

An Agent-Based Model of COVID-19 Diffusion to Plan and Evaluate Intervention Policies

Gianpiero Pescarmona, Pietro Terna, Alberto Acquadro, Paolo Pescarmona, Giuseppe Russo, Emilio Sulis, and Stefano Terna

Abstract A model of interacting agents, following plausible behavioral rules into a world where the Covid-19 epidemic is affecting the actions of everyone. The model works with (i) infected agents categorized as symptomatic or asymptomatic and (ii) the places of contagion specified in a detailed way. The infection transmission is related to three factors: the characteristics of both the infected person and the susceptible one, plus those of the space in which contact occurs. The model includes the structural data of Piedmont, an Italian region, but we can easily calibrate it for other areas. The micro-based structure of the model allows factual, counterfactual, and conditional simulations to investigate both the spontaneous or controlled development of the epidemic.

The model is generative of complex epidemic dynamics emerging from the consequences of agents' actions and interactions, with high variability in outcomes, but frequently with a stunning realistic reproduction of the successive contagion waves in the reference region. There is also an inverse generative side of the model, coming

G. Pescarmona
University of Torino, Italy, e-mail: gianpiero.pescarmona@unito.it

P. Terna
University of Torino, Italy; Fondazione Collegio Carlo Alberto, Italy e-mail: pietro.terna@unito.it

A. Acquadro
University of Torino, Italy e-mail: alberto.acquadro@unito.it

P. Pescarmona
University of Groningen, The Netherlands e-mail: p.p.pescarmona@rug.nl

G. Russo
Centro Einaudi, Torino, Italy e-mail: russo@centroeinaudi.it

E. Sulis
University of Torino, Italy; e-mail: emilio.sulis@unito.it

S. Terna
tomorrowdata.io e-mail: stefano.terna@tomorrowdata.io

from constructing a meta-agent optimizing the vaccine distribution among people groups—characterized by age, fragility, work conditions—to minimize the number of symptomatic people, using genetic algorithms.

1 A quick introduction to our agent-based epidemic model

The starting point is a compartmental model with Susceptible, Infected, and Recovered people (S.I.R.), but adding both a more detailed breakdown of the subjects involved in the contagion process [1] and a multi-scale framework to account for the interaction at different dimensional, and spatial levels [2]. From the virus micro-level, we move to individuals and up to the collective behavior of the population.

Following [3], we know that the analysis based on the assumption of heterogeneity strongly differs from S.I.R. compartmental structures modeled by differential equations. Their work ponders when it is better to use agent-based models and when it would be better to use differential equation models. Differential equation models assume homogeneity and perfect mixing of characteristics within compartments, while agent-based models can capture heterogeneity in agent attributes and the structure of their interactions. We follow the second approach.

- The model works with:
 - i infected agents categorized as symptomatic or asymptomatic and
 - ii the places of contagion specified in a detailed way, thanks to agent-based modeling capabilities.
- The infection transmission is related to three factors: the infected person's characteristics and those of the susceptible one, plus those of the space in which a contact occurs.

Finally, we subscribe the call of [4] to «cover the full behavioural and social complexity of societies under pandemic crisis» and we work arguing that «the study of collective behavior must rise to a “crisis disciplin” just as medicine, conservation, and climate science have, with a focus on providing actionable insight to policymakers and regulators for the stewardship of social systems», as in [5].

Now a look at the structure of the whole presentation.

In Section 1.1, we discuss models and specifically agent-based models; in Section 1.2, the biochemical support to agents' intrinsic susceptibility construction; in Section 1.3, the structure of the model, with the daily sequence of the agents' actions. Section 2 introduces a detailed description of the internal model mechanisms, with conditional actions in Section 2.1, parameters in Section 2.2 and agents' interaction in Section 2.3.

A technique for contagion representation is reported in Section 3. Then we explore simulation cases in Section 4, building several batches of runs in Section 4.1 and comparing extreme situations in Sections 4.2.

Section 4.3 introduces the actual epidemic data of the reference region. With those data, we verify factual and counterfactual analyses in Section 5. Considering the possibility of calculating infection indicators without delays (Section 5.4), we experiment with the effect of adopting the control measure with 20 days of anticipation (Section 5.5). In Section 5.6 we verify another counterfactual policy, that of concentrating the efforts uniquely in defense of fragile persons. Section recap 5.7 summarizes the results.

The final application of the model is dedicated to a planning exercise on vaccination campaigns (Section 6). We introduce an analysis of the vaccine mechanism in the perspective of our model (Section 6.1) and using genetic algorithms to optimize the behavior of a meta-agent deciding the sequence of the vaccinations (Section 6.7). Finally, a specific, realistic case is deeply analyzed in Section 6.3.

1.1 Why model? Why agents? Why another model?

Why another model, and most of all, why model? With [6]:

The choice (...) is not whether to build models; it's whether to build explicit ones. In explicit models, assumptions are laid out in detail, so we can study exactly what they entail. On these assumptions, this sort of thing happens. When you alter the assumptions that is what happens. By writing explicit models, you let others replicate your results.

With even more strength:

I am always amused when these same people challenge me with the question, "Can you validate your model?" The appropriate retort, of course, is, "Can you validate yours?" At least I can write mine down so that it can, in principle, be calibrated to data, if that is what you mean by "validate" a term I assiduously avoid.

To reply to "why agents?", with [7] we define in short what an agent-based model is:

An agent-based model consists of individual agents, commonly implemented in software as objects. Agent objects have states and rules of behavior. Running such a model simply amounts to instantiating an agent population, letting the agents interact, and monitoring what happens. That is, executing the model—spinning it forward in time—is all that is necessary in order to "solve" it.

More in detail:

There are, ostensibly, several advantages of agent-based computational modeling over conventional mathematical theorizing. First, [...] it is easy to limit agent rationality in agent-based computational models. Second, even if one wishes to use completely rational agents, it is a trivial matter to make agents heterogeneous in agent-based models. One simply instantiates a population having some distribution of initial states, e.g., preferences. That is, there is no need to appeal to representative agents. [...] Finally, in most social processes either physical space or social networks matter. These are difficult to account for mathematically except in highly stylized ways. However, in agent-based models it is usually quite easy to have the agent interactions mediated by space or networks or both.

