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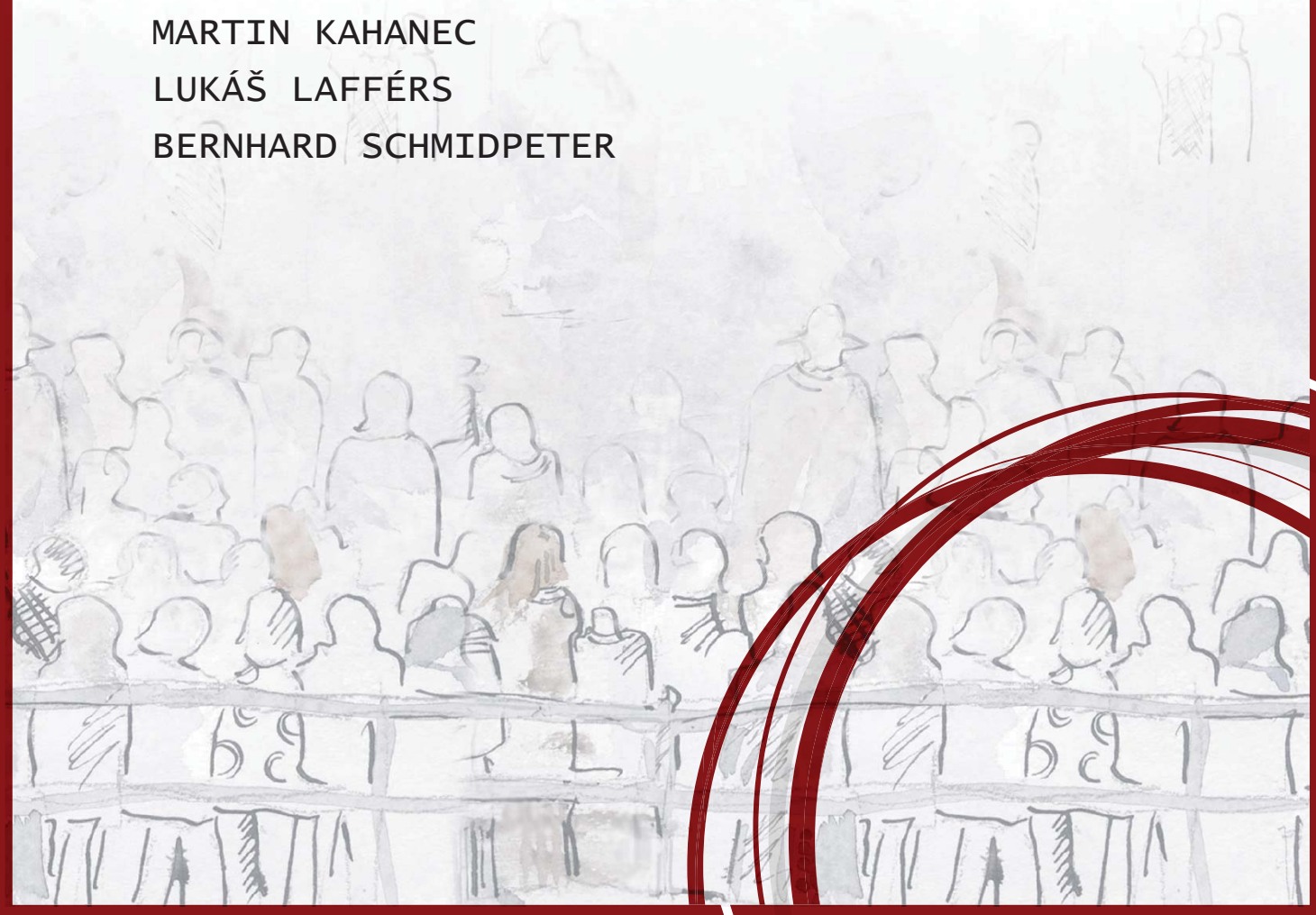

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The Impact of Mass Antigen Testing for COVID-19 on the Prevalence of the Disease*

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Abstract

More than a year since the first outbreak in China in December 2019, most countries are still struggling to contain the COVID-19 pandemic. Mass antigen testing has been proposed as an instrument to mitigate the spread of the disease and allow the economy to re-open. We investigate the potential benefits of mass antigen testing for the mitigation of the pandemic, using data from a uniquely designed testing that took place in Slovakia in autumn 2020. As the first country in the world, Slovakia implemented and repeated mass rapid antigen testing. After the first round of nation-wide testing, only districts above an ex-ante unknown prevalence threshold were re-tested. Comparing districts in the neighborhood above and below the threshold using a quasi-experimental design, we find that repeated mass antigen testing reduces infections by about 25-30% and results in a decrease in R_0 of 0.3 two weeks after the testing. These effects peaked about 15 days after the second round of testing and gradually dissipated afterward. These results suggest that mass testing could be an effective tool in curbing the spread of COVID-19, but for lasting effects it would need to be conducted regularly in relatively short intervals.

JEL Codes: D04, I18, J22

Keywords: COVID-19, COVID-19 policies, Causal impact, Antigen testing, Mass testing, Non-pharmaceutical interventions

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1 Introduction

More than one year after the first documented case of COVID-19, most countries are still struggling to contain this highly contagious disease. According to data provided by John Hopkins University, more than 100 million people around the world have been infected and more than 2 million people have died of COVID-19 as of January 25, 2021.[1] To protect their most vulnerable citizens and to slow down the spread of the disease, many governments have imposed strict policy measures such as requiring social distancing, stay-at-home orders, as well as local and nation-wide lock-downs (e.g. Chernozhukov, Kasahara and Schrimpf, 2020). While there is evidence that some of these policies have been successful in at least slowing the numbers of infections, they also have directly and indirectly affected labor supply and demand, investment, consumption, expectations and other economic variables, taking a heavy toll on economies. World GDP is projected to fall more than 4% in 2020 and the decline in GDP is even more pronounced in advanced economies (IMF, 2020). Economic distress caused by the applied policy measures has also affected broader aspects of individuals' lives and well-being by, for example, increasing domestic violence (Arenas-Arroyo, Fernandez-Kranz and Nollenberger, 2020). Facing such detrimental effects on both the economy and society, and with the prospect of widely accessible vaccination still distant – especially for low-income countries –, policy makers have been looking for economically less damaging alternative ways of containing the pandemic.[2] Mass testing for COVID-19 as a potential tool of containing the pandemic has received particular attention. Regional and local mass testing has been carried out in several countries such as the UK, China, South Korea, Austria, Luxembourg, and Slovakia. Evidence on whether and how mass testing can work to curb the spread of COVID-19 is scant, however, there are also few guidelines as to how effective single mass testing events can be in comparison to alternative testing strategies. Informing policy makers on the question whether mass testing can be an effective and cost-efficient way of re-opening the economy is an urgent call.

Proponents of mass testing maintain that it indeed is a cost-effecient policy for identifying and quarantining potentially infectious individuals. This would in turn help to reduce cases and the spread of the diseases (e.g. Pavelka, Van-Zandvoort, Abbott, Sherratt, Majdan, CMMID COVID-19 working group, Jarcuska, Krajci, Flasche and Funk, 2020) and, as a result, may enable policy makers to cautiously open shops, restaurants, and services. Opponents of mass testing argue that it can create a false sense of security and may lead individuals to behave less carefully; see the discussion in Mahase (2020). Cheaper rapid antigen (Ag) test often used in mass testing events are also not as sensitive

[1] The data was taken from https://coronavirus.jhu.edu/.

[2] Many countries also build on a mass Covid vaccination strategy to re-open the economy in the future. Delivery shortages as well as uncertainty about the share of the public which is willing to get finally vaccinated do not make this a viable short-term strategy, however.

as the more expensive PCR (polymerase chain reaction) tests, possibly leading to a high rate of false positives. This can undercut the credibility of the screening process and Covid measures in general. In contrast to the initial intention of mass testing, it may also confine a large share of workers wrongly in quarantine putting an unjustified pressure on the economy (Pettengill and McAdam, 2020). Related to this discussion is also the important question how long the potential benefits of mass testing will last.[3]

In this study, we evaluate the impacts of mass testing on the spread of COVID-19 pandemic as a potential strategy for re-opening the economy. Exploiting a unique quasi-experimental setting whereby districts in Slovakia above an ex-ante unknown threshold of positive test incidence had to repeat the mass testing event, we are able to identify the impact of repeated mass testing on the level (prevalence) and velocity (R_0) of the spread of the disease.

2 Literature review

Up until now, numerous papers have been written regarding the impact of COVID-19 on the economy and the potential benefits of different policies on the spread of the infection, labor markets, and the economy as a whole; see, for example, Brodeur, Gray, Islam and Bhuiyan (2020) for a literature review. Rapid mass testing as a policy tool to re-open the economy and stop the spread of the disease has until recently received only limited attention Mina, Parker and Larremore (2020).

It has been suggested that rapid antigen testing on which mass testing events rely may play an important role in mitigating the pandemic Baqaee, Farhi, Mina and Stock (2020). The cheap and broad availability of these tests and the relatively short duration until test results are available make them a useful tool with potentially high returns relatively to their costs (Atkeson, Droste, Mina and Stock, 2020). In addition, recent evidence points to a relatively high sensitivity and specificity, even if the rates of false positivity and false negativity are not trivial (see e.g. Mina, Miller, Quigley, Prentiss, McKinnon and Comer (2020)).

Several studies have developed theoretical models to evaluate possible effects of antigen testing. Using a behavioral SIR model for the US, (Atkeson et al., 2020) propose that a simple and cheap two-step procedure may yield the best results from a cost-benefit perspective; for example, a low specificity antigen testing followed by a high-specificity confirmatory antigen testing of those who tested positive.Their derived conclusion depends crucially, however, on public compliance with quarantine of those who test positively (and their contacts). Indeed, one important caveat underscored by the authors

[3] Platiel, Zheng and Walensky (2020) investigate what screening policies would permit U.S. students to return to college. They model a hypothetical cohort of students under various assumptions on reproductive numbers and tests of varying frequencies.

is that the costs and benefits of mass testing depend on whether mass testing increases or decreases risky behavior. From the theoretical point of view, the consequences are ambiguous. Testing generates a range of signals to which individuals may respond differently. For example, those who have tested positively may altruistically self-quarantine, or, alternatively, they may stop worrying about contracting the virus (as they already have it) and start behaving carelessly, not adequately caring about the health risks their behaviour poses for others.

Mina, Parker and Larremore (2020) have developed a simple theoretical model to study the effect of testing on infections, explicitly modeling the effect of social distancing and social activity as network formation problems. They find that testing and isolating can work but also that testing increases the range of social networks as individuals feel more secure. Interestingly, they find that if testing capacities are limited, the optimal behavior is to leave some capacities unused to avoid the adverse social contentedness effect of testing.

Using a theoretical model on reopening universities, Platiel et al. (2020) argue that rapid testing can be effective, but that testing has to be conducted in very short-time intervals. Their results indicate that students need to be screened every 2 days, in addition to general vigilance and good prevention practice. Their conclusions are derived for a hypothetical cohort of students, however. While interesting, the results still beg the questions if these policies also work in practice.

