The Effectiveness of Strategies to Contain SARS-CoV-2: Testing, Vaccinations, and NPIs ⋆

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One-sentence summary

Among many measures, large-scale rapid testing has had the largest effect on reducing SARS-CoV-2 infections in Germany in 2021

Background:
Spread disease, initially NPIs, now testing and vaccines; seasonality unclear.
Objective: Provide a model that allows studying these things in conjunction, allowing for different virus strains.
Results: Along the transition to vaccination-induced herd immunity, testing is most effective, also thanks to family structures. Seasonality contributed its share.
Conclusions: Frequent rapid testing should remain part of strategies to contain CoViD-19.

JEL Classification: C63, I18
Keywords: Covid-19, agent based simulation model, public health measures

[Tobias 1]

Commenting is on!
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Since early 2020, the CoViD-19 pandemic has presented an enormous challenge to humanity on many dimensions. The development of highly effective vaccines holds the promise of containment in the medium term. However, most countries find themselves many months—and often years—away from reaching vaccination-induced herd immunity. In the meantime, it is of utmost importance to employ an effective mix of strategies for containing the virus. The most frequent initial response was a set of non-pharmaceutical interventions (NPIs) to reduce contacts between individuals. While this has allowed some countries to sustain equilibria with very low infection numbers[1], most have seen large fluctuations of infection rates. Containment measures have become increasingly diverse and now include testing, more nuanced NPIs, and contact tracing. Neither these policies' effect nor the influence of seasonal patterns or more infectious virus strains are well understood in quantitative terms. This paper develops a model incorporating all these factors. The framework allows to combine a wide variety of data in a timely fashion, making it useful to predict the effects of various interventions. We apply the model to Germany and show that rapid testing had the largest impact on the reduction in infections by almost 80% during the month of May 2021. We conclude that rapid tests have a large role to play at least as long as vaccinations have not been offered to an entire population.

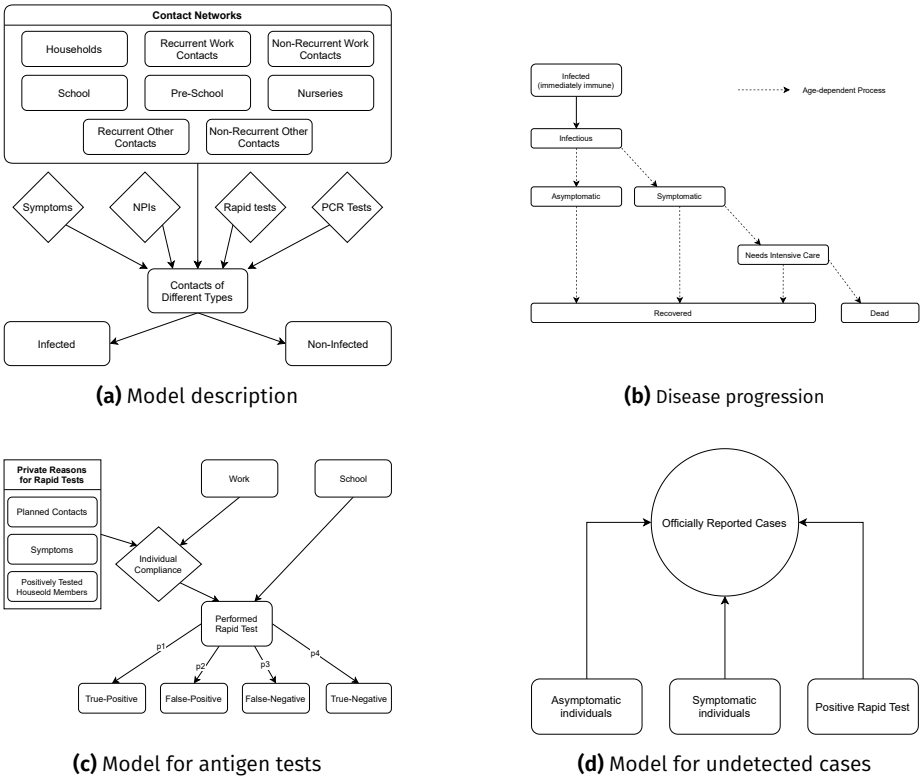
[HM 1]
Cite some paper on herd immunity, maybe vaccine data

At the core of our agent-based model are physical contacts between heterogeneous agents (Figure 1a).[2] Each contact between an infectious individual and somebody susceptible to the disease bears the risk of transmitting the virus. Contacts occur in the household, at work, at school, or in other settings (leisure activities, grocery shopping, medical appointments, etc.). Some contacts recur regularly, others occur at random. Random contacts are typically assortative in age and geographical location. Empirical applications can take the population structure from census data and the types and frequency of contacts from diary data measuring contacts before the pandemic (e.g. Mossong, Hens, Jit, Beutels, Auranen, et al., 2008).[3] The dimensions are chosen so that the most common NPIs can be modeled in great detail by reducing the number of contacts in a particular setting or the risk of transmitting the disease for a type of contact. For example, a mandate to work from home will reduce the number of work contacts to zero for a fraction of the working population. Schools and daycare can be closed entirely, operate at reduced capacity—including an alternating schedule—,or implement mitigation measures like masking requirements or air filters (Lessler, Grabowski, Grantz, Badillo-Goicoechea, Metcalf, et al., 2021). Curfews may reduce the number of contacts in non-work/non-school settings.

1. See Contreras, Dehning, Mohr, Bauer, Spitzner, et al. (2021) for a theoretical equilibrium at low case numbers which is sustained with test-trace-and-isolate policies.

2. We provide a detailed comparison to other approaches in 2. The model most closely related to ours is described in Hinch, Probert, Nurtay, Kendall, Wymatt, et al. (2020).

3. Hoang, Coletti, Melegaro, Wallinga, Grijalva, et al. (2019) provide access to multiple data sets on contact types and frequencies at http://www.socialcontactdata.org/ covering countries from all continents except North America and Australia.



(a) Model description

(b) Disease progression

(c) Model for antigen tests

(d) Model for undetected cases

Figure 1. Model description

Note: ...

In any setting, measures like masking requirements would reduce the probability of infection associated with a contact.

In our model, susceptibility to contracting the SARS-CoV-2 virus is dependent on age. A possible infection progresses as shown in Figure 1b. We differentiate between an initial period of infection without being infectious or showing symptoms, being infectious (presymptomatic or asymptomatic), showing symptoms, requiring intensive care, and recovery or death. The probabilities of transitioning between these states depend on age; their duration is random within intervals calibrated to medical literature (for a detailed description see Section 5.1). Conditional on the type of contact, infectiousness is independent of age (Jones, Biele, Mühlemann, Veith, Schneider, et al., 2021).

[HM 2]
Seems broadly standard, can we cite someone?

The model includes several other features, which are crucial to describe the evolution of the pandemic in 2020-2021. New virus strains with different profiles regarding infectiousness can be introduced. Agents may receive a vaccination. With a probability of 75% (Hunter and Brainard, 2021), vaccinated agents become immune and they do not transmit the virus (Levine-Tiefenbrun, Yelin, Katz, Herzel, Golan, et al., 2021; Petter, Mor, Zuckerman, Oz-Levi, Younger, et al., 2021; Pritchard,

Matthews, Stoesser, Eyre, Gethings, et al., 2021).[4] During the vaccine roll-out, priority may depend on age and occupation.

We include two types of tests. Polymerase chain reaction (PCR) tests directly reveal whether an individual is infected or not. PCR tests require some days to be processed and there are always aggregate capacity constraints. In contrast, rapid antigen tests yield immediate results. Specificity and sensitivity of these tests is set according to data analysed in Brümmer, Katzenschlager, Gaeddert, Erdmann, Schmitz, et al. (2021) and Smith, Gibson, Martinez, Ke, Mirza, et al. (2021); sensitivity depends on the timing of the test relative to the start of infectiousness. Figure 1c shows our model for rapid test demand. Schools may require students to be tested regularly. Rapid tests may be offered by employers for on-site workers. Individuals may demand tests for private reasons, which include having plans to meet other people[5], showing symptoms of CoViD-19, and because a household member tested positively for the virus. We endow each agent with an individual compliance parameter. This parameter determines whether she takes up rapid tests offered by employers or follows up on private reasons. The thresholds are lower for tests in a private setting than for tests at the workplace.

[HM 3]
True? @Janos

Modelling a population of agents according to actual demographic characteristics means that we can use a wide array of data to identify and estimate the model's many parameters.[6] Mobility data is used to model the reductions in work contacts. School and daycare policies are incorporated directly from official directives. Infection rates by age and geographical region are estimated to match to officially recorded numbers; so is the prevalence of virus strains. The model yields total infections, but only a fraction of those will be officially recorded. We model the fraction of known cases as depicted in Figure 1d.[7] A further advantage is that the simulated data have a structure that resembles datasets used for regression models, which

[HM 4]
Add a sentence.