And now, "why another?" As a commitment to our creativity, using our knowledge to understand what is happening. Indeed, with arbitrariness: it is up to others and time to judge. Modeling the Covid-19 pandemic requires a scenario and the actors.

As any model, also this one is based on assumptions: time will tell whether these were reasonable hypotheses. Modeling the Covid-19 pandemic requires a scenario and the actors. As in a theater play, the author defines the roles of the actors and the environment. The characters are not real, they are prebuilt by the author, and they act according to their peculiar constraints. If the play is successful, it will run for a long time, even centuries. If not, we will rapidly forget it. Shakespeare's Hamlet is still playing after centuries, even if the characters and the plot are entirely imaginary. The same holds for our simulations: we are the authors, we arbitrarily define the characters, we force them to act again and again in different scenarios. However, in our model, the micro-micro assumptions are not arbitrary but based on scientific hypotheses at the molecular level, the micro agents' behaviors are modeled in an explicit and realistic way. In both plays and simulations, we compress the time: a whole life to two or three hours on the stage. In a few seconds, we run the Covid-19 pandemic spread in a given regional area.

1.2 Here a Gianpiero's section on the biochemical of the contagion

1.3 Our model

With our model, we move from a macro compartmental vision to a meso and micro-analysis capability. Its main characteristics are:

- scalability: we take in account the interactions between virus and molecules inside the host, the interactions between individuals in more or less restricted contexts, the movement between different environments (home, school, workplace, open spaces, shops); the movements occur in different parts of the daily life, as in [8];
in detail, the scales are:
 - *micro*, with the internal biochemical mechanism involved in reacting to the virus, as in [9], from where we derive the critical importance assigned to an individual attribute of intrinsic susceptibility related to the age and previous morbidity episodes; the model indeed incorporates the medical insights and consistent perspectives of one of its co-authors, former full professor of clinical biochemistry, signing also the quoted article; a comment on Lancet [10] consistently signals the syndemic character of the current event: «Two categories of disease are interacting within specific populations—infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and an array of non-communicable diseases (NCDs)»;
 - *meso*, with the open and closed contexts where the agents behave, as reported above;

- *macro*, with the emergent effects of the actions of the agents;
- *granularity*: at any level, the interactions are partially random and therefore the final results will always reflect the sum of the randomness at the different levels; changing the constraints at different levels and running multiple simulations should allow the identification of the most critical points, where to focus the intervention.

Summing up, S.I.s.a.R. [11] is an agent-based model designed to reproduce the diffusion of the COVID-19 using agent-based modeling in NetLogo [12]. We have Susceptible, Infected, symptomatic, asymptomatic, and Recovered people: hence the name S.I.s.a.R. The model works on the structural data of Piedmont, an Italian region, but we can quite easily calibrate it for other areas. It can reproduce the events following a realistic calendar (national or local government decisions, as in Section 2.2), via its script interpreter. The model is online at <https://terna.to.it/simul/SIsaR.html>, from where it is also possible to run the code without installation. Into the *Info* sheet of the model, we have more than 20 pages of Supporting Information about both the structure and the calibration of the model.

The micro-based structure of the model allows factual, counterfactual, and conditional simulations to investigate both the spontaneous or controlled development of the epidemic. Examples of counterfactual situations are those considering:

- i different timing in the adoption of the non-pharmaceutical containment measures;
- ii alternative strategies focusing exclusively on the defense of fragile people.

The model generates complex epidemic dynamics, emerging from the consequences of agents' actions and interactions, with high variability in outcomes, but frequently with a stunning realistic reproduction of the contagion waves that occurred in the reference region.

We take charge of the variability of the epidemic paths within the simulation, running batches of executions with 10,000 occurrences for each experiment.

Following [13], the AI and inverse generative side of the model comes from constructing a meta-agent optimizing the vaccine distribution among people groups—characterized by age, fragility, work conditions—to minimize the number of symptomatic people (deceased persons come from there).

We can characterize the action of the planner both:

- i introducing ex-ante rules following “plain” or “wise” strategies that we imagine as observers or
- ii evolving those strategies via the application of a genetic algorithm.

The genome is a matrix of vaccination quotas by people groups, with their time range of adoption.

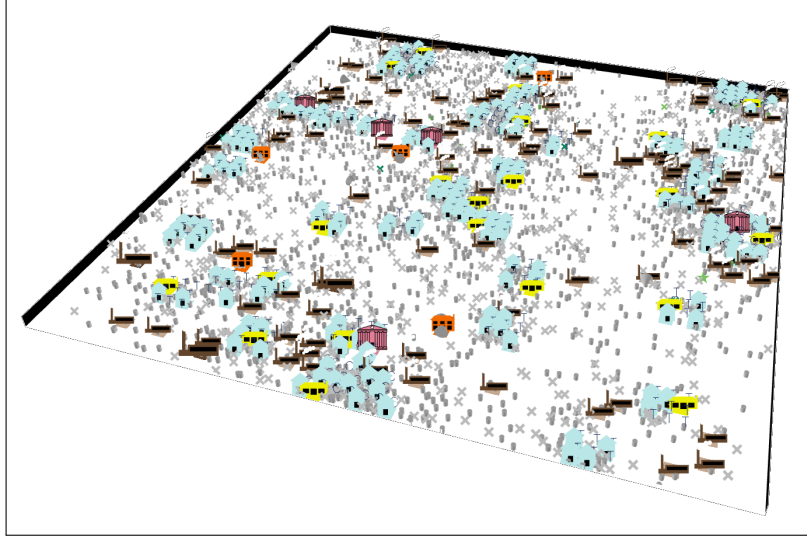


Fig. 1: A 3D representation of the model world

2 How S.I.s.a.R. works

We have two initial infected individuals in a population of 4350 individuals, on a scale of 1:1000 with Piedmont. The size of the initial infected group is out of scale: it is the smallest number, ensuring the epidemic's activation in a substantial number of cases. Initial infected people bypass the incubation period. For implausibility reasons, we never choose initial infected people among persons in nursing homes or hospitals. The presence of agents in close spaces—such as classrooms, factories, homes, hospitals, nursing homes—is made with realistic numbers, not to be read in scale: e.g., a classroom contains 25 students, a home two persons, large factories up to 150 employees, small ones up to 15, etc.; the movements occur in different parts of the daily life, as in [8].

