Implications of a highly transmissible variant of SARS-CoV-2 for children

Viner and colleagues1 reviewed data on SARS-CoV-2 transmission involving school-age children, found no evidence that children are more likely to transmit SARS-CoV-2 than adults and suggested that children are likely the safer population group among which social distancing measures could be relaxed. We reconstructed the age groups that sustain resurgence COVID-19 in the USA by combining mobility data of >10 million individuals, age-specific COVID-19 attributable death data and school case data, and came to consistent conclusions.2 In autumn 2020, after in-person schooling or hybrid models resumed in most US school districts,3 our analyses indicate the reproduction number (R) from children and teens was well below 1 at the population level, and that children aged 0–9 years contributed less than 5% and teens 10–19 years less than 10% to onward spread. While asymptomatic infections remained largely unreported,3 to onward spread. While asymptomatic infections remained largely unreported,3 our analyses indicate the reproduction number (R) from children and teens was well below 1 at the population level, and that children aged 0–9 years contributed less than 5% and teens 10–19 years less than 10% to onward spread. While asymptomatic infections remained largely unreported,3 mounting evidence from the USA and internationally indicates that school-age children do not contribute substantially to the spread of 2020 SARS-CoV-2 variants, unlike the spread of other respiratory pathogens.

Since winter 2020, a novel and highly transmissible SARS-CoV-2 lineage, B.1.1.7, is expanding globally. Large-scale surveillance data from England suggest that reproduction numbers for B.1.1.7 are 1.79 (1.22–2.49) times higher than those of other lineages.4 To assess the transmission potential of B.1.1.7 from school-age children in the USA, we adjusted age-specific reproduction numbers for currently circulating SARS-CoV-2 lineages3 with the inferred transmission advantage of the B.117 lineage.4 The predicted reproduction numbers for B.117 remain slightly but not certainly below 1 for children and are around 1 for teens. This suggests that B.117 and similar variants could qualitatively change the nature of SARS-CoV-2 spread in school settings from sinks of infection to amplifiers of infection, but concurrently our data and modelling provide no overwhelming evidence that stuttering transmission chains will turn into exponential growth in school settings and further fuel resurgent COVID-19 epidemics where B.117 is spreading.

School reopening remains a priority to put an end to the many harms inflicted on children during the COVID-19 pandemic1 and rebuild their mental health and futures, even as SARS-CoV-2 continues to evolve.4 We cannot tell where and when the highly transmissible B.117 or other highly transmissible variants of concern may become the dominant SARS-CoV-2 strain. It seems prudent to expand SARS-CoV-2 testing (including in schools), scale up molecular surveillance to detect B.117 spread in community settings and redouble and focus test-and-trace efforts on B.117 cases. Our calculations suggest that, where B.117 is spreading, more stringent mitigation measures may become necessary as schools reopen, including improved ventilation and air cleaning in schools, outdoor classes where feasible to reduce transmission and even potentially closing some schools as an integral part of comprehensive epidemic control measures. Meanwhile, vaccinating high-risk individuals as rapidly as possible to prevent COVID-19 deaths and hospitalisations, and then adults aged 20–49 to reduce spread1 is our strongest asset in the race against highly transmissible COVID-19.

Oliver Ratmann 1, Samir Bhatt, 1, 2 Seth Flaxman 1
1 Department of Mathematics, Imperial College London, London, UK
2 Department of Public Health, University of Copenhagen, Copenhagen, Denmark

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