Prevention and Control (5). Similar to our findings, a study from Denmark reported 47 Omicron BA.2 reinfections that occurred 20–60 days after a primary BA.1 infection (M. Stegger et al., unpub. data, https://www.medrxiv.org/content/10.1101/2022.02.19.22271112v1).

The first limitation of our study is that the number of cases was small. Second, we cannot exclude that some cases might have been concurrent infections with different subvariants, notably in the 3 cases that had a 7-day interval between the detection of 2 subvariants. In Marseille, the short time between emergence of different Omicron subvariants might have favored co-infections with different subvariants circulating within the population (Figure). Co-infections can be missed if the quantitative PCR has inadequate sensitivity, and whole-genome sequencing might fail to detect a variant with low prevalence in a patient. Finally, because most reinfection cases were identified from samples transferred to our laboratory by external entities, we were unable to describe COVID-19 vaccination and clinical status of the patients. Nonetheless, our results suggest that the currently used definitions for SARS-CoV-2 reinfection require revision with regard to the duration between primary and secondary infections.

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Human Parainfluenza Virus in Homeless Shelters before and during the COVID-19 Pandemic, Washington, USA

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To determine the epidemiology of human parainfluenza virus in homeless shelters during the COVID-19 pandemic, we analyzed data and sequences from respiratory specimens collected in 23 shelters in Washington, USA, during 2019–2021. Two clusters in children were genetically similar by shelter of origin. Shelter-specific interventions are needed to reduce these infections.

Human parainfluenza virus (HPIV) contributes to acute respiratory tract infection burden in young children (1) and adults (2). Persons experiencing homelessness are among those at risk for respiratory viral complications caused by chronic disease burden, mental illness, and social inequities. Homeless shelters might lack resources to reduce viral transmission by using nonpharmaceutical interventions (NPIs). We describe HPIV epidemiology in homeless shelters in King County, Washington, USA, before and during the COVID-19 pandemic.

We analyzed respiratory virus surveillance data from 2 previously described homeless shelter studies (3,4) conducted during October 2019–May 2021. Eligible participants were residents at 1 of 23 homeless shelters who were ≥3 months of age and had a cough or ≥2 other acute respiratory illness symptoms. At enrollment, consenting participants or guardians completed questionnaires, and upper respiratory specimens were collected; each enrollment was considered 1 encounter. Once a month, persons were eligible to enroll, regardless of symptoms. Beginning April 1, 2020, enrollment expanded to residents and staff, regardless of symptoms. Participants could enroll multiple times; encounters were linked by name and birthdate.

We tested samples by using a TaqMan reverse transcription PCR platform that included influenza virus (A, B, C), respiratory syncytial virus, HPIV (1–4), human coronaviruses, rhinovirus, enterovirus, human bocavirus, human parechovirus, human metapneumovirus, adenovirus, and SARS-CoV-2 (beginning January 1, 2020). A cycle threshold value was generated. We typed HPIV-positive specimens, performed whole-genome sequencing by using hybrid capture on specimens that had a cycle threshold value <22 (Appendix, https://wwwnc.cdc.gov/EID/article/28/11/22-1156-App1.pdf), and submitted genomes to GenBank (Appendix Table 1). We aligned shelter consensus genomes generated with corresponding HPIV type genomes from GenBank, generated type-specific phylogenetic trees, and visualized trees by using NextStrain Auspice software (https://github.com). We analyzed the data descriptively by using SAS software version 9.4 (https://www.sas.com).

During October 2019–May 2021, the study conducted 14,464 encounters with 3,281 unique participants (median age 37 years, range 0.3–85 years; 16% children; 17% shelter staff) (Appendix Figure 1). Among 1,569 encounters with positive virus test results, 32 (2%) encounters from 29 unique participants were HPIV positive (median age 29 years, range 0.3–64 years; 62% children, 45% female, 52% white, 100% resident; 10% had ≥1 chronic condition) (Appendix Table 2). Most HPIV-positive encounters

Table. Human parainfluenza virus detection across 23 homeless shelters, King County, Washington, USA, October 2019–May 2021*

Time period	Type of shelter	Total	Human parainfluenza virus, no. (%) positive	Human parainfluenza virus types
Before April 1, 2020	Shelters: family (sites D, E, O)	303	16 (5.3)	HPIV-1, n = 5; HPIV-3, n = 6; HPIV-4, n = 5;
	Shelters: adults 18–25 y (site C)	89	1 (1.1)	HPIV-1, n = 1
	Shelters: adults ≥18 y (sites A, B, F, L)	845	3 (0.4)	HPIV-1, n = 2; HPIV untyped, n = 1
	Shelters: adults ≥50 y (site M)	453	3 (0.7)	HPIV-1, n = 2; HPIV untyped, n = 1
After April 1, 2020	Shelters: family (sites D, E, H, N, O, OF, OG)	4,764	8 (0.2)	HPIV-3, n = 5; HPIV untyped, n = 3
	Shelters: adults 18–25 y (sites C, OH)	1,228	0	NA
	Shelters: adults ≥18 y (sites A, B, F, G, J, K, L, OB, OD)	6,078	1 (0.02)	HPIV untyped, n = 1
	Shelters: adults ≥50 y (sites I, M, OA, OC, OE)	661	0	NA
Total		14,421†	32 (0.2)	HPIV-1, n = 10; HPIV-3, n = 11; HPIV-4, n = 5; HPIV untyped, n = 6

*A Washington State Stay-At-Home ordinance we issued on March 23, 2020. HPIV, human parainfluenza virus; NA, not available.
†n = 43 encounters for which dates were missing were not included (none involved human parainfluenza-positive specimens).