4. 75% is lower than what is usually reported for after the second dose of the Biontech/Pfizer vaccine, which is most commonly used in Germany. We choose it because our model neither includes booster shots, nor does it allow vaccinated individuals who became immune to transmit the disease(Levine-Tiefenbrun et al., 2021; Petter et al., 2021; Pritchard et al., 2021).

5. A positive test will make them reduce their contacts; this is why tests impact the actual contacts in Figure 1.

6. See section S.XXX of the supplementary materials for an overview.

7. EQUATIONS FOR THE FOURTH PICTURE (find names!):
The probabilities for Figure d are the following:

$$x = \frac{n\_newly\_infected \times share\_known\_cases \times P(PCR|S)}{n\_symptomatic\_and\_untested}$$
$$y = \frac{n\_newly\_infected \times share\_known\_cases \times P(PCR|S)}{n\_asymptoptomatic\_and\_infected\_and\_untested}$$
$$z = \frac{P(PCR|RT)}{survey}$$

where untested means that individuals are not waiting on the result of a previously administered rapid test.

allows additional plausibility checks by re-running the same models on the model-generated data.

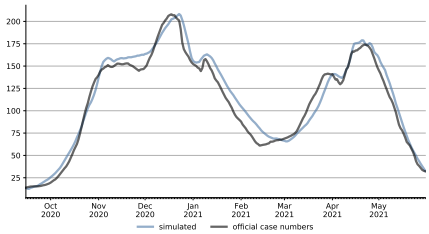
[HM 5]
Only keep if we actually do something like that.

We apply this model to Germany. In March and April 2020, the country broke the first wave of the pandemic fairly quickly. Between mid-May and mid-September, daily new infections were below 20 per Million and day. We model the period mid-September 2020 to the end of May 2021. We pick the starting date for two reasons. First, we do not include the first wave because the environment was very different (e.g., aggregate PCR test capacity was much lower and we would require a very different model for calculating the share of known cases) Second, a large fraction cases during summer of 2020 were traced to international travel (Hodcroft, Zuber, Nadeau, Vaughan, Crawford, et al., 2021; Koch-Institut, Seifried, Böttcher, Oh, Michel, et al., 2021) but the precise number is difficult to model.

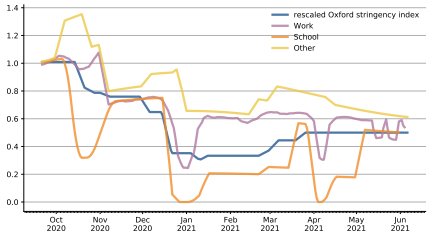
[HM 6]
Cite our world in data/RKI

Figure 2 describes the evolution of the pandemic and of its drivers. The black line in Figure 2a shows officially recorded cases; the black line in Figure 2b the Oxford Response Stringency Index (Hale, Atav, Hallas, Kira, Phillips, et al., 2020), which tracks the tightness of non-pharmaceutical interventions. We transform the index so that lower values represent higher levels of restrictions. A value of zero means all measures incorporated in the index are turned on. The value 1 represents the situation in mid-September, with restrictions on gatherings and public events, masking requirements, but open schools and workplaces (the raw value of the index at that point is 49.5). In the seven weeks between mid September and early November, cases increased by a factor of 10. Restrictions were somewhat tightened in mid-October and again in early November. New infections remained constant throughout November, before rising again in December, which prompted the most stringent lockdown to this date. Schools and daycare centers were closed again, so were customer-facing businesses except for grocery and drug stores. From the peak of the second wave just before Christmas until the trough in mid-February, newly detected cases decreased by almost three quarters. The third wave in the spring of 2021 is associated with the B.1.1.7 strain, which became dominant in March. See Figure 2d. In early March, some NPIs were being relaxed; e.g., hairdressers and home improvement stores were allowed to open again to the public.

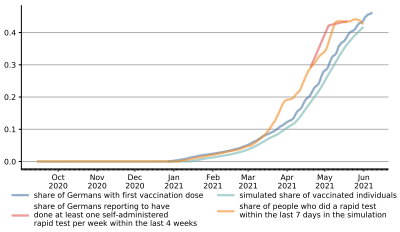
By this time, the set of policy instruments had become much more diverse. Around the turn of the year, the first people were vaccinated with a focus on older age groups and medical personnel (Figure 2c. By the end of May, just over 40% had received at least one dose of a vaccine. Around the same time, rapid tests started to replace PCR tests in many medical and nursing facilities. These had to be administered by medical doctors or in pharmacies. At-home tests approved by authorities became available in mid-March, rapid test centers were opened and one test per person and week was made available free of charge. Depending on the state, customers were only allowed to enter certain stores with a recent negative rapid test result. These developments are characteristic of many countries: The initial focus on NPIs to slow the spread of the disease has been accompanied by vaccines and a growing



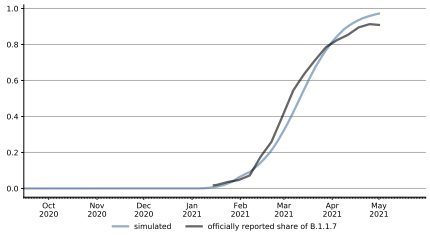
(a) Recorded cases: Empirical and simulated



(b) Stringency of NPIs and changes in infectious contacts by type



(c) Tests and vaccinations



(d) Fraction of B.1.1.7 strain among measured infections

Figure 2. Evolution of the pandemic, its drivers, and model fit, September 2020 to May 2021

Note: All aggregates; See S.XXX for statistics by age group and by geographical region. Also more disaggregated data.
Sources: ...

acceptance and use of rapid tests. At broadly similar points in time, novel strains of the virus have started to pose additional challenges.

Our model is able to track these developments very well. The blue line in Figure 2a shows our model's predictions are very close to officially recorded cases in the aggregate. This is also true for infections by age and geographical region, which are shown in the supplementary materials (Figures 11 and 12, respectively). We can disentangle various mechanisms due to the distinct temporal variation in the drivers of the pandemic. Next to the stringency index, the three lines in Figure 2b summarize how contact reductions, increased hygiene regulations, and seasonality evolved since early September for each of the three broad contact networks. For example, a value of 0.75 for the work multiplier means that if the environment was the same as in September (levels of infection rates, no rapid tests or vaccinations, only the wildtype virus present), infections at the workplace would be reduced by 25%. This reduction is the product of the effect of contact reductions, increased hygiene regulations, and seasonality. Along with the levels of infections, these measures determine the spread of SARS-CoV-2 in 2020. See Appendix for separate plots of the three factors. Two aspects are particularly interesting. First, all lines broadly follow the stringency index and they would do so even more if we left out seasonality and school vacations (roughly the last two weeks of October, two weeks each around Christmas and Easter, and some days in late May). Second, the most stringent regulations are associated with the period of strong decreases in new infection between late December 2020 and mid-February 2021. The measures were not enough, however, to stop the B.1.1.7 variant from spreading in the subsequent period. The steep drop in recorded cases during May 2021 is associated with at least weekly rapid tests to around 42 percent of the population, a vaccination rate that rose from 28% to 43%, and mostly seasonality impacting a fall in the relative infectiousness of contacts outside of work and school.

[HM 7]
Reference

Figure 3 consider the relative effects of rapid tests, vaccinations, and of seasonality during 2021, assuming NPIs to have evolved the same way as in the baseline scenario. Figure 3a shows the model fit (the blue line, same as in Figure 2a), a scenario without any of the three factors (red line), and three scenarios turning these factors off one by one. Figure 3b does the same for total infections in the model. Figure 3c employs Shapley values to decompose the difference in total infections between the scenario without any of the three factors and our main specification.

Until mid-March, there is no visible difference between the different scenarios. Seasonality hardly changes, and only few vaccinations or rapid tests were administered. Even thereafter, the effect of the vaccination campaign is surprisingly small at first sight. Whether considering recorded or total infections, the final level is always the highest in case the vaccination campaign had been running in isolation (red lines). The Shapley value decomposition shows that vaccinations contribute about 15% to the cumulative difference between scenarios. Reasons for this are the slow start—it took until 24 March until 10% of the population had received their first

[HM 8]
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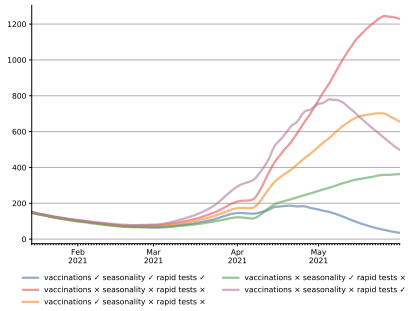
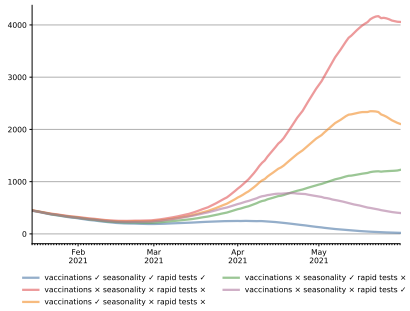
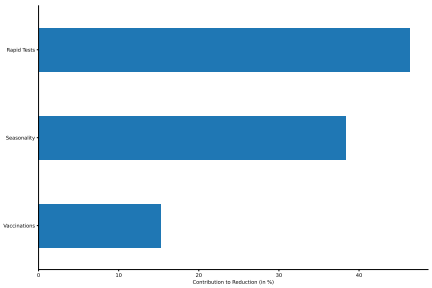


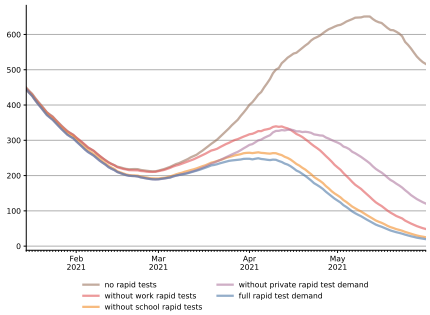
(a) Recorded cases: 2021 scenarios



(b) Total cases: 2021 scenarios



(c) Decomposition of effects for Figure 3b.