Pettengill and McAdam (2020), in contrast, doubt that widespread and cheaper rapid antigen testing can end the COVID-19 pandemic. On the one hand, antigen testing produces nontrivial numbers of false positives, which can undercut the credibility of testing programs and compliance with quarantine orders. Specifically, this is the case if false positivity is revealed to the tested by e.g. confirmatory testing. Using cheaper and faster, but less precise tests may also put a large drag on the economy by placing a lot of false positive workers in isolation. One the other hand, the imperfect sensitivity of antigen testing implies that significant numbers of infected individuals are not identified as such. If they receive the corresponding signal (e.g. a negative test certificate), this is likely to increase their risky behavior and worsen the pandemic.

Empirical estimates of the impacts of antigen testing on the spread of COVID-19 is scant. Callaway and Li (2020) evaluate Tennessee's open testing policy using a bounding approach which allows for non-randomly missing test data. Using bordering states as controls, they show that increased accessibility of testing reduced overall cases (which are not fully observed), confirmed cases, and work trips among counties with fast-growing numbers of confirmed cases.

Closest related to our study is the work by Pavelka et al. (2020), exploring the impact of mass antigen testing in Slovakia by comparison of the infections in different munic-

ipalities in different rounds of antigen-testing and also using a microsimulation model. They find that the decrease in prevalence compared to a scenario of unmitigated growth cannot be fully explained by non-pharmaceutical interventions implemented before the mass antigen testing. They interpret this finding as evidence of an impact of antigen testing (and the ensuing isolation and quarantine of positively tested individuals) on the spread of the virus. In another study using the data from mass antigen testing in Slovakia, Bod'ová and Kollár (2020) study the spatial patterns of the epidemic in Slovakia. They conclude that the mitigating effect of repeater testing increased with the measured prevalence in the earlier round of testing.

To the best of our knowledge no study about the impact of antigen mass testing on the level and velocity of the spreading of COVID-19 exploring a quasi-experimental setting has yet been has been published.[4]

3 Mass antigen-testing in Slovakia

In late 2020, Slovakia became the first country in the world that introduced nation-wide mass rapid antigen testing intended to detect new COVID-19 cases early and halt the spread of the disease. With a total population of 5.5 million people, residents aged between 10 and 65 years and older adults in employment, or about 80% of the population were eligible for voluntary tests.

Before conducting mass testing, Slovakia implemented several containment measures to control infection, such as partial schools closing and restrictions on indoor gastronomy and leisure activities. During the week prior to the first wave of mass testing, authorities asked citizens to limit their movement. The Slovakia's government also conducted preliminary pilot testing from October 23 to 25, 2020, in four districts: Bardejov, Dolný Kubín, Námestovo and Tvrdošín.

The first wave of nation-wide testing was organised from October 31 to November 1, 2020. Around twenty thousand healthcare professionals and forty thousand army personnel and volunteers helped to test residents of all the country's 79 districts. In total, $5,276,832$ rapid antigen tests (SD Biosensor Standard Q antigen tests) were conducted during this period, and overall 84-87% of the eligible population was tested (Pavelka et al., 2020). Štefánik (2021) provides key statistics related to the COVID-19 pandemic Slovakia and compares it to neighboring countries.

Even though the participation in mass testing was voluntary, citizens who agreed to be tested received medical certificates with the result of their test. Those people who had

[4] An insightful exception is the analysis that has been published in a blog by Šuster (2021), which, similarly to our paper, compares COVID-19 trends in the districts that were tested and the were not tested in the second round of Ag-testing in Slovakia.

not participated in mass testing or had positive results were instructed to quarantine for ten days (in the latter case - together with their household members and self-traced contacts). All employers had to ask their employees for medical certificates, and authorities conducted random inspections in public venues. As a result, although participation in the testing was formally voluntary, these measures created impelling incentives to participate.

A second wave of mass testing occurred from November 7 to 8, 2020. Individuals living in counties with a prevalence rate of seven positive results per 1 000 tests or 0.7% were asked to re-test. Importantly for our identification, the threshold as well as the timing was ex-ante unknown to citizens and chosen at random. There is no epidemiological or any other foundation for choosing a prevalence of 0.7% threshold. The decision about the second mass testing was announced on November 2, 2020. This enables us to treat the second wave as an experiment, with some districts 'treated' and others 'non-treated' with the second wave. Similar approaches have recently been used in Šuster (2021).

Overall, the second round of testing was conducted in the 45 counties. As it was the case during the first round, participation was voluntary but citizens without medical certification of their test results had to quarantine.[5]

We distinguish three types of districts:

1. Districts with non-pharmaceutical measures, a pilot, and two waves of testing
2. Districts with non-pharmaceutical measures and the first wave of testing only
3. Districts with non-pharmaceutical measures and two waves of testing (1st and 2nd)

In the analysis below we take the last type as the treatment group, the second type as the control group, and the first type is omitted (see Figure 1).

4 Data and methodology

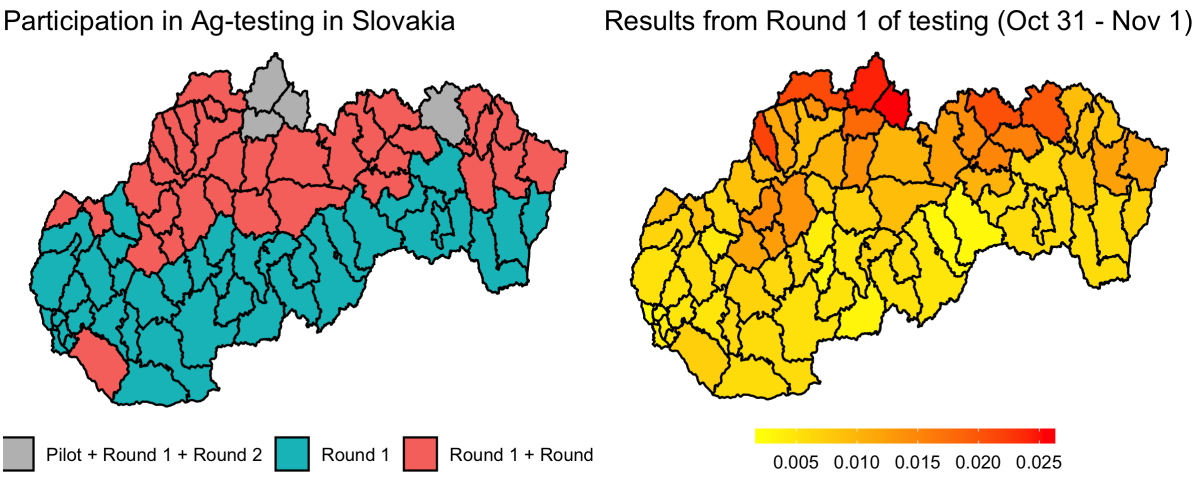
We make use of the public data provided by the Institute of Health Analyses an analytical unit that supports the Ministry of Health of the Slovak Republic.[6] The data contains information on infections, PCR and rapid antigen testing, as well as the hospitalizations on the district level for all 79 districts in Slovakia. [7]

[5] As a result of the mass testing efforts, 50,466 tests turned positive, with the proportion of positives in all tested varying from 3.91% during the pilot to 1.01% in the first wave of mass testing and 0.62% in the second wave (Pavelka et al., 2020).

[6] `https://github.com/Institut-Zdravotnych-Analyz/covid19-data`

[7] Due to data availability of PCR test results, we merged the districts in the two largest cities, Bratislava and Košice. The number of districts thus dropped from 79 to 72.

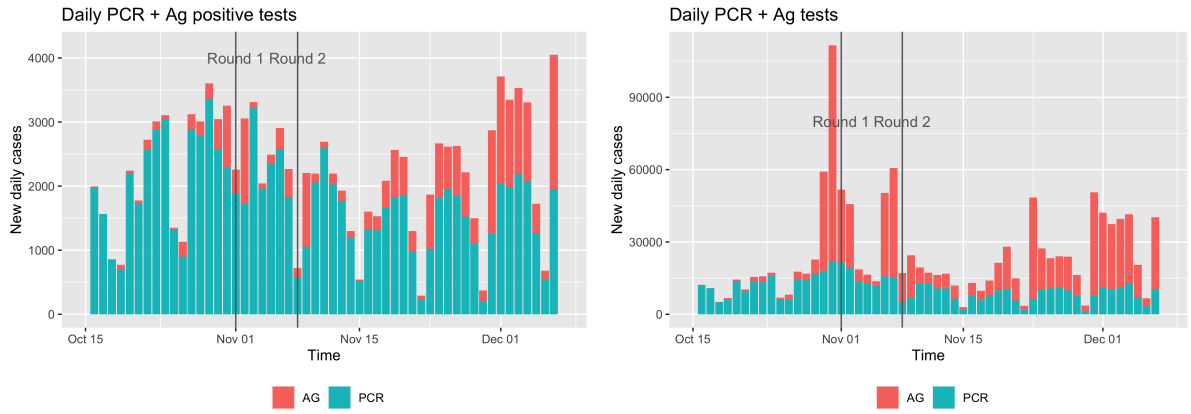
Figure 1: Participation of different districts in mass Ag-testing and results from the first round.



4.1 Situation in Slovakia

This subsection visualizes the epidemiological situation in Slovakia prior to and after two rounds of nationwide mass antigen-testing.

Figure 2: Evolution of positive tests and the number of administered tests.



As it is apparent from the figure, the number of positive PCR tests went down after the mass testing. At the same time, we see an increase in antigen positive tests. Around mid October, antigen testing sites were introduced at various places in Slovakia, where it was possible to get tested for free. Availability of these free antigen tests increased over time as demonstrated in the right pane of Figure 2. With the wider availability, it is likely that a larger share of the population has switched to Ag-tests, rather than the more expensive PCR tests. Given that there was a large variation in the antigen testing capacities on the district level, we included antigen tests into our analysis.

Figure 3 shows that positivity of PCR tests went up in the week after the round 2 of testing and the went down the week after. Positivity of the antigen tests appeared to

be somewhat more stable with a spike approximately 10 days after the second round of testing.

Figure 3: Evolution of percent positive for PCR and Ag-tests.

