Stepping Back to School
A step-by-step look at COVID introduction, spread, and exportation

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Results as of February 24, 2021

What do we already know?
Closing schools early in the COVID-19 pandemic has been associated with significant reductions in disease transmission at the community level, yet K-12 schools that have returned to in-person learning have reported few outbreaks. Modeling suggests that this discrepancy can be largely explained by school-based countermeasures that were not in place when schools closed. Guidance from CDC and state departments of health, including Washington, agree on the importance of these countermeasures. But we also know that schools are not islands: COVID-19 burden in the community is an important determinant of school-based risk. And while diagnostic testing of symptomatic individuals and contacts of known positives is essential, our testing the waters report found that “asymptomatic” testing of all students and staff added little value on top of other countermeasures. Even with vaccines soon available to staff, substantial uncertainties remain and no zero-risk scenarios are available.

What does this report add?
We use a detailed computer model of COVID-19 to explore health risks associated with in-person learning in three steps: 1. introduction to school, 2. outbreak spread within school, 3. exportation from schools. Whereas previous work, including our own, considered schools holistically and in the context of a specific place and time, this step-by-step approach enables new insights into risks and mitigation strategies. On introduction, we find that the rate at which COVID-19 is introduced to the classroom is proportional to the prevalence of COVID-19 in the community. Introductions are mostly likely to occur after a weekend or holiday. Few tools are available to reduce the introduction rate, although high-frequency asymptomatic testing helps. On spread within school, many tools affect the size of school-based COVID-19 outbreaks following introduction. Here, we find that outbreaks will be small if countermeasures are sufficient to limit in-school spread ($R < 1$). But detection and immediate action are essential to prevent large outbreaks if COVID-19 is able to spread ($R > 1$), perhaps due to gaps in non-pharmaceutical interventions (NPI) or more transmissible variants. Outbreaks are larger in high schools, and can be mitigated significantly though hybrid scheduling. Finally, the risk of exporting COVID-19 from K-12 schools to households and the broader community depends on the combination of how often the virus is brought into schools and how many get infected at school.

What are the implications for public health practice?
The best way to avoid COVID-19 in schools is to reduce prevalence in the community. But unless the virus is eliminated, school introductions are going to happen because countermeasures slow, but do not stop, introductions. Countermeasures should focus on reducing the possibility of spread within schools so as to limit the outbreak size. To avoid larger outbreaks resulting from more transmissible variants or weak in-school countermeasures, discovering outbreaks early and reacting aggressively is critical. Children are at low risk of severe outcomes, but vulnerable staff and family members should seek vaccination, when available.

¹We thank Mandy Izzo, Jen Schripsema, and Kate Davidson for their support in reviewing this report.
Executive summary

Returning K-12 students to in-person learning remains controversial during the COVID-19 pandemic. Modeling results, including results shared in our first three reports, broadly conclude that health risks are non-zero, but can be mitigated using multicomponent countermeasures including symptom screening, masks, distancing, ventilation, avoiding large groups, and classroom cohorting. We have also seen that risks can be further reduced using hybrid scheduling and by phasing in younger learners first; however, these approaches result in fewer in-person school days [1]. Schools are not islands, and we have found that school-based COVID-19 risks increase with disease activity levels in the community [2].

In this report, we take a different approach to studying the relationship between COVID-19 and the classroom while summarizing and adding new insights and nuance to our previous findings. Our previous work used the model to study a particular place at a point in time, most recently King County, WA in early October, when the case detection rate was 75 per 100,000 over 2 weeks. But the COVID-19 activity level varies substantially over time and from place to place. As of February 22nd, 2021, the case detection rate in King County is 124 per 100,000 over 2 weeks [3], having recently fallen from a high of 460 per 100,000 on December 12th. Instead of simulating a specific context, we use the model to identify fundamental COVID-19 transmission relationships in three steps. First is the rate at which COVID-19 is introduced from households or the community to a classroom setting. Once introduced, the second step explores what determines the size of the resulting outbreak. The third step considers the role schools may play in exporting COVID-19 back out to households.

We find that the introduction rate is proportional to the prevalence of COVID-19 in the community. Each 0.1% increase in prevalence results an increase in the daily introduction rate by 3.1 per 100,000, see Figure 2A. For a 500 person school, this increase is equivalent to one additional introduction over a 65 day period. COVID-19 is most likely to be introduced by a student, and after a break, such as a weekend or holiday. For a given prevalence of COVID-19 in the community, few countermeasures significantly reduce the rate of introductions, the exception being frequent rapid testing.

Upon introduction to a classroom setting, many factors affect the number of students and staff who may become infected through linked in-school transmission. Unlike introduction rate, outbreak size varies non-linearly with factors such as symptom screening, diagnostic screening, and contact tracing, meaning that outbreaks can grow quickly if insufficient countermeasures are in place. Specifically, we identify two regimes: 1. if in-school virus transmissibility is low ($R_{0,sch} < 1$), outbreaks will be small and additional countermeasures such as frequent “asymptomatic” testing will add little value, 2. if in-school transmission is high, large outbreaks are possible with more transmissible variants or if countermeasures are insufficient. In this case, it is critical to identify outbreaks while they are still small and take immediate action to terminate growth. Outbreaks can be identified by testing individuals flagged by symptom screening or by frequent “asymptomatic” testing. Actions to terminate in-school spread include contact tracing and reactive closures.

Students and staff with COVID-19 are infectious to their families and broader community. Results here are unsurprising: the frequency of exports from schools depends on the number of people infected in schools, which in turn is affected by the introduction rate and outbreak size.

To mitigate school-based health risks, public health should continue to seek countermeasures that reduce the prevalence of COVID-19 in the community. While introductions and subsequent outbreaks are difficult to eliminate, readily available countermeasures are effective in limiting in-school spread and stopping outbreaks before they reach many individuals. With sufficient countermeasures, “outbreaks” will usually not spread beyond the source and may not even be noticed. More infectious variants [4, 5] may increase in-school transmissibility despite countermeasures, thereby necessitating outbreak discovery and reactive countermeasures. Recently clinical trials of vaccines from Pfizer [6], Moderna [7], and others have demonstrated high efficacy in preventing adults from severe disease, hospitalization, and death, but we do not have data to know if vaccination prevents asymptomatic infection and onward transmission [8].
Key inputs and assumptions

The results presented in this report were generated using the same agent-based model that we have used in our previous work, Covasim. This computer model simulates individuals; interpersonal contacts at home, school, work, and in the community; COVID-19 disease progression and transmission; and public health countermeasures like diagnostic testing, contact tracing, isolation, and quarantine. Please refer to our technical report [9] for background details about the model, or view the source code on GitHub. Contact networks are generated by SynthPops.

In this analysis, we use new methodology to map relationships between COVID-19 risk and various countermeasures and contextual factors, thereby allowing these results to better generalize across settings. We explore key inputs and assumptions to determine the link between these factors and school-based transmission. Results are quantified along the three principle steps depicted in Figure 1 and defined below.

