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Transmission of SARS-CoV-2 by children to contacts in schools and households: a prospective cohort and environmental sampling study in London

Rebecca Cordery, Lucy Reeves, Jie Zhou, Aileen Rowan, Patricia Watber, Carolina Rosadas, Michael Crone, Marko Storch, Paul Freemont, Lucy Mosscrop, Alice Cowley, Gina Zelent, Kate Bisset, Holly Le Blond, Sadie Regmi, Christian Buckingham, Ramlah Junaideen, Nadia Abdulla, Joseph Eliahoo, Miranda Mindlin, Theresa Lamagni, Wendy Barclay, Graham P Taylor, Shiranee Sriskandan

Summary

Background Assessing transmission of SARS-CoV-2 by children in schools is of crucial importance to inform public health action. We assessed frequency of acquisition of SARS-CoV-2 by contacts of pupils with COVID-19 in schools and households, and quantified SARS-CoV-2 shedding into air and onto fomites in both settings.

Methods We did a prospective cohort and environmental sampling study in London, UK in eight schools. Schools reporting new cases of SARS-CoV-2 infection to local health protection teams were invited to take part if a child index case had been attending school in the 48 h before a positive SARS-CoV-2 PCR test. At the time of the study, PCR testing was available to symptomatic individuals only. Children aged 2–14 years (extended to <18 years in November, 2020) with a new nose or throat swab SARS-CoV-2 positive PCR from an accredited laboratory were included. Incidents involving exposure to at least one index pupil with COVID-19 were identified (the prevailing variants were original, α, and δ). Weekly PCR testing for SARS-CoV-2 was done on immediate classroom contacts (the so-called bubble), non-bubble school contacts, and household contacts of index pupils. Testing was supported by genome sequencing and on-surface and air samples from school and home environments.

Findings Between October, 2020, and July, 2021 from the eight schools included, secondary transmission of SARS-CoV-2 was not detected in 28 bubble contacts, representing ten bubble classes (participation rate 8·8% [IQR 4·6–15·3]). Across eight non-bubble classes, 3 (2%) of 62 pupils tested positive, but these were unrelated to the original index case (participation rate 22·5% [9·7–32·3]). All three were asymptomatic and tested positive in one setting on the same day. In contrast, secondary transmission to previously negative household contacts from infected index pupils was found in six (17%) of 35 household contacts rising to 13 (28%) of 47 household contacts when considering all potential infections in household contacts. Environmental contamination with SARS-CoV-2 was rare in schools: fomite SARS-CoV-2 was identified in four (2%) of 189 samples in bubble classrooms, two (2%) of 127 samples in non-bubble classrooms, and five (4%) of 130 samples in washrooms. This contrasted with fomites in households, where SARS-CoV-2 was identified in 60 (24%) of 248 bedroom samples, 66 (27%) of 241 communal room samples, and 21 (11%) 188 bathroom samples. Air sampling identified SARS-CoV-2 RNA in just one (2%) of 68 of school air samples, compared with 21 (25%) of 85 air samples taken in homes.

Interpretation There was no evidence of large-scale SARS-CoV-2 transmission in schools with precautions in place. Low levels of environmental contamination in schools are consistent with low transmission frequency and suggest adequate cleaning and ventilation in schools during the period of study. The high frequency of secondary transmission in households associated with evident viral shedding throughout the home suggests a need to improve advice to households with infection in children to prevent onward community spread. The data suggest that SARS-CoV-2 transmission from children in any setting is very likely to occur when precautions are reduced.

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London Health Protection Teams (R Cordery MD, A Cowley MSc, G Zelent MSc, K Bisset MSc, H Le Blond MSc, S Regmi MFPH, M Mindlin BMBCh), **COVID-19 EpiCell** (T Lamagni PhD), **United Kingdom Health Security Agency, London, UK; Department of Infectious Disease** (L Reeves MSc, J Zhou PhD, A Rowan PhD, P Watber MSc, C Rosadas PhD, M Crone MBBCh, M Storch PhD, Prof P Freemont PhD, L Mosscrop MSc, C Buckingham BSc, R Junaideen MBBCh, N Abdulla MBChB, Prof W Barclay PhD, Prof G P Taylor DSc, Prof S Sriskandan PhD), **Statistical Advisory Service** (J Eliahoo MSc), **Imperial College London, London, UK; The National Institute for Health and Care Research Health Protection Research Unit in Healthcare-associated infections and Antimicrobial Resistance, London, UK** (Prof S Sriskandan)

Correspondence to: Prof Shiranee Sriskandan, Department of Infectious Disease, Imperial College London, London W12 0NN, UK **s.sriskandan@imperial.ac.uk**

Introduction

The potential to control influenza outbreaks via school closures is well recognised.[1,2] Control of COVID-19 incidence and mortality was also associated with school closures,[3] although many other non-pharmaceutical interventions were instituted alongside, making the direct effects of school closure hard to determine.[4] Importantly, any benefits of school closures must be weighed against the unquestionable harms to children and to wider society, noting that COVID-19 poses a much lower risk to children than to adults in terms of illness severity.[5]

Initial studies suggested children are at reduced risk of testing positive for SARS-CoV-2 than adults,[6] but more

Research in context

Evidence before this study

Schools are believed to act as amplifiers of infectious disease epidemics but, in contrast to influenza, COVID-19 causes paucisymptomatic illness in children. Whether SARS-CoV-2-infected children pose a risk to others in schools is of importance; studies suggest that children might be as infectious as adults. At study inception we searched the literature for studies relating to COVID-19 and transmission by children. We found various rapid systematic reviews but no systematic contact-tracing studies in schools. Contemporary contract-tracing studies of influenza and scarlet fever describe secondary attack rates of approximately 25% in children exposed in classrooms. During this study, observational studies in other countries and one trial suggested that the SARS-CoV2 transmission risk to close contacts in schools might be as low as 1–3%.