(d) Effects of different types of testing

Figure 3. The effect of different interventions on recorded and actual infections

Note: All aggregates; See S.XXX for statistics by age group and by geographical region.
The decomposition is based on Shapley values where the individual contribution of a channel is its average contribution over different sizes of coalitions (combinations with other channels). The individual contribution to a coalition is the difference between the effect size of the coalition with the particular channel and without.

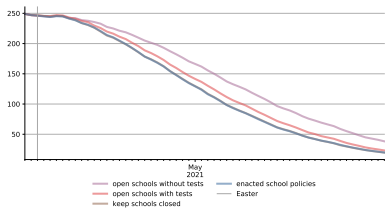
vaccination, the 20% mark was reached on April 19th —and the focus on older individuals. These groups contribute less to the spread of the disease than others due to a lower number of contacts, see 5. It is important to note that the initial focus of the campaign was to prevent deaths and severe disease; the case fatality was rate considerably lower during the third wave when compared to the second (4.4% between October and February and 1.4% between March and June). It is important to note that by the end of our study period, when first-dose vaccination rates reached around 40% of the population, the numbers of new cases would have started to decline.

Seasonality has a large effect in slowing the spread of SARS-CoV-2. By May 31, both observed and recorded cases would be reduced by a factor of four if only seasonality mattered. However, in this period, cases would have kept on rising throughout, just at a much lower pace. Nevertheless, we estimate it to be a quantitatively important factor determining the evolution of the pandemic, explaining most of the early changes and almost 40% of the cumulative difference by the end of May.

The largest effect—almost one half when considering the decompositions—comes from rapid testing. Here, it is crucial to differentiate between recorded cases and actual cases. Additional testing means that infections become known which would otherwise remain undetected. Figure 3a shows that this means that until late April, recorded cases are higher than in the scenario where none of the three mechanisms is turned on. Compared to the scenario with vaccinations only, this point is reached only around mid May and it would be June for the comparison with the seasonality-only scenario. The effect on total cases, however, kicks in immediately and strongly. Despite the fact that only a small fraction of the population performed weekly rapid tests in March (X%), the rise in new infections would be limited by XXX% relative to the scenario without vaccinations, tests, or seasonality.

[HM 9]
Put in

Tests



(a) Effects of different schooling scenarios after Easter

(b) Effects of different work scenarios after Easter

Figure 4. Effects of different scenarios for schooling and working from home

Note: All aggregates; See S.XXX for statistics by age group and by geographical region.

Two particularly important areas:

- Schooling: large costs to pupils, ... social inequalities
- Work: Q2 / 2020 saw the largest drop in GDP in a long time;

Results

- Advantage of testing in both cases: Recurrent. Small cost relative to other stuff. Certain publicness.
- Mandatory tests: Screening effect: ...

Points to mention.

- If anything too optimistic regarding vaccinations
- Social structure / conditional block testing in families important (?)
- Trump-effect: More testing = more cases true for how long?
- Towards the end, total tests go down: Preferences for tests remain the same, but infection rates lower

[HM 10]
Janos, please fill up

1 Supplementary Material

(1) Model

(2) Data

(3) Identification and Estimation

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2 Literature Review

We build on two strands of literature: Recent extensions of the epidemiological SEIR model and agent-based simulation models.

The traditional SEIR model is not fine-grained enough to model nuanced policies. This has motivated a large number of researchers to extend the standard model to allow for more heterogeneity and flexibility. Examples are Grimm, Mengel, and Schmidt (2020), Donsimoni, Glawion, Plachter, and Wälde (2020) and Acemoglu, Chernozhukov, Werning, and Whinston (2020) who develop multi group SEIR models to analyze the effects of targeted lockdowns and Berger, Herkenhoff, and Mongey (2020) who extend the SEIR model to analyze testing and conditional quarantines. For a more comprehensive review see Avery, Bossert, Clark, Ellison, and Ellison (2020). Others have used the results of a standard SEIR model as input for economic models that estimate the cost of policies (e.g. Dorn, Khailaie, Stöckli, Binder, Lange, et al. (2020)).

While the popularity of the SEIR model is mainly due to its simplicity, the extensions are quite complex. It is unlikely that there will be a SEIR model that combines all proposed extensions. Moreover, the extensions do not address other key issues: The main parameter of the SEIR model, the basic reproduction number (R_0), is not policy-invariant. It is a composite of the number of contacts each person has and the infection probability of the contacts. In fact, policy simulations are done by setting R_0 to a different value but it is hard to translate a real policy into the value of R_0 it will induce. In other words, SEIR models are not suited for evaluating the effect of policies which have never been experienced before.

Another commonly used model class in epidemiology are agent-based simulation models. In these models individuals are simulated as moving particles. Infections take place when two particles come closer than a certain contact radius (e.g. Silva, Batista, Lima, Alves, Guimarães, et al. (2020) and Cuevas (2020)). While the simulation approach makes it easy to incorporate heterogeneity in disease progression, it is hard to incorporate heterogeneity in meeting patterns. Moreover, policies are modeled as changes in the contact radius or momentum equation of the particles. The translation from real policies to corresponding model parameters is a hard task.

Hinch et al. (2020) is a recent extension of the prototypical agent-based simulation model that replaces moving particles by contact networks for households, work and random contacts. This model is similar in spirit to ours but focuses on contact tracing rather than social distancing policies.

The above assessment of epidemiological models is not meant as a critique. We are aware that these models were not designed to predict the effect of fine-grained social distancing policies in real time and are very well suited to their purpose. We invite epidemiologists to provide feedback and collaborate to improve our model.

3 Background: The Situation in Germany

4 Model

4.1 Summary

To predict and quantify the effects of a wide variety of fine-grained social distancing policies, vaccinations and rapid testing, we propose a different model structure. Our model inherits many features from prototypical agent-based simulation models but replaces the contacts between moving particles by contacts between individuals who work, go to school, live in a household and enjoy leisure activities.

The structure of the model is depicted in Figure 1a.

The background characteristics include age, county and occupation of each simulated individual. Contact models are functions that map individual characteristics into a predicted number of contacts. Currently, we distinguish between eight types of contact models which are all listed in Figure 1a: households, recurrent and random work contacts, recurrent and random leisure contacts, and nursery, preschool, and school contacts.

The predicted number of contacts is translated into infections by a matching algorithm. There are different matching algorithms for recurrent contacts (e.g. classmates, family members) and non-recurrent contacts (e.g. clients, contacts in supermarkets). The infection probability can differ for each contact type. All types of contacts can be assortative with respect to geographic and demographic characteristics.

The infection probabilities of contacts vary with contact type, age of the susceptible person, and the virus strain of the infected person. Moreover, they follow a seasonal pattern. The strength of the seasonality effect is higher for contacts that are easy to be moved to an outside location in summer (such as leisure contacts) and smaller for contacts that take place inside even in summer (e.g. work contacts).

Once a person is infected, the disease progresses in a fairly standard way which is depicted in Figure 1b. Asymptomatic cases and cases with mild symptoms are infectious for some time and recover eventually. Cases with severe symptoms additionally require hospitalization and lead to either recovery or death.

After rapid tests become available, people who work or go to school can receive rapid tests there. Moreover, people can decide to make a rapid test if they develop symptoms, have many planned contacts or observe cases in their contact network. People who have a positive rapid test demand a confirmatory PCR test with a certain probability. Moreover, PCR tests can be demanded because of symptoms or randomly.

This rich model of PCR and rapid tests leads to a share of detected cases that varies over time and across age groups. It also allows to quantify the effect of changes in testing policies on the dynamic of infections.

People who have symptoms, received a positive test, or had a risk contact can reduce their number of contacts across all contact types endogenously. The extent to which this is done is calibrated from survey data.

The model makes it very simple to translate policies into model quantities. For example, school closures imply the complete suspension of school contacts. A strict lockdown implies shutting down work contacts of all people who are not employed in a systemically relevant sector. It is also possible to have more sophisticated policies that condition the number of contacts on observable characteristics, risk contacts or health states.

