Reopening Schools in the Pandemic Did Not Increase Covid-19 Incidence and Mortality in Brazil

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ABSTRACT:

Background: School closures due to Covid-19 have left 1.6 billion students around the world without in-person classes for a prolonged period of time. To date, no study has documented whether reopening schools in developing countries during the pandemic causally increased aggregate Covid-19 incidence and mortality with appropriate counterfactuals.

Methods: We take advantage of the fact that 131 municipalities in São Paulo State, Brazil, reopened schools for in-person activities between October and December 2020, to estimate the causal effects of school reopening on municipal-level Covid-19 effective potential growth and deaths. We estimate treatment effects through a differences-in-differences strategy, comparing how outcomes evolved between municipalities that reopened schools and all others, before and after school reopening. We also estimate heterogeneous treatment effects by local characteristics. Last, we estimate the effects of school reopening on a local mobility index to shed light on the mechanisms behind its impacts on disease activity.

Findings: We find that school reopening did not increase Covid-19 incidence or mortality on average, up to 12 weeks after reopening. The counterfactual is critical for our conclusions: comparing only municipalities that reopened schools before and after reopening would lead us to conclude the opposite. Reopening schools did not affect disease activity even in poorer municipalities, in those with low-quality school infrastructure, with higher senior population share, or with higher baseline disease activity. We also find no effects of school reopening on the local mobility index.

Interpretation: While keeping schools open during the pandemic could still increase risks for school staff and students' families, our findings suggest that it did not contribute to the aggregate disease activity. This was the case not only because schools typically represent only a small fraction of the overall municipal population, but also because counterfactual mobility during the pandemic was already substantial even in the absence of in-person classes – making the marginal health benefits of keeping schools closed negligible in the aggregate.

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Background

In the absence of effective treatments for Covid-19, governments around the world resorted to non-pharmaceutical interventions aimed at reducing infections.[1] Among these interventions, school closures due to Covid-19 have left 1.6 billion students without in-person classes for a prolonged period of time[1-2]. The lion's share of those students are in developing countries, with limited access to devices and connectivity required to adequately learn remotely;[3] as such, the educational costs of school closures are potentially very large.[1-4] Beyond learning outcomes, school closures have also been shown to adversely affect children's and adolescents' well-being and development,[1] and to substantially increase the risk of dropouts.[5] As a result, seven million additional students worldwide are expected not to return by the time in-person classes resume.[3]

Despite all those costs, a survey with 370,000 respondents across 15 countries indicated that closing schools is the civil liberty citizens are most ready to forego in order to save lives in the pandemic.[4] That could be reasonable if in-person classes were a key contributor to Covid-19 incidence and mortality. On the one hand, there are reasons to believe that could be the case. First, recent evidence suggests that even though children are not systematically at high risk of infection from in-person classes, the likelihood that their families and school staff get infected increases significantly when schools are open.[6-7] Children infected with SARS-CoV-2 may be asymptomatic or have only mild symptoms, indistinguishable from common upper respiratory tract infections, allowing them to spread the virus even if they feel well; as a matter of fact, children are often key transmitters in other viral epidemics, like influenza. The risks might be especially high in developing countries, where robust mitigation measures are less likely to be in place at schools allowed to reopen during the pandemic.[8] Second, non-pharmaceutical measures, from lockdowns to restricting opening hours of bars and restaurants, have been shown to contribute to slowing down disease activity[9-15]. Schools were typically closed as part of those measures, as in-person classes concentrate more people (and for longer time periods) than most other establishments – features akin to super-spreader events.[16-19] In effect, prior studies have documented an association between school closures and reduced transmission of viral respiratory illnesses,[1] and school closures have been widely promoted as a leading mitigation strategy during pandemics.[2] Last, the mobility of primary caregivers is expected to increase when children are in school, potentially boosting transmission above and beyond the school setting.[20-21]

On the other hand, there are also reasons to believe that keeping schools open in the pandemic might not systematically contribute to Covid-19 incidence and mortality, especially in developing countries. First, there is no evidence that children are more likely to transmit SARS-CoV-2 than adults, unlike other respiratory viruses. In fact, studies suggest that children, particularly those at primary school, are among the safest groups for whom social distancing could be gradually foregone.[22] Second, many developing countries have refrained from imposing mobility restrictions a few months into the pandemic[19]; in particular, bars and restaurants were open for most of the time when schools were already closed.[23] Accordingly, data from cell tower triangulation attest that, in large cities like São Paulo, mobility was never below 50% even during the strictest periods of lockdown.[24] As such, reopening schools in the pandemic might have only small or no additional effects on disease activity relative to the counterfactual in which schools remain closed but mobility is basically unrestricted otherwise.

Determining whether in-person school activities contribute to aggregate Covid-19 incidence and mortality is, however, difficult: contrasting cases and deaths in municipalities that reopened schools, before and after reopening, will typically lead one to conclude that in-person classes increase disease activity – even if they, in fact, do not. The reason is that cases tend to subsequently increase in locations where they have been unusually low in the recent past, which, statistically, is more likely where in-person classes were allowed to return than elsewhere. For that very reason, merely comparing average Covid-19 incidence and mortality across locations that authorized schools to reopen to those that did not tends to over-estimate the effects of reopening during the pandemic. Undertaking such comparisons under appropriate counterfactuals is, hence, crucial. To our knowledge, no study to date has documented the aggregate effects of keeping schools open in developing countries during the pandemic with appropriate counterfactuals. The few studies that attempt to capture the causal aggregate effects of school reopening during the pandemic are based on data for developed country settings[24-25], with mixed results.

Brazil has been hard hit by the pandemic: as of March 2021, nearly 300,000 deaths had been attributed to Covid-19.[26] The country ranks 22nd in deaths per 1,000 inhabitants worldwide, in the upper-middle range of Covid-19 impacts among developing countries.[26] São Paulo State, the setting of our study, is the most populous in the country and, as such, also experienced the country's highest death toll: approximately 66,000 deaths as of March 2021.[23]São Paulo was also pretty typical in terms of its educational responses to the pandemic: it closed schools from March 16, 2020, and did not reopen them until September that year. Remote learning strategies were rolled out from April onwards, heavily based on broadcasting content on open television. The State's educational response in the pandemic was rated close to the median quality in the country; while roughly 90% of its 3.6 million students report to have studied daily during school closures, on average, they dedicated no more than 2.5 hours a day to school activities.[26] São Paulo State authorized municipalities to reopen schools for optional activities (remedial classes for students lagging behind, and extra-curricular activities such as psychological counselling) from September 8 to high-school students, and from October 7 to primary- and middle-school students. Regular in-person classes to high-school students were authorized to return from November 3. Municipalities had autonomy to decree whether schools could reopen, as long as safe reopening protocols were in place; in particular, all school staff head to wear personal protective equipment, alcohol had to be made available at the school gate, and attendance was limited (e.g. at 35% capacity in regions where the severity of the pandemic was high). The State Secretariat of Education estimates that 1,700 schools were in fact open for in-person activities and at least 2 million students did go to school during that period [27], especially as many public-school students rely on them for support beyond educational activities (e.g. school meals).

In this paper, we test the hypothesis that reopening schools to primary-, middle- and high-school students in the pandemic under appropriate safety protocols did not increase Covid-19 EPG and deaths in São Paulo State.

Methods

We rigorously test this hypothesis by taking advantage of the staggered school reopening in São Paulo State between October and December 2020. We draw on official data on municipal decrees to code which municipalities had reopened schools each week, between October 7 and December 17, 2020 (when the school year ended). Although the State had authorized municipalities to reopen schools for optional activities to high-school students since early September, very few had actually done so even by mid-October (see Figure 1). 131 out of the 645 municipalities in the State eventually reopened schools; as Figure 1 shows, reopening decisions were staggered.

