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#stayhome to contain Covid-19: Neuro-SIR – Neurodynamical epidemic modeling of infection patterns in social networks

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ABSTRACT

An innovative neurodynamical model of epidemics in social networks – the Neuro-SIR – is introduced. Susceptible–Infected–Removed (SIR) epidemic processes are mechanistically modeled as analogous to the activity propagation in neuronal populations. The workings of infection transmission from individual to individual through a network of social contacts, is driven by the dynamics of the threshold mechanism of leaky integrate-and-fire neurons. Through this approach a dynamically evolving landscape of the susceptibility of a population to a disease is formed. In this context, epidemics with varying velocities and scales are triggered by a small fraction of infected individuals according to the configuration of various endogenous and exogenous factors representing the individuals' vulnerability, the infectiousness of a pathogen, the density of a contact network, and environmental conditions. Adjustments in the length of immunity (if any) after recovery, enable the modeling of the Susceptible–Infected–Recovered–Susceptible (SIRS) process of recurrent epidemics. Neuro-SIR by supporting an impressive level of heterogeneities in the description of a population, contagiousness of a disease, and external factors, allows a more insightful investigation of epidemic spreading in comparison with existing approaches. Through simulation experiments with Neuro-SIR, we demonstrate the effectiveness of the #stayhome strategy for containing Covid-19, and successfully validate the simulation results against the classical epidemiological theory. Neuro-SIR is applicable in designing and assessing prevention and control strategies for spreading diseases, as well as in predicting the evolution pattern of epidemics.

1. Introduction

At the time of writing this paper more than 5,332,908 people across the world had been infected by SARS-Cov-2, and more than 342,540 persons had died of Covid-19,[1] the disease caused by the new coronavirus. While the Covid-19 pandemic continues taking a heavy toll in human lives, wreaking havoc on businesses, and posing an unprecedented threat to public health and global economy, the need for gaining insights into the spreading dynamics of contagious diseases in social networks, is more important than ever before. This is because our world becomes increasingly interconnected, thus accelerating not only the global development, but also the transmission of infections from individual to individual and from country to country. While human history is rich in deadly pandemics, never before was the world so interrelated and interconnected as it is now, thus creating an entirely new context within which the dynamics of spreading diseases should be studied and modeled. The bubonic plague (Black Death), caused by *bacillus pestis*, a microorganism originating from black rats and transmitted to humans by fleas, was far deadlier than Covid-19, as it killed almost one third of Europe's population in the mid-14^{th} century. Nevertheless, at that time, the bubonic plague was spreading up through Europe at a speed of 320–640 km per year (McEvedy, 1988), whereas now, Covid-19 is sweeping the globe in a timescale of hours and days, thus revealing the tremendous impact of interconnectedness on the transmission velocity of communicable diseases. Meanwhile, other factors, such as age, general health condition, length of incubation period, infectiveness, strength of immune system, external conditions (e.g. weather, pollution, sanitary conditions), and length of immunity after recovery; significantly affect one's vulnerability to a contagious disease, thus having a strong bearing on the spreading patterns of infections. In this regard, the modeling and study of the diffusion patterns of epidemics, in relation to the underlying transmission network and the heterogeneities in the individuals' susceptibility to a contagious disease, is of paramount importance for designing prevention and control strategies, especially when there are no medicines and vaccines, as in the case of the Covid-19 outbreak.

In this study we expand on an idea for future research, mentioned in our previous work (Lymperopoulos, 2015; Lymperopoulos & Ioannou, 2016), regarding the potential of integrate-and-fire neural networks to capture the dynamics of epidemics. In our earlier work, we have

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[1] https://coronavirus.jhu.edu/map.html.

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shown that the integrate-and-fire mechanism and neural firing rate models, precisely reproduce activity patterns in social media, thus indicating that they are suitable for modeling contagion dynamics in social networks (Lymperopoulos, 2017; Lymperopoulos & Ioannou, 2015, 2016). By further elaborating on our proposal to use integrate-and-fire neural networks for epidemic modeling, we come up with the Neuro-SIR model, thus initiating a novel modeling framework for the dynamics of epidemics, based on a neural networks perspective.

Recent epidemiological studies on the transmission dynamics of SARS-CoV-2 and its impact on the evolution of Covid-19 outbreaks, outline several factors affecting the spreading process of the disease, caused by the new coronavirus. Social distancing and usage of personal protective equipment (PPE) (Chu et al., 2020; Leclerc et al., 2020); infection transmission through asymptomatic individuals (Huang et al., 2020; Nishiura et al., 2020; Sakurai, Sasaki, Kato, Hayashi, Tsuzuki, Ishihara, et al., 2020; Wang et al., 2020), various modes of transmission, even without contact (airborne) or through contaminated objects (Chan et al., 2020; Liu, Liao et al., 2020; Somsen, van Rijn, Kooij, Bem, & Bonn, 2020; Stadnytskyi, Bax, Bax, & Anfinrud, 2020); the incubation period of Covid-19 (Lauer et al., 2020; Yu, Zhu, Zhang, & Han, 2020); susceptibility and disease severity relative to age and health condition (Ludvigsson, 2020; Wu et al., 2020; Zheng et al., 2020); and exogenous factors, such as ventilation, crowding, and sanitation (Asadi, Bouvier, Wexler, & Ristenpart, 2020; Chia et al., 2020; La Rosa, Bonadonna, Lucentini, Kenmoe, & Suffredini, 2020; Leclerc et al., 2020; Lu et al., 2020; Morawska & Cao, 2020); determine the course of a Covid-19 epidemic wave. Consequently, a realistic modeling of the spreading dynamics of Covid-19, and of other infectious diseases, entails an approach, capable of accommodating the aforementioned factors into a mechanistic description of the dynamics of an epidemic. Neuro-SIR, is such a model. Based on the dynamics of leaky integrate-and-fire neurons, it features the mechanisms for the representation of the foregoing aspects of an epidemic process, through an isomorphism between the activity propagation in a neuronal population, and the spreading of a pathogen in a social network. As a result, the transmission of a pathogen in a network of individuals, is considered analogous to the transmission of action potentials through synaptic connections among neurons.

The infection mechanism is represented by the activation dynamics of leaky integrate-and-fire neurons, which is analogous to the development of a contagious disease, through multiple exposures to infected individuals, as described in recent epidemiological studies pertaining to the propagation of tuberculosis (Ackley et al., 2019; Lee, Proulx, Menzies, & Behr, 2016). The heterogeneous susceptibility to a disease is analogous to varying activation thresholds in leaky integrate-and-fire neurons, and the incubation period of a disease can be represented by the transmission delays of action potentials. Viral loads, social distancing, contact network, and usage of personal protective equipment, can be depicted on the configuration of the topological aspects, and connectivity strengths of the interaction network of leaky integrate-and-fire neurons. Exogenous factors affecting the spreading dynamics of a disease, are represented by external signals, supplied to a neural network. Viral transmission through asymptomatic infected individuals, can be represented by another layer of neural activity, connected to the layer of neurons representing susceptible individuals, who develop symptoms after the infection. Finally, the immunity period (if any) conferred by a disease, corresponds to the refractory period of leaky integrate-and-fire neurons following their activation.

The aforementioned adaptability and flexibility of Neuro-SIR, substantially exceeds the modeling capacity of existing state-of-the-art Susceptible–Infected–Recovered epidemic models. These models cannot accommodate the foregoing aspects in the transmission dynamics of pathogens, and the description of the infection mechanism. Also, they cannot support a high level of heterogeneity in a population of individuals, nor the impact of exogenous factors on the spreading dynamics of a disease. Present state-of-the-art epidemic models, are based on differential equations (three for the SIR model) describing the transition of individuals between the compartments of an epidemic process assuming a retransmission factor (R_0), a contact rate between infected and susceptible individuals, as well as a recovery rate for the infected population. The social network structure is incorporated into the differential equations of these models through the inclusion of the statistical properties of the degree distribution of various types of complex networks (e.g. scale-free, random, small-world) (Barthélemy, Barrat, Pastor-Satorras, & Vespignani, 2004, 2005; Boguná, Pastor-Satorras, & Vespignani, 2003; Dickison, Havlin, & Stanley, 2012; Kuperman & Abramson, 2001; May & Lloyd, 2001; Moreno, Pastor-Satorras, & Vespignani, 2002; Newman, 2002; Pastor-Satorras & Vespignani, 2001). While this approach has led to a tremendous progress in the study of epidemics with major developments in theoretical and practical fields, there exist limitations in the incorporation of several factors that affect the dynamics of epidemic spreading in a social network. As a result, a limited exploration of the evolution of epidemic processes can be performed within the existing modeling framework.

Recently, many models aiming at predicting the trend of Covid-19 infections in various countries, were based on SIR modeling approaches in order to estimate the retransmission factor of Covid-19, with a view to generating forecasts about the evolution of the total number of Covid-19 infections (Anastassopoulou, Russo, Tsakris, & Siettos, 2020; Fanelli & Piazza, 2020; Hamzah et al., 2020; Kuniya, 2020; Pandey, Chaudhary, Gupta, & Pal, 2020; Peng, Yang, Zhang, Zhuge, & Hong, 2020; Roda, Varughese, Han, & Li, 2020; Sun et al., 2020; Zhou, Ma et al., 2020). The limited capacity of existing SIR models to incorporate many of the aforementioned factors into the dynamics of an epidemic process, compromises the estimation of the retransmission factor of a disease, thereby leading to low precision in the forecasts of the cumulative number of Covid-19 cases.

Recent research (Huang et al., 2020; Nishiura et al., 2020; Sakurai et al., 2020; Wang et al., 2020) regarding the transmission of Covid-19, suggests that a considerable amount of symptomatic infections in a population of individuals, are caused through contacts with asymptomatic persons, who are infected with Covid-19 without developing symptoms. Asymptomatic individuals are mainly undetected by health care systems, since there are no signs calling for a medical examination that could lead to quarantine, and contact tracing. The only way to detect asymptomatic individuals and determine their proportion in a population is trough massive testing of a population for Covid-19. As a result, asymptomatic individuals continue interacting with other people throughout an epidemic wave, thus giving rise to new symptomatic infections in the part of the population, who develop symptoms when infected with Covid-19.

The incorporation of the aforementioned transmission mode into the Neuro-SIR modeling approach, can take place through the creation of another layer of neurons representing asymptomatic individuals. The layer of asymptomatic individuals is connected with a predefined density to the layer of the individuals, who can become symptomatic when infected. The layer of asymptomatic individuals creates spikes through a poison process, thus simulating the random contacts between individuals of the symptomatic layer and individuals of the asymptomatic layer. In this way the activity of the asymptomatic layer, which continues during the entire length of a simulation process, affects the evolution of the symptomatic development of an epidemic. Section 5.3 deals with a simulation experiment of this type, thus demonstrating how asymptomatic individuals can intensify the progression of an epidemic.

The modeling approach to epidemic spreading, introduced by Neuro-SIR, substantially widens the scope of epidemic spreading scenarios, which can be investigated through simulations, in comparison with existing epidemic models. Some of the enhancements brought in by Neuro-SIR are described below:

- Investigation of the impact of social distancing, usage of personal protective equipment, transmission of viral load and severity of illness, on the spreading of a disease, through the configuration of the statistical properties of the interaction network, and the adjustment of its link weights.
- Investigation of the impact of varying susceptibility levels among individuals in the progression of a contagious disease, through the configuration of the distribution of the infection thresholds of a simulated population of individuals, susceptible to a particular disease.
- Investigation of the impact of the length of the incubation period of a disease on the evolution of an epidemic, through the configuration of time delays in the transmission of an infection.
- Investigation of the impact of exogenous factors such as ventilation, crowding, weather, sanitary conditions, and pollution, on the spreading of a disease, through the configuration of external signals increasing or decreasing the individuals' susceptibility to a disease.
- Investigation of the impact of varying immunity periods on the spreading pattern of a disease, through the configuration of the refractory periods of a neuronal population representing individuals.
- Investigation of the impact of the asymptomatic transmission of a disease on the epidemic spreading pattern, through the configuration of an additional asymptomatic infection propagation layer interacting with the layer of the symptomatic progression of a disease.

The versatility of Neuro-SIR enables the simulation and examination of complex epidemic spreading scenarios, which cannot be investigated by existing SIR-based epidemic models. Neuro-SIR by functioning at the micro-level dynamics of a network dynamical system, that is, the dynamics at a node level, provides a high level of flexibility in setting up diverse values for the system parameters per individual, and per contact link. As a result, the variability of contexts, within which, the dynamics of epidemics are examined, is substantially enhanced. Being an adaptable and intuitive model, Neuro-SIR can serve as an overarching platform for an insightful exploration of an epidemic spreading process by substantially extending the repertoire of questions that can be addressed. As such, it can be a valuable tool for the development and assessment of strategies aiming at the prevention and containment of outbreaks of infectious diseases.

