

ISSN-L: 2735-6817 | ISSN (online): 2735-6817



# Spatial spread of an epidemic in the context of cellular automata

# Propagación espacial de una epidemia en el contexto de autómatas celulares

Marcelo Cargnelutti Rossato<sup>1</sup> and <sup>10</sup> João Frederico da Costa Azevedo Meyer<sup>1</sup>

Marcelo Cargnelutti Rossato: marcelocrossato@gmail.com

<sup>1</sup> Instituto de Matemática, Estatística e Computação Científica, Universidade Estadual de Campinas, São Paulo, Brasil

Recepción: 2023-04-05 | Aceptación: 2023-08-25 | Publicación: 2023-10-29

Recommended Citation: Cargnelutti Rossato, M et al. (2023). 'Spatial spread of an epidemic in the context of cellular automata'. Rev. model. mat. sist. biol. 3(E), e23E04, doi:10.58560/rmmsb.v03.n02.023.03



This open access article is licensed under a Creative Commons Attribution International (CC BY 4.0) http://creativecommons.org/licenses/by/4.0/. Support:

#### 2 of 8

# ABSTRACT

COVID-19 is a disease that has surpassed the mark of 760 million confirmed cases and has caused more than 6.8 million deaths, revealing the importance of seeking and studying strategies to control its spread. Therefore, the objective of this work is to analyze two of the main mechanisms proposed to control the spread of this disease: confinement and vaccination. To do so, a cellular automaton based on a SCEIRDV compartmental model was constructed, with susceptible, confined, exposed, infected, recovered, dead, and vaccinated individuals. Simulations were performed in scenarios with and without confinement and/or vaccination, mainly analyzing the number of deaths, the average number of infected individuals per day, and the maximum number of individuals simultaneously infected. It was concluded that both strategies contributed to the reduction of these indicators, especially when adopted together.

### **Keywords:**

Epidemiology, Compartmental models, Cellular automata, COVID-19

# RESUMEN

COVID-19 es una enfermedad que superó la marca de 760 millones de casos confirmados, además de causar más de 6.8 millones de muertes, lo que revela la importancia de buscar y estudiar estrategias para controlar su propagación. Por lo tanto, el objetivo de este trabajo es analizar dos de los principales mecanismos propuestos para controlar la propagación de esta enfermedad: el confinamiento y la vacunación. Para hacerlo, se construyó un autómata celular basado en un modelo compartimental SCEIRDV, con susceptibles, confinados, expuestos, infectados, recuperados, muertos y vacunados. Se realizaron simulaciones en escenarios con y sin confinamiento y/o vacunación, analizando principalmente el número de muertes, el promedio de infectados por día y el máximo de individuos simultáneamente infectados. Se concluyó que ambas estrategias contribuyeron a la reducción de estos indicadores, especialmente cuando se adoptaron conjuntamente.

### **Palabras Claves:**

Epidemiología, Modelos compartimentales, Autómatas celulares, COVID-19

2020 AMS Mathematics Subject Classification: Primary: 92B05; Secondary: 92D30, 37B15

## **1** INTRODUCTION

**C** OVID-19 is a serious respiratory infection that has a high transmissibility rate, recording more than 760 million cases and 6.8 million deaths around the world (World Health Organization, 2023). A major initial concern, in addition to the number of infections and deaths, was that there were too many people infected simultaneously, which could lead to hospital overload.

Consequently, some social isolation measures were suggested as a strategy to try to avoid this overload and, at the same time, a great mobilization was initiated for the development of a vaccine for the disease. Thus, this work aims to analyze the influence of confinement and vaccination measures in controlling the spread of COVID-19 through the construction of a mathematical model that represents the spatial spread of this disease and the performance of simulations considering the different possible scenarios.