In Fig. 1 we have a 3D representation of the model world—as one of the possible random maps that the simulation generates— with persons in gray, houses in cyan, nursing homes in orange, hospitals in orange, schools in yellow, factories (with shops and offices) in brown. Persons have a cylinder as shape, if regular; a capital X if fragile; temporary their colors can be: red, if symptomatic; violet, if asymptomatic; turquoise, if symptomatic recovered; green, if asymptomatic recovered.

We use random maps to have a neutral representation of the structure of the space when doing the batches of 10,000 repetitions of the simulation.

We can set:

- min and max duration of the individual infection;
- the length of the incubation interval;

- the critical distance, i.e., the radius of the possibility of infection in open air, with a given probability;
- the correction of that probability, due to the personal characteristics of both active and the passive agents; passive agents, as receivers, can be robust, regular, fragile, and extra fragile.

We have two main types of contagion: (a) within a radius, for people moving around, also if only temporary present in a house/factory/nursing home/hospital (in schools we only have students and teachers); (b) in a given space (room or apartment) for people resident in their home or in a hospital or in a nursing home or being in school or in a working environment.

People in hospitals and nursing homes can be infected in two ways: (a) and (b). Instead, while people are at school, they can only receive the disease from people in the same classroom, where only teachers and students are present, so this is a third infection mechanism (c). In all cases, the personal characteristics of the recipients are decisive.

We remark that workplaces are open to all persons, as clients, vendors, suppliers, external workers can go there. In contrast, schools are mainly reserved for students and school operators and are less affected by contact with other types of agents.

All agents have their home, inside a city, or a town. The agents also have usual places (UPs) where they act and interact, moving around. These positions can be interpreted as free time elective places. When we activate the schools, students and teachers have both UPs and schools; healthcare operators have both UPs and hospitals or nursing homes; finally, workers have both UPs and working places. In each day (or tick of the model), we simulated realistic sequences of actions.

Fig. 2 describes what happens during every *day* in our simulated world, with each day as a sequence of several actions.

2.1 Conditional actions

Agents' movements in space, to go to work, school, and other UPs are subject to two interrelated general conditions.

- I Symptomatic persons are at home or in a hospital or a nursing home and do not move.
- II People not constrained by *condition I* can move if (primary rule) there are no general limitations (e.g., lockdown) *OR* one of the following specific sub-conditions applies:
 - a. agents who are hospital healthcare operators or nursing home healthcare operators;
 - b. all people, according to the probability of moving of the whole non-symptomatic agents (2.2, iii);
 - c. regular people, according to the probability of moving of the regular non-symptomatic agents (2.2, iv);

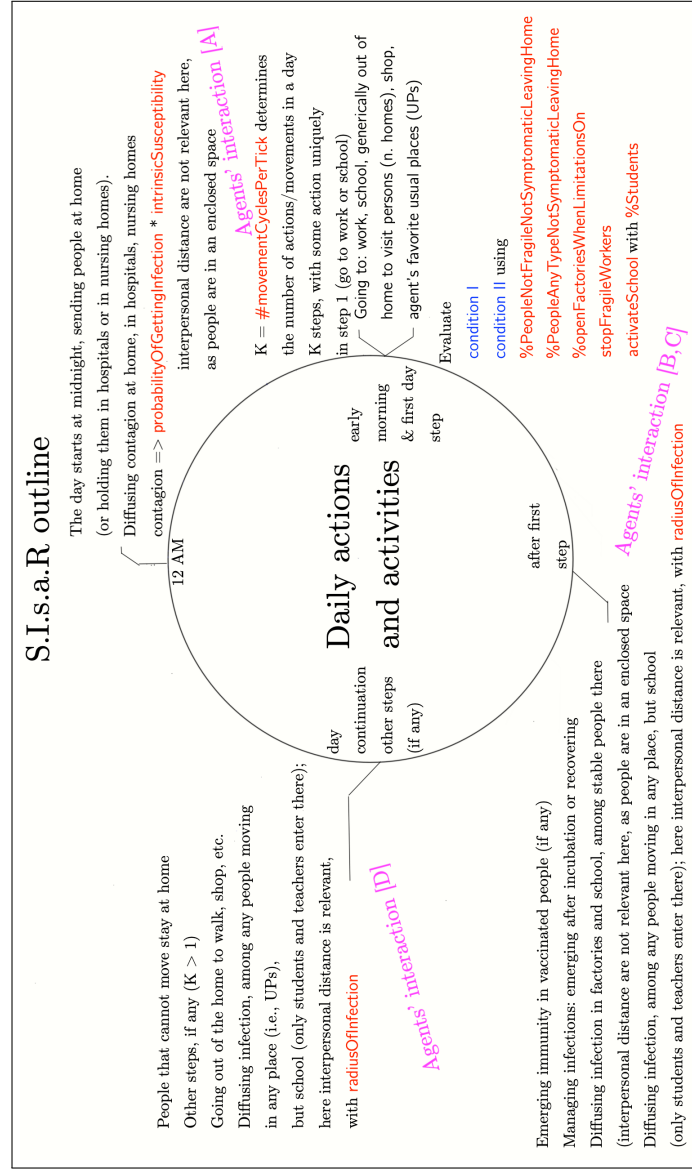


Fig. 2: A day in the simulation, with N repetitions where N is the duration of a specific outbreak; look at: Section 2.1 for the rules of the conditional actions; Section 2.2 for the parameter definitions; and Section 2.3 for details on the agent interactions

- d. workers, if all the factories are open or it is open their workplace (2.2, v);
- e. teachers, if the schools are open (2.2, vi);

- f. students, if the schools are open, but with a possible quota limitation (2.2, vii).