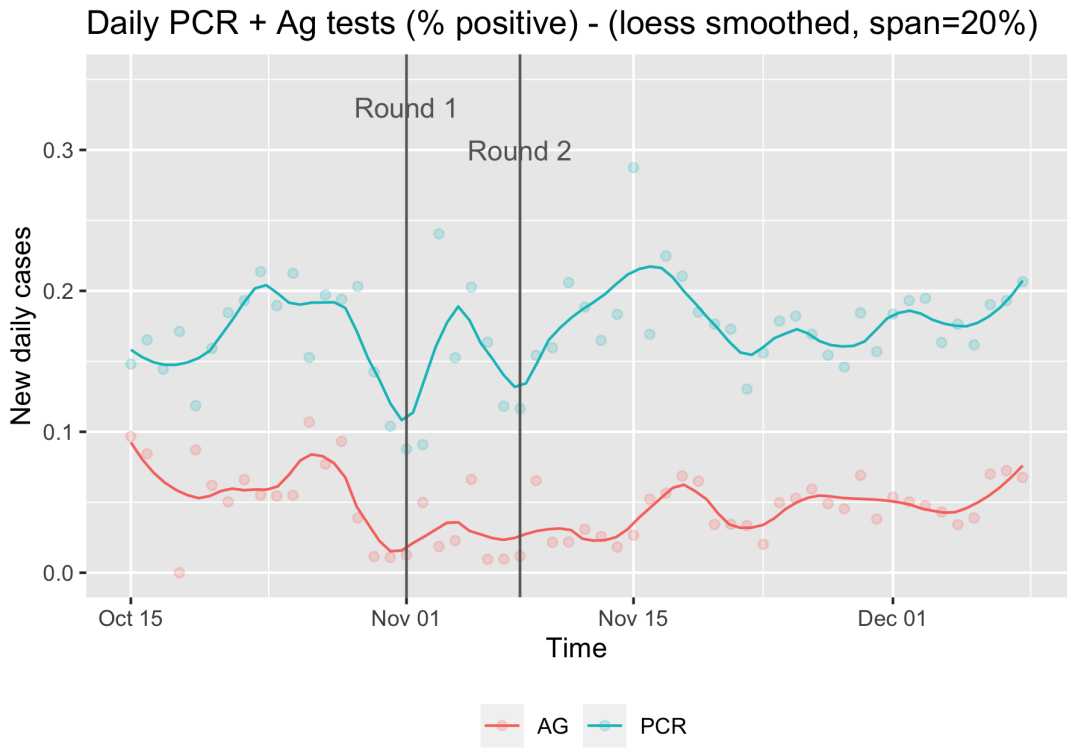
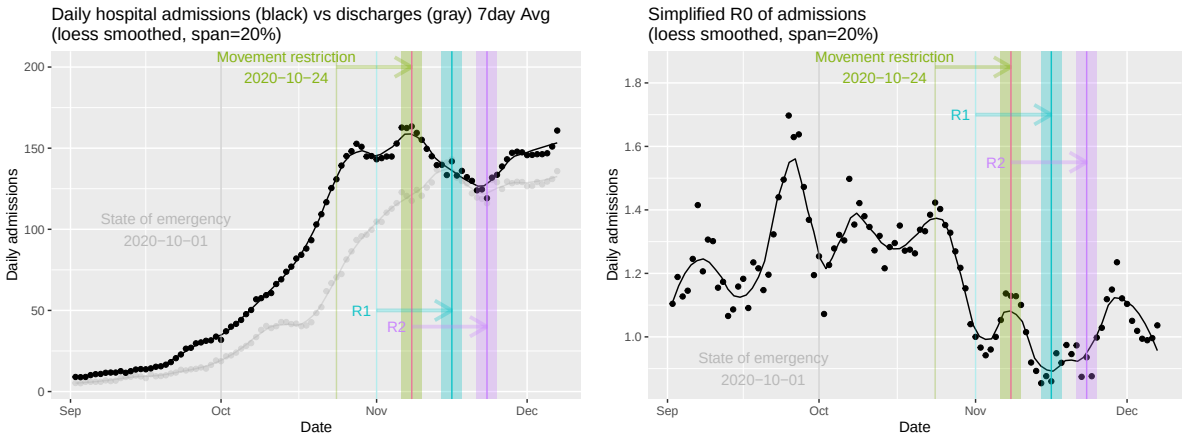


Figure 4: Hospital admissions, discharges and R_0. Arrows point 15 days ahead.



Hospitalizations (Figure 4) decreased around the weekend of the second round of Ag testing. The right pane shows the simplified R_0 of hospital admissions.[8] From these figures alone it is not possible to disentangle the potential effect of the Ag-testing as several

[8]Simplified R_0 evaluated at time T is equal to $\left(\sum_{\tau=T-1}^{T-7} y_\tau\right) / \left(\sum_{\tau=T-6}^{T-12} y_\tau\right)$, where τ is a day. This measure was used in epidemic nowcasting in Germany (Hamouda et al., 2020).

other policy measures were in place, such as closed schools and movement restrictions. However, we see some improvement in the R_0, which fell below 1 for approximately two weeks. Data on hospitalizations are independent of the testing capacities and therefore contain a lot of information about the epidemic situation although with a time lag.[9]

4.2 Effect of mass testing on infection prevalence

Estimating the causal effect of the mass testing is difficult, as without strong modelling restrictions, it is not possible to disentangle the effect of testing from the other interventions, such as movement restrictions or school closures. In other words, we lack valid counterfactual observations.

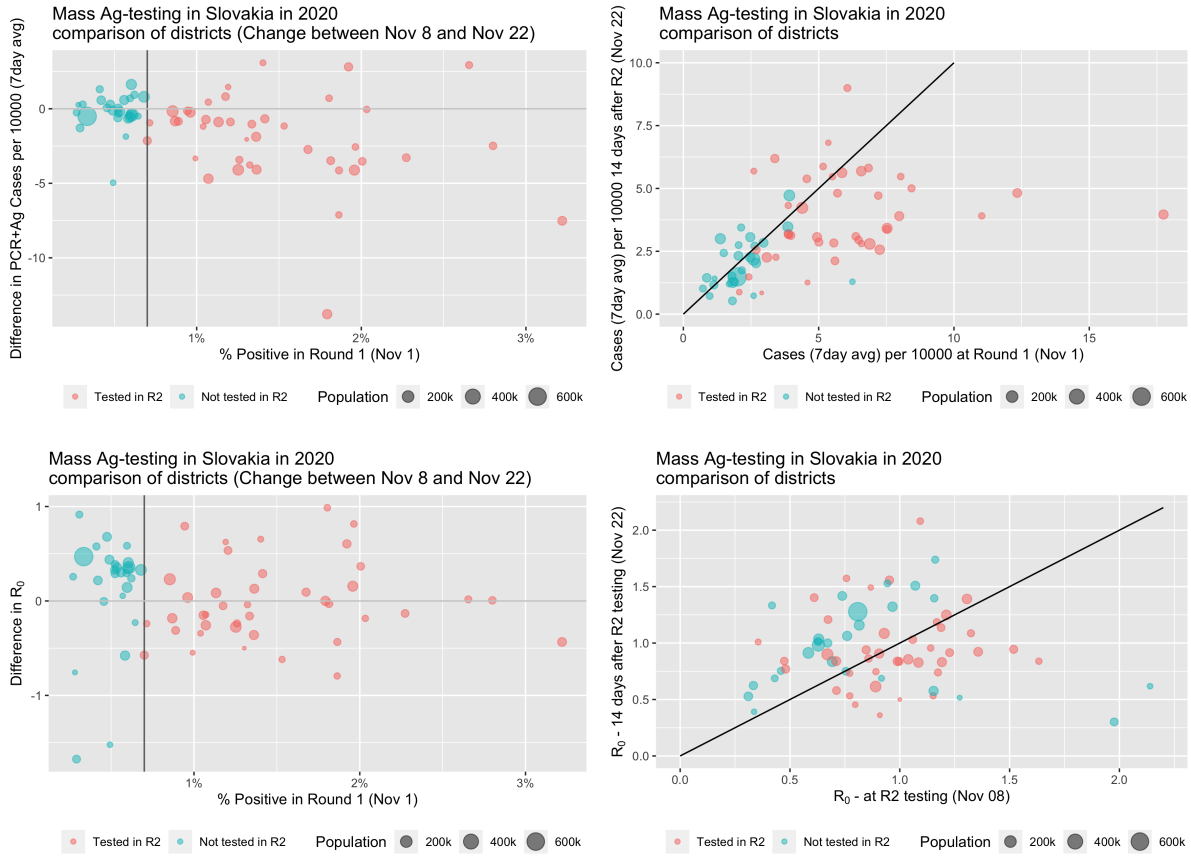
In the second round of mass Ag-testing in Slovakia, only districts with at the prevalence over 0.7% from the first round participated. Given that the threshold 0.7% was decided and announced ex-post (one day after the first round and 5 days before the second round), we can explore the source of randomness that this discontinuity induced to identify the effect of the second round on the infection spread.

We first explore the association between the prevalence from the first round with the difference between normalized positive cases (PCR or antigen) by 68 districts in Figure 5.[10] We observe that tested districts experienced larger drops in infections than the untested regions as well as larger drops in the reproduction number R_0.

[9] There is no reliable data on the numbers of hospitalization on the district level in Slovakia. Not all districts have hospital ane the are many spillovers from the neighborhood districts.

[10] Recall that we removed four districts that participated in the pilot testing one week before the first round. The epidemic situation in these four districts was far worse than in the rest of the country.

Figure 5: Association between the results from the first round of mass testing and the R_0 and various metrics two weeks after the Round 2.



5 Data analysis

To measure the causal impact of treatment (i.e. going through two instead of one round of testing) on the prevalence and velocity (as measured by R_0) of the COVID-19 disease, we consider the following two simple regression models:

$$\Delta y_i = \beta_0 + \beta_1 above_i + \epsilon_i \tag{1}$$

or

$$y_{it} = \beta_0 + \beta_1 (above_i \cdot t) + \beta_2 t + \beta_3 above_i + \epsilon_i \tag{2}$$

Where

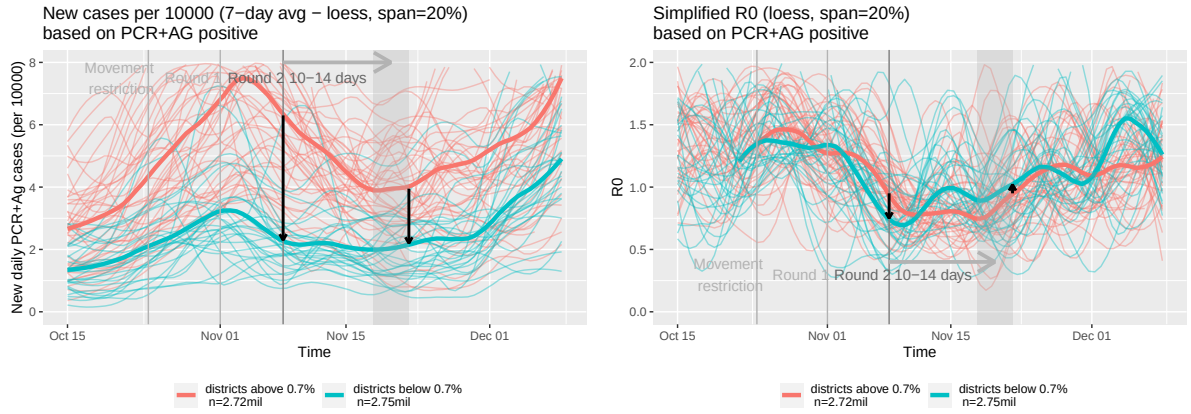
- i stands for a particular district
- t stands for a time: $t = 0$ for Nov 8, 2020 and $t = 1$ for Nov 22, 2020
- $above_i$ is a binary indicator if a district had infection rates above 0.7% in the first round of mass antigen-testing and therefore had to participate in the second round
- $\Delta y_i = y_{i1} - y_{i0}$ is the change in the output variable, we consider different outcome variables
  - 7day rolling average of new PCR+antigen positive tests (per 10 000 inhabitants)
  - logarithm of a 7day rolling average of new PCR+antigen positive tests (per 10 000 inhabitants)
  - simplified R_0
  - logarithm of simplified R_0

The model based on equation (1) compares the difference in outcomes at the time of Round 2 and 14 days after in treated and non-treated districts; it is the simplest possible model. Equation (2) describes a difference-in-differences setup. While equation (1) compares the simple differences in the outcome variable; equation (2) also models a linear trend. While linear trend is apparently a highly restrictive simplification, the high variability in the infection curves on the district level (as seen on Figure 6) do not allow for a credible estimation of more complex models. Note that the estimator $\hat{\beta}_1$ based on equation (1) is the same as the estimator $\hat{\beta}_1$ based on equation (2). The standard errors for these two estimators are, however, different because these two models have different numbers of degrees of freedom.