## 2 SUGGESTED MODEL

One of the first compartmental models in the area of epidemiology was developed by Kermack and McKendrick (1927), in which a system of three differential equations was described to represent the variation of susceptible, infected and recovered individuals. The compartments to be used in a model must be chosen considering the characteristics of the disease to be analyzed and the objective of the model (Hethcote, 2000). Therefore, to describe the spread of COVID-19, we developed a compartmental SCEIRDV model, in which:

- S represents susceptible individuals, who are likely to be infected by the disease or to take measures to protect themselves, such as self-confinement or vaccination;
- C represents confined people, who protect themselves from the disease by remaining isolated and not visiting anyone or receiving visitors;
- E represents those who have had recent exposure to the disease and have not yet developed symptoms, who are less likely than infected people to infect susceptible;
- I represents the infected who have been exposed to the disease for a few days and are symptomatic, being more likely to infect the susceptible;
- R represents recovered individuals, who had the disease and developed a temporary immunity to it;
- D represents those who died from the disease, since deaths from natural causes were not considered, and;
- V represents vaccinated individuals, who cannot be contaminated during the vaccine effect interval, even having contact with exposed or infected people.

The compartment for dead individuals could be removed without affecting any results, as they do not participate in the transmission dynamics, but we have included it as a simple way to count their numbers. While it may be more informative to assume that vaccination does not provide full protection against infection and does not prevent vaccinated individuals from transmitting the disease, we are considering the best possible scenario, in which vaccinated individuals cannot become infected and therefore cannot transmit the disease. Although it is generally considered that exposed individuals cannot transmit the disease, we decided to consider that they can infect at a lower rate than infected individuals, as was done by Chowell and Brauer (2009).

To carry out the simulations, a cellular automaton was developed, which is a model of discrete states, time and space, in which a state is assigned to each region of the domain, which can be called a cell (De Vries *et al.*, 2006). Thus, an automaton of dimension  $100 \times 100$  was constructed by the authors, which can be understood as a matrix  $100 \times 100$  in which each element represents the state (susceptible, confined, exposed, infected, recovered, dead or vaccinated) of the individual living in the corresponding cell.

Initially, only 1 was considered infected near the center of the domain, while all other 9999 individuals were susceptible. In Figure 1, the possible state changes are presented, associated with the parameters that influence these changes.



Figure 1: Diagram of the compartmental model SCEIRDV.

Instead of considering that the spatial spread of the disease would occur only through contacts between direct neighbors and following the classic diffusion equation, it was defined that, at each instant of time (which corresponds to one day), each person would randomly interact with others three people within 5 units horizontally and vertically of their position. When there is contact between a susceptible individual and an exposed or infected individual, the susceptible individual may become exposed to the disease with probabilities  $p_e$  or  $p_i$ , respectively. It should also be noted that the confined and dead do not participate in these interactions, being excluded from the interaction options of other individuals.

It is assumed that individuals exposed to the disease develop symptoms and go to the infected compartment after an incubation time of  $t_e$  days and, after  $t_i$  days, an infected person can die with probability  $p_m$  or recover with probability  $p_r = 1 - p_m$ . A recovered person becomes susceptible again after  $t_r$  days. Finally, at each instant of time, every susceptible individual can confine himself with probability  $p_c$ or be vaccinated with probability  $p_v$ . The confined remain totally isolated for a period of  $t_c$  days, then returning to being susceptible, and the vaccinated remain protected during an interval of  $t_v$  days and then also return to the susceptible compartment.

In this model, we use stochastic components for the probabilities that an individual will be infected, confined, vaccinated, and to determine whether the individual recovers or dies after an infection. However, the times in which he remains vaccinated, confined, exposed, infected or recovered are presented in a deterministic way. An analogous completely stochastic model could be constructed using the relation  $p = \frac{1}{t}$ , in which, for example, a confined individual would have a probability of  $\frac{1}{t_c}$  to leave the confinement each day rather than becoming deterministically susceptible after  $t_c$  days.

To estimate the parameters accurately, we reviewed several articles. Li *et al.* (2020) reported an average disease incubation period of 5.2 days in the first 425 COVID-19 patients in Wuhan, while Elias *et al.* (2021) analyzed 99 studies published between January 1, 2020 and January 10, 2021, and found an average incubation time of 6.38 days. Additionally, Wu *et al.* (2022) obtained a mean incubation time of 6.57 days in 142 studies with a total of 8112 patients, which could be even lower considering specific strains. Thus, we set  $t_e = 6$  days.