![Figure 1: The relationships between schools and the broader community is typically studied holistically, but in this work we decompose the problem into three steps: 1. introduction to schools, 2. outbreak spread within schools, and 3. exportation back to households and the broader community.](image)

**Step 1 Introduction:** An introduction event occurs when a student or staff member who was infected outside of school *physically attends school while infectious*. Because the frequency of introductions depends on time, school size, and the percent of the school population who has already been infected and now has immunity, we present results in terms of the **introduction rate**, calculated as

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\text{Introduction rate} = \frac{\text{Number of introduction events recorded during the simulation}}{\text{School-person-days of exposure}}
\] (1)

To be included in the denominator, an individual must be a possible source of introduction into the school. For this reason, the denominator excludes members of the school community who have recovered from COVID-19 and therefore pose no risk of introduction. Vaccines provide tremendous individual protection against severe disease and death, but we have not removed vaccinated individuals from the denominator due to uncertainty surrounding the efficacy of the vaccine on reducing transmission.

**Step 2 Outbreak:** Given an introduction, we count the number of students, teachers, and staff who acquire COVID-19 as a direct result of the source infection, a single “epidemiologically linked” cluster. While
some define an outbreak as two or more linked infections, here an outbreak/cluster can be a single individual (the source of the introduction).

Step 3 **Exportation**: For individuals infected at school as part of a school-based transmission cluster, we explore the risk they pose to their families.

Infections simulated in the model begin with a latent period during which time the infected individual is not infectious. Following this period, viral load increases resulting in heightened infectivity before potentially developing symptoms [10]. We assume that symptom screening implemented in school populations will only detect symptomatic individuals, but that diagnostic screening with PCR or antigen-based tests can detect asymptomatic individuals as well, depending on test sensitivity. As in previous work, we assume that the probability of experiencing symptoms increases linearly with age from a low of 50% for children to a high of 90% for the elderly [11]. Much uncertainty remains about variations in susceptibility, symptomaticity, and infectivity by age and other factors.

This analysis revisits many scenarios and countermeasures addressed in our previous work. We consider the following school schedules:

1. **Full**: Students, teachers, and staff return to in-person learning 5 days per week.

2. **Hybrid**: Students are split into “A” and “B” groups. The A group attends in-person on Monday and Tuesday and the B group attends on Thursday and Friday. Teachers and staff are physically present all days except Wednesday.

3. **K-5**: Elementary schools conduct in-person learning 5 days per week while middle and high schools continue remote learning.

We assume full attendance; there is no remote alternative to in-person learning.

We also consider the following school-based diagnostic screening (asymptomatic testing) scenarios explored in our testing the waters report.

- **None**: Diagnostic testing continues as usual in the model, but no diagnostic screening is conducted.

- **PCR-based**: Diagnostic screening one time or at specified intervals. Most scenarios assume results would be available the next day, a potentially optimistic assumption. All students, teachers, and staff are included in screening.

- **Antigen-based**: Antigen tests have lower sensitivity and specificity compared to PCR, but results are available immediately. We model that positives will quarantine while awaiting follow-up PCR confirmation, a 3-day delay. If the PCR result is negative, the individual may return to school, and if positive, they will enter isolation and contact tracing will be initiated. We also consider weekly antigen testing for just teachers & staff.

Importantly, both PCR and antigen tests will be negative during the latent period, and only become positive in the infectious period (with imperfect sensitivity). The relationship between viral load, PCR or antigen test positivity, and infectiousness remains uncertain and could impact our findings. For example, if antigen tests have very low sensitivity until long after an infected individuals becomes infectious, their value in preventing introductions would be lower than we have assumed. Assumed properties of these diagnostics are described in Appendix B.

Finally, we continue to account for the importance of countermeasures as an instrumental part of mitigating risk:
**Symptom screening:** A percentage of students, teachers, or staff who are scheduled to attend school on a particular day will be screened for symptoms. Unless otherwise stated, the coverage of symptom screening is 50% (without correlation from day-to-day), and the coverage of symptom screening is one of the parameters that we will sweep from 0% to 100%. The model is agnostic to the symptom screening location (home or school). Individuals who are currently experiencing symptoms due to COVID-19 will screen positive (if screened), but note that not all COVID-19 infections will experience symptoms, and for those that do, symptoms will only develop after a brief period of elevated infectivity (as described above). Individuals without COVID-19 can screen positive due to other influenza-like illness (ILI) with 0.2% per day (without day-by-day correlation).

Individuals who screen positive will begin isolation on the same day, and 50% will seek a confirmatory PCR diagnostic test, which takes 2 days to return results. If the results are negative, the individual will return to school on the next in-person day. If the results are positive, contact tracing may be conducted.

**Contact tracing:** When a student, teacher, or member of staff is diagnosed with COVID-19 by a PCR diagnostic test, there is a 75% chance that the individual will be reached by case investigators and provide a list of contacts including one or more teachers, staff, and student contacts. We assume that 95% of school contacts can be reached, and begin a 14-day quarantine period starting on the same day as the index case was diagnosed (an optimistic assumption). Outside of schools, contact tracing is a normal part of the Covasim model, and is happening in the background of school scenarios.

**Non-pharmaceutical interventions (NPI):** NPI include countermeasures such as hand hygiene, masks, physical distancing, and ventilation. Instead of simulating each of these factors independently, we model that NPI will reduce the per-contact daily transmission probability in schools. In the past we have assumed the reduction would be 25%, and while that reduction remains the baseline value used in this report, we will consider a range of values for in-school transmissibility, thereby accounting for both NPI and conversely, increases in transmissibility due to variants such as B.1.1.7.

As with all disease modeling, this analysis has underlying assumptions that may impact our results:

- Our simulations are generated using demographic data, household size distribution and school enrollment data from King County, WA. Unlike previous work, we are not explicitly fitting the model to the epidemic in Washington State, but rather exploring the relationships that drive transmission risk for a given population. We find in Appendix A that these underlying population-specific data do not dramatically affect our findings, meaning these results may generalize to other communities.

- As in our previous work, we model distinct types (preschools, elementary schools, middle schools, high schools, and universities) with different contact structures corresponding to the patterns commonly observed for students at these kinds of schools in the US [12]. We improve upon our previous modeling by capturing the relationship between school size and school type using data from Washington State [13, 14], see Figure 15.

- While in-school contacts are simulated in detail, we do not explicitly model school-based sports, after-school care, transportation, or other similar pathways that may contribute to transmission. However, we do explore the impact of weaker student cohorting in Appendix C.

- Compared to adults 20-64, we assume that children 0-9 and 10-19 are 33% and 66% as susceptible to infection [15]. We explore two other susceptibility variations in Appendix D.

- The probability of an infected individual becoming symptomatic increases linearly with age from 50% for 0-9 to 90% for those aged 80+ [11].
• Symptoms begin concurrently with or within a few days after the period of peak infectiousness.

• Infectiousness is elevated during the post-latent phase, and varies between individuals, but we do not assume that asymptomatic infections are less infectious, nor do we assume that children are less infectious than adults [16]. This assumption means that students are as infectious as school staff per contact, but difference in contact patterns between students and staff will generate some transmission asymmetries in student-student, student-staff, and staff-staff transmissions as have been observed in recent school data from Germany [17] and in elementary schools in Georgia State [18].