Another key advantage of the model is that the number of contacts an individual has of each contact type can be calibrated from publicly available data (Mossong et al., 2008). This in turn allows us to estimate policy-invariant infection probabilities from time series of infection and death rates using the method of simulated moments (McFadden, 1989). Since the infection probabilities are time-invariant, data collected since the beginning of the pandemic can be used for estimation. Moreover, since we model the testing strategies that were in place at each point in time, we can correct the estimates for the fact that not all infections are observed.

Last but not least, performing simulations whose starting point is set amidst the pandemic requires special adjustments to arrive at a realistic distribution of courses of diseases. We solve the initial conditions problem by matching reported infections to individuals in our data while also correcting for reporting lag and undetected cases.

In the following sections we describe each of the model components in more detail.

4.2 Modeling Numbers of Contacts

Consider a hypothetical population of 1,000 individuals in which 50 were infected with a novel infectious disease. From this alone, it is impossible to say whether only those 50 people had contact with an infectious person and the disease has an infection probability of 1 in each contact or whether everyone met an infectious person but the disease has an infection probability of only 5 percent per contact. SEIR models do not distinguish contact frequency from the infectiousness of each contact and combine the two in one parameter that is not invariant to social distancing policies.

To model social distancing policies, we need to disentangle the effects of the number of contacts of each individual and the effect of policy-invariant infection probabilities specific to each contact type. Since not all contacts are equally infectious, we distinguish different contact types.

The number and type of contacts in our model can be easily extended. Each type of contacts is described by a function that maps individual characteristics, health states and the date into a number of planned contacts for each individual. This allows to model a wide range of contact types.

In our empirical application we distinguish the following types of contacts:

- Households: Each household member meets all other household members every day.
- Recurrent daily non-work contacts representing very close family or friendship relations outside the household.
- Recurrent weekly non-work contacts representing friendships, sports groups, charity work, or other activities. We randomize over the days on which the meetings take place.
- Random non-work contacts: Contacts with randomly drawn other people, which are assortative with respect to region and age group. This contact type reflects contacts during leisure activities, grocery shopping, medical appointments, etc..
- Recurrent daily work contacts, capturing direct colleagues.
- Recurrent weekly work contacts, representing meetings with clients, superiors, or more distant colleagues. We randomize over the days on which the meetings take place.
- Random work contacts: Working adults' contacts with randomly drawn other people, which are assortative in geographical location
- Schools: Each student meets all of his classmates every day. Class sizes are calibrated to be representative for Germany and students have the same age. Schools are closed on weekends and during vacations, which vary by states. School classes also meet three pairs of teachers every school day. The pairs are meant to represent interactions between teachers.
- Preschools: Children who are at least three years old and younger than six may attend preschool. Each group of nine children interacts with the same two adults every day. The children in each group are of the same age. The remaining mechanics are similar to schools.
- Nurseries: Children younger than three years may attend a nursery and interact with one adult. The age of the children varies within groups. The remaining mechanics are similar to schools.

[HM 11]
I started updating this to understand it better myself. In the end, I think it will be useful to group into HH / work / schools and add an introductory sentence for each group. E.g., that work is for everybody who is working — except there is a different model for teachers (?), ...

[HM 12]
True?

[HM 13]
True? age?

[HM 14]
Not updated from here on. Mayb

The number of random and recurrent contacts at the workplace, during leisure activities and at home is calibrated with data provided by Mossong et al. (2008). For details see Section 4.2. In particular, we sample the number of contacts or group sizes from empirical distributions that sometimes depend on age. It is also possible to use economic or other behavioral models to predict the number of contacts.

Theoretically, each contact type can have its own infection probability. However, to reduce the number of free parameters and thus avoid a potential over-fitting we only estimate different infection probabilities for the areas work, school, preschool and nurseries, households and other contacts.

4.3 Reducing Numbers of Contacts Through Policies

The main motivation of our model is to predict the effect of policies that affect the number of contacts people have. Examples range from school closures and lockdowns to more nuanced policies such as mandatory quarantines for symptomatic individuals or a class splitting policy where only half of the students come to school in person and the other half joins digitally with weekly rotation.

Instead of thinking of policies as completely replacing how many contacts people have, it is often more helpful to think of them as adjusting the pre-pandemic number of contacts.

Therefore, we implement policies as a step that happens after the number of contacts is calculated but before individuals are matched.

On an abstract level, a policy is a functions that modifies the number of contacts of one contact type. For example, school closures simply set all school contacts to zero. A lockdown where only essential workers are allowed to work means that approximately two thirds of the working population have zero work contacts and the rest has the same number of contacts as before.

This, in conjunction with our fine-grained contact types, allows us to easily implement a wide variety of policies. Allowing policies to depend on the health states of the entire population means that adaptive lockdowns where, for example, schools close when a certain threshold of infections is surpassed at the county level would be as simple as determining which counties are above the threshold and then setting all school contacts in these counties to zero.

The dependency of policies on health states also makes it possible to model contact tracing. For example, a policy could check whether each child has a classmate who's received a positive test result and then bar all children of that class from attending school.

Some policies can be easily implemented if the background characteristics are suitably extended. For example, a schooling policy of splitting and rotating classes, where each half attends school every other week can be implemented by storing whether the child would attend in even or odd weeks in the background characteristics and then using that information in the policy function.

For some policies the exact effect on each contact type is not easy to determine. If this refers to a policy during the estimation period, it is possible to estimate such parameters by fitting the model to time series data of infection rates. This is only possible if the policy was not active during the whole estimation period and thus the infection probabilities can be identified separately. If instead it refers to a policy that we want to simulate, we make a scenario analysis in which the model is simulated with several assumptions about how the policy affects the number of contacts.

4.4 Endogenous Contact Reductions

Policies are not the only way in which the number of contacts are reduced compared to the pre-pandemic level. It is important to model those other channels. Otherwise, the effect of policies would be overestimated and policy recommendations based on the model would be biased.

Examples of endogenous contact reductions are manifold: symptomatic people stay at home; Members of risk groups try to reduce their number of contacts more strongly than others; People self-isolate if they know they had a risk contact.

Since we model the number of contacts as arbitrary functions of background characteristics and health states, it is easy to implement such considerations.

In our current empirical application we only model that symptomatic people reduce their number of contacts across all contact types (except for households) by 70 %. Within households they reduce contacts by 50%. We are working on extending this to allow for formal and informal contact tracing as well as quarantines after positive test results. For an application of our model showcasing private contact tracing in the context of the Christmas holidays see Gabler, Raabe, Röhrl, and Gaudecker (2020).

4.5 Matching Individuals

The empirical data described above only allows to estimate the number of contacts each person has. In order to simulate transmissions of Covid-19, the numbers of contacts has to be translated into actual meetings between people. This is achieved by our matching algorithm:

As described in section 4.2, some contact types are recurrent (i.e. the same people meet regularly), others are non-recurrent (i.e. it would only be by accident that two people meet twice). The matching process is different for recurrent and non recurrent contact models.

Recurrent contacts are described by two components: 1) A variable in the background characteristics. An example would be a school class identifier which could come from actual data or be drawn randomly to achieve representative class sizes. 2) A deterministic or random function that takes the value 0 (non-participating) and 1 (participating) and can depend on the weekday, date and health state. This can be used to model vacations, weekends or symptomatic people who stay home (see section 4.4 for details).

The matching process for recurrent contacts is then extremely simple: On each simulated day, every person who does not stay home meets all other group members who do not stay home. The assumption that all group members have contacts with all other group members is not fully realistic, but seems like a good approximation to reality, especially in light of the suspected role of aerosol transmission for Covid-19 (Anderson, Turnham, Griffin, and Clarke, 2020; Morawska, Tang, Bahnfleth, Bluyssen, Boerstra, et al., 2020).

```
while are_unmatched_contacts_left:

    contact_type, i = draw_contact_type_and_individual()

    for _ in remaining_contacts[i, contact_type]:

        group_j = draw_group_of_other_person()
        j = draw_other_person_from_that_group(group_j)

        if infection_takes_place(i, j):
            update_health_state_of_freshly_infected()

        remaining_contacts[i, contact_type] -= 1
        remaining_contacts[j, contact_type] -= 1
```

Listing 1. Pseudo-code of the matching algorithm for non-recurrent contacts.

The matching in non-recurrent contact models is more difficult and implemented in a two stage sampling procedure to allow for assortative matching. Currently most contact models are assortative with respect to age (it is more likely to meet people from the same age group) and county (it is more likely to meet people from the same county) but in principle any set of discrete variables can be used. This set of variables that influence matching probabilities introduce a discrete partition of the population into groups. The first stage of the two stage sampling process samples on the group level. The second stage on the individual level.

Below, we first show pseudo code for the non-recurrent matching algorithm and then describe how the algorithm works in words.