Studies have provided estimates of the infectious period for the disease ranging from 5 to 14 days. The analysis by Acuña-Zegarra *et al.* (2020) estimated infectious periods of 5.97 days for asymptomatic patients and 10.81 days for symptomatic patients. Moreover, viral loads were found to peak around 10 days after symptom onset, according to Zou *et al.* (2020). Byrne *et al.* (2020) reported that the average time from symptom onset to two negative RT-PCR tests was 13.4 days. In this work, we use  $t_i = 10$  days.

The duration of protection provided by the COVID-19 vaccine is difficult to pinpoint precisely, but studies suggest a decline in its effectiveness between 3 to 6 months after vaccination. Research has shown that the high initial antibody titers induced by mRNA vaccines diminish by this time frame (Barouch, 2022), with mean antibody levels 4 months after vaccination falling to just 6.3% of peak levels (Khoury *et al.*, 2021). Furthermore, a reduced efficacy against hospitalization within 3 to 4 months post-vaccination has been reported by Collie *et al.* (2022). Based on these observations, we have set  $t_v = 120$  days.

While Reynolds *et al.* (2020) reported detecting neutralizing antibodies against SARS-CoV-2 at 16-18 weeks after infection, other studies suggest the potential for reinfection with an average time of 50.5 days (Dos Santos *et al.*, 2021). Additionally, Seow *et al.* (2020) observed that individuals could maintain high levels of neutralizing antibodies for up to 60 days after infection. In order to establish a parameter between these observed values, we define  $t_r = 90$  days.

It is assumed that an individual will remain confined for an average of 30 days and may face isolation again in the future. Hence, we set  $t_c = 30$  days. The mortality rate is calculated by dividing the number of deaths from the disease by the total number of cases, using data reported by World Health

Organization (2023). This yields  $p_m \approx \frac{6,800,000}{760,000,000} \approx 0.009$ .

For any interaction between an infected person and a susceptible person, the probability of exposure for the susceptible person is assumed to be 3%. Therefore, we define  $p_i = 0.03$ . In the case of an interaction with an exposed individual, it is assumed that the probability of exposure for the susceptible person is half of that, or  $p_e = 0.015$ .

#### **3** SIMULATIONS

In order to analyze the influence of confinement and vaccination on the spread of the disease, the results obtained in simulations with and without confinement and/or vaccination will be presented, mainly evaluating the number of deaths, average infected per day and maximum number of simultaneously infected individuals.

Table 1 presents the parameters used for each simulation, with periodic boundary conditions and a total simulation time of 1,095 days (approximately 3 years). The values of  $p_c$  and  $p_v$  differ in each simulation, and will be discussed in detail for each of the considered scenarios.

Table 1: Parameters

Parameter	Value	Meaning		
$t_e$	6 days	Incubation time		
$t_i$	10 days	Infectious period		
$t_c$	30 days	Consecutive confinement time		
$t_r$	90 days	Period of immunity from disease		
$t_{v}$	120 days	Period of vaccine immunity		
$p_m$	0.009	Mortality rate		
$p_i$	0.03	Infection rate by infected individuals		
$p_e$	0.015	Infection rate by exposed individuals		
$p_c$	0.01 - 0.03	Confinement rate		
$p_v$	0.001 - 0.005	Vaccination rate		

First, in the simulation without confinement and without vaccination, that is, with  $p_c = p_v = 0$ , a total of 403 deaths were obtained, which is about 4% of the initial population of 10,000 individuals. The evolution of the number of susceptible, exposed, infected, recovered and dead is shown in Figure 2.

It is possible to observe that, without any containment measures, the virus continues to spread with great intensity even when there is a high number of deaths, with an average of 405.7 people infected per day. The maximum number of people simultaneously infected is 1,255, occurring on day 927. Figures 3a and 3b show the day with the highest number of infected people and the scenario after 1,095 days, respectively.