• Diagnostic tests are positive with the stated sensitivity only when individuals are infectious, and are thus assumed to not detect infections during the latent (pre-infectious) period.

• COVID-19 transmission within schools is highly variable and challenging to estimate. For this reason, we often sweep across values of the in-school transmission risk. For scenarios in which we are exploring other variables, we assume a per-contact, per-day transmission probability that is close to 1.2%, which factors in NPI such as masks. When combined with other assumptions and countermeasures, this translates to a basic reproduction number \( R_0 \) within schools that is less than one, closer to 0.7 with the full schedule.

• In the main analysis, elementary and middle schools are cohorted, meaning that student interactions are limited to their classroom peers, but teachers and staff have random interactions with other teachers and staff within the same school. The sensitivity analysis in Appendix C explores implications if these “bubbles” break due to transportation, after-school care, or other logistical challenges. High schools are not cohorted, due to practical considerations around flexible schedules.

• The model does not include reactive transitions back to remote learning after detection of COVID-19. We expect that given a sufficiently large school-based outbreak, schools may close in-person learning entirely to reduce transmission.

• The simulation does not explicitly account for “seasonality” or other factors that may cause transmission to increase or decrease, but these factors would affect the prevalence of COVID-19 in the community, which we explore in detail.

• Diagnostic screening scenarios reach 100% of the intended target group, e.g. students, when in reality individual consent might not be received from everyone. Therefore, diagnostic screening results in this modeling analysis should be viewed as an upper bound on the possible impact of this countermeasure.

• Contact tracing within a school-based setting is assumed to occur on the same day as PCR confirmation, which we model with a two day delay. In reality, it might take a few additional days to find and notify contacts.

• Symptom screening is applied to 50% of individuals attending in-person school on a given day. This screening will not identify asymptomatic or pre-symptomatic infections. We explore how symptom screening might impact results by sweeping the screening probability through the full range.

• This modeling does not allow the possibility of reinfection due to immune waning or variants like B.1.351 and P.1 that may escape natural- or vaccine-derived immunity.

• Opening schools for in-person learning is challenging logistically and financially. This modeling work does not address those challenges.
Main results

Step 1: Introducing COVID-19 from the community into the classroom

We first explore how often COVID-19 is brought into the classroom from external sources. Because each student, teacher, or staff member who attends a school could be the source of an introduction, the number of introductions is proportional to school size. Similarly, the number of introductions increases proportionally to the length of time considered. Perhaps more subtle is that introductions are expected to decrease as immunity increases. For example, in a school population in which 20% have immunity from a previous infection, the virus has 20% fewer pathways into the school, and therefore 20% fewer introductions would be expected.

To capture these established relationships with school size, susceptibility, and time, the results we present will be quantified as an introduction rate. This rate is calculated as the ratio of two numbers. The numerator contains the number of introduction events in each simulation. To count as an introduction, the student or staff member must visit school in-person while infectious. The denominator is the cumulative number of school-person days among those at risk of introducing a case of COVID-19. This is calculated by adding up the number of students, teachers, and staff, excluding individuals who have recovered and therefore are not at risk of new introductions, day-by-day over the course of the simulation, starting from the day schools open and including weekends. The vaccine is not yet available for children and the extent to which the vaccine blocks transmission remains uncertain, so we have not removed vaccinated individuals from the denominator in this analysis. The units of the introduction rate are “introductions per school-person days,” and because the results are small, we will often report results as introductions per 100,000 (school person days). The introduction rate will be on the y-axis of many plots in this section.

Example: Consider as an example an introduction rate of 20 per 100,000. To determine how many introductions are expected to happen, we need some more information. For this example, consider a school of 500 (all susceptible) people over a period of 30 days. Then, to get the number of introductions, multiply the introduction rate by the time period, \( \frac{20}{100,000} \times 500 \times 30 \), to get 3. If 20% of the school people had been previously infected, the effective school population size would be \( (1 - 0.2) \times 500 = 400 \) people, and the resulting number of introductions this school would expect over 30 days would be \( \frac{20}{100,000} \times 400 \times 30 = 2.4 \).

We begin by varying the prevalence of COVID-19 in the model to see how COVID-19 activity levels in the community impact the introduction rate. We find that the introduction rate is proportional to prevalence: each 0.1% increase in community prevalence results in increase in the introduction rate by 3.1 introductions per 100,000 susceptible school-person days, see Fig. 2A. For a 500 person school, this works out to one additional introduction over the course of 65 days, assuming nobody has immunity.

Prevalence of COVID-19 in the population is not directly measurable, but can be estimated using various models and heuristics. We have produced model-based estimates of prevalence for various regions within the state of Washington [19]. Related work has produced simple calculations that estimate prevalence from directly observable quantities such as the number diagnosed over the past two-week period and the test positivity rate [20].

Few countermeasures are able to dramatically reduce the introduction rate. In-school interventions, such as NPI, prevent in-school transmission, but do not have an impact on the introduction rate. Nonetheless, we find that the introduction rate can be reduced by

1. Frequent “asymptomatic” testing using rapid tests

\(^2\)Prevalence is the percent of the population that is currently infected with COVID-19, including those who are not yet showing symptoms and those who may never show symptoms.
2. High compliance with daily symptom screening

3. Bringing back only K-5 students

**Introduction rate: school type**

We find that returning only elementary (K-5) students to in-person learning leads to a lower introduction rate compared to middle or high schools. Students in kindergarten through grade 5 are assumed to be less susceptible to COVID-19 compared to older students and adults. This reduced susceptibility leads to a lower COVID-19 prevalence among K-5 students than the general community. The result is that the introduction rate for the elementary schools scales more slowly with community prevalence compared to the other school types considered, see Figure 2A.

One factor working against the K-5 population is that their infections are assumed more likely to be asymptomatic, and therefore less likely to be caught by symptom screening before entering the school while infectious. We discuss the impact of symptom screening below.

**Introduction rate: diagnostic screening**

Asymptomatic testing at high coverage and with high frequency is one of the more effective ways to reduce the introduction rate, see Figure 2B. **Weekly diagnostic screening of all students, teachers, and staff with a rapid antigen test reduces the introduction rate by approximately 50% compared to no diagnostic screening. With fortnightly antigen screening, the introduction rate is reduced by approximately 25%.** Less frequent asymptomatic testing or screening only teachers and staff results on minimal reductions compared to no diagnostic screening.

In the model, infected individuals test negative during the latent (pre-infectious) period, after which they will test positive with high sensitivity until recovering. Because we are testing the designated school population at the beginning of the day before school starts, rapid antigen tests, which take approximately 15 minutes to deliver results, are able to prevent introductions that day. While we have not assumed that these tests would be able to detect pre-infectious individuals, this result is sensitive to the test being able to detect infectious individuals at the beginning of their infectious period.

**Introduction rate: school scheduling**

Much like elementary (K-5) is at lower risk of introductions compared to middle and high schools, we find that the K-5 phase-in approach has a **25% lower introduction rate compared to a full 5-day-per-week schedule.** The hybrid scheduling approach has a similar introduction rate to the K-5 phase in. The reason hybrid is below full is that infected individuals often become infectious before developing symptoms. Symptom screening cannot identify these pre-symptomatic individuals, who will attend school in-person while infectious and be counted as an introduction. With the hybrid schedule, delay between in-person days means that infected individuals are less likely to be scheduled for in-person learning on the first day they become infectious. The additional gap allows for a greater chance of symptom onset, for those developing symptoms, and therefore an introduction rate reduction by symptom screening.