We first randomly draw a contact type and individual. For each contact of the drawn contact type that person has, we first draw the group of the other person (first stage). Next, we calculate the probability to be drawn for each member of the group, based on the number of remaining contacts, i.e. people who have more remaining contacts are drawn with a higher probability. This has to be re-calculated each time because with each matched contact, the number of remaining contacts changes. We then draw the other individual, determine whether an infection takes place and if so update the health state of the newly infected person. Finally, we reduce the number of remaining contacts of the two matched individuals by one.

The recalculation of matching probabilities in the second stage is computationally intensive because it requires summing up all remaining contacts in that group. Using a two stage sampling process where the first stage probabilities remain constant over time makes the matching computationally much more tractable because the number of computations increases quadratically in the second stage group size.

4.6 Course of the Disease

The following medical parameters describing the progression of the disease are taken from systematic reviews (e.g. He, Lau, Wu, Deng, Wang, et al. (2020)). After an infection occurs, the disease progresses in the way depicted in Figure 1b.

First, infected individuals will become infectious after one to five days. About one third of people remain asymptomatic. The rest develop symptoms about one to two days after they become infectious. Modeling asymptomatic and pre-symptomatic cases is important because those people do not reduce their contacts or demand a test and can potentially infect many other people (Donsimoni et al., 2020). The probability to develop symptoms with Covid-19 is highly age dependent with 75% of children not developing symptoms (Davies, Klepac, Liu, Prem, Jit, et al., 2020).

A small share of symptomatic people will develop strong symptoms that require intensive care. The exact share and time span is age-dependent. An age-dependent share of intensive care unit (ICU) patients will die after spending up to 32 days in intensive care. Moreover, if the ICU capacity was reached, all patients who require intensive care but do not receive it die.

It would be easy to make the course of disease even more fine-grained. For example, we could model people who require hospitalization but not intensive care. So far we opted against that because only the intensive care capacities are feared to become a bottleneck in Germany.

We allow the progression of the disease to be stochastic in two ways: Firstly, state changes only occur with a certain probability (e.g. only a fraction of infected individuals develops symptoms). Secondly, the number of periods for which an individual remains in a state is drawn randomly. The parameters that govern these processes are taken from the literature[8]and age-dependent.

4.7 Testing

Our model includes both PCR tests which are scarce and take time until the result becomes available and rapid tests which are done after an individual's contacts are determined but before the contacts take place.

PCR testing consists of three stages. Firstly, we model who demands a PCR test. Demand functions map from individual characteristics to a probability which is the probability for this individual to demand a test. There can be multiple demand functions where each function may describe a different channel. For example, individuals who experience symptoms or have a risk contact may ask for a test. Or, the ministry of education requires a negative test result from every teacher every second week.

8. Detailed information on the calibration of the disease parameters is available as part of our online documentation.

After the probabilities for each individual and every demand model are computed, individuals who demand a test as well as the channel is sampled.

The second stage is the allocation phase in which demand and supply for tests are matched. The number of available tests can be inferred from official data and used to model shortages in supply. When demand exceeds supply, some individuals might be given preferred access to tests because of their own vulnerability or their potential to become a super-spreader.

In the last and third phase, administered tests are processed. This step can become a bottleneck in the testing process if there are not enough laboratories or necessary resources available to evaluate all tests.

In our empirical estimation we use a very simplified testing model where the number of tests to be distributed is calculated from estimates for the ratio of known to all infections.[9] Using these estimates as well as data on the test distribution over age groups by the RKI[10] we allocate tests firstly among the symptomatic and then randomly allocate tests to newly infected to fit the German test distribution.

Rapid tests are modeled much simpler. Every day before individuals have contacts they can decide to be tested. For example students that plan to attend school that day and have not done a rapid test in the last three days get a rapid test. Then they immediately receive the test result. After they have received their test result individuals can react to it by reducing their contacts. For example positively tested individuals may not go to work and reduce their household contacts to some degree. Who reduces their contacts to what degree depends on a quarantine compliance attribute.

[HM 15]

If we get econ readers, we should not use language like this because we do not have a choice model.

Our rapid tests include false positives and false negatives. The sensitivity of rapid tests in our model depends on when the individual has or will become infectious. This way we can account for the fact that rapid tests are likely to be false positive before infectiousness starts.

4.8 Initial Conditions

Consider a situation where you want to start a simulation with the beginning set amidst the pandemic. It means that several thousands of individuals should already have recovered from the disease, be infectious, symptomatic or in intensive care at the start of your simulation. Additionally, the sample of infectious people who will determine the course of the pandemic in the following periods is likely not representative of the whole population because of differences in behavior (number of contacts, assortativity), past policies (school closures), etc.. The distribution of

9. The Dunkelzifferradar project publishes daily estimates of the dark figure of infections under https://covid19.dunkelzifferradar.de/

10. https://ars.rki.de/Content/COVID19/Main.aspx

courses of diseases in the population at the begin of the simulation is called initial conditions.

To come up with realistic initial conditions, we match reported infections from official data to simulated individuals by available characteristics like age and geographic information. The matching must be done for each day of a longer time frame like a month to have individuals with possible health states. Then, health statuses evolve until the begin of the simulation period without simulating infections by contacts. We also correct reported infections for a reporting lag and scale them up to arrive at the true number of infections.

5 Calibration and Estimation

The model is described by a large number of parameters that govern the number of contacts a person has, the likelihood of becoming infected on each contact, the likelihood of developing light or strong symptoms or even dying from the disease as well as the duration each stage of the disease takes.

Most of these parameters can be calibrated from existing datasets or the medical literature. Only the infection probabilities have to be estimated inside the model by fitting it to time series data of case numbers and fatality rates.

5.1 Medical Parameters

This section discusses the medical parameters used in the model, their sources and how we arrived at the distributions used in the model.[11] See Figure 1b for a summary of our disease progression model.

5.1.1 Duration until Infectiousness and Symptoms. The first medical parameter we need is the length of the period between infection and the start of infectiousness, the so called latent period. We infer it from two other measures that are more common in the medical literature: Firstly, the time between infection and the onset of symptoms, the incubation period. Secondly, the time between the start of infectiousness and the onset of symptoms. We assume that the latency period is the same for symptomatic and asymptomatic individuals.

Once individuals become infectious a share of them goes on to develop symptoms while others remain asymptomatic. We rely on data by Davies et al. (2020) for the age-dependent probability to develop symptoms. It varies from 25% for children and young adults to nearly 70% for the elderly.

11. Additional information can be found in the online documentation.

The incubation period is usually estimated to be two to twelve days. A meta analysis by McAloon, Collins, Hunt, Barber, Byrne, et al. (2020) comes to the conclusion that "The incubation period distribution may be modeled with a lognormal distribution with pooled μ and σ parameters (95% CIs) of 1.63 (95% CI 1.51 to 1.75) and 0.50 (95% CI 0.46 to 0.55), respectively." For simplicity we discretize this distribution into four bins.

The European Centre for Disease Prevention and Control reports that people become infectious between one and two days before symptoms start.[12]

Taking these estimates together, we arrive at a latent period of one to five days.

5.1.2 Duration of Infectiousness. We assume that the duration of infectiousness is the same for both symptomatic and asymptomatic individuals as evidence suggests little differences in the transmission rates between symptomatic and asymptomatic patients (Yin and Jin (2020)) and that the viral load between symptomatic and asymptomatic individuals are similar (Zou, Ruan, Huang, Liang, Huang, et al. (2020), Byrne, McEvoy, Collins, Hunt, Casey, et al. (2020), Singanayagam, Patel, Charlett, Bernal, Saliba, et al. (2020)).

Our distribution of the duration of infectiousness is based on Byrne et al. (2020).

For symptomatic cases they arrive at zero to five days before symptom onset (figure 2) and three to eight days of infectiousness afterwards.[13]Thus, we arrive at 0 to 13 days as the range for infectiousness among individuals who become symptomatic (see also figure 5).

5.1.3 Duration of Symptoms. We use the duration to recovery of mild and moderate cases reported by Bi, Wu, Mei, Ye, Zou, et al. (2020, Figure S3, Panel 2) for the duration of symptoms for asymptomatic and non-ICU requiring symptomatic cases.

These numbers are only used for mild cases. We do not disaggregate by age. Note that the length of symptoms is not very important in our model given that individuals stop being infectious before their symptoms cease.

5.1.4 Time from Symptom Onset to Admission to ICU. The data on how many percent of symptomatic patients will require ICU is pretty thin. We rely on data by the US CDC (Stokes, Zambrano, Anderson, Marder, Raz, et al. (2020)) and the OpenABM-Project. Table 1 shows our derivations for the probabilities of requiring intensive care per age group.

For those who will require intensive care we follow Chen, Qi, Liu, Ling, Qian, et al. (2020) who estimate the time from symptom onset to ICU admission as 8.5 ± 4 days.

12. This is similar to He et al. (2020) and in line with Peak, Kahn, Grad, Childs, Li, et al. (2020).

13. Viral loads may be detected much later but eight days seems to be the time after which most people are culture negative, as also reported by Singanayagam et al. (2020).

Table 1. Shares of symptomatic patients who will require ICU care by age groups.