(72%) occurred before April 1, 2020, and the highest HPIV-positive percentage was observed in family shelters (Table).

Six of 32 encounters involved viral co-infections with HPIV (rhinovirus, adenovirus, human bocavi-rus, enterovirus, and human parechovirus). Participants with HPIV infection reported symptoms at 25 (78%) encounters. Commonly reported symptoms included rhinorrhea (95%), cough (74%), sore throat (53%), and subjective fevers (47%) (Appendix Table
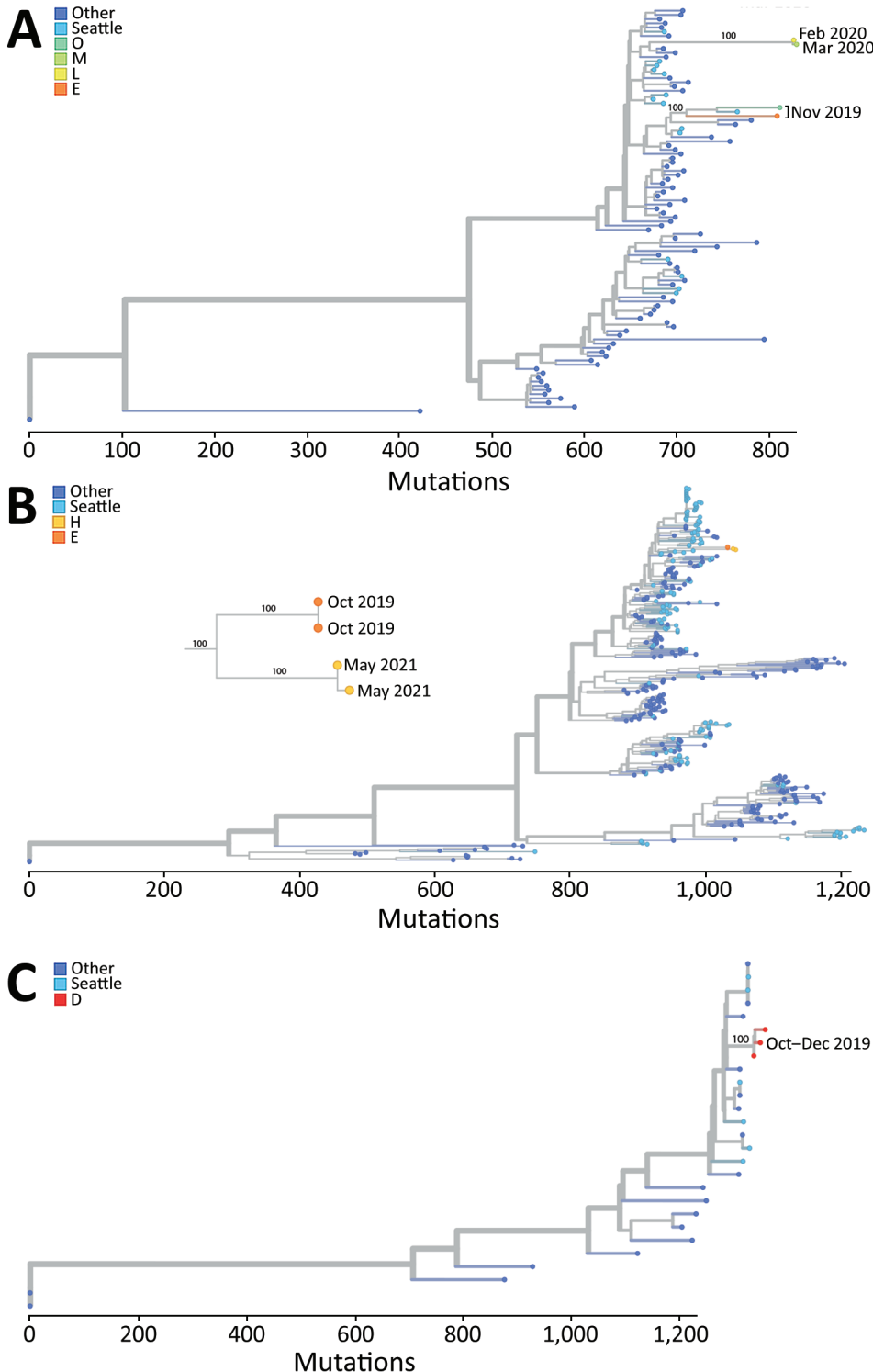

Figure. Phylogenetic trees of human parainfluenza viruses in homeless shelters, King County, Washington, USA, October 2019–May 2021. A) Human parainfluenza virus 1; B) human parainfluenza virus 3; C) human parainfluenza virus 4a. Letters in keys indicate different homeless shelters from which sequenced specimens were collected. Other indicates genomic data from locations not in Seattle, Washington. Seattle indicates genomic data from Seattle other than homeless shelters in this study.