**Introduction rate: symptom screening**

We have shown in previous work that daily symptom screening is one of the more effective countermeasures. However, blocking symptomatic individuals from in-person learning has only a modest impact on the rate of COVID-19 introductions. Figure 3 shows the dependence of the introduction rate on the daily screening probability for two prevalence levels. **Screening all students, teachers, and staff results in a 33%**
Figure 2: Simulation results showing how the rate at which COVID-19 is introduced to schools depends on the prevalence of COVID-19 in the community by A) school type, B) diagnostic screening, and C) reopening scenario. The rate of introductions increases linearly with community prevalence, the slope is 3.1 per 0.1% increase in the top panel when considering all schools combined. Shaded regions represent 95% confidence interval in the mean (solid line), variance of the full distribution is larger.
**reduction in the introduction rate compared to no symptom screening** for the scenario with 2% prevalence of COVID-19 in the community. The impact is similar in magnitude, but smaller in absolute scale, when the prevalence is lower, 1.1% and 0.2% are shown.

![Graph showing the importation rate of COVID-19 into schools decreases with increasing levels of symptom screening. Three levels of community prevalence are shown, 0.2% in red, 1.1% in blue, and 2% in green. As we have seen previously, higher prevalence results in higher levels of the importation rate, and the decreasing trend is more apparent at higher prevalence. For reference, 50% symptom screening is assumed in other results.](image)

Figure 3: The importation rate of COVID-19 into schools decreases with increasing levels of symptom screening. Three levels of community prevalence are shown, 0.2% in red, 1.1% in blue, and 2% in green. As we have seen previously, higher prevalence results in higher levels of the importation rate, and the decreasing trend is more apparent at higher prevalence. For reference, 50% symptom screening is assumed in other results.

The impact of symptom screening is not larger because symptoms often develop after a period of infectiousness, if symptoms ever develop, and therefore symptom screening will catch only individuals who develop symptoms concurrently with becoming infectious. While the limited impact of symptom screening here seems contradictory to our previous results, in fact symptom screening remains a powerful countermeasure. However, symptom screening acts primarily to reduce outbreak size rather than prevent introductions in the first place, as we shall see in the next section.

**Timing of introductions by day of week and around breaks**

Introductions are most likely to occur on the first day of school or after a weekend or holiday, see Figure 4. This is simply because introductions on Monday come from individuals who became infectious on Saturday, Sunday, or that Monday. In comparison, introductions on other weekdays are from individuals newly infectious that day only. This observation has public health implications for when to conduct testing, namely that tests should be conducted after a period away from school to reduce introductions.
Students are likely responsible for the majority of introductions

Our modeling assumptions imply that the majority of introductions, nearly 90%, will be due to students. This result comes from several factors. Students are assumed to be less susceptible to COVID-19 infection, by 66% for those under 10 and by 33% for 10-19 year olds, resulting in lower prevalence and hence fewer introductions per-individual. However, this effect is overwhelmed by the fact that there are approximately 20 students per teacher and slightly fewer non-teaching staff than teachers, resulting in a 11:1 student to staff ratio [14, 21]. A further small consideration is that, while students and staff are assumed to have similar levels of exposure to the broader community, student households are about 33% larger than staff households on average, thereby increasing student exposure.

This result from the model is consistent with recent data from schools in Germany, which showed that students are the introduction source in 80% of introductions [17]. However, other studies have indicated that teachers and staff are the most common source of introductions. Data from the UK shows that staff initiated 73% of school outbreaks [22]. Recent data from elementary schools in Cobb County, Georgia, also suggest that educators may play a significant role in seeding and spreading COVID-19; 4 of 9 identified outbreaks had an educator as the index, however 40% of contacts could not be reached for testing and educators may be more likely to get tested [18].

One difference is that we are assuming that all students comply with a return to in-person learning, resulting in an 11:1 student:staff ratio. The ratio might be lower in some of these datasets due to variations in school structures, remote learning options, and family choice. In the elementary school data from Georgia, 80% of elementary students were in-person, and the ratio of students to staff was lower at 4:1 [18]. Another potential difference is that school outbreak data can only identify the first individual who is diagnosed, the true infection source could have been undiagnosed, potentially due to asymptomatic infection. We do model that children are less likely to develop symptoms than adults, however there is considerable uncertainty surrounding this assumption. The age difference between students and staff results in a small bias towards identifying adults as the outbreak source. We account for this shift in Figure 5B, finding that the majority of introduction sources, as identified by the first individual diagnosed, would still be from students.

Note, however, that in this analysis we do not adjust for the possibility that staff would have higher
Figure 5: In each pie chart, the size of each wedge is proportional to the number of introductions started by students, teachers, or staff. In the left column, charts reflect the true (hidden) source of introductions. The right column more accurately reflects the distribution of individuals who would be identified as the introduction source, even if not the true source. The top row is a per-individual perspective showing that a given student is less likely to introduce COVID-19 than a member of staff. The bottom row shows the total for all members of a school. In short, teachers and staff are more likely to be infected and detected, but the roughly 10-fold higher number of students makes them the more likely source of introductions.

levels of testing, and therefore could be biased even more towards being the first diagnosed case in a school outbreak.
Introduction rate: teacher and staff vaccination

COVID-19 vaccines are effective in preventing severe disease and death, but there is much uncertainty around their ability to prevent acquisition and asymptomatic transmission. Nonetheless, because the majority of introductions are from students, vaccinating teachers and staff will not stop introductions.

We compared baseline simulations, which do not have vaccination, to a scenario in which a hypothetical “perfect” vaccine is given to all teachers and staff. This vaccine scenario is intentionally optimistic to give an upper bound on the impact of vaccination. The vaccine scenario is optimistic for two reasons: 1) we assume all teachers and staff accept the vaccine and are fully vaccinated before the start of in-person learning, and 2) the vaccine has 100% efficacy in blocking acquisition. Under this scenario, no teachers or staff can become infected with COVID-19. While the available vaccines are highly efficacious against hospitalization and death, and also reduce the likelihood of mild symptomatic infections, their efficacy against preventing acquisition of an asymptomatic infection and subsequent transmission remains unknown.

As expected, these vaccination scenarios result in only a small reduction in the introduction rate. This is because teachers and staff are only responsible for 11% of introductions in this modeling work, see the bottom left pie chart in Figure 5.

Figure 6: The impact of three vaccine scenarios on the introduction rate. We compare a perfect vaccine administered to all teachers and staff (blue) to no vaccination (red). The impact of vaccination on the size of outbreaks is negligible at low levels of the in-school transmission probability, and increases with increasing transmission probability. Outbreak data from schools that have opened is consistent with a low transmission probability.