Added value of this study

Our study forensically investigated onward transmission of SARS-CoV-2 to classroom bubble contacts of infected pupils who had been in school with symptomatic SARS-CoV-2 for at least one day, as well as contacts in the wider school and household contacts of the same cases. It did not rely on self-reported test results or passive surveillance, and used genome sequencing to support findings. Done at a time with many precautions in place, transmission to this small cohort of bubble contacts was not detected (ie, transmission was well below what has been reported for other respiratory infections).
This phenomenon was not because children were incapable of transmitting SARS-CoV-2. Indeed, children accounted for a secondary attack rate of at least 17% in the household setting. Notably, 40% of apparent transmission events in the household could be excluded using genome sequencing, which underlines the risk of inferring transmission from PCR or antigen results alone. The study identified an unexpected cluster of three asymptomatic SARS-CoV-2-positive pupils in the same class; although one pupil was undoubtedly infected and infectious to their household, the other pupils might have represented transient mucosal contamination while in the presence of a more infectious case. Our study found many challenges that could undermine future transmission research unless addressed. Importantly, the study provides the first systematic evaluation of environmental contamination with SARS-CoV-2 associated with infected children, showing that the household is a site of intense contamination compared with schools, both on surfaces and in the air, and highlighting the risks of whole household quarantine to household contacts if mitigation strategies are not advised or put in place.

Implications of all the available evidence

In the setting of the heightened precautions in place at the time of study, the risk of onward transmission to children from a single case of SARS-CoV-2 in schools is substantially less than other respiratory infections that have been studied in the prepandemic period, and markedly less than in the household. With such precautions in place, the added home quarantine of entire classes or bubbles appears unnecessary if the infected child is isolated. However, the heightened precautions put into place as a result of the COVID-19 pandemic abrogated many childhood respiratory infections. Therefore, although the results are reassuring, they cannot be extrapolated to settings where precautions are relaxed or more transmissible strains prevail. Conversely, the findings show that more stringent measures are required to reduce the risk of onward transmission from children in households highlighting the intense environmental and air contamination identified in households compared with schools, which act as a marker of viral shedding. These measures include own-room isolation, increased ventilation, and enhanced cleaning when handling personal items that might be more contaminated than other surfaces. To more rigorously evaluate transmission of infectious agents and increase participation, anonymised testing could be considered.

recent asymptomatic surveillance and household studies suggest children are as likely to acquire infection as adults.[7,8] Few studies have been done on whether children testing positive for SARS-CoV-2 pose an onward transmission risk in schools, although shedding of virus by children and adults reportedly does not differ.[9] Outbreaks can provide an opportunity to study onward transmission; however, uncertainty about timing and transmission direction complicate interpretation. One study[10] in English schools did identify children as a source of onward transmission in a small number of cases, but such outbreaks comprised just two cases on average. Another report described an outbreak at the time of school reopening, which coincided with reduced precautions, and was characterised by attack rates that varied greatly between year groups.[11]

Transmission of respiratory infection in schools is rarely quantified except during major outbreaks. Clinical attack rates of 20–30% are reported in schools affected by influenza A;[12] indeed, a secondary attack rate of 23% was observed in one year group after confirmation of one H1N1 case in a school.[13] The role of silent infection and onward transmission from such cases is not well studied and raises a concern that SARS-CoV-2 might be similarly transmissible.

Forward contact tracing offers an opportunity to search actively for secondary infections in a controlled manner. Before the COVID-19 pandemic and the institution of several interventions to limit the spread of SARS-CoV-2, we did a scarlet fever forward contact tracing study[14] in schools and observed outbreak strains spread to over one quarter of classroom contacts, despite treatment and isolation of index cases, alongside evidence of pathogen dispersal into air.[14] We adapted our protocol to investigate transmission of SARS-CoV-2 by children in schools and

households. The Transmission of Coronavirus-19 in Kids (TraCK) study aimed to assess the risk posed by a SARS-CoV-2-infected child who attends school via longitudinal sampling of the child, school and household contacts, and associated environments (surfaces and air), to evaluate and inform interventions to limit the spread of COVID-19.

Methods

Study design and participants

We did a prospective cohort and environmental sampling study in eight schools in London, UK. Schools reporting new cases of SARS-CoV-2 infection to local health protection teams were invited to take part if a child (index case) had been attending school in the 48 h before a positive SARS-CoV-2 PCR test. At the time of the study, PCR testing was available to symptomatic individuals only. Contextual information relating to prevailing regulations are in the appendix (p 8). Parents or guardians of notified cases were invited to allow their child and wider household to participate in the study. If the school was willing to support the study, parents or guardians of contacts were also invited to allow their child to participate in the study.

Children aged 2–14 years (extended to <18 years in November, 2020) with a new nose or throat swab SARS-CoV-2 positive PCR from an accredited laboratory were included. Detailed findings from cases will be reported elsewhere.

Bubble contacts were pupils who were required to isolate at home due to direct contact in class with a case that was symptomatic for at least 1 day before PCR confirmation. Non-bubble school contacts were pupils from a control class in the same school that was adjacent in terms of age group or proximity, selected by the headteacher, who were not required to isolate. Household contacts were adults and children of any age normally resident with the case, and required to isolate. Household contacts who had not already tested positive before the start of study sampling were considered naive. The study was approved by a research ethics committee (REC 18/LO/0025; IRAS 225006). Written, informed consent was obtained from all participants or parents or guardians, and written assent was obtained from participants younger than 18 years.

Procedures

Nasopharyngeal (throat followed by nose) swabs were taken by the research team from each participating contact as soon as possible (ideally <48 h) after case identification, and thereafter weekly for a total of four visits (three visits from Dec 1, 2020). In households, surface and air samples were obtained in three locations (index case's bedroom, communal room, and bathroom) at the first visit and thereafter weekly for a minimum of four visits (three visits from December 1, 2020); in some households, sampling was done more frequently in the first 2 weeks. In schools, surface and air samples were

