

# MATHEMATICAL MODELS OF CONFINEMENT AND DECONFINEMENT

Satyanad Kichenassamy

## ▶ To cite this version:

Satyanad Kichenassamy. MATHEMATICAL MODELS OF CONFINEMENT AND DECONFINEMENT. 2020. hal-02551210v1

## HAL Id: hal-02551210 https://hal.science/hal-02551210v1

Preprint submitted on 22 Apr 2020 (v1), last revised 6 May 2020 (v2)

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers. L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

### MATHEMATICAL MODELS OF CONFINEMENT AND DECONFINEMENT

#### SATYANAD KICHENASSAMY

ABSTRACT. We introduce modifications of space-extended SIR models by taking into account confinement of part of the population as well as their partial mobility. We introduce distinct compartments for major regions of the country, and for confined classes within each region. It is shown that a deconfinement procedure that depends on the infection levels of different regions can stall the growth of the epidemic in the less infected regions while avoiding complete lockdown. Deconfinement may therefore be achieved without saturation of the health system.

**Keywords:** Covid-19, SARS-Cov-2, SIR model, confinement and deconfinement, lockdown.

#### 1. INTRODUCTION

Confinement measures have stalled the Covid-19 epidemic, but did not quell it. It is therefore necessary to devise a procedure for deconfinement that limits the number of new cases it will necessarily induce. We propose a strategy that, in a somewhat counter-intuitive fashion, both allows a measure of population mobility and prevents all regions from reaching a peak of infection at the same time. The main idea is to limit the allowed flux of population in terms of the gradient of infection.

This proposal takes stock of the current consensus in epidemiology, and seeks to introduce spatial variation without losing the advantages of the compartmental approach. Indeed, the SIR model and its variants, by summarizing spatial interaction in the mass-action term, can only be modified into a space-dependent setup in two ways: by modifying the mechanism of interaction, and by introducing different compartments for different regions. Since we are dealing with short periods, we neglect births. Age-related transmission could be included in the obvious manner. It follows from a recent survey of spatially-extended TSIR models (Bjørnstad, Grenfell, Viboud and King, 2019) that all earlier approaches amount to replacing terms of the form  $\beta SI/N$  in the usual equations by  $\beta S(I + \iota)/N$ ,<sup>1</sup> where  $\iota$  depends on the model.

<sup>&</sup>lt;sup>1</sup>Or more generally,  $\beta S(I + \iota)^{\alpha}/N$ . The extension of our considerations to this case is immediate.

#### SATYANAD KICHENASSAMY

We propose here a particular form of  $\iota$ , and show its relevance on a example.

The implicit assumption that the infectious periods are exponentially distributed could be removed by introducing further compartments, as was shown by Keeling and Grenfell (2001) and Lloyd (2002), but we do not consider this straightforward extension here.

We construct our models by successive generalizations, starting from a standard SIR model, spelling out the meaning of the assumptions introduced at each step. We first deal with the gradual reinstatement of communication between regions, and then deal in the third section with the issue of intraregional deconfinement<sup>2</sup>. The conclusions of the paper are then summarized.

### 2. Communication between regions

2.1. A model for each region. Consider first a spatially extended SIR model with independent regions  $A_i$ , in which there are  $S_i$  susceptibles,  $I_i$  infectious and  $R_i$  recovered. With obvious notation, if there is no interaction between regions, the unknowns satisfy (primes denote time derivatives)

(1) 
$$S'_i = -\beta_i S_i I_i / N_i$$

(2) 
$$I'_i = \beta_i S_i I_i / N_i - (\gamma + d) I_i$$

(3) 
$$R'_i = \gamma I_i.$$

The coefficients  $\beta_i$  could depend on time. Indeed, if confinement is successful, we expect  $R_{0i} := \beta_i / \gamma$  to decrease. Here, we have assumed that the recovery rate and the death rate are the same for all regions – which is reasonable if the health system is the same throughout the country. The introduction of further compartments within regions will be performed in the next section.

