Household Transmission of Severe Acute Respiratory Syndrome Coronavirus 2 in the United States: Living Density, Viral Load, and Disproportionate Impact on Communities of Color

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Background. Households are hot spots for severe acute respiratory syndrome coronavirus 2 transmission.

Methods. This prospective study enrolled 100 coronavirus disease 2019 (COVID-19) cases and 208 of their household members in North Carolina though October 2020, including 44% who identified as Hispanic or non-White. Households were enrolled a median of 6 days from symptom onset in the index case. Incident secondary cases within the household were detected using quantitative polymerase chain reaction of weekly nasal swabs (days 7, 14, 21) or by seroconversion at day 28.

Results. Excluding 73 household contacts who were PCR-positive at baseline, the secondary attack rate (SAR) among household contacts was 32% (33 of 103; 95% confidence interval [CI], 22%–44%). The majority of cases occurred by day 7, with later cases confirmed as household-acquired by viral sequencing. Infected persons in the same household had similar nasopharyngeal viral loads (intraclass correlation coefficient = 0.45; 95% CI, .23–.62). Households with secondary transmission had index cases with a median viral load that was 1.4 $\log_{10}$ higher than those without transmission (P = .03), as well as higher living density (more than 3 persons occupying fewer than 6 rooms; odds ratio, 3.3; 95% CI, 1.02–10.9). Minority households were more likely to experience high living density and had a higher risk of incident infection than did White households (SAR, 51% vs 19%; P = .01).

Conclusions. Household crowding in the context of high-inoculum infections may amplify the spread of COVID-19, potentially contributing to disproportionate impact on communities of color.

Keywords. SARS-CoV-2; household transmission; viral load; secondary attack rate; living density.

Households are hot spots for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) transmission [1]. Person-to-person transmission is difficult to control in shared living spaces. For those who isolate at home with young children in small living spaces, following guidelines to physically distance and use separate sleeping, eating, and lavatory facilities is difficult [2]. Because the period of peak infectiousness starts prior to the onset of symptoms, spread can occur before these measures are taken [3–5].

Meta-analyses of the secondary household attack rate (SAR) in the first 6 months of the pandemic ranged from 15% to 20% [6, 7], but these analyses incorporated both retrospective and prospective analyses [6, 7]. Prospective testing of household contacts (HCs) regardless of symptom status is required to capture all secondary cases. Previously identified risk factors associated with increased transmission include the presence of symptoms [6–8] and high viral load in the index case [9]. Among HCs, spouses and those aged >18 years (ie, adults compared with children) are more likely to acquire infection [6, 7, 10]. Measuring secondary household attack rates in vulnerable communities and identifying risk factors for transmission in these communities are critical.

The University of North Carolina (UNC) COVID-19 Household Transmission Study (CO-HOST) is the largest, single-site, observational, household cohort in the United States to date and the most ethnically and racially diverse. Weekly sampling for quantitative viral loads combined with SARS-CoV-2 antibody testing at baseline and at 1 month provided an extended period to evaluate transmission. Our objective in this study was to measure the incident SAR in a setting where

infected individuals were asked to quarantine at home and given standard guidance. Household and individual demographics as well as daily symptoms and weekly viral loads were collected to identify risk factors and timing of household transmission.

METHODS

Study Design and Enrollment

In the CO-HOST study, we evaluated SARS-CoV-2 acquisition among persons undergoing quarantine in their home after a care-seeking household member (the index case) tested positive for SARS-CoV-2. Recruitment occurred between April 2020 and October 2020, prior to the introduction of SARS-CoV-2 vaccines in the United States. Inclusion criteria for the index cases were age >18 years with a positive qualitative nasopharyngeal (NP) swab for SARS-CoV-2 RNA using polymerase chain reaction (PCR) performed at the UNC hospital clinical laboratory, willingness to self-isolate at home for a 14-day period, living with at least 1 HC who was also willing to participate, and living within driving distance (<1 hour) from the study site. Inclusion criteria for HCs of index patients included age >1 year and currently living in the same home as the index case without plans to live elsewhere during the 28-day study. All participants (or their parents/guardians) gave written, informed consent. Minors aged >7 years provided assent.

As shown in Supplementary Figure 1, all households were visited on day 1 by the study team. NP and nasal midturbinate (NMT) swabs were collected for SARS-CoV-2 PCR testing, and blood samples were collected for serology. All study participants received instruction on self-collection of NMT swabs and completed baseline questionnaires that included basic demographic and household information, abbreviated medical history, symptoms, recent travel history, and exposure to confirmed cases of coronavirus disease 2019 (COVID-19).