In the experimental part of this study we demonstrate the ability of Neuro-SIR to accurately reproduce well-studied and prevalent patterns of epidemic spreading. In this regard, we statistically verify the outstanding fit of standard epidemic evolution curves to the stylized facts of the corresponding patterns produced by Neuro-SIR. In doing so, we confirm that the proposed model complies with the classical epidemiological theory. As an introductory and validating usage of Neuro-SIR, we experimentally assess the effectiveness of the #stayhome policy to contain Covid-19. With the social isolation, quarantine, and restriction in movements being the only options for controlling the outbreak of Covid-19, since there are no medicines and vaccines, social distancing is a major strategy for enduring this unprecedented storm. To demonstrate the effectiveness of this policy, experiments are performed with Neuro-SIR using varying densities in the contact network of a synthetic population. The experimental results comply with the expectations, and show that as the connectivity of a network decreases, so does the rate of new infections and the speed of epidemic spreading. Reducing the density of the network below a certain value inhibits the penetration of a disease to a large part of a population. Furthermore, we illustrate the ability of Neuro-SIR to capture the dynamics of recurrent epidemics, where the recovery from a disease does not confer permanent immunity. In this regard, we visualize epidemic waves in a population, in relation to the individuals' dynamically evolving susceptibility to a disease. Through the simulation of recurrent epidemics, it is shown that Neuro-SIR can be easily extended to an SIRS (Susceptible–Infected–Recovered–Susceptible) epidemic model, and possibly to other multi-compartmental epidemics models through the proper transformations.

The rest of the paper is organized as follows. Section 2 reviews relevant literature and summarizes key findings and knowledge. Section 3 deals with the formulation and the description of the dynamics of Neuro-SIR. Section 4 explains the methods applied in the implementation of the model and the simulation experiments. Section 5 describes the results of the experimental work performed through Neuro-SIR, and validates the simulation outcomes against classical epidemiological theory. Section 6 discusses the modeling capacity of Neuro-SIR, and its potential for future research and practical use. Finally, Section 7 presents the concluding remarks of this study.

2. Related work

Epidemic modeling constitutes a well established scientific field, and a vast research area with a long history. Its beginnings date back at least in the late 1920's, when the physician Anderson McKendric published his seminal work on the mathematical theory of epidemics (Kermark & Mckendrick, 1927). An exhaustive review of this huge scientific topic is not only beyond the scope of this study, but is also infeasible even to merely mention hundreds of papers dealing with this topic. Nevertheless, in this section we discuss important findings from the relevant literature, with the aim of providing an extensive background for further exploration of this fertile research field. The basic concept of epidemic modeling is the classification of a population into discrete compartments depending on the individuals' state in relation to a disease. For a thorough account of the mathematical modeling of epidemics through the compartmental approach, we refer the reader to introductory material on the epidemiological theory (Anderson, Anderson, & May, 1992; Bailey et al., 1975; Hethcote, 2000; Kermark & Mckendrick, 1927; Murray, 2007). In the simplest versions of epidemic models, the population is divided into three classes, that is, Susceptibles (S), Infected (I), and Recovered (R) (or Removed). In the Susceptibles (S) class belong the members of a population, who are vulnerable to a particular contagious disease. The Infected (I) class contains the individuals who have contracted the disease, and therefore they can transmit it to Susceptibles. The Recovered (or Removed) (R) class comprises the individuals, who have been cured (or died) after contracting the disease. More complicated versions of epidemic models incorporate additional classes, such as Exposed (E), Quarantined (Q), Vaccinated (V), Isolated (W), or Dead (D) (Del Valle, Hethcote, Hyman, & Castillo-Chavez, 2005; Hethcote, 2000).

The main mathematical framework of the compartmental models is the construction of the differential equations describing the transition of individuals between states, according to specific rates. In early versions of compartmental epidemic models, the dynamics of spreading diseases was studied on the assumption of a fully mixed population, where an individual's probability of contacting another individual, was equal for all the members of a population. Recent models incorporate the notion of network into the dynamics, with a view to capturing the effect of the individuals' contact network on the evolution of an epidemic within a population. For instructive material on the adaptation of principal compartmental epidemic models (SI, SIS, SIR, SIRS) to a contact network structure, we refer the reader to representative work in this field (Barrat, Barthelemy, & Vespignani, 2008; Dorogovtsev, Goltsev, & Mendes, 2008; Newman, 2002, 2018; Vespignani, 2012). The investigation of epidemic spreading on networks has led to the theoretical development of this field with analytical mathematical expressions, and exact solutions to network-adapted epidemic models, thus facilitating the exploration of the role of various topological and transmission parameters, in the propagation of a disease in a population of networked individuals (Barthélemy et al., 2004, 2005; Boguná et al., 2003; Dickison et al., 2012; Kuperman & Abramson, 2001; May & Lloyd, 2001; Moreno et al., 2002; Newman, 2002; Pastor-Satorras &

Vespignani, 2001). The incorporation of the interaction network in these epidemic models is performed through the inclusion of the statistical properties (e.g. 1^{st} and 2^{nd} moment of the degree distribution, and mean-field approximation of the degree distribution) into the differential equations describing the transition dynamics between the states of a compartmental epidemic model. The exploration of the evolution of epidemics in relation to the topological properties of a social contact network, revealed crucial insights into the dynamics of contagious diseases. It has been shown that the propagation of epidemics in scale-free networks is characterized by critical behavior, such as the absence of epidemic threshold, that is, the spreading rate value, beyond which, a disease becomes prevalent (Boguná et al., 2003; Moreno et al., 2002; Pastor-Satorras & Vespignani, 2001). Also, the fast propagation of infections in heterogeneous complex networks (Barthélemy et al., 2004, 2005), characterize the spreading dynamics of contagions in social networks.

Another strand of research in epidemic modeling deals with the investigation of immunization techniques, which can inhibit the outbreak of diseases in a population of individuals (Cohen, Havlin, & Ben-Avraham, 2003; Ferguson et al., 2006; Pastor-Satorras & Vespignani, 2002; Schneider, Mihaljev, Havlin, & Herrmann, 2011). These studies explore methods and strategies towards the identification of the topological properties of individuals in a social network, who should be vaccinated, as well as the optimization of the size of the immunized population, with a view to preventing the widespread propagation of contagious diseases. The role of the individuals' mobility and transportation networks in the propagation of diseases, is another research topic attracting the attention of the research community dealing with the modeling of world-wide epidemics (Bajardi et al., 2011; Belik, Geisel, & Brockmann, 2011; Brockmann & Helbing, 2013; Colizza, Barrat, Barthelemy, Valleron, & Vespignani, 2007; Colizza, Barrat, Barthélemy, & Vespignani, 2006). This research area is of particular interest for identifying metapopulation mathematical models, suitable for epidemics spreading across countries through transportation links, as well as for assessing control policies aiming at preventing the propagation of diseases through mobility restrictions, and other transportation constrains. Another aspect investigated in epidemic modeling, is the role of the decreasing availability of health care resources, while an epidemic advances in a population, at rate faster than the ability of a health system to respond to the treatment needs of the infected individuals (Böttcher, Woolley-Meza, Araújo, Herrmann, & Helbing, 2015).

The unprecedented health and hygienic crisis that the world is currently faced with, due to the Covid-19 pandemic, evidences the need for further developing the modeling of epidemics. The prediction of disease propagation patterns, and the formation of proper prevention strategies, especially when medicines and vaccines are not available, represent a huge challenge for the scientific community. In this regard, it is anticipated that the epidemic modeling field will continue expanding at a fast pace, with a view to providing reliable tools for analyzing the spreading dynamics of infectious diseases, and assisting their prediction and control. To this end, this study aims at making a significant contribution by proposing a novel modeling approach, with significant potential for a more realistic modeling of epidemics.

3. Model

We describe the dynamics of a Susceptible–Infected–Removed (or Recovered) (SIR) epidemic process in a social network composed of N individuals, whose susceptibility to a disease obeys the dynamics of the membrane potential of leaky Integrate-and-Fire neurons. Susceptible *(S)* individuals can contract an infectious disease *D*, through contacts with infected individuals *(I)*. Removed *(R)* individuals have either recovered from the disease *D* with permanent immunity, or died due to an irreversible health damage caused by the infection. One's contacts with other individuals in the same population *P*, are represented by undirected edges C_{ij} in a graph *G*. An individual's contacts C_{ij} represent close socialization with family members, friends, and colleagues, but also chance encounters with other members of the population *P* representing passengers in transport means, clients in shops, restaurants, spectators in theaters etc.. In an extended conceptualization of the dynamics, the population *P* might comprise not only individuals, but also objects, shared among individuals of the population *P*. Such a context gives rise to a mixed interaction graph G featuring individual-to-individual and individual-to-object links, by means of which a pathogen *p* (e.g. virus, bacterium) causing a disease *D*, might be transmitted. In the present model formulation, we deal with a population *P* comprising exclusively individuals. Initially, at time t_0, a very small fraction N_I of the population *P*, is in the infected state *(I)*, that is, $N_I/P \ll 1$. At time t_0, the remaining part of the population N_S, is in the susceptible state *(S)*. As the disease propagates in the population *P*, susceptible *(S)* individuals become infected *(I)*, and infected individuals either recover or die, thus shifting to the removed *(R)* state, according to a process, described later in this analysis. The part of the population *P* comprising the Recovered and Removed individuals *(R)* is denoted by N_R. Each individual *i* in the population *P* receives $C_I(i)$ connections from infected individuals, $C_S(i)$ connections from susceptible individuals, and $C_R(i)$ from recovered individuals. The numbers $C_I(i)$, $C_S(i)$, $C_R(i)$ evolve over time as the disease spreading unfolds, however at any time *t*, $C_I(i)+C_S(i)+C_R(i) = C(i)$, which means that the number of each individuals' connections is fixed to a value from the degree distribution characterizing the density and topological properties of the graph *G*.

An individual's Susceptibility (S) to the disease D, evolves dynamically according to the mechanism regulating the membrane potential of a leaky Integrate-and-Fire neuron. In the absence of any contact with infected individuals, one's susceptibility remains at a resting value S_{rest}, which is analogous to the resting potential (V_{rest}) of a leaky Integrate-and-Fire neuron. A Susceptible *(S)* individual *i* gets infected through contacts with infected individuals, whereby each close interaction of with an infected *(I)* individual *j* transmits a pathogenic load to the contacted susceptible *(S)* individual *i*. As a result, at each time *t* a susceptible *(S)* individual *i* carries a dynamically evolving amount of infectious agents, obtained through *i's* contacts with infected individuals in the population *P*. We denote the pathogenic load of a susceptible individual *i* at time *t*, as $I_i^{net}(t)$. This quantity represents the pathogenic load accumulated in *i's* organism after contacting infected *(I)* individuals *j* in the population *P* through the $C_I(i)$ links of the graph *G*. The susceptibility $S_i(t)$ of the individual *i* evolves over time according to Eq. (1).

$$\tau \frac{dS_i(t)}{dt} = -(S_i(t) - S_{rest}) + I_i^{net}(t) \tag{1}$$

Eq. (1) is analogous to the depolarization mechanism of the membrane potential $V(t)$ of a leaky integrate-and-fire neuron (Brunel, 2000; Dayan & Abbott, 2001; Gerstner & Kistler, 2002; Rieke, Warland, Van Steveninck, Bialek, et al., 1999; Tuckwell, 1988). By dividing both sides of Eq. (1) with the time constant τ, we obtain two dynamically evolving terms. The quantity $-(S_i(t) - S_{rest})/\tau$ constitutes a drift term driving *i's* susceptibility to the resting value S_{rest}. The exponential relaxation of *i's* susceptibility to the resting value S_{rest} with time constant τ, represents *i's* immunity mechanism defying the invading infective agents, thereby protecting the susceptible individual *i* from developing the disease *D*. The gain term of the differential equation describing the evolution of *i's* susceptibility is the quantity $I_i^{net}(t)/\tau$, which represents the infection force increasing *i's* susceptibility to the disease *D*. As a result, the transition of an individual from the susceptible state *(S)* to the infected state *(I)*, is controlled by the leaky integration of the pathogenic load that a susceptible individual receives through contacts with infected individuals. When *i's* susceptibility $S_i(t)$ crosses a threshold value S_i^{Thres} while increasing, that is, when $S_i(t) = S_i^{Thres}$ and $dS_i(t)/dt > 0$, the individual *i* moves to the infected state *(I)*.

This means that the transition from the susceptible state *(S)* to the infected state *(I)* occurs through a threshold mechanism, whereby the leaky integration of the pathogenic load transmitted to the susceptible individual *i*, through contacts with infected individuals, leads to the development of a critical level of infection signifying the onset of the disease *D* in the individual *i*.