Step 2: Spread of COVID-19 within schools

The second step of this analysis explores determinants of outbreak size following introduction of COVID-19 into a school. Briefly, these results come from infecting one person, selected at random, from each elementary, middle, and high school represented in the model. There are no other infections in these simulations, and the virus is only able to spread at school so that we can measure the number of people infected directly.

In these simulations, virus transmission in workplaces and the general community is disabled, leaving only school and household transmission. We allow transmission at home because of the close connection between households and schools, and to be able to quantify the number of exportations from the school to households in the next section. Importantly, outbreak size includes only students, teachers, and staff. Family members infected in households are excluded from this part of the analysis.
by the initial “seed” infection as well as in the resulting cluster, which we follow to completion. **Outbreak size is simply the number of students, teachers, and staff who get infected as part of the transmission cluster stemming from the source infection.**

Unlike introductions, which are difficult to prevent, we find that **many interventions are able to limit the number of students, teachers, and staff infected during school-based outbreaks.** One of the strongest determinants of outbreak size is the transmissibility of COVID-19 within school settings. Non-pharmaceutical interventions such as face coverings, hand hygiene, increased ventilation, and physical distancing all reduce transmissibility. Alternatively, novel COVID-19 variants such as B.1.1.7 have been found to be more transmissible compared to the wild-type strain. Ultimately, we do not fully know how transmissible COVID-19 is in a classroom setting, and indeed every classroom, grade, and school is likely different based on environmental factors and compliance.

**Outbreak size: NPI and variants**

Most outbreaks will be small, but the average outbreak size increases with the in-school transmission probability, as expected. However, the rate at which outbreaks grow with increasing transmission probability is not linear, but rather exponential, see Figure 7(top). In-school transmissibility, which is affected by factors such as NPI and eventually more transmissible variants, is represented by a parameter called beta ($\beta_s$) in the model. The value of beta scales the probability that an infected individual transmits COVID-19 to a susceptible individual in school on any given day. Outbreak sizes grow exponentially, rather than linearly, because larger values of beta lead to higher transmission probabilities, which leads to more infections, which leads to more infections, and so on. This relationship highlights the importance of NPI to decrease the risk of transmission in school and prevent large outbreaks, particularly in the face of more transmissible variants.

Outbreak size has a strong dependence on school type, see again the top panel of Figure 7. What we find is that high schools are at considerably higher risk for large outbreaks compared to middle and elementary schools. This finding is likely driven by two main factors. First, we have assumed that susceptibility to COVID-19 increases with age so that older students are more likely to acquire the virus than younger students. Second, while elementary and middle schools are cohorted into roughly 20 person groups, we have not assumed that high schools would be able to implement cohorting due to individualized schedules. Finally, high schools tend to be larger than other school types, thereby allowing the virus to spread farther within a single school. Based on these results, we recommend extra surveillance in the form of enhanced symptom screening and/or diagnostic screening for these schools, combined with immediate action on a positive to quarantine contacts and close the school if appropriate.

While the relationship with outbreak size is exponential, transmissibility ($\beta_s$) has a linear relationship with the reproduction number of COVID-19 in schools, $R_{0,s}$, see Figure 7(bottom). The reproduction number quantifies how many secondary infections directly caused by the initial “seed” infection in an otherwise completely susceptible school population. As predicted by classical epidemiology, low values of $R_{0,s} < 1$ generally result in small outbreaks, and the outbreak size grows exponentially as transmissibility, and hence $R_{0,s}$, increases. However unlike classical theory, these model-based results include the impact of variations in infectivity between individuals and within the time-course of a single infection, school cohorting structure, scheduling factors, and age effects.

The size of individual outbreaks is not well represented by the mean outbreak size. In the middle panel of Figure 7, we show the size of individual outbreaks that happened in the model as points. Large outbreaks are apparent only on the right side of the figure, where the in-school transmission probability is high. Another way to visualize outbreak sizes is shown in the third panel of Figure 7. Here we see that most outbreaks are small, and often there is no spread beyond the source. The risk of a large outbreak grows with the in-school transmission probability.

School data from around the world consistently shows that without mitigating countermeasures in
place, schools are capable of producing large outbreaks. Studies that have explored the role of school closures on the community-wide reproduction number have found closing schools early in the epidemic to be associated with reducing transmission [23, 24, 25]. And schools that reopened without sufficient countermeasures, such as in Israel [26] and a high school in Georgia State [27], lead us to believe that $R_0$ in schools without NPIs is above 1. But schools that have reopened with countermeasures have not observed large outbreaks [28, 29], including data from the State of Washington showing the majority (64%) of outbreaks involved only two or three people [30]. Elementary school outbreak data from Georgia shows limited distancing in all 9 reported outbreaks and inadequate mask usage by students in 5 of 9 [18]. Combined, these results suggest that masks, distancing, and other NPIs are able to bring $R_0$ below 1. While we have previously modeled that these NPIs reduce the per-contact transmissibility of COVID-19 in school settings by 25%, the true baseline and impact of NPIs are variable and unknown. Regardless, new variants of concern could be more than 50% more transmissible [4], and could increase transmissibility towards a level that would promote greater levels of spread within schools.
Figure 7: Average outbreak size by school type (top), individual simulated outbreak data-points to show large outliers (2nd), distribution of outbreak sizes (3rd), and the calculated basic reproduction number in schools, $R_{0,s}$ (bottom), all depend on the level of transmission occurring within schools (x-axis). We show the individual outbreaks observed in the model as dots in the second panel because the average outbreak size (top) does not well characterize the large variance in this distribution. The third panel shows outbreaks are almost always small when transmission is low, and large outbreaks become increasingly likely and increasingly large as transmission increases, particularly in high schools. The dashed horizontal line in the top two panels is at an outbreak size of one, which is the minimum because the source individual is included.
Outbreak size: teacher and staff vaccination

Vaccines against COVID-19 are powerful against preventing hospitalization and death; however, the extent to which these vaccines prevent asymptomatic infections, and subsequent spread, remains uncertain. Nonetheless, we can use the model to explore the extent to which vaccinating teachers and staff may reduce the size of outbreaks within schools. With strong student cohorting, teachers and staff may serve as a “bridge” between one group of students and the next. If vaccines prevent spread of COVID-19, vaccinating these populations may reduce outbreak sizes.

As in the step focused on introductions, we compare an optimistic “perfect” vaccine scenario against no vaccine. Results presented in Figure 8 show little separation between vaccination scenarios when the in-school transmission probability is low. Here, countermeasures alone are sufficient to prevent large outbreaks; the virus is rarely using the teachers and staff as “bridges” to connect otherwise separate student cohorts. From limited outbreak data, it does appear as though the transmission probability is in this low-transmission regime; however, the transmission probability will be higher in schools that are not able to implement countermeasures or as more transmissible variants become dominant. When the transmission probability is higher, the impact of the vaccine can be seen in the figure, resulting in outbreaks that are smaller compared to the no-vaccine scenario.

Regardless of the impact on outbreak size, we know that COVID-19 vaccines are highly effective in protecting at-risk individuals from severe disease and death. All individuals should seek vaccination as eligibility permits.