All participants received a daily symptom questionnaire via email until no symptoms were reported for 2 consecutive days. HCs who remained asymptomatic received the questionnaire for 21 days to monitor for new symptoms. On days 7, 14, and 21, a study staff member conducted home visits to collect self-collected NMT swabs. At the final study visit (day 28), participants were asked about COVID-19–related care-seeking and underwent serologic testing.

Laboratory Analyses

Details for all laboratory methods are found in the Supplementary Materials. Quantitative PCR testing for SARS-CoV-2 was performed using a Centers for Disease Control and Prevention reverse-transcription quantitative-PCR protocol authorized by the US Food and Drug Administration for emergency use, as previously described [11]. Serology was performed using enzyme-linked immunoassay (ELISA) that detects antibodies to the receptor binding domain of the spike protein with high sensitivity and specificity [12]. When ELISA results were not available (ie, in children who did not undergo venipuncture), results from a BioMedomics COVID-19 immunoglobulin (Ig) M/IgG Rapid Test [13–15] were used.

To determine the prevalence of the 614G variant, which predominated in North Carolina at the time of the study, in our study samples, we developed a real-time PCR assay to target a 107 bp region encompassing the D614G mutation in the S1 segment of the SARS-CoV-2 spike protein.

To determine whether secondary cases were acquired outside the household rather than due to household transmission, we performed high-density amplicon sequencing on NP/NMT swab samples from households with late secondary cases to assign SARS-CoV-2 clades and determine the genetic relatedness of viral isolates within and between households.

Statistical Analyses

We summarized baseline demographic characteristics and underlying conditions of index cases and HCs, as well as their household demographics. We determined if baseline NP SARS-CoV-2 viral loads were correlated within households by the intraclass correlation coefficient (ICC), which compares within vs between household variation.

We evaluated the secondary household attack rate among household members of persons who tested positive for SARS-CoV-2. For each household, if multiple participants were positive at enrollment, the index case was defined as the person with the earliest onset of infection based on reported onset of symptoms and known date(s) of PCR test positivity. If this was ambiguous, baseline antibody positivity was used as evidence of less recent infection. We calculated the SAR as the risk of incident infection among HCs, defined as the proportion of contacts who were PCR-negative at baseline but then developed SARS-CoV-2 infection during the 28-day study follow-up, confirmed by either PCR or antibody seroconversion. Those with evidence of prior infection (antibody-positive and PCR-negative) or HCs who reported the same COVID-19 exposure outside the household as the index case were excluded from the analysis. To avoid misclassification of asymptomatic infection, HCs who tested PCR-negative by weekly nasal swabs (days 7, 14, and 21) but without day 28 antibody results were also excluded. A 95% confidence interval (CI) for the SAR was calculated using a robust variance estimation for the intercept term in a logistic regression model to account for outcome dependence within a household.

Potential risk factors for secondary transmission within the household, including characteristics of index cases, households, and HCs, were examined. These risk factors and details of symptom severity evaluation are described in the Supplementary Materials. Statistical significance was tested using either the Fisher exact test or χ^2 test for categorical variables and the Mann-Whitney test for continuous variables. To determine if the association between index race-ethnicity and SAR was related to housing density or viral load (which

were correlated with race-ethnicity and/or transmission in unadjusted analyses), we calculated the odds ratio of the race-ethnicity–specific SAR, with the 95% CI estimated using the same robust variance estimation described above, then added the other risk factors as covariates in the logistic regression model to calculate adjusted odds ratios.

Analyses were performed using R 4.0.2 (R Core Team, Vienna, Austria). All hypothesis tests were 2-sided at a significance level of 0.05 with no adjustments for multiple comparisons.

The University of North Carolina Institutional Review Board approved the study.