Age Group	Share
0-9	0.00005
10-19	0.00030
20-29	0.00075
30-39	0.00345
40-49	0.01380
50-59	0.03404
60-69	0.10138
70-79	0.16891
80-100	0.26871

Notes: The data is taken from Stokes et al. (2020) and the OpenABM-Project.

This aligns well with numbers reported for the time from first symptoms to hospitalization: Gaythorpe, Imai, Cuomo-Dannenburg, Baguelin, Bhatia, et al. (2020) report a mean of 5.76 with a standard deviation of 4. This is also in line with the durations collected by the Robert-Koch-Institut.

We assume that the time between symptom onset and ICU takes 4, 6, 8 or 10 days with equal probabilities. These times mostly matter for the ICU capacities.

5.1.5 Death and Recovery from ICU.

We take the survival probabilities and time to death and time until recovery from intensive care from the OpenABM Project.

They report time until death to have a mean of 11.74 days and a standard deviation of 8.79 days. Approximating this with the normal distribution, we have nearly 10% probability mass below 0. We use it nevertheless as several other distributions (such as chi squared and uniform) were unable to match the variance. Discretizing the distribution leads to 41% of individuals who will die from Covid-19 after one day in intensive care, 22% day after 12 days, 29% after 20 days and 7% after 32 days. Again, we rescale this for every age group among those that will not survive.

They report a mean duration of 18.8 days until recovery and a standard deviation of 12.21 days. Approximating this with the normal distribution, we have over 5% probability mass below 0. Of those who recover in intensive care, 22% do so after one day, 30% after 15 days, 28% after 25 days and 18% after 45 days.

5.2 Number of Contacts

We calibrate the parameters for the predicted numbers of contacts from contact diaries of over 2000 individuals from Germany, Belgium, the Netherlands and Luxembourg (Mossong et al., 2008). Each contact diary contains all contacts an individual had throughout one day, including information on the other person (such as age and gender) and information on the contact. Importantly, for each contact individuals

entered of which type the contact (school, leisure, work etc.) was and how frequent the contact with the other person is.

Thus, we can use the empirical distributions from this data as pre-pandemic number of contacts.

5.3 Assortative Matching

As mentioned in section 4.5, the probability that two individuals are matched can depend on background characteristics. In particular, we allow this probability to depend on age and county of residence. While we do not have good data on geographical assortativity and just roughly calibrate it such that 80% of contacts are within the same county, we can calibrate the assortative mixing by age from the same data we use to calibrate the number of contacts.

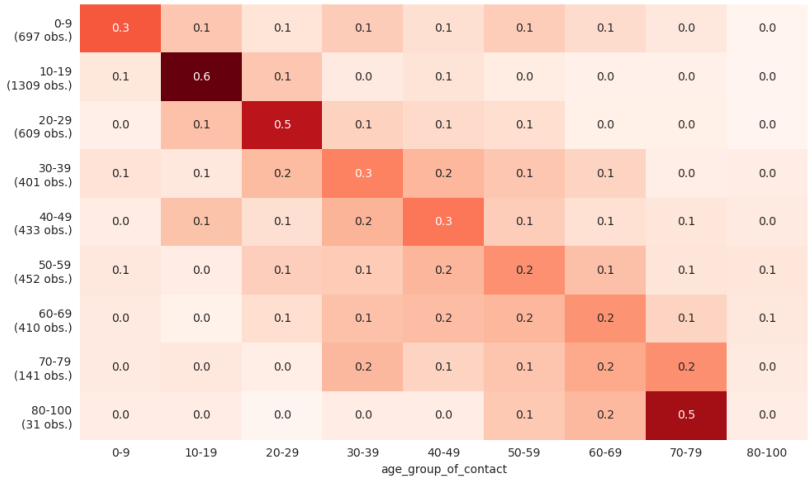

Figure 5. Distribution of random non-work contacts by age of participants.

Notes: The figure shows the distribution of random non-work contacts by age groups. A row shows the share of contacts a certain age group has with all other age groups. Higher values are colored in darker red tones. The diagonal represents the share of contacts with individuals from the same age group.

Figure 5 shows that assortativity by age is especially strong for children and younger adults. For older people, the pattern becomes more dispersed around their own age group, but within-age-group contacts are still the most common contacts.

[HM 16]
So no work in here? How about school? Can we add a column with marginals and another one with work / (school?), too?

5.4 Infection Probabilities

To calibrate infection probabilities outside of the model, it would be important to know the exact duration and distance of each contact type as well as viral loads. Since this is not available in any dataset, we estimate those parameters inside the model with the method of simulated moments (McFadden, 1989) by minimizing the

distance between simulated and observed infection rates. Since our model includes a lot of randomness, we average simulated infection rates over several model runs.

Currently, we use data for Germany from August until November. We do not use earlier periods to save computational time. Moreover, we would be worried that the there are seasonal effects that we currently do not model.

To avoid overfitting and simplify the numerical optimization problem, we only allow for four different probabilities: 1) for contacts in schools, preschools and nurseries. 2) for work contacts. 3) for households. 4) for leisure activities.

5.5 Policies

In our empirical application we distinguish four groups of contact types: households, education, work and other contacts. For households we assume that the individuals' contacts in their households do not change over our estimation period. For nurseries, preschools and schools we implement vacations as announced by the German federal states as well as school closures. For the moment we ignore both emergency childcare and that lack of childcare leads working parents to stay home. For our work models[14]we use the reductions in work mobility reported in the Google Mobility Data (Google, 2021) to calibrate our work policies. Reductions in work contacts are not random but governed through a work contact priority where the policy changes the threshold below which workers stay home. Figure 6 shows the share of workers that go to work in our model over time.

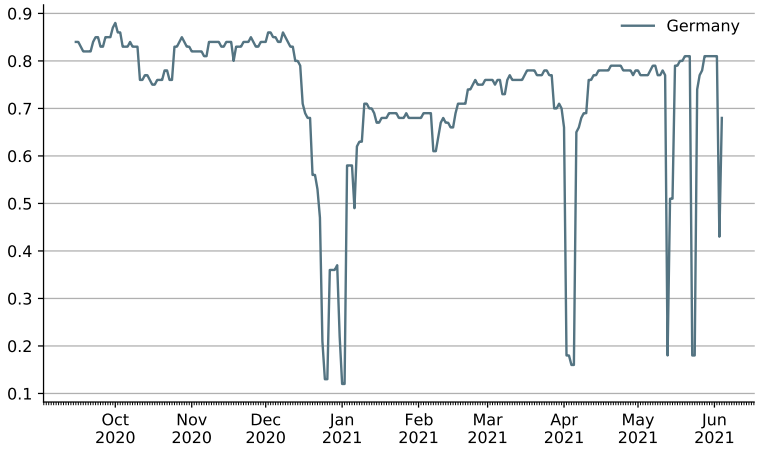

Figure 6. Share of Workers with Work Contacts

Notes: The figure shows the work mobility as reported by Google (2021). We take this as a proxy of the share of workers who are not in home office, i.e. who still have physical work contacts. The figure interpolates over weekends as we handle weekend effects through information on work on weekends in the German census data we use.

For the last group of contacts which cover things like leisure activities, grocery shopping etc. we have no reliable data by how much policies reduce them. In addition, they are likely to be affected by social and psychological factors such as pandemic fatigue and vacations. Because of this we estimate them like the infection probabilities to fit the time series data. We use very few change points and tie them to particular events such as policy announcements or particular holidays.

5.6 Rapid Test Demand

In our model, there are five reasons why rapid tests are done:

(1) someone plans to have work contacts

(2) someone is an employees of an educational facility or school pupil

(3) someone lives in a household where someone has tested positive or developed symptoms

(4) someone has developed symptoms but has not received a PCR test

(5) someone plans to participate in a weekly non-work meeting

For work contacts, we know from the COSMO study (Betsch, Korn, Felgendreff, Eitze, Schmid, et al. (2021), 20th/21st of April) that 60% of workers who receive a test offer by their employer regularly use it. We assume this to be time constant.

In addition, there are some surveys that allow us to trace the expansion of employers who offer tests to their employees. Mid march, 20% of employers offered

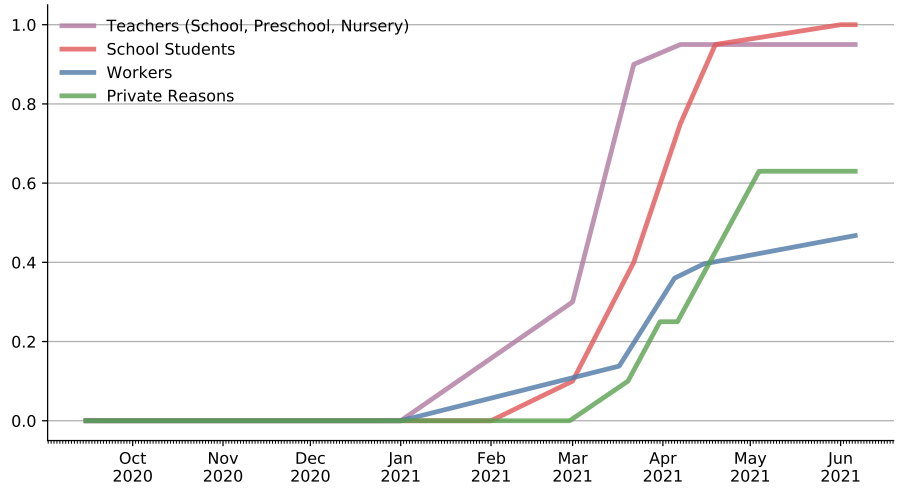

Figure 7. Share of Individuals that do a Rapid Test. Talk about the interpretation of each line.