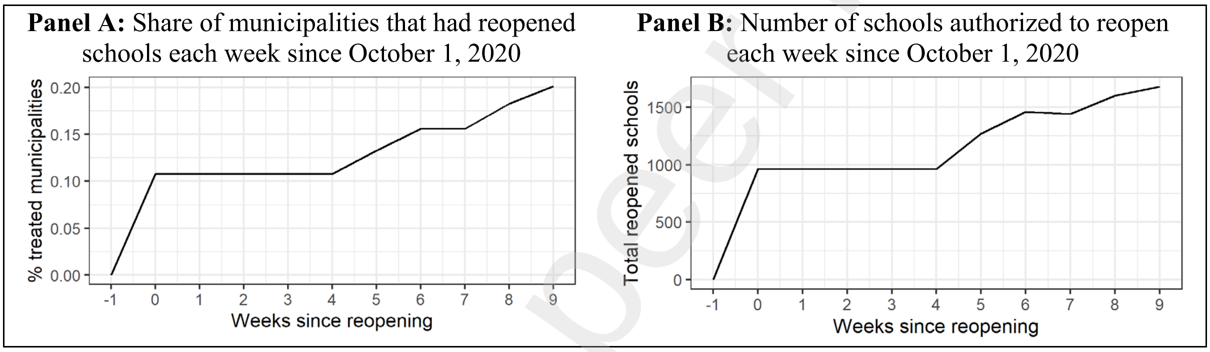


Figure 1 – Staggered school reopening in São Paulo State during the pandemic. Panel A showcases the share of municipalities in São Paulo State with decrees authorizing schools to resume in-person optional activities to middle- and high-school students each week. Panel B showcases the number of schools authorized to reopen each week, summing over all schools in those municipalities. Because decree data shared by the State Secretariat of Education starts on October 28, we need an imputation procedure to assign reopening status for the first 3 weeks of the October. In the main paper, we assume that all municipalities with an active decree by October 28 had already authorized school reopening by October 7. In supplementary materials (Table D3), we show that results are robust to the polar extreme assumption that no municipalities had authorized school reopening until October 28. In both cases, we assume no municipality had authorized schools to reopen before October; in effect, schools could not offer in-person activities to primary- and middle-school students until then. We drop two municipalities from our sample that authorized schools to reopen in October but later revoked their decrees.

For each cohort of municipalities that reopened schools (defined by the week at which their authorization decree was made effective), we estimate treatment effects by comparing the Covid-19 effective potential growth (EPG) and deaths between municipalities that reopened schools and those that did not, before and after in-person activities returned in the former. EPG is obtained by multiplying Covid-19's attack rate per 10^5 inhabitants over the last 14 days (i.e. the density of cases) by its an approximation of its effective reproduction number over the last 3 days (i.e. the growth rate of cases)[29]. Because in-person activities return at different weeks across cohorts, we first estimate cohort-specific average treatment effects, and then combine them by weighting each estimate by its relative group size when computing average treatment effects for the whole sample.[30] Because we do not have data on which schools actually reopened, we estimate intention-to-treat effects (ITT) based on municipalities' authorization decrees, through Ordinary Least Squares regressions.

We estimate the impacts of school reopening on municipal-level Covid-19 EPG and deaths, using publicly available data from municipal Health Secretariat and Brazil.io repository[31]. We restrict attention to data from August 7, 2020 – 2 months before the State authorized schools to reopen, to test for the absence of differential trends across treatment and control municipalities before in-person classes returned – until the end of 2020. Since EPG is not measured in easily interpretable units, we estimate treatment effects on (1) its percentile rank, capturing changes in municipality's position in the State ranking sorted by Covid-19 EPG (with positive effect sizes indicating a relative worsening of the local disease activity), and (2) the probability of displaying high EPG, equal to 1 if EPG is above the cutoff for the highest-level cluster (see supplementary materials), and 0 otherwise. When it comes to Covid-19 deaths, we estimate treatment effects on weekly deaths , and on cumulative deaths until the end of that week. We transform death counts to logarithms, such that effect sizes can be interpreted as percentual changes.

In all regressions, we control for municipal characteristics (income, population, number of schools, number of students per 1,000 inhabitants, and average quality of school infrastructure) from the 2010 Brazilian population census, and their baseline per capita Covid-19 cases and deaths (cumulative between January and September, immediately before school reopening). School infrastructure is the first principal component from a vector of school characteristics from the 2019 Brazilian school census (whether the school had a working kitchen, working bathrooms, trash collection, adequate sanitation infrastructure and access to drinking water, and its average class size).

We also compute risk diagrams, separately for municipalities that authorized schools to reopen at some point between October and December 2020, and for those that did not. Risk diagrams capture the progress of disease activity over time within municipalities in each group, relative to the recent state of the pandemic in the State.

Next, we estimate heterogenous treatment effects with respect to (a) baseline Covid-19 deaths; (b) average quality of school infrastructure; (c) per capita income; and (d) 65+ year-old population share. In each case, we split the sample according to the sample median, and focus on whether effects are statistically different from zero within the highest risk / most vulnerable sub-sample.

Last, we estimate treatment effects on municipality's average mobility index in that week. This outcome is publicly available from Google data on daily municipal mobility[32], based on cell phone location. The data is computed from the universe of Android phones in the municipality; for each phone, mobility is captured as the daily amount of time outside of home (inferred from phone location at sleeping hours). The outcome we have access to is an index, capturing changes in mobility relative to the municipal average in the week of February 15, 2020, measured in percentage points (see supplementary materials).

Results

Figure 2 starts by showcasing how disease activity progressed over time across treatment (Panel A) and control municipalities (Panel B), highlighting in both panels the 60 days immediately after the State authorized in-person classes to return. The risk diagrams showcase the value of counterfactuals: restricting attention to municipalities that eventually reopened schools, in Panel A, would lead us to incorrectly conclude that school reopening increased disease activity. Within treated municipalities, the attack rate and the recent growth rate of Covid-19 cases increased notably during the first 2 months since in-person classes were authorized to returned; in effect, average EPG even moved from lighter to darker shades (associated with higher EPG clusters) in the weeks following school reopening. Panel B, however, shows that the exact same trends in EPG take place in municipalities that did not authorize schools to reopen in 2020.