RESULTS

Study Enrollment

Between April 2020 and October 2020, the UNC CO-HOST study enrolled 102 households across the central Piedmont Region of North Carolina (Supplementary Figure 2). After excluding participants who did not complete follow-up,
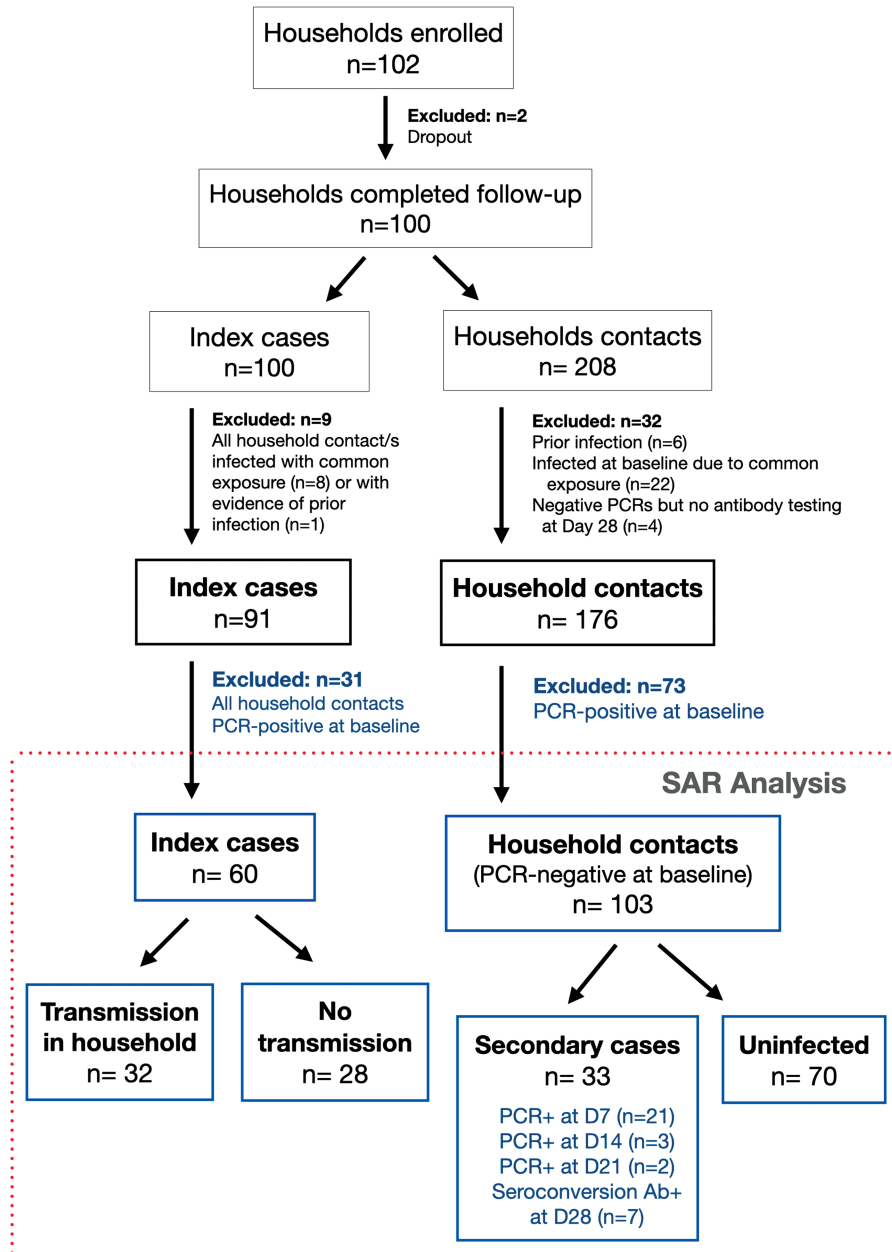
Figure 1. COVID-19 Household Transmission Study enrollment and SAR. Among 100 households that completed the 28-day follow-up, household contacts were excluded if they had evidence of prior infection (negative PCR and positive antibody test at enrollment), were possibly infected at the same time as the index case based on a common exposure event, or negative PCR testing could not be confirmed with a negative antibody test at day 28. Of the remaining 176 household contacts of 91 index cases, 41% (73) were already PCR-positive at baseline and thus excluded from the primary SAR analysis. During study follow-up, 33 incident severe acute respiratory syndrome coronavirus 2 cases were identified, yielding a SAR of 32% (33 of 103). Among the 33 secondary cases, 22 were identified by both PCR and seroconversion from day 1 to day 28, 4 were identified by PCR only, and 7 were identified based on seroconversion. Abbreviations: Ab+, antibody positive; D, day; PCR, polymerase chain reaction; SAR, secondary household attack rate.

had evidence of prior infection, or those who were possibly infected at the same time as the index case based on a common exposure, 91 households were included in the baseline analysis (Figure 1). Households were enrolled a median of 6 days (interquartile range [IQR], 4–7) after symptom onset of the designated index case, which was reassigned in 11 households. Baseline characteristics of the 91 index cases and 176 HCs are listed in Table 1. The median index case age was 37 years (IQR, 23–49), while the age of HCs ranged from 2 to 77 years with 34% aged <18 years. Overall, 44% of participants identified as other than White,

non-Hispanic race-ethnicity, and 33% of adults were obese (body mass index >30 kg/m^2; Table 1, other comorbidities in Supplementary Table 1).

