

Children are not COVID-19 super spreaders: time to go back to school

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Since the first reports of SARS-CoV-2 infections in China, doctors, parents and policy-makers have been aware that COVID-19 is 'not just another respiratory virus' in children. There is a large discrepancy in case rate and prognosis between young children and older adults that has caught everyone by surprise, and for which the mechanisms remain unknown. As community testing has demonstrated a significant number of children with no or subclinical symptoms,¹ key questions needs answering: are there low rates of confirmed infection in children because children are not becoming infected and/or infectious, or is COVID-19 in children usually such a benign upper respiratory illness that does not even cause infants or immune suppressed children to need hospital admission? If children are infected, are they infectious to each other and/or to adults? If so, how long for?

The implications of asymptomatic but potentially infectious children in the community are important. If, as for influenza,² children are the primary drivers of household SARS-CoV-2 transmission, then silent spread from children who did not alert anyone to their infection could be a serious driver of community transmission. On this presumption but without evidence, school closures were implemented almost ubiquitously around the world to try and halt the potential spread of disease despite early modelling that suggested this would have less impact than most other non-pharmacological interventions.³

Early contact tracing data from Shenzhen, China, appeared to confirm a role for children in transmission. Although apparently presenting with more benign disease or even without symptoms, similar attack rates were found in children and adults

in individual households.⁴ However, the story has subsequently evolved.

Some regions have implemented wide-spread community testing, such as South Korea and Iceland. Both countries found children were significantly underrepresented. In Iceland, this is true both in targeted testing of high-risk groups compared with adults (6.7% positive compared with 13.7%) and in (invited) population screening, there were no children under 10 found to be positive for SARS-CoV-2 compared with 0.8% of the general population.⁵ Subsequently, early pre-print data from the town in Vo, Italy, showed similar findings. With 86% of their population screened following the first death in late February, no children under 10 years were found to be positive (compared with 2.6% of the general population).⁶ This was despite a number of children found to be living with adults who had COVID-19, but where it was not transmitted (or was unable to be detected). Data from contact tracing in Japan demonstrated lower attack rates in children,⁷ and recent pre-print data from Guangzhou province in China have also demonstrated a much lower secondary attack rate for children than their adult counterparts (OR 0.23 compared with adults >60 years).⁸

Evidence is therefore emerging that children could be significantly less likely to become infected than adults. On the other hand, children could have a more transient upper respiratory infection with minimal viral shedding, or the less likely scenario of showing minimal symptoms despite significant viral shedding. A further key question is the ability of infected children to spread SARS-CoV-2. A collection of international family clusters found that children were not likely to be the index case in households, only being responsible for around 10% of clusters.⁹ Data from Guangzhou have supported this, finding an even lower rate of children as index cases in households at 5%.⁸ A case study of a cluster in the French Alps included a child with COVID-19 who failed to transmit it to any other person, despite exposure to more than a hundred children in different schools and a ski resort.¹⁰ In New South Wales, Australia none of 735 students and

128 staff contracted COVID-19 from nine child and nine adult initial school cases despite close contact.¹¹ In The Netherlands, separate data from primary care and household studies suggests SARS-CoV-2 is mainly spread between adults and from adult family members to children.¹²

Until there is high-quality sero-surveillance data, these questions will not be able to be answered with certainty. It is possible that biases in population selection for testing or false-negative swabs due to difficulties sampling in children contribute to existing findings. However, these data so far have been consistent across regions and continue to push the evidence in one direction. Sero-surveillance will not be produced quickly despite ongoing studies (<https://whatsthestory.web.ox.ac.uk/>) due to the logistics of mass blood sampling in children and global issues with large-scale antibody testing quality control. In the meantime, schools remain closed and policy-makers around the world are considering their options for releasing the pressure on lock-downs as case numbers and death start to plateau and fall.

In addition, there has been very little evidence so far on the effects of COVID-19 on children with comorbidities. This contrasts significantly to COVID-19 disease in adults. All three children who required intubation in a large Chinese cohort study had comorbidities (including leukaemia, hydronephrosis and intussusception),¹³ but it is not clear if COVID-19 was the reason for their needing intensive care. Limited data on children post liver transplant,¹⁴ with inflammatory bowel disease on immunosuppression¹⁵ and cancer,¹⁶ are reassuring. Limited data from the USA CDC are available on children with comorbidities; however, 23% of confirmed cases were found to have a comorbid condition (most commonly respiratory), and 77% of patients with known hospitalisation status (n=37) had a comorbidity (including all six cases admitted to PICU).¹⁷ Despite these mostly reassuring data, most public health interventions have assumed children with comorbidities such as primary or secondary immune dysfunction or respiratory/cardiac illness to be at increased risk, and in the UK this has led to advice for these children to shield completely, as for adults known to be at risk. Many paediatric specialists are concerned that a blanket assumption that immune-suppressed children of any kind are all at increased risk will cause considerable long-term educational and social harm to these children.

At the current time, children do not appear to be super spreaders. Sero-surveillance data will not be available to

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confirm or refute these findings prior to the urgent policy decisions that need to be taken in the next few weeks such as how and when to re-open schools. Policies for non-pharmacological interventions involving children are going to have to be made on a risk–benefit basis with current evidence available.

Governments worldwide should allow all children back to school regardless of comorbidities. Detailed surveillance will be needed to confirm the safety of this approach, despite recent analysis demonstrating the ineffectiveness of school closures in the recent past.¹⁸ The media highlight of a possible rare new Kawasaki-like vasculitis that may or may not be due to SARS-CoV2 does not change the fact that severe COVID-19 is as rare as many other serious infection syndromes in children that do not cause schools to be closed. Individualised risk assessment and decision-making by clinicians should occur for those considered at exceptional risk (such as in immediately after bone marrow transplant) or where there are other older family members at significant risk.

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