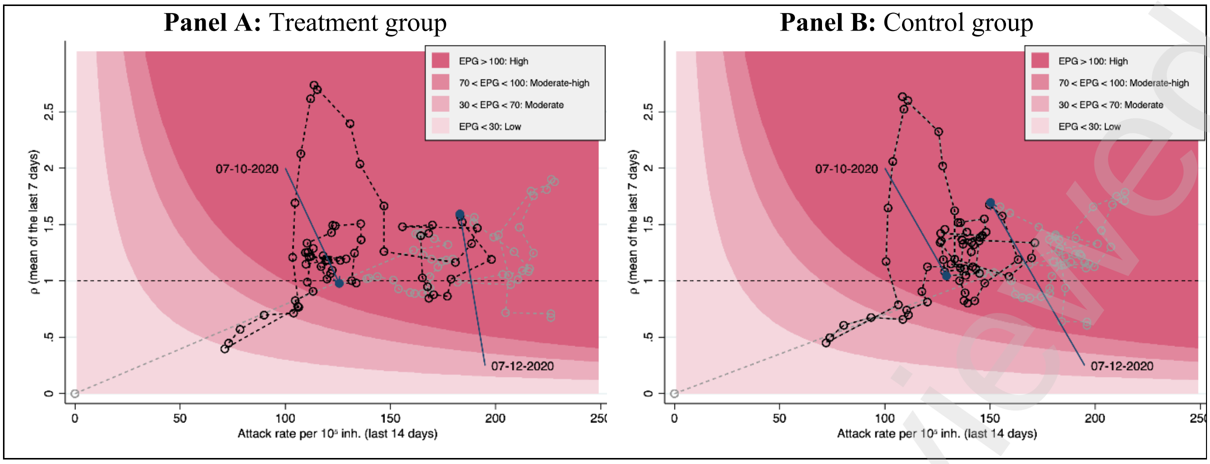
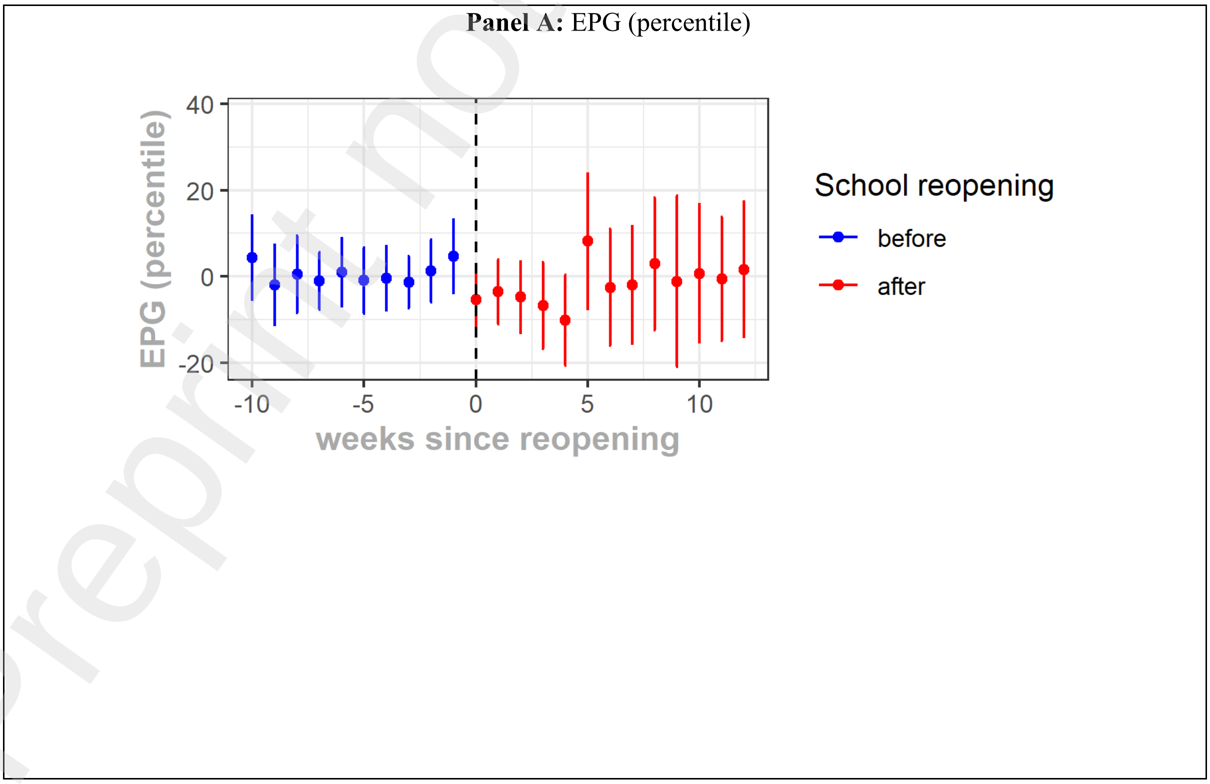


Figure 2 – Risk diagrams for municipalities that reopened schools and those that did not**.** Panel A presents the risk diagram for municipalities that authorized schools to reopen at some point between October and December 2020, while Panel B presents the risk diagram for those that did not. In both panels, diagrams display the 14-day attack rate (A_{14}) in the X-axis, and the average level of epidemic growth during the last week (ρ_7) in Y-axis. The value of A_{14} is obtained from the number of reported or estimated cases.[29]

Next, Figure 3 displays average treatment effects on EPG percentile rank (Panel A), probability of high EPG (Panel B), deaths (Panel C), and cumulative deaths (Panel D), estimated week-by-week through differences-in-differences. The figure also allows us to validate our identification assumption, conducting falsification tests by estimating average differences between the treatment and control groups week-by-week before school reopening. Reassuringly, no statistical differences between groups arise before reopening, and estimates are concentrated around zero – very precisely so for the two EPG measures and cumulative deaths. In the supplementary materials, Table D2 compiles cohort-specific treatment effects and average treatment effects of school reopening on those outcomes up to 12 weeks after the reopening, confirming that school reopening did not causally increase Covid-19 EPG or deaths in the aggregate. Results are more precise for the first weeks after reopening because all cohorts have data for those weeks, while only earlier cohorts have data later on.