To enable a more immediate perception of the aforementioned $S \rightarrow I$ transition mechanism, in the experiments section, we visualize the susceptibility evolution pattern deriving from the epidemic spreading in a networked population. At this point, it should be emphasized that recent epidemiological studies suggest that a threshold mechanism, better describes the contagion dynamics of an infectious disease. In particular, case studies have shown that the infection risk of tuberculosis increases with multiple exposures to infected individuals following a sigmoidal pattern, whereby a sudden transition to a high infection risk occurs after a certain number of contacts with infected individuals (Ackley et al., 2019; Lee et al., 2016). Consequently, the Neuro-SIR model, through the threshold activation mechanism of leaky integrate-and-fire neurons, more precisely captures the contagion dynamics of communicable diseases, as it incorporates the epidemiological empirical evidence of the phase transition in the infection risk, as the number of contacts with infected individuals increases. For simplicity, in the formulation of the Neuro-SIR model we adopt a heavyside threshold function to regulate the transition from the Susceptible state *(S)* to the Infected state *(I)*. This function is described by Eq. (2).

$$\theta(i) = \begin{cases} 1, & transition\ to\ state\ I\ occurs \\ & when\ S_i(t) = S_i^{Thres}\ \ and\ \ dS_i(t)/dt > 0 \\ 0, & state\ \ S,\ \ while\ \ S_i(t) < S_i^{Thres} \end{cases} \tag{2}$$

When an individual *i* moves from the susceptible state *(S)* to the infected state *(I)*, he/she passes on a pathogenic load to his/her connections $C(i)$ in the graph *G*, thus increasing the susceptibility of the individuals *j* connected to the individual *i*. The Infected and Removed individuals *j* connected to *i*, are not affected by the transmission of pathogens from the individual *i*. In the formulation of the Neuro-SIR model, the transmission of pathogens is analogous to the propagation of an action potential from an activated neuron to its adjacent neurons through synapses. After the transmission of the pathogenic load to his/her connections, the infected individual *i* shifts to the Removed (R) state, according the following process. After getting infected, the individual *i* transmits pathogens to his/her connection and then, in a way similar to the onset of the refractory period in a neuron after its activation, he/she enters the Removed *(R)* state. Actually, the Removed state *(R)* includes the sub-states Quarantine, Recovery, or Death following an individual's infected *(I)* state. In other words, after transmitting pathogens to his/her contacts, an infected *(I)* individual for a certain period (defined later), no longer affects the dynamics of the disease propagation, since he/she either retreats to his/her home or a hospital to receive medical care (quarantine). Subsequently, if the treatment is successful, an infected individual recovers from the disease, otherwise he/she passes away. Both cases are incorporated into Neuro-SIR through the notion of the refractory period following the activation of a neuron.

Setting up an infinitely long refractory period, Neuro-SIR corresponds to the scenario in which an infected individual moves infinitely to the Removed *(R)* state, either through recovery from a disease with permanent immunity, or because of death. Obviously, the neural refractory period is the analog of the immunity period after recovery, hereafter denoted Im^{Period}. Consequently, an endless refractory period represents the Removed *(R)* state. In the experimental part of this study, we examine the behavior of Neuro-SIR with both endless and limited refractory periods. In the former case, Neuro-SIR simulates the dynamics of an Susceptible *(S)*–Infected *(I)*–Removed *(R)* (SIR) epidemic process, while in the latter it models the Susceptible *(S)*–Infected *(I)*–Recovered *(R)*–Susceptible *(S) (SIRS)* epidemic process, whereby the immunity after the recovery from a disease is not permanent, and therefore a Recovered individual becomes Susceptible again, thus giving rise to recurrent epidemics. To accommodate the entire state transition dynamics into the threshold mechanism of Neuro-SIR, we rewrite Eq. (2) as follows:

$$\theta(i) = \begin{cases} -1, & transition\ to\ state\ R\ after\ the\ infection, \\ & S_i(t) = S_{reset}\ while\ Im^{Period}\ is\ \text{“}on\text{"} \\ 1, & transition\ to\ state\ I\ occurs \\ & when\ S_i(t) = S_i^{Thres}\ \ and\ \ dS_i(t)/dt > 0 \\ 0, & state\ \ S,\ \ while\ \ S_i(t) < S_i^{Thres}\ \ and\ Im^{Period}\ is\ \text{“}off\text{"} \end{cases} \tag{3}$$

After describing the state transition mechanism of Neuro-SIR for endless and limited immunity periods (Im^{Period}), we continue the formulation of the model with the analysis of the pathogen transmission dynamics from infected *(I)* to susceptible *(S)* individuals. As mentioned earlier, the term $I_i^{net}(t)$ in Eq. (1) represents the cumulative pathogenic load transmitted to a susceptible *(S)* individual *i*, from infected *(I)* individuals *j*, connected to i according to the adjacency matrix of the graph *G*. $I_i^{net}(t)$ is a composite term, whose structure and dynamics are analyzed below.

Initially, we describe the dynamics of the case of permanent transition to the Removed *(R)* state, after the infection. In this situation, an infected individual *j* transmits pathogens to his/her connection only once, since after the infection the individual *j* moves permanently to the Removed *(R)* state. The pathogens have effect only on individuals who are in the susceptible state *(S)*. Removed *(R)* individuals are not affected by pathogens, as their susceptibility is locked in the S_{reset} value for an infinite period, in the case that we currently analyze. The amount of pathogens transmitted to a susceptible (S) individual *i* at time *t* through a contact with an infected *(I)* individual *j*, is equal to $W_{ij}^p * \delta(t - t_j - d_{tr})$. With W_{ij}^p we denote the strength of the pathogenic load transmitted through the contact between *i* and *j*. We assume that the pathogenic load is entirely conveyed with a single instantaneous contact, and therefore it has the form of a delta function concentrating all the amount of pathogens transmitted from *j* to *i*, at a single time point. To account for the incubation period of a pathogen *P*, that is, the time distance between the contact with an infected individual, and the potential development of symptoms of the disease *D* caused by pathogen *P*, we include a transmission delay d_{tr}. With t_j we denote the time point at which the individual *j* gets infected. It is presumed that the pathogenic load W_{ij}^p undergoes an exponential decay with time constant τ_p. This is due to the pathogen's not fully invading an individual's organism through a proper doorway (e.g. respiratory gates, eyes), and also due to the response of the immune system. As a result the cumulative pathogenic load $I_i^{net}(t)$ driving one's susceptibility to the threshold value S_i^{Thres}, evolves dynamically according to Eq. (4):

$$\tau_p \frac{dI_i^{net}}{dt} = -I_i^{net} + \tau_p \sum_{j=1}^{N} W_{ij}^p \delta(t - t_j - d_{tr}) \tag{4}$$

Eq. (4) describes the dynamics of pathogen transmission under the assumption of permanent transition to the Removed state *(R)* after the infection. In this case, as mentioned before an infected individual *j* transfers pathogens to his/her contacts only once. However, by setting up a limited immunity period Im^{Period}, an infected individual can get reinfected, thereby transmitting pathogens to his/her connections more than once. In such a case, Eq. (4) can be rewritten in the following form, where *M* is the number of times an individual *j* gets infected by a pathogen *p*.

$$\tau_p \frac{dI_i^{net}}{dt} = -I_i^{net} + \tau_p \sum_{j=1}^{N} W_{ij}^p \sum_{k=1}^{M} \delta(t - t_j^k - d_{tr}) \tag{5}$$

Table 1
Parameters of the Neuro-SIR model.

Parameter	Description
$S_i(t)$	Susceptibility of the individual i at time t. It is analogous to the membrane potential value of a leaky integrate-and-fire neuron
S_{rest}	Resting value of the susceptibility. It is analogous to the resting potential of the membrane of a leaky integrate-and-fire neuron
S_{reset}	Reset value at which the susceptibility remains clamped during the immunity period
$I_i^{net}(t)$	Momentary value of the pathogenic load accumulated on an individual i, through contacts with infected individuals j. It is analogous to the momentary value of the presynaptic current received by a leaky integrate-and-fire neuron
τ	Time constant of the susceptibility evolution. It is analogous to the time constant of the membrane potential evolution in leaky integrate-and-fire neurons
S_i^{Thres}	Threshold value of the susceptibility of the individual i, for the transition to the infected state. It is analogous to the firing threshold of integrate-and-fire neurons
Im^{Period}	Length of the immunity period conferred through recovery from a disease. It is analogous to the refractory period of a leaky integrate-and-fire neuron
W_{ij}^{p}	Pathogenic load transferred from the infected individual j to the individual i through their connection. It is analogous to the synaptic strength in interconnected leaky integrate-and-fire neurons
d_{tr}	Transmission delay of the pathogen representing the incubation period of an infection. It is analogous to the transmission of action potentials in a network of leaky integrate-and-fire neurons
τ_p	Time constant of the decay of the pathogenic load. It is analogous to the time constant of the decay of the action potentials generated by leaky integrate-and-fire neurons
$F_i^{ext}(t)$	Strength of external factors influencing an individuals' susceptibility to a disease. It is analogous to external signals received by sensory neurons
G	The graph consisting of connections C_{ij} representing contacts between individuals in a population P

Eqs. (4) and (5) are analogous to the equations describing the dynamics of the presynaptic current that a leaky integrate-and-fire neuron receives from activated neighboring neurons. Eq. (1) in conjunction with the state transition mechanism (3), and Eq. (4) or Eq. (5) depending on the length of the immunity period, form the Neuro-SIR epidemic model that we are going to demonstrate through experiments, pertinent to various contexts in the propagation of an epidemic in a social network.

As we mentioned in the introduction, Neuro-SIR has the capacity to capture the impact of external factors on the dynamics of epidemic spreading in a social network. Factors, such as weather, pollution, sanitary conditions can significantly affect one's susceptibility to a disease, and therefore they influence the epidemic spreading patterns. To take into consideration, the effect of the external environment on the propagation dynamics of an infectious disease, we incorporate an additional term into the differential equation (1). This term is denoted as $F_i^{ext}(t)$, and functions as an extra control parameter of an individual's susceptibility dynamics. A positive value in the $F_i^{ext}(t)$ increases *i's* susceptibility, while a negative values has the opposite effect. After including the term $F_i^{ext}(t)$ in Eq. (1), we obtain Eq. (6). Eq. (6) is analogous to the equation describing the subthreshold dynamics of the membrane potential of sensory neurons, which apart from presynaptic current, they also receive signals from external sources. As a result, Eq. (6) combined with the state transition mechanism (3), and Eq. (4) or Eq. (5) according to the preferred immunity period length, frame the adaptation of the Neuro-SIR model to an epidemic spreading process with external influencing factors. In the experimental part of this study, we illustrate the behavior of Neuro-SIR in such a context.

$$\tau \frac{dS_i(t)}{dt} = -(S_i(t) - S_{rest}) + I_i^{net}(t) + F_i^{ext}(t) \tag{6}$$

Table 1, summarizes the parameters of Neuro-SIR.

4. Methods

The implementation of Neuro-SIR has been carried out by setting up a network dynamical system, where each node represents an individual from the population P. The dynamics of each node, that is, the evolution of the susceptibility of each individual, and the transition between the states of the model, is regulated by the differential equations and the state switching mechanism, presented in the previous section. As a result, in a network with N nodes, we have a network dynamical system featuring: (a) A set of N differential equations with the form of Eq. (1), or Eq. (6) in the case of adding external factors influencing the epidemic spreading. (b) A set of N coupled differential equations with the form of Eq. (4) or Eq. (5) depending on the length of the immunity period (permanent or limited). These equations control the evolution of the susceptibility of each node representing an individual in the population P, within which an epidemic is spreading. The network dynamical system is solved numerically and monitored for predefined lengths of time depending on the duration of the epidemic spreading simulation. The numerical integration of the differential equations of the model is carried out through an exact clock-driven integration, with a time step of *0.1* ms. The connection matrix of the networked individuals is constructed as an Erdős–Rényi undirected random graph (Erdős & Rényi, 1960), with a predefined connection probability p_c among nodes. We consider that a random graph, better captures the topological properties of connections between individuals in an environment where contacts among individuals occur in families, work places, café, restaurants, clubs, athletic events, theaters, shops, public transport means, gyms, sidewalks, etc.. Nevertheless, any other topological structure of complex networks (Boccaletti, Latora, Moreno, Chavez, & Hwang, 2006), such as scale-free, or small-world could be incorporated into the dynamics for analyzing the behavior of the model. Using an Erdős–Rényi random graph as the contact network of the N individuals of the population P, the number of undirected connections C_{ij} among individuals, is given by formula (7).

$$C = p_c \frac{N \cdot (N-1)}{2} \tag{7}$$

Considering that the total number of undirected links in a random graph is equal to $N \cdot (N-1)/2$, the density d_G of the network population P is given by the formula (8).

$$d_G = \frac{C}{N \cdot (N-1)/2} \tag{8}$$

Substituting formula (7) for C in formula (8), yields $d_G = p_c$, that is, the density of the connection matrix of the networked population P, is equal to the connection probability p_c. In the experimental section of the study, we perform several simulations of the epidemic spreading using various densities, with a view to demonstrating that the #stayhome policy for containing Covid-19 is indeed a highly effective strategy for

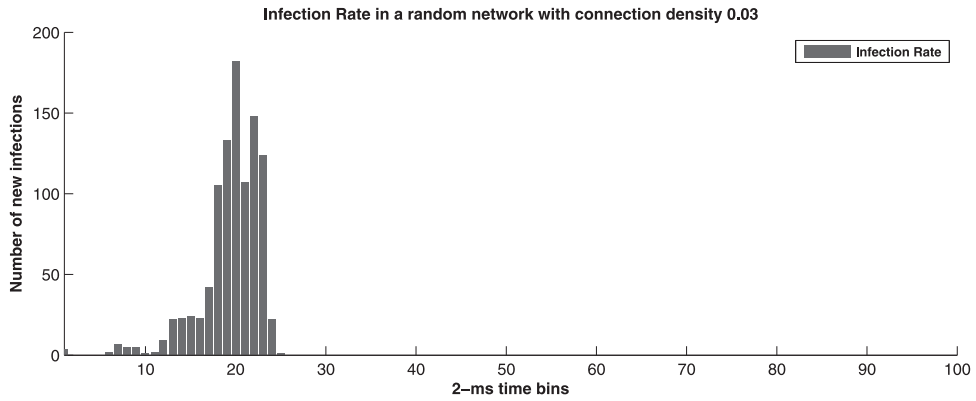


Fig. 1. Infection rate in a random network with density 0.03.

