

Increased Residential Clustering of COVID-19 Cases Associated With SARS-CoV-2 Variant of Concern B.1.1.7

Joe Flannagan, Katherine A. Twohig, Emma Carter, Dimple Y. Chudasama, Theresa Lamagni, and Gavin Dabrera

Background: The emergence of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) B.1.1.7 variant in England in 2020 and subsequent global spread emphasized the need to understand epidemiologic characteristics of SARS-CoV-2 variants. A diagnostic proxy for this variant, referred to as S-gene target failure, provided a rich dataset to assess transmissibility of the variant in an analysis of clustering in residential settings.

Methods: We used a pair-matched case-control study design to estimate odds of onward transmission within households with S-gene target failure index cases versus nontarget failure index cases. We defined cases as the index in a household cluster (clustered case) and controls as a case with no subsequent household cluster (sporadic). We matched clustered and sporadic cases one-to-one on specimen week, geography, and property type. We used conditional logistic regression, adjusting for age, sex, ethnicity, and symptom status, to assess odds of residential clustering.

Results: Our study population comprised 57,244 individuals with specimen dates from 23 November 2020 to 4 January 2021. Crude analysis yielded 54% increased odds (odds ratio [OR] = 1.5; 95% confidence interval [CI] = 1.5, 1.6) of residential clustering associated with S-gene target failure; the association remained in the fully adjusted model (OR = 1.6, 95% CI = 1.5, 1.6). Stratified analyses by region showed increased odds of residential clustering associated with target failure in all regions apart from the Southwest, where we observed lower precision. Similar adjusted odds ratios with precise confidence intervals remained in stratified analyses by property category.

Conclusion: We observed increased odds in all property types, consistent with greater transmissibility of the B.1.1.7 variant in this high-risk setting.

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From the National COVID-19 Epidemiology Cell, National Infection Service, Public Health England, Colindale, London, United Kingdom.

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The authors report no conflicts of interest.

Data are incorporated into the article and material contained within. Individual level data cannot be shared due to ethical/privacy reasons. Code availability: Custom code using Stata 15.

All data were collected within statutory approvals granted to Public Health England for infectious disease surveillance and control. Information was held securely and in accordance with the Data Protection Act 2018 and Caldicott guidelines.

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Correspondence: Joe Flannagan, National COVID-19 Epidemiology Cell, National Infection Service, Public Health England, Colindale, London, NW9 5EQ, United Kingdom. E-mail: feedback.c19epi@phe.gov.uk.

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SARS-CoV-2 variant B.1.1.7 was initially identified in England in December 2020 as part of the national collaboration to sequence SARS-CoV-2 cases; early cases were identified in the South East of England and London before later detection across England.¹ B.1.1.7, designated “alpha” by the World Health Organization, became the most frequently detected variant in England and spread internationally to several other countries.² Understanding the case severity and transmissibility of this variant are therefore key to the national and international pandemic response. While case severity has been studied in detail in the United Kingdom using national surveillance data, existing evidence for increased transmissibility is contingent on ecological studies.^{1,3,4} Analyses of household transmission are an important alternative to ecologic analyses, providing rich data for characterizing transmission in a high-risk setting for SARS-CoV-2.^{5,6} Analytical processes were established in England to identify residential clustering of SARS-CoV-2 by enhancing routine laboratory data.

Whole-genome sequencing of SARS-CoV-2 is available for only a subset of confirmed cases but S-gene target failure has become an internationally recognized proxy indicator for the B.1.1.7 variant.^{7,8} In brief, one of the mutations in the B.1.1.7 variant, consisting of a deletion of six nucleotides on the S-gene, causes a drop out in the S-gene target in specific diagnostic assays. These assays are used as part of the national community-based testing programme in England, to which samples for SARS-CoV-2 testing are directed from across the country. Since the week commencing 23 November 2020 up to the end of this study period, more than 90% of all samples that have a result for S-gene target status and were sequenced as B.1.1.7 has shown S-gene target failure. As of 21 December 2020, this number rose to 99% and, of wild-type sequenced cases where S-gene status was known, only 0.05% had target failure.⁸ Therefore, these circumstances allowed S-gene target failure data from the national testing programme to be used as a proxy for variant for epidemiologic analyses.