Similar to the colors in Figure 2, the black squares represent the dead, the green ones are recovered, the red ones are infected and the orange ones are exposed to the disease. The only difference is that the susceptibles, previously displayed in yellow, are now represented by white squares. It is noted that at the end of the simulation there are still several foci of infection, similarly to the day when there is the highest number of infected people.



Figure 2: Result of the first simulation, without confinement and vaccination.



Figure 3: Spatial distributions of the simulation without confinement and vaccination.



Figure 4: Evolution of the confinement rate over time.



Figure 5: Result of the second simulation, with confinement and without vaccination.

To carry out the second simulation, it was considered that the confinement rate should start high and be reduced over time, since there are several factors that lead people not to isolate themselves at home for a long time, such as the need to going out to work or the desire to meet friends and family, for example.

Thus, an initial confinement probability of  $p_c = 0.03$  was defined, which was linearly reduced until reaching  $p_c = 0.01$  at the end of 2 years (730 days), remaining constant at this value until the end of the analyzed period, as can be observed in Figure 4.

Again without vaccination  $(p_v = 0)$ , the simulation resulted in 403 deaths, the same as the previous scenario. A summary of this simulation can be seen in Figure 5.

By reducing the number of susceptibles through confinement, it is observed that the oscillations of all compartments are smaller and the peak of infected individuals in one day is also reduced by one third to 837, which can help to avoid overloading hospitals. Even so, the average number of people infected per day is 398.1 people, which indicates that, although confinement manages to reduce the number of people infected simultaneously, there is no significant reduction in the total number of infections, they only occur in a slightly more homogeneous way over the days. The spatial distribution of the population on day 686, when the number of infected people peaked, and at the end of 1,095 days are shown in Figures 6a and 6b, respectively.



Figure 6: Spatial distributions of the simulation with confinement and without vaccination.

The colors are the same as shown in Figure 3, with the

inclusion of cyan for confined individuals. It is observed that the high number of confined means that the infection cannot reach as many people at the same time as in the previous case, but there is still a considerable oscillation in the number of infected even after approximately 3 years.

The third scenario serves to analyze the impact of vaccination alone, therefore confinement is not carried out ( $p_c = 0$ ). Estimating that people could start vaccinating approximately 1 year after the onset of the disease and that the vaccination rate would increase as more vaccines were purchased and more people could be vaccinated, it was defined that  $p_v = 0$ initially, spiking to  $p_v = 0.001$  at t = 365 days and growing linearly up to  $p_v = 0.005$  at t = 730 days, remaining constant until the end of the analyzed period. The graph of the vaccination rate over time is shown in Figure 7.



Figure 7: Evolution of the vaccination rate over time.

The evolution of the disease in this case is shown in Figure 8, which shows that there were 306 deaths, a reduction of about 24% compared to previous cases.



Figure 8: Result of the third simulation, with vaccination and without confinement.

In this simulation, a large difference is observed in the number of infected people before and after the start of vaccination. While there is a peak of 1,022 infected on day 180,

the highest number of individuals simultaneously infected after the start of vaccination (disregarding the first days, as the vaccine began to be applied at a time when the number of infected people was close to maximum value) was 523 people, on the day 582.

In this way, it is possible to observe that the vaccination was able to reduce both the maximum number of infected in one day and the average of infected over the days, which dropped from about 400 to 301.7. The spatial spread on the days when there is the highest number of infected people after the start of vaccination and after 1,095 days can be seen in Figures 9a and 9b, respectively.



Figure 9: Spatial distributions of the simulation with vaccination and without confinement.

In this case, blue squares are included to represent those vaccinated. Analyzing the two scenarios, it is observed that in Figure 9a, while the vaccination rate was still rising, there are some large groups of susceptibles nearby, which facilitates the spread of the disease. Meanwhile, it is noted that at the end of the 1,095 days, susceptibles are often surrounded by vaccinated and recovered, which makes it difficult for the disease to spread.