3). HPIV-positive specimens occurred every month during October 2019–April 2020 (Appendix Figure 2). Only 2 HPIV infections were identified during May 2020–April 2021, despite an average of 954 monthly encounters. Six HPIV infections occurred during May 2021 (Appendix Figure 3).

Of 32 HPIV-positive specimens, we identified 3 of the 4 HPIV serotypes: 10 HPIV-1, 11 HPIV-3, and 5 HPIV-4. Six specimens were untypeable. Sequencing of 16 specimens generated 11 sequences (4 HPIV-1, 4 HPIV-3, and 3 HPIV-4a) from 6 shelters (Figure). HPIV-1 sequences formed 2 clusters (100% bootstrap support for each cluster) by collection date in a maximum-likelihood tree that included 94 GenBank HPIV-1 genomes. Both HPIV-3 and HPIV-4a sequences formed single genetic clusters (100% bootstrap support for each cluster) in a maximum-likelihood tree that included 397 GenBank HPIV-3 and 24 HPIV-4a genomes. The HPIV-3 clusters involved HPIV-positive specimens from shelters E (October 2019) and H (May 2021); both shelters housed adults and children. In shelter E, HPIV-3–positive specimens resulted from 6 encounters involving 5 unique participants (all children) spanning 9 days, and 2 specimens were sequenced. In shelter H, 5 HPIV-3 encounters involving 4 unique participants (all children) spanned 17 days, and 2 specimens were sequenced. The sequenced HPIV-3 specimens from shelters E and H, each from unique persons, formed 2 subclusters, each with 100% bootstrap support, corresponding to shelter and collection date.

Respiratory viruses are increasingly appreciated as major pathogens in homeless shelters (5,6), We identified HPIV infections in shelter residents of all ages, although predominantly in children. Family shelters that have mixed populations of adults and children had the greatest percentage of HPIV detections. Two pediatric HPIV-3 clusters occurred before and during the COVID-19 pandemic with genetic clustering by shelter. After the Washington stay-at-home ordinance on March 23, 2020, overall numbers of HPIV infections decreased. These reductions (7) were probably in part caused by community implementation of NPIs because respiratory droplets are probably the main mode of HPIV transmission (8). However, HPIV has been detected on environmental surfaces (9), and shelter site resources might not enable adequate social distancing and air quality.

The pediatric HPIV-3 cases illustrate the need for mitigation guidance to reduce intrashelter HPIV transmission, particularly because younger children have higher upper respiratory tract viral levels than older persons (10). Limitations of this study included potential selection bias, a lack of site-specific NPI data, cross-sectional study design, and inability to compare concurrent shelter results to community HPIV epidemiology. These HPIV data provide information on site-specific characteristics to inform public health guidance.

About the Author

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Presence of *Spirometra mansoni*, Causative Agent of Sparganosis, in South America

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We report molecular identification of an adult *Spirometra mansoni* tapeworm retrieved from a crab-eating fox (*Cerdocyon thous*) in Colombia, confirming presence of this parasite in South America. This tapeworm is the causative agent of human sparganosis, commonly reported from Southeast Asia, and represents the second congeneric species with known zoonotic potential in the Americas.

Sparganosis is a neglected human zoonosis caused by migrating larval stages of the broad tapeworm genus *Spirometra* (Diphyllobothriidea), whose natural definitive hosts include wild and domestic canids and felids. The life cycle of this tapeworm involves 2 intermediate hosts: a freshwater copepod crustacean as the first and various vertebrates, mostly amphibians, as the second. Human infections are commonly reported from Southeast Asia and propagate most often in the form of subcutaneous sparganosis; however, the larvae can enter other organs or parts of central nervous system and cause damage.

Taxonomy of *Spirometra* remains highly complicated. Numerous species of *Spirometra* have been described, often poorly (*1*), and representatives of just 6 species-level lineages have been characterized molecularly so far, a key prerequisite to achieve a convincing tapeworm identification when only strobila fragments or larval stages are available. Limitations of morphologic characters of *Spirometra* are numerous and include characters' great intraspecific and even intra-individual variability (overview of problematic traits in *2*). Molecular sequence data thus represent the only unequivocal method of species identification.

Previous phylogenetic analysis of *Spirometra* has shown that the geographic distribution of the 6 lineages respects continental borders (*2*). North

[1]These authors contributed equally to this article.

Human Parainfluenza Virus in Homeless Shelters before and during the COVID-19 Pandemic, Washington, USA

Appendix.

Supplemental Methods

We analyzed data from 2 previously described studies: 1) a randomized control trial of an influenza test and treat (NCT04141917) (*1*) and a SARS-CoV-2 surveillance study (*2*) of 23 homeless shelters across King County, Washington during October 2019–May 2021. These studies took place sequentially and data from cross-sectional survey responses and respiratory specimens were collected at study enrollment. Before April 1, 2020, eligible participants were shelter residents ≥3 months of age who had a cough or at least 2 other acute respiratory illness symptoms, including subjective fever, headache, sore throat, rhinorrhea, shortness of breath, and myalgias, and were recruited from shelter site staffed kiosks. For participants <18 years of age, diarrhea, rash, and ear pain or discharge were also included. Monthly enrollment of participants not meeting the symptom requirements were also permitted to enroll in the study for shelter surveillance. From April 1, 2020, onward, enrollment eligibility included residents and staff regardless of symptoms when SARS-CoV-2 community circulation was detected. Participants were not followed-up longitudinally, but were permitted to enroll multiple times. Multiple participant encounters were linked by name and birthdate. One-day surge testing events took place as part of Public Health Seattle and King County SARS-CoV-2 contact tracing efforts starting on March 30, 2020, in which study participation was offered to all residents and shelter staff with or without symptoms.