The objective of this analysis was to leverage the S-gene target failure data from the national community-based testing

programme to rapidly characterize transmissibility of the B.1.1.7 variant within households.

METHODS

In England, there is a statutory requirement to report all positive SARS-CoV-2 tests to Public Health England's (PHE) Second Generation Surveillance System, a laboratory reporting system.⁹ For a subset of laboratories (Lighthouse laboratories operating the TaqPath assay; Thermo Fisher Scientific, Waltham, MA) test results were accompanied by a result for S-gene target failure. These laboratories provided the majority of community testing and received specimens nationwide and so have very little geographical skew. During our study period, testing was available in person or via post for anyone who requested it, it was advised for individuals who had symptoms of COVID-19, and it was routine for certain groups such as those residing or working in long-term care facilities (regardless of symptom status). Positive SARS-CoV-2 patients were matched using residential address data in PHE's surveillance system to reference databases to derive Unique Property Reference Number and Basic Land and Property Unit.¹⁰ The surveillance system holds two addresses for each case from: National Health Service (NHS) summary care record (a minimum data set for all persons registered with the NHS) and laboratory information management system. The latter address was used preferentially as it should reflect the address at time of testing, as opposed to the centrally held NHS address which may not include recent or temporary address changes. We used Basic Land and Property Unit classes to classify property type according to usage classifications held by local authority planning departments. We defined household clusters as an index case followed by one or more laboratory confirmed SARS-CoV-2 cases at the same private dwelling (based on Unique Property Reference Number) within 14 days.

Data were extracted on 24 March 2021 and the study population consisted of SARS-CoV-2-positive cases who: had a first positive specimen date between 23 November 2020 and 4 January 2021 (when there was a high specificity of S-gene target failure testing for variant B.1.1.7); were tested in a Lighthouse laboratory using TaqPath assay; and resided in a private dwelling, that is, terraced (a house that shares both side walls with another house), semidetached (one sidewall is shared) or detached (no shared side walls) house, or a flat. Cases with indeterminate S-gene target failure results and cases involved in ongoing clusters (a cluster with at least one case within the 14 days before data extraction) were excluded. We defined an index case in a cluster as the case with the earliest specimen date. We excluded any households that had laboratory confirmed cases in the preceding 90 days under the assumption that this would independently reduce the number of susceptible persons in household and potential observed clustering effects. Coindex households were defined as more than one case having the same earliest positive specimen date and were also excluded. To retain as many index cases as possible in the analysis, we identified secondary cases from all

national laboratory confirmed case data irrespective of availability of S-gene target status.

We defined cases as the index in a residential cluster (a household with more than one case within a 14-day rolling window based on specimen dates) and controls as a case with no SARS-CoV-2 cases in the household in the 14 days after their specimen date; these will hereby be referred to as clustered cases and sporadic cases, respectively. We pair-matched clustered and sporadic cases based on specimen week, geography (lower-layer super output area), and property type, that is, terraced, semidetached or detached house, or flat. Lower-layer super output areas are small geographical areas of England predefined nationally based on residential population size; they have a minimum population of 1,000 people and a mean of 1,500 people.

We used a conditional logistic regression model to account for pair-matching within the data. An adjusted model was built using a forward stepwise approach that included age, sex, ethnicity, and symptom status. Age and sex were a priori confounders, whereas ethnicity and symptom status were included due to the results of our stepwise approach, no other variables were considered for inclusion. Due to slight geographic differences in testing coverage from the Lighthouse laboratories using the TaqPath assay, we conducted a fully adjusted analysis stratified by PHE center. PHE centers are large geographic areas of England based on health service boundaries; public health response or access to healthcare does not substantially differ between centers. Stratification by property type was also performed to evaluate any differences of the impact of S-gene target failure in different types of properties, as a measure of different household dynamics. In addition, a sensitivity analysis was undertaken to expand the definition of a coprimary case to anyone with a specimen date within 2 days of the earliest specimen date in the cluster and assess the effects on the results.