5.1 All districts

In the full sample of 68 districts, we observe that differences in both new cases and simplified R_0 between tested and non tested districts shrank between Rounds 1 and 2 (Figure 6). The districts with very high infection prevalence improved more than the districts with very low infection prevalence. While the districts with an infection rate around the 0.7% threshold from Round 1 are similar regarding their pandemic situation; This is not not the case for the districts with very high and very low prevalence. Some of the differences may be explained by the regression to mean effect. We explore this possibility in Subsection 5.2.

Figure 6: Evolution of infections and R_0 in the districts below and above the threshold 0.7% for the full sample.



We used population-weighted ordinary least squares to estimate models based on equations (1) and (2). We estimate that the change in infections between Round 2 and 14 days after are approximately 2.3 cases (per 10 000 inhabitants) lower in the districts tested also in the second round, which amounts to a change of about -36%. We estimate the decrease in simplified R_0 to be approximately by 0.28 larger in the treated districts than the non-treated ones. This corresponds to a decrease in R_0 by 31%.

5.2 Restricted sample

In order to have groups of districts that are more comparable from the epidemiological point of view than is the case in the full sample, we restrict the sample only to a comparably sized group of districts that are closer to the threshold level 0.7%. The choice of the districts is presented in Figure (7): the "above" the threshold group of districts that were tested include districts where the prevalence as measured in the Round 1 was between 0.7% and 1% with a cumulative population size of around 650 000; the "below" group consists of districts with a prevalence in Round 1 in the range between 0.6% and 0.7% with population of approximately 750 000.

Table 1: Full Sample: Regression results based on Equation (1)

	Dependent variable:			
	Diff in Cases	Diff in log Cases	Diff in R_0	Diff in log R_0
	(1)	(2)	(3)	(4)
Tested in R2	-2.252^{***}	-0.358^{***}	-0.279^{***}	-0.314^{***}
	(0.574)	(0.108)	(0.096)	(0.103)
(Intercept)	-0.125	-0.092	0.271^{***}	0.306^{***}
	(0.405)	(0.076)	(0.068)	(0.073)
Observations	68	68	68	68
R^2	0.189	0.143	0.114	0.123

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

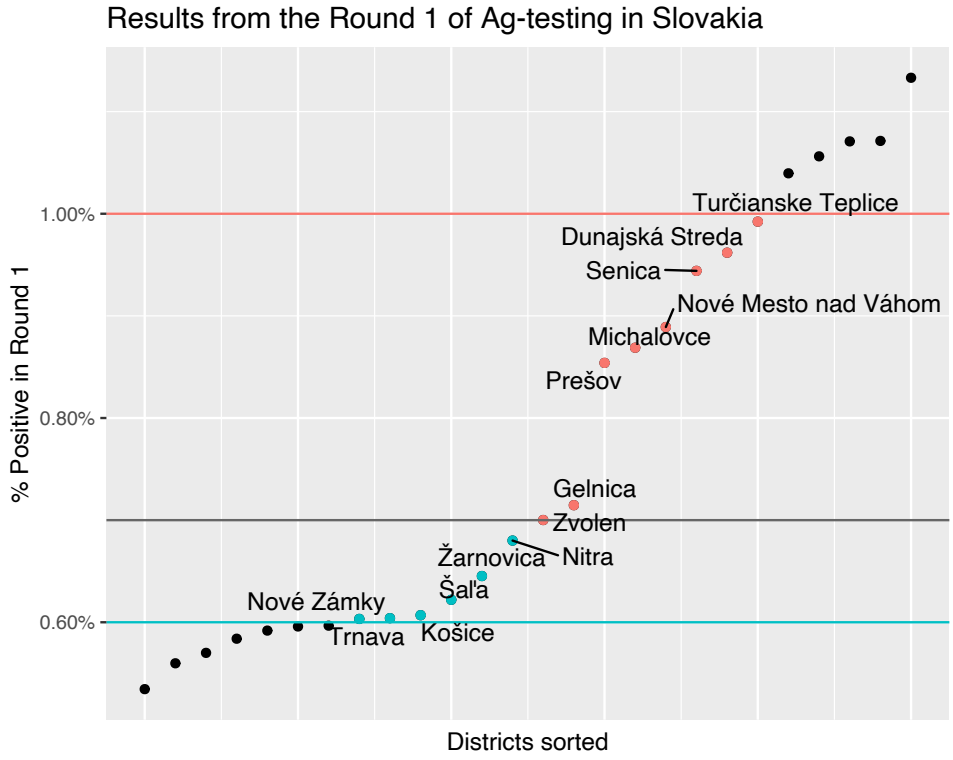
Table 2: Full Sample: Regression results based on Equation (2)

	Dependent variable:			
	Cases	log Cases	R_0	log R_0
	(1)	(2)	(3)	(4)
Tested in R2 × Time	-2.252^{***}	-0.358^{**}	-0.279^{***}	-0.314^{***}
	(0.632)	(0.144)	(0.100)	(0.112)
Tested in R2	4.047^{***}	1.005^{***}	0.186^{***}	0.235^{***}
	(0.447)	(0.102)	(0.071)	(0.079)
Time	-0.125	-0.092	0.271^{***}	0.306^{***}
	(0.446)	(0.101)	(0.071)	(0.079)
(Intercept)	2.243^{***}	0.739^{***}	0.787^{***}	-0.302^{***}
	(0.315)	(0.072)	(0.050)	(0.056)
Observations	136	136	136	136
R^2	0.463	0.535	0.105	0.114

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

There is a trade-off between making the reference groups too small (high statistical uncertainty) and too large (possible regression to mean effect). We will explore the sensitivity to the choice of the groups in the following subsection.

Figure 7: Choice of districts in our restricted sample.



Results from the estimated regressions are presented in Tables 3 and 4 and visualized in Figure 8. While we estimate that the change in infections between Round 2 and 14 days after are smaller than in the full sample (1 versus 2.3 per 10 000 inhabitants), the percentage difference is very similar at 35%. We estimate the decrease in simplified R_0 to be 0.32 larger in the re-tested districts, which is about 38% (versus 0.28 and 31% in the full sample, respectively). We observe that with a smaller number of districts, the statistical precision of our estimates decreased compared to the full sample. Only the coefficients for R_0 in the model based on equation (1) remain statistically significant at the customary 0.05 level.

Table 3: Main Specification: Regression results based on eq (1)

	Dependent variable:			
	Diff in Cases	Diff in log Cases	Diff in R_0	Diff in log R_0
	(1)	(2)	(3)	(4)
Tested in R2	-1.007^{**}	-0.350^{*}	-0.332^{**}	-0.385^{**}
	(0.454)	(0.185)	(0.152)	(0.168)
(Intercept)	0.328	0.136	0.328^{***}	0.395^{***}
	(0.309)	(0.126)	(0.103)	(0.114)
Observations	14	14	14	14
R^2	0.291	0.230	0.286	0.305

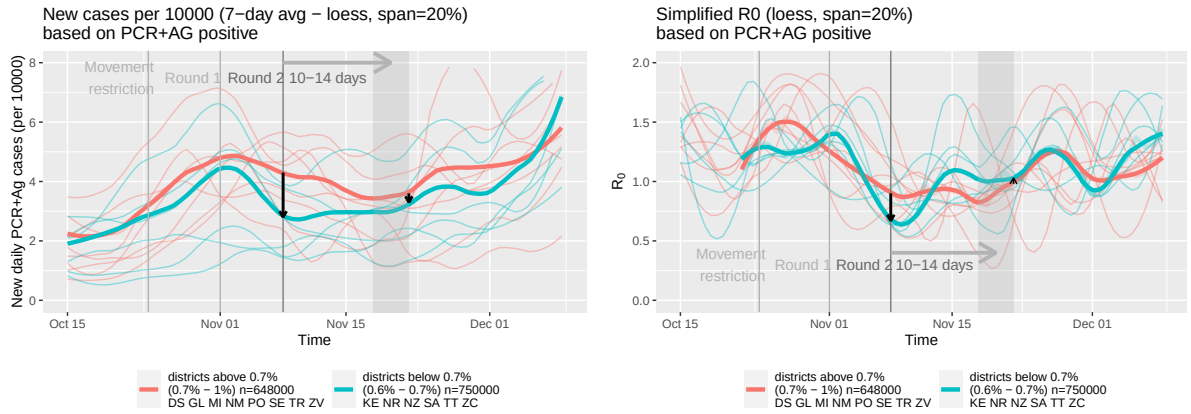
Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

Table 4: Main Specification: Regression results based on eq (2)

	Dependent variable:			
	Cases	log Cases	R_0	log R_0
	(1)	(2)	(3)	(4)
Tested in R2 × Time	−1.007	−0.350	-0.332^{*}	-0.385^{*}
	(0.904)	(0.293)	(0.173)	(0.189)
Tested in R2	1.451^{**}	0.463^{**}	0.285^{**}	0.315^{**}
	(0.639)	(0.208)	(0.123)	(0.133)
Time	0.328	0.136	0.328^{**}	0.395^{***}
	(0.616)	(0.200)	(0.118)	(0.128)
(Intercept)	2.769^{***}	0.941^{***}	0.699^{***}	-0.378^{***}
	(0.435)	(0.141)	(0.083)	(0.091)
Observations	28	28	28	28
R^2	0.193	0.181	0.286	0.318

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

Figure 8: Evolution of infections and R_0 in the districts below and above the threshold 0.7% for the restricted sample.



5.3 Size of the reference groups

In order to explore the sensitivity of the regression results to the choice of the reference groups, we estimate a sequence of regression for different group sizes. Figure 9 presents these results. On the horizontal axis we have the maximal size of both the "below" and "above" groups. The black line denotes the regression coefficient and the red and cyan lines stand for the 95% confidence intervals from models based on equations (1) and (2) respectively.[11] On the left hand size of all these four graphs, we have results based on relatively small reference groups, consisting from only a few districts where the statistical uncertainty is very high. On the right hand side we have the results based on the full sample. The grey vertical dashed line shows the results for the maximal reference group of size of 750 000, which is close to the choice made in the restricted sample from the previous section.[12]

Several patterns become apparent. The downward shift of the curve of coefficient $\hat{\beta}_1$ in the upper left pane suggests a regression to mean effect if the outcome of interest is the level of infections, much less so on a log scale. The regression coefficients for R_0, however, appears very stable for different population sizes of the reference groups.