2.2 Parameter definition

We define here the parameters of Fig. 2, also with the short names used in program scripts, in round brackets. The values of the parameters are reported in detail in Appendix 1 - Parameter values (Section 8).

- i *probabilityOfGettingInfection* (**prob**) is the base probability of getting infected, to be multiplied by the *intrinsicSusceptibility* factor (ii); it is activated if the subject is within a circle of radius (viii) with an infected person; values at (8, i);
- ii the *intrinsicSusceptibility* is based on *intrinsicSusceptibilityFactor* set to 5 in Eq. 1

$$\text{intrinsicSusceptibility} = \text{intrinsicSusceptibilityFactor}^{\text{groupFragility}} \quad (1)$$

with *groupFragility* exponent set to:

- 1 for extra-fragile persons,
- 0 for fragile persons,
- 1 for regular persons,
- 2 young people from 0 to 24 years old;

- iii *%PeopleAnyTypeNotSymptomaticLeavingHome* (**%PeopleAny**) determines, in a probabilistic way, the number of people of any kind going around in case of limitations/lockdown; the limitations operate only if the lockdown is on (into our simulated world, from day 20); values at (8, iii);
 - iv *%PeopleNotFragileNotSymptomaticLeavingHome* (**%PeopleNot**) determines, in a probabilistic way, the number of regular people going around in case of limitations/lockdown; as above, the limitations operate only if the lockdown is on (into our simulated world, from day 20); values at (8, iv);
- the parameters iii and iv in some time interval change very frequently, reproducing into the model the uncertainty of the decisions that were happening in the real world in the same periods;

NB, the parameters iii and iv produce independent effects, as in the following examples: (a) the activation of *%PeopleAny* at 31, 0 and, simultaneously, of *%PeopleNot* at 31, 80, means that people had to stay home on that day, but people specifically not fragile could go out in 80% of the cases; (b) *%PeopleAny* at 339, 80 and, simultaneously, *%PeopleNot* at 339, 100 means that fragile and not fragile persons cannot always go around, but only in the 80% of the cases; instead, considering uniquely non-fragile persons they are free to go out; the construction is an attempt to reproduce a fuzzy situation; in future versions of the model, we will define the quotas more straightforwardly:

- %FragilePeopleNotSymptomaticLeavingHome;
 - %NotFragilePeopleNotSymptomaticLeavingHome;
- v *%openFactoriesWhenLimitationsOn* (%Fac) determines, in a probabilistic way, what factories (small and large industries, commercial surfaces, private and government offices) are open when limitations are on; if the factory of a worker is open, the subject can go to work, avoiding restrictions (but uniquely in the first step of activity of each day); values at (8, v);
- vi *stopFragileWorkers* (sFW) is *off* (set to 0) by default; if *on* (set to 1), fragile workers (i.e., people fragile due to prior illnesses) can move out of their homes following the iii and iv parameters, but cannot go to work; the regular case is that the workers (fragile or regular) can go to their factory (if open) also when limitations are on; values at (8, vi);
alternatively, we also have the *fragileWorkersAtHome* parameter; if *on* (set to 1) the total of the workers is unchanged, but the workers are all regular; we can activate this counterfactual operation uniquely at the beginning of the simulation;
- vii when *activateSchools* (aSch) is *on* (set to 1), teachers and students go to school avoiding restrictions (but uniquely in the first step of activity of each day); *%Students* (%St) limits to its value the quota of the students moving to school; the residual part is following the lessons from home; values at (8, vii);
- viii following *radiusOfInfection* (radius), the effect of the contagion—outside enclosed spaces, or there, but for temporary presences—is possible within that distance; values at (8, viii).

2.3 Agents' interaction

We underline that our simulation tool is not based on micro-simulation sequences, calculating the contagion agent by agent, on the base of their characteristics and ex-ante probabilities. It implements a true agent-based simulation, with the agents acting and, most of all, interacting, thus generating continuously contagion situations.

Each run creates a population with expected characteristics, but also with random specifications, to assure the heterogeneity in agents. The daily choices of the agents are partially randomized, to reproduce real-life variability.

Contagions arise from agents' interactions, in four situations, as specified in Fig. 2:

- A - in houses (at night), hospitals, nursing homes;
- B - in schools, workplaces in general, among people stable there;
- C - in the places above (excluding schools) by people temporary there and in open spaces (UPs above);
- D - interactions mainly in open spaces (UPs above).

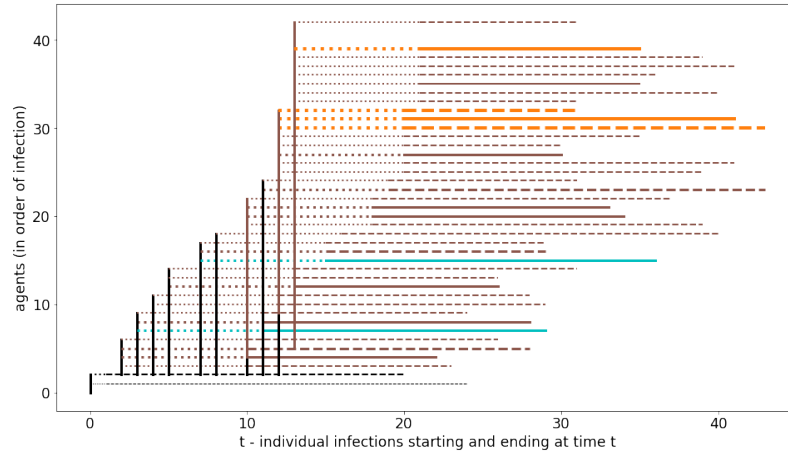


Fig. 3: A case with containment measures, first 40 infections: workplaces (brown) and nursing homes (orange) strictly interweaving

3 Contagion representation

PROVVISORIO

collegare a Appendix 2 - A gallery of contagion sequences (Section 9)

- The model allows analyzing the sequences of contagions in simulated epidemics, reporting the places where the contagion occur.
- We represent each infected agent as a horizontal segment (from the starting date to the final date of the infection) with vertical connections to other agents if they receive the disease.

We represent the new infected agents via further segments at an upper level.