Household Characteristics

The median size of households was 4 persons (Supplementary Table 2). However, in 38% of households, at least 1 household member chose not to participate. Households with a non-White index case were more likely to live in a home less than 2000 square feet in size (71% vs 43%, $P = .01$). This led to a higher "living density" for non-White households: 43% had more than

Table 1. Demographics of Study Participants

Characteristic	Index Cases, n (%)		Household Contacts, n (%)	
	91		176	
Male	43	(47)	87	(49)
Female	48	(53)	89	(51)
Race/Ethnicity				
White, non-Hispanic	52	(57)	96	(55)
Non-White	39	(43)	77	(44)
Black or African American	10	(11)	17	(9.7)
Hispanic/Latinx	26	(29)	58	(33)
Other, non-Hispanic	3	(3.3)	2	(1.1)
Unknown	0	(0.0)	3	(1.7)
Spanish speaking				
Yes	13	(14)	28	(16)
No	78	(86)	148	(84)
Age, years				
0–12	2	(2.2)	38	(22)
13–17	6	(6.6)	22	(13)
18–24	20	(22)	23	(13)
25–49	41	(45)	56	(32)
50–64	18	(20)	27	(15)
>65	4	(4.4)	10	(5.7)
Education (excluding those aged <18 years)				
Total responses for adults aged >18 years	80		113	
High school or lower	36	(45)	54	(48)
College degree	23	(29)	34	(30)
Graduate degree	21	(26)	25	(22)
Occupation (excluding those aged <18 years)				
Total responses for adults aged >18 years	83		116	
Education	3	(3.6)	6	(5.2)
Healthcare worker	11	(13)	9	(7.8)
Retail/Hospitality/Other frontline worker	19	(23)	22	(19)
Student	7	(8.4)	12	(10)
White collar worker	23	(28)	34	(29)
Other (trade and arts)	6	(7.2)	6	(5.2)
Not working outside the home	14	(17)	27	(23)
Comorbidities (excluding those aged <18 years)				
Diabetes	4	(4.8)	9	(7.8)
High blood pressure	12	(15)	24	(21)
Body mass index, kg/m^2				
>30	28	(34)	38	(33)
25–29.9	24	(29)	31	(27)
>30 and 1 or more comorbidity				
Adults aged >18 years (n = 83 index, 116 HC)	16	(19)	22	(19)
Adults aged >50 years (n = 22 index, 37 HC)	7	(32)	12	(32)

Abbreviation: HC, household contact.

3 household members living in a home with fewer than 6 rooms compared with 8% of White households ($P < .001$).

SARS-CoV-2 viral burden was correlated within households (Figure 2). When comparing the baseline NP viral load within vs between households, viral burden showed significant clustering within households (ICC = 0.45; 95%, CI 0.23–0.62; $P < .001$). Differences in viral load are not attributable to D614G mutation in the viral spike protein that has been associated with increased viral load and infectivity [16], as 90 of 92 (98%) of genotyped SARS-CoV-2 isolates contained the 614G mutant, while only 2 of 92 were wild type at this locus.

Secondary Attack Rate Among Household Contacts

The incident SAR among HCs was 32% (33 of 103; 95% CI, 22%–44%). Among 91 households, 73 of 176 (41%) HCs tested PCR-positive at baseline and were excluded from the primary SAR analysis (Figure 1, Supplementary Table 3 for demographics of baseline infected cases). Among the remaining 103 HCs of 51 index cases, 33 incident SARS-CoV-2 infections were observed during the 28-day study follow-up (SAR = 32%; 33 of 103). Of these 33 secondary cases, 22 were identified by both PCR and seroconversion from day 1 to day 28, 4 were identified by PCR only, and 7 were identified by seroconversion alone. The majority of secondary cases in the household experienced symptoms (27 of 33), while 18% (n = 6) remained asymptomatic.

Secondary household transmission occurred early, within the first week following enrollment for the majority of cases (n = 21 of 26 for those identified by PCR; Figure 1). Five cases were detected by PCR after the first week of enrollment, at day 14 or day 21. Four of these late secondary cases occurred in households of 5 or more (including 2 from the same household), which suggests the possibility of sequential transmission within the household. High-density amplicon sequencing of viral isolates from these late secondary cases and others in their household confirmed that 4 of 5 were indeed due to household transmission (1 isolate failed sequencing) and not community-acquired (Figure 3).

Risk Factors for Household Transmission

At the household level, 44% of households (40 of 91) had at least 1 infected household member at enrollment in addition to the index case, rising to 69% (63 of 91) of households 1 month later. Sixty households contained susceptible HCs at enrollment and were thus included in the risk factor analysis.

