ARTICLE TYPE

A new stochastic approach to the spread of envinromental events enhanced by the wind.

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Abstract

The purpose of this paper is to present a stochastic approach of the expansion of adverse environmental events which are enhanced by the wind. Our model follows a scheme based on the well-known Susceptible-Infected-Recovered (SIR) model. The expansion takes place in a grid where the cells represent an individual (or even a group) in the population under study. The state of the cells is modelled by a random variable which is a measure of the intensity and direction of the wind. As time passes, the status of the cells is determined according to both an update criterion and a probabilistic neighbourhood relationship. In addition, in building the expansion model, we have defined a set of adjustable parameters, which will have an effect on the rate of propagation of the phenomenon.

KEYWORDS:

grid, spread of the phenomenon, neighbourhood relationship, update criterion

1 | INTRODUCTION

The problem of modelling how environmental phenomena spread through the population has been one of the most studied for decades.

However, this field suffers from many problems arising from the lack of quality information which we need to check whether our hypotheses are true or not. Even so, the development of mathematical models has made it possible to address important issues such as vaccination and immunization policies ^{1,2,3}, population dynamics ^{4,5}, the evolution of infectious or degenerative diseases ^{6,7} or even in the malware propagation through computer devices ⁸.

On the other hand, it is well known that climate is one of the most important variables in the propagation of various environmental phenomena such as forest fires, or of certain pests transported by vectors. In this paper, we focus on wind as a main climate variable involved in the propagation of ageneric environmental phenomenon. We will assume as a starting hypothesis that the spread of the phenomenon moves in the same direction as the wind and takes advantage of its intensity to cover further distances. Moreover, our proposal will follow a variant of the well-known SIR model, a discrete SID scheme, where D means that our cell is dead.

In previous work, the spread was studied using non-adaptive neighbourhood relations, which produced symmetric outcomes ^{9,10}. Our approach considers that the neighbourhood relations should be flexible and probabilistic since the probability of infection for each cell will be determined by both the direction and the intensity of the wind. This assumption leads to the

fact that our results are probably not symmetric and will be wind dependent.

Finally, the wind direction θ is considered as a random variable $\theta \sim Unif(0, 2\pi)$. However, we know that the intensity of the wind depends on the location of the country among others, so its distribution is neither known (a priori) nor set. In order to avoid this situation we work with percentiles. Hence, we consider $\rho \in [0, 1]$, where ρ refers to the percentile on this unknown distribituion, and in this sense we can also consider it as a random variable $\rho \sim Unif(0, 1)$.

2 | NOTATION

N odd positive integer.

n integer such that N = 2n + 1

K positive integer.

 $\mathbb{M}_{N\times N}(S)$ set of square matriz with size N and entries in $S\neq\emptyset$

3 | OUR MODEL

Definition 3.1. Let $M = (m_{ij})_{i,i=0}^{N-1} \in \mathbb{M}_{N \times N}(\{0,1\})$, such that only $m_{n,n} = 1$. Then, we say that M is a **primitive matrix**.

Example 1. If N = 3, then

$$M := \begin{pmatrix} 0 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

is a primitive matrix

Definition 3.2. The different states of our grid will be stored in a family of matrices $\mathcal{E} := \{M^k\}_{k=0}^K$, where M^0 is a primitive matrix and $M^k \in \mathbb{M}_{N \times N}(\{0,1,2\})$. Apart from that, every single entry m_{ij}^k of M^k refers to the state of this entry in the generation k, where:

$$m_{ij}^{k} := \begin{cases} 0 & \text{if } is \text{ susceptible} \\ 1 & \text{if } is \text{ infected} \\ 2 & \text{if } is \text{ dead} \end{cases}$$

Let $Q: [0, 2\pi[\mapsto \{1, 2, 3, 4\}]$ be the function which returns us the quadrant of θ , and $T: [0, 2\pi[\mapsto [0, \frac{\pi}{2}]]]$ be the function which transports the angle θ to its similar in the first quadrant.

Now, we are going to introduce the function f, which will be very important for us in order to build the spread model, because we will use it to define the infection probabilities of diagonal elements, as shown in Equation (4)

$$f: [0, \frac{\pi}{2}] \mapsto [0, 1]$$

$$\theta \mapsto \begin{cases} \tan \theta & \text{si } \theta \in [0, \frac{\pi}{4}] \\ \cot \theta & \text{si } \theta \in]\frac{\pi}{4}, \frac{\pi}{2}] \end{cases}$$

$$(1)$$

Note that, by trigonometric properties f is symmetric with respect to the axis $x = \frac{\pi}{4}$ and the maximum is reached at the intersection with it, where $f(\frac{\pi}{4}) = 1$.

Definition 3.3. Let the generation $0 \le k \le K$ where $k \in \mathbb{N}$. Then, we consider the set I_k as the set of index pairs of the infected elements of M^k . So,

$$I_k := \{(i,j): m_{ij}^k = 1\}$$
 (2)

Remark 3.1. Note that M^0 will always be a primitive matrix, then $I_0 = \{(n, n)\} \neq \emptyset$.

As mentioned above, our goal is to get a function:

the probability of this cell of being infected in the following step.

Definition 3.4. Let $\mathcal{P} := \{u_i\}_{i=0}^4$ be an arbitrary partition of the interval [0, 1], such that

$$0 = u_0 \le u_1 \le u_2 \le u_3 \le u_4 = 1$$

Then, we say that the intensity affects up to the i-th layer if and only if

$$u_i \leq \rho \leq u_{i+1}$$

Let us suppose that we are in generation k, and $(\rho, \theta) \in [0, 1] \times [0, 2\pi]$. Therefore, the intensity ρ affects up to the m-th layer. It is clear that $m \in \{0, 1, 2, 3\}$ and $\xi = T(\theta) \in [0, \frac{\pi}{2}]$.

First of all, let us suppose that m > 0 then ρ affects up to one layer, at least. Thus, we develop an **enlargement process**, which aims to provide the model with more infection capacity for further cells, depending on the intensity of the wind in each generation. So, we build the matrix A_m by means of this **enlargement process** which is described in Equation (4).

$$A_{1} = \begin{pmatrix} \sin \xi & f(\xi) \\ 0 & \cos \xi \end{pmatrix}$$

$$\vdots$$

$$A_{n} = \begin{pmatrix} a_{00} & a_{01} & \cdots & a_{0n} \\ a_{10} & a_{11} & \cdots & a_{1n} \\ \vdots & \ddots & \vdots \\ a_{n0} & a_{n1} & \cdots & a_{nn} \end{pmatrix}$$

$$A_{n+1} = \begin{pmatrix} \frac{a_{00}}{C} & \cdots & \frac{a_{0n-2} + a_{0n-1}}{C} & \frac{a_{0n-1}}{C} \\ & & \frac{a_{0n-1} + a_{1n-1}}{C} \\ \vdots & & \vdots \\ & \frac{a_{n-1} - 1}{C} \end{pmatrix}, \quad C \ge 1$$

As we can see above, the recursive process adds one column and one row to the previous matrix. In order to calculate the new entries, we only carry out divisions by a constant C or averages between two inputs which have just been calculated. Note that $A_m \in \mathbb{M}_{(m+1)\times(m+1)}([0,1])$, where $A_m = (a_{rl})_{r,l=0}^m$.

Let's observe that we have constructed the matrix A_m over ξ . In order to get the matrix for angle θ , we are going to use A_m . For this reason we consider $B = \left(b_{ij}\right)_{i,j=0}^{2m} \in \mathbb{M}_{(2m+1)\times(2m+1)}([0,1])$, such that

$$B = \left(\frac{0 \, \middle| \, A_m}{0 \, \middle| \, 0}\right) \in \mathbb{M}_{(2m+1) \times (2m+1)}([0,1])$$

If $Q(\theta) = 1$ then B will be the matrix for θ . Otherwise, it is sufficient to carry out:

- Make the symmetry on the vertical line x = 0 of B, when $Q(\theta) = 2$
- Make the symmetry on the diagonal line y = -x of B, when $Q(\theta) = 3$

• Make the symmetry on the horizontal line y = 0 of B, when $Q(\theta) = 4$.

Therefore, we can assume that the matrix B is built on θ and oriented correctly. The square matrix B provides us the probability of infection values for an arbitrary cell infected. But, it is clear that, $\forall (i,j) \in I_k \neq \emptyset$ has infectious capacity, hence we define the matrix $P^{(i,j)} = \left(p_{rl}^{(i,j)}\right)_{r,l=0}^{N-1} \in \mathbb{M}_{N \times N}([0,1])$, where the entries satisfy (5):

$$p_{rl}^{(i,j)} := \begin{cases} b_{(r-i+m),(l-j+m)} & if \ (r,l) \in D \\ 0 & \text{otherwise} \end{cases}$$
 (5)

and

$$D := \{i - m, \dots, i + m\} \times \{j - m, \dots, j + m\} \cap \{0, \dots, N - 1\}^2$$

In other words, the square matrix $P^{(i,j)}$ of size N stores the infection probabilities of the cells around the infected cell (i,j), but places them in the corresponding position. That is, it places the infection probability of cell $(k,l) \in \{0,\ldots,N-1\}^2$ in row k and column l. It also satisfies that $p_{ij}^{(i,j)} = 0$.

Finally, we only have to add this kind of matrix $P^{(i,j)} \forall (i,j) \in I_k$. Thus, our neighbourhood relationship is

$$R^{k}(\rho,\theta) = \sum_{(i,j)\in I_{k}} P^{(i,j)} \tag{6}$$

Remark 3.2. Note that $R^k(\rho, \theta)$ is a summation, so its entries can exceed the unity. Thus, $R^k(\rho, \theta) \in \mathbb{M}_{N \times N}(\mathbb{R})$. Thanks to the update criterion, this fact has no influence.

For the case where m = 0, we will set an arbitrary probability p_0 , and we will consider that B satisfies (7)

$$B = \begin{pmatrix} p_0 & p_0 & p_0 \\ p_0 & 0 & p_0 \\ p_0 & p_0 & p_0 \end{pmatrix}$$
the wind is not enough for this climatic variable to have any effect on the

When $\rho < u_1$ it is considered that the intensity of the wind is not enough for this climatic variable to have any effect on the spread of the phenomenon. Therefore, all the elements in the environment of the infected cell have the same probability of being infected.

Once the probabilities have been determined, we need to establish a criterion that allows us to update the state of our grid. For ease of notation, we consider $P^k = R^k(\rho, \theta)$, such that $P^k = \left(p_{ij}^k\right)_{i,j=0}^{N-1}$. Thus, the entries of M^{k+1} will verify Equation (8)

$$\forall \ m_{ij}^{k} = 2 \implies m_{ij}^{k+1} = 2
\forall \ m_{ij}^{k} = 1 \implies m_{ij}^{k+1} = 2
\forall \ m_{ij}^{k} = 0 \implies m_{ij}^{k+1} := \begin{cases} Be(p_{ij}) \ si \ 0 \le p_{ij}^{k} < 1 \\ 1 \ si \ p_{ij}^{k} \ge 1 \end{cases}$$
(8)

Remark 3.3. If our cell is infected, then it could remain as an infectious cell during a particular number of steps, after that it dies. This **delay** will be a tuning parameter in our model which will be denoted as Δ_{ID} .

From (8) it can be deduced that we assume that $\Delta_{ID} = 1$. But to generalise the algorithm we could consider $\Delta_{ID} \ge 1$ and update our state according to this parameter, i.e.

$$\forall \ 1 \le m_{ij}^k < 2 \ \Rightarrow \ m_{ij}^{k+1} = m_{ij}^k + \frac{1}{\Delta_{ID}}$$

Observation 1. Let us note that $Be(p_{ij})$ is a Bernouilli random variable with probability of success p_{ij}^k , that is, we obtain 0 in case of failure in the test, or 1 in case of success. In this way, we add to the model the uncertainty characteristic of the phenomenon under study.

Therefore, we can state that our model will be **stochastic** in that its operation depends on random variables. At the same time, we have to be careful in the **implementation**, since computers work with **pseudo-random** numbers.

Example 2. Let us suppose that we have a square grid with size N = 5. Let M^0 be the initial state such that

Since only $m_{2,2}^0 = 1$, then $I_0 = \{(2,2)\}$. Let us suppose that $\theta = \frac{\pi}{4}$ then θ is in the first quadrant. Moreover, the intensity ρ is in the second part of the interval, i.e., m = 1. Hence, we only have one non-zero row and column. Thus, applying the equations (4) and (6), we get

Now, it is clear that $I_1 = \{(1,3), (2,3)\}$. Let us suppose that, $\theta = \frac{5\pi}{6}$, ρ such that m = 2 and C = 2. Thus, it is clear that $T(\theta) = \frac{\pi}{6}$, so if we apply our **enlargement process**, we get

$$A_{1} = \begin{pmatrix} \frac{1}{2} & \frac{\sqrt{3}}{3} \\ 0 & \frac{\sqrt{3}}{2} \end{pmatrix} \Rightarrow A_{2} = \begin{pmatrix} \frac{1}{4} & \frac{3+2\sqrt{3}}{12} & \frac{\sqrt{3}}{6} \\ \frac{1}{2} & \frac{\sqrt{3}}{3} & \frac{5\sqrt{3}}{12} \\ 0 & \frac{\sqrt{3}}{2} & \frac{\sqrt{3}}{4} \end{pmatrix} \underbrace{\longrightarrow}_{Q(\theta)=2} B = \begin{pmatrix} \frac{\sqrt{3}}{6} & \frac{3+2\sqrt{3}}{12} & \frac{1}{4} & 0 & 0 \\ \frac{5\sqrt{3}}{6} & \frac{3+2\sqrt{3}}{12} & \frac{1}{4} & 0 & 0 \\ \frac{5\sqrt{3}}{12} & \frac{\sqrt{3}}{3} & \frac{1}{2} & 0 & 0 \\ \frac{\sqrt{3}}{4} & \frac{\sqrt{3}}{2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

Let us observe that θ is in the second quadrant, therefore, we have had to rotate the matrix B according to the second quadrant. If we apply this matrix for every infected element, we get

Finally, we have to add the results and apply the update criterion. Thus,

Definition 3.5. If the element (i, j) is infected in generation k. Then its **marginal infectiousness** is the number of infections it would produce in generation k + 1, assuming that all its exposed cells can be infected.

Property 3.1. If (i, j) is infected and let $\mathcal{P} := \{u_i\}_{i=0}^4$ be an arbitrary partition of the interval $[0, 1], p_0 \in [0, 1]$ and $C \in \mathbb{R}$. Then,

1. The expected marginal value of infections per generation E for our model is

$$E(\mathcal{P}, C, p_{0}) = 8 u_{1} p_{0} + (u_{2} - u_{1}) \int_{0}^{\frac{\pi}{4}} g(\theta, C, 0) d\theta$$

$$+ (u_{3} - u_{2}) \int_{0}^{\frac{\pi}{4}} \left(\sum_{i=0}^{1} g(\theta, C, i) + g_{0}(\theta, C) \right) d\theta$$

$$+ (1 - u_{3}) \int_{0}^{\frac{\pi}{4}} \left(\sum_{i=0}^{2} g(\theta, C, i) + g_{0}(\theta, C) + g_{0}(\theta, C^{2}) + g_{1}(\theta, C) \right) d\theta$$

$$(9)$$

2. The probability P that no one will be infected by the marginal effect of (i, j) is

$$P(\mathcal{P}, C, p_0) = u_1 (1 - p_0)^8 + (u_2 - u_1) \int_0^{\frac{\pi}{4}} h(\theta, C, 0) d\theta$$

$$+ (u_3 - u_2) \int_0^{\frac{\pi}{4}} h(\theta, C, 1) h_1(\theta, C) d\theta$$

$$+ (1 - u_3) \int_0^{\frac{\pi}{4}} h(\theta, C, 2) h_1(\theta, C) h_1(\theta, C^2) h_2(\theta, C) d\theta$$
(10)

Proof. By symmetry we can assume without loss of generality that $\theta \in [0, \frac{\pi}{4}]$ and ρ affects up to the *m*-th layer. Thus, in this case $\theta \sim Unif(0, \frac{\pi}{4})$, and $0 \le m \le 3$.

1. Let

 $\mathbb{I} := \{ \text{ number of infected by the marginal effect of } (i,j) \}$

Then it is clear that \mathbb{I} , depends on θ and m. Therefore, as we know distributions of θ and ρ (i.e. m as well), we can compute the expected value of \mathbb{I} according to our parameters:

$$\begin{split} E(\mathcal{P},C,p_0) &= E[\mathbb{I}] = \int\limits_0^{\frac{\pi}{4}} \frac{4}{\pi} \sum_{k=0}^3 E[\mathbb{I}|\theta,\,m=k] \, P(m=k) \, d\theta \\ &= 8 \, u_1 \, p_0 \, + \, (u_2 - u_1) \int\limits_0^{\frac{\pi}{4}} g(\theta,C,0) \, d\theta \, + \, (u_3 - u_2) \int\limits_0^{\frac{\pi}{4}} \left(\, \sum_{i=0}^1 \, g(\theta,C,i) \, + \, g_0(\theta,C) \right) d\theta \\ &+ \, (1 - u_3) \int\limits_0^{\frac{\pi}{4}} \left(\, \sum_{i=0}^2 \, g(\theta,C,i) \, + \, g_0(\theta,C) \, + \, g_0(\theta,C^2) \, + \, g_1(\theta,C) \right) d\theta \end{split}$$

Where, we have defined the functions as follows

$$g(\theta, C, i) = \frac{4}{\pi} \frac{\sin \theta + \tan \theta + \cos \theta}{C^i}, \quad g_0(\theta, C) = \frac{4}{\pi} \frac{\sin \theta + 2 \tan \theta + \cos \theta}{2 C}$$

$$g_1(\theta, C) = \frac{4}{\pi} \frac{\sin \theta + 6 \tan \theta + \cos \theta}{(2 C)^2}$$
(11)

2. Let the event

 $N := \{ \text{no element is infected by the marginal effect of } (i, j) \}$

Then, as the previous case, the event N depends on θ and m. Therefore, we can also compute P(N) using the distributions of θ and ρ . Thus,

$$\begin{split} P(\mathcal{P},C,p_0) &= P(N) = \int_0^{\frac{\pi}{4}} \frac{4}{\pi} \sum_{k=0}^3 P(N \mid \theta, m = k) P(m = k) d\theta \\ &= u_1 (1 - p_0)^8 + (u_2 - u_1) \int_0^{\frac{\pi}{4}} h(\theta,C,0) d\theta + (u_3 - u_2) \int_0^{\frac{\pi}{4}} h(\theta,C,1) h_1(\theta,C) d\theta \\ &+ (1 - u_3) \int_0^{\frac{\pi}{4}} h(\theta,C,2) h_1(\theta,C) h_1(\theta,C^2) h_2(\theta,C) d\theta \end{split}$$

Where we have also defined the functions as follows

$$h(\theta, C, i) = \frac{4}{\pi} \prod_{j=0}^{i} \left(1 - \frac{\sin \theta}{C^{i}} \right) \left(1 - \frac{\tan \theta}{C^{i}} \right) \left(1 - \frac{\cos \theta}{C^{i}} \right)$$

$$h_{1}(\theta, C) = \left(1 - \frac{\sin \theta + \tan \theta}{2C} \right) \left(1 - \frac{\cos \theta + \tan \theta}{2C} \right)$$

$$h_{2}(\theta, C) = \left(1 - \frac{\sin \theta + 3 \tan \theta}{(2C)^{2}} \right) \left(1 - \frac{\cos \theta + 3 \tan \theta}{(2C)^{2}} \right)$$

$$(12)$$

Corollary 3.1. If C = 2, $u_1 = 0.1$, $u_2 = 0.5$, $u_3 = 0.9$ and $p_0 = 0.25$. Then, for the primitive state of our lattice k = 0, the expected value of infections is $E \approx 2.53548$. Furthermore, the probability that, $I_1 = \emptyset$, is $P \approx 0.01804$.

Example 3. By Property 3.1, the expected marginal value of infections E and the probability P that no one will be infected depend on the partition P, on p_0 and also on the constant C. Therefore, all of these parameters will be a tuning parameters, which may have an effect on the rate of phenomenon spread.

If we focus on the constant C and given a partition \mathcal{P} of the interval [0,1], such that $u_1=0.1,\,u_2=0.5,\,u_3=0.9$ and $p_0=0.25$, we can see the behaviour of it in the Figure 1.

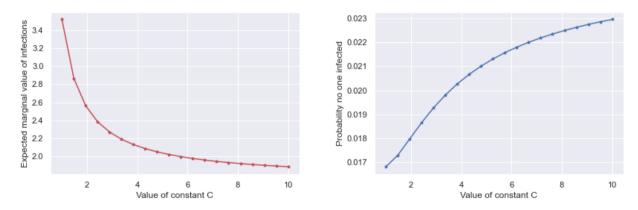


Figure 1 The expected values of infections according to the value of C (left). Probability of no one infected according to the value of C(right).

Clearly, when the value of C increase, the probability of infection for further cells decrease as we divide by a larger number. Therefore, there will be a gradual fall for the expected marginal value and slight grew for the probability of no one infected.

4 | CONCLUSIONS

In this study, we have presented a grid-based stochastic model of the propagation of adverse environmental events enhanced by the wind. The model highlights two key elements such as neighbourhood relationship among the cells of the grid and update criterion. The results show a non-symmetrical expansion and at the same time the spread and scope of the phenomenon depend on climate variables. In addition, when building the expansion model, we have defined a set of adjustable parameters, which will have an effect on the rate of propagation of the phenomenon.

Regarding future work, we could change our SID model to Susceptible-Exposed-Infected-Dead (SEID) scheme. This means insert one more step, an Exposition level and hence, determine another parameter such as Δ_{EI} which represents the delay between the Exposed and the Infected steps. More, we could consider these delay and perhaps similar parameters as climate functions as well. In other words, they may depend on temperature and humidity, among others climate variables.

Finally, as future work, we plan to make a comparison between our results and those provided by the models proposed in reference 12. At the same time, we intend to obtain data on real phenomena with sufficient quality to test our model.

References

- 1. Anderson RM, May RM. Infectious diseases of humans: dynamics and control. Oxford university press . 1992.
- 2. Eubank S, Guclu H, Anil Kumar V, et al. Modelling disease outbreaks in realistic urban social networks. *Nature* 2004; 429(6988): 180–184.
- 3. Hufnagel L, Brockmann D, Geisel T. Forecast and control of epidemics in a globalized world. *Proceedings of the national academy of sciences* 2004; 101(42): 15124–15129.
- 4. Malthus TR, Winch D, James P. Malthus: 'An Essay on the Principle of Population'. Cambridge University Press . 1992.
- 5. Wangersky PJ. Lotka-Volterra population models. Annual Review of Ecology and Systematics 1978; 9(1): 189-218.
- 6. Kermark M, Mckendrick A. Contributions to the mathematical theory of epidemics. Part I. *Proc. r. soc. a* 1927; 115(5): 700–721.
- 7. Kuang Y, Nagy JD, Eikenberry SE. Introduction to mathematical oncology. Chapman and Hall/CRC . 2018.
- Signes-Pont MT, Cortés-Castillo A, Mora-Mora H, Szymanski J. Modelling the malware propagation in mobile computer devices. *Computers & Security* 2018; 79: 80–93.
- 9. Signes Ponnt M, Mora H, Cortés CA. The Susceptible-Infectious-Recovered (SIR) model of disease expansion: a new approach. In: ; 2017.
- 10. Signes-Pont MT, Cortés-Plana JJ, Mora H, Mollá-Sirvent R. An epidemic model to address the spread of plant pests. The case of *Xylella fastidiosa* in almond trees. *Kybernetes* 2020.
- 11. Huang CY, Chin WCB, Wen TH, Fu YH, Tsai YS. EpiRank: Modeling bidirectional disease spread in asymmetric commuting networks. *Scientific reports* 2019; 9(1): 1–15.
- 12. Signes Pont MT, Ramirez-Martinez DE, García-Chamizo JM, Mora H, others . A multigrid approximation to the expansion of Xylella fastidiosa in almond trees. 2019.