RESULTS

Of the 1,164,935 SARS-CoV-2 cases, from both community and other testing routes, during the study period, 42% ($n = 485,538$) were tested in a Lighthouse laboratory and so had a result for S-gene target failure. Of those, 238,805 met the eligibility criteria with the biggest reasons for exclusion being multiple index cases within a household. Pair-matching resulted in a study population of 57,244 individuals: 28,622 sporadic cases and 28,622 clustered cases (Table 1). Women and girls made up 53% (15,066) of sporadic cases and 51% (14,564) of clustered cases. The largest age groups for sporadic cases were 30–39 (22%; 6,207) followed by 40–49 (21%; 5,891) and for clustered cases 20–29 (21%; 6,075) and 30–39 (22%; 6,422; Table 1). More than a quarter of the study population were from London (30%), followed by the Southeast (16%). The most common residential setting was terraced households (39%), followed by semidetached (31%).

The matched case-control population included 37,055 (65%) individuals who tested positive for S-gene target

TABLE 1. Characteristics of Cases and Controls Included in the Pair-matched Case–Control Study

	Sporadic Cases		Clustered Cases		χ ²
	Countx	%	Count	%	
Total	28,622		28,622		
S-gene target failure					
True	17,678	62	19,377	68	220.88
False	10,944	38	9,245	32	
Household type					
Terraced	11,091	39	11,091	39	N/A
Semidetached	8,798	31	8,798	31	
Detached	4,636	16	4,636	16	
Flat	4,097	14	4,097	14	
Specimen week					
w/c 23 Nov 2020	1,570	5	1,570	5	N/A
w/c 30 Nov 2020	1,884	7	1,884	7	
w/c 7 Dec 2020	3,655	13	3,655	13	
w/c 14 Dec 2020	6,666	23	6,666	23	
w/c 21 Dec 2020	5,877	21	5,877	21	
w/c 28 Dec 2020	8,744	31	8,744	31	
w/c 4 Jan 2021	226	1	226	1	
PHE Center					
East Midlands	1,126	4	1,126	4	N/A
East of England	3,702	13	3,702	13	
London	8,657	30	8,657	30	
Northeast	1,551	5	1,551	5	
Northwest	3,547	12	3,547	12	
Southeast	4,689	16	4,689	16	
Southwest	372	1	372	1	
West Midlands	3,421	12	3,421	12	
Yorkshire and Humber	1,557	5	1,557	5	
Age					
<10	1,001	3	1,077	4	319.05
10–19	3,474	12	3,095	11	
20–29	6,075	21	5,152	18	
30–39	6,422	22	6,207	22	
40–49	4,889	17	5,891	21	
50–59	3,919	14	4,584	16	
60–69	1,863	7	1,967	7	
70+	979	3	649	2	
Sex					
Female	15,066	53	14,564	51	17.63
Male	13,556	47	14,058	49	
Ethnicity					
Asian	3,886	14	4,734	17	162.26
Black	1,530	5	1,114	4	
Mixed	663	2	597	2	
Other	1,275	4	1,339	5	
Unknown	353	1	296	1	
White	20,915	73	20,542	72	
Symptom status					
No symptoms	3,633	13	3,008	11	66.54
Symptomatic	24,989	87	25,614	89	

All individuals included are the index case in a household and have specimen dates from 23 November 2020 to 4 January 2021 (based on date of first positive SARS-CoV-2 test). Cases were defined as the index in a residential cluster and controls as a case with no SARS-CoV-2 cases in the household in the subsequent 14 days. w/c indicates week commencing.

failure. There was a higher proportion of target failure cases among clustered cases (68%), compared with sporadic cases (62%). Crude analysis yielded a 54% increased odds (OR = 1.5; 95% CI = 1.5, 1.6) of residential clustering associated with S-gene target failure, and this association remained after adjusting for age, sex, ethnicity, and symptom status (OR = 1.6 95% CI = 1.5, 1.6; Table 2).

We saw differences in household clustering between age and ethnic groups in the fully adjusted model with the greatest magnitude of effect seen for those of Asian ethnicity (Table 2). Where the index case was of Asian ethnicity there was a 39% (OR = 1.4, 95% CI = 1.3, 1.5) increased odds of residential clustering compared with instances where the index case was white. Where the index in a household was black, there was a reduced odds of subsequent household clustering (OR = 0.8, 95% CI = 0.7, 0.8). Furthermore, if the index in a household reported symptoms at the time of testing there was 24% (OR = 1.2, 95% CI = 1.2, 1.3) increased odds of residential clustering, when controlling for geography, week of specimen, household type, S-gene target failure status, sex, age, and ethnicity (Table 2).