Secondary transmission in the household was associated with a higher NP viral load in index cases at enrollment. The median NP viral load among index cases was 1.4 $\log_{10}$ higher in households with secondary cases detected during the study vs those with no incident cases in the household ($P = .03$; Table 2). This difference persisted when the analysis was restricted to index cases who were still antibody-negative and thus more
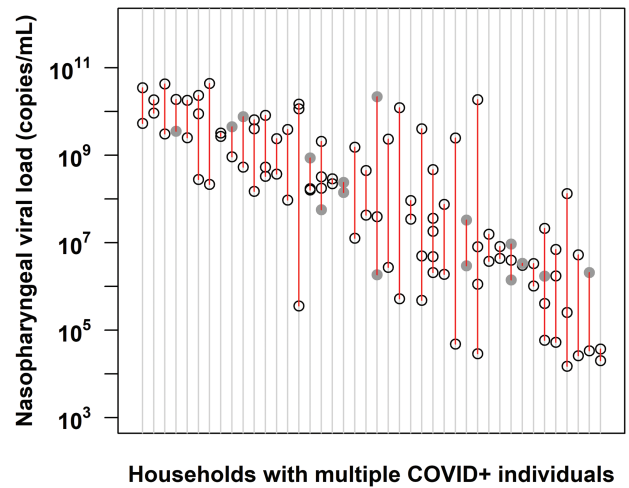

Figure 2. Severe acute respiratory syndrome coronavirus 2 viral burden is correlated within families. The viral load obtained at enrollment from nasopharyngeal (NP) swabs in households with multiple COVID-19–positive household members is shown (n = 42 households). Each vertical row in red depicts an individual household, with circles delineating the log viral load of each member within the household. Gray-shaded circles represent values derived from a nasal midturbinate swab if NP sampling was not performed. This was based on a linear regression equation generated from more than 100 study participants with positive viral load from both NP and nasal midturbinate swabs [11]. Households are depicted across the x-axis in order of decreasing viral load. Data drawn from 148 participants. The intraclass correlation coefficient = 0.45; 95% confidence interval, .23–.62; P value < .001. Abbreviation: COVID-19, coronavirus disease 2019.

recently infected [16, 18] (Figure 4). Symptom severity was not associated with household transmission, though secondary transmission did occur in households of the 4 index cases who were hospitalized (Table 2).

Households with non-White index cases were more likely to experience incident transmission in the household (Table 2), despite there being no difference in index case viral loads by race-ethnicity (median NP viral load for White vs non-White: 8.3 vs 8.3 $\log_{10}$ copies/mL). This corresponds to a SAR of 51% (95% CI, 33%–69%) in households with a non-White or Hispanic index case compared with 19% (95% CI, 10%–35%) in White, non-Hispanic households ($P = .008$). Higher living density, defined as more than 3 household members living in a home with fewer than 6 rooms, was associated with a greater odds of transmission (OR, 3.3; 95% CI, 1.02–10.9; $P = .047$; Table 3); a greater proportion of non-White/Hispanic households met this definition of high living density (42%, 11 of 26) compared with White, non-Hispanic households (12%, 4 of 34; $P = .01$). However, after adjusting for viral load and living density, Hispanic/non-White race-ethnicity remained associated with secondary household transmission (Table 4).