- With colors, line thickness, and styles, we display multiple information.
- This enables understanding at a glance how an epidemic episode is developing. In this way, it is easier to reason about countermeasures and, thus, to develop intervention policies.

Examples.

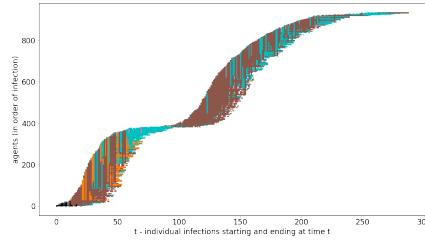


Fig. 4: A Case with containment measures, the whole epidemics: workplaces (brown) and nursing homes (orange) and then houses (cyan), with a bridge connecting two waves

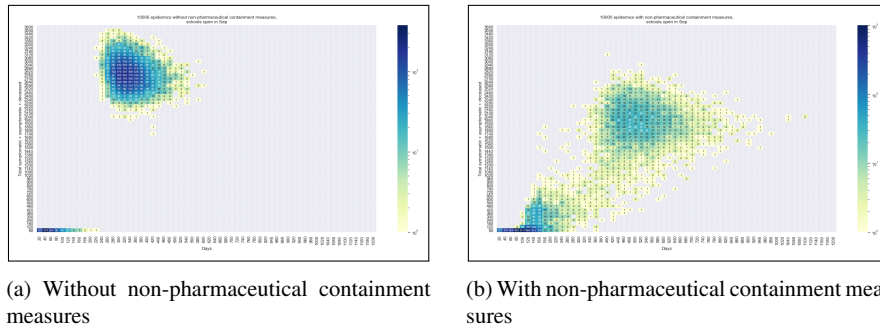


Fig. 5: Starting point: 10,000 epidemics in Piedmont

4 Exploring cases

4.1 Simulation batches

4.2 Epidemics without and with control

In Figs. 5a and 5b we . . .

4.3 Actual data

The critical points for our experiments with the simulation model of the epidemic in Piedmont are Summer and Fall 2020, as in Fig. 6a, where we have the time series of the first part of Piedmont's actual epidemic. The blue line represents the cumulative number of infected persons. Initially, only symptomatic cases were accounted for, but after the 2020 Summer, more generalized tests and asymptomatic patients are included.

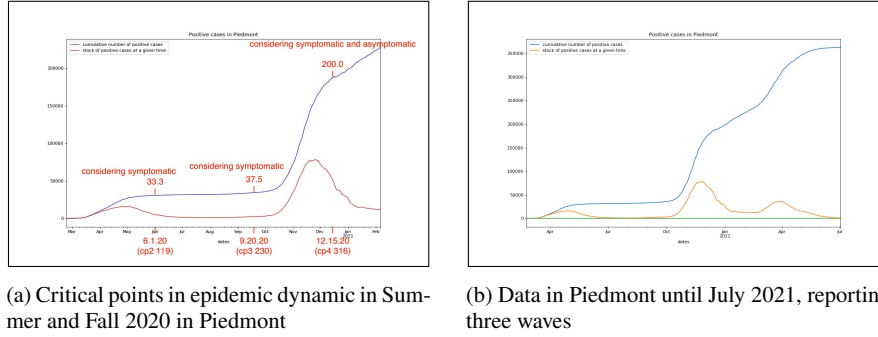


Fig. 6: Actual data

From <http://www.protezionecivile.it/web/guest/department>, the Italian Civil Protection Department web site, we find at <https://github.com/pcm-dpc/COVID-1>, i.e., the repository of regional data. Initially, only symptomatic cases were accounted for, but after the 2020 Summer, more generalized tests and asymptomatic patients are included. We had data about symptomatic infected people in the first wave, but from October 2020, data are mixed. In the above *git* repository, in October and November, we had “Positive cases emerged from clinical activity”, unfortunately then reported as “No longer populated” (from the end of November, our observation) and “Positive cases emerging from surveys and tests, planned at national or regional level”, again “No longer populated” (from the end of November, our observation). As a consequence, the subdivision between symptomatic and asymptomatic cases is no longer possible.

Considering the dynamic of the data in Fig. 6a, we search within the simulation batch for cases with both:

- i numbers of infected persons quite similar at cp2 and at cp3; besides, numbers not too different from those of the figure;
With *cp*, we indicate the internal check points of the simulation program. In Fig. 6a we also report the number of days from the beginning of the epidemic for each check point.
- ii the number of infected persons at cp4 has to be significantly greater than those at the previous check point.

In a lot of cases, epidemics satisfying condition (i) fail to match condition (ii); both the situations happen only in less than the 1.5% of the instances in the batch of ten thousand epidemic. We can conclude that the second wave registered in Piedmont after the Summer stability is due to new infected agents coming from outside and restarting the contagion process.

Other critical points in our analysis are the day on which the vaccination campaign starts, 373 of the simulation (Feb. 12th, 2021), and the day of the effectiveness of the initial vaccinations, 40 days later, day 413 (Mar. 22nd, 2021). At those dates, within

(1000) cum. v.	Jun 1, 20 sym.	Sep 9, 20			Dec 15, 20			Feb 1, 21		May 1, 21		Dec 15, 20		to end	days
		all	sympt.	totalInf.	sympt.	totalInf.	sympt.	totalInf.	sympt.	totalInf.	sympt.	totalInf.	sympt.	totalInf.	
count	1407.0	1407.0	1407.0	1407.0	1044.0	1044.0	1005.0	1005.0	980.0	980.0		1044.0	1044.0	1044.0	
mean	35.6	72.7	40.0	84.1	180.4	462.1	354.1	900.4	623.8	1563.3		726.6	1810.9	620.9	
std	14.1	42.6	16.7	52.8	134.6	354.6	213.8	535.4	217.9	527.0		221.9	544.0	110.8	

Table 1: Second wave, new infections from outside, without specific measures

the simulations, we can find either the presence of many infected agents or of few ones, as effectively was the situation in Piedmont.

As a documentation, in Fig. 6b the time series cover the whole the period.