2.2. Introduction of communication between regions. Let us now assume that the susceptibles can be infected by contact with infectious individuals from other regions:

(4) 
$$S'_{i} = -\beta_{i}S_{i}\left(I_{i} + \sum_{j \neq i} \iota_{ij}\right)/N_{i}$$

(5) 
$$I'_{i} = \beta_{i}S_{i}\left(I_{i} + \sum_{j \neq i} \iota_{ij}\right)/N_{i} - (\gamma + d)I_{i}$$

(6)  $R'_i = \gamma I_i,$ 

<sup>&</sup>lt;sup>2</sup>With sufficient computing power, it would be possible to subsume the second issue under the first, treating each home as a different "region," but this would not necessarily be appropriate, since communication between urban centers differs qualitatively from communication within a city.

with

(7) 
$$\iota_{ij} = \sum_{j \neq i} M_{ij} I_j,$$

(8) 
$$M_{ij} = \frac{m_{ij}}{(1 + [I_i/N_i - I_j/N_j]^2/K^2)},$$

where the parameters  $m_{ij}$  and K define the deconfinement strategy, and  $N_i$  is the total population of region  $A_i$ . Note that  $0 \le M_{ij} \le m_{ij}$ , and that the second inequality becomes an equality when the infection rates in the two regions are equal.

Special cases include the following.

- If K is very large, the  $M_{ij}$  are very close to the  $m_{ij}$ . If in addition all the  $m_{ij} = 1$ , the  $S_i$  susceptibles interact with all the infected. This represents the stage of full deconfinement.
- If on the contrary all the  $m_{ij} = 0$ , the value of K becomes irrelevant, and in this situation, all regions boundaries are closed.
- To encourage communication between  $A_i$  and  $A_j$  is to make  $M_{ij}$  closer to 1, by increasing  $m_{ij}$  or K.
- A larger value of K means that the mobility is *less* dependent on the infection levels of the regions. Conversely, a very small K means that the model is *very sensitive* to differences in infection levels.

2.3. An example. As a simple illustration that the suggested mechanism actually has the expected consequences, let us consider the case of three regions  $A_1$ ,  $A_2$  and  $A_3$ , in which the rates of infection are initially 20%, 2% and 10% respectively. We take  $\beta = 3$ ,  $\gamma = 1$  (hence  $R_0 = 3$ ), d = 0.1. We assume that region  $A_1$  interact only with  $A_1$  ( $m_{23} = 0$ ).

All figures describe the time evolution of the proportions of infected patients  $I_i/N_i$  for various choices of parameters.

Figure 1 describes the situation where the regions are disconnected. There are three distinct infection peaks, reflecting the differences in initial infection levels.

Figure 2 describes the opposite situation, when all regions are allowed to interact freely. All three populations now exhibit much closer peaks. Even though  $A_2$  and  $A_3$  interact only through  $A_1$ , the evolution is almost identical in all three regions except for small times.

Figure 3 shows the result of favoring exchanges between  $A_1$  and  $A_3$ , that are much more infected initially than  $A_2$  ( $m_{12} = 0.3$ ,  $m_{13} = 1$ ). In this case, by taking K relatively large (K = 3), one also takes to some extent account their differences in infection as the epidemic progresses. The peaks are not as clustered, but are still close.

Figure 4 shows the result of favoring exchanges between  $A_1$  and  $A_3$  as before,  $(m_{12} = 0.3, m_{13} = 1)$ , but giving now greater importance to the differences in infection levels (K = 0.3). The peaks are more separated.



FIGURE 1. Infection levels in regions 1 (blue), 2 (red) and 3 (yellow), with confined regions  $(m_{12} = m_{13} = 0)$ .



FIGURE 2. Infection levels in regions 1 (blue), 2 (red) and 3 (yellow), with strong interaction between regions and no flux limitation  $(m_{12} = m_{13} = 1, K = 1000)$ .

Figure 4 shows the result of favoring exchanges between  $A_1$  and  $A_3$ , with the same values of the  $m_{ij}$  ( $m_{12} = 0.3$ ,  $m_{13} = 1$ ), but giving greater importance to the differences in infection levels (K = 0.3). The infection peaks are more separated.