Study consent was obtained from all persons ≥18 years of age or from a guardian for persons <18 years of age. Study participant assent was required for those persons 13–17 years of age. After consent was obtained, an enrollment questionnaire was administered collecting information sociodemographics, current tobacco use, self-reported chronic conditions, and illness

course symptoms. Loss of taste or smell was included after April 1, 2020. Collected sociodemographic data included shelter site location, birthdate, sex at birth, race and ethnicity, pregnancy status, and current tobacco use status. Self-reported chronic conditions included neurologic disease, cardiovascular disease, asthma, bronchitis, chronic obstructive pulmonary disease, hepatic disease, diabetes mellitus, immunosuppression, and cancer or another condition not listed. Illness course questions in the survey included self-reported symptoms and illness duration. Questionnaire symptoms included rhinorrhea, cough, sore throat, fatigue, myalgias, headaches, subjective fevers, shortness of breath, sweats, nausea or vomiting, chills, diarrhea, rash, ear pain or discharge and loss of taste or smell (added after April 1, 2020). Participants not reporting any of these symptoms were considered asymptomatic up until study enrollment. Shelter facility specifics including targeted resident demographics that the shelter served were obtained from shelter management. Survey questionnaires were administered on an electronic tablet at the time of respiratory sample collection and data was stored on Research Electronic Data Capture. A respiratory sample was also collected at enrollment by using midturbinate sterile nylon flocked swabs (Copan Diagnostics, https://www.copanusa.com) with anterior nares swabs briefly used during July 22, 2020–November 1, 2020, because of COVID-19 pandemic–associated supply changes. Respiratory specimens were initially obtained by study staff. However, procedure was converted to study staff supervised self-collected swab specimens with the community spread of SARS-CoV-2. We prepared this analysis by using deidentified study data. The University of Washington IRB (study no. 00007800) approved this study.

Specimens were stored at 4°C in universal transport medium. Respiratory specimens were tested by using the TaqMan RT-PCR platform (Thermo Fisher OpenArray, https://www.thermofisher.com) that included influenza virus (A, B and C), respiratory syncytial virus (RSV-A and RSV-B), human parainfluenza (HPIV 1-4), human coronaviruses (HCoV-NL63, HCoV-OC43, HCoV-229E, HCoV-HKU1), rhinovirus, enterovirus, human bocavirus, human parechovirus, human metapneumovirus, adenovirus and SARS-CoV-2 (from January 1, 2020 onward). Specimens collected from January 1, 2020, onward were tested for SARS-CoV-2 with those collected after February 25, 2020 tested prospectively. For the purposes of this study, we categorized SARS-CoV-2 inconclusive results as negative. HPIV co-infections were defined as detection of HPIV and $\geq$1 other respiratory virus. A cycle threshold (Ct) was generated for each virus-positive sample.

Human Parainfluenza Typing

RNA was extracted from specimens by using the MagnaPure 96 DNA and Viral NA Small Volume Kit, (Roche, https://www.roche.com). Viral NA Universal SV 4.0 protocol (200 µL input, 50 µL elution) and were used for HPIV typing and sequencing. All 32 HPIV-positive specimens were typed by using a multiplex RT-PCR for HPIV-1, -3, and -4 (*3*) and a separate PCR for HPIV-2 (*4*).

Amplification reactions of 35-µL reactions contained 10 µL of extracted RNA and the AgPath-ID One Step RT-PCR enzyme and master mix (Ambion, https://www.thermofisher.com). Multiplex reactions contained 250 nmol/L each of forward and reverse primers for HPIV-1 and HPIV-3, 375 nmol/L each of forward and reverse primers for HPIV-4, and 100 nmol/L each of probes for HPIV-1, HPIV-3, and HPIV-4. HPIV-2 reactions contained 250 nmol/L each of forward and reverse primers and 100 nmol/L of probe. Amplification was conducted in ABI 7500 Thermocyclers (https://www.thermofisher.com) by using 10 minutes of reverse transcription at 48°C, 10 minutes of denaturation at 98°C, and 40 cycles of 15 seconds at 95°C and 45 seconds at 60°C. Specimens were considered positive if the PCR Ct value was <40 based on established cutoffs for laboratory-developed tests. HPIV-positive specimens that were not positive for HPIV 1-4 are listed as untyped.

Genomic Sequencing

HPIV sequencing was attempted on specimens that had with Ct values <22 by using an oligonucleotide probe capture panel targeting common respiratory viruses including HPIV. Sequencing libraries were prepared from extracted RNA by using the Respiratory Virus Oligo Panel version 2 (Illumina, https://www.illumina.com) according to the manufacturer's instructions. In brief, RNA was converted to double-stranded complementary DNA, and precapture libraries were prepared and normalized by using the Illumina RNA Prep with Enrichment (L) Tagmentation Kit. After hybridization with capture probes, the resulting enriched library was cleaned by using AMPure XP beads (https://www.beckmancoulter.com). Libraries were quantified by using the Quant-IT HS dsDNA Kit (https://www.thermofisher.com) on the Victor Nivo Multimode PlateReader https://www.thermofisher.com), and library size was checked by using the Agilent 4200 TapeStation system (https://www.agilent.com).