Stratified analyses by region showed increased odds of residential clustering associated with S-gene target failure with precise confidence intervals in all regions apart

TABLE 2. Results of Univariable and Fully Adjusted Multivariable Conditional Logistic Regression Models

	Univariable	Multivariable
	Odds Ratio (95% CI)	Odds Ratio (95% CI)
SGTF		
No	1.0 (base)	1.0 (base)
Yes	1.5 (1.5–1.6)	1.6 (1.5–1.6)
Sex		
Female	1.0 (base)	1.0 (base)
Male	1.1 (1.0–1.1)	1.1 (1.0–1.1)
Age		
<10	1.1 (1.0–1.2)	1.1 (1.0–1.2)
10–19	0.9 (0.9–1.0)	0.9 (0.9–1.0)
20–29	0.9 (0.8–0.9)	0.9 (0.8–0.9)
30–39	1.0 (base)	1.0 (base)
40–49	1.3 (1.2–1.3)	1.3 (1.2–1.3)
50–59	1.2 (1.2–1.3)	1.2 (1.2–1.3)
60–69	1.1 (1.0–1.2)	1.1 (1.0–1.2)
70+	0.7 (0.6–0.8)	0.7 (0.6–0.8)
Ethnicity		
Asian	1.4 (1.3–1.5)	1.4 (1.3–1.5)
Black	0.8 (0.7–0.8)	0.8 (0.7–0.8)
Mixed	0.9 (0.8–1.0)	1.0 (0.9–1.1)
Other	1.1 (1.0–1.2)	1.1 (1.0–1.2)
Unknown	0.9 (0.8–1.1)	0.9 (0.8–1.1)
White	1.0 (base)	1.0 (base)
Symptom status		
No	1.0 (base)	1.0 (base)
Yes	1.3 (1.2–1.3)	1.2 (1.2–1.3)

from the Southwest, where we observed a lower precision. Similar adjusted odds ratios with precise confidence intervals remained in stratified analyses by property category. Results of the fully adjusted sensitivity analysis, which expanded the definition of coprimary cases to include anyone testing positive within 2 days of the index case and excluded households with these coprimary cases, included 35,266 individuals and showed the same association with precise confidence intervals (OR = 1.6, 95% CI = 1.5, 1.7; eTable; <http://links.lww.com/EDE/B925>).

DISCUSSION

In our analysis, we used a pair-matched case-control study design to estimate odds of onward transmission within households with S-gene target failure index cases versus nontarget failure index cases. We found that if the index in a household had the B.1.1.7 variant, there was 1.56 times the odds of residential clustering in the household within a 14-day period. This corroborates previous findings from ecologic studies suggesting that the B.1.1.7 variant is more transmissible than previously circulating SARS-CoV-2 but demonstrates even stronger evidence of increased transmissibility. Our alternative method at a household level and demonstration of the impact of increased transmissibility within household settings allowed for this increased strength of evidence. This improved understanding can help inform United Kingdom and international infection prevention and control policies and improve pandemic modeling. Stratified odds analysis showed that this relationship was similar in every household type and in every PHE region except in the Southwest, where the confidence intervals were not precise. This lack of precision is likely due to lower numbers of individuals in the Southwest eligible for inclusion and the Lighthouse Laboratories processing fewer tests from the region.

Other studies have shown that, in individuals with SARS-CoV-2, those who are symptomatic are more infectious than those who are asymptomatic, and our study shows this remains true for the B.1.1.7 variant plus estimates the difference in transmissibility based on symptom status for this variant.¹¹ However, it should be noted that our data will have likely misclassified some presymptomatic cases as asymptomatic as it relates to the absence of symptoms at time of testing only. This misclassification may have skewed the odds ratio toward one, thereby leading to an underestimate in the true effect size of symptom status on clustering. It is possible that both symptom status and transmission are associated with increased virulence of the case.

It has been shown that people of Asian ethnicity are at increased risk of infection compared with people of White ethnicity.¹² Our findings, showing that where the index case in a household was of Asian ethnicity there was an increased risk of residential clustering compared with index cases of White ethnicity, indicates this may in part be related to increased transmission within households.¹² This result possibly reflects

differing composition of households between ethnic groups such that people of Asian ethnicity are less likely to live alone and more likely to live in a multigenerational household.¹³ Alternatively it may reflect a difference in behaviors such as time spent in close proximity to other household members. Further research is needed to elucidate the reasons behind this finding.