14. We distinguish non-recurrent work contacts, daily work contacts and weekly work contacts.

tests to their employees (DIHK, 2021). In the second half of March, 23% of employees reported being offered weekly rapid tests by their employer (Ahlers, Lübker, and Jung, 2021). This share increased to 60% until the first days of April Fernsehen (2021). Until mid April 70% of workers were expected to receive a weekly test offer (ÄrzteZeitung, 2021). However, according to surveys conducted in mid April (Betsch et al., 2021), less than two thirds of individuals with work contacts receive a test offer. Starting on April 19th employers were required by law to provide two weekly tests to their employees (Bundesanzeiger, 2021). We assume that compliance is incomplete and only 80% of employers actually offer tests.

Sources still missing below this

We assume that employees in educational facilities start getting tested in 2021 and that by March 1st 30% of them are tested weekly. The share increases to 90% for the week before Easter. At that time both Bavaria and Baden-Württemberg were offering tests to teachers and North-Rhine Westphalia and Lower Saxony were already testing students and tests for students and teachers were already mandatory in Saxony. After Easter we assume that 95% of teachers get tested twice per week.

Tests for students started later so we assume that they only start in February and only 10% of students get tested by March 1st. Relying on the same sources as above we approximate that by the week before Easter this share had increased to 40%.

After Easter we assume the share of students receiving twice weekly tests to based on tests already being mandatory in North-Rhine Westphalia and Bavaria while still being voluntary in Baden-Württemberg. There tests become mandatory on April 19th.

Add a section on how we calibrate rapid test demand; Mainly describe the datapoints we have and say that we usually interpolate linearly in between data points. (Only exception to that is private rapid test demand, which we fit to data)

6 Model Validation

This compares simulated data from our model with empirical data from Germany. We at observed infections, fatality rates, the spread of the B117 mutation, vaccinations and rapid test demands. Where available we do not only look at aggregated statistics but also analyze the model fit for age groups.

summarize the fit; Some of this needs to go in the appendix. I suggest the age group and federal state fit goes there???

6.1 Observed Infections

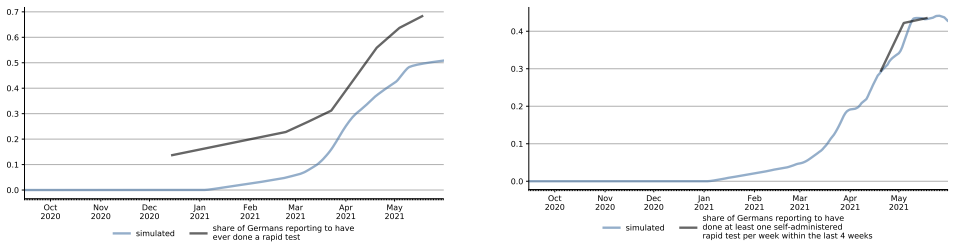

Figure 8. Share of Individuals With Rapid Tests

Notes: The figure compares the share of individuals who have ever done a rapid test or done a rapid test within the last week in our simulations to the shares reported in the COVID-19 Snapshot Monitoring survey. The left panel compares the share of individuals who have ever done a rapid test. The right panel compares the share of individuals who have done a rapid test within the last seven days in our simulation compared to the share reporting to have done at least weekly rapid tests in the last four weeks in the COSMO survey. Overall our calibration of rapid tests are slightly conservative. The overall share is below that in the study. We fit the share of weekly tests quite exactly. However, the study only covers adults while our share also includes children who are tested very regularly when attending school.

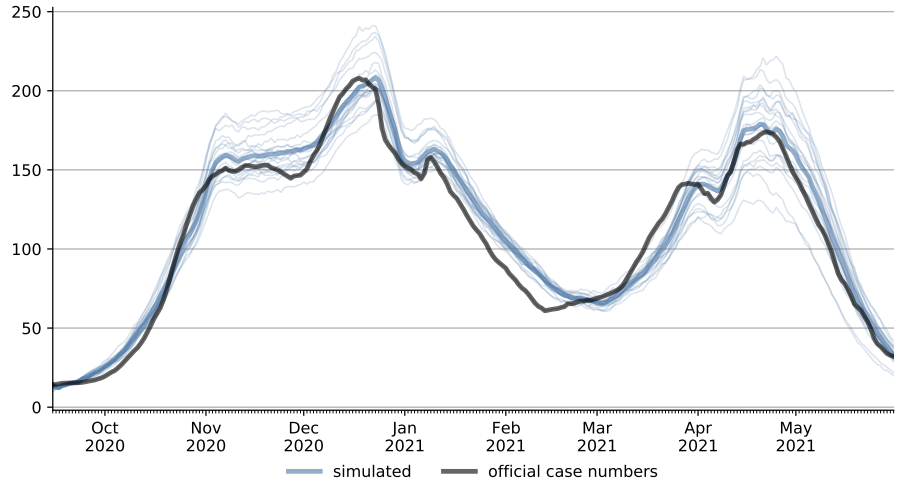

Figure 10. Simulated and Empirical Infections

Notes: The figure shows the weekly incidence rates per 100,000 people for the reported versus the simulated infections rates.

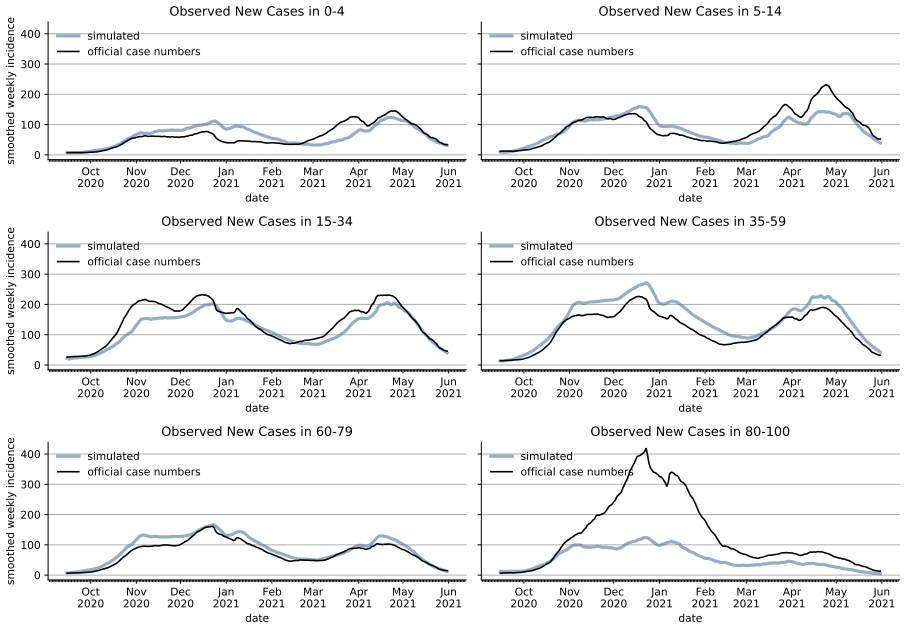

Figure 11. Simulated and Empirical Infections by Age Group

Notes: The figure shows the weekly incidence rates per 100,000 people for the reported versus the simulated infections rates for different age groups.