Table 2
Parameters values in experiment 1.

Parameter	Value
S_{rest}	−52
S_{reset}	−70
τ	15 ms
S_i^{Thres}	Threshold values assigned to individuals from a normal distribution with mean = −50 and std = 0.6
Im^{Period}	2900 ms
W_{ij}^{p}	5
d_{tr}	9 ms
τ_p	5 ms
G	Random graph with 1000 nodes
d_G	Network density — case 1: 0.03, case 2: 0.015, case 3: 0.01, case 4: 0.00825

slowing down the infection rate, and preventing the spreading of the disease. In a random graph, the average degree of each node is equal to $p_c \cdot (N-1)$, and the probability of each node having a degree k is given by formula (9)

$$P(k) = e^{-\langle k \rangle} \frac{\langle k \rangle^k}{k!} \tag{9}$$

The degrees of the nodes in a random graph come from a Poisson distribution. When the average degree $\langle k \rangle$ is less than *1* the network consists of small subgraphs. When $\langle k \rangle > 1$, the network features a giant component and small subgraphs. An Erdős–Rényi random graph is characterized by small clustering and the small world effect.

Neuro-SIR is able to incorporate a high level of heterogeneity into the dynamics of epidemic spreading in a social network. In this regard, it is possible to set up varying values among the individuals of a population for a large number of parameters. For instance, experiments can be performed using heterogeneous values in the configuration of the population for the susceptibility resting value S_{rest}, the time constant of the susceptibility evolution τ; the susceptibility threshold value S_i^{Thres} for the transition from the Susceptible *(S)* to the Infected *(I)* state; the length of the immunity period Im^{Period}, either globally or per individual; the pathogenic load W_{ij}^{p} transmitted from an infected to a susceptible individual; the transmission delay d_{tr} of the pathogenic load per link, and even then the strength of external factors influencing the spread of a disease. Consequently, Neuro-SIR has a tremendous capacity in simulating highly diverse epidemic spreading scenarios, thus providing insights into the dynamics of the propagation of a disease in a highly flexible way surpassing existing methods. Considering the large parameter space of the Neuro-SIR network dynamical system, we proceeded to our experiments using a limited level of heterogeneity among the individuals of the synthetic population for two reasons. First, because we aim to reproduce epidemic spreading patterns, generated by existing well-studied models, with long history in the epidemiology. Such models function at the macroscopic dynamics of epidemic spreading in a population, and therefore they are not configurable at a node level, in way which can be compared with Neuro-SIR. By reproducing well-known epidemic spreading patterns through Neuro-SIR, we verify and validate its compatibility with the existing epidemiological theory. Second, the exploration of the behavior of Neuro-SIR in various contexts of heterogeneities among individuals, requires detailed epidemiological data per individual, and formulation of realistic epidemic spreading scenarios. Such an endeavor, is beyond the scope of this study, which aims at introducing a new epidemiological model.

In view of the foregoing considerations, we performed our experiments using heterogeneous values in the susceptibility threshold of the individuals in the synthetic networked population, as well as in the number of the individuals' connections, which follow a Poisson distribution, as mentioned earlier. The values of the susceptibility thresholds assigned to the individuals of the population, are obtained from a normal distribution with a standard deviation properly configured, so that a very small number of individuals have a susceptibility threshold S_i^{Thres}, slightly below the susceptibility resting value S_{rest}. As a result, these individuals shift to the infected state *(I)* immediately with the start of the simulation, thus triggering the epidemic spreading process. Using heterogeneous values in the susceptibility threshold S_i^{Thres}, we also incorporate into the dynamics of the epidemic spreading, the variability in the sensitivity to an infection in a population due to factors, such as age, health condition, genetic predisposition. During the simulation of the epidemic spreading in a social network, we monitor the evolution of the susceptibility of the population, and record the values as they change per *0.1* ms (integration time step). We also record the *node-id*, and the time point at which a susceptible individual gets infected. Furthermore, we record the infection rate per *2* ms, that is, the number of individuals who get infected per *2* ms. After the end of each simulation experiment, we process the aforementioned recorded information, with a view to producing figures illustrating various patterns of the epidemic spreading process.

5. Results

5.1. Simulation experiment 1 – SIR epidemic process and #stayhome strategy simulation

The first experiment deals with the simulation of a Susceptible → Infected → Removed epidemic spreading process in a population of *1000* individuals, who are connected in a random network. The experiment examines the infection rate, the speed of epidemic spreading, and the evolution of the total number of individuals who got infected, in relation to the density of the individuals' interaction network. We are going to demonstrate, that the Neuro-SIR model verifies the efficiency of the #stayhome strategy, which was employed as the only weapon

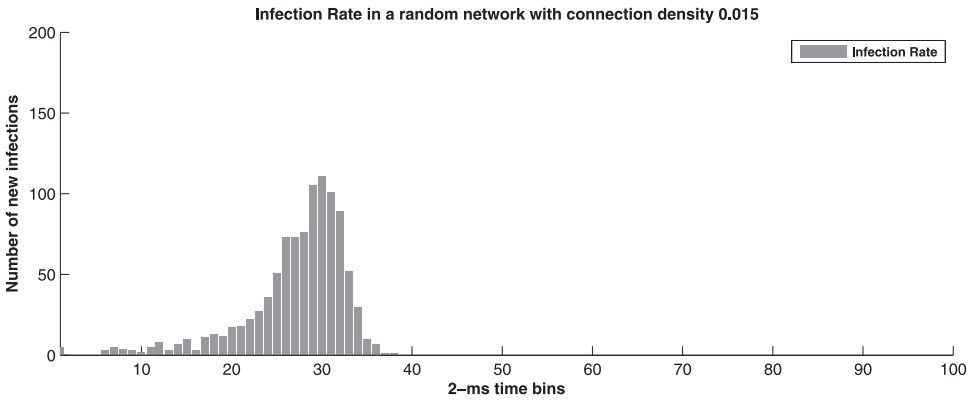


Fig. 2. Infection rate in a random network with density 0.015.

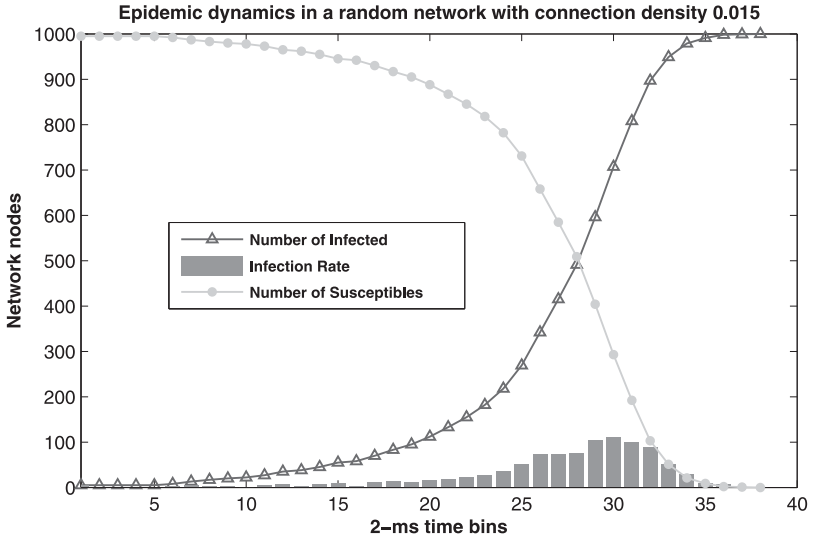


Fig. 3. Infection rate and evolution of the cumulative number of infected and susceptible individuals in a random network with density 0.015.

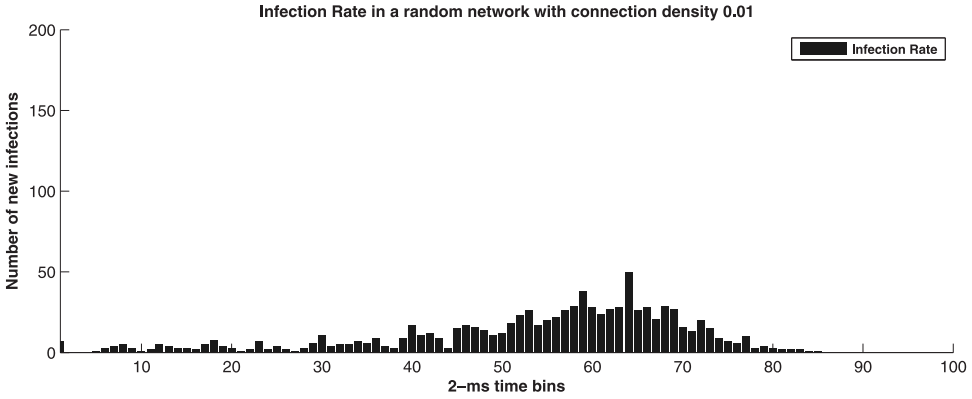


Fig. 4. Infection rate in a random network with density 0.01.

against the spreading of the Covid-19 disease. As mentioned in the Methods section the susceptibility threshold values in the population of the *1000* individuals are obtained from a normal distribution. All the details about the parameter values used in experiment 1 are listed in Table 2. In what follows we describe the epidemic dynamics using as control parameter the density of the social network. Results are presented for network densities d_G equal to *0.03*, *0.015*, *0.01*, and *0.00825*. The immunity period Im^{Period} is set to *2900* ms, a value substantially higher than the length of the experiment which is set to *200* ms. With this configuration the model simulates an Susceptible–Infected–Removed epidemic process. Fig. 1 illustrates the infection rate per *2* ms in the population of *1000* individuals, who are connected in random network with density $d_G = 0.03$. The mean number of the individuals' contacts is $d_G \cdot (N - 1)$, that is, approximately *30*. As mentioned in the Methods section the individuals' number of connections follows a Poisson distribution, and therefore the mean degree although indicative of the individuals' connectivity, does not constitute a homogeneous degree value for all the individuals of the synthetic population. It can

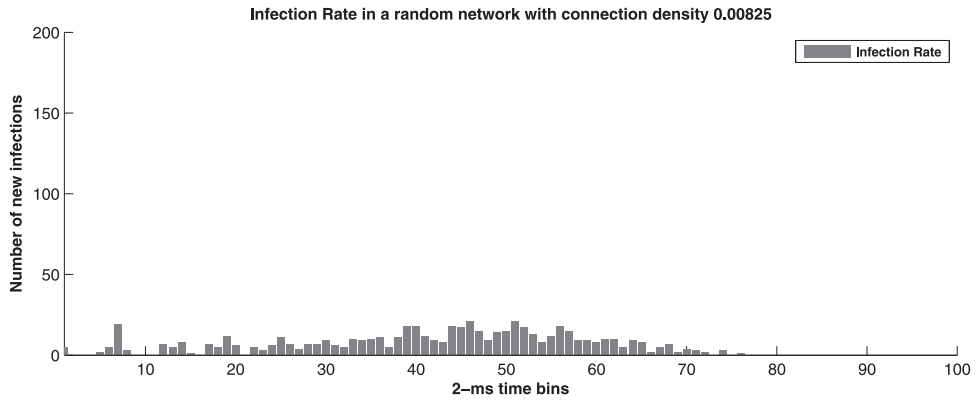


Fig. 5. Infection rate in a random network with density 0.00825.

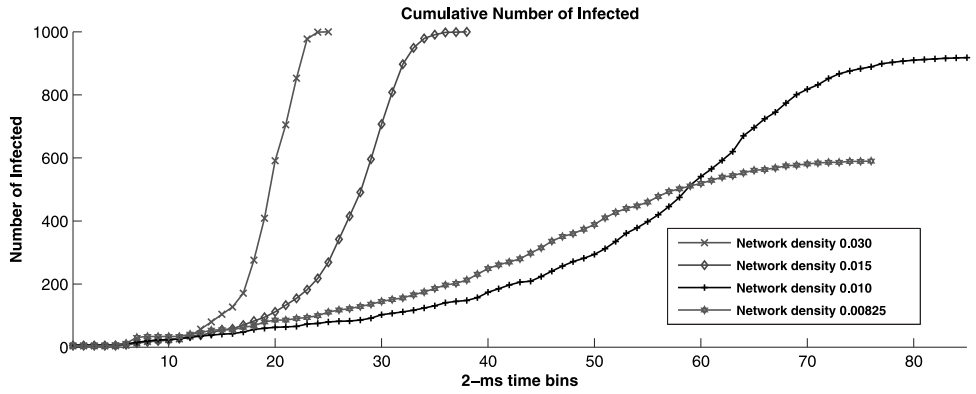


Fig. 6. Comparison of the cumulative growth of infected individuals for various network densities.

be seen in Fig. 1 that the infection rate exhibits a sharp increase, and the duration of the epidemic is short, as it ends very early in relation to the *200* ms of the duration of the experiment.