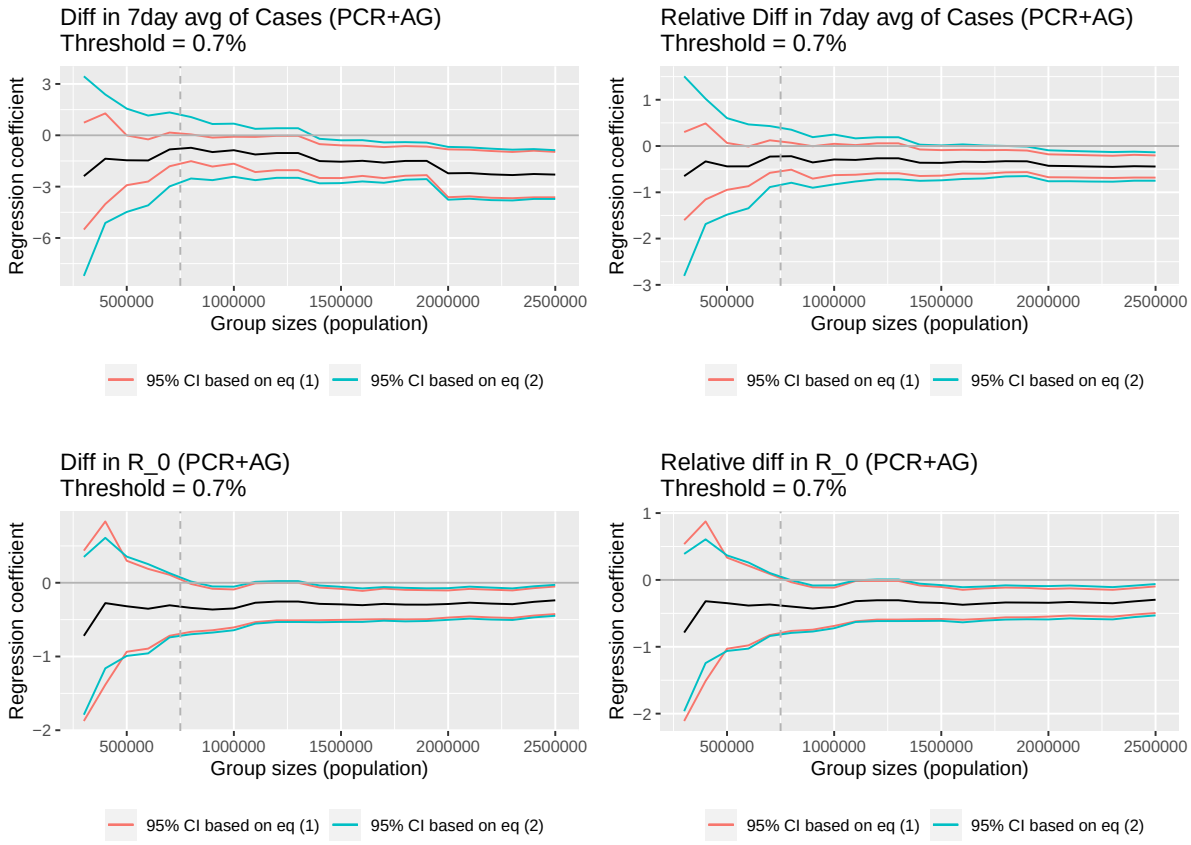
Overall, the reduction due to the second round of testing in the reported infection cases is about 30% and the reduction in R_0 is about 0.3. Statistical uncertainty connected to these estimates is sizeable and 95% confidence intervals for many specifications include zero. However, we observe that the estimates become statistically significant above certain

[11] More precisely, we sequentially include non-treated districts in the "below" group (and similarly, treated districts in the "above" group) until the cumulative population in these group crosses a specified threshold.

[12] These results are not completely the same, as the range of percentages that correspond to this line would not consist of round numbers, such as 0.6% and 1% in the restricted sample.

threshold sizes of "above" and "below" groups are reached (circa 1.3 million for cases and 0.8 million for R_0) .

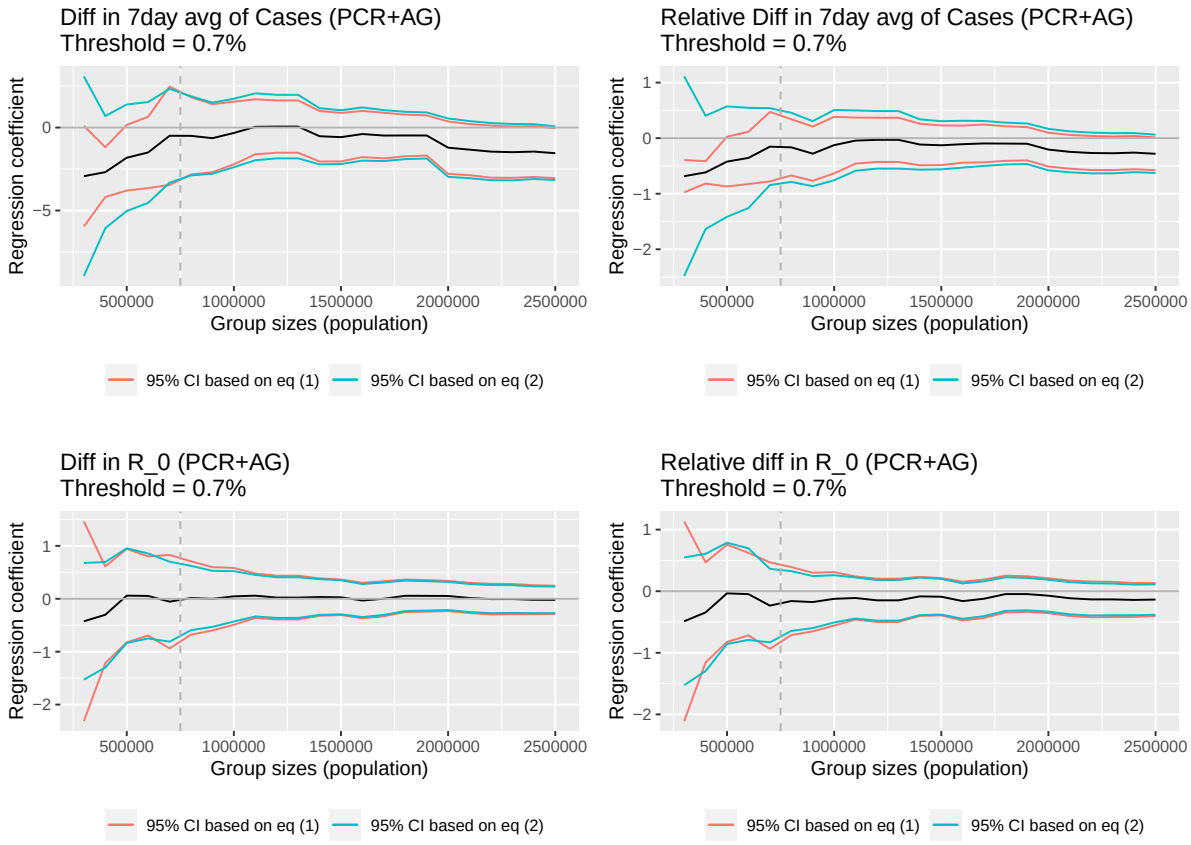
Figure 9: Estimated regression coefficient $\hat{\beta}_1$ with confidence intervals based on equations (1) and (2) as a function of the size of the groups below and above the threshold.



5.4 Effects at different times

In order to shed some light on how lasting these effects are, we first look at the effect on our outcome variables three weeks after Round 2 (instead of two weeks considered above).

Figure 10: Estimated regression coefficient $\hat{\beta}_1$ with confidence intervals based on equations (1) and (2) as a function of the size of the groups below and above the threshold. The effect is now calculated three weeks after Round 2.

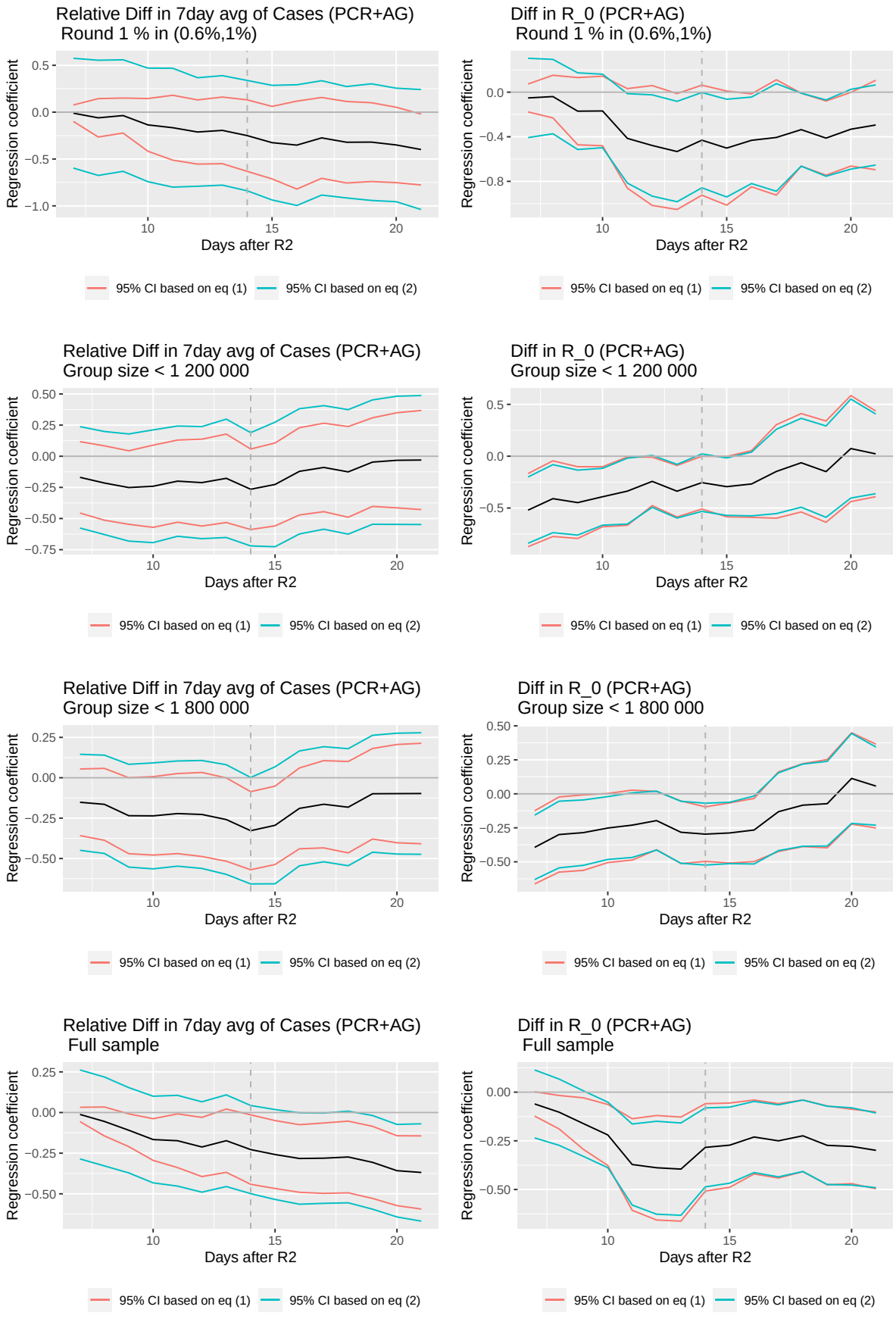


The two lower panels in Figure 10 show that the effects for R_0 vanish. These findings suggest that antigen mass testing does not effect the speed of the disease spread for longer than three weeks. For very large group sizes (over 2 million) the regression coefficient for relative differences in infection cases are negative (-27%), but the statistical uncertainty is high, with 95% confidence intervals (-55%, 0.5%) and (-58%, 3.7%) per model based on Equations 1 and 2, respectively. Downward slopes of the curves in the upper panel of Figure 10 suggest that the results may be driven by the regression to mean effect. This evidence indicates that it is difficult to draw any strong claims about longer-term effects. It is, however, natural to expect some form of attenuation in time as the epidemic trajectories of different districts are subject to different shocks, so the level of noise increases, accumulating over time.