Information on vaccination starting 10 January 2021 is publicly available; by that date around two million people had received at least one dose and 375,000 had received two doses in England out of a population of about 56 million.¹⁴ At this time, vaccination was focused on residents and staff of long-term care facilities, health and social care workers, those aged over 70 years, and clinically extremely vulnerable individuals.¹⁵ While we do not have vaccine status information for the individuals in our study population, given that our study only goes up to 4 January 2021 and was focused on residential dwellings, it is likely the vast majority will not have been vaccinated and, therefore, vaccine status, if included, would not have affected our results.

Pair-matching on household type, week of specimen collection, and lower-layer super output area helped ensure that clustered and sporadic cases did not vary on these key characteristics. Inclusion of specimen collection week and lower-layer super output area as matching factors helped account for the uneven distribution of B.1.1.7 variant cases across time and geography, as well as differential implementation of various social distancing measures at a regional level. Matching on household type helped limit variation between clustered and sporadic cases as the number and proximity of residents in a household varies by property type, which effects the potential for household transmission. Matching therefore helped to create comparable populations and other key confounding variables were controlled for through adjusting in our model. We therefore recommend application of this approach to rapidly study household transmission of new SARS-CoV-2 variants where enough variant cases are sequenced or where a reliable proxy is available for a large number of variant cases. This is also of benefit for other emerging communicable diseases where the surveillance infrastructure is available.

Strict inclusion and matching criteria and the use of S-gene target failure status resulted in the inclusion of only a portion of all cases in the study period. However, the inclusion criteria applied was required for this study design as it largely related to ascertaining the exposure (S-gene target failure status) and measuring the outcome (residential clustering). This study design still included over 50,000 cases which far exceeds the number that could have been included based on sequencing results and still provided enough power to detect differences between clustered and sporadic cases. While we showed that S-gene target failure status was a very good proxy during the study period and allowed for inclusion of a larger study population, it must be noted that, since this proxy cannot be entirely accurate, using it will have meant our results are

slightly less accurate than if only genetically sequenced cases were included.^{7,8} However, this is compensated by the larger scale of S-gene target failure testing than sequencing for cases during the study period, and therefore increased numbers of SARS-CoV-2 cases eligible for inclusion. Limiting the study population to private dwellings means our results are less generalizable to the whole English population as other residential settings, such as long-term care facilities, will likely have different transmission dynamics. The study period overlapped with the implementation of social distancing measures including stay at home orders for some regions of England; this means the observed effect will be more likely attributable to household transmission but does mean that observations were undertaken when people's behaviors were altered and so may be less applicable to other time periods.

We did not have access to household composition data that would allow us to identify single-person households but we have no reason to believe that being in a single-person household would differentiate by S-gene target failure status and therefore do not believe this to have biased the results of our analysis. Had we had access to household composition data, we could have investigated this further as well as undertaken further analyses such as calculating secondary attack rates. Furthermore, standard limitations of case-control studies such as the potential for bias and the lack of independence exist within this study as with all case-control studies. For this study, in particular, while the outcome is not common, it is also not rare and so the methodology of sampling controls from the tested population rather than the general population may mean the odds ratio may be somewhat higher than the risk ratio. The odds ratio provides utility in characterizing the trend in household transmission with the alpha variant but estimates provided may not be as optimal as a risk ratio. In this study, a stepwise approach was used to build the regression model which has some limitations such as the potential for biases like collider bias. However, at this stage of the pandemic, testing was widely accessible to members of the public with published guidance for all symptomatic persons to be tested; we therefore do not consider it is likely that having the B.1.1.7 variant and/or being part of a residential cluster had an association with the likelihood of being tested in a Lighthouse laboratory using the relevant assay.

The findings of this study would be complemented by an analysis of household data that contains denominators of all individuals in the household. This would allow for the calculation of secondary attack rates and assist with assessing additional drivers of transmission. Notwithstanding, this study still provides important evidence that expands our understanding of the transmission of the B.1.1.7 variant in the most common residence types in England.

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