Fig. 2 shows the infection rate with the network density of the interconnected individuals set to a lower value. In this simulation run d_G is equal to *0.015*, and therefore the mean of the individuals' degree distribution is approximately *15*. In comparison with the infection rate of the previous case ($d_G = 0.03$), a significant decrease is noticed. The maximum value of the infection rate is substantially lower and the epidemic lasts longer. In Fig. 3 we visualize two additional epidemic spreading patterns along with the infection rate. In particular, Fig. 3 shows the evolution of the cumulative number of individuals who got infected during the epidemic, as well as the evolution of the total number of susceptible individuals. It can be seen that at the end of the epidemic outbreak, the disease had penetrated the entire network, and therefore all the individuals got infected at some point during the disease spreading process. As a result, the evolution of the total number of susceptibles in the population follows an inverse pattern in relation to the cumulative number of the individuals who got infected.

The sigmoidal pattern of the cumulative growth of the number of infected individuals, and the inverse pattern describing the evolution of the total number of susceptibles, will be further analyzed later. As a matter of fact, the epidemic patterns illustrated in Fig. 3 constitute telltale signs of the compatibility of the Neuro-SIR dynamics with the classic epidemiological theory, which relies on sigmoidal functions for the description of the evolution of the total number of infected individuals during an epidemic.

The effective control of the infection rate through the reduction of the network density, is more pronounced in Fig. 4 depicting the propagation of the epidemic in a network with density d_G equal to *0.01*. In this network, the mean of the individuals' degree distribution is approximately *10*. It can be observed, that the infection rate is substantially smoother and the epidemic lasts significantly longer in comparison with the previous cases. Such an outcome is the goal of the #stayhome campaign for the containment of Covid-19.

By decreasing the density of a social network, the infection rate becomes less bursty and the epidemic progresses at a lower speed. These effects yield valuable societal benefits, as they allow health systems to respond more effectively to the requirements for medical care with an immediate impact on the reduction of the death toll caused by an infectious and dangerous disease like Covid-19. Fig. 5 illustrates the infection rate in a random network with density d_G equal to *0.00825*, which corresponds to an approximate mean value of *8* in the individuals' degree distribution. Although the density of the network is close to the previous value ($d_G = 0.01$), the infection rate is substantially lower and smoother in comparison with the previous case, thus evidencing the existence of a critical value in the density of the network, below which the intensity of the epidemic outbreak changes suddenly. This is also obvious in Fig. 6 depicting the evolution of the total number of individuals, who got infected in the aforementioned cases of network densities.

In Fig. 6 it can be observed that as the network density decreases so does the slope of the sigmoidal curve describing the evolution of the cumulative number of infected individuals. For network densities equal to *0.03* and *0.015*, the infection spreads to the entire population. However, in the case of $d_G = 0.015$ the spreading phase takes longer.

The difference in the patterns of the epidemic outbreak is sharper, when we compare the penetration of the infection in the population in random networks with densities *0.01* and *0.00825*. Although the network density values are close, there is a substantial difference in the total number of individuals who got infected, as well as in the slope of the cumulative growth of the infected population. With a network density of *0.01* the disease penetrates the largest part of the population, however with a network density of *0.00825*, hardly a *60%* of the

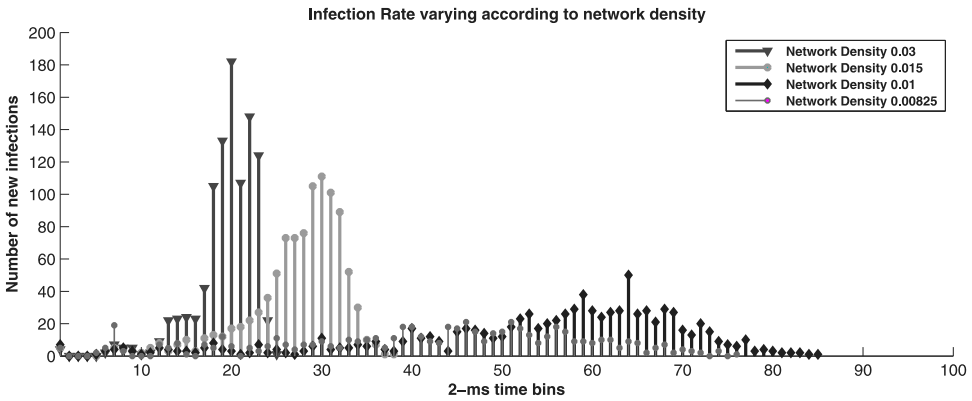


Fig. 7. Comparison of infection rates for various interaction network densities.

population is infected until the end of the epidemic spreading which terminates noticeably earlier than in the case of a network with density $d_G = 0.01$. This observation is very important when it comes to implementing "lockdown" strategies for the containment of an infectious disease. The evidence obtained from the simulations that we discuss, suggests that there is no much tolerance in the network densities values separating the containment of a disease from the widespread infection of a population.

Fig. 7 illustrates a comparative chart of the infection rates in relation to the network density. This diagram summarizes the previous discussion on the significant effect of the interaction network density on the intensity and the duration of epidemics in social networks. The denser a contact network is, the higher the infection rate will be. As a result, "lockdown" policies constitute effective reaction measures against the spreading of infections, especially when no medicines or vaccines are available. It is also noticeable, that the duration of an epidemic is inversely proportional to the density of the interaction network. Nevertheless, while the short duration of an epidemic might sound beneficial, one has to consider the limited capacity of health systems to adequately respond to the influx of infected individuals in need of intense and long-lasting medical care, thus unavoidably leading to increased death rates.

Fig. 8 provides an additional form of visualizing the epidemic spreading patterns for the network densities discussed so far. Each dot in Fig. 8 represents the time point at which an individual from the synthetic population got infected. With higher network densities the epidemic spreading looks like a short and forceful wave. On the contrary, when the network density decreases, the epidemic wave spreads further in time, and features a smoother form without concentrations.

5.2. Validation

Classic epidemiological theory (Anderson et al., 1992; Bailey et al., 1975; Kermark & Mckendrick, 1927) uses the logistic growth equation to describe the evolution of the total number of the individuals, who get infected from a disease during an epidemic. The logistic growth equation is generally suitable for the mathematical modeling of various self-limiting process, such as the population growth in relation to the carrying capacity of an environment, the diffusion of innovations, and the tumor expansion among others (Murray, 2007). The adaptation of the logistic growth equation to the description of the evolution of the cumulative number of distinct infected individuals in a population of individuals, takes the form of Eq. (10).

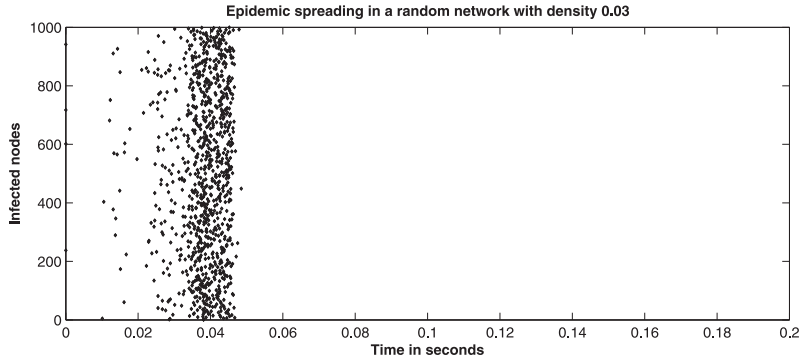
$$x(t) = x_0 \frac{e^{\beta t}}{1 - x_0 + x_0 e^{\beta t}} \tag{10}$$

In Eq. (10), x_0 represents the initial fraction of the infected population ($x_0 \ll 1$), from whom a disease starts spreading in the remaining population of susceptible individuals. The parameter β represents the probability of a susceptible individual getting infected after contacting an infected one in a fully mixed population. In other words, β is the transmission parameter – analogous to R_0, mentioned worldwide in the daily news about the spread of Covid-19. In order to validate the compliance of Neuro-SIR with the established epidemiological theory, we fitted the Eq. (10) to the cumulative growth pattern of the individuals, who got infected in a synthetic networked population, through the simulation of an epidemic process using Neuro-SIR. Fig. 9 shows the excellent goodness-of-fit of Eq. (10) to the cumulative growth pattern of infected individuals in a synthetic contact network with density *0.015*. To enable the fitting process of Eq. (10), we normalized the total number of infected individuals to *1*. It can be seen that the *R-square* was estimated at *0.9906*, and the RMSE at *0.03689*, thus confirming the precise fit of Eq. (10) to the Neuro-SIR results.

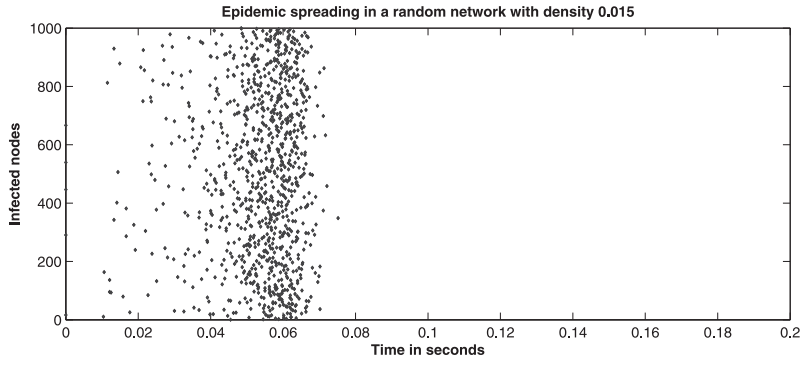
5.3. Simulation experiment 2 – Examining the role of asymptomatic transmission in the progression of a contagious disease

In this simulation experiment we are going to demonstrate how the Neuro-SIR model can be adapted to an epidemic spreading scenario, where asymptomatic transmission of an infection, progresses in parallel with the transmission of a contagious disease through contacts between symptomatic and susceptible individuals. To illustrate the effects of the asymptomatic transmission of a disease to the evolution of an epidemic, we are going to use as reference case, the simulation results (Figs. 5, 6, 7, 8(d)) pertaining to the development of an epidemic in a network of 1,000 susceptible individuals connected through a random network with density 0.00825.

To implement the aforementioned scenario we created another layer of 100 neurons representing asymptomatic individuals. The choice of the 1/10 proportion in the size of the population who do not develop symptoms when infected, is based on recent findings suggesting a 6% to 41% of asymptomatic cases of Covid-19 in relation to the number of confirmed cases (Davies et al., 2020). Each individual in the asymptomatic layer randomly generates contact events with individuals from the symptomatic layer, through a Poison process with a frequency of 10 Hz. The density of the connections between the asymptomatic and symptomatic layers is equal to 0.01. This means that each asymptomatic individual is on average connected to 10 individuals in the symptomatic layer. Therefore, each time a contact event is triggered by the Poison process for an asymptomatic individual, then this individual transmits a viral load to 10 individuals on average in the symptomatic layer. The viral load transmitted by asymptomatic individuals is configured to a lower value (=2.7), in relation to the viral load transmitted by symptomatic infected individuals (=5). This difference is an approximation of recent epidemiological findings suggesting that the SARS-CoV-2 viral load transmitted by asymptomatic or milder cases of Covid-19 patients, is lower than the viral load transmitted by



(a)



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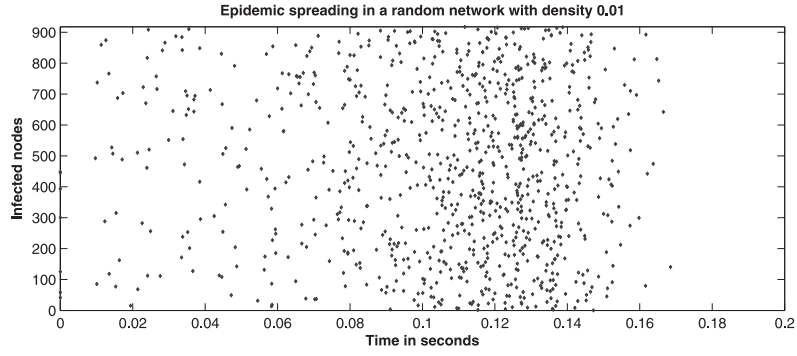

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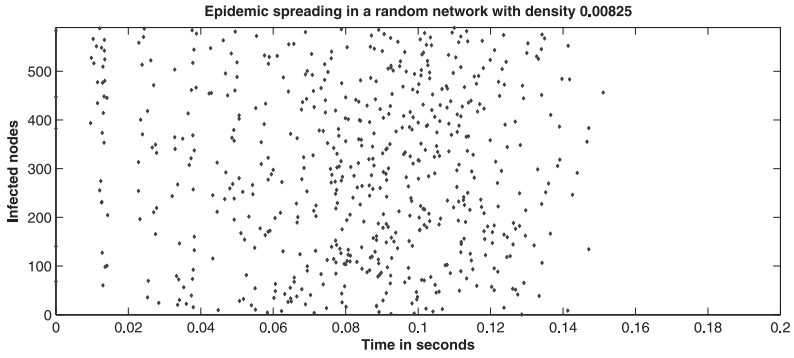

(d)

Fig. 8. Visualization of the epidemic spreading per individual and network density. Each dot represents the time point at which an individual of the networked population got infected. (a) Epidemic spreading with network density *0.03*, (b) Epidemic spreading with network density *0.015*, (c) Epidemic spreading with network density *0.01*, (d) Epidemic spreading with network density *0.00825*.