5 Factual and counterfactual analyses

In Fig. 7 we collect the heat-maps of the cases in Sections 5.1, 5.2, 5.3, 5.5, and 5.6.

5.1 Spontaneous second wave, without specific measures

5.2 Second wave, new infections from outside, without specific measures

1407 cases; epidemics stable in Summer 2020 out of 10,000, rule: at Jun 1, 20 select if sym. (10, 70], actual v. 33.3 & at Sep 20, 20 select if sym. (20, 90], actual value 37.5; 1044 cases; at Dec 15, 20, rule: sym.+asym.>Sep 20, 20, actual value: 200.0.

Fig. 7b and Table 1 refer to . . .

5.3 Second wave, new infections from outside, with new specific measures

5.4 Here a Stefano's section on calculating the infection indicator without delays

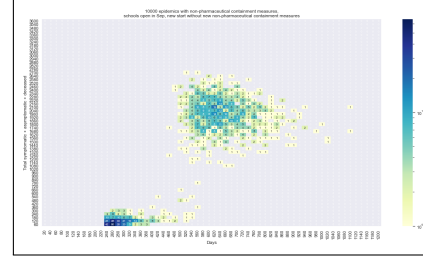
See Fig. 8 about . . .

Methodology:

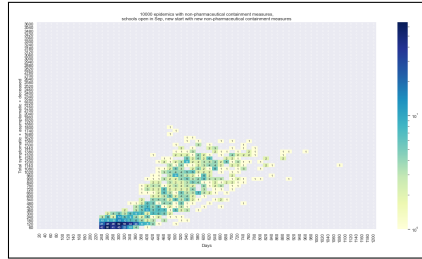
https://github.com/tomorrowdata/COVID-19/blob/main/notebooks/Rt_on_italian_national_data.ipynb



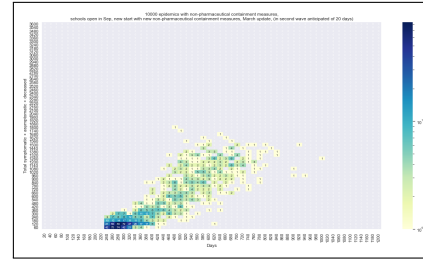
(a) First wave with non-pharmaceutical containment measures, spontaneous second wave, without specific measures



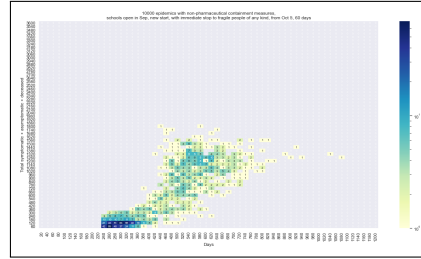
(b) First wave with non-pharmaceutical containment measures, forcing the second wave, without specific measures



(c) First wave with non-pharmaceutical containment measures, forcing the second wave, with new specific non-ph. containment measures



(d) First wave with non-pharmaceutical containment measures, forcing the second wave, with new specific non-ph. containment measures, 20 day anticipation



(e) First wave with non-ph. containment measures, forcing the second wave; in second wave, uniquely stop fragile people, including fragile workers

Fig. 7: Heat-maps of the factual and counterfactual analyses

5.5 Second wave, new infections from outside, with new specific measure anticipation of -20 days

N.B.: (i) anticipation limit Oct 5.; (ii) also the ending date of each measure is anticipated of 20 days.

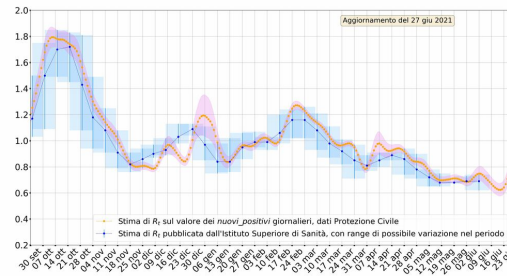


Fig. 8: In blue the R_t values as reported by the Istituto Superiore di Sanità and in red the calculation published regularly at <https://mondoeconomico.eu> by Stefano Terna

5.6 Second wave, new infections from outs., stop fragile people. 60 days from Oct. 5

Schools are always working 100% in this case.

5.7 To recap

6 Planning vaccination campaigns

6.1 Here an Alberto's section on *How vaccines work*

6.2 Planning a vaccination campaign using genetic algorithms (GAs), with non-pharmaceutical containment measures in action

We simulate the vaccination campaigns with the GAs BehaviorSearch program, with a maximum step number of 865, related to the actual duration of case I. In the case of "y" use, the simulation never ends; when applying the GAs quotas ex-post, the simulation stops when no more infected people exist. In any case, if necessary, we have a hard finish button.

6.2.1 Vaccination groups

We take into consideration seven groups in order of decreasing fragility but also considering the exposure to contagion:

gl extra fragile people with three components;

- due to intrinsic characteristics: people in nursing homes;
- due to risk exposure:
 - nursing homes operators;
 - healthcare operators;

g2 teachers;

g3 workers with medical fragility;

g4 regular workers;

g5 fragile people without special characteristics;

g6 regular people, not young, not worker, and not teacher;

g7 young people excluding special activity cases (a limited number in *g1*).

6.3 A specific realistic case

6.4 Vaccination quotas, *plain* strategy

6.5 Vaccination quotas, *wise* strategy

6.6 GAs quotas in the experiment, with vac. people spreading or not spreading the infection

6.7 An experiment with GA

6.7.1 Time dynamics without vaccinations

6.7.2 Time dynamics with *plain* vac. strategy, vac. people still spreading the infection

6.7.3 Time dynamics with *wise* vac. strategy, vac. people still spreading the infection

6.7.4 Time dynamics with best GAs strategy, vac, people still spreading the infection

7 A new model and future developments

8 Appendix 1 - Parameter values

We report here the values of parameters of Fig. 2, also with the short names used in program scripts, in round brackets. Look at Section 2.2 for the definition. Day numbering is related to actual dates via the Table 2. Day 1 is fixed at Feb 4th, 2020.