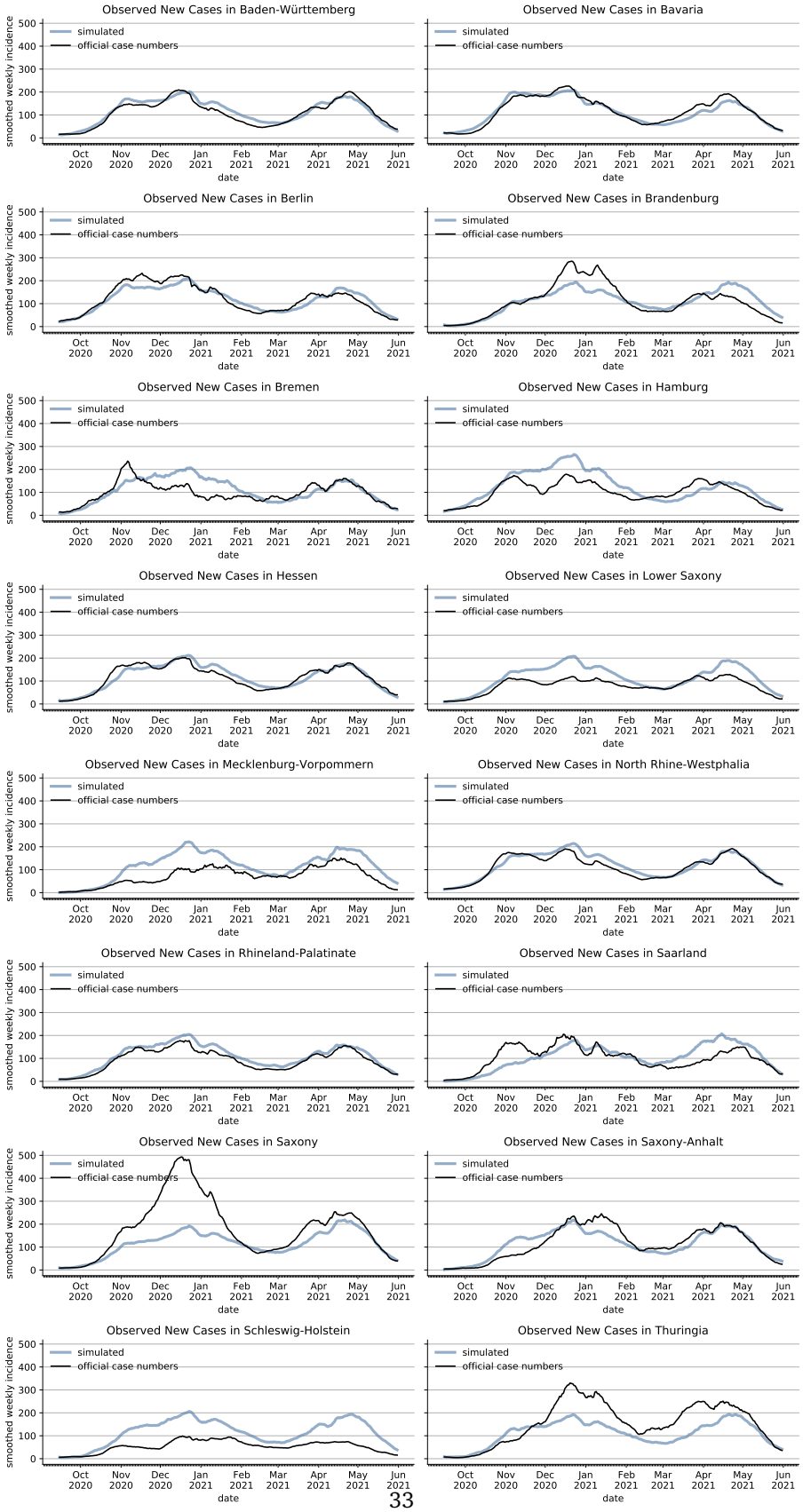

Figure 12. Simulated and Empirical Infections by Federal State

Notes: The figure shows the weekly incidence rates per 100,000 people for the reported versus the simulated infections rates for different federal states.

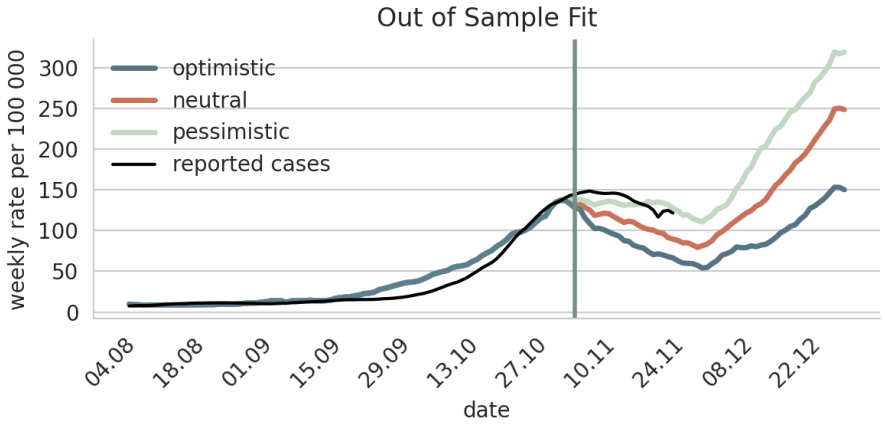

Figure 13. Predicted effect of the "Lockdown Light" on infection rates.

Notes: For the time period until the beginning of November, the figure shows the weekly incidence rates of infections per 100,000 people from reported (black) versus simulated (blue) data. With the start of November, the projections of the three scenarios, optimistic (blue), neutral (red), and pessimistic (mint green), are shown until the beginning of the Christmas holidays. The actual incidence rates (black) are reported until the 24th November.

7 The Spread of the B117 Mutation

We don't have this figure in the repo yet, but the fit is perfect

7.1 Out-of-Sample Fit

We can assess the out-of-sample fit by projecting the effect of the lockdown light and comparing it to case numbers until mid of November. It is important to note that this is not just a simple extrapolation of a time trend because the lockdown light only started after the estimation period. The out-of-sample fit can be assessed in Figure 13.

The model correctly predicts the effect of the lockdown light with reasonable accuracy. In particular, the actual case numbers are between our neutral and pessimistic projection. The plot also shows that ending the lockdown light on November 30, as was originally planned, would lead to an explosive growth in case numbers in all scenarios.

8 Results

This is the results section.

The results can be found in 'figures/results'. This includes both figures and tables for lookup of numbers and summary tables.

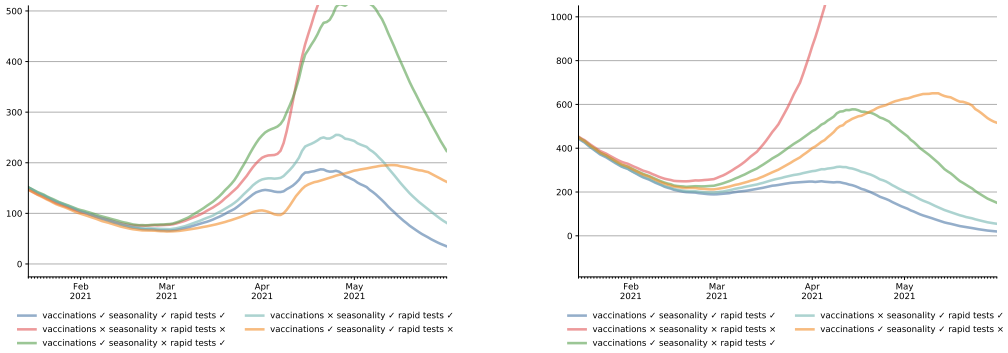

Figure 14. The Effect of Policies on Observed and Unobserved Cases

Notes: ...

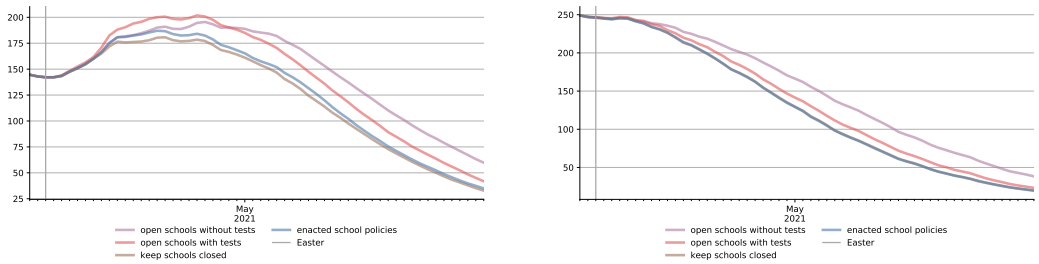

Figure 15. The Effect of Different School Scenarios on Observed and Unobserved Cases

Notes: K: One of the scenarios starts too early. Will be fixed with the next full simulations run.

scenario	predicted total infections among 5-14 year olds from Easter until 2021
educ open after easter without tests	7
educ open after easter with tests	5
close educ after easter	4
baseline	4

9 Conclusion

We propose a simulation based model of infectious disease transmission that is designed to predict the effects of fine-grained social distancing policies. In particular, the model can be used to model policies such as several ways of splitting school classes or work reduction policies that affect essential and non-essential workers differentially. Both policies would be hard to implement in standard SEIR or agent based simulation models.

To predict the effects of such policies, it is not only important to have a way of expressing such flexible policies in terms of model quantities, but also to incorporate heterogeneity in disease progression as well as meeting patterns. We calibrate age dependent disease progression parameters from the medical literature and age dependent contact frequencies from contact diaries. Moreover, we distinguish ten types of contacts out of which some are only relevant for certain age groups.

The model has a good fit on past German case numbers and passes an out of sample validation despite a drastic change in the policy environment between the estimation period and the validation period.

Despite these encouraging results we still see the model as work in progress. We plan to implement more features, in particular allowing age and symptoms to affect the probability to seek and receive a test, opening the way to show the effect of different testing policies, such as those proposed by Tröger et al. (2020). Moreover, the estimation of the infection probabilities and the model fit will improve as more data becomes available.

We invite researchers from any discipline, but particularly epidemiologists to provide feedback on the model and welcome collaborations.