Human Parainfluenza Pipeline Description

Consensus genomes were generated by using a custom bioinformatic pipeline (https://github.com/greninger-lab/revica). In brief, raw reads were trimmed by using Trimmomatic version 0.39 (https://bioweb.pasteur.fr) and the settings ILLUMINACLIP:2:30:10:1:true, SLIDINGWINDOW: 4:20, LEADING: 3, TRAILING: 3, and MINLEN: 35. Trimmed reads were mapped to a multi-fasta reference containing complete genomes of human parainfluenza virus by using BBMap version 38.96 (https://anaconda.org). The reference with the highest median coverage was selected as the initial reference for consensus calling. Trimmed reads were then mapped again to the initial reference by using BBMap, and the resulting alignment was used to call a consensus genome by using Samtools version 1.15 (http://www.htslib.org) and iVar version 1.3.1 (https://github.com). A minimum coverage of 3, a minimum base quality of 15, and a minimum frequency threshold of 0.6 were required to call consensus. Regions with less than the minimum coverage were called Ns. This process of reference-based consensus calling was iterated 3 times, and leading and trailing Ns were trimmed to generate a final consensus. Generated consensus genomes were submitted to GenBank under accessions nos. ON778017–ON778027 (Appendix Table 1).

Computational Analysis

Publicly available parainfluenza genome sequences were downloaded from GenBank. Consensus genomes generated for this project were aligned with others of the same subtype from GenBank by using MAFFT version 7.453 (https://mafft.cbrc.jp). Maximum-likelihood phylogenetic trees were generated for each subtype, and bootstrap values were calculated by using IQ-Tree version 2.2.0 (www.iqtree.org). Trees were visualized by using NextStrain Auspice software (https://docs.nextstrain.org). We analyzed our data descriptively by using SAS software version 9.4 (https://www.sas.com).

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Appendix Table 1. GenBank accession numbers for human parainfluenza virus sequences

Sequence name	Collection date	Virus type	Shelter of origin	Participant age, years	GenBank accession no.
PIV1_1	2019 Nov 4	1	O	31	ON778017
PIV1_2	2019 Nov 22	1	E	1	ON778018
PIV1_3	2020 Feb 28	1	L	46	ON778019
PIV1_4	2020 Mar 31	1	M	64	ON778020
PIV3_1	2019 Oct 15	3	E	1	ON778021
PIV3_2	2019 Oct 21	3	E	3	ON778022
PIV3_3	2021 May 13	3	H	3	ON778023
PIV3_4	2021 May 25	3	H	4	ON778024
PIV4a_1	2019 Oct 28	4	D	11	ON778025
PIV4a_2	2019 Nov 14	4	D	2	ON778026
PIV4a_3	2019 Dec 3	4	D	0	ON778027

Appendix Table 2. Demographics and health characteristics of study participants*

Characteristic.	Human parainfluenza virus†	Other respiratory viruses‡	No respiratory virus detected§
No. unique participants	29	973	2,996
Age, years			
Overall, median (range)	20 (0.3–64)	33 (0.3–85)	37 (0.3–85)
<5	12 (44.4)	99 (10.2)	154 (5.1)
5–11	3 (11.1)	93 (9.6)	189 (6.3)
12–17	1 (3.7)	43 (4.4)	101 (3.4)
18–49	7 (25.9)	485 (49.9)	1,635 (54.6)
50–64	4 (14.8)	212 (21.8)	752 (25.1)
≥65	0	41 (4.2)	164 (5.5)
Sex			
M	15 (51.7)	570 (58.6)	1,815 (60.6)
F	13 (44.8)	389 (40.0)	1,127 (37.6)
Other	0	2 (0.2)	16 (0.5)
Prefer not to say	1 (3.5)	12 (1.2)	38 (1.3)
Race			
White	15 (51.7)	398 (40.9)	1,208 (40.3)
Black or African American	8 (27.6)	321 (33.0)	950 (31.7)
Asian	0	17 (1.8)	114 (3.8)
American Indian or Alaskan Native	1 (3.5)	30 (3.1)	121 (4.0)
Native Hawaiian or Pacific Islander	1 (3.5)	64 (6.6)	129 (4.3)
Other	1 (3.5)	64 (6.6)	263 (8.8)
Prefer not to say	3 (10.3)	79 (8.1)	211 (7.0)
Ethnicity			
Hispanic	3 (10.3)	155 (15.9)	440 (14.7)
Non-Hispanic	25 (86.2)	797 (81.9)	2,502 (83.5)
Unknown	1 (3.5)	21 (2.2)	54 (1.8)
Pregnancy status for women of child-bearing age	n = 5	n = 258	n = 770