Day	Date	Day	Date	Day	Date	Day	Date
25	28- 2-2020	200	21- 8-2020	375	12- 2-2021	550	6- 8-2021
50	24- 3-2020	225	15- 9-2020	400	9- 3-2021	575	31- 8-2021
75	18- 4-2020	250	10-10-2020	425	3- 4-2021	600	25- 9-2021
100	13- 5-2020	275	4-11-2020	450	28- 4-2021	625	20-10-2021
125	7- 6-2020	300	29-11-2020	475	23- 5-2021	650	14-11-2021
150	2- 7-2020	325	24-12-2020	500	17- 6-2021	675	9-12-2021
175	27- 7-2020	350	18- 1-2021	525	12- 7-2021	700	3- 1-2022

Table 2: The days of the simulation and their equivalent dates in the calendar

The values adopted in the experiments reported in this work are the following.

- i The values of *probabilityOfGettingInfection* (prob) are: 0.05 (starting phase); 0.02 at day 49 (adoption of non-pharmaceutical measures); 0.035 at day 149 (some relaxation in compliance); 0.02 at day 266 (again, compliance to rules).
- ii *intrinsicSusceptibility* is set discussing Eq. 1 in (2.2, ii).
- iii The values of *%PeopleAnyTypeNotSymptomaticLeavingHome* (%PeopleAny) are: at (day) 20, 90; at 28, 80; at 31, 0; at 106, 80; at 110, 95; at 112, 85; at 117, 95; at 121, 90; at 259, 90; at 266, 80; at 277, 50; at 302, 70; at 320, 90; at 325, 50; at 329, 80; at 332, 50; at 336, 80; at 337, 50; at 339, 80;
- iv The values of *%PeopleNotFragileNotSymptomaticLeavingHome* (%PeopleNot) are: at (day) 31, 80; at 35, 70; at 36, 65; at 38, 15; at 42, 25; at 84, 30; at 106, 0; at 302, 90; at 325, 50; at 332, 50; at 337, 50; at 339, 100; at 349, 90;
- v The values of *%openFactoriesWhenLimitationsOn* (%Fac) are: at (day) 38, value4 0; at 49, 20; at 84, 70; at 106, 100; at 266, 90; at 277, 70; at 302, 80; at 320, 90; at 325, 30; at 329, 90; at 332, 30; 336, 90; at 337, 30; at 339, 100;
- vi *stopFragileWorkers* (sFW): by default, 0; in one of the experiments we used sFW with set to 1 (on) at day 245 and to 0 (off) at day 275;
- vii The values of *activateSchools* (aSch) are: at (day) 1, on; at 17, off; at 225, on; at 325, off; at 339, on;
the values of *%Students* (%St) are: at (day) 0, 100; at 277, 50; at 339, 50; at 350, 50 (repeated values are not relevant for the model, but for the use of the programmer-author);
- viii The value of *radiusOfInfection* (radius) is 0.2; in the model, space is missing of a scale, but forcing the area to be in the scale of a region as Piedmont, 0.2 is equivalent to 20 meters; we have to better calibrate this measure with movements and probabilities; this is a critical step in future developments of the model.

9 Appendix 2 - a gallery of contagion sequences

A gallery of contagion sequences in Table 3.

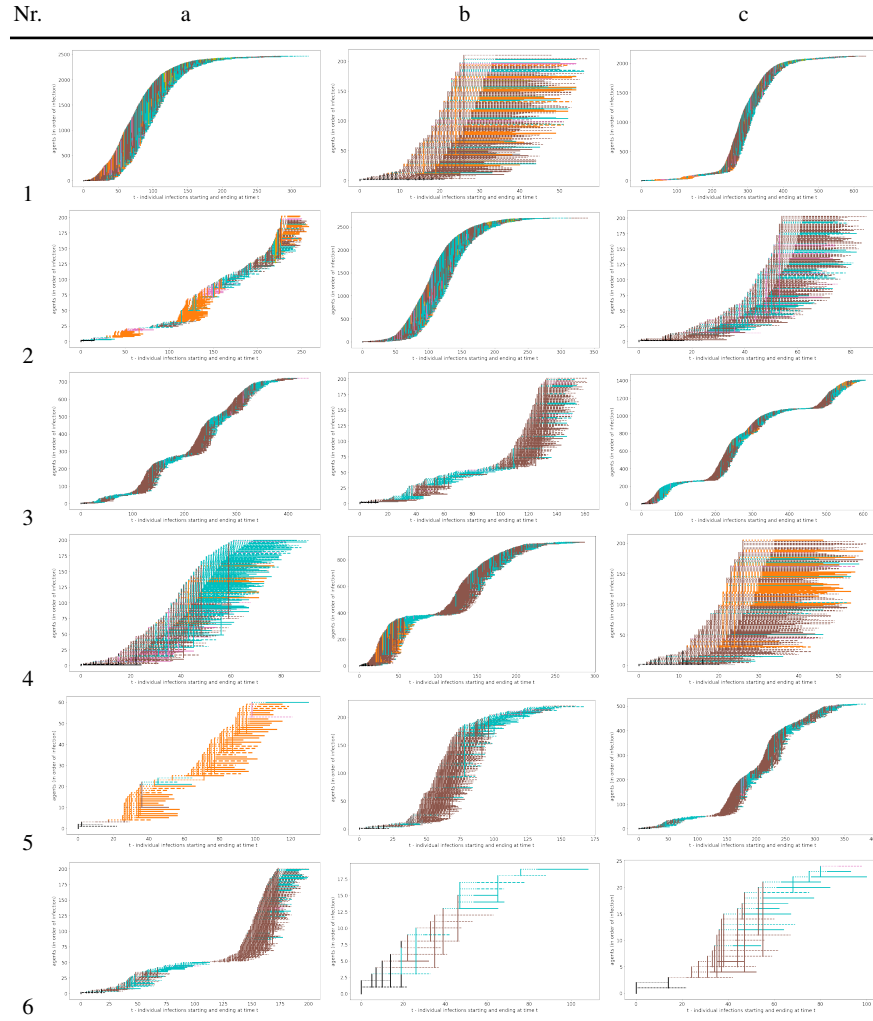


Table 3: Gallery of sequences

- 1a Case 1, without containment measures: contagions in nursing homes (orange), workplaces (brown), homes (cyan), hospitals (pink)
- 1b Case 1, without containment measures, first 200 infections with the main contribution of nursing homes (orange) and workplaces (brown)
- 1c Case 2, without containment measures: nursing homes (orange) as starter
- 2a Case 2, without containment measures, first 200 infections: nursing homes (orange) as starter and around day 70 a unique contagion at home continuing the epidemic