![Figure 8: The impact of a perfect vaccine for all teachers and staff on the average size of outbreaks. We compare this perfect vaccination scenario (blue) to no vaccination (red). The impact of vaccination on the size of outbreaks is negligible at low levels of the in-school transmission probability and increases with increasing transmission probability. School outbreak data suggests that the transmission probability in schools that have opened is low.](image-url)

Outbreak size: symptom screening

We showed above that screening most students, teachers, and staff for symptoms of COVID-19 infection each day before in-person learning had a modest impact on the rate of introductions. We now explore the impact of symptom screening on the number of infections that may occur after the virus has been brought into the classroom. To generate these results we consider the full range of probabilities that each student,
teacher, or staff is screened for symptoms on each school day. There is no day-to-day correlation assumed, so an individual who skips symptom screening one day does not have an elevated chance of being skipped on the next in-person day. Individuals who screen positive are not allowed to enter school, and we assume at baseline that half (50%) of screen positives will receive a diagnostic test that returns results in 2 days. If that diagnostic result comes back positive, contact tracing is initiated with 75% probability, immediately reaching 95% of in-school contacts. For reference, the baseline probability of symptom screening used in other parts of this analysis is 50%.

![Figure 9](image-url)

**Figure 9: Outbreak size depends on the daily probability of screening students, teachers, and staff for symptoms of COVID-19 infection. Mean outbreak size gets progressively smaller as the probability of symptom screening increases. The impact is larger when there is more transmission happening in schools (orange) compared to when there is less (red).**

Results displayed in Figure 9 show that symptom screening can significantly reduce outbreak size. At all levels of the screening probability, outbreaks are larger when in-school transmission is higher, as we saw in the previous section. The impact is greatest near the left-hand side of the figure where increasing the symptom screening probability from 0% to 20% reduces the outbreak size from 14 to 8 for the 2% transmission probability scenario.

For all levels of in-school transmission, a plateau is observed beyond which point additional symptom screening has little to no additional benefits. This point is reached almost immediately for the low transmission setting (red, transmission probability of 0.8% per contact, per day), and symptom screening adds little value in this scenario. On the other hand, the point of diminishing returns doesn’t happen until near 75% coverage in the high in-school transmission scenario (orange, transmission probability of 2.4% per contact, per day).

Symptom screening can only prevent symptomatic individuals from entering school, but many infected with COVID-19 will become infectious before developing symptoms. For this reason, even 100% symptom screening does not limit the outbreak size to 1, representing just the initial seed infection. Instead, the seed transmits to one or more others before the outbreak is even detected.

Therefore, **symptom screening acts primarily to identify outbreaks that have already started.** When in-school transmission is low, $R_{0,s} < 1$, these outbreaks will likely burn out on their own, often with zero
additional infections. In this scenario, symptom screening adds little value. But when in-school transmission is higher such that $R_{0,s} > 1$, possibly due to poor adherence to countermeasures or more transmissible variants, exponential growth in schools could cause large outbreaks. Symptom screening is one way to detect these outbreaks. But simply detecting the outbreak is not sufficient to stop it; immediate action such as contact tracing or reactive closures must be taken to terminate transmission chains in this $R_{0,s} > 1$ regime.

In these simulations the “action” taken on positive diagnosis is that, with 75% probability, the index case will be reached by cases investigators, who will notify 95% of school-based contacts. Stronger actions, for example conducting contact tracing from all diagnosed cases or closing schools, will have a greater impact, thereby increasing the value of symptom screening (and vice versa).

**Outbreak size: diagnostic screening**

Diagnostic screening using PCR- or antigen-based tests acts similarly to symptom screening in the sense that testing asymptomatic individuals enables discovery of ongoing outbreaks. Again we find two regimes, see Figure 10. When transmission conditions in school are such that $R_{0,s} < 1$, diagnostic screening adds little value over no diagnostic screening. This finding is consistent with what we found “with countermeasures” in the testing the waters report [31].

![Diagram](image_url)

**Figure 10:** The impact of various diagnostic screening scenarios (colored lines) on the mean outbreak size, as a function of the per-contact, per-day transmission probability in schools. When the transmission probability is small, $R_{0} < 1$ and the results of all diagnostic screening scenarios is similar. However, as the transmission probability increases, possibly due to poorly implemented NPI or novel variants, testing all students, teachers, and staff weekly (yellow is PCR, orange is antigen) maintains small outbreak sizes whereas no asymptomatic testing (red) results in large outbreak.

As compliance with NPIs falls and as more-transmissible variants come to dominate, we shift towards the right side of this figure; however, it is challenging to pinpoint a precise location on the x-axis due to variations between and within individuals schools. As with symptom screening, high transmission probability result in $R_{0,s} > 1$ and COVID-19 is likely to spread once introduced. Only in this regime do we find significant value in diagnostic screening. Screening only teachers and staff weekly (blue) adds marginal value beyond no diagnostic screening. Monthly frequency is an improvement, but additional reductions in
outbreak size can be realized by fortnightly diagnostic screening of all students, teachers, and staff. Weekly screening further reduces outbreak size, but the value add is marginal over the fortnightly frequency. Test type, PCR versus antigen, is of less relevance here as compared to preventing introductions in the first place, where we found that the speed of antigen testing was important in preventing introductions.

Note that these simulations assume that half (50%) of individuals are screened for symptoms before each in-person learning day. The value of asymptomatic testing in the $R_0 > 1$ regime would be even higher were symptom screening not implemented or screening with lower probability. Diagnostic and symptom screening both are able to identify ongoing outbreaks, and therefore work similarly to identify COVID-19 outbreaks, thereby preventing large outbreaks, see Figure 11.

![Figure 11: Dots show individual outbreaks realized during simulations under various transmission probabilities (panels, top=2.6% and bottom=1.3%) and diagnostic screening scenarios (rows within panels). Note that the scale of the x-axis is not aligned between the top and bottom panels because outbreaks are much larger when the in-school transmission probability is higher. As more and more diagnostic screening is conducted, the large outbreaks are identified and stopped.](image)

**Outbreak size: school scheduling**

We saw previously that hybrid and K-5 phase-in school schedules resulted in a lower introduction rate than the full 5-day schedule. The impact of these school schedules on the average size of outbreaks is shown in Figure 12. Outbreak size increases with the transmission probability in schools, however what is surprising in this figure is that hybrid school scheduling (blue) results in outbreaks that are considerably smaller than K-5 (green) or full (red), on average.
Hybrid scheduling has many advantages that result in small outbreaks, even at heightened in-school transmission levels. First, children are only in school two days per week, and teachers and staff only in-school four days per week. Second, class sizes are half as big, resulting in approximately half as many onward transmissions. Third, the A/B groups do not mix directly, so there is an additional cohorting effect. The infection can spread from one group to the other though the teacher. Fourth, symptom screening will work better for hybrid because the 5-day gap between schools days allows time for symptoms to develop, in those ever developing symptoms. These symptoms can be detected by symptom screening, triggering contact tracing to stop an ongoing outbreak. Finally, the infectious period is not much longer than 7-10 days in the model, so infected individuals will likely spend only 2 or 4 days in school while infectious, thereby limiting the potential for onward spread.