Characteristic.	Human parainfluenza virus†	Other respiratory viruses‡	No respiratory virus detected§
Pregnant	1 (20.0)	5 (1.9)	13 (1.7)
Not pregnant	2 (40.0)	75 (29.1)	128 (16.6)
Prefer not to say	2 (40.0)	178 (69.0)	629 (81.7)
Smoking status			
Current tobacco use	6 (20.7)	405 (41.6)	1368 (45.7)
Chronic conditions			
None	26 (89.7)	699 (71.8)	2081 (69.5)
≥1 chronic condition	3 (10.3)	274 (28.2)	915 (30.5)
Neurologic disease	0	23 (2.8)	63 (2.6)
Cardiovascular disease	0	23 (2.4)	95 (3.2)
Asthma	1 (3.5)	117 (12.0)	393 (13.1)
Bronchitis	1 (3.5)	24 (2.5)	93 (3.1)
Chronic obstructive pulmonary disease	1 (3.5)	37 (3.8)	116 (3.9)
Hepatic disease	1 (3.5)	21 (2.2)	85 (2.8)
Diabetes mellitus	1 (3.5)	62 (6.4)	199 (6.6)
Immunosuppression	0	14 (1.4)	36 (1.2)
Cancer	1 (3.5)	16 (1.6)	57 (1.9)
Other	0	11 (1.1)	31 (1.0)
Shelter staff	0	109 (11.2)	550 (18.4)
No. encounters	32	1,537	12,895

*Values are no. (%) where indicated. Participant groups were not mutually exclusive because some participants might have encounters for which human parainfluenza was detected and others for which other virus or no virus was detected.
†n = 2 encounters for which participant age is missing and were not included in the age analysis.
‡There were n = 22 encounters for which an inconclusive test result for severe acute respiratory syndrome virus 2 was recategorized as a negative result; there were no other pathogens detected in these specimens and 17 of these specimens came from asymptomatic participants.
§n = 1 encounter for which participant age is missing and was not included in the age analysis.

Appendix Table 3. Participant symptoms among those with human parainfluenza only*

Symptom	Human parainfluenza only	No virus detected
Encounters	26	12,895
Unique participants	25	2,996
Encounter symptoms, no. (%)		
Asymptomatic	7 (26.9)	10,700 (83.0)
Symptomatic	19 (73.1)	2,195 (17.0)
Rhinorrhea	18 (94.7)	1,277 (58.2)
Cough	14 (73.7)	1,070 (48.8)
Sore throat	10 (52.6)	662 (30.2)
Subjective fevers	9 (47.4)	459 (20.9)
Fatigue	8 (42.1)	749 (34.1)
Myalgias	6 (31.6)	711 (32.4)
Chills	6 (31.6)	444 (20.2)
Diarrhea	6 (31.6)	296 (13.5)
Shortness of breath	5 (26.3)	348 (15.9)
Sweats	4 (21.1)	427 (19.5)
Headaches	3 (15.8)	711 (32.4)
Ear pain or discharge	3 (15.8)	148 (6.7)
Rash	3 (15.8)	125 (5.7)
Nausea or vomiting	2 (10.5)	497 (22.6)
	n = 8	n = 1,904
Loss of taste or smell†	0	34 (1.6)
Influenza-like illness‡	9 (47.4)	353 (16.1)
COVID-19-like illness§	9 (47.4)	328 (14.9)
Influenza-like illness and COVID-19-like illness symptoms	9 (47.4)	315 (14.4)

*COVID-19, coronavirus disease.
†Loss of taste or smell was collected from April 1, 2020, onward.
‡Defined as fever with cough or sore throat.
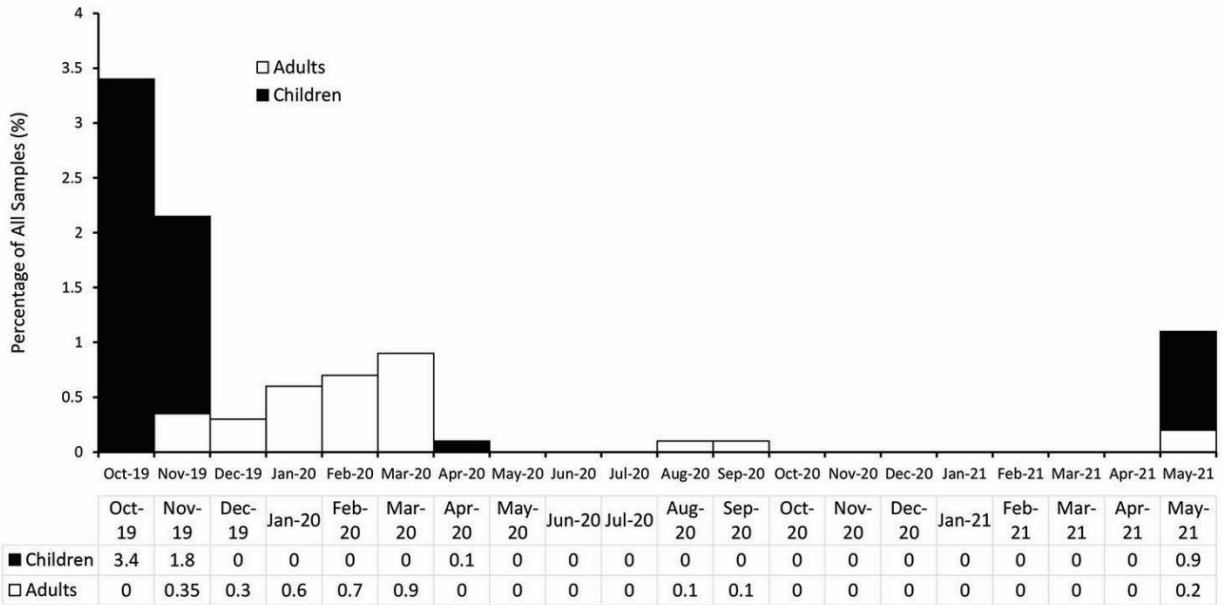§Defined as fever with cough or shortness of breath.