- 2b Case 3, without containment measures: an initial deep effect of contagions in workplaces (brown) and homes (cyan)
- 2c Case 3, without containment measures, first 200 infections: the initial deep effect of contagions in workplaces (brown) and homes is due in the initial steps to fragile persons, also asymptomatic
- 3a Case 4, with containment measures: another case of strong contribution of workplaces (brown) and homes (cyan) to epidemic diffusion
- 3b Case 4, with containment measures, first 200 infections: after day 100 we observe many significant cases of fragile workers diffusing the infection
- 3c Case 5, with containment measures: workplaces (brown), hospitals (pink), nursing homes (orange) and homes (cyan), then workplaces
- 4a Case 5, with containment measures, first 200 infections: in the beginning workplaces (brown), hospitals (pink), nursing homes (orange) and homes (cyan) interweaving
- 4b Case 6, with containment measures: workplaces (brown), nursing homes (orange), homes (cyan)
- 4c Case 6, with containment measures, first 200 infections: workplaces (brown) and nursing homes (orange) strictly interweaving
- 5a Case 7, with containment measures: the effect of nursing homes (orange)
- 5b Case 8, with containment measures: a highly significant effect of workplaces (brown)
- 5c Case 8, with containment measures, stopping fragile workers at day 20, with a positive result, but home contagions (cyan) keep alive the pandemic, which explodes again in workplaces (brown)
- 6a Case 8, with containment measures, stopping fragile workers at day 20, with a positive effect, but home contagions (cyan) keep alive the pandemic, which explodes again in workplaces (brown), first 200 infections with evidence of the event around day 110 with the new phase due to a unique asymptomatic worker
- 6b Case 8, with containment measures, stopping fragile workers and any case of fragility at day 15, also isolating nursing homes
- 6c Case 9, with containment measures: a spontaneously stopping epidemic in short period

References

1. A. Scala, A. Flori, A. Spelta, E. Brugnoli, M. Cinelli, W. Quattrociocchi, F. Pammolli, *Scientific Reports* **10**(1), 13764 (2020). DOI 10.1038/s41598-020-70631-9. URL <https://doi.org/10.1038/s41598-020-70631-9>
2. N. Bellomo, R. Bingham, M.A.J. Chaplain, G. Dosi, G. Forni, D.A. Knopoff, J. Lowengrub, R. Twarock, M.E. Virgillito, *Mathematical Models and Methods in Applied Sciences* **30**(08), 1591 (2020). URL <https://doi.org/10.1142/S0218202520500323>
3. H. Rahmandad, J. Sterman, *Management Science* **54**(5), 998 (2008)
4. F. Squazzoni, J.G. Polhill, B. Edmonds, P. Ahrweiler, P. Antosz, G. Scholz, E. Chappin, M. Borit, H. Verhagen, F. Giardini, N. Gilbert, *Journal of Artificial Societies and Social*

- Simulation **23**(2), 10 (2020). DOI 10.18564/jasss.4298. URL <http://jasss.soc.surrey.ac.uk/23/2/10.html>
5. J.B. Bak-Coleman, M. Alfano, W. Barfuss, C.T. Bergstrom, M.A. Centeno, I.D. Couzin, J.F. Donges, M. Galesic, A.S. Gersick, J. Jacquet, A.B. Kao, R.E. Moran, P. Romanczuk, D.I. Rubenstein, K.J. Tombak, J.J. Van Bavel, E.U. Weber, Proceedings of the National Academy of Sciences **118**(27) (2021). DOI 10.1073/pnas.2025764118. URL <https://www.pnas.org/content/118/27/e2025764118>
 6. J. Epstein, Journal of Artificial Societies and Social Simulation **11**(4), 12 (2008). URL <http://jasss.soc.surrey.ac.uk/11/4/12.html>
 7. R. Axtell, Why agents? On the varied motivations for agent computing in the social sciences. Tech. rep., Center on Social and Economic Dynamics Brookings Institution (2000)
 8. A. Ghorbani, F. Lorig, B. de Bruin, P. Davidsson, F. Dignum, V. Dignum, M. van der Hurk, M. Jensen, C. Kammler, K. Kreulen, L.G. Ludescher, A. Melchior, R. Mellema, C. Păstrăv, L. Vanhée, H. Verhagen, Review of Artificial Societies and Social Simulation (2020). URL <https://rofasss.org/2020/04/25/the-assocc-simulation-model/>
 9. F. Silvagno, A. Vernone, G.P. Pescarmona, Antioxidants **9**(7), 624 (2020). DOI 10.3390/antiox9070624. URL <http://dx.doi.org/10.3390/antiox9070624>
 10. R. Horton, Lancet (London, England) **396**(10255), 874 (2020). URL <https://www.thelancet.com/action/showPdf?pii=S0140-6736%2820%2932000-6>
 11. P. Terna, G. Pescarmona, A. Acquadro, P. Pescarmona, G. Russo, S. Terna. SIsaR, An Agent-Based Model of the Diffusion of Covid-19 Using NetLogo, with Susceptible, Infected, symptomatic, asymptomatic, and Recovered People (2020). URL <https://terna.to.it/simul/SIsaR.html>
 12. U. Wilensky. Netlogo (1999). URL <http://ccl.northwestern.edu/netlogo/>
 13. T.M. Vu, C. Probst, J.M. Epstein, A. Brennan, M. Strong, R.C. Purshouse, Genetic and Evolutionary Computation Conference : [proceedings]. Genetic and Evolutionary Computation Conference **2019**, 1356 (2019). DOI 10.1145/3321707.3321840. URL <https://pubmed.ncbi.nlm.nih.gov/33083795>