Among susceptible HCs, those who shared a bathroom with the index case were at higher risk of acquiring infection (Supplementary Table 4). Obesity and being female were also associated with a higher risk of incident infection, though these
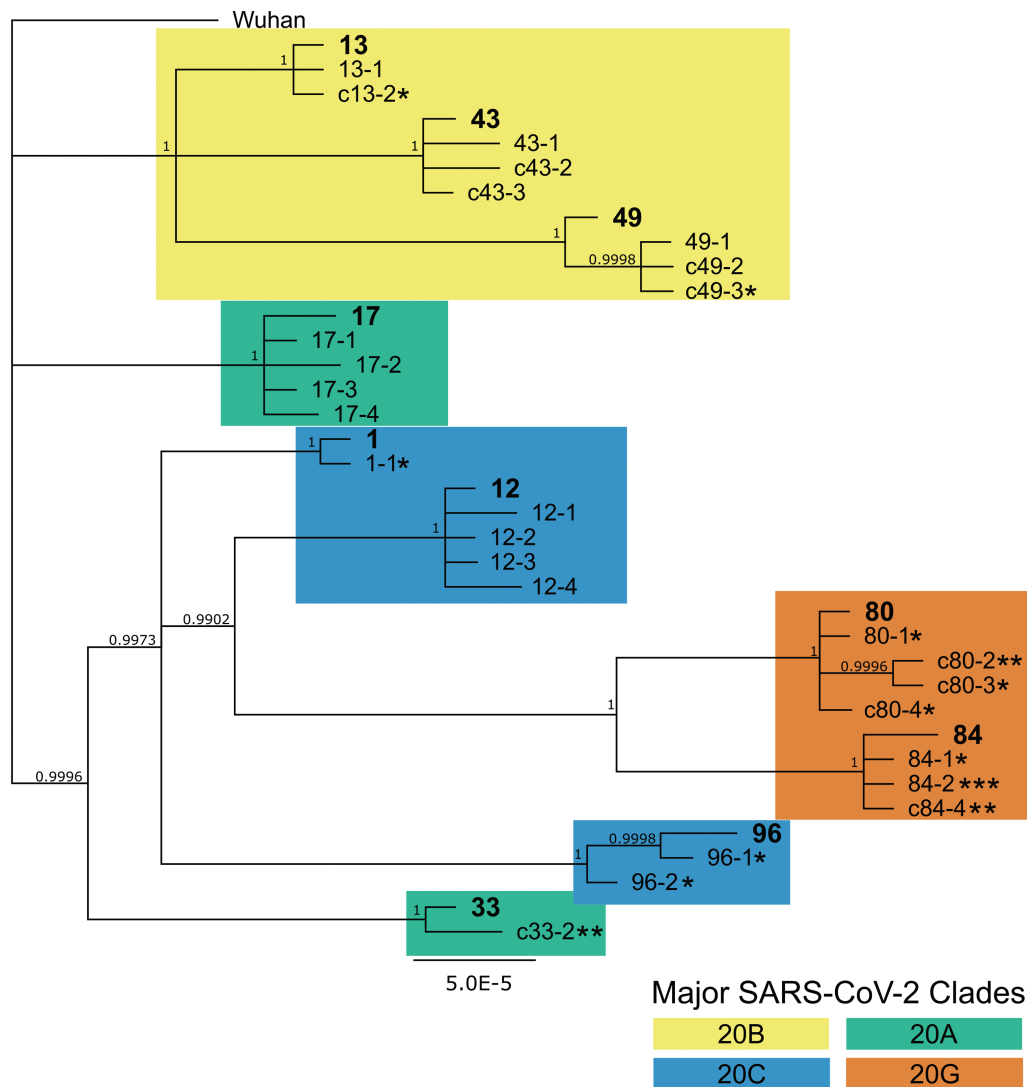
Figure 3. Bayesian phylogeny showing high relatedness within household infections, indicating household transmission. High-density amplicon sequencing was performed on all available viral isolates from 10 households with secondary infections to assess relatedness between infections. Whole-genome sequences were assembled according to the Wuhan reference genome, assigned to major clades, and then used for Bayesian phylogeny reconstruction. Index cases within each numbered household are in bold. Household contacts are numbered sequentially starting with the index case number, eg, X-1, X-2. Minors are indicated with the letter "c" prior to the case number. Each asterisk indicates 1 study week preceding a positive quantitative polymerase chain reaction (PCR) test, ie, * indicating a D7 positive test, ** indicating a D14 positive test, and *** indicating a D21 positive test. Household contacts without asterisks were PCR-positive at baseline. Abbreviation: SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

associations were not statistically significant. Though a slightly greater percentage of participants in households without secondary transmission reported wearing a mask at home in the week prior to enrollment (22% vs 13% for index cases and 30% vs 20% in HCs), these differences were not statistically significant.

DISCUSSION

Household transmission is one of the main drivers of the SARS-CoV-2 pandemic. By incorporating timely recruitment of index cases, prospective sampling to 21 days regardless of symptom status, and confirmatory viral sequencing in a subset of households, we show that household transmission occurs in a substantial proportion of COVID-19–positive households, with racial-ethnic disparities in secondary attack rates and higher risk of infection in more crowded households. Our data also suggest that those infected with a high viral load are not only more likely to transmit virus to other household members but they may seed other high viral load infections, putting the entire household at higher risk for more severe illness.