Figure 12: The impact of three school schedules, full (red), hybrid (blue), and K-5 phase-in (green) on the mean outbreak size varies with the in-school transmission probability. Outbreaks are expected to be small for all school schedules when the transmission probability is low, and size increases with transmission probability. Of the three schedules considered, the hybrid approach results in the smallest outbreaks, especially as in-school transmission increases. Solid curves represent the mean and the shaded regions capture 95% confidence intervals in the mean; individual outbreaks vary significantly in size.

The primary advantage of the K-5 phase-in approach is that these younger students are less susceptible to acquiring infection. But as the in-school transmissibility increases, due to partially implemented countermeasures or more transmissible variants, the benefit of lower susceptibility becomes overwhelmed.

We note that these results focus on the COVID-19 health risks of the various school schedules. Younger learners may have a more difficult time learning remotely, and therefore may receive a greater educational and social benefit from the in-person time. Also, while not simulated here, combining hybrid scheduling with K-5 (or even K-1 or K-2) phase-in would further reduce outbreak sizes. While hybrid learning is easy to simulate in a computer, we appreciate that this learning modality adds complexity to school schedules and teaching requirements, and may therefore not be feasible.
Step 3: Exportation of COVID-19 from schools

Students, teachers, and staff infected at school can bring the infection home to their families. The rate at which this occurs is proportional to the number of individuals infected at school, a product of the introduction rate and outbreak size given introduction. Few interventions are available to stop an infectious individual from unknowingly infecting their household members, particularly if the infection is asymptomatic or during the pre-symptomatic period. Instead, the best course of action is upstream by lowering community infection rates and implementing countermeasures fully. If school-based transmission is conducive to large outbreaks, adding strong symptom or diagnostic screening and quickly reacting to detected infections will ensure outbreaks do not grow too large.

Just as we simulated the introduction of one infection into each school in order to observe the progression of the outbreak, we could similarly simulate the introduction of COVID-19 into a household. The resulting percent of the household that acquires the virus is called the “household secondary attack rate,” or SAR for short. Data from careful studies and contact tracing usually estimates the SAR to be around 15-20% [32, 33], although there is considerable variation between studies. In the CDC MMWR reporting on data from elementary schools in Georgia, 18 of 69 household members of cases tested positive resulting in a household secondary attack rate of 26% [18].

Of course, this finding would still be cause for concern if many students and staff were acquiring COVID-19 at school. However, in most schools that have reopened, observed levels of in-school transmission are low, likely the $R_{0,s} < 1$ regime. In this case, even though occasional introductions will occur, outbreaks will be small on average, likely only the source individual and perhaps one other. This regime is consistent with observed data [34] as well as a recent study of 17 schools in Wisconsin [35]. However, even though the risks in this regime are not much higher than an all-remote learning scenario, multi-generational families may want to continue remote learning until vulnerable individuals are vaccinated. Under the vaccine roll-out plan in some states, including Washington, individuals over the age of 50 in multi-generational households are currently eligible to receive vaccination.
Appendix

A Does the setting matter?

In our main analysis, we used demographic and school enrollment/size distribution data from Seattle-King County to generate a simulated population. In order to test how well our results generalize to other settings across the state, we conducted a sensitivity analysis varying the population networks. We used three alternative counties to test the results: Spokane, Franklin, and Island Counties. Briefly, these locations vary from Seattle King County and each other in terms of age distribution as well as school size. Spokane and Franklin have more school-aged children as a share of the population than Seattle, whereas Island County has fewer children overall (see Figure 14). The three counties also have larger elementary schools on average than Seattle and smaller middle and high schools (see Figure 15).

Figure 13: Introduction rate (top) and outbreak size (bottom) for three Washington counties (Franklin, Island, Spokane) and the Seattle metro area. While small differences exist due to population age structure, school sizes, and student to teacher/staff ratios, the resulting trends are similar.
Figure 14: Age distribution of the Seattle metro area, Spokane, Franklin, and Island Counties from the 2019 American Community Survey [36].

Figure 15: Distribution of school size (students enrolled per school) in the four locations by type of school. Each histogram shows the data for the different locations. The left, middle, and right columns show data for elementary, middle, and high schools, respectively. Data on enrollment sizes come from the Washington Office of the Superintendent of Public Instruction [13, 14].
We find that the relationships between community prevalence of COVID-19 and introduction rate (linear) as well as in-school transmissibility and outbreak size (non-linear) are consistent even when we vary the population, see Figure 13. However, we see a small upward shift in introduction rate for Franklin Counties, indicating that for the same community-wide prevalence, there is a higher rate of COVID-19 introduction in schools for these settings. This finding is potentially due to the higher percentage of children aged 5-19 in Franklin County, 25%, compared to other locations such as Seattle, 16%.

B Assumptions about diagnostics

This analysis includes the potential benefit of one-time or routine test-based screening in schools as a congregate setting. Nothing specific in this analysis requires the screening tests to be physically performed at school; however, that might be easier logistically and lead to higher coverage levels. Two types of tests are considered:

- **PCR:** PCR tests are the clinical “gold standard” and have high sensitivity and specificity. In the Covasim model, PCR tests will return a positive result if the individual is currently in the infectious stage, and a negative result otherwise. In clinical settings, PCR tests can return positive results in the late stages, during which time individuals are potentially no longer infectious. In our model, these individuals would be the recovered stage, and the PCR test would return a negative result. Therefore, we may have under-estimated “false positive” results from PCR-based screening tests conducted in the post-infectious period. PCR tests typically take one or more days to return results. In screening scenarios, we assume results are available on the next day, a potentially optimistic assumption. For confirmation of screen- or antigen-positive individuals, we assume results will be available in 2 days. For routine diagnostic testing, we also assume that test results (based on PCR) are available in 2 days.

- **Antigen:** Antigen tests are less expensive than PCR and return results quickly, on the order of 15 minutes; however, they are less likely to identify true positives and more likely to generate false positives compared to PCR. There are numerous antigen-based tests, here we focus on the Abbott BinaxNOW™Ag CARD test, as these tests are broadly available within Washington State. Based on an FDA fact sheet specific to the BinaxNOW test, when applied to individuals who have experienced symptoms onset within the past 7 days, we model a sensitivity of 97.1% and a specificity of 98.5% [37]. For those not experiencing symptoms, or if symptom onset was more than 7 days in the past, we model a test sensitivity of 90% and maintain the specificity at 98.5%.

There is much uncertainty about the properties of antigen tests when used for screening the general population. The above numbers are based on just 35 positive examples. More research is needed to quantify the properties of these tests. The sensitivity of antigen-based tests correlates with the amount of virus present, often called the viral load. For this reason, this type of test may be less able to detect early infections, and therefore we may have over-estimated the impact of antigen-based diagnostic screening.

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4The Abbott BinaxNOW™Ag CARD test is authorized for use at the Point of Care (POC), i.e., in patient care settings operating under a CLIA Certificate of Waiver, Certificate of Compliance, or Certificate of Accreditation. Screening in congregate populations, such as schools, is considered off-label use.
C The value of cohorting students

In the base analysis, students in elementary and middle school are tightly grouped into “cohorts,” also called “pods,” whereas we have assumed that high school students are not cohorted due to scheduling complexities. Each student in a cohort will have connections to all other students in that same cohort, as well as their direct teacher as well as about one non-teaching staff member. The teachers and non-teaching staff are connected to each other, thereby enabling the virus to spread from one cohort to the next.