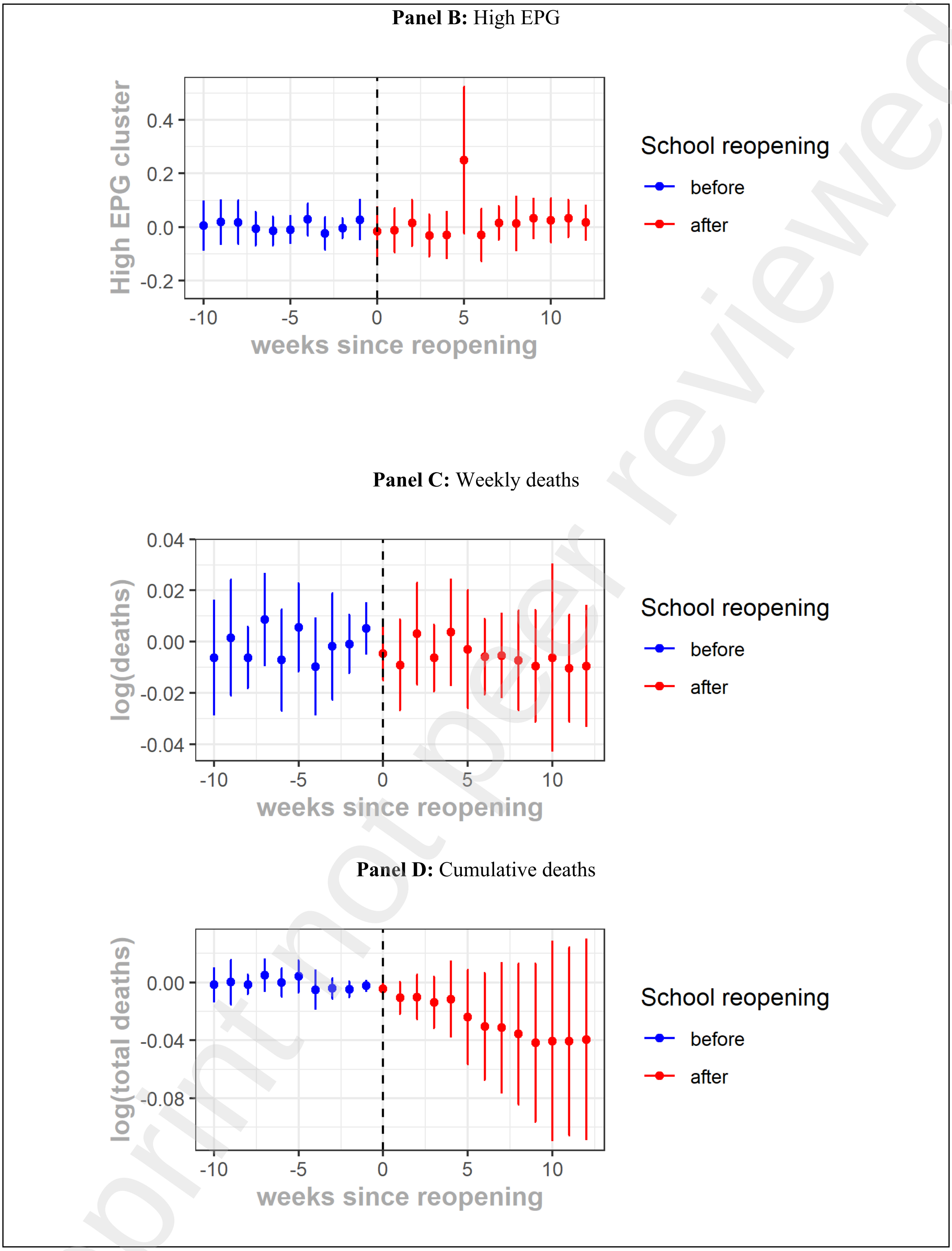


Figure 3 - Week-by-week average treatment effects estimated through differences-in-differences. This figure presents the average treatment effects[28] of school reopening on EPG percentile rank (Panel A), probability of high EPG (Panel B), deaths (in log; Panel C) and cumulative deaths (in log; Panel D). We aggregate treatment effects across all treatment cohorts at each week since school reopening. All panels show 95% confidence intervals. Falsification tests to validate our empirical strategy (week-by-week treatment effects estimated before actual reopening, to test for differential pre-trends before the onset of

in-person activities) are shown in blue. Treatment effects are shown in red. Table D3 in supplementary materials shows that results are robust to the assumption that no municipalities had authorized school reopening until October 28.

Last, Table 1 presents average treatment effects of school reopening on EPG percentile rank (Column 1), probability of high EPG (Column 2), deaths (Column 3), cumulative deaths (Column 4), separately for below- and above-median per capita income, quality of school infrastructure, senior population share, and baseline Covid-19 deaths. We find no systematic effects of school reopening even for the highest risk or most vulnerable sub-samples.

	EPG Percentile rank (1)	High EPG cluster (2)	log(weekly deaths) (3)	log(cumulative deaths) (4)
School infrastructure				
Below median	3.879	0.008	0.005	-0.005
	(2.707)	(0.021)	(0.003)	(0.008)
Above median	-3.718	0.031	-0.003	-0.022
	(3.057)	(0.028)	(0.005)	(0.016)
Per capita income				
Below median	-4.495	-0.023	0.002	-0.015*
	(3.329)	(0.029)	(0.003)	(0.006)
Above median	1.157	0.027	-0.001	-0.010
	(2.547)	(0.022)	(0.003)	(0.009)
65+ year-old share				
Below median	-0.831	0.006	0.001	-0.002
	(2.645)	(0.019)	(0.002)	(0.006)
Above median	0.311	0.019	0.003	0.011
	(3.531)	(0.016)	(0.006)	(0.010)
Baseline severity of the pandemic				
Below median	-8.421***	0.010	-0.001	-0.007
	(3.395)	(0.016)	(0.002)	(0.007)
Above median	1.003	0.042	-0.011	-0.028
	(4.809)	(0.044)	(0.008)	(0.016)

Table 1 - Heterogenous treatment effects estimated through differences-in-differences. This table presents average treatment effects of school reopening[30] on EPG percentile (column 1), High EPG (column 2), deaths (in log; column 3) and cumulative deaths (in log; column 4) estimated for different sub-samples. At the different rows, we split the sample according to the median of the following variables: quality of school infrastructure, per capita income, senior population share, and baseline disease activity. Treatment effects are calculated as the average of the treatment effects in each week (as in Figure 3) for all weeks after school reopening. Bootstrapped standard errors clustered at the municipality level in parentheses. *p<0.05, **p<0.01, ***p<0.001. Table D3 in supplementary materials shows that results are robust to the assumption that no municipalities had authorized school reopening until October 28.

In supplementary materials, Table D2 also documents average treatment effects on the afore-mentioned outcomes, and on the local mobility index. School reopening is associated with a 1 percentage point higher mobility, a small effect that is not significantly different from zero at conventional statistical levels. Most importantly, supplementary materials show that local mobility was already increasing dramatically before school reopening across all municipalities. Strikingly, mobility reached pre-pandemic levels by late November 2020, even in the control group.

Last, Table D4 reports differences-in-differences estimates for Covid-19 cases among 6-17-year-olds, documenting that school reopening also did not increase incidence even in school-aged children, and even when using Covid-19 cases among young adults within each municipality as an additional counterfactual.

Discussion

We find that reopening schools during the pandemic did not systematically affect Covid-19 EPG or deaths in São Paulo State. Average treatment effects are not statistically significant at any week after reopening, for any of the outcomes. This is not because we lack statistical precision: effect sizes on EPG percentile rank and the probability of high EPG are nearly zero in most weeks and, if anything, average treatment effects on deaths are even negative, although small and not statistically different from zero at conventional significance levels. We show that school reopening did not significantly affect disease activity even in municipalities with low-quality school infrastructure, low per capita income, high senior population share, or most severely affected by the pandemic. For the latter, we can rule out with over 95% confidence that school reopening moved municipalities one or more positions up in the Covid-19 EPG State ranking.

Extrapolating conclusions from studies conducted in rich countries about the impacts of school reopening during the pandemic might be inappropriate, for two main reasons. On the one hand, in developing countries, schools might be unable to properly implement safe reopening protocols, which might be key to mitigating the risk of accelerating the pandemic[8]. If that is the case, reopening schools in developing countries during the pandemic potentially represents a higher risk of increasing Covid-19 incidence and mortality. On the other hand, in developing countries, mobility during the pandemic might be much higher than in developed countries even in the absence of in-person classes. If that is the case, reopening schools during the pandemic in those countries might actually represent a lower risk of increasing aggregate disease activity. Evidence from the studies conducted in developed countries that we reviewed is mixed[24-25], and their empirical strategy can only estimate short-term effects of school reopening on disease activity (2-3 weeks after in-person classes returned). In contrast, we estimate the short- and medium-term causal effects of school reopening on Covid-19 endemic potential growth and deaths in a typical developing country setting, up to 12 weeks after in-person classes returned. Taken together, this work suggests that reopening schools in developing countries during the pandemic is unlikely to contribute to aggregate risk in the presence of safe reopening protocols.

Even in developing country settings, most studies only analyze risks within the school community, rather than at the aggregate[19,33], and none of them estimates effects under appropriate counterfactuals. Studies that estimate the effects of school reopening by simply comparing cases within municipalities, before and after in-person activities returned, are likely to detect false positives. The reason is regression to the mean – the statistical tendency for negative (positive) shocks to be followed by positive (negative) ones[34] –; concretely, locations that allow schools to re-open are statistically more likely to have experienced unusually low Covid-19 cases before in-person classes returned. In effect, our risk diagrams show that restricting attention to municipalities that reopened schools would have led us to incorrectly conclude that reopening increased disease activity in those municipalities. Analogously, merely comparing average Covid-19 incidence and mortality across locations that authorized schools to reopen to those that did not would tend to over-estimate the effects of reopening during the pandemic; such locations typically differ in many dimensions, particularly when it comes to their previous trends for Covid-19 cases, hospitalizations and deaths. Analyzing a comparison group with similar pre-trends for Covid-19 EPG and deaths is a critical element of study design to assign a causal interpretation to empirical findings.