FIGURE 3. Infection levels in regions 1 (blue), 2 (red) and 3 (yellow), favoring exchanges between regions with similar levels of infection initially  $(m_{12} = 0.3, m_{13} = 1)$ ,



FIGURE 4. Infection levels in regions 1 (blue), 2 (red) and 3 (yellow), favoring exchanges between regions with similar levels of infection initially  $(m_{12} = 0.3, m_{13} = 1)$ , with significant flux limitation (K = 0.3).



FIGURE 5. Infection levels in regions 1 (blue), 2 (red) and 3 (yellow), favoring exchanges between regions with similar levels of infection initially  $(m_{12} = 0.3, m_{13} = 1)$ , with significant flux limitation (K = 0.0003).

Figure 5 shows that, with the same values of  $m_{ij}$  but with a much larger effect of infection differences (K = 0.0003), the result is similar to the fully confined situation.

Figure 6 assumes  $\beta = 1.6$ ,  $\gamma = 1$ ,  $m_{12} = m_{13} = 1$  and K = 1000 and K = 0.03 respectively. The separation of peaks is again apparent. In addition, a plateau formation is seen.

We have discussed deconfinement strategies at the region level. Now, the SIR model does not distinguish, within a given region, infected individuals in or out of quarantine, nor susceptibles that are confined at home, as opposed to those that may move more freely (such as health workers for example). These may be handled in a fairly simple manner, by introducing new compartments within regions, and will be discussed elsewhere.

#### 3. CONCLUSION

We have proposed a modified space-extended SIR model containing restrictions on travel between regions, that favors communication between regions with a similar level of infection. We have proposed a model for confinement and deconfinement on this basis, in which public policies act by modifying the time dependence of the parameters in the model. We have shown on an example that this model allows for communication between regions while avoiding that all regions reach their peak at the same time.



FIGURE 6. Infection levels in regions 1 (blue), 2 (red) and 3 (yellow), with  $\beta = 1.6$ ,  $\gamma = 1$ ,  $m_{12} = m_{13} = 1$ , K = 1000 (top) and K = 0.03 (bottom).

It appears that these models are a natural extension of recently proposed models, and are consistent with the current concepts in use in epidemiology, that should only be modified with care, since they are the outcome of the comparison of theory and observations for over a century (Serfling (1952), Hethcote (2000)). The class of models in this paper enables one to plan a progressive deconfinement, that can be controlled in real time according to the evolution of the disease, while avoiding excessive restrictions on citizens' freedom.

#### SATYANAD KICHENASSAMY

#### References

- Ottar N. Bjørnstad, Bryan T. Grenfell, Cecile Viboud, and Aaron A. King, "Comparison of alternative models of human movement and the spread of disease," http://dx.doi.org/10.1101/2019.12.19.882175
- [2] M. Choisy, J.-F. Guégan,2 and P. Rohani, "Mathematical Modeling of Infectious Diseases Dynamics," Chapter 22 of the Encyclopedia of Infectious Diseases: Modern Methodologies, (M.Tibayrenc, ed.), Wiley, 2007.
- [3] Herbert W. Hethcote, "The Mathematics of Infectious Diseases," SIAM Review, 42: 4 (2000), 599-653.
- [4] Matt J. Keeling and Bryan T. Grenfell, "Understanding the persistence of measles: reconciling theory, simulation and observation," Proc. of the Royal Society of London, B (2002) 269, 335343.
- [5] Alun L. Lloyd, "Destabilization of epidemic models with the inclusion of realistic distributions of infectious periods," Proc. of the Royal Society of London, B (2001) 268, 985-993.
- [6] Robert E. Serfling, "Historical review of epidemic theory," Human Biology, Vol. 24, No. 3 (September, 1952), pp. 145-166

UNIVERSITÉ DE REIMS CHAMPAGNE-ARDENNE, LABORATOIRE DE MATHÉMATIQUES (CNRS, UMR 9008), MOULIN DE LA HOUSSE, B. P. 1039, F-51687 REIMS, FRANCE

E-mail address: satyanad.kichenassamy@univ-reims.fr