The incident SAR in this study was 32%, rising to 51% in minority households. While a meta-analysis of household transmission studies conducted in the first 6 months of the pandemic

Table 2. Potential Risk Factors for Severe Acute Respiratory Syndrome Coronavirus 2 Transmission From Index Cases

Index Cases	All Indexes, n (%)	Household Transmission, n (%)	No Transmission, n (%)	P Value
	60 (100)	32 (53)	28 (47)	—
Age, years				
<18	7 (12)	4 (13)	3 (11)	NS
18–50	45 (75)	25 (78)	20 (71)	
>50	8 (13)	3 (9)	5 (18)	
Sex				
Female	33 (55)	15 (47)	18 (64)	NS
Male	27 (45)	17 (53)	10 (36)	
Mask wearing at home prior to enrollment (missing n = 3)	10 (18)	4 (13)	6 (22)	NS
Race-Ethnicity				
White, non-Hispanic	34 (57)	13 (41)	21 (75)	.01[a]
Black or African American	7 (12)	5 (16)	2 (7)	
Other, non-Hispanic	4 (7)	4 (12)	0 (0)	
Hispanic/Latinx	15 (25)	10 (31)	5 (18)	
Symptom severity				
Mild	12 (21)	4 (14)	8 (29)	.07
Moderate/Severe	41 (72)	21 (72)	20 (71)	
Hospitalized	4 (7)	4 (14)	0 (0)	
Duration of symptoms at enrollment, median (IQR), days	5 (4–7)	5 (4–7)	6 (4–7)	NS
Nasopharyngeal viral load ($\log_{10}$ copies/mL) at enrollment (missing n = 6), median (IQR)	8.3 (5.9–9.5)	8.8 (7.3–10.1)	7.4 (5.3–8.7)	.03
Comorbidities for adults aged >18 years (missing n = 1 for diabetes, n = 3 for obesity)				
Diabetes	0 (0)	0 (0)	0 (0)	NS
Obesity, body mass index >30 kg/m^2	21 (42)	13 (50)	8 (33)	NS
Education for adults aged >18 years (missing n = 3)				
High school or less	21 (42)	13 (52)	8 (32)	NS
College degree	17 (34)	9 (36)	8 (32)	
Graduate degree	12 (24)	3 (12)	9 (36)	

P values only reported if ≤ .10, otherwise noted as NS.

Abbreviations: IQR, interquartile range; NS, not significant.

[a]Compares White, non-Hispanic vs all other categories.

(prior to circulation of new variants) found a much lower overall household SAR of 17% (95% CI, 14%–19%), it noted significant heterogeneity between studies (ranging from 4% to 45%) and combined both retrospective studies based on contact tracing data and prospective analyses [6]. As expected, prospective studies with increased frequency of testing regardless of symptom status generally show higher infection rates [7]. In the United States, a retrospective study in New York that included household testing offered regardless of symptom status reported a SAR of 38% [17], while 2 prospective studies following households in Utah and Wisconsin (58 households, SAR 29%) [18] and Tennessee and Wisconsin (101 households, SAR 53%) [10] also reported higher SARs. Altogether, these studies document high secondary household attack rates within US households.

To our knowledge, this is the first study to show increased transmission in non-White US households. Though they experience similar case fatality rates, African American/Black and Hispanic populations in the United States experience disproportionately higher rates of SARS-CoV-2 infection [19–21]. These racial disparities are likely due to differences in healthcare access and exposure risk that are driven by systemic societal inequities rather than individual biological or behavioral characteristics [22–25]. Our limited findings are consistent with this explanation. While the sample size precluded full investigation of drivers of increased transmission in minority households, we found that high living density/household crowding was more common in non-White households, while viral load and reported masking in the home did not differ by race-ethnicity.

We also found that SARS-CoV-2 viral burden was correlated within households. Increased viral load increases infectivity in vivo [26]. A study of 282 clusters in Spain showed an increased risk of transmission with shorter time to onset of symptoms among contacts as viral load of the index cases increased [9]. Since greater viral burden (high viral load or lower cycle threshold values by PCR) is associated with disease severity [27–30], our findings imply that when a person is hospitalized, others in the same household may be at a higher risk for a similar outcome than would be predicted based on their individual risk factors alone. An inoculum effect may underlie this finding [31] and also explain why secondary cases in households appear to be overdispersed, with either most or all members infected, or none at all [6, 32, 33].
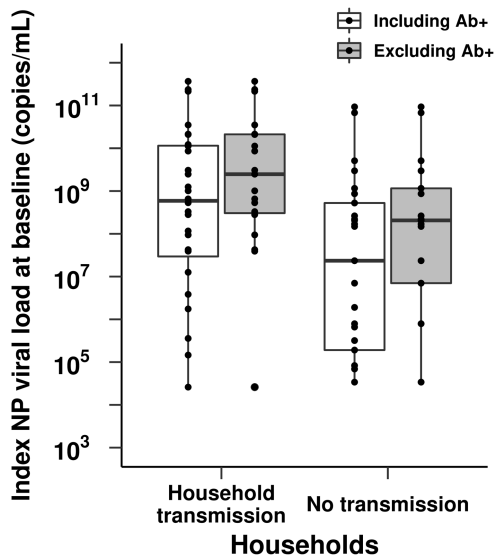
Figure 4. Association of index NP viral load and transmission in the household. Households with secondary cases were more likely to have index cases with high NP viral load compared with households without secondary transmission (median NP viral load log 8.8 vs 7.4 copies/mL, respectively; $P = .03$). Index cases who were not yet antibody-positive at enrollment, as a marker of more recent infection, are depicted to the right in gray. Abbreviations: Ab+, antibody positive; NP, nasopharyngeal.