Finally, the last simulation analyzes the combined effects of confinement and vaccination. Thus, it starts with  $p_c = 0.03$  which decreases linearly to  $p_c = 0.01$  in t = 730 days, while  $p_v = 0$  during the first year, jumping to  $p_v = 0.001$  after 365 days and growing linearly up to  $p_v = 0.005$  at t = 730 days, as used in the two previous simulations. Combining the two mechanisms to control the spread of the disease, there were only 261 deaths, about 35% less than in the first simulation, in which there was no form of control. The evolution of the disease in this simulation is illustrated in Figure 10.

Up to day 365, the results obtained are identical to those shown in Figure 5 and the maximum number of infected individuals is 719 on day 344. After the start of vaccination, there is the lowest peak among all the simulations, with only 515 infected simultaneously on day 528. Figures 11a and 11b illustrate this peak of infected people and the population distribution at the end of the simulation, respectively.

Contrary to the large waves of infection that were observed mainly in Figures 3a and 6a, those infected seem to be more spread out and fewer in this simulation, showing that vacci-



Figure 10: Result of the fourth simulation, with confinement and vaccination.



Figure 11: Spatial distributions of the simulation with confinement and vaccination.

nation and confinement managed to curb a little the spread of the disease. Still in this simulation, the lowest average of infected per day was reached, 282.9. A summary of the results obtained in the simulations, considering the peak of infected people after the start of vaccination in the cases in which it was carried out, is presented in Table 2.

Table 2: Summary of simulation results

Simulation	1	2	3	4
Number of deaths	403	403	306	261
Peak of infected	1255	837	523	515
Average number of infected	405.7	398.1	301.7	282.9

Analyzing the table, it is possible to observe that in the second simulation, where there was only confinement, there was no reduction in the number of deaths and the average of infected was less than 2% lower, but the peak of infected was reduced by about 33% when compared to the case without any containment measures.

Meanwhile, considering only vaccination, there was a 24% reduction in the number of deaths, a 58% reduction in the maximum number of simultaneous infections and an av-

erage number of infected people almost 26% lower. Finally, in the simulation with both containment measures, 35% fewer deaths, 59% lower peak of infected people and 30% lower average number of infected people were observed.

#### **4 FINAL CONSIDERATIONS**

In this work, the authors developed a cellular automaton based on a SCEIRDV compartmental model to simulate the evolution of COVID-19 using parameters based on real studies on the disease. Analyzing the results summarized in Table 2, it is possible to observe that the implementation of confinement and vaccination measures separately has already caused a reduction in some infection and mortality rates, but the joint application of these two measures has reduced them the number of deaths at about 35%, the peak of infected at almost 59% and the average of infected at more than 30%, being the most favorable strategy among those analyzed.

In future work, there are several implementations that can be considered, such as changing the infection and/or mortality rates over time to consider the different variants of the disease, for example. In addition, a completely stochastic model can be built for comparison and the scenario in which vaccinated people can also be infected can be considered. Finally, another idea is to introduce houses or clusters into the model, to directly consider the possibility of exposure to the disease through other individuals living in the same environment.