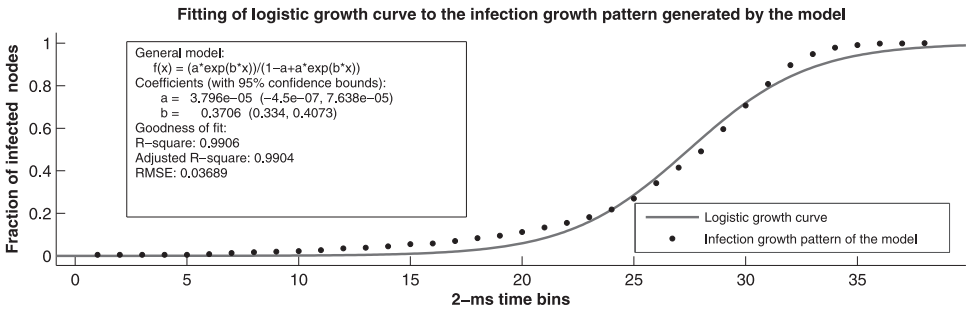

Fig. 9. The precise fit of Eq. (10) to the Neuro-SIR cumulative growth pattern of infected individuals in a synthetic contact network with density *0.015*.

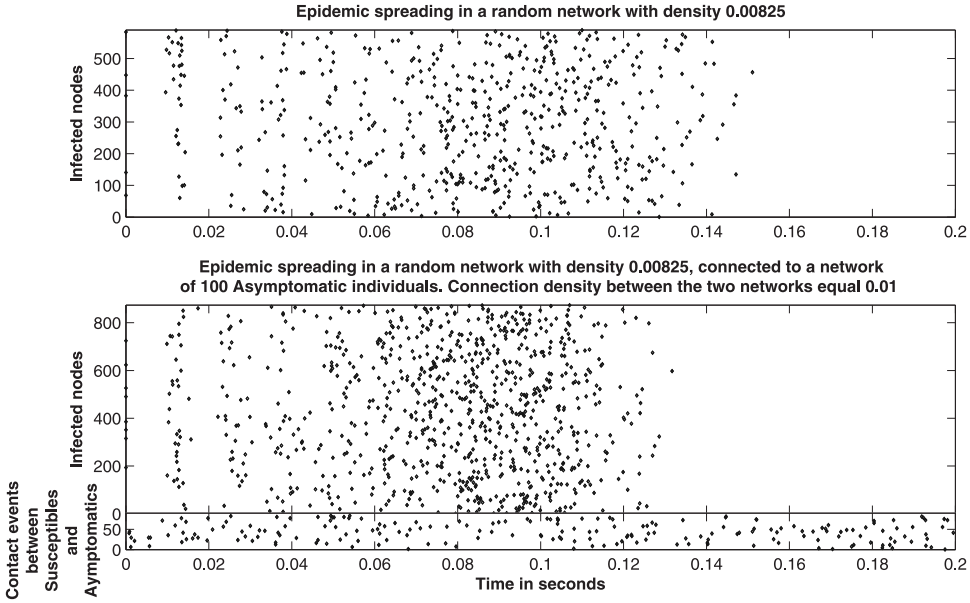

Fig. 10. The evolution of infection events in the symptomatic layer, with (middle) and without (upper) asymptomatic transmission. Contact events from asymptomatic individuals are illustrated in the lower part of the figure.

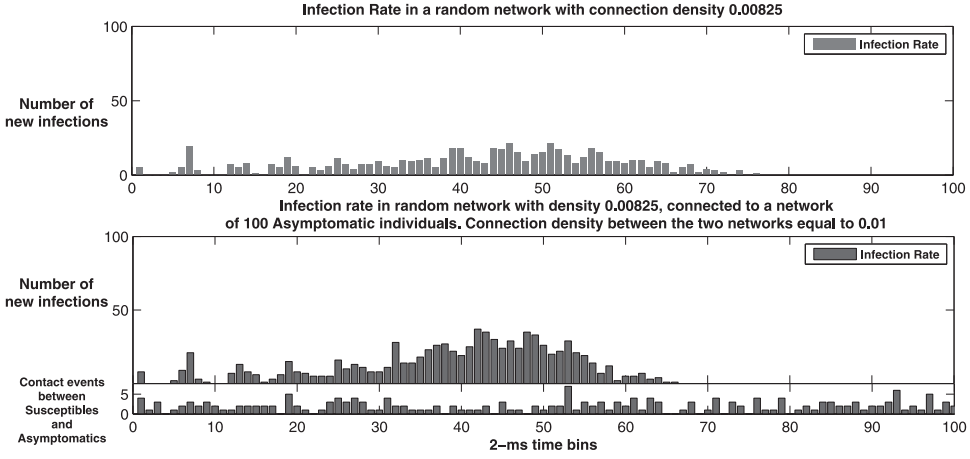

Fig. 11. The infection rate in the layer of susceptibles who can develop symptoms, with (middle) and without (upper) asymptomatic transmission. Rate of contact events from asymptomatic individuals are illustrated in the lower part of the figure.

severe cases of Covid-19 (Ludvigsson, 2020; Wu et al., 2020; Zheng et al., 2020).

Fig. 10, in the middle, shows the infection events in the layer of susceptible individuals, who become symptomatic when infected, in relation to the contact events with asymptomatic individuals, illustrated in the lower part of Fig. 10. The upper part of Fig. 10 shows the evolution of the infection events without asymptomatic transmission. It can be seen that when asymptomatic transmission takes place, the infection events increase and occur in a shorter time interval. The contact events between susceptible and asymptomatic individuals, after a time point do not give rise to new infection events, as the remaining susceptibles have a higher infection threshold, which cannot be crossed

Table 3
Parameters values in experiment 2.

Parameter	Value
S_{rest}	−52
S_{reset}	−70
τ	15 ms
S_i^{Thres}	Threshold values assigned to individuals from a normal distribution with mean = −50 and std = 0.6
Im^{Period}	2900 ms
W_{ij}^{p}	5
d_{tr}	9 ms
τ_p	5 ms
G	Random graph with 1000 nodes
d_G	Network density: 0.00825
Poison Group PG	100 neuron representing asymptomatic infected individuals
Contact rate between PG and G layers	10 Hz (10 contacts per second per asymptomatic individual)
W_{PG}^{G} (connection weight between PG and G)	2.7
d_{PG}^{G} (connection density between PG and G)	0.01

through the accumulation of the relatively low viral load transmitted by asymptomatic individuals.

Fig. 11, in the middle, illustrates the infection rate in the layer of susceptibles who can develop symptoms, in the presence of interaction with asymptomatics, as well as without asymptomatic transmission, in the upper part of the figure. The lower part of the figure illustrates the contact rate between individuals of the symptomatic and asymptomatic layers. As expected, the existence of asymptomatic transmission increases the infection rate in the layer of susceptible-symptomatic individuals, and shortens the epidemic wave, thus making it more intense.

Fig. 12, illustrates the development of the total number of infected individuals in the susceptible-symptomatic layer with and without asymptomatic transmission. It can be seen that the asymptomatic transmission substantially increases the total number of symptomatic infections. In the simulation experiment, 874 individuals were infected in the susceptible-symptomatic layer in the presence of asymptomatic transmission, while 590 were infected in the case without asymptomatic transmission. The simulation results comply with recent epidemiological findings (Aguilar, Faust, Westafer, & Gutierrez, 2020; Huang et al., 2020) about Covid-19 suggesting that the asymptomatic transmission may significantly add to the growth of the Covid-19 pandemic. The parameter values used in this experiment are provided in Table 3.

5.4. Simulation experiment 3 – SIRS epidemic process

In this experiment, we demonstrate the capacity of Neuro-SIR to function as a Susceptible → Infected → Recovered → Susceptible *(SIRS)* epidemic model. In an *SIRS* epidemic process, the immunity conferred after the recovery from a disease is limited, and therefore individuals get reinfected, thus giving rise to repetitive epidemic waves. To simulate this scenario we assign a short immunity period to the individuals of the synthetic population. We remind that according to the Neuro-SIR formulation, the immunity period is analogous to the refractory period of leaky integrate-and-fire neurons. During the immunity period, an individual's susceptibility remains clamped to a reset value, and therefore contacts with other infected individuals do not drive his/her susceptibility towards the infection threshold. After the expiration of the immunity period, an individual becomes susceptible again, and he/she can be reinfected. The outcome of this simulation experiment is shown in Fig. 13 illustrating three epidemic waves in a synthetic contact network with density *0.02225* and immunity period equal to *165ms*. The parameter values used in this experiment are detailed in Table 4.

Table 4
Parameters values in experiment 3.

Parameter	Value
S_{rest}	−52
S_{reset}	−54
τ	15 ms
S_i^{Thres}	Threshold values assigned to individuals from a normal distribution with mean = −50 and std = 0.6
Im^{Period}	165 ms
W_{ij}^{p}	5
d_{tr}	9 ms
τ_p	5 ms
G	Random graph with 1000 nodes
d_G	Network density: 0.02225

Fig. 14 illustrates the dynamics of the evolution of the individual's susceptibility $S_i(t)$, during the immunity and vulnerability periods. As discussed in the "Model" and "Methods" sections, the dynamics of an individual's susceptibility in the SIRS epidemic process, is driven by the differential equations (1) and (5). The trajectories of the dynamically evolving susceptibilities from the reset value to the infection threshold, for all the interconnected individuals of the simulation experiment 3, are depicted in Fig. 14. It can be seen that recurrent epidemics occur in waves, whereby vulnerable individuals contract the disease through infection cascades transmitting significant pathogenic load from the infected to the susceptibles. As discussed earlier, higher densities in the contact network lead to stronger and more solid infection bursts. Neuro-SIR models these bursts through the dynamics of excitation waves appearing in neural networks, and other excitable media.

5.5. Simulation experiment 4 – SIRS epidemic process with external factors

The fourth simulation experiment deals with a SIRS epidemic process, which is affected by external factors. The experiment aims to demonstrate the ability of Neuro-SIR to model the epidemic spreading, when external conditions influence the dynamics of the propagation of a disease. As already discussed, climate, pollution, and sanitary conditions are some of the exogenous factors accelerating or decelerating an epidemic. In this regard, we examine the case in which an external factor (e.g. weather) increases the individuals' susceptibility in a pathogen, thereby reinforcing the spreading of a disease. The external force is modeled as a step signal with strength equal to 0.001, applied on the network dynamical system for *30* ms starting from the 200^{th} ms of the simulation. In this case, the dynamics of the individuals' susceptibility is driven by the differential equations (5) and (6). The exogenous force increases the individuals' susceptibility during the *30* ms of its interaction with the system, and therefore the second epidemic wave appears earlier, and in a denser form in comparison with the corresponding wave of the previous simulation experiment, as shown in Figs. 15(b) and 15(c). The earlier appearance of the second wave brings closer the third epidemic wave, since the immunity period is set to a fixed value. The difference between the epidemic spreading pattern influenced by external conditions, and the respective pattern without exogenous factors is obvious in Fig. 15(b) depicting the former, and in Fig. 15(c) illustrating the latter. Fig. 15(a) shows the profile of the external force applied on the epidemic process. A detailed list of the parameter values of the simulation experiment 4 is provided in Table 5.

6. Discussion

Neuro-SIR, through its capacity to incorporate a large number of endogenous and exogenous factors into the spreading dynamics of a

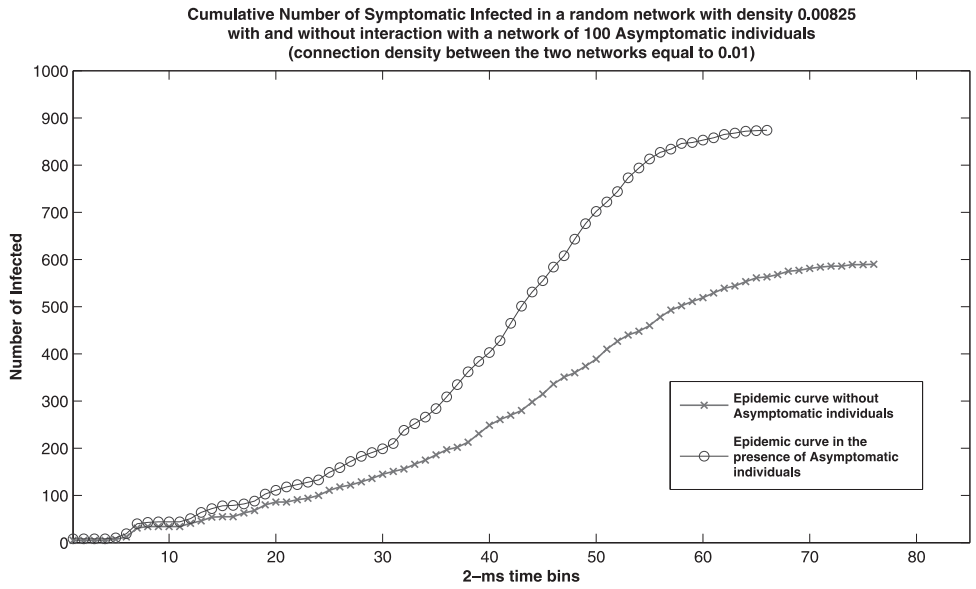

Fig. 12. The evolution of the cumulative number of infected individuals in the symptomatic layer with and without the presence of asymptomatic transmission.

