Murray *et al.*, School-Based Surveillance of Respiratory Pathogens on "High-Touch" Surfaces. *Front*
774 *Pediatr* **9**, 686386 (2021).
- 775 43. N. H. L. Leung *et al.*, Respiratory virus shedding in exhaled breath and efficacy of face masks. *Nat Med* **26**,
776 676-680 (2020).
- 777 44. A. Rössler, L. Riepler, D. Bante, D. von Laer, J. Kimpel, SARS-CoV-2 Omicron Variant Neutralization in
778 Serum from Vaccinated and Convalescent Persons. *N Engl J Med* **386**, 698-700 (2022).
- 779 45. F. P. Lyngse *et al.*, Household transmission of the SARS-CoV-2 Omicron variant in Denmark. *Nat*
780 *Commun* **13**, 5573 (2022).
- 781 46. J. A. Backer *et al.*, Shorter serial intervals in SARS-CoV-2 cases with Omicron BA.1 variant compared
782 with Delta variant, the Netherlands, 13 to 26 December 2021. *Euro Surveill* **27**, (2022).
- 783 47. J. L. H. Tsui *et al.*, Genomic assessment of invasion dynamics of SARS-CoV-2 Omicron BA.1. *Science*
784 **381**, 336-343 (2023).
- 785 48. M. Moriyama, W. J. Hugentobler, A. Iwasaki, Seasonality of Respiratory Viral Infections. *Annu Rev Virol*
786 **7**, 83-101 (2020).
- 787 49. J. Shaman, V. E. Pitzer, C. Viboud, B. T. Grenfell, M. Lipsitch, Absolute humidity and the seasonal onset
788 of influenza in the continental United States. *PLoS Biol* **8**, e1000316 (2010).
- 789 50. R. E. Baker *et al.*, Epidemic dynamics of respiratory syncytial virus in current and future climates. *Nat*
790 *Commun* **10**, 5512 (2019).
- 791 51. V. E. Pitzer *et al.*, Environmental drivers of the spatiotemporal dynamics of respiratory syncytial virus in
792 the United States. *PLoS Pathog* **11**, e1004591 (2015).
- 793 52. D. A. Rankin *et al.*, Circulation of Rhinoviruses and/or Enteroviruses in Pediatric Patients With Acute
794 Respiratory Illness Before and During the COVID-19 Pandemic in the US. *JAMA Netw Open* **6**, e2254909
795 (2023).
- 796 53. R. H. M. Price, C. Graham, S. Ramalingam, Association between viral seasonality and meteorological
797 factors. *Sci Rep* **9**, 929 (2019).

- 798 54. S. Park, Y. Lee, I. C. Michelow, Y. J. Choe, Global Seasonality of Human Coronaviruses: A Systematic
799 Review. *Open Forum Infect Dis* **7**, ofaa443 (2020).
- 800 55. Y. Li *et al.*, Global patterns in monthly activity of influenza virus, respiratory syncytial virus, parainfluenza
801 virus, and metapneumovirus: a systematic analysis. *Lancet Glob Health* **7**, e1031-e1045 (2019).
- 802 56. A. S. Monto *et al.*, Coronavirus Occurrence and Transmission Over 8 Years in the HIVE Cohort of
803 Households in Michigan. *J Infect Dis* **222**, 9-16 (2020).
- 804 57. G. Luca *et al.*, The impact of regular school closure on seasonal influenza epidemics: a data-driven spatial
805 transmission model for Belgium. *BMC Infect Dis* **18**, 29 (2018).
- 806 58. R. M. Eggo, J. G. Scott, A. P. Galvani, L. A. Meyers, Respiratory virus transmission dynamics determine
807 timing of asthma exacerbation peaks: Evidence from a population-level model. *Proc Natl Acad Sci U S A*
808 **113**, 2194-2199 (2016).
- 809 59. C. B. Hall *et al.*, Respiratory Syncytial Virus Infections within Families. *New England Journal of Medicine*
810 **294**, 414-419 (1976).
- 811 60. X. Carbonell-Estrany, J. Quero, I. S. Group, Hospitalization rates for respiratory syncytial virus infection in
812 premature infants born during two consecutive seasons. *Pediatr Infect Dis J* **20**, 874-879 (2001).
- 813 61. I. M. Longini, Jr., J. S. Koopman, A. S. Monto, J. P. Fox, Estimating household and community
814 transmission parameters for influenza. *Am J Epidemiol* **115**, 736-751 (1982).
- 815 62. School Facility ReOpening Survey. (Washington State Office of Superintendent of Public Instruction,
816 2021).
- 817 63. P. Lemey *et al.*, Unifying viral genetics and human transportation data to predict the global transmission
818 dynamics of human influenza H3N2. *PLoS Pathog* **10**, e1003932 (2014).
- 819 64. T. Bedford *et al.*, Global circulation patterns of seasonal influenza viruses vary with antigenic drift. *Nature*
820 **523**, 217-220 (2015).
- 821 65. C. Viboud *et al.*, Synchrony, waves, and spatial hierarchies in the spread of influenza. *Science* **312**, 447-
822 451 (2006).
- 823 66. V. Charu *et al.*, Human mobility and the spatial transmission of influenza in the United States. *PLoS*
824 *Comput Biol* **13**, e1005382 (2017).
- 825 67. F. Reicherz *et al.*, Waning Immunity Against Respiratory Syncytial Virus During the Coronavirus Disease
826 2019 Pandemic. *J Infect Dis* **226**, 2064-2068 (2022).
- 827 68. S. Hamid *et al.*, Seasonality of Respiratory Syncytial Virus - United States, 2017-2023. *MMWR Morb*
828 *Mortal Wkly Rep* **72**, 355-361 (2023).
- 829 69. A. Gimma *et al.*, Changes in social contacts in England during the COVID-19 pandemic between March
830 2020 and March 2021 as measured by the CoMix survey: A repeated cross-sectional study. *PLoS Med* **19**,
831 e1003907 (2022).
- 832 70. M. I. Paredes *et al.*, Local-Scale phylodynamics reveal differential community impact of SARS-CoV-2 in
833 metropolitan US county. *medRxiv*, 2022.2012.2015.22283536 (2022).
- 834 71. FluView Interactive. (National Center for Immunization and Respiratory Diseases (NCIRD) Centers for
835 Disease Control and Prevention, 2023).
- 836 72. Movement Range Maps. (Meta Data for Good, Humanitarian Data Exchange, 2023).
- 837 73. J. A. Salomon *et al.*, The US COVID-19 Trends and Impact Survey: Continuous real-time measurement of
838 COVID-19 symptoms, risks, protective behaviors, testing, and vaccination. *Proc Natl Acad Sci U S A* **118**,
839 (2021).
- 840 74. B. Rader *et al.*, Mask-wearing and control of SARS-CoV-2 transmission in the USA: a cross-sectional
841 study. *Lancet Digit Health* **3**, e148-e157 (2021).
- 842 75. Influenza Hospitalization Surveillance Network (FluSurv-NET). (National Center for Immunization and
843 Respiratory Diseases (NCIRD) Centers for Disease Control and Prevention, 2023).
- 844 76. COVID-19 Data Dashboard. (Washington State Department of Health, 2023).
- 845 77. E. Goldstein, S. Cobey, S. Takahashi, J. C. Miller, M. Lipsitch, Predicting the epidemic sizes of influenza
846 A/H1N1, A/H3N2, and B: a statistical method. *PLoS Med* **8**, e1001051 (2011).
- 847 78. B. Carpenter *et al.*, Stan: A Probabilistic Programming Language. *Journal of Statistical Software* **76**, 1 - 32
848 (2017).
- 849 79. S. Abbott *et al.*, Estimating the time-varying reproduction number of SARS-CoV-2 using national and
850 subnational case counts. *Wellcome Open Research* **5**, (2020).
- 851 80. S. N. Wood, Fast stable restricted maximum likelihood and marginal likelihood estimation of
852 semiparametric generalized linear models. *Journal of the Royal Statistical Society: Series B (Statistical*
853 *Methodology)* **73**, 3-36 (2011).

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888 Data acquisition or curation: ACP, CH, RB, CM, DR, BC, MT, KSF, KK, BP, ZA, JL, TRS, EM, LRS, JSt, LG,
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896 All authors have seen and approved the manuscript.
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898 **Competing Interests**

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907 **Data and materials availability**

908 Aggregated epidemiological and mobility data and code to reproduce analyses and figures will be made available in
909 a GitHub repository and Dryad at the time of publication acceptance. Access to deidentified study participant data
910 requires a signed data access agreement with the Seattle Flu Alliance and can be made available to researchers
911 whose proposed use of the data is approved by study investigators. Requests for data access should be submitted to
912 data@seattleflu.org. Mobility metrics were generated using SafeGraph Weekly Patterns and Social Distancing
913 datasets and Meta Data for Good Movement Range Maps, which were originally made freely available to academics
914 in response to the COVID-19 pandemic. The SafeGraph Weekly Patterns dataset is currently available to academics
915 for non-commercial use through an institutional university subscription or individual subscription to Dewey
916 (<https://www.deweydata.io/>). The data access agreement with Dewey does not permit sharing of the raw data. Meta
917 Data for Good Movement Range Maps are publicly accessible through the Humanitarian Data Exchange
918 (<https://data.humdata.org/dataset/movement-range-maps>).

919 **Disclaimer**

920 The findings and conclusions in this report are those of the authors and do not necessarily represent the official
921 position of the US National Institutes of Health or the US government.