We study the impact of Round 2 for different durations of the possible effects in more detail. Figure 11 visualizes the regression coefficients of interest for log Cases and R_0 as a function of the number of days after Round 2 for the following specifications:

(1) Districts with Round 1 results between $(0.6\%, 1\%)$ as described in Subsection 5.2
(2) Districts with the "above" and "below" group sizes each less than 1 200 000
(3) Districts with the "above" and "below" group sizes each less than 1 800 000
(4) The full sample

Figure 11: Estimated regression coefficient $\hat{\beta}_1$ with confidence intervals based on equations (1) and (2) as a function of time at which the outcome was measured. Results are shown for different subsamples. Dashed vertical line denotes two weeks.



For the specification described in Subsection 5.2 (subsample with Round 1 results between $(0.6\%, 1\%)$) the effect on log cases is estimated at -30% after 16 days, but with very wide confidence intervals. The curve for R_0 shows a hump shape with the biggest effect around 13–15days after the Round 2.

In the second specification with group sizes less than 1 200 000, we observe that the effect starts to fade out after two weeks with R_0 being around 0 after three weeks.

Very similar patterns may be seen in the specification with the group sizes that have population size less than 1 800 000.

The full-sample shows a similar pattern to the first specification. As it may be subject to a stronger regression to mean effect, we should be cautious when interpreting these numbers.

Overall, we consider the second and third specifications as the most salient to triangulate the true effect. Our best conjecture is that the effect of the second round, conditional on the first round, was a 25-30% reduction in prevalence and reduction in R_0 by approximately 0.3. After three weeks the effects diminished, with point estimates for the reduction in prevalence at less than 10% and the effect on R_0 being close to zero; however, the coefficients after three weeks are estimated with a large statistical uncertainty and cannot be distinguished from zero.

5.5 Placebo specifications

In order to investigate whether our results might be driven by some spurious correlations, we conducted similar analysis as under our main specifications presented above but with different arbitrary placebo thresholds for defining the "above" and "below" groups: 1.2% and 0.5%. The regression results, figures and sensitivity to the size of references groups are presented in the Appendices A.1 and A.2.

For the placebo specification with threshold 1.2% we see a negative but non-significant regression coefficient of interest for all the reference group sizes that did not include any non-tested district. All the other coefficients, both for thresholds 1.2% and 0.5%, are not significant and very close to zero. The curves for coefficients for R_0 are non-significant and closely match the horizontal axes, supporting the causal interpretations of the results with the true threshold 0.7%.

We also tried to artificially change the date at which the districts were treated. Instead of the true date Nov 8, we set Nov 1 as the date of placebo Round 2 instead, and then we measured outcomes 14 days after (on Nov 15). Figure 18 in Appendix A.3 shows that the estimated effects for this placebo test are non-significant and close to zero, apart for the specification with very large group sizes for the logarithm of R_0. This essentially states that the velocity of the disease in districts with worse epidemiological

situations improved more than in the districts with better situations. This is intuitive as we have seen this pattern in most of the other specifications and can be attributed to the regression to mean effect.

6 Discussion and limitations of results

We underline several points related to the causal interpretation of our regression coefficients.

Our effects can be only interpreted as an effect on the districts for which the infection prevalence is in the proximity of 0.7% as measured by the antigen tests in the Round 1 of testing.

We estimate the effect of the second round only, but *conditional* on that the first round occurred one week before. Given that there were many more infections isolated during the first round of testing than in the second case, we conjecture that the effect of the first round may have been even higher than the numbers that we estimate for Round 2.

It is important to stress that the week after each round of the mass nation-wide antigen-testing, citizens who were not tested were required to self-quarantine. We do not disentangle these effects but our estimates have to be interpreted as a joint effect of Round 2 antigen-testing together with all the policy restrictions associated with this round of testing.

Given the high variability of the infection curves in the particular districts, we employed a simple specification that only allowed us to make crude comparisons. We also explored the possibility if mobility patterns are predictive for the effects, but there was only little variation between the type of mobilities that we had data on the district level (see Figure 19 in Appendix for workplace mobility).[13]

There are different explanations why the effect of antigen-testing started to slowly fade away in time after approximately 15 days. Given the lack of reliable mobility patterns data on the district level we do not offer any data-driven explanations. We conjecture that different behaviour changes and non-compliance with stringency measures between the tested and non-tested districts could potentially explain part of the effect reduction. This is however an open question and the level of statistical uncertainty is high.

This paper does not discuss nor address the cost-effectiveness of the nationwide antigen-testing. From the epidemiological point of view, we estimate that antigen-testing in Slovakia likely resulted in a sizable reduction in infections and very likely slowed down the infection spread. From an economic perspective, such positive epidemiological effects potentially benefited the economy. However, the overall effect remains ambiguous, as

[13] We merged our dataset with Google's Community Mobility Reports (GoogleLLC, 2021)

we do not measure the other direct and indirect effects on labor supply and demand, other economic variables, or later policy responses. Whether this was an efficient use of resources is a different question that requires further study.

7 Conclusion

We explore the quasi experimental setup of the mass nationwide antigen-testing in the Fall of 2020 in Slovakia, where only districts with an infection prevalence above 0.7% from the first round of testing were tested in the second round. We exploit the natural variation in the results from the first round that resulted in some districts being slightly below and some slightly above the threshold 0.7%. Comparing these groups of districts we estimate that the first and second rounds of testing together with the implied mobility restrictions led to approx. 25–30% decrease of new infections and 0.3 decrease in R_0 14 days after the second round when compared with the first round of testing only. We estimate that the effect slowly faded out and three weeks after the second round the difference was less than 10% whereas the effect on R_0 was close to zero. Our results are robust with respect to a number of robustness and placebo tests.

The policy implications are that antigen testing could be an effective instrument of COVID-19 mitigation. It also could, to the extent it slows down the spread of the disease, help the economy either directly through its impacts on labor supply, demand, investment and other economic variables, or by enabling the governments to avoid harsher non-pharmaceutical interventions. It is however important to recognize the limitations of the testing: it is costly and binds significant resources, the effects dissipate with time, and it may affect other important variables besides the spread of the disease, such as the levels of trust in society, people's expectations, voters' preferences, and many others.

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A Sensitivity and robustness

A.1 Placebo 1 - different threshold (1.2%)

Figure 12: Choice of districts in the placebo with threshold 1.2%.

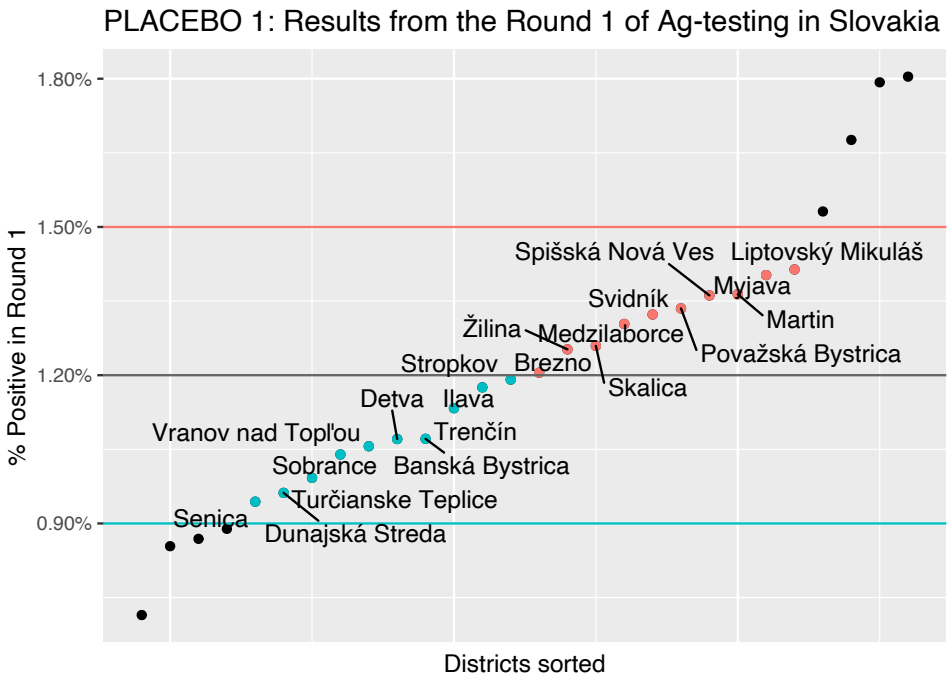


Figure 13: Evolution of infections and R_0 in the districts below and above the Placebo threshold 1.2%.

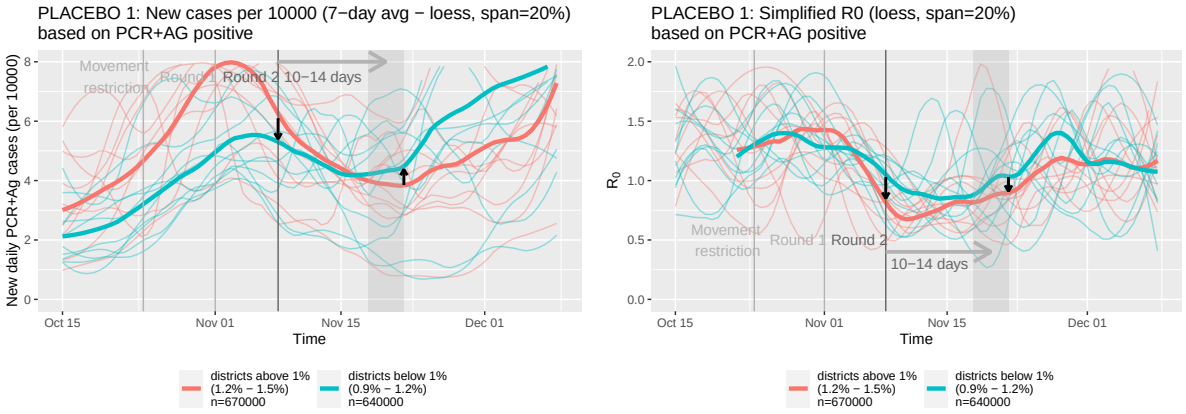


Figure 14: Regression coefficient as a function of maximal size of the groups below and above the threshold. Vertical dash line stands for the size of the placebo specification groups and vertical blue line depicts the size of the below group that does not include any district that was not tested in R2. The results to the left of this blue line are all tested in R2.