The null effects that we estimate, even for the most vulnerable municipalities (low income and low-quality school infrastructure) or those with the highest exposure to risks of Covid-19 (high senior population share and high baseline disease activity), are not because schools did not actually reopen regardless of municipal decrees, or because, where they did, students did not actually come to school: as it is typical in developing countries, public schools play a bigger role in their communities, supporting students' livelihoods with meals and providing emotional and psychological support[17]; accordingly, the State Secretariat of Education estimates that about 2 million students did come to school. School reopening in the pandemic did not increase municipal-level Covid-19 incidence or deaths not only because school communities represent a small fraction of the overall population, but also because counterfactual mobility during the pandemic was already substantial even without in-person classes, making the marginal health benefits of keeping schools closed negligible in the aggregate.

We also show that school reopening did not increase Covid-19 cases even among municipalities' school-age population, relative to the counterfactual. Having said that, reopening schools in the pandemic could still pose risks for school staff and students' families – especially if robust protocols to prevent infections at the school setting are not in place. Additional research is needed to document the direct impacts of school reopening on those populations with appropriate counterfactuals. That would require drawing on individual-level data on Covid-19 cases, hospitalizations and deaths for students, school staff, and their families, both for schools that reopened and for those that did not, parsing any differences in previous trends in disease activity across them. Documenting the joint distribution of educational gains and health costs from keeping schools open in the pandemic is a necessary step to inform citizens and policy makers about the trade-offs involved in those decisions, as the Covid-19 pandemic is expected to linger for the foreseeable future, especially in developing countries. Complementary approaches, from seroprevalence surveys[35-36] to identifying which variants of the virus are circulating in these settings[37], are also needed to further inform risk assessments in this context.

Contributors

GL, CAB, JC and OLN take responsibility for the integrity of the data and the accuracy of the data analysis. GL and OLN decided to publish the paper. CAB was responsible for compiling the data. GL, CAB and OLN drafted the manuscript. GL, CAB and OLN contributed to statistical analysis. CAB led data management. GL, CAB, JC and OLN critically revised the manuscript. All authors had full access to all the data in the study and had responsibility for the decision to submit for publication.

Declaration of interests

GL and OLN received fees from the Inter-American Development Bank (IADB) for the design of this study. JC is an IADB staff member. CAB declares no competing interests.

Data sharing

After the publication of this study, the authors will make all data and statistical code used in this manuscript and supplementary materials publicly available at the website https://www.ssrn.com/index.cfm/en/the-lancet/.

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Research in context

Evidence before this study

We searched PubMed for research articles published from database inception until March 2, 2021, with no restrictions on language using the terms "SARS-CoV-2" and "school reopening." We also searched the main Economics journals for articles that implement quasi-experimental methods to evaluate the effects of school reopening on health outcomes. So far, the evidence of schools closures is mixed. Some studies pointed to a potential acceleration of cases among school-age individuals, but others failed to find any significant effect of reopening on cases. All studies except two lacked clear counterfactuals, and provided only descriptive evidence of the progression of cases.

Added value of this study

This is one of the first studies to implement a quasi-experimental design to evaluate the aggregate effects of school reopening during the SARS-CoV-2 pandemic, and the first one in the context of developing countries. Our institutional background is closer to the ideal experiment and allows some important extensions to the previous literature. First, we are able to compare municipalities that reopened schools and those that did not, and to conduct a range of falsification tests to rule pre-existing differences across them before in-person school activities returned. Our setting also allows examining effects over a much longer horizon than previous studies. As we are able to estimate the effects of school reopening up to 12 weeks after in-person activities return, we can examine its effects on the number of Covid-19 deaths, not only on reported cases.

Implications of all the available evidence

Our results imply that reopening school in developing countries during the pandemic is unlikely to affect the aggregate number of cases or deaths in the presence of safe reopening protocols. This finding also holds even for municipalities most severely affected by the pandemic, with a higher share of population at risk, or where the quality of school infrastructure is subpar.

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Supplementary materials

Reopening Schools in the Pandemic Did Not Increase Covid-19 Incidence and Mortality in Brazil

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Appendix A: Data description

Appendix A1: General description

In this paper, we combine information from several sources. We summarize these sources in Table A1:

Table A1: Summary of data sources

Name	Source	Year	Aggregation level
Population projections	IBGE	2019	Municipality
Census data	IBGE	2010	Municipality
School census	INEP	2019	Municipality
Mobility reports	Google	2020	Municipality-week
Covid cases microdata	SUS	2020	Municipality-week
Pandemic Evolution	Brasil.io	2020	Municipality-week
Schools status	SEDUC	2020	Municipality-week

In Table A2, we provide a description of all variables used in the paper:

Table A2: Variable description

Variable	Description	Source
Panel A: Dependent variables		
Log(deaths)	log of weekly new deaths plus one	Pandemic evolution
Log(accumulated deaths)	log of accumulated deaths plus one	Pandemic evolution
EPG percentiles	see separate description	Pandemic evolution
EPG (cluster)	see separate description	Pandemic evolution
Cases for school-age youngsters	weekly cases for individuals with 13 to 18 years-old per-thousand	Covid cases microdata
Mobility	see separate description	Mobility reports
Panel B: Other variables		
Treatment	municipality reopened of schools	School status
Income	average household per capita income	Census data
Population	municipality projected population in thousands of individuals (2019)	Population projection
Number of schools	total number of schools in the municipality	School census
Number of students	total number of students per thousand in the municipality	School census
School infrastructure	see separate description	School census

Appendix A2: Specific variable description

Three important variables in the paper require a lengthier discussion: endemic potential growth (EPG), mobility school infrastructure.

EPG

EPG measures the growth of the pandemic at any point in time. In order to generate it, we first calculate for every day the propagation velocity of each municipality. Let c_{mt} be the number of notified cases in municipality m and day t. Then, the propagation velocity is given by:

$$\rho_{mt} = \frac{\sum\limits_{s=t-1}^{t+1} c_{ms}}{\sum\limits_{s=t-6}^{t-4} c_{ms}}$$

Next, we approximate the total number of infectious individuals in a certain municipality as the number of total cases notified in the last fourteen days, that is:

$$A_{mt} = \sum_{s=t-14}^{t-1} c_{ms}$$

Then, the EPG is defined as:

$$EPG_{mt} = \rho_{mt} * A_{mt}$$

However, there are three potential problems with this definition. First, sometimes negative cases are registered to correct for previous mistakes in the data. This would generate negative values for the EPG. Thus, we ignore any negative values in the data.

Second, the variable is not defined when $\sum_{s=t-6}^{t-4} c_{ms} = 0$, which would force us to drop all occurrences of this problem (approximately 2% of the sample). To correct this, we divide by one instead of zero in those instances.

Let $\tilde{c}_{mt} = \max\{0, c_{mt}\}$. Then, we calculate a corrected version of the variable as:

$$E\tilde{P}G_{mt} = \frac{\sum\limits_{s=t-1}^{t+1} \tilde{c}_{ms} * \sum\limits_{s=t-14}^{t-1} \tilde{c}_{ms}}{\max\left\{ \sum\limits_{s=t-6}^{t-4} \tilde{c}_{ms}, 1 \right\}}$$

Finally, the third problem is that this measure is sensible to clogged cases. If cases are not reported for a few days, and the notification of accumulated cases is made after that, the numerator of the propagation velocity variable tends to be very high, and its numerator tends to be zero (which we replace by one). Therefore, the EPG variable has very high values in these cases. To avoid the presence of outliers, we replace EPG's with values greater than 10,000 as the EPG value of the previous day.

Once we calculated the EPG and corrected for outliers, we average these variables at the municipality-week level. Treatment effect on EPG does not necessarily have a direct interpretation, so we compute two dependent variables of interest:

1. **EPG (percentile)**: For each day of the sample, we compute the EPG percentiles for every municipality. Therefore, this variable indicates the ordering of each individual municipality on that specific day.
2. **EPG (cluster)**: We implemented the Hartigan-Wong clustering algorithm that divides municipalities into three levels according to the size of the EPG. Our dependent variable is a dummy variable for municipalities in the cluster with more severe endemic propagation (the cluster's inferior limit is an EGP of 1296).

Mobility

Our variable of mobility is based on Google reports. This is calculated using mobile-phone GPS information. Google calculates daily mobility information for more than 400 municipalities in the São Paulo state. We have information for approximately 78% of the treated municipalities and 56% of the control municipalities.

Google provides information for different types of mobility, including: 1) retail and recreation; 2) grocery and pharmacy, 3) parks, 4) transit stations; and 5) workplaces. We average these five mobilities to generate an aggregate one. The data started being collected on February 15 of 2020. Then, information is provided as the mobility percentage point deviation from the baseline.

School infrastructure

There are several potentially different ways to measure the school infrastructure, which tend to be correlated. We opt to build an aggregate measure using the principal component method. We select several different measures of school infrastructure that are available in the school Census of 2019, and that might be relevant to the degree of propagation of the disease in the school environment.

We select the following variables: the availability of a bathroom, school staff's bathroom, shower, kitchen, garbage collection, piped water, basic sanitation, and the average number of students per class.

We implement the principal component method and select as our aggregate school infrastructure measure the first estimated component. This first component accounts for approximately 40% of the total variance of the selected variables.

Appendix B: Methodological details

Appendix B1: Estimation method

To estimate the effect of school reopening on the outcomes of interest, we rely on the difference-in-difference approach. That is, we compare the trends of the dependent variables for treated and non-treated municipalities. However, recent literature suggests that a straight-forward two-way fixed-effect regression is not appropriate in the context of an application with multiple periods and staggered treatment (GOODMAN-BACON, 2018).

To circunvent this problem, we implement Callaway and Sant'Anna (2020) estimator. First, we divide the treatment group into cohorts according to the week schools reopened schools. Then, we estimate a cohort-time treatment effect by:

$$\delta(g,t) = \mathbb{E}\left[\left(\frac{G_{gm}}{\mathbb{E}(G_{gm})} - \frac{\frac{\hat{p}(X)*C}{1-\hat{p}(X)}}{\mathbb{E}\left[\frac{\hat{p}(X)*C}{1-\hat{p}(X)}\right.}\right) * (Y_t - Y_{t-1})\right]$$

where g is the cohort of treatment, t is the relative week, G_g is a dummy for municipality being treated and in cohort g, C is a dummy for the never treated control group, $\hat{p}(X)$ is a propensity score estimate based on covariates X and Y is the outcome of interest. We include the following variables as controls: per capita income, number of students, school infrastructure and the number of baseline cases and deaths.

We provide different aggregation procedures. First, we aggregate (Table 1 of the main text) at the cohort level. This is simply the average treatment effects for each cohort. We also aggregate (Figures 1 to 4) the treatment effects at the relative time level. To do that, we weight the average by the relative size of each cohort. Finally, we aggregate all treatment effects as the simple average of the treatment effects in each period.

We calculate standard-errors using Callaway and Sant'Anna (2020) standard procedure. This is done by block-bootstraping standard-errors using at the municipality level. The block-bootstrap procedure is necessary to allow arbitrary correlations between regression errors for the same municipality at different points in time.

Appendix B2: Closest neighbor matching

As a robustness check, we also implement a nearest neighbor propensity score matching. We implement the procedure sequentially, starting the process by considering a sample consisting of the first cohort and the control group (never treated municipalities). We estimate a Probit model as:

$$G_{gm} = \boldsymbol{\beta} \boldsymbol{X}_m + \epsilon_m$$

Then, we calculate the propensity score for this sample as the predicted value from the estimate above. We include the following variables as controls: per capita income, population, number of students, school infrastructure, and the number of baseline cases and deaths.

For each municipality in the treated cohort, we find the municipality in control with the closest propensity score without replacement. After we match all municipalities in the first cohort, we build a new sample, including the second cohort of treatment and the control group without the municipalities matched in the first step of the process.

Then, we re-estimate the Probit model for this alternative sample and match all municipalities in the second cohort. We repeat this procedure until we find a match to all cohorts of treatments.

We implement this sequential algorithm for two alternative samples. The first one is the full sample of the study, including all 644 São Paulo municipalities. As discussed in Appendix A, we do not have mobility data for all municipalities. Thus, we implement this procedure again for the alternative sample with available information on mobility.

Appendix C: Descriptive statistics

Appendix C1: Full sample

In Table C1, we present descriptive statistics separately for ever and never treated municipalities. The number of new and accumulated cases and deaths is calculated at the last week of September:

Table C1: Descriptive statistics in the baseline (end of September)

	Never treated	Ever treated
New cases per thousand	0.79	0.76
New deaths per thousand	0.03	0.02
Accumulated deaths per thousand	0.44	0.49
Income per capita	674.12	804.85
Population (thousands)	38.77	200.37
Number of schools	19.48	67.94
Number of students (thousands)	7.25	34.34
School infrastructure	-0.013	-0.003
Municipalities	514	129

Next, in Figures C1 to C4, we show trends in the dependent variable for each group separately:

Figure C1: Trends in EPG (percentile)

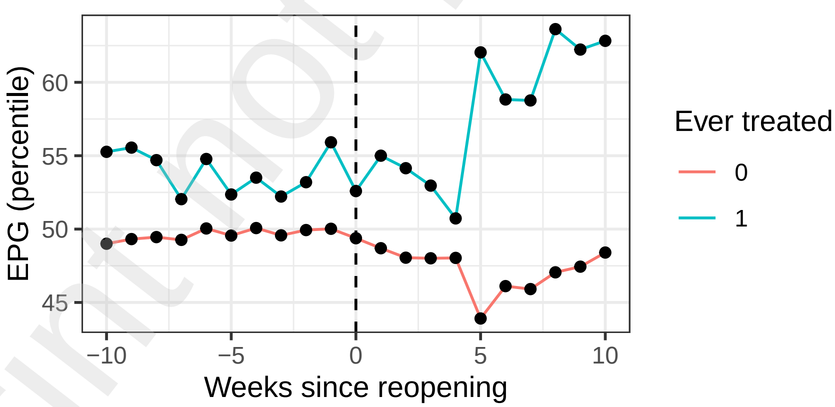


Figure C2: Trends in high EPG cluster

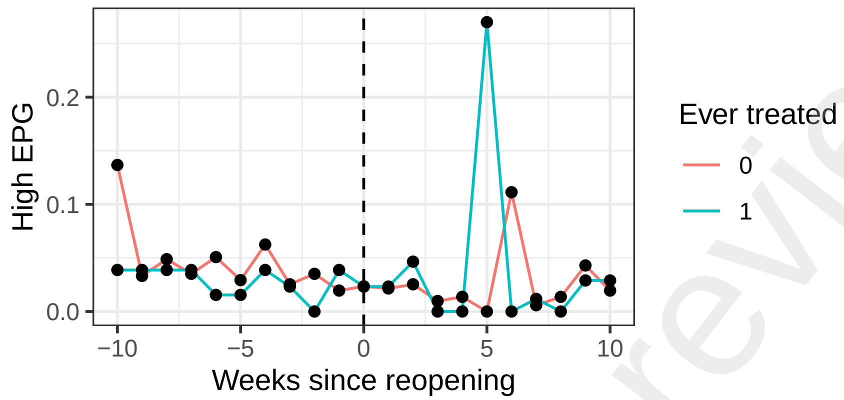


Figure C3: Trends in log(deaths)

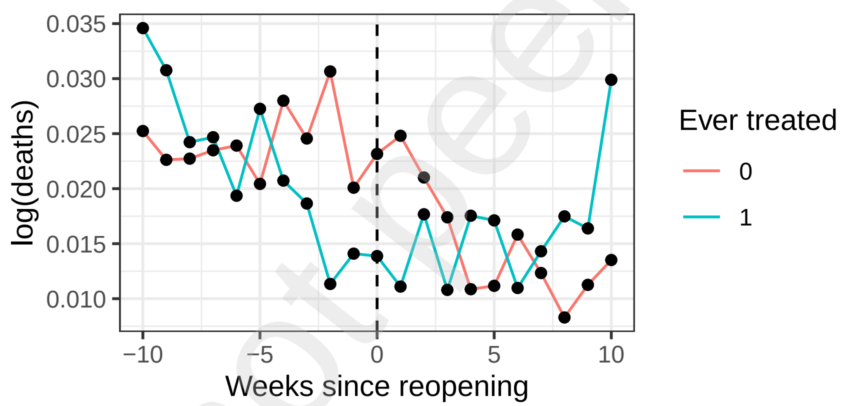
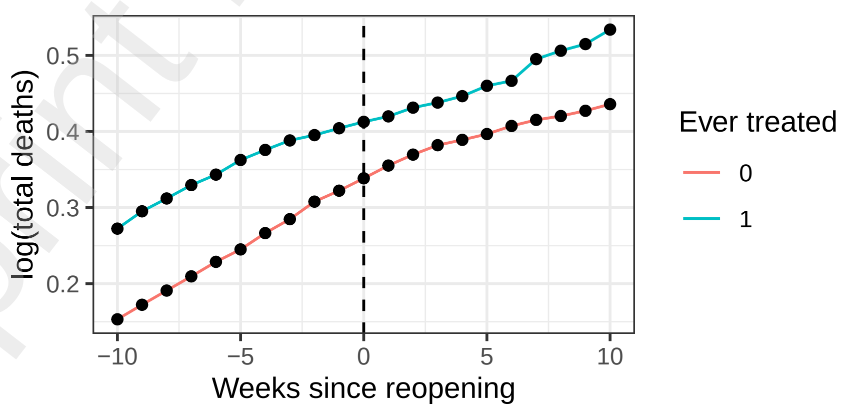


Figure C4: Trends in log(total deaths)



Appendix C2: Matched sample

Now, we present in Figures C5 to C8 the trends in the variables of interest for the treated group and the matched control group. We include sample standard-errors only for the average in the control group since we still present the trends for the universe of ever treated municipalities.

Figure C5: Trends in EPG (percentile) with matched control group

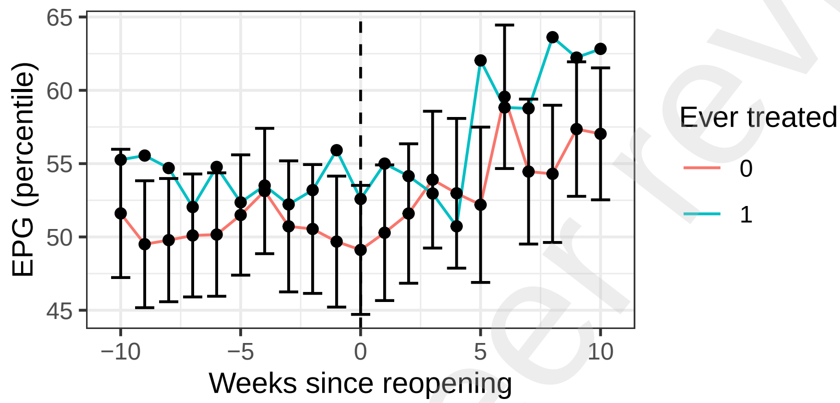


Figure C6: Trends in high EPG cluster with matched control group

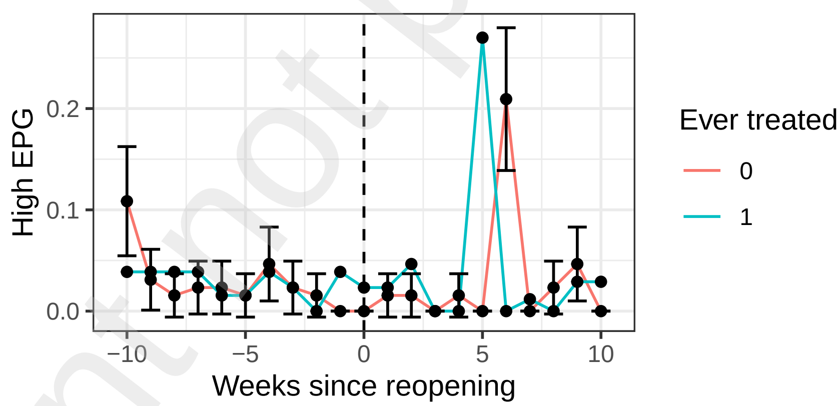


Figure C7: Trends in log(deaths) with matched control group

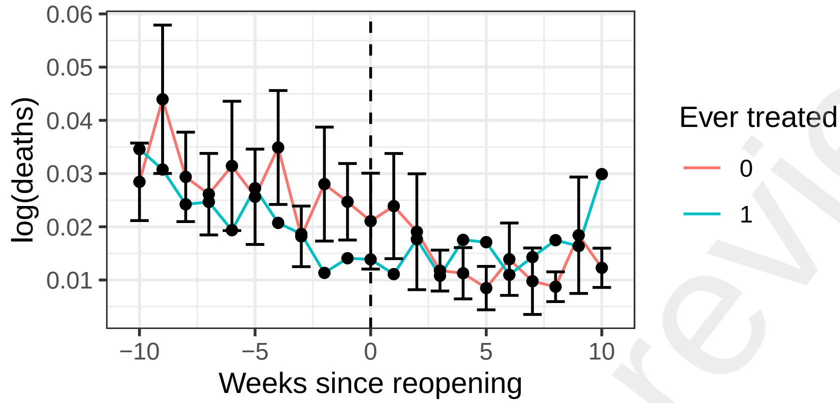
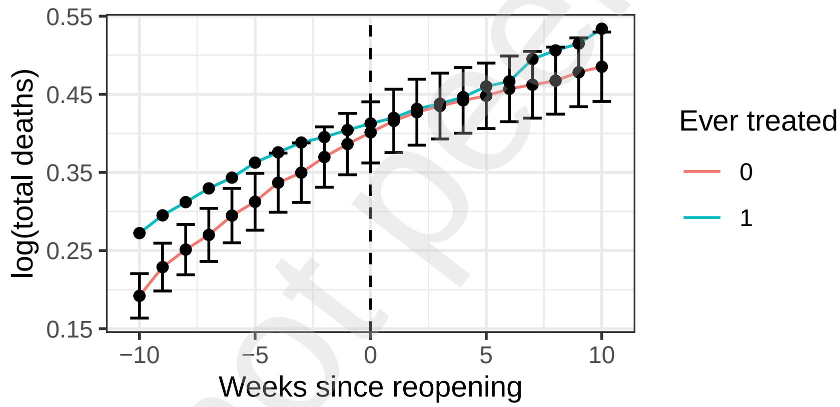


Figure C8: Trends in log(total deaths) with matched control group



Appendix D: Supplementary results

Appendix D1: Additional results

In Table D1, we show naive estimates of the school reopen. In the first panel, we compare ever treated municipalities in the weeks before and after reopening. In the second one, we compare ever treated and the never treated group. In both panels, we find negative adverse effects of the school reopening on the pandemic for almost all variables. However, these results are likely to be driven by the pandemic's evolution and differences between the groups and not the policy implementation itself. These naive results illustrate the importance of the counterfactual approach considered in the main text.

Table D1: Naive comparisons (not interpreted to be causal)

	EPG (percentile)	EPG (cluster)	log(deaths)	log(total deaths)
Panel A: Pre-post comparison				
Treatment effect	10.542***	0.053***	0.001	0.320***
	(1.051)	(0.017)	(0.001)	(0.007)
Panel B: Treatment x control comparison				
Treatment effect	9.377***	0.060***	0.001	0.059***
	(0.919)	(0.015)	(0.001)	(0.008)

Table D2 compiles cohort-specific treatment effects and average treatment effects of school reopening on EPG percentile rank (Column 1), probability of High EPG (Column 2), deaths (Column 3), accumulated deaths (Column 4), and the mobility index (Column 5), estimated through differences-in-differences. We find that school reopening did not systematically affect Covid-19 EPG or deaths up to 12 weeks after the reopening: average treatment effects are not statistically significant in the aggregate for any of the outcomes. If anything, the average effect on EPG percentile rank and deaths is actually negative, while that on the likelihood of being featured above the 92th EPG percentile rank is nearly zero. The table also showcases the value of our estimation technique: there is substantial variation in cohort-specific estimates . Last, school reopening is associated with a 1 percentage point higher mobility, a small effect that is not significantly different from zero at conventional statistical levels.

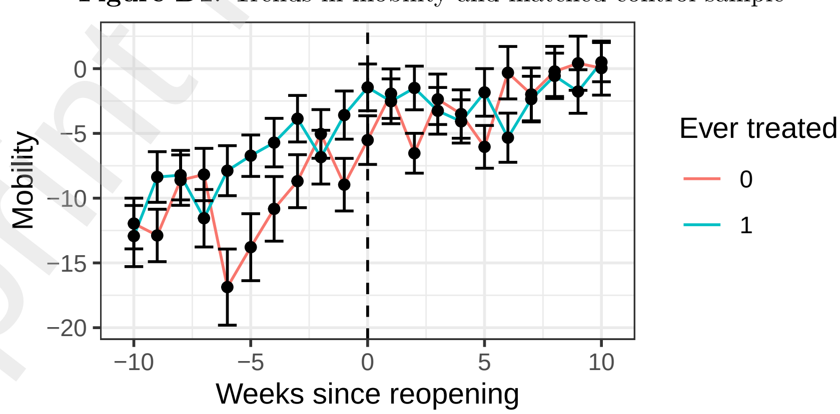
Table D2: Difference-in-difference treatment effects aggregated by group

Cohort treatment	EPG (percentile)	EPG (cluster)	log(deaths)	log(total deaths)	mobility
0	2.224	0.050*	-0.009	-0.032***	1.928
	(3.543)	(0.020)	(0.006)	(0.013)	(1.116)
5	-10.526	-0.059	0.015	0.022	-0.023
	(6.183)	(0.080)	(0.020)	(0.028)	(1.889)
6	-21.027***	-0.202	0.002	-0.017	0.112
	(11.577)	(0.188)	(0.004)	(0.011)	(1.473)
8	-17.990***	0.023	0.001	0.010	4.761***
	(7.291)	(0.032)	(0.007)	(0.009)	(1.440)
9	-1.241	0.068	-0.008	-0.020	0.006
	(5.712)	(0.053)	(0.007)	(0.012)	(2.644)
Aggregate TE	-5.047	0.006	-0.003	-0.017	1.005
	(2.903)	(0.029)	(0.004)	(0.009)	(1.015)

In order to shed some light on this question, we can examine some mechanisms through which school reopening might affects the evolution of the pandemic. As discussed in the main text, the reopening effects might generate negative effects by: 1) increasing the mobility and 2) generating agglomerations in schools. We evaluate the reduced-form of the policy on the first potential mechanism.

We show (In Figure D1) illustrative evidence that the reopening does not affect the trends in the mobility of the municipalities that reopened schools. This evidence suggests that, conditional on other activities being allowed, school reopening does not dramatically increase circulation of individuals. The fact that we do not find significant aggregate effects also suggests that disease transmissions that might occur in the school environment are not numerous enough to generate aggregate affects at the municipality level.

Figure D1: Trends in mobility and matched control sample



Appendix D2: Robustness checks

Now, we consider two variations in our estimates to test the sensibility of our main results to two different decisions underlying them. First, we do not know exactly when municipalities in the first cohort of treatment reopened schools. As stated in the main text, schools reopened on October 7, but the São Paulo state secretary did not keep track of municipalities that reopened schools until the fourth week of October. After that, they kept a weekly record of the municipalities that reopened and those that did not.

Thus, we know which municipalities reopened schools until the fourth week of October but not exactly when each of them reopened schools. In the main text, we assumed that all municipalities reopened schools in the first week of October and that no school reopened after that. The second important decision was to exclude EPG outliers from the sample, as discussed in Appendix A2.

In Table D3, we show that our main conclusions are dependent on these decisions. We test the sensibility of the results first to the first decision by making the radically opposite hypothesis, that is, that no school reopened on the first week of October and that all municipalities in the first cohort reopened schools in the fourth week. To test the sensibility to the second decision, we present our main results without excluding outliers. We can see that in both robustness checks our results barely change from the main ones.

Table D3: Robustness checks

	Sample with outliers	Schools opening later
EPG (percentile)	-2.786	-3.272
	(2.463)	(2.766)
EPG (cluster)	-0.002	-0.003
	(0.027)	(0.026)
log(deaths)		0.003
		(0.004)
log(total deaths)		-0.008
		(0.010)

Next, in Table D4, we evaluate whether the school reopening affected school-age children. We estimate a triple-difference strategy, where we not only compare treated and control municipalities but also the trends in cases for school-age children (ages 7-18) and youngsters (ages 19-22).

Table D4: Treatment effects for school-age children

	Cases school-age children	
Relative week of reopening	Difference-in-difference	Triple-difference
0	0.033	0.029
	(0.020)	(0.033)
5	-0.008	0.002
	(0.022)	(0.003)
6	-0.019	-0.010
	(0.020)	(0.018)
Aggregate TE	0.014	0.011
	(0.013)	(0.012)

Finally, we show in Table D5 and additional robustness check. We show our main difference-in-difference estimates only for the matched sample (discussed in Appendix B2) in order to be sure that our results are not driven by different group characteristics.

Table D5: Difference-in-difference treatment effects (matched sample)

Cohort treatment	EPG (percentile)	EPG (cluster)	log(deaths)	log(total deaths)	mobility
0	1.818	0.009	0.001	-0.009	1.227
	(2.693)	(0.008)	(0.002)	(0.008)	(0.697)
5	-14.284*	-0.070	0.009	0.020	-1.180
	(7.059)	(0.077)	(0.010)	(0.023)	(1.141)
6	-10.670**	-0.045	0.002	-0.010	-1.136
	(5.063)	(0.113)	(0.004)	(0.006)	(1.192)
8	-19.919***	0.041	-0.007	0.001	
	(8.280)	(0.040)	(0.010)	(0.016)	
9	0.470	0.068	-0.006	-0.016	-1.372
	(5.524)	(0.055)	(0.007)	(0.013)	(3.028)
Aggregate TE	-4.621	0.002	0.001	-0.005	0.516
	(2.538)	(0.017)	(0.002)	(0.006)	(0.867)