Appendix Table 4. Sequences of primers and probes used in this study*

| HPIV type	Primer/probe name	Primer/probe sequence, 5'→3'
HPIV-1	Forward primer 1	CGA GCG AGT CGA TTT ATT ACC A
	Reverse primer 1	CAA TCC GGT TAA CAT AAT TTG A
	Probe 1	Cy5-TGG CAT TAA AAG AG+G C+AG +GA-BHQ
HPIV-2	Forward primer 2	TGC ATG TTT TAT AAC TAC TGA TCT TGC TAA
	Reverse primer 2	GTT CGA GCA AAA TGG ATT ATG GT
	Probe 2	VIC-ACT GTC TTC AAT GGA GAT AT-MGB
HPIV-3	Forward primer 3	TGC TGT TCG ATG CCA ACA A
	Reverse primer 3	ATT TTA TGC TCC TAT CTA GTG GAA GAC A
	Probe 3	FAM-TTG CTC TTG CTC A-MGB
HPIV-4	Forward primer 4	TGC CAA ATC GGC AAT AA ACA
	Reverse primer 4	GGC TCT GGC AGC AAT CAT AAG
	Probe 4	VIC-TGA TTC TGC ATT GAT GTG G-MGB

*HPIV, human parainfluenza virus.
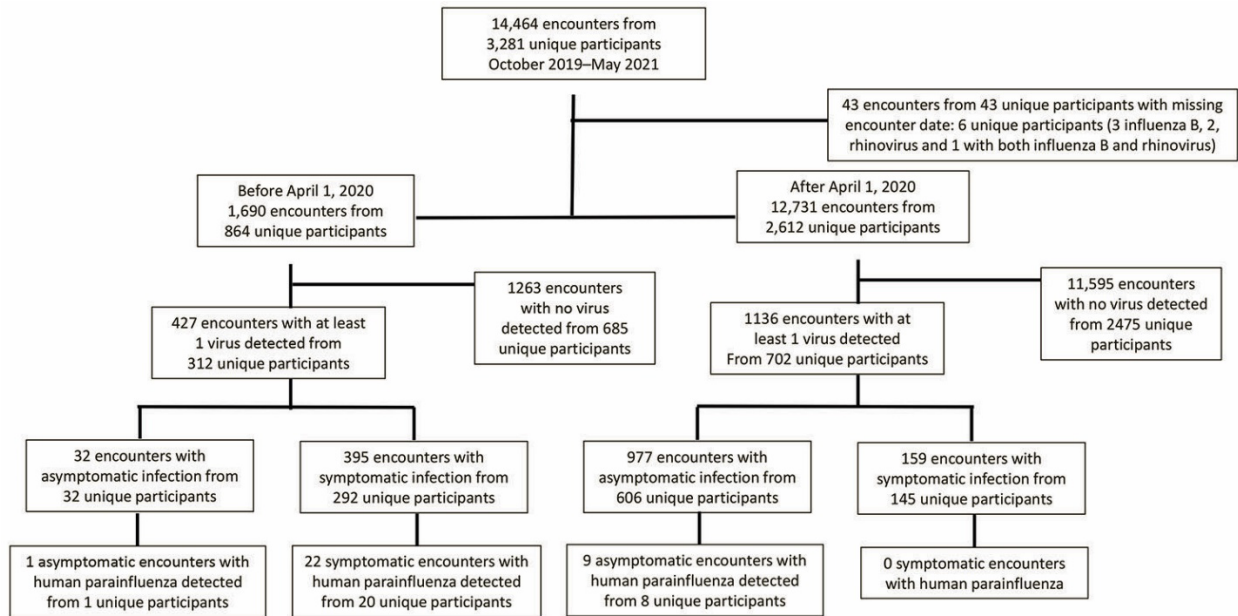
Appendix Figure 1. Homeless shelter study flowchart for human parainfluenza, King County, Washington, USA.

	Oct-19	Nov-19	Dec-19	Jan-20	Feb-20	Mar-20	Apr-20	May-20	Jun-20	Jul-20	Aug-20	Sep-20	Oct-20	Nov-20	Dec-20	Jan-21	Feb-21	Mar-21	Apr-21	May-21
■ Children	3.4	1.8	0	0	0	0	0.1	0	0	0	0	0	0	0	0	0	0	0	0	0.9
□ Adults	0	0.35	0.3	0.6	0.7	0.9	0	0	0	0	0.1	0.1	0	0	0	0	0	0	0	0.2