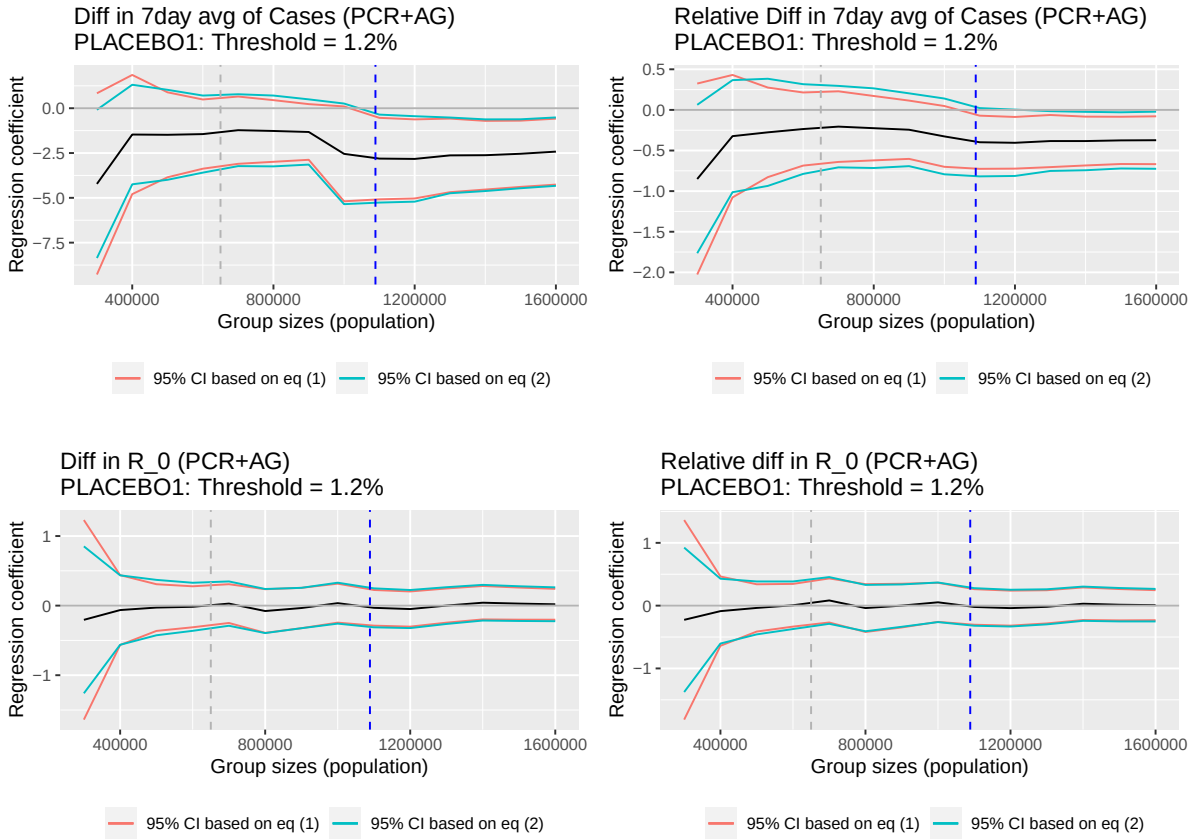


Table 5: Placebo 1: Regression results based on eq (1)

	Dependent variable:			
	Diff in Cases	Diff in log Cases	Diff in R_0	Diff in log R_0
	(1)	(2)	(3)	(4)
Tested in R2	−1.325	−0.229	−0.052	−0.005
	(0.849)	(0.197)	(0.150)	(0.181)
(Intercept)	−1.101*	−0.279*	0.016	−0.011
	(0.607)	(0.141)	(0.107)	(0.130)
Observations	20	20	20	20
R^2	0.119	0.070	0.007	0.00005

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

Table 6: Placebo 1: Regression results based on eq (2)

	Dependent variable:			
	Cases	log Cases	R_0	log R_0
	(1)	(2)	(3)	(4)
Tested in R2 × Time	−1.325	−0.229	−0.052	−0.005
	(0.989)	(0.246)	(0.160)	(0.186)
Tested in R2	0.889	0.169	−0.210*	−0.246*
	(0.700)	(0.174)	(0.113)	(0.131)
Time	−1.101	−0.279	0.016	−0.011
	(0.707)	(0.176)	(0.114)	(0.133)
(Intercept)	5.280***	1.611***	1.070***	0.046
	(0.500)	(0.125)	(0.081)	(0.094)
Observations	40	40	40	40
R^2	0.293	0.241	0.197	0.167

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

A.2 Placebo 2 - different threshold (0.5%)

Figure 15: Choice of districts in the placebo with threshold 0.5%.

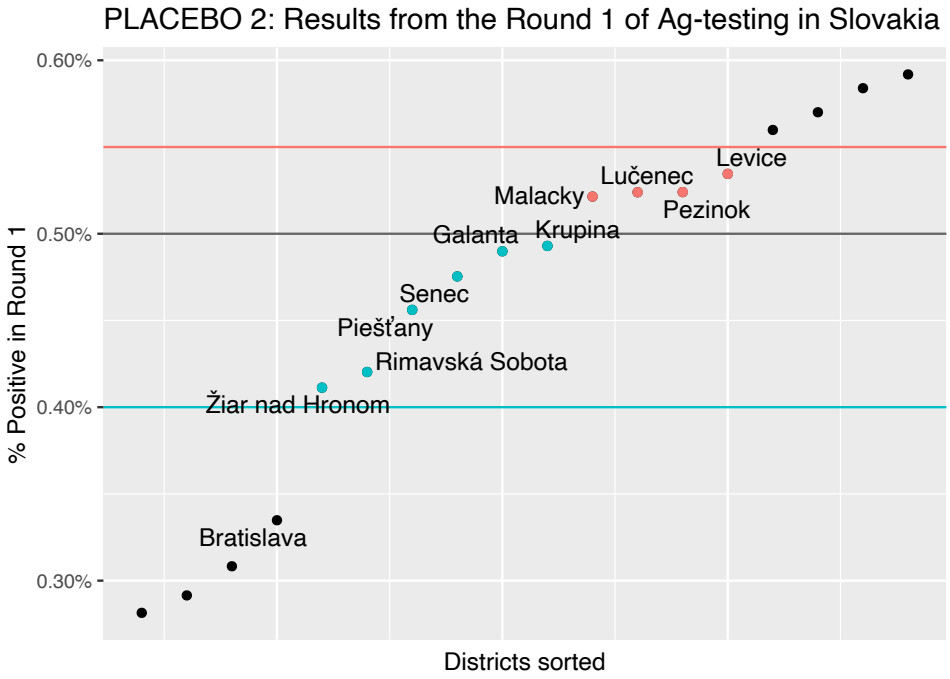


Figure 16: Evolution of infections and R_0 in the districts below and above the Placebo threshold 0.5%.

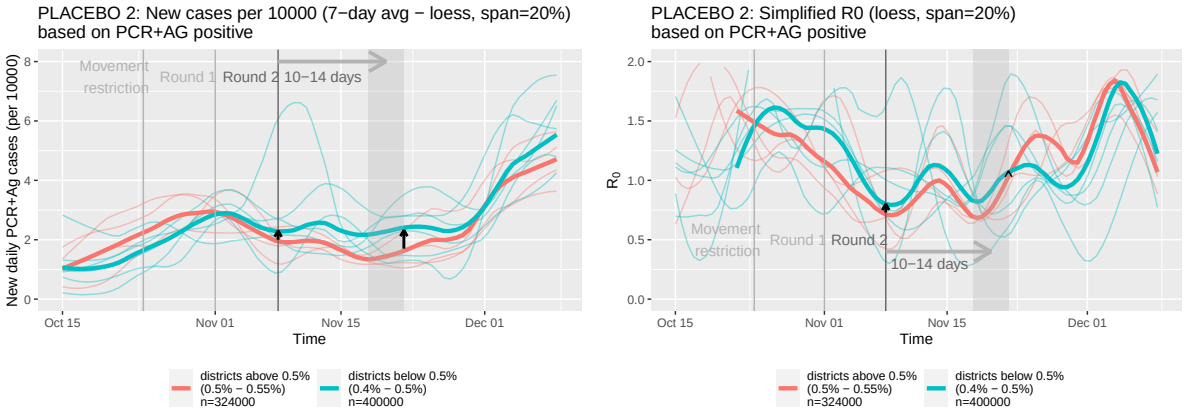


Figure 17: Regression coefficient as a function of maximal size of the groups below and above the threshold. Vertical dash line stands for the size of the placebo specification groups and vertical blue line depicts the size of the above group that does not include any district that was tested in R2. The results to the left of this blue line are all based on districts not tested in R2.

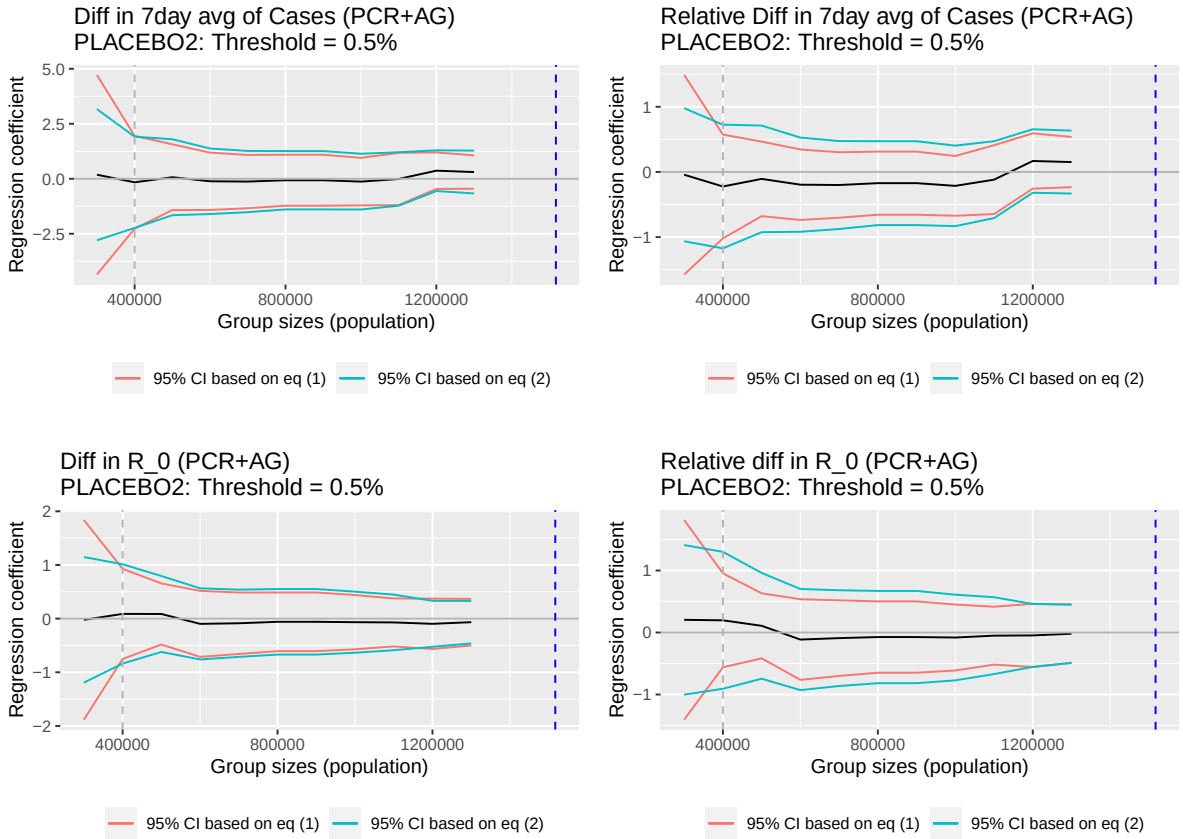


Table 7: Placebo 2: Regression results based on eq (1)