#### REFERENCES

- Acuña-Zegarra, M.A., Santana-Cibrian, M. and Velasco-Hernandez, J.X. (2020) 'Modeling behavioral change and covid-19 containment in mexico: A trade-off between lockdown and compliance'. *Mathematical biosciences*, 325, p. 108370.
- Barouch, D.H. (2022) 'Covid-19 vaccines—immunity, variants, boosters'. New England Journal of Medicine, 387(11), pp. 1011–1020.
- Byrne, A.W., McEvoy, D., Collins, A.B., Hunt, K., Casey, M., Barber, A., Butler, F., Griffin, J., Lane, E.A., McAloon, C. *et al.* (2020) 'Inferred duration of infectious period of sars-cov-2: rapid scoping review and analysis of available evidence for asymptomatic and symptomatic covid-19 cases'. *BMJ open*, 10(8), p. e039856.
- Chowell, G. and Brauer, F. (2009) 'The basic reproduction number of infectious diseases: computation and estimation using compartmental epidemic models'. *Mathematical and statistical estimation approaches in epidemiology*, pp. 1–30.
- Collie, S., Nayager, J., Bamford, L., Bekker, L.G., Zylstra, M. and Gray, G. (2022) 'Effectiveness and durability of the bnt162b2 vaccine against omicron sublineages in south africa'. New Engl and Journal of Medicine, 387(14), pp. 1332–1333.
- De Vries, G., Hillen, T., Lewis, M., Müller, J. and Schönfisch, B. (2006) A course in mathematical biology: quantitative modeling with mathematical and computational methods. SIAM.
- Dos Santos, L.A., de Góis Filho, P.G., Silva, A.M.F., Santos, J.V.G., Santos, D.S., Aquino, M.M., de Jesus, R.M., Almeida, M.L.D., da Silva, J.S., Altmann, D.M. *et al.* (2021) 'Recurrent covid-19 including evidence of reinfection and enhanced severity in thirty brazilian health-care workers'. *Journal of Infection*, 82(3), pp. 399–406.
- Elias, C., Sekri, A., Leblanc, P., Cucherat, M. and Vanhems, P. (2021) 'The incubation period of covid-19: A meta-analysis'. *International Journal of Infectious Diseases*, 104, pp. 708–710.
- Hethcote, H.W. (2000) 'The mathematics of infectious diseases'. SIAM review, 42(4), pp. 599–653.

Kermack, W.O. and McKendrick, A.G. (1927) 'A contribution to the math-

ematical theory of epidemics'. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772), pp. 700–721.

- Khoury, J., Najjar-Debbiny, R., Hanna, A., Jabbour, A., Ahmad, Y.A., Saffuri, A., Abu-Sinni, M., Shkeiri, R., Elemy, A. and Hakim, F. (2021) 'Covid-19 vaccine–long term immune decline and breakthrough infections'. *Vaccine*, 39(48), pp. 6984–6989.
- Li, Q., Guan, X., Wu, P., Wang, X., Zhou, L., Tong, Y., Ren, R., Leung, K.S., Lau, E.H., Wong, J.Y. *et al.* (2020) 'Early transmission dynamics in wuhan, china, of novel coronavirus–infected pneumonia'. *New England journal of medicine.*
- Reynolds, C.J., Swadling, L., Gibbons, J.M., Pade, C., Jensen, M.P., Diniz, M.O., Schmidt, N.M., Butler, D.K., Amin, O.E., Bailey, S.N. *et al.* (2020) 'Discordant neutralizing antibody and t cell responses in asymptomatic and mild sars-cov-2 infection'. *Science immunology*, 5(54), p. eabf3698.
- Seow, J., Graham, C., Merrick, B., Acors, S., Pickering, S., Steel, K.J., Hemmings, O., O'Byrne, A., Kouphou, N., Galao, R.P. et al. (2020) 'Longitudinal observation and decline of neutralizing antibody responses in the three months following sars-cov-2 infection in humans'. *Nature* microbiology, 5(12), pp. 1598–1607.
- World Health Organization (2023) Covid-19 weekly epidemiological update, edition 135, 22 march 2023. https://www.who.int/emergencies/ diseases/novel-coronavirus-2019/situation-reports.
- Wu, Y., Kang, L., Guo, Z., Liu, J., Liu, M. and Liang, W. (2022) 'Incubation period of covid-19 caused by unique sars-cov-2 strains: a systematic review and meta-analysis'. *JAMA network open*, 5(8), pp. e2228008– e2228008.
- Zou, L., Ruan, F., Huang, M., Liang, L., Huang, H., Hong, Z., Yu, J., Kang, M., Song, Y., Xia, J. *et al.* (2020) 'Sars-cov-2 viral load in upper respiratory specimens of infected patients'. *New England journal of medicine*, 382(12), pp. 1177–1179.

**Recommended Citation:** Cargnelutti Rossato, M *et al.* (2023). 'Spatial spread of an epidemic in the context of cellular automata'. Rev. model. mat. sist. biol. 3(E), e23E04, doi:10.58560/rmmsb.v03.n02.023.03



This open access article is licensed under a Creative Commons Attribution International (CC BY 4.0) http://creativecommons.org/licenses/by/4.0/. Support: