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SHORT COMMUNICATION

Contribution to COVID-19 spread modelling: a physical phenomenological dissipative formalism

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Abstract
In this study, we propose an evolution law of COVID-19 transmission. An infinite ordered lattice represents population. Epidemic evolution is represented by a wave-like free spread starting from a first case as an epicentre. Free energy of the virus on a given day is defined equal to the natural logarithm of active infected cases number. We postulate a form of free energy built using thermodynamics of irreversible processes in analogy to isotherm wave propagation in solids and non-local elastic damage behaviour of materials. The proposed expression of daily free energy rate leads to dissipation of propagation introducing a parameter quantifying measures taking by governments to restrict transmission. Entropy daily rate representing disorder produced in the initial system is also explicitly defined. In this context, a simple law of evolution of infected cases as function of time is given in an iterative form. The model predicts different effects on peak of infected cases $I_{\max}$ and epidemic period, including effects of population size N, effects of measures taking to restrict spread, effects of population density and effect of a parameter T similar to absolute temperature in thermodynamics. Different effects are presented first. The model is then applied to epidemic spread in Tunisia and compared with data registered since the report of the first confirmed case on March 2, 2020. It is shown that the low epidemic size in Tunisia is essentially due to a low population density and relatively strict restriction measures including lockdown and quarantine.

Keywords COVID-19 · Transmission · Lattice · Wave · Free energy · Dissipation

1 Introduction

First cases of pneumonia unknown etiologies have been declared in Wuhan, China, since December 8, 2019. Pneumonia starts with severe acute respiratory infection symptoms and some cases developed acute respiratory distress syndrome with failure complications. On January 7, 2020, Chinese centre for disease control and prevention identified a new coronavirus (Chen et al. 2020).

COVID-19 is a human coronavirus include in the gender beta coroanvirus group 2b, family coronaviridae. It is the third strain of virus of the coronavirus family (CoV), isolated in humans in the context of an epidemic after SARS-CoV in China (2002) and MERS-CoV in Saudi Arabia (2012). Examination of the COVID-19 genome showed genetic similarity to SARS-CoV about 79.5%. Human to human transmission takes place by either respiratory droplets or close contacts. According to the world health organization, COVID-19 is a virus with unique characteristics that causes respiratory disease and which spreads via oral and nasal droplets (Kolifarhood et al. 2020).

On March 2, the first case has been declared in Tunisia.

Actually, COVID-19 is causing a disease representing a planetary problem for public health and negative impact on humanity (Boccaletti et al. 2020).

The objective of this paper is to propose a simple model to predict COVID-19 transmission using early data of the outbreak. Majority of epidemic transmission models are based on compartmental mathematical models dividing population in different interacting groups and assuming different rates of transmission between them. Solutions are conducted using integration of differential equations and principle of conservation (Kermack and McKendrick 1927). Population is generally assumed as a closed system, the probabilistic formalism of transmission between individuals of different

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groups leads to saturation and a population size effect on epidemic size and epidemic period. Models that are more sophisticated include also Monte Carlo numerical simulations for stochastic models and more realistic epidemic networks. See for example a review by House et al. (2013) and recent studies by Kim et al. (2020), Liang (2020) and Li et al. (2020) among several others for COVID-19 modelling.

Epidemic networks and lattice methods have their origin in social science and computer science (see for example a review by Keeling and Eames 2005). Lattice models are representation of an ordered network in which epidemic transmission is similar to a wave-like spread in regular grid representing connected individuals. Epidemic starts from an epicentre and spreads out in a roughly circular manner. Figure 1 illustrates an example in two dimensions. Lattice models are suitable for example for forest-fire models (Bak et al. 1990) where nodes represent trees that burn leaving empty sites. Keeling and Eames (2005) interpreted this representation as similar to epidemic transmission.

This paper proposes, in this context, a phenomenological model of propagation and dissipation in analogy to elastic wave propagation and a size and temperature-dependent elastic damage material model (Ben Hassine et al. 2019; Limam et al. 2014). The advantage of a formalism inspired from thermodynamics is that different effects emerge from principles.

2 Materials and methods

Wave propagation is particularly studied in biomechanical applications for example in evaluation of dental implant stability (Vayron et al. 2015). In addition, elastic damage models are considered in geomechanics and biomechanics to model shock waves dissipation. For example, Nelms et al. (2017) applied a finite element elasto-plastic damage model

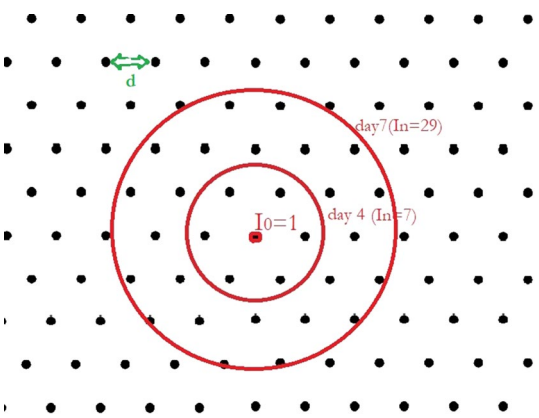

Fig. 1 Lattice representation of population with a wave-like epidemic spread

in order to evaluate mechanical shock waves decay in cement microstructure. Fovargue et al. (2018) developed a model for kidney stone fragmentation in shock wave lithotripsy assuming elastic damage behaviour with reduction in the apparent Young modulus, in a context of thermodynamics of generalized standard materials behaviour (Lemaître and Chaboche 1978). Non-local damage mechanics are particularly considered to model shock waves dissipation. For example, Lu et al. (2005) developed a non-local damage approach to model damage wave slow propagation in solids. Non-local damage approaches predict a size effect on macroscopic constitutive behaviour (Pijaudier-Cabot and Bazant 1987). Size effect phenomenon was experimentally observed in biomechanics since the time of Galileo.

Furthermore, Limam et al. (2014) and Ben Hassine et al. (2019) proposed that, for isotherm processes, a scaling law should be associated with specific entropy S depending on size N and that the specific free energy φ should be written $\varphi = U - TS(N)$. In fact, internal energy is extensive and specific internal energy U should be independent of N. On the contrary, entropy is non-additive at small scales and become additive as size N tends to infinity (Tsallis 2009). Irreversible isotherm processes assume equilibrium at successive states and allow heat transfer in agreement with Fourier law. For this isotherm case, specific free energy φ linearly decreases as temperature increases. This was confirmed experimentally. We can cite, for example, structural health monitoring related to concrete and composite structures (Wang et al. 2018; Moll et al. 2019).

On the contrary, Carlioz et al. (2019) showed that material damage corresponding to high velocity process and to sudden and abrupt nature of a crack nucleation process is adiabatic rather than isothermal with temperature rising at crack tip during dynamic crack propagation due to heat accumulation at solid boundaries.

In this paper, in the case of epidemic transmission context, isotherm open system hypothesis will be considered in analogy to isothermal damage process allowing heat transfer. Physicians who modelled epidemics spread using information entropy concept and epidemic thermodynamics, assumed this hypothesis. For example Koivu-Jolmaa and Annilaa (2018) proposed an isotherm natural process based on thermodynamics of open systems to predict Ebola virus transmission using statistical mechanics and analogy to chemical potentials. Tsallis and Tirnakli (2020) developed analytical model to predict COVID-19 transmission based on non-extensive Tsallis Entropy.

The proposed scientific method proposed in this paper is developed in the same context of isotherm open systems considered for example by Koivu-Jolmaa and Annilaa (2018) and Tsallis and Tirnakli (2020), but with a free energy postulated equal to the natural logarithm of active infected cases and written in the form $U - TS(N)$. The proposed approach

can be viewed as part of epidemic physical models. It represents also a complement to compartmental mathematical models and complex network based on sophisticated approaches taking in to account real interaction between different groups and heterogeneous fields of motion and density depicted in real maps (Gomez et al. 2020). We adopt a method based on an empirical macroscopic description of epidemic spread inspired from mechanics and thermodynamics. It is noted that phenomena related to effects of ambient temperature or population size on COVID-19 epidemic transmission were recently reported in the literature. The proposed model is presented and explained in Sect. 3. In Sect. 4, the model is applied to study different effects including measures to restrict spread, effect of population density and size and effect of a parameter T similar to absolute temperature in thermodynamics. Epidemic evolution in Tunisia is also analysed. We consider for comparison, data from national observer for new and emergent diseases (https://www.onmne.tn) until June 12.

We present also an "Appendix". Firstly, this appendix aims to present the considered mechanical model (Ben Hassine et al. 2019). We present the simplified case of unidirectional behaviour, which is macroscopic, and size dependent. This model is therefore non-local in the sense of damage mechanic. Secondly, we present some phenomenological similarities between virus spread and mechanical wave spread. Finally, this appendix includes also a table with both, epidemiology and damage mechanics to list the similarities, parameters, Equations local or non-local and their interpretation.

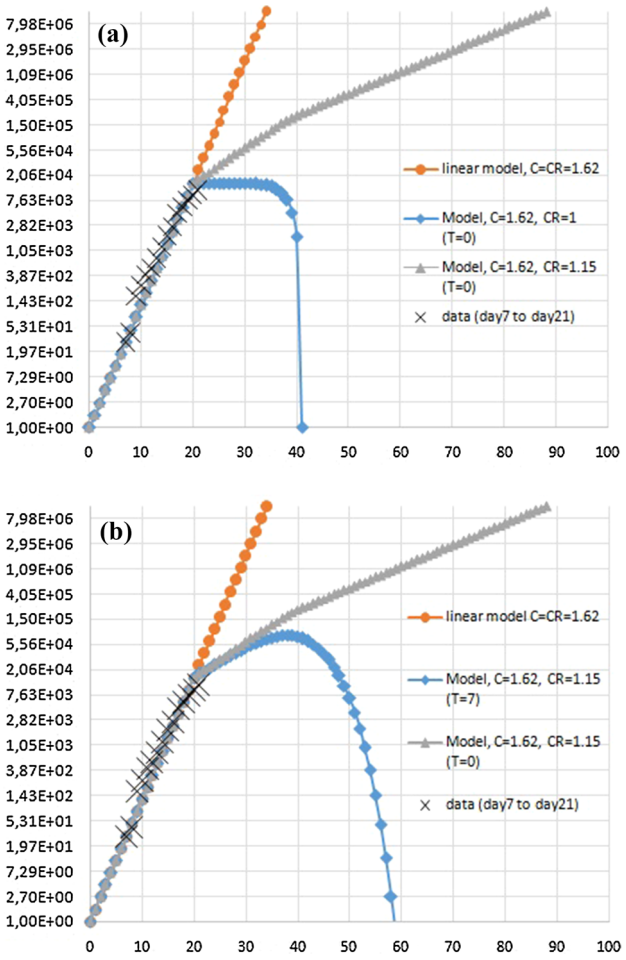

Fig. 2 Infected cases as function of time (day n): analogy to wave spread in elastic damage material

3 Theory

Consider first a simple mathematical model given by Eqs. (1) and (2). I_n is the number of infected people on day n. Theses Equations correspond to epidemic theoretical free transmission in a population of size N defined in a finite roughly circular convex domain part of a perfect infinite ordered lattice of connected people and starting from an epicentre $I_0 = 1$ belonging to the domain. Population density is inversely proportional to the square of distance d as depicted in Fig. 1. Coefficient C defined by Eq. (1) is considered as an intrinsic characteristic of population density and independent of population size. It is clear that it decreases when distance d increases which means that when population density decreases. It represents the number of transmission between every infected person at wave front to other persons. A theoretical free transmission in the lattice corresponds to the linear curve with a slope ln[C] in a semi-logarithmic scale as depicted in Fig. 2a, b for a domain representing, for example, a typical dense city with $N = 12$ Million and $C = 1.62$. In that case, population size N will be reached at $n_s = \frac{\ln(N)}{\ln(C)}$, on day 34; meanwhile epidemic will continue to propagate in the lattice outside the population domain, as the considered population is fixed but transmission to the outside was made possible by hypotheses.

$$I_{n+1} = CI_n \tag{1}$$

$$I_n = C^n I_0 \quad (I_0 = 1) \tag{2}$$

In reality transmission, process is dissipative and the linear curve of slope ln[C] corresponds only to early stage. It can be deduced, for example, from initial data curves fitting in a semi-logarithmic curve as shown in Fig. 2. This coefficient should be reduced by measures imposed by governments including lockdown and quarantine. It is reduced also by population behaviour including social distancing, personal hygiene, for example by wearing a mask in fear of the spread of the virus. This is similar to the effect described by, for example, Kim et al. (2020) or Liang (2020).This

behaviour emerges naturally after first deaths inducing a disorder and long range interaction in the initial lattice of Fig. 1, which means physically that entropy should increase.

The idea of the proposed model can be highlighted when we made an empirical analogy of the linear curve as depicted in Fig. 2 with energy as function of time of elastic wave propagation in a rod obeying Hooke's law of elasticity and submitted to harmonic imposed power. Therefore, we defined by analogy free energy of the virus spread by $\varphi = \ln(I_n)$ which gives a constant daily rate in the case of linear curve as depicted in Fig. 2 and defined by Eqs. (1) and (2).

In reality, material behaviour as epidemic spread is dissipative and wave velocity will decrease due to material damage. In analogy to reduction in Young modulus of elasticity in damage mechanics (Kachanov 1958), we should introduce a reduction in C in a semi-logarithmic scale. Therefore, we postulate the evolution model of infected cases I_n on day n, given by Eqs. (3–7), where T is a parameter similar to absolute temperature in thermodynamics and where a first case is $I_0 = 1$.

$$\frac{I_{n+1}(T)}{I_n(T)} = C(n)\frac{\left(1 - D_n\right)}{\left(1 + F_n\right)^T} \tag{3}$$

$$D_n = 0 \text{ for } n \le 21 \text{ and } D_n = \frac{J_{n-21}}{J_{n+1}} \text{ for } n > 21 \tag{4}$$

$$F_n = 0 \text{ for } n \le 21 \text{ and } F_n = \frac{J_n}{N} \text{ for } n > 21 \tag{5}$$

$$\text{With } \frac{J_{n+1}}{J_n} = C(n)\left(J_0 = 1\right) \tag{6}$$

$$C(n) = C \text{ for } n \le 21 \text{ and } C(n) = CR \text{ for } n > 21 \tag{7}$$

In order to give a physical sense to these Equations, we define first a free energy rate of the virus noted $\varphi_{n+1} - \varphi_n = \Delta\varphi$ given by Eq. (8). In a thermodynamically consistent isotherm framework, Helmholtz free energy is defined by the rate $\Delta\varphi = \Delta U - T\Delta S$, where ΔU is internal energy rate and ΔS is entropy rate given, respectively, by Eqs. (9) and (10) in the case of virus spread analogy and identified from Eq. (8).

$$\Delta\varphi = \ln\left(\frac{I_{n+1}}{I_n}\right) = \ln\left(C(n)\left(1 - D_n\right)\right) - T\ln\left(1 + F_n\right) \tag{8}$$

$$\Delta U = \ln\left(C(n)\left(1 - D_n\right)\right) \tag{9}$$

$$\Delta S = \ln\left(1 + F_n\right) \tag{10}$$

In order to consider lockdown and quarantine effect, C is decreased from day 22 and noted CR, a coefficient between 1 and C (Eq. 7). Equation (4) is introduced to model recovering or death from day 22 with parameter D_n. This is justified by recent studies reporting that observed duration of viral shedding among survivors was between 8 and 37 days (Zhou et al. 2020). This means an average of 22 days corresponding to a first death. This means also different behaviours of governments and people for different time intervals $n \le 21$ and $n > 21$ before and after this event. Equations (4, 6 and 7) define different coefficients C and CR to distinguish between no measures before the event and measures taking after the event. Evolution law of damage defined by these Equations is independent of population size and temperature and can be considered as an intrinsic property of virus spread but dependent upon people density through coefficient C and measures taking through coefficient CR. C and CR are therefore depend on different cities or countries. Equations (3) to (7) were inspired using similarities with non-local damage behaviour as shown in Table 1. As explained in the "Appendix" non-local damage predicts a size effect and local damage does not. In Table 1, the particular case of local damage behaviour corresponding to $T = 0$ is also presented, where Eq. (11) is obtained from Eq. (3) using Eqs. (4) and (6). We can underline here that Eq. (11) is a conservation Equation eliminating recovered or deceased individuals from infected individuals to obtain only active infected cases with a delay of 22 days. This Equation is similar to Equations defined by compartmental models as for example SIR or SEIR models. Meanwhile, the advantage of the present physical approach is that some intrinsic characteristics of virus spreading are considered in the model, including local interaction between individuals leading to its transmission and non-local interactions leading to its dissipation due to an entropic effect as explained in analogy to the non-local damage model presented in the "Appendix".

$$\frac{I_{n+1}(0)}{I_n(0)} = C(n)\left(1 - D_n\right) = \frac{J_{n+1}}{J_n}\left(1 - \frac{J_{n-21}}{J_{n+1}}\right) = \frac{J_{n+1} - J_{n-21}}{J_n} \tag{11}$$

Its application with $CR = 1.15$ ignoring entropy dissipative effect which means with $T = 0$, leads to the trilinear curve presented in Fig. 2a, b where population size will be reached with an epidemic period $n_s = 89$ days for the considered example in Fig. 2. Figure 2a shows also the theoretical case of an ideal lockdown with $CR = 1$ and $T = 0$. This leads to an epidemic period of 40 days due to a recovering rate higher than infection rate in that case with an epidemic peak of 11,200, reached on day 21 and independent of N. Evolution law of damage given by Eqs. (4, 6 and 7) is defined exclusively by C and CR and remains

Table 1 Phenomenological analogy between damage mechanics and virus spread

Mechanical model	Virus spread
Time (t)	Time (day n)
Cartesian scale	Semi-logarithmic scale
Young modulus E	Coefficient C
Material density constant	Density of population constant
Unidirectional semi-infinite rod $x \in [0, +\infty[$ considered system $x \in]0, L[$ system size L	Population of size N part of infinite population
constant imposed power at $x=0$	First case
Local elasticity	*Linear spread (Eqs. 1 and 2)*
Stored energy proportional to $\varphi \propto Et$	Free energy of the virus $\varphi_n = \ln(I_n) = n\ln(C)$
Specific internal power proportional to Young modulus $\frac{\partial \varphi}{\partial t} \propto E$	Free energy rate of the virus $\Delta\varphi_n = \frac{\varphi_{n+1}-\varphi_n}{n+1-n} = \ln\left(\frac{I_{n+1}}{I_n}\right) = \ln(C)$
Local damage (without size and temperature effect)	*Non linear spread (without size and temperature effect) (Eq. 11)*
Macroscopic damage parameter due to a shock wave D	Damage parameter of virus spread D_n
Specific internal power proportional to $\frac{\partial \varphi}{\partial t} \propto (1-D)E$	Free energy rate of the virus $\Delta\varphi = \ln\left(\frac{I_{n+1}}{I_n}\right) = \ln\left(C(n)\left(1-D_n\right)\right)$
Independent of L	Independent of N
Non local damage (with size and temperature effect)	*Non linear spread (with size and* temperature effect) Eqs. (3–10)
Specific internal power proportional to $\frac{\partial \varphi_L}{\partial t} \propto (1-D-Tk(L)\ln(1+D))E$	Free energy rate of the virus $\Delta\varphi = \ln\left(\frac{I_{n+1}}{I_n}\right) = \ln\left(C(n)\left(1-D_n\right)\right) - T\ln\left(1+F_n\right)$
Specific internal energy $U \propto (1-D)E$	Internal energy rate $\Delta U = \ln\left(\frac{I_{n+1}}{I_n}\right) = \ln\left(C(n)\left(1-D_n\right)\right)$
Independent of L	Independent of N
Specific entropy $S(L) \propto k(L)\ln(1+D)E$	Entropy rate $\Delta S = \ln\left(1+F_n\right)$
Depend on L	Depend on N

independent of population size. This evolution law of damage induces a decreasing of internal energy rate defined by Eq. (9) which remains also independent of population size and temperature and equal to free energy rate when $T=0$. Effects of T and N are rather due to entropy production. Similar hypothesis was considered for a damage evolution law as an intrinsic characteristic of the material independent of specimens size and temperature see Ben Hassine et al. (2019) and (Limam et al. (2014).

Equation (5) is introduced to consider population size effect, also from day 21. It is worth mentioning that the considered free energy is choosing with an entropy rate $\Delta S = \ln\left(1+F_n\right)$, null before damage initiation ($F_n = 0$ for $(n \leq 21)$) and always positive, which means that entropy increases according to the second law of thermodynamics and contributes to dissipate free energy of the virus. Theoretical free transmission in an ordered lattice case given by Eqs. (1) and (2) can be obtained when considering $CR = C$, and $T=0$, in analogy to absolute zero state in thermodynamics, where entropy effect vanishes. Parameter T should be understood as for example hygiene measures in the system which can be linked also to ultraviolet rays increasing with ambient temperature rising. When increased it contributes to increase entropy effect and consequently to decrease free energy rate and epidemic spread. This is in agreement with recent environment studies shown also through statistical analysis of data that transmission decreases as ambient temperature increases, see for example Prata et al. (2020) and Liu et al. (2020).

Figure 2b shows an example applying the proposed model with $N=12$ Million, $C=1.62$, $CR=1.15$ and $T=7$. Introducing entropic effect, epidemic size is decreased with a peak of 79,000 infected cases and an epidemic period of 56 days. Parameters were chosen to give an order of epidemic comparable to a dense city like Wuhan (Liang 2020; Li et al. 2020); . C was identified from the first slope of data using a regression analysis between day 8 and day 21. This choice of linear behaviour was not arbitrary. In fact, we assumed that virus spread similarly to material behaviour is linear at early stages of loading. Similarities between local elasticity and linear spread are depicted in Table 1 ("Appendix").

4 Results

4.1 Restriction measures (CR) effect

Figure 3 presents an example of the model applied first with $N=12$Million, $C=1.62$, $CR=1.15$ and $T=7$ in a Cartesian scale and then with the same parameters but more restricted measures traduced by a reduction in C on day 21 to $CR=1.1$. It can be observed that $CR=1.1$ describes stricter measures that induce a decreasing of epidemic size and slightly increase epidemic period. The maximum of positive cases Imax is decreased from 79,000 to 46,000. Figure 3 presents

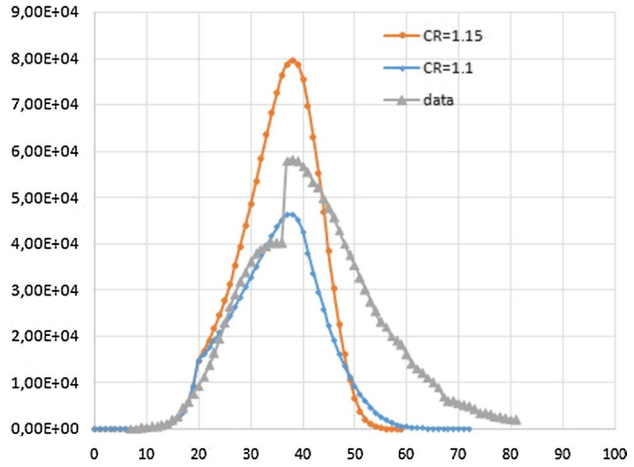

Fig. 3 Restriction measures effect on infected cases curve ($N=12E6$, $C=1.62$, $T=7$)

also real data of infected active cases in Wuhan, China (Liang 2020; Li et al. 2020), until epidemic wave end on March 2020. Real data show that I_{max} is equal to 57,900 cases, which means that CR is effectively between 1.1 and 1.15. It is observed also that the epidemic real period is larger than the predicted one. This can be explained by a possibility of a second epicentre with a shift in time and space from the first one. This can be confirmed also by the shift in the data curve shape. It can be noted here that the model assumes a unique epicentre. A superposition can be made to include the effect of a second epicentre.

4.2 Population size (N) effect

Figure 4 shows effect of population size N on infected cases in a Cartesian scale. C, CR and T are fixed and the size N is changed from 3 to 12 and 24 Million. It is deduced that when population size increases epidemic size and period increase, with respective maximums I_{max} of 25,000, 79,000 and 128,000 reached, respectively, on days 30, 39 and 42with, respectively, epidemic periods of about 41, 56 and 60 days. When N increases the ratio (I_{max}/N) decreases and, respectively, given by 0.83%, 0.66% and 0.53%. Figure 4 presents also real data of infected active cases in Wuhan, China. Comparison shows an agreement with data. Estimated population size in Wuhan is 11 Million.

4.3 Tunisian case, population density effect and T effect

Figure 5 presents data in Cartesian scale in Tunisia until April 25. On this date, our ministry of health reported 38 deaths and 194 recovered cases. The model is depicted and reproduces actual data tendencies, considering $C=1.3$ corresponding to initial data fitting in semi-logarithmic scale. It represents quarantine and lockdown effects thereafter with $CR=1.115$.The model reproduces data tendencies with $I_{max}=912$ and an epidemic period of 92 days, which means an epidemic spread end at the beginning of June if the same measures are maintained. It is noted that a reduction in C from 1.62 to 1.3 induces a reduction in epidemic peak of about 50 times as deduced when comparing Fig. 5 with Fig. 3. Furthermore, it is noted in Fig. 5 that parameter T when changed from 7 to 25 slightly decreases epidemic. Furthermore, Fig. 6 presents updated real data until June 12. A good agreement is observed between data and the model.

5 Discussion

An evolution law of COVID-19 based on analogy with wave propagation in elastic solids and a non-local damage model is proposed. The key coefficient C is obtained by a linear fitting of initial slope of data in a semi-logarithmic scale between day 8 and day 21. Results are very sensitive to this parameter, considered as an intrinsic parameter of

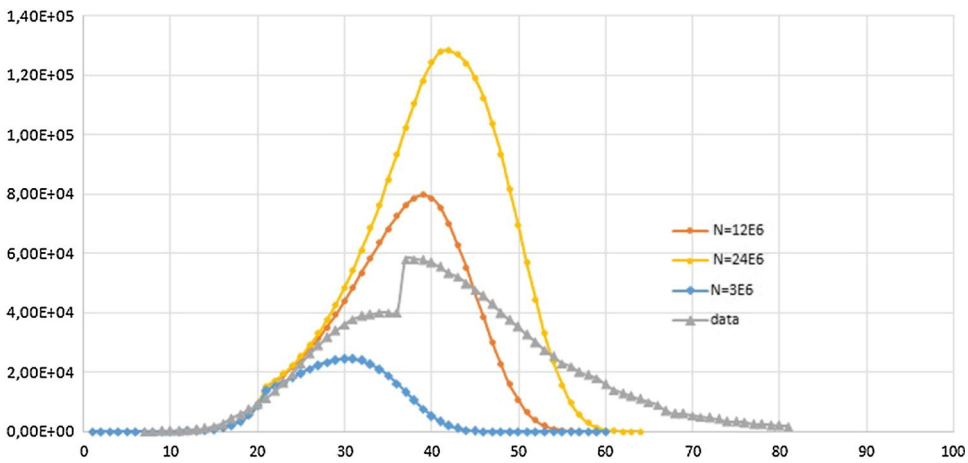

Fig. 4 Population size effect on infected cases curve ($C=1.62$, $CR=1.15$, $T=7$)

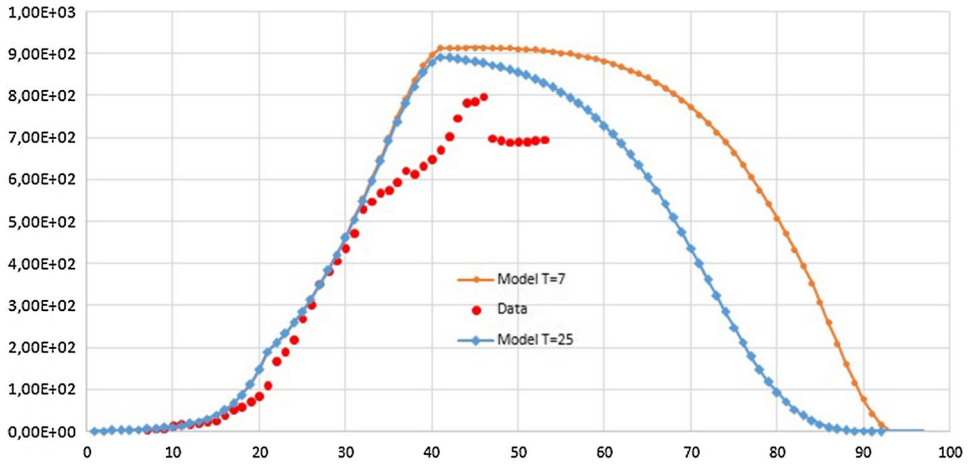

Fig. 5 COVID-19 spread in Tunisia ($N=12E6$, $C=1.3$, $CR=1.115$, $T=7$, $T=25$)

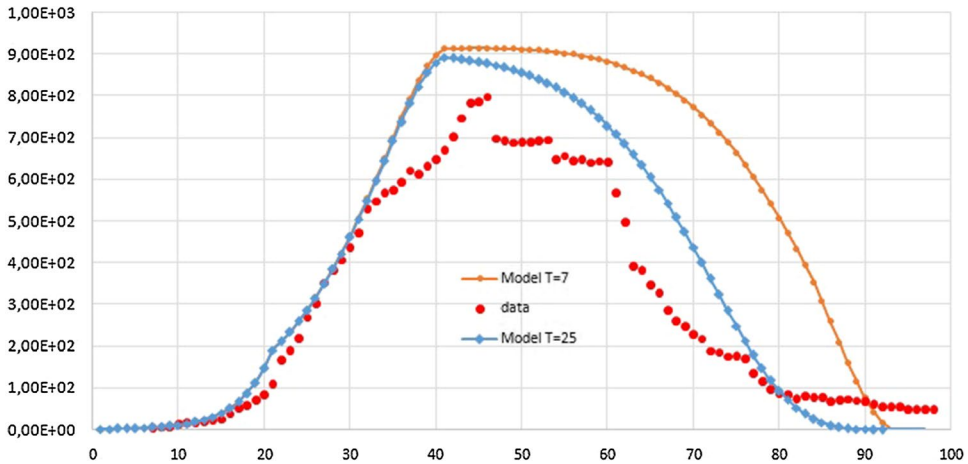

Fig. 6 COVID-19 spread in Tunisia with updated data until June 12

population density. For populations of a comparable size, but with coefficients, respectively, $C=1.3$ and $C=1.62$, results show that epidemic size can increase very fast. The second important parameter is CR. It traduces measures like lockdown and quarantine. When controlled, which means decreased, it decreases epidemic size.

6 Conclusion

The low predicted epidemic size in Tunisia is essentially due to a low population density ($C=1.3$) and strict restriction measures ($CR=1.115$). Population density is inversely proportional to the square of distance d depicted in Fig. 1 which explains its important effect on transmission. Information exchange, democratization of access to knowledge and long-range non-local interaction between humans at a planetary level contribute to dissipate pandemic and help humanity to be prepared to a possibility of a second wave.

Author contributions OL proposed the model and wrote the manuscript. ML developed medical aspects of the model and wrote the manuscript.

Compliance with ethical standards

Conflict of interest The authors have no conflicts of interest to declare for this study.

Appendix

Presentation of the mechanical non-local damage model

The model was applied to unidirectional isotherm behaviour of concrete under quasi-static compression. It was validated using comparison with experimental tests. It was derived in a context of generalized standard behaviour of material verifying Clausius–Duhem inequality (Limam et al. 2014). Constitutive macroscopic behaviour (Eq. 12)

exhibits a size and ambient temperature effects on macroscopic strength and secant modulus of elasticity with decreasing as size and temperature increase.

Consider a rod of length L and imposed displacements $u(0)$ and $u(L)$ at its boundaries.

Macroscopic elastic damageable constitutive behaviour associating macroscopic stress σ_L to macroscopic strain $\varepsilon_L = \frac{u(L)-u(0)}{L}$ is given by Eq. (12).

$$\sigma_L = \frac{\partial \varphi_L}{\partial \varepsilon_L} = \left(1 - D - Tk(L)D\left(1 - \frac{D}{2}\right)\right)E\varepsilon_L \tag{12}$$

This macroscopic behaviour derives from a chosen specific free energy φ_L given by Eq. (13) where D is damage parameter between 0 and 1, E is Young modulus and k is an increasing positive function with horizontal asymptote. A unique evolution law of damage parameter D as function of loading history was considered. This law is independent of the size L and absolute temperature. Evolution law of damage was considered as an intrinsic characteristic of material.

$$\varphi_L = \left(1 - D - Tk(L)D\left(1 - \frac{D}{2}\right)\right)E\frac{\varepsilon_L^2}{2} \tag{13}$$

It is important to note here that specific free energy was obtained through a second-order Taylor series development $\ln(1+D) \approx D - \frac{D^2}{2}$ and can be written as follows:

$$\varphi_L = (1 - D - Tk(L)\ln(1+D))E\frac{\varepsilon_L^2}{2} \tag{14}$$

This specific free energy $\varphi_L = U - TS(L)$ was defined as function of a specific internal energy U and specific entropy S given, respectively, by Eqs. (15) and (16).

$$U = (1 - D)E\frac{\varepsilon_L^2}{2} \tag{15}$$

$$S(L) = k(L)\ln(1+D)E\frac{\varepsilon_L^2}{2} \tag{16}$$

Specific internal energy U is independent of L and internal energy is proportional to L; meanwhile specific entropy $S(L)$ depends on L and is size dependent. This hypothesis is based on physical principles. For discrete physical systems with weak correlation between microstates, entropy associated with a given macro-state is not necessary additive at small sizes. However, it becomes additive for large sizes. This means also that specific entropy increases as function of system size and tends asymptotically to a constant for large sizes (Tsallis 2009).

In Eq. (12), stress is the sum of tow components the first one is derived from the internal energy and given by Eq. (17).

$$\frac{\partial U}{\partial \varepsilon_L} = (1 - D)E\varepsilon_L \tag{17}$$

The second component is an entropic stress given by Eq. (18)

$$-T\frac{\partial S(L)}{\partial \varepsilon_L} = -T\kappa(L)\ln(1+D)\,E\varepsilon_L \tag{18}$$

Physicians state that entropic forces are long range (March's principle). This means that long-range non-local interactions should be defined in order to obtain macroscopically Eq. (18).

Figure 1 is a simple representation drawn to understand phenomenologically virus spread.

The key ideas to explain virus spread is that:

1. Without exchange of information between individuals, virus is spreading by transmission or diffusion due to close contact. This means that transmission is a local phenomenon. This means also that persons represented by nodes in Fig. 1 are not informed about what happens far from them.
2. Long range exchange of information and interaction between persons induce a non-local entropic force opposite to virus spread in analogy to mechanical entropic stress given by Eq. (18). Dissipation of spread is therefore a non-local phenomenon.

Table 1 presents, firstly, similarities between elastic wave propagation in a semi-infinite rod submitted to a constant power and free linear spread. Similarities are extended thereafter to shock wave propagation in a semi-infinite rod modelled as a non-local damage medium. Resolution of dynamics with non-local equations remains possible but complicated. The presented Equations are therefore phenomenological and macroscopic.

References

Bak P, Chen K, Tang C (1990) A forest fire model and some thoughts on turbulence. Phys Lett A 147:279–300

Ben Hassine W, Loukil M, Limam O (2019) A damage model predicting moderate temperature and size effects on concrete in compression. Comput Concr 23(5):321–327

Boccaletti S, Ditto W, Mindlin G, Atangana A (2020) Modeling and forecasting of epidemic spreading: the case of Covid-19 and beyond. Chaos Solitons Fract 135:109794

Carlioz T, Dormieux L, Lemarchand E (2019) Thermodynamics of crack nucleation. Contin Mech Thermodyn. https://doi.org/10.1007/s00161-020-00863-7

Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, Qiu Y, Wang J, Liu Y, Wei Y, Xia J, Yu T, Zhang X, Zhang L (2020) Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet 395:507–513. https://doi.org/10.1016/S0140-6736(20)30211-7

Fovargue D, Mitran S, Sankin G, Zhang Y, Zhong P (2018) An experimentally-calibrated damage mechanics model for stone fracture in shock wave lithotripsy. Int J Fract. https://doi.org/10.1007/s10704-018-0283-x

Gomez J, Prieto J, Leon E, Rodriguez A, (2020) A General agent-based model for transmission of infectious diseases: studying the COVID-19 propagation in Bogota, Colombia. medRxiv preprint https://doi.org/10.1101/2020.04.06.20056119

House T, Ross JV, Sirl D (2013) How big is an outbreak likely to be? Methods for epidemic final-size calculation. Proc R Soc A 469:20120436. https://doi.org/10.1098/rspa.2012.0436

Kachanov L (1958) Time of the rupture process under creep conditions. IzvestiiaAkademiiNauk SSSR, OtdelenieTeckhnicheskikhNauk 8:26–31

Keeling MJ, Eames KTD (2005) Networks and epidemic models. J R Soc Interfaces 2:295–307

Kermack WO, McKendrick AG (1927) A contribution to mathematical theory of epidemics. Proc R Soc A 115:700–721

Kim S, Seo YB, Jung E (2020) Prediction of COVID19 transmission dynamics using mathematical model considering behavior changes. Epidemiol Health. https://doi.org/10.4178/epih.e2020026

Koivu-Jolmaa M, Annilaa A (2018) Epidemic as a natural process. Math Biosci 299:97–102

Kolifarhood G, Aghaali M, Saadati HM, Taherpour N, Rahimi S, Izadi N, Saeed S, Nazari H (2020) Epidemiological and clinical aspects of COVID-19; a narrative review. Arch Acad Emerg Med 8(1):e41

Lemaître J, Chaboche JL (1978) Aspects phénoménologiques de la rupture par endommagement. J Méc Appl 2:317–365

Li Y, Wang B, Peng R, Zhou C, Zhan Y, Liu Z, Jiang X, Zhao B (2020) Mathematical modeling and epidemic prediction of COVID-19 and its significance to epidemic prevention and control measures. Ann Infect Dis Epidemiol 5(1):10521

Liang K (2020) Mathematical model of infection kinetics and its analysis for COVID-19, SARS and MER. Infect Genet Evol 82:104306. https://doi.org/10.1016/j.meegid.2020.104306

Limam O, Aidi M, Zenzri H (2014) Structural nominal concrete strength derived by statistical mechanics. Phys A 395:48–57

Liu J, Zhou J, Yao J, Zhang X, Li L, Xu X, He X, Wang B, Fu S, Niu T, Yan J, Shi Y, Ren X, Niu J, Zhu W, Li S, Luo B, Zhang K (2020) Impact of meteorological factors on the COVID-19 transmission: a multicity study in China. Sci Total Environ 726:138513

Lu J, Zhang X, Mai Y (2005) A preliminary study on damage wave in elastic-brittle materials. Int J Damage Mech 14:127–147. https://doi.org/10.1177/1056789505045934

Moll J, Kexel C, Pötzsch S, Rennoch M, Herrmann AS (2019) Temperature affected guided wave propagation in a composite plate complementing the Open Guided Waves Platform. Sci Data 6:191. https://doi.org/10.1038/s41597-019-0208-1

Nelms M, Rajendran AM, Hodo W, Mohan R (2017) Shock wave propagation in cementitious materials at micro/meso scales. AIP Conf Proc 1793:120009. https://doi.org/10.1063/1.4971691

Pijaudier-Cabot G, Bazant ZP (1987) Non local damage theory. J Eng Mech 113:1512–1533

Prata DN, Rodrigues W, Bermejo PH (2020) Temperature significantly changes COVID-19 transmission in (sub)tropical cities of Brazil. Sci Total Environ. https://doi.org/10.1016/j.scitotenv.2020.138862

Tsallis C (2009) Computational applications of nonextensive statistical mechanics. J Comput Appl Math 227:51–58

Tsallis C, Tirnakli U (2020) Predicting COVID-19 peaks around the world. MedRxiv preprint https://doi.org/10.1101/2020.04.24.20078154

Vayron R, Nguyen VH, Bosc R, Naili S, Haïat G (2015) Finite element simulation of ultrasonic wave propagation in a dental implant for biomechanical stability assessment. Biomech Model Mechanobiol. https://doi.org/10.1007/s10237-015-0651-7

Wang H, Tan G, Wang W, Liu Z (2018) Effect of temperature and spring-mass systems on modal properties of Timoshenko concrete beam. Struct Engg Mech 65:4. https://doi.org/10.12989/sem.2018.65.4.389

Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, Xiang J, Wang Y, Song B, Gu X, Guan L, Wei Y, Li H, Wu X, Xu J, Tu S, Zhang Y, Chen H, Cao B (2020) Clinical course and risk factors for mortality of adult in patients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet 395:1054–1062. https://doi.org/10.1016/S0140-6736(20)30566-3