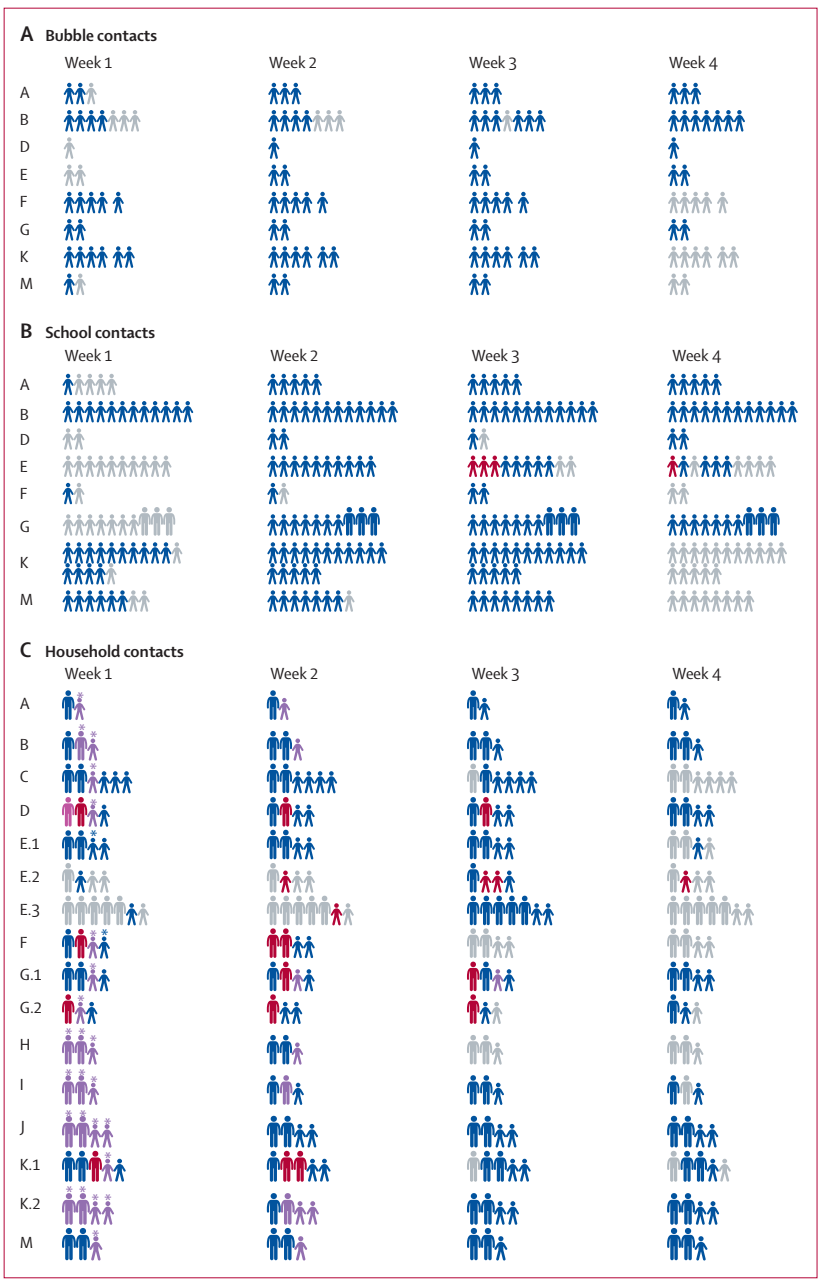

Figure 1: **Pictograms of individual bubble and non-bubble school and household contacts in each week of sampling**

(A) Bubble contacts (ie, 28 pupils). (B) Non-bubble school contacts (ie, 62 pupils and three staff). (C) Household contacts (ie, 63 adult and child household contacts) and index cases. Colour of icons indicates research swab test result in each week of study: blue icons indicate a negative swab result, red icons indicate SARS-CoV-2 newly detected during study, purple icons indicate SARS-CoV-2 detected and previously known to be positive, grey icons indicate individuals not swabbed in that week or not recruited yet. Two of three pupils identified incidentally are included in both panels B and C (ie, school and household contact pictograms); although these pupils were swabbed weekly, their associated households were recruited only after week 2. Within each panel, the figure position is consistent in each week and represents individual participants so can be compared between weeks 1–4. Individual settings are on different rows. Longitudinal sampling was limited to 3 weeks rather than 4 weeks for part of the study, hence some individuals were not swabbed in week 4. *26 participants reported to the study team as having tested positive before research swabbing (child index cases, adult household contacts, and child household contacts).

See Online for appendix

	Bubble size (excluding cases)	Number of cases bubble exposed to	Number of bubble participants	Number of bubble contacts testing PCR positive†				Bubble contact anti-NP total IgG	
				Week 1	Week 2	Week 3	Week 4	Positive on first sample	Positive on last sample
A	28	1	3	0/2	0/3	0/3	0/3	1/3	1/3
B	25	1	7	0/4	0/4	0/6	0/7	2/7	1/7
D*	40	1	1	..	0/1	0/1	0/1	0/1	0/1
E*	38	1	2	..	0/2	0/2	0/2	0/2	0/2
F†	46	2	5	0/5	0/5	0/5	ND	0/5	0/5
G‡	12	4	2	0/2	0/2	0/2	0/2	0/2	0/2
K§	139	11	6	0/6	0/6	0/6	ND	1/6	1/6
M	29	1	2	0/1	0/2	0/2	ND	0/2	0/2
Total number of children in bubbles	357	22	28	0/20	0/25	0/27	0/15	4/28	3/28

Data are n or n/N. A–M are different educational settings each of which had at least one case to which a bubble was exposed. Bubble participants all provided at least one swab. ..=not done. *Swabbing delayed until day 7 after case confirmed. †Includes two different bubbles exposed to one case each. One non-participant bubble contact tested positive in community test (included in household study). ‡Bubble exposed to two adult and two child cases. §Includes two different bubbles exposed to four cases and seven cases.

Table 1: **Transmission to contacts in bubble classrooms**

obtained weekly in three locations (bubble classroom, school contact classroom, and washrooms). Sampling started as soon as practically possible after case identification, ideally in less than 48 h (appendix pp 9–10).

Nasopharyngeal swabs were tested for SARS-CoV-2 envelope gene RNA and human ribonuclease P gene RNA by an accredited, quantitative RT-PCR followed by genome sequencing (appendix p 9–10).[15] Results were reported in real time to participants and subject to statutory reporting and associated regulations. Environmental samples were tested by a research laboratory[16] (appendix p 10).

To provide evidence of previous SARS-CoV-2 exposure, oral fluid samples were collected from contacts on each sampling occasion (appendix p 11) then tested for total IgG against SARS-CoV-2 nucleoprotein by the reference laboratory.[17]

Statistical analysis

The study was pragmatic and enrolled as many bubble contacts as possible within the school year. The study aimed to recruit 40 bubble contacts; a sample size of 28 was sufficient to identify a difference in infection rate of 3% versus 25% (the frequency identified in an earlier contact tracing study) with 94·49% power (appendix p 9) Fisher's exact test was used to compare proportions of household contacts with positive serology results (Stata version 15). Human target RNAs were compared using Mann Whitney U test (GraphPad Prism version 9.0). This study is registered at ISRCTN, 13773960.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

The study commenced Oct 9, 2020, and recruitment ended July 18, 2021. Eight schools participated (settings A, B, D, E, F, G, K, and M), of which five were primary, two secondary, and one was a special educational needs school. Participants comprised 28 bubble contacts, 62 non-bubble school pupil contacts, and 47 household contacts. In the course of the study, 428 combined nose and throat swabs and oral fluid samples were obtained from contacts of index cases. Environmental sampling from schools and households comprised 1620 surface samples, of which 446 were from schools, and 218 air samples, of which 68 were from schools.

Bubble contacts were recruited from ten bubbles in eight schools. The participation rate among bubble contacts was low (median 8·8%; IQR 4·6–15·3%), being highest in primary schools and lowest in special educational needs and secondary schools. In total, 28 bubble contacts were followed up weekly, with the first swab taken within a median of 6 days (IQR 5·25–10·0) of exposure in class to at least one index case. Onward transmission of SARS-CoV-2 to the 28 participating bubble contacts was not detected over the sampling period (figure 1A, table 1). Only four (14%) of 28 participating bubble contacts had serological evidence of previous exposure to SARS-CoV-2 and there was no evidence of seroconversion between first and last sample. One bubble contact, who had not consented to take part in the study as a contact, developed a fever and reported a positive community test 6 days after exposure to the index in their class. The child was recruited as a case along with their household, but all of the child's study sample PCRs were negative; seroconversion occurred at 4 weeks, but the timing of infection was unclear.

62 pupils and three staff (non-bubble school contacts) were recruited from the same eight schools. School contacts' participation rates were higher than bubble contacts', median 22·5% (IQR 9·7–32·3%; table 2). In seven (88%) of eight schools, no school contacts were found to be infected with SARS-CoV-2. Of those tested, 13 (20%) of 65 had antibodies indicating previous SARS-CoV-2 infection, with no serological change to indicate new SARS-CoV-2 exposure during the sampling period. In setting E, a secondary school, consent to sample contacts was delayed until 11 days after symptom onset in the index case. All non-bubble school contacts tested negative in the first round of sampling but in the second round, unexpectedly, SARS-CoV-2 was detected in three (30%) of ten pupils (figure 1B, table 2). All three were asymptomatic; in one, the viral load increased from 293 240 E-gene copies per swab to 5 999 560 copies per swab 3 days later and onward transmission to a sibling who shared their bedroom (84 040 E-gene copies per swab) was observed. The other two asymptomatic school contacts had very low viral loads; the first, who had tested negative 7 days earlier, had 280 E-gene copies per swab but further testing was declined. The second had 560 E-gene copies per swab; samples 7 days earlier and 4 days later were PCR-negative, and anti-SARS-CoV-2 antibodies were already present in oral fluid in their first samples. We felt it possible that these low viral levels did not represent true infections, but transient mucosal contamination while in the company of another pupil with active high-titre infection. The original index case in setting E was identified after community PCR testing; by the time the school contacts were first sampled, the index case already had a negative PCR test but was still quarantined. We inferred that infection in these non-bubble school contacts was not linked directly to the original index case.

16 households took part, comprising 47 household contacts and 16 index cases who were each an index or co-primary case to a bubble class. The number of households exceeded the number of bubbles that participated because, in four settings, household contacts agreed to take part, but the relevant schools withdrew. In another setting, the school agreed to take part, but a household declined; a separate case from the same class was identified by community testing however, and their household was recruited. For setting E, household contacts of two of the three newly identified asymptomatic non-bubble school contact infections were included. All of the index cases were symptomatic except these two.

Households were sampled a median of 3 days (IQR 3–4) after onset of symptoms in the index case. Of the household contacts, three children and nine adults were already reported to be infected at the start of sampling. Initial analysis focussed on 35 household contacts who were considered naive (ie, were not reported to already be infected at the start of sampling, of which 11 (31%) of 35 were children; table 3)

Over the sampling period, ten new infections were detected among naive household contacts in nine adults and one child (table 3, figure 1C). In two households, genome sequencing revealed that the index case was, however, unrelated to the four new adult household contact infections (two adults per household), hence these represented secondary introduction from the community (table 3; appendix p 5). In all other households, genome sequencing was consistent with household transmission (appendix p 5). Transmission by children therefore resulted in infection of six (17%) of 35 previously-negative household contacts. Only one (3%) of 35 oral fluid samples suggested previous COVID-19 among naive household contacts at the start of sampling, although this rose to six (23%) of 26 by the end of sampling (p=0·035). Just six

	Group size	Number of cases at start of study in school	Number of participants	Number of participants testing PCR positive or number swabbed				Participant anti-nucleoprotein total IgG	
				Week 1	Week 2	Week 3	Week 4	Positive on first sample	Positive on last sample
A	30	1	5	0/1	0/5	0/5	0/5	3/5	1/5
B	22	1	12	0/12	0/12	0/12	0/12	1/12	1/12
D*	27	1	2	..	0/2	0/1	0/2	0/2	1/2
E*	30	1	10	..	0/10	3/8	1/5	2/10	1/8
F	11	2	2	0/1	0/1	0/2	ND	0/2	0/1
G†	24	4	7	..	0/7	0/7	0/7	1/7	2/7
G.ad			3	..	0/3	0/3	0/3	0/3	0/3
K	306	26	16	0/14	0/16	0/16	ND	3/16	4/16
M	30	1	8	0/6	0/7	0/8	ND	3/8	3/7
Total	480	37	65	0/34	0/63	3/62	1/34	13/65	13/61

Data are n or n/N. A–M are different educational settings each of which had at least one case to which a bubble was exposed. Non-bubble school contacts all provided at least one swab. ..=not done. G.ad=adults. *Swabbing of school contacts started one week after initial case. †Four cases in school included two children and two adults. Non-bubble contacts, seven children and three adults.

Table 2: **Transmission to non-bubble school contacts**

	Household size*	Number of child cases at start of swabbing†	Number of adult cases at start of swabbing	Number of naive contacts	Number of contacts testing positive				Naive contact crevicular fluid anti-nucleoprotein total IgG	
					Week 1	Week 2	Week 3	Week 4	Positive on first sample	Positive on last sample
A	2	1	0	1	0/1	0/1	0/1	0/1	0/1	0/1
B	3	1	1	1	0/1	0/1	0/1	0/1	0/1	0/1
C‡	6	1	0	5	0/5	0/5	0/4	..	0/5	0/5
D§¶	4	1	0	3	2/3	1/3	1/3	0/3	0/3	1/3
E.1§	4	1	0	3	0/3	0/3	0/3	..	0/3	0/3
E.2‖	4	1	0	3	1/3	..	..	..	0/3	..
E.3§	7	1	0	6	0/6	..	..	..	0/6	..
F	4	2	0	2	1/2	2/2	..	..	0/2	0/2
G.1§	4	1	0	3	0/3	1/3	1/3	0/3	0/3	2/3
G.2§	3	1	0	2	1/2	1/2	1/1	0/1	1/2	2/2
H	3	1	2	0	0/0	0/0	ND	..	0/0	0/0
I	3	1	2	0	0/0	0/0	0/0	..	0/0	0/0
J	4	2	2	0	0/0	0/0	0/0	..	0/0	0/0
K.1§¶	5	1	0	4	1/4	2/4	0/3	0/2	0/4	1/4
K.2	4	2	2	0	0/0	0/0	0/0	0/0	0/0	0/0
M	3	1	0	2	0/2	0/2	0/2	..	0/2	0/2
Total	63	19	9	35	6/35	7/26	3/21	0/11	*1/35	*6/26

Data are n or n/N. A–M and the associated numbers are individual households associated with each educational setting. Where more than one household was associated with a setting, individual households are indicated by an additional number (E.1, E.2, G.1, G.2, K.1, K.2. ..=not done due to intervening holiday or withdrawal from study. *Includes index child case: each household had 100% participation rate at time of consent. †Includes index child case plus any other child already identified as infected. ‡Naive household contacts include three children. §Naive household contacts include one child. ¶Household contacts with different genomic sequences to index case are indicated. ‖Naive household contacts include two children.

Table 3: **Transmission events in participating household contacts in each setting**

household contacts had been partially or fully vaccinated at the time of study; these were two adults each in settings K.1, K.2, and M.

12 household contacts who were reported to be already infected before study team arrival were also sampled sequentially, but were not included in the initial analysis due to uncertainty of transmission direction. To gain greater insight into the potential frequency of secondary attack, symptom and testing history were reviewed. Three child household contacts were reported to be positive before research sampling; on the basis of symptom onset and date of testing, it was deduced that these child household contacts had been secondarily infected by the index case in the home. Nine adults (from five households) were also reported to be positive before research sampling. For five (45%) of nine adults, test results or symptoms predated that of the index case, suggesting that the child was not the source of infection within the household. For four (36%) of nine adults, their infection was believed to arise from the index case. Taking these additional cases into consideration, the 16 index case children resulted in a maximum of 13 new cases in 47 household contacts (28% secondary attack rate).

School environmental sampling commenced a median of 7 days (IQR 4·5–9·5) after onset of symptoms in the index pupil. Surface sampling identified SARS-CoV-2 in only four (2%) of 189 samples from bubble classrooms, two (2%) of 127 samples in school contact classrooms, and five (4%) of 130 samples from school bathrooms (figure 2A–C). Where detected, viral copy numbers were at the lower limits of detection except the edge of an index child's chair in a bubble classroom that had more than 10^4 E-gene copies per swab in week 1, 6 days after the bubble class was sent home and before deep cleaning. The same items were sampled in each location on a weekly basis (appendix pp 2–3); no item became positive on subsequent sampling. Air sampling was done weekly in the same locations, as soon as possible after children vacated those rooms, except when availability of equipment components limited this. Only one (2%) of 68 air samples was positive. This result was at the limit of detection, in week 2 in a school that had experienced numerous staff infections, in a control (non-bubble) classroom not known to have any pupil COVID-19 cases.

We considered the possibility that air samples might only be positive when a room is in active use. To provide context, we did environmental sampling in a university building (appendix p 4). We identified SARS-CoV-2 in three (30%) of ten surface samples from a small office 4 days after use by a staff member with confirmed COVID-19, but not in any other office or location in the same building, or on follow-up (none out of the 96 samples). We also detected low levels of SARS-CoV-2 in an air sample from the same office 4 days after use; all air

samples were negative when retested 2 weeks later (appendix p 4).

Environmental sampling from households commenced a median of 3 days (IQR 3·0–4·3) after onset of symptoms in the index case. In contrast to schools, overall 262 (22%) of 1174 surface samples were contaminated with SARS-CoV-2 in 16 households. Focusing on samples taken on the first visit and thereafter weekly, there was a trend to declining virus detection over time (figure 2 D–F). The most frequent surface contamination was identified in index case bedrooms, where 60 (24%) of 248 samples tested positive, and communal rooms, where 66 (27%) of 241 samples tested positive. In bathrooms, 21 (11%) of 188 surface samples tested positive, consistent with increased bathroom surface cleaning. Personal items relating to the child, such as pillows, and digital equipment, such as mobile phones, remote controls, and digital toys, were more persistently positive over the sampling period whereas other sample types became negative within 2–3 weeks, including pet fur (appendix p 6). Surface human RNA levels were higher in households than schools (appendix p 7).

Overall, 42 (28%) of 150 air samples obtained in households were contaminated with SARS-CoV-2. Focusing on samples taken on the first visit and thereafter weekly, air samples were positive in four (18%) of 22 samples taken in the index child's bedroom, 13 (31%) of 42 samples in the communal room, and four (19%) of 21 samples in the bathroom (figure 2D–F). Virus levels in the air were highest in the room with an infected child and infected adults. The index child and household contacts were always in the communal (living) room at the time of sampling except in three settings where the index child was only in the bedroom during sampling and one setting where the child moved between rooms. There was no apparent association between the type of household (apartment or house) and air contamination. Air samples in households and schools did not differ significantly with regard to human RNA (appendix p 7).

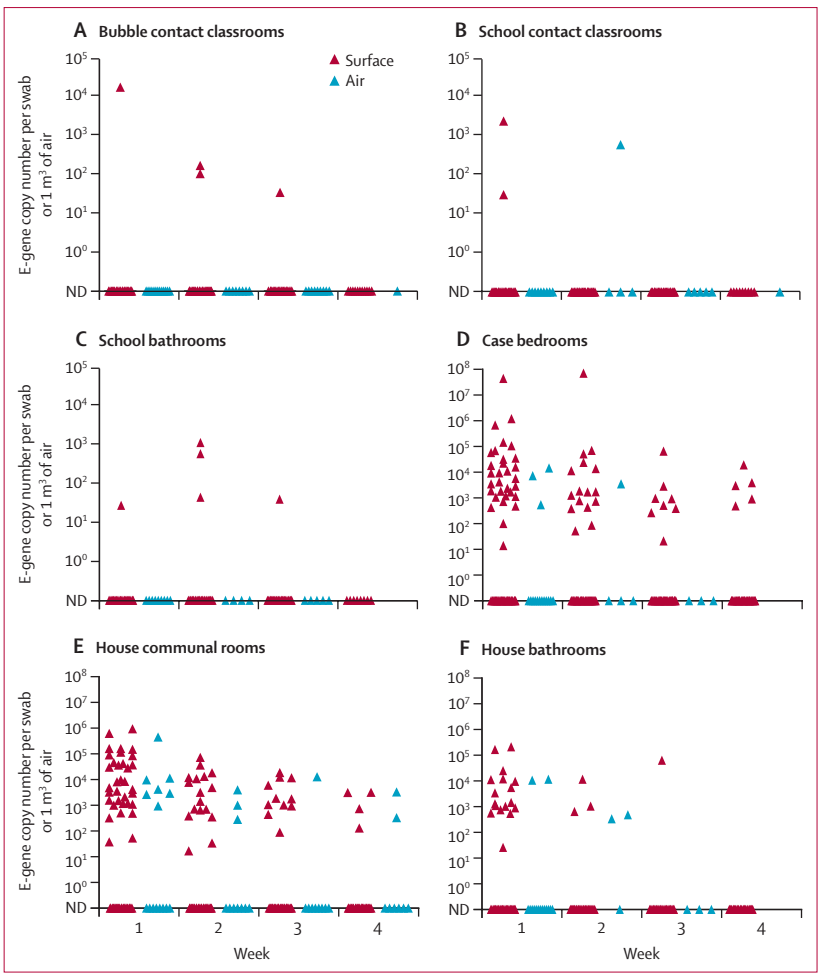

Figure 2: **Environmental contamination with SARS-CoV-2 in schools and households**
A–C are schools. D–F are households. (A) Bubble contact classroom. (B) School contact classroom. (C) School bathroom used by bubble. (D) Child's bedroom. (E) Communal room. (F) Bathroom used by child. Samples obtained at start of sampling and thereafter weekly are shown. Red indicates surface samples and blue indicates air samples. Surface and air samples were obtained from the same items and locations weekly in each school and households. Data shown as absolute E-gene copy number and represent samples from eight schools (one special educational needs and disabilities school, two secondary schools, and five primary schools) and 16 households. Note y-axis range differs between schools and households. ND=not detected.

Discussion

This study, done during a time of enhanced precautions, did not actively detect transmission from index pupils to bubble contacts, or to other pupils in school who were not close contacts, although the low participation rates might mean that infrequent transmission events were missed. The study was adequately powered to rule out the secondary attack rates reported in pupils exposed to influenza in school; to that extent we can be confident that the interventions in place reduced that risk. Our findings in classrooms contrasted with a secondary attack rate of 17% in household contacts of the same index cases, albeit that exposure would have been greater in households. When household contacts who had already been tested were included in our analysis, the secondary attack rate in households with a child index case was 28%.

One apparent transmission incident in a control class, which was not isolating, involved three asymptomatic pupils, not linked to the original index case in that school. One of these pupils had a high viral load, leading to infection of a secondary case in their household and, we believe, accounted for transient carriage, as previously reported,[18] rather than true infection in two other pupils, as E-gene copy numbers were similar to environmental samples and detected only once.

Environmental surface and air sampling were done to understand mechanisms of transmission and as a surrogate for measuring viral shedding by all those using the rooms that were sampled. This showed little or no contamination in schools, including of surfaces touched frequently by children, providing reassurance regarding the school environment during a period of enhanced

vigilance, underlined by a difference in human RNA detection between surfaces in households and schools. This contrasted with the repeated identification of virus on household items frequently touched by children, and in the air around the home, particularly where the child was present. This is perhaps not surprising because the dimensions of domestic rooms are approximately four times smaller than classrooms and provides some insight into the risks of virus acquisition in the two settings. The detailed environmental sampling identified digital equipment and personal items as potential fomite vectors or as metrics of infectivity. The high proportion of air samples that were positive in the households compared with school underlines the greater risks associated with smaller rooms and is a reminder that air might remain positive for SARS-CoV-2 for some time if not well ventilated. Control sampling in a different educational setting showed low levels of SARS-CoV-2 RNA in the air 4 days after an office was used by a staff member with COVID-19. We considered the possibility that air sampling in schools was negative for SARS-CoV-2 because the children were not present in the room; however, control human RNA was no different in the air between schools and households. The low or absent levels of SARS-CoV-2 RNA in the bubble classroom provides reassurance that there was no ongoing infection in members of the bubble who returned to school by weeks 2–3.

Our findings are consistent with other studies showing transmission to bubble contacts was uncommon, with 1–2% co-primary or secondary infections identified where larger numbers were sampled.[19–22] Our findings are also consistent with the approximately 1·5% asymptomatic infection rate reported in a trial[23] of daily lateral-flow testing. The infrequency of transmission to other pupils contrasts with transmission frequency of other respiratory infections in schools, including group A streptococcus and influenza.[12–14] The result presumably reflects the multiple interventions in place during the pandemic period or the heterogeneity of infection in COVID-19, where most transmission is caused by a minority of infections.[24,25]

Our study prospectively examined transmission from the same children to contacts in both schools and households; the secondary attack rate in households was higher than expected, and was in contrast to that seen in schools. Our findings are, however, consistent with other household studies[26–28] that show age does not appreciably affect transmissibility, although younger children might serve as a more frequent source of infection than older children. Quarantine for household contacts, in place throughout our study, might increase exposure of household members to index cases unless mitigated by protective measures, noting that household size has been associated with urban caseloads.[29] It was notable that in all households with no onward transmission to naive contacts, householders had ensured that the affected child was isolated from others, without sharing a bedroom, while still affording care and supervision.

Our study adopted a forensic approach to contact tracing, so as not to miss infections that were cleared early or those arising from ongoing transmission in the bubble. The study relied on identification of index cases via community PCR testing, as such index cases in this study were symptomatic. We took combined nose and throat swabs to increase opportunity for virus detection and used human ribonuclease P as a sample control to ensure that negative results could be trusted. Furthermore, almost all swabs were taken by the study team, although a small number of contacts were permitted to take swabs themselves if supervised by the study team. Genome sequencing identified transmission events that were genuine while also refuting others, highlighting a risk of estimating transmission rates relying on PCR results alone: 40% of inferred household transmissions were eventually excluded. The study was designed to investigate bubble sizes of approximately 10–15 pupils, but interpretation of bubble changed over time, and by autumn 2020, bubble sizes routinely included 30–200 primary-school-aged and secondary-school-aged pupils.[30] The environmental sampling is the most detailed to date in both schools and households, revealing prolonged positivity particularly among items belonging to the child, and is indicative of ongoing viral shedding in the household setting.

There are several limitations to our study. Firstly, the study was done at a time of heightened and constantly changing interventions, in particular physical distancing in schools, reduced class sizes, and closures. Transmission in schools might well alter when interventions relax, and when more transmissible variants arise, as occurred in late 2021.[31] Secondly, participation rates of contacts were very low, compared with participation rates of higher than 40% in our earlier contact tracing study[14] also involving throat swabs, lowering confidence in ruling out any bubble transmission. Low participation is reported in other studies in England that detect asymptomatic SARS-CoV-2 infections,[32] contrasting with high participation in Nordic countries.[21] Deterrents to participation reported anecdotally were the legal requirement to notify new infections, quarantine effect on participants, study team making home visits, low risk in children, and inclusion of older pupils. Participation by school contacts was consistently higher than bubble contacts, underlining the challenge of recruiting bubble contacts sent home to isolate because schools use disparate methods to contact parents. The greatest barrier to participation was the recognition that newly identified infections would result in quarantine for entire households or classes, such that participation was actively discouraged by some groups, in contrast to predicted responses at study inception. Finally, although our study benefited from the objective starting point of positive index cases who attend school,

there is a risk of bias in reliance on voluntary participation that is hard to correct for in a small study. School participation was influenced by staffing levels, data protection issues, and parental and governing body concerns; this might have biased the type of schools that took part. The requirement for consent from parents and assent from children who were isolating might have excluded certain groups at greater risk of infection. Representation from a larger number of participants would require expansive resources and, with the prevailing restrictions, might not necessarily lead to more inclusive recruitment. Strategies to enhance capacity for health research in community settings such as schools are much needed.

Notwithstanding low participation, the study confirmed that the interventions put into place in the 2020–21 school year prior to spread of the omicron variant reduced SARS-CoV-2 secondary attack rates that were previously reported for influenza and scarlet fever. These interventions likely rendered additional bubble quarantine unnecessary. We conclude that future forensic-level research on transmission of infectious agents might provide more meaningful information if the results are unlinked to identifiable data, or any form of notification or requirement to isolate (ie, without real-time reporting, and conducted within schools). With reduced interventions and the advent of new variants, it might be prudent to evaluate schools-based transmission in such a silent study.

Contributors

SS and RC contributed to study conceptualisation. RC supervised the study site. GPT, WB, PF, and SS supervised the laboratory. SS, RC, GPT, WB, TL, and MM contributed to study methods. LR, JZ, AR, PW, CR, MC, MS, LM, AC, GZ, KB, HLB, SR, CB, RJ, and NA contributed to data collection. JZ and LR contributed to data visualisation. LR, GPT, and JZ contributed to data validation. JZ and JE contributed to data analysis. SS, WB, GPT, RC, TL, and MM contributed to funding acquisition. SS wrote the original draft. RC, GPT, WB contributed to the review and critical editing. All authors contributed to the final drafts.

Declaration of interests

All authors declare no competing interests.

Data sharing

Deidentified data relating to the study are available on reasonable request to the corresponding author.

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References

1 Cauchemez S, Valleron AJ, Boëlle PY, Flahault A, Ferguson NM. Estimating the impact of school closure on influenza transmission from Sentinel data. *Nature* 2008; **452:** 750–54.

2 Earn DJ, He D, Loeb MB, Fonseca K, Lee BE, Dushoff J. Effects of school closure on incidence of pandemic influenza in Alberta, Canada. *Ann Intern Med* 2012; **156:** 173–81.

3 Auger KA, Shah SS, Richardson T, et al. Association between statewide school closure and COVID-19 incidence and mortality in the US. *JAMA* 2020; **324:** 859–70.

4 Brauner JM, Mindermann S, Sharma M, et al. Inferring the effectiveness of government interventions against COVID-19. *Science* 2021; **371:** eabd9338.

5 Swann OV, Holden KA, Turtle L, et al. Clinical characteristics of children and young people admitted to hospital with covid-19 in United Kingdom: prospective multicentre observational cohort study. *BMJ* 2020; **370:** m3249.

6 Viner RM, Mytton OT, Bonell C, et al. Susceptibility to SARS-CoV-2 infection among children and adolescents compared with adults: a systematic review and meta-analysis. *JAMA Pediatr* 2020; **175:** e204573.

7 Riley S, Ainslie KEC, Eales O, et al. REACT-1 round 6 updated report: high prevalence of SARS-CoV-2 swab positivity with reduced rate of growth in England at the start of November 2020. http://hdl.handle.net/10044/1/83912 (accessed March 28, 2022).

8 McLean HQ, Grijalva CG, Hanson KE, et al. Household transmission and clinical features of SARS-CoV-2 infections. *Pediatrics* 2022; **149:** e2021054178.

9 Jones TC, Biele G, Mühlemann B, et al. Estimating infectiousness throughout SARS-CoV-2 infection course. *Science* 2021; **373:** eabi5273.

10 Ismail SA, Saliba V, Lopez Bernal J, Ramsay ME, Ladhani SN. SARS-CoV-2 infection and transmission in educational settings: a prospective, cross-sectional analysis of infection clusters and outbreaks in England. *Lancet Infect Dis* 2021; **21:** 344–53.

11 Stein-Zamir C, Abramson N, Shoob H, et al. A large COVID-19 outbreak in a high school 10 days after schools' reopening, Israel, May 2020. *Euro Surveill* 2020; **25:** 2001352.

12 Zhao H, Joseph C, Phin N. Outbreaks of influenza and influenza-like illness in schools in England and Wales, 2005/06. *Euro Surveill* 2007; **12:** e3–4.

13 Health Protection Agency West Midlands H1N1v Investigation Team. Preliminary descriptive epidemiology of a large school outbreak of influenza A(H1N1)v in the West Midlands, United Kingdom, May 2009. *Euro Surveill* 2009; **14:** 19264.

14 Cordery R, Purba A, Begum L, et al. Frequency of transmission, asymptomatic shedding, and airborne spread of *Streptococcus pyogenes* among schoolchildren exposed to scarlet fever: a longitudinal multi-cohort molecular epidemiological contact tracing study in England, UK. *Lancet Microbe* 2022; **3:** e366–75.

15 Rowan AG, May P, Badhan A, et al. Optimized protocol for a quantitative SARS-CoV-2 duplex RT-qPCR assay with internal human sample sufficiency control. *J Virol Methods* 2021; **294:** 114174.

16 Zhou J, Otter JA, Price JR, et al. Investigating SARS-CoV-2 surface and air contamination in an acute healthcare setting during the peak of the COVID-19 pandemic in London. *Clin Infect Dis* 2020; **73:** ciaa905.

17 Hoschler K, Ijaz S, Andrews N, et al. SARS antibody testing in children: development of oral fluid assays for IgG measurements. *Microbiol Spectr* 2022; **10:** e0078621.

18 Lin YC, Cheng CY, Chen CP, Cheng SH, Chang SY, Hsueh PR. A case of transient existence of SARS-CoV-2 RNA in the respiratory tract with the absence of anti-SARS-CoV-2 antibody response. *Int J Infect Dis* 2020; **96:** 464–66.

19 Brandal LT, Ofitserova TS, Meijerink H, et al. Minimal transmission of SARS-CoV-2 from paediatric COVID-19 cases in primary schools, Norway, August to November 2020. *Euro Surveill* 2021; **26:** 2002011.

20 Dawson P, Worrell MC, Malone S, et al. Pilot investigation of SARS-CoV-2 secondary transmission in kindergarten through grade 12 schools implementing mitigation strategies—St. Louis County and City of Springfield, Missouri, December 2020. *MMWR Morb Mortal Wkly Rep* 2021; **70:** 449–55.

21 Zimmerman KO, Brookhart MA, Kalu IC, et al. Community SARS-CoV-2 surge and within-school transmission. *Pediatrics* 2021; **148:** e2021052686.

22 Ehrhardt J, Ekinci A, Krehl H, et al. Transmission of SARS-CoV-2 in children aged 0 to 19 years in childcare facilities and schools after their reopening in May 2020, Baden-Württemberg, Germany. *Euro Surveill* 2020; **25:** 2001587.

23 Young BC, Eyre DW, Kendrick S, et al. Daily testing for contacts of individuals with SARS-CoV-2 infection and attendance and SARS-CoV-2 transmission in English secondary schools and colleges: an open-label, cluster-randomised trial. *Lancet* 2021; **398:** 1217–29.

24 Chen PZ, Bobrovitz N, Premji Z, Koopmans M, Fisman DN, Gu FX. Heterogeneity in transmissibility and shedding SARS-CoV-2 via droplets and aerosols. *eLife* 2021; **10:** e65774.

25 Adam DC, Wu P, Wong JY, et al. Clustering and superspreading potential of SARS-CoV-2 infections in Hong Kong. *Nat Med* 2020; **26:** 1714–19.

26 Miller E, Waight PA, Andrews NJ, et al. Transmission of SARS-CoV-2 in the household setting: a prospective cohort study in children and adults in England. *J Infect* 2021; **83:** 483–89.

27 Thompson HA, Mousa A, Dighe A, et al. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) setting-specific transmission rates: a systematic review and meta-analysis. *Clin Infect Dis* 2021; **73:** e754–64.

28 Paul LA, Daneman N, Schwartz KL, et al. Association of age and pediatric household transmission of SARS-CoV-2 infection. *JAMA Pediatr* 2021; **175:** 1151–58.

29 Federgruen A, Naha S. Crowding effects dominate demographic attributes in COVID-19 cases. *Int J Infect Dis* 2021; **102:** 509–16.

30 Department for Education Evidence Summary Coronavirus (COVID-19) - children, young people and education settings July, 2021. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1000025/Evidence_Summary_-_July_2021.pdf (accessed Dec 20, 2021).

31 Chudasama DY, Tessier E, Flannagan J, et al. Surge in SARS-CoV-2 transmission in school-aged children and household contacts, England, August to October 2021. *Euro Surveill* 2021; **26:** 2101019.

32 Elliott P, Bodinier B, Eales O, et al. Rapid increase in omicron infections in England during December 2021: REACT-1 study. *Science* 2022; **375:** 1406–11.