This study has several limitations. First, although we enrolled most households within 24 to 48 hours of a positive SARS-CoV-2 test result, delays in testing meant that it was common for others in the household to be PCR-positive at enrollment. While 33 HCs met our end point as incident SARS-CoV-2 cases, 73 HCs were infected at study baseline and hence could not be categorized definitively as due to household transmission and were excluded from the analysis. Thus, we likely underestimated the true SAR, and the resultant small sample size was not sufficient to investigate all drivers of household transmission. We were similarly limited

in our ability to do adjusted analyses beyond a simplistic exploration of whether living density might account for the observed racial disparity in SAR. In the households with multiple infected household members at baseline, we cannot be certain that the designated index case was the source of infection for all infected household members. This may have affected our evaluation of index case risk factors associated with transmission.

Additionally, we were unable to adequately assess the effects of age, mask-wearing, and the presence of symptoms on transmission. We recruited adult index cases, and in 38% of households, at least 1 household member (most often young children) declined to participate. While mask use was queried, mask use prior to any COVID-19 diagnosis in the household was not specifically elicited. All index cases except 1 were tested because they were symptomatic.

In conclusion, SARS-CoV-2 transmits early and often among household members. While masking, physical distancing, and quarantining the whole household may reduce or prevent transmission beyond the household, these strategies are less effective within the household, especially in the setting of high viral load infections and crowded living spaces. Frequent point-of-care testing and post-exposure prophylaxis in those at risk for severe illness [34] and ultimately widespread and equitable distribution of vaccines [35] are needed to lessen the impact of COVID-19 within households and vulnerable communities.

Supplementary Data

Notes

Table 3. Potential Household-Level Risk Factors for Severe Acute Respiratory Syndrome Coronavirus 2 Transmission

Households	All Households, n (%)	Infected, n (%)	Uninfected, n (%)	P Value
	60 (100)	32 (53)	28 (47)	-
Household size, mean	3.8	4.2	3.4	.03
Living space (missing n = 3), sq ft				
<2000	29 (51)	18 (60)	11 (41)	NS
>2000	28 (49)	12 (40)	16 (59)	
Number of rooms[a]				
2 or fewer	5 (8)	2 (6)	3 (10)	.06
3–5	27 (45)	19 (59)	8 (29)	
6 or more	28 (47)	11 (34)	17 (61)	
More than 3 people and fewer than 6 rooms	15 (25)	13 (41)	2 (7)	.003
Home ownership (missing n = 1)				
Renting apartment	4 (7)	2 (7)	2 (7)	NS
Renting home	17 (29)	11 (36)	6 (21)	
Own home	38 (64)	18 (58)	20 (71)	

P values only reported if ≤ .10, otherwise noted as NS.

Abbreviation: NS, not significant.

[a]Number of rooms includes bedrooms, kitchen, and common rooms but not bathrooms or garage.

Table 4. Risk Factors for Severe Acute Respiratory Syndrome Coronavirus 2 Transmission in the Household

Index or Household Risk Factor	Susceptible House-hold Contacts	Incident Sec-ondary Cases	Secondary Household Attack Rate (95% CI)	Odds Ratio (95% CI)	Adjusted Odds Ratio (95% CI)
Non-White	41	21	51% (33–69)	4.4 (1.5–13.0)	4.8 (1.5–15.4)
White, non-Hispanic	62	12	19% (10–35)	-	
Higher index nasopharyngeal viral load[a]	-	-	-	3.5 (1.5–8.1)	3.6 (1.5–8.5)
High living density[b]	29	15	52% (27–75)	3.3 (1.02–10.9)	1.4 (.4–4.6)
Not high living density	74	18	24% (15–37)	-	

Adjusted odds ratio adjusts for viral load as a continuous variable and race-ethnicity and living density as dichotomous variables.

Abbreviation: CI, confidence interval.

[a]Odds ratio for 3 $\log_{10}$ increase in the index viral load (VL). For example, the odds of transmission in a household where the index VL is 1×10^9 copies/mL is 3.5 times greater than in a household where the index VL is 1×10^6 copies/mL.

[b]Defined as more than 3 people occupying fewer than 6 rooms.

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