Appendix Figure 2. Homeless shelter human parainfluenza virus–positive specimens, October 2019–May 2021, King County, Washington, USA.
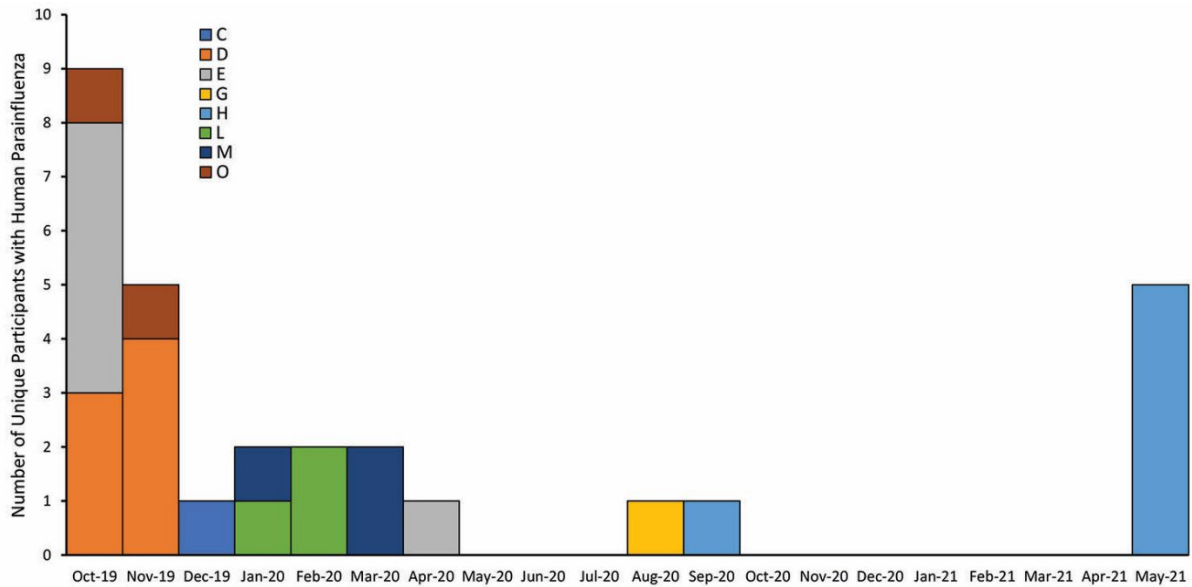
Appendix Figure 3. Unique participants with human parainfluenza virus by shelter site, King County, Washington, USA.
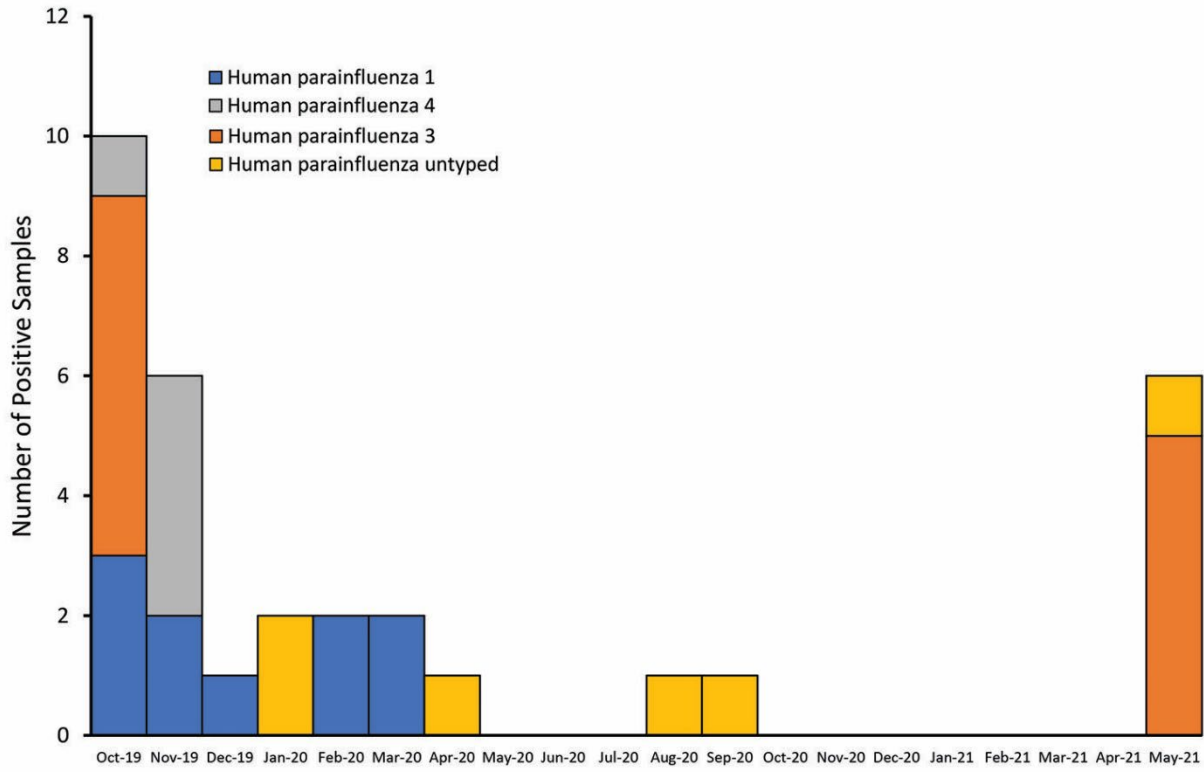
Appendix Figure 4. Human parainfluenza virus–positive specimens by human parainfluenza virus type, King County, Washington, USA.