For schools using the hybrid A/B group approach to scheduling, the class is split into two equally sized groups and students from the A group have contacts with other A students and teacher/staff, and similar for B. In this case, the teacher and staff serve to connect the A and B groups, but there are no direct connections between A and B students.

In practice, we do not expect student cohorts to be perfect due to transportation and other logistics, after-school care, sports, and other factors. Here, we explore the impact less-ideal student cohorting might have on the size of outbreaks. Note that cohorting happens downstream of introductions, and thus will not affect the introduction rate.

For this analysis, we take the tightly cohorted student groups within each school and randomly choose 10% or 25% of the contacts to randomly “rewire” to some other student in the school. Thus the indicated percent of student-to-student edges will be reassigned to some other student, thereby weakening the cohorting effect from our baseline assumption.

We find that cohorting is protective, as expected, see Figure 16. The magnitude of the impact increases with the in-school transmission probability, and thus cohorting becomes more important when other countermeasures cannot be implemented fully or after more-transmissible variants are introduced. The impact is larger for middle schools than elementary schools due primarily to the lower assumed susceptibility of younger students.

One limitation of this sub-analysis is that we continue to assume that contact tracing works equally well for in- and out-of-cohort contacts. Of school based cases, we assume that 75% are reached and provide a list of contacts that includes 95% of school-based contacts. This high value is because we assume that within-cohort contacts will be easy to identify, it is the essentially the full cohort. The limitation of this sub-analysis is that we have not assumed that out-of-cohort contacts will be harder to identify. With this in mind, the value of cohorting will be even greater than these simulation results suggest due to better chances of identifying contacts.

Moreover, these results suggest that student cohorting reduces outbreak sizes, particularly in middle school and as in-school transmission increases.
Figure 16: We explore the impact of mixing between student cohorts in elementary (left) and middle (right) schools. At baseline, students are not assumed to mix (red). Here we “rewire” 10% (blue) or 25% (green) of the edges in the simulated network to represent mixing between cohorts. As in other figures, the shaded region represents 95% in the mean, true variance in outbreak size is much larger.

D Sensitivity of the susceptibility of children

Most scientific studies that have explored the relationship between age and susceptibility have found the children are less likely to acquire COVID-19. Note that susceptibility is different from the probability of acquiring symptoms given an infection, which also varies with age. Biological and behavioral mechanisms to explain age effects have been identified in the literature [38].

In this sensitivity analysis, we consider three variations on the susceptibility of children relative to adults:

• **Baseline**: Children 0-9 and 10-19 are 33% and 66% as susceptible to COVID-19 infection [15].

• **Children 56% as susceptible**: All children under age 20 are 56% as susceptible based on meta-analysis [39]. This scenario is similar to the 50% reduction for those under age 20 presented in [11].

• **Children fully susceptible**: Children are fully susceptible in the sense that they are more likely to acquire infections as adults. Support for this scenario is more limited, but a household study revealed equal susceptibility [40].

Results are presented in Figure 17. Regarding the introduction step, we see that the introduction rate is higher for the scenario in which children are fully susceptible compared to the baseline and 56% reduced alternatives. With equal susceptibility, children are at higher prevalence for a given population prevalence, and are therefore more likely to cause introductions. The similarity of the other two scenarios comes because 56% is near the middle of 33% (0-9) and 66% (10-19), although a difference would emerge if the results were broken out by school type.

With respect to the second step, outbreak, we find some considerable differences. The fully susceptible scenario results in larger outbreaks compared to the other two. Interestingly, the baseline scenario resulted in larger outbreaks than the result from the meta-analysis using 56% for all children under age 20. This finding is due to the upward sloping shape of these susceptibility curves. Moving the susceptibility of children aged 10-19 from 66% down to 56% results in considerable smaller outbreaks whereas moving children aged 0-9 from 33% to 56% has a smaller effect.
Moreover, our baseline assumptions [15] are slightly conservative compared to the 56% susceptibility number from meta-analysis [39]. These two scenarios have a similar rate of introductions, but the equal 56% susceptibility scenario results in larger outbreaks, on average. If children are fully susceptible, equal to adults, then introductions will be more frequent and outbreaks will be larger, on average, therefore compounding to more frequent exportations to households and the community.

![Figure 17: Sensitivity analysis comparing introduction rate (left) and mean outbreak size (right) for three variations on the susceptibility of children relative to adults. The baseline assumptions (red) are compared against scenarios in which children under 20 are 56% as susceptible (blue) or equally susceptible (green) compared to adults. Shaded regions represent 95% confidence intervals in the mean, true variance is larger.](image)

E Methods

This analysis uses the same Covasim model that we have used in our previous three school-based reports. In those works, we first used a calibration algorithm to automatically adjust model input parameters to that the output of the model looked like King County at the time of the report. We then simulated various hypothetical school reopening “scenarios” to learn the impact schools may have on the broader community and estimate the number of students, teachers, and staff who may acquire a COVID-19 infection of a period of time, typically three months.

We are now using the Covasim model in two new ways so was to gain a deeper understanding of what causes risk in school settings. None of these simulations require calibration, as we are no longer trying to make the model output match the epidemic in any particular location. The first new method was used for the simulations that estimate the introduction rate. Here, we configure the model to maintain a set community prevalence and count how often COVID-19 is introduced into schools once they reopen. The ability to set the community prevalence on a given simulation allowed us to sweep over prevalence levels across multiple simulations, aggregating statistics for each that were ultimately compiled into the plots.

The second new way in which we used the model was for the outbreak and exploration parts of the analysis. For these, we start the Covasim model without any infections. Then, for each elementary, middle, and high school in the model, we pick one student, teacher, or staff at random and infect them with a simulated COVID-19 infection. From each of these “seed” infections, the virus may spread to others in the school or at home, but we have disabled community and workplace transmission layers to prevent technical challenges.

Each simulation represents 223,000 individuals, a fraction of the entire community. Nonetheless, population size is not an important factor because the introduction rate normalized by the population size and
the outbreak size is measured within a specific school. For outbreak/exporation simulations, we ensure that the model runs long enough that all seeded outbreaks have terminated so as to avoid a downward bias in outbreak size that would otherwise have been caused by stopping too soon.

The model keeps track of who infects whom. We examine this infection “tree” to see when COVID-19 was brought into a school population and if the source of the introduction was a student, teacher, or staff member. We also keep track of how many others get infected at school, and the number of exports to households and the community, all on this single branch of the tree. Figures come from analyzing all such branches, each one representing an importation event and/or outbreak.

Each data point is replicated 20 times with different underlying population files. Figures show the mean and 95% confidence intervals in the mean determined by bootstrapping across replicates and fitting splines to the resulting mean and variance trends. For the outbreak size analysis, the mean does not well characterize the distribution. Many of the outbreaks are very small, but sometimes they can be very large. This variance is driven in part by school size and type, but also individual-level heterogeneity in COVID-19 infections (some simulated people are more infectious than others). For this reason, we also display bubble charts showing the size of individual outbreaks.
References