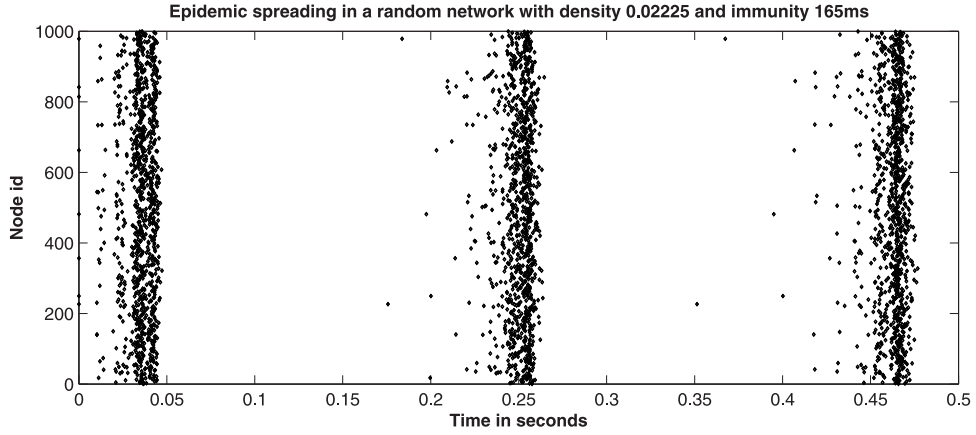

Fig. 13. Simulation of recurrent epidemic waves through Neuro-SIR by assigning short immunity periods to the individuals of the synthetic population.

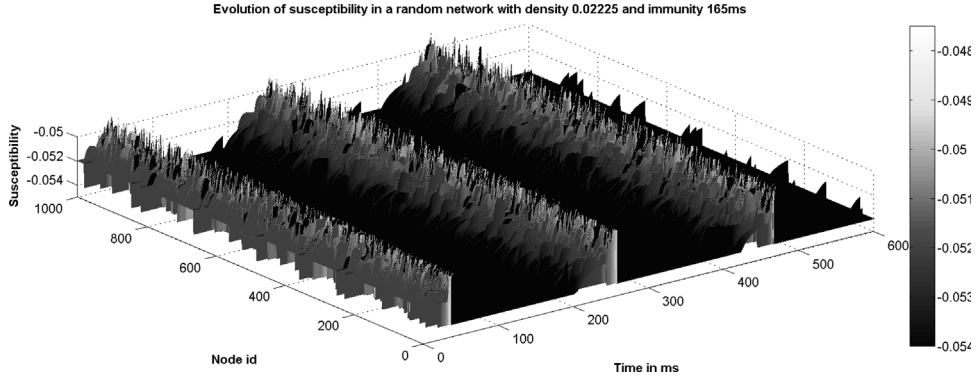

Fig. 14. Visualization of the evolution of the individuals' susceptibility in a random network with density 0.02225 and immunity period equal to 165 ms.

contagious disease, could be adapted to the modeling of the propagation of Covid-19, and of other contagious diseases, thus enabling the study of the dynamics of epidemics in a more insightful way. Recent research suggests that the spreading dynamics of Covid-19 is a multifaceted process. These aspects should be considered and included in modeling the propagation pattern of this disease. In this regard, Neuro-SIR can accommodate the following aspects pertaining to the spreading dynamics of Covid-19, as well as of other infectious diseases.

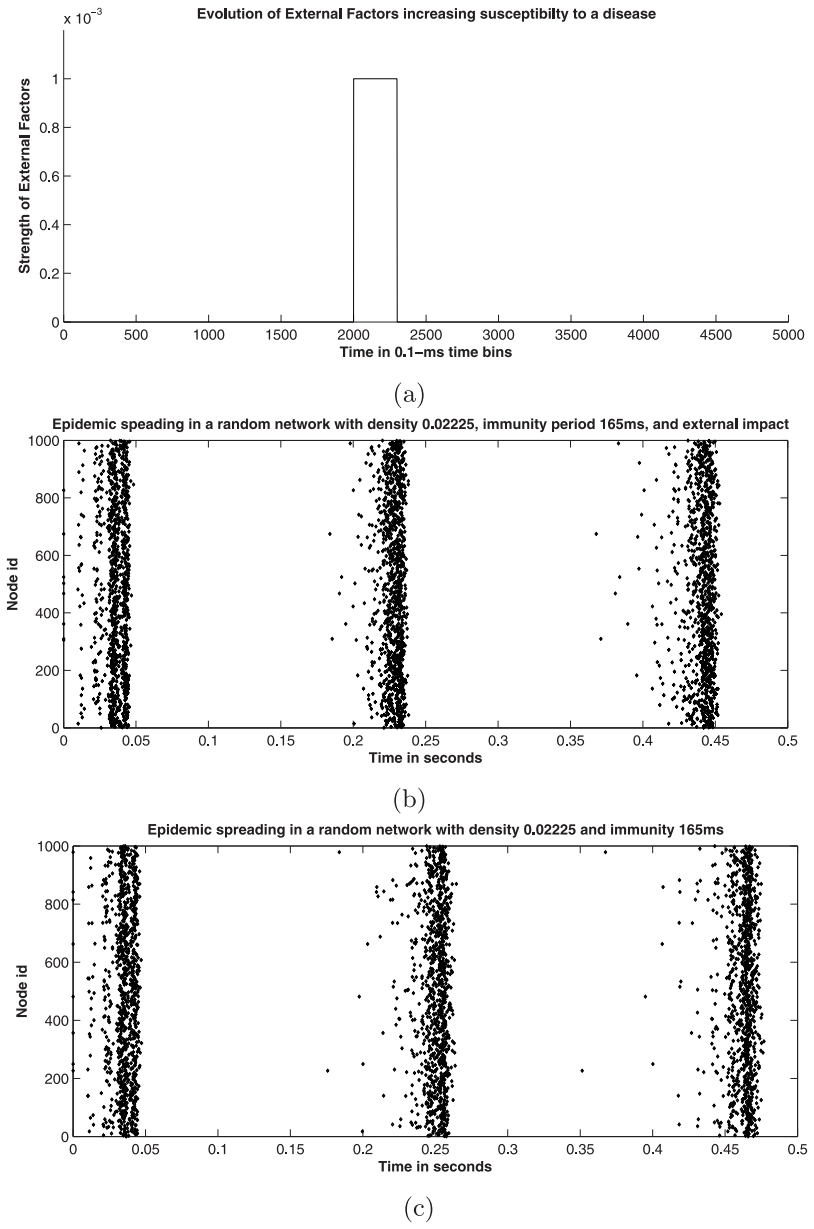

Fig. 15. (a) Profile of the external force applied on the epidemic spreading process, (b) Epidemic spreading pattern affected by the external force, (c) Epidemic spreading pattern without external factors.

i. *Use of personal protective equipment (PPE):* The impact of such preventing measures on the spreading of Covid-19 (Chu et al., 2020; Leclerc et al., 2020), could be simulated through the proper adjustment of the connection weights among individuals. Usage of PPE corresponds to smaller connection weights leading to less transmission risk of a contagious disease.
ii. *Social distancing:* Complying with social distancing rules has a significant impact on the containment of Covid-19 (Chu et al., 2020; Leclerc et al., 2020), and therefore incorporating this kind of prevention measures into the modeling of Covid-19, and similar contagious diseases, is important for a realistic simulation of an epidemic. Social distancing measures can be incorporated into Neuro-SIR, through the adjustment of the connection density of a social network.
iii. *Transmission of a disease through asymptomatic infected individuals:* Recent research confirms that asymptomatic individuals infected with Covid-19, considerably contribute to the spreading of Covid-19 (Huang et al., 2020; Nishiura et al., 2020; Sakurai et al., 2020; Wang et al., 2020). As it was demonstrated in experiment 5.3, Neuro-SIR supports the study of the dynamics of a contagious disease through interconnected multi-layer networks. In the implementation of the experiment 5.3, the role of asymptomatic individuals in the propagation of a contagious disease was studied through the creation of a group of simulated individuals, who are connected to another network comprising susceptible individuals, who can become symptomatic patients. The group of asymptomatic individuals comes into contact with susceptible individuals through a Poison process, whereby the asymptomatics remain active throughout the simulation. This approach imitates the situation, in which asymptomatic individuals with Covid-19 are not tested and quarantined, thereby forming a constant source of infections, substantially affecting

Table 5
Parameters values in experiment 4.

Parameter	Value
S_{rest}	−52
S_{reset}	−54
τ	15 ms
S_i^{Thres}	Threshold values assigned to individuals from a normal distribution with mean = −50 and std = 0.6
Im^{Period}	165 ms
W_{ij}^{p}	5
d_{tr}	9 ms
τ_p	5 ms
G	Random graph with 1000 nodes
d_G	Network density: 0.02225
$F_i^{ext}(t)$	0.001 for *30* ms starting from the 200th ms of the simulation experiment

the epidemic dynamics in the population of susceptible individuals, who can develop symptoms. To incorporate the lower risk of Covid-19 transmission through contacts between asymptomatic and susceptible individuals, the connection weight between them was configured with a lower value in relation to the connection weights between susceptible individuals, who develop symptoms. By studying the role of asymptomatics in the propagation of Covid-19 through Neuro-SIR, valuable insights can be provided into the design of the proper strategy for the testing of large portions of a population. The identification of the proportion of asymptomatics, could subsequently lead to simulation scenarios and development of strategies for the containment of their number, and their impact on the transmission of Covid-19.

iv. *Modes of transmission:* Covid-19 can be transmitted through various modes (Chan et al., 2020; Liu, Liao et al., 2020; Somsen et al., 2020; Stadnytskyi et al., 2020), such as physical contact, droplet, contaminated surfaces, or airborne. Each of these transmission modes is associated with a different level of risk, as the transmitted viral load varies in relation to the transmission mode. A possible implementation of this aspect through Neuro-SIR, is to configure an individual's connection weights to other people or objects, as a distribution of values, whose parameters could be estimated from relevant epidemiological data. Regarding the airborne transmission, that is, the propagation of a pathogen through aerosols in medical indoor places, or other internal settings with poor ventilation, it is possible to incorporate the impact of this type of transmission into Neuro-SIR, through the inclusion of exogenous factors increasing the susceptibility of individuals to a contagious disease, when located in such places. The adjustment of the impact of this type of external factors, could be performed in consultation with empirical studies quantifying the probability of the airborne transmission of Covid-19, or other similar diseases.

v. *Incubation period of a contagious disease:* Neuro-SIR enables the incorporation of the incubation period of infections through the adjustment of the value of the transmission delay. The transmission delay can either be set to a fixed value, or take values from a distribution, so it can more realistically describe variable lengths of the incubation period. For instance, recent research (Lauer et al., 2020; Yu et al., 2020) suggests that the incubation period of Covid-19 is on average 5–6 days, but it is also possible to last for 14 days. By including such variabilities into the spreading dynamics of contagious diseases, it is possible to more realistically simulate their propagation pattern.

vi. *Heterogeneous susceptibility:* The susceptibility of people to Covid-19 and other infectious diseases, as well as the severity of the illness in infected persons, may depend on many factors (Ludvigsson, 2020; Wu et al., 2020; Zheng et al., 2020), such as age and general health condition. Neuro-SIR can be configured so that the individuals' susceptibility follows the distribution pattern corresponding to real epidemiological data, thereby enabling a more realistic simulation of the propagation dynamics of a contagious disease, like Covid-19.

vii. *Exogenous factor:* External conditions, such as weather, pollution, sanitary conditions, crowding, ventilation, aerosols, can be incorporated into Neuro-SIR, through the inclusion of exogenous factors affecting the individuals' susceptibility (Asadi et al., 2020; Chia et al., 2020; La Rosa et al., 2020; Leclerc et al., 2020; Lu et al., 2020; Morawska & Cao, 2020), and therefore the spreading dynamics of contagious diseases, as shown in Section 5.5. Consequently, the adjustment of the values of such parameters, so they realistically describe the quantitative impact of the external environment on the evolution of an epidemic, enables a more precise simulation of the propagation of a contagious disease in a population.

viii. *Dynamically evolving susceptibility of a population to a contagious disease:* Fig. 14 provided a graphical representation of the dynamical evolution of the susceptibility of a population to an infectious agent. This dynamic susceptibility landscape, actually constitutes a map of the evolution of the risk regarding the development of outbreaks of a disease. Incorporating real time information about the number, frequency and proximity of contacts in a population of susceptible individuals, as well as information about exogenous factors as mentioned before, enables the reliable estimation of the community transmission risk of an infectious disease, thus permitting the timely organization and implementation of containment policies.

Simulating the dynamics of the transmission of a contagious disease in complex settings comprising all the aforementioned factors, is crucial to understanding the impact of various factors on how, when and in which conditions infectious diseases spread faster or slower. Neuro-SIR has the capacity to replicate complicated transmission scenarios in highly heterogeneous populations, affected by endogenous and exogenous factors. As a result, Neuro-SIR can significantly support the design and implementation of containment measures that could break the transmission chain of Covid-19, or other contagious diseases.

A proposal for future research could be based on the calibration and adaptation of Neuro-SIR to more realistic infection transmission scenarios. The implementation of such simulation experiments entails the estimation of the parameters of the model from real epidemiological data pertaining to particular infectious diseases, such as Covid-19. The information collected through test-and-trace programs for the control of the spreading of Covid-19, as well as demographic information and location data about the individuals who were infected by Covid-19, could be used for the configuration of highly realistic simulations enabling a more insightful study of the spreading dynamics of Covid-19. Such simulations could serve as platforms for the evaluation of "what-if" scenarios in relation to the assessment of possible measures for the containment of the spreading of Covid-19. A potential obstacle in implementing this direction, is the difficulty of getting access to such data, as it pertains to very sensitive private information. Nevertheless, as more research results are under way regarding the role of various transmission modes (including airborne), the role of viral load causing transmission of Covid-19, as well as the role of environmental and other exogenous factors that could trigger superspreading events; future research should focus on the adaptation of Neuro-SIR to these findings, with a view to developing a highly realistic simulation environment for the study of the dynamics of Covid-19, and of other contagious diseases. For instance, recent research (Liu, Yan et al., 2020) suggests that the viral load transmitted from severe cases of Covid-19 patients, is 60 times higher than the viral load passed on by Covid-19 patients in mild condition. Also, another research (Jones et al., 2020) examining the viral load in relation to the age of the Covid-19 patients suggests that there is no considerable difference between the viral load transmitted by patients in the age group of 1–20, and the age group of 21–100.

Another aspect of the transmission dynamics of Covid-19, relates to indoor crowding situations creating superspreading settings for the transmission of Covid-19. Recent research (Leclerc et al., 2020) indicates that confined indoor spaces, such as workplaces, shopping malls, transport means and churches among others, function as rapid transmission clusters fueling the propagation of Covid-19. Furthermore, a recent study (Chu et al., 2020) quantifies the transmission risk of Covid-19 in relation to social distances, and usage of personal protective equipment (PPE). Such information can be incorporated into the configuration of the parameter values of a Neuro-SIR implementation comprising multilayer networks with spatio-temporal structure representing varying interaction and activity networks (e.g. workplaces, gyms, shopping centers). Such implementations capture the dependence of the Covid-19 transmission risk, in relation to the location, activity, social distancing, and the individuals' contact network. Configuring the Neuro-SIR model in this way, enables the simulation of real epidemiological contexts, within which the spreading dynamics of Covid-19 should be examined. The calibration of Neuro-SIR in accordance with the epidemiological findings, relative to the transmission of Covid-19, constitutes a major goal of future research aiming at exploiting the full potential of the modeling capacity of the proposed approach.

Using the dynamics of leaky integrate-and-fire neurons to describe the transmission process of a contagious disease in a networked population, is of course an approximation of the real transmission process. It is more realistic than existing approaches, but nevertheless it cannot incorporate all the levels of details, associated with the dynamics of the propagation of a contagious disease. Replicating the behavior of a pathogen and the reaction of an individual's immunity system, requires a much more complex approach gearing towards the implementation of model pathogens, and model individuals with multiple layers of neurons in order to simulate more complex behaviors. Such an approach, increases significantly the computational requirements, and necessitates a more detailed description of the transmission dynamics of a disease. However, Neuro-SIR following a micro-level approach in the modeling of epidemic processes, incorporating endogenous and exogenous factors, and a high level of heterogeneity in the parameters of the model, paves the way towards a more realistic description of the dynamics of epidemics. Another major challenge that Neuro-SIR is faced with, relates to the access to the proper data for the estimation of the parameters of the model. Such information is not publicly available, as it pertains to very sensitive private information, owned by healthcare systems and administrative authorities. Consequently, the simulation of realistic scenarios regarding the propagation of Covid-19 or other infectious diseases, requires close cooperation with relevant organizations and authorities, which can provide access to anonymized data, suitable for the configuration of the Neuro-SIR model.

Recent research on the epidemiological characteristics, and the spreading dynamics of Covid-19, indicates that the transmission of Covid-19 constitutes a complex process involving several factors that affect the evolution of Covid-19 outbreaks. In particular, current epidemiological studies analyze: (a) The role of the SARS-CoV-2 viral load in the transmission of Covid-19 (Jones et al., 2020; Liu, Yan et al., 2020; To et al., 2020; Wölfel et al., 2020; Zou, Ruan et al., 2020). (b) The impact of social distancing and personal protective equipment on the propagation of Covid-19 (Chu et al., 2020; Leclerc et al., 2020). (c) The transmission of respiratory diseases through multiple contacts with infected persons (Ackley et al., 2019; Lee et al., 2016). (d) The incubation period of SARS-CoV-2 (Lauer et al., 2020; Yu et al., 2020). (e) The role of asymptomatic infected persons in the propagation process of Covid-19 (Huang et al., 2020; Nishiura et al., 2020; Sakurai et al., 2020; Wang et al., 2020). (f) The role of external factors (indoor places, air-conditioning, water environments, airborne transmission, contaminated surfaces) (Asadi et al., 2020; Chia et al., 2020; La Rosa et al., 2020; Leclerc et al., 2020; Lu et al., 2020; Morawska & Cao, 2020) in the spreading dynamics of Covid-19. Neuro-SIR complies with these findings, since it is capable of accommodating the aforementioned factors in the spreading dynamics of a contagious disease, as explained in the foregoing discussion, and demonstrated through the experiments performed in this study.

In addition, Neuro-SIR is also compatible with the classical epidemiological theory, and established epidemic models (Anderson et al., 1992; Bailey et al., 1975; Kermark & Mckendrick, 1927), such as the SIR and SIRS, as it was demonstrated in Section 5.1, 5.3, 5.4 and 5.5, dealing with the simulation experiments, and the validation of Neuro-SIR (Section 5.2) against the fundamental modeling concepts of epidemiology. The versatility of the Neuro-SIR model allows its realistic adaptation to the spreading process of Covid-19, as well as to the propagation of other infectious diseases. The flexibility and adaptability of Neuro-SIR to complex epidemiological processes, enable the study of the dynamics of infectious diseases in a more informative and insightful way. As a result, Neuro-SIR has the potential to significantly contribute to the investigation of epidemic processes, by experimenting with the configuration of critical factors that affect the propagation pattern of a contagious disease.

7. Conclusion

We introduced a novel neurodynamical model for the description of the dynamics of epidemic spreading through contacts in a population of individuals. In this regard, we initiated the modeling of the infection transmission process, through an isomorphism between disease spreading in a social network, and activity propagation in a network of leaky integrate-and-fire neurons. This approach enables the modeling of epidemic processes in highly heterogeneous populations and contagiousness contexts, in terms of susceptibility to a disease, pathogen infectiveness, proximity and number of contacts, disease incubation period, immunity, and exogenous factors affecting the propagation of an infection. As such, Neuro-SIR serves as a realistic platform for the investigation of epidemic spreading patterns in a significantly more extensive way than existing methods. Considering that epidemics, such as Covid-19, pose an unprecedented threat to public health with tremendous economic and societal ramifications worldwide, it is vital that prevention and containment strategies for epidemics are carefully investigated and planned before being implemented. Recent empirical evidence relative to the spreading velocity and diffusion of Covid-19, suggests that small differences in the timing and scope of lockdown measures, might have a vast impact on the size and duration of the outbreak of this disease. As a result, a thorough analysis of the effect of various parameters on the spreading dynamics of epidemics is an imperative for designing and implementing effective containment policies. Neuro-SIR can significantly help in this direction, as it functions at the micro-level dynamics of the propagation of a disease, thereby enabling the incorporation of multiple sources of heterogeneities in the epidemic spreading dynamics. Consequently, through Neuro-SIR it is possible to investigate more complex scenarios in relation to the impact of various endogenous and exogenous factors, and levels of variations in the individuals' vulnerability and exposure to a pathogen. Furthermore, Neuro-SIR can be used in conjunction with real epidemiological data featuring details regarding the demographic characteristics and general health status of an infected population; the contagiousness of a disease; the density of contact network; and external factors, such as climate, pollution, and sanitary conditions. This information can be incorporated into Neuro-SIR, thus giving rise to a powerful platform for examining the effectiveness of various prevention and control policies for an infectious disease, through simulations with real parameter values.

The results of the experiments performed in this study demonstrate that Neuro-SIR precisely reproduces standard epidemiological patterns, thereby confirming the validity and compatibility of the model with the existing knowledge in the epidemiological field. Consequently, Neuro-SIR constitutes a reliable basis for the exploration of the dynamics

of epidemics in a more enhanced way, by taking advantage of its flexibility and ability to simulate complex experimental contexts. In this regard, we provided introductory evidence of the efficiency of Neuro-SIR by investigating how the density of a contact network affects the penetration speed and scope of a disease in a population with inhomogeneous vulnerabilities to a pathogen. This experiment aimed at demonstrating the enormous impact of social connectivity in the propagation of a contagious disease, and how important it is to minimize the social interaction, since a small increase in the density of a contact network can make the difference between a widespread propagation, and the eradication of a disease. It was also shown that Neuro-SIR can be easily transformed into an SIRS epidemic model by properly configuring the length of the immunity period.

Through the simulation experiments it was shown that Neuro-SIR reproduces the stylized facts pertaining to the evolution of the total number of infected and susceptibles during an epidemic process in a networked population of individuals, thus complying with the existing knowledge and established SIR and SIRS epidemic models (Anderson et al., 1992; Bailey et al., 1975; Hethcote, 2000; Kermark & Mckendrick, 1927; Murray, 2007). Also the investigation of the role of the network density on the spreading dynamics of epidemics, indicates that Neuro-SIR is in line with state-of-the-art SIR and SIRS epidemic models (Barthélemy et al., 2004, 2005; Boguná et al., 2003; Dickison et al., 2012; Kuperman & Abramson, 2001; May & Lloyd, 2001; Moreno et al., 2002; Newman, 2002; Pastor-Satorras & Vespignani, 2001) incorporating the network structure into the dynamics through a mean field approach. Both Neuro-SIR and the aforementioned approaches, show that denser social networks accelerate and intensify the epidemics of contagious diseases, and therefore social distancing is crucial to inhibiting the progression of infections in a population of individuals.

However, Neuro-SIR through a micro-level approach to the dynamics of epidemics, based on the isomorphism between the activity propagation in neural networks, and the spreading of diseases in social networks, substantially expands the modeling framework of epidemics by allowing the incorporation of additional factors affecting the propagation of infectious diseases. The expanded modeling capacity of Neuro-SIR is in line with recent epidemiological findings pertaining to: (a) The role of the viral load on the spreading of Covid-19 (Jones et al., 2020; Liu, Yan et al., 2020; To et al., 2020; Wölfel et al., 2020; Zou, Ruan et al., 2020). (b) The containment of the propagation of Covid-19 through social distancing and use of personal protective equipment (Chu et al., 2020; Leclerc et al., 2020). (c) The impact of the incubation period of Covid-19 on the transmission dynamics (Lauer et al., 2020; Yu et al., 2020). (d) The role of the asymptomatic transmission in the spreading of Covid-19 (Huang et al., 2020; Nishiura et al., 2020; Sakurai et al., 2020; Wang et al., 2020). (e) The impact of exogenous factors on the propagation of Covid-19 (Asadi et al., 2020; Chia et al., 2020; La Rosa et al., 2020; Leclerc et al., 2020; Lu et al., 2020; Morawska & Cao, 2020).

Additionally, we illustrated the incorporation of asymptomatic transmission and exogenous factors in the propagation dynamics of a disease, thus providing an extensive platform for further investigation of the dynamics of epidemics. We also expanded on the ability of Neuro-SIR to capture the infection mechanism through multiple exposures to infected individuals. Recent epidemiological research suggests that there is a threshold in the number of exposures of a susceptible individual to infected ones, so that a contagious disease can be transmitted (Ackley et al., 2019; Lee et al., 2016). The infection threshold featured in Neuro-SIR, which is reached through contacts with many infected individuals, realistically incorporates the empirically confirmed contagion mechanism through multiple exposures. As a result, Neuro-SIR models the transmission of infectious diseases in a realistic way, thereby providing a modeling platform consistent with the epidemiological theory. Another aspect with a significant effect on the propagation patterns of infectious diseases, is the influence of external factors such as weather conditions, pollution, and the hygienic state of an environment within which the dynamics of an epidemic is examined. Such exogenous factors can either enable or hinder the transmission of an infection, thus drastically affecting the spreading pattern of a contagious disease. For this reason, it is important to include in an epidemic model both the intensity, and the temporal structure of time-varying external factors affecting the individuals' vulnerability to an infection. In this regard, we performed an experiment illustrating the ability of Neuro-SIR to involve exogenous factors into the dynamics of epidemics. This is achieved by dynamically changing the individuals' susceptibility to a pathogen through the integration of time-varying external signals driving one's susceptibility either closer to the infection threshold, or away from it. When the individuals' susceptibility has increased due to external conditions, then infection cascades of large size can easily occur, as the individuals' are closer to the infection threshold and few contacts with infected persons suffice to trigger widespread contagions.

Overall, Neuro-SIR constitutes a novel and overarching approach to the modeling of the dynamics of epidemics. It is a highly configurable mechanistic epidemic model incorporating endogenous and exogenous factors realistically describing the dynamics of propagating infections in heterogeneous populations. Consequently, Neuro-SIR can be a valuable tool for epidemiologists and governments, especially in times of public health, hygienic and economic crisis, caused by the outbreak of contagious diseases.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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