	Dependent variable:			
	Diff in Cases	Diff in log Cases	Diff in R_0	Diff in log R_0
	(1)	(2)	(3)	(4)
Tested in R2	−0.298	−0.242	0.057	0.121
	(0.688)	(0.255)	(0.262)	(0.236)
(Intercept)	0.046	0.100	0.284	0.316*
	(0.460)	(0.170)	(0.176)	(0.158)
Observations	10	10	10	10
R^2	0.023	0.102	0.006	0.032

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

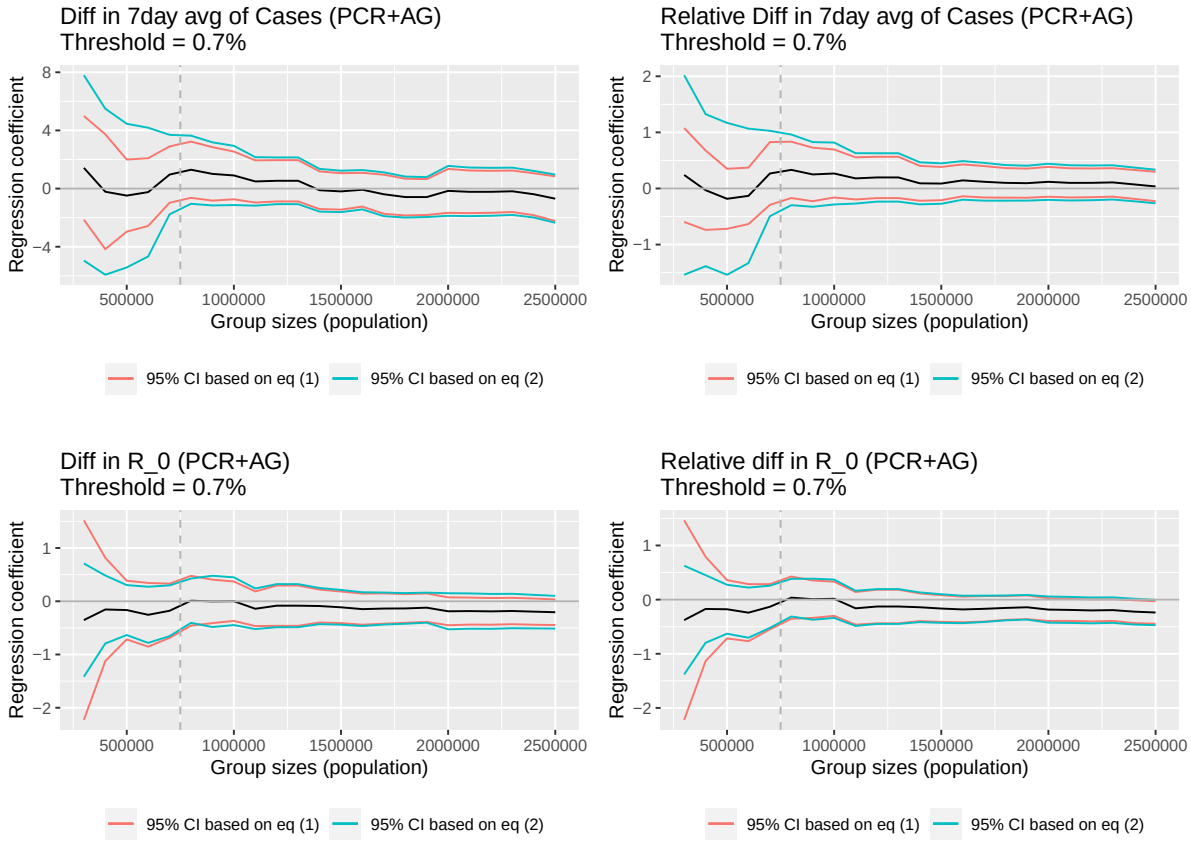
Table 8: Placebo 2: Regression results based on eq (2)

	Dependent variable:			
	Cases	log Cases	R_0	log R_0
	(1)	(2)	(3)	(4)
Tested in R2 × Time	−0.298	−0.242	0.057	0.121
	(0.795)	(0.372)	(0.370)	(0.436)
Tested in R2	−0.454	−0.128	−0.169	−0.163
	(0.562)	(0.263)	(0.261)	(0.308)
Time	0.046	0.100	0.284	0.316
	(0.532)	(0.249)	(0.247)	(0.291)
(Intercept)	2.347***	0.727***	0.856***	−0.283
	(0.376)	(0.176)	(0.175)	(0.206)
Observations	20	20	20	20
R^2	0.135	0.122	0.177	0.168

Note: Districts weighted by their population size. *p<0.1; **p<0.05; ***p<0.01

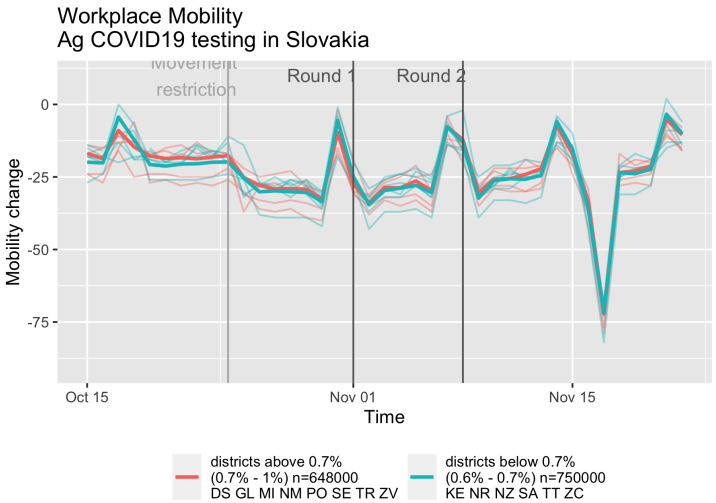
A.3 Placebo 3 - different treatment date (Nov 1)

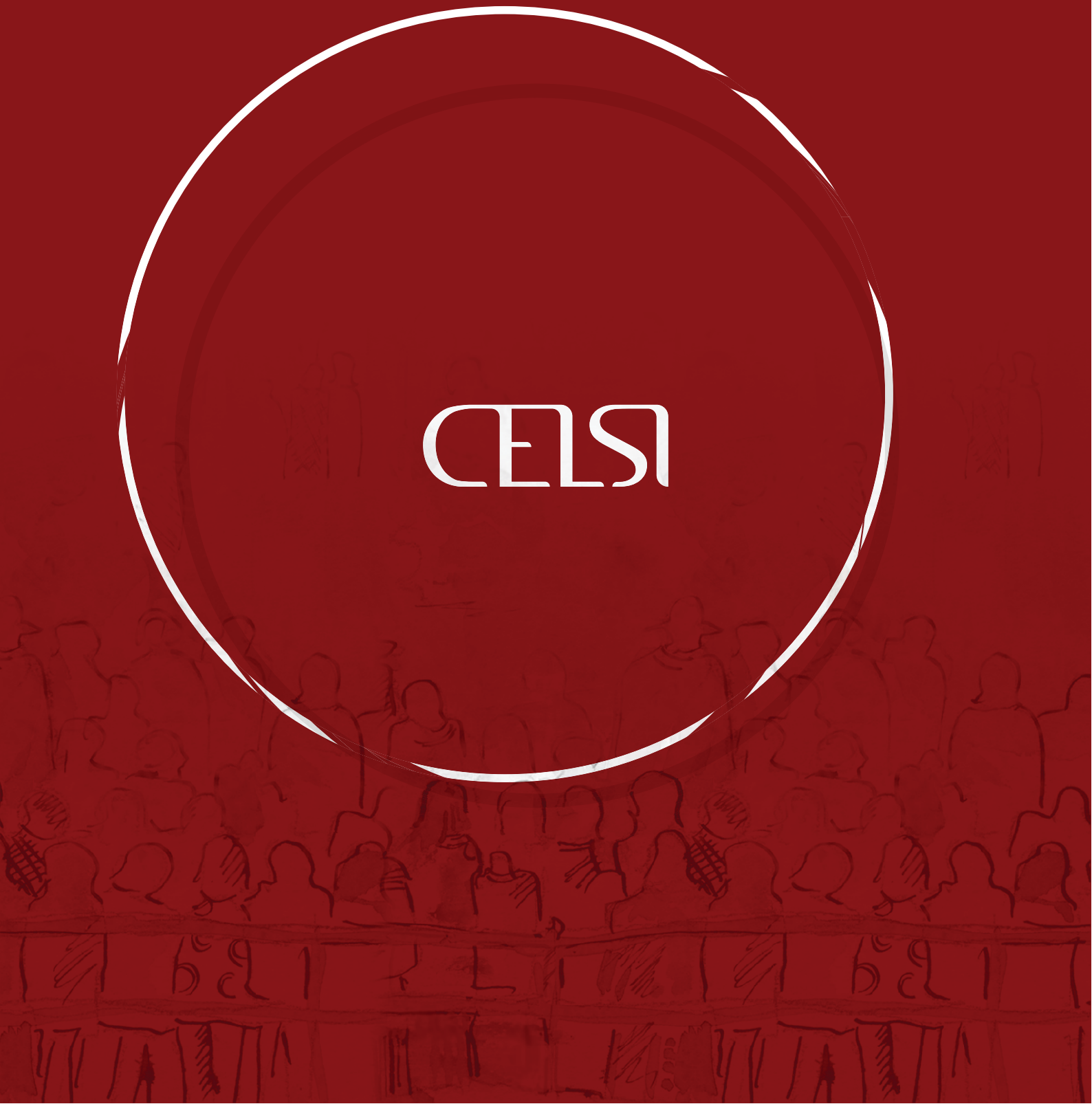
Figure 18: Estimated regression coefficient $\hat{\beta}_1$ with confidence intervals based on equations (1) and (2) as a function of the size of the groups below and above the threshold. Round 2 date was (incorrectly) set for Nov 1.



B Additional figures

Figure 19: Google mobility data - transport.

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