THE EFFECTS OF DIFFUSION OF INFORMATION ON EPIDEMIC SPREAD — A MULTILAYER APPROACH

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In this work, the aim is to study the spread of a contagious disease and information on a multilayer social system. The main idea is to find a criterion under which the adoption of the spreading information blocks or suppresses the epidemic spread. A two-layer network is the base of the model. The first layer describes the direct contact interactions, while the second layer is the information propagation layer. Both layers consist of the same nodes. The society consists of five different categories of individuals: susceptibles, infective, recovered, vaccinated and precautioned. Initially, only one infected individual starts transmitting the infection. Direct contact interactions spread the infection to the susceptibles. The information spreads through the second layer. The SIR model is employed for the infection spread, while the Bass equation models the adoption of information. The control parameters of the competition between the spread of information and spread of disease are the topology and the density of connectivity. The topology of the information layer is a scale-free network with increasing density of edges. In the contact layer, regular and scale-free networks with the same average degree per node are used interchangeably. The observation is that increasing complexity of the contact network reduces the role of individual awareness. If the contact layer consists of networks with limited range connections, or the edges sparser than the information network, spread of information plays a significant role in controlling the epidemics.

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1. Introduction

In modern societies, epidemics are a growing thread despite all advanced technological tools and practices. Social interaction patterns play the crucial role for the majority of the contagious diseases spreading mechanism as well as the precaution practices. Therefore, the in-depth understanding of the role of inter-woven social networks during an epidemic spread is a very active area of research and attracts considerable interest. The real social impact of the realistic predictive model studies can be understood when the severeness of both human and economy wise results of an epidemic are considered. Nevertheless, the social interactions are complicated relations of competing interests, hence, the modeling of the social phenomena requires a good understanding of the real interaction patterns and the dynamics among the members of the society. In the last 20 years, the interest in the social networks has intensified. Recent realistic models of real-world complex systems [1–3] have improved the understanding of a large variety of complex social interactions. As the societies become more technology-oriented, new channels of communication and interaction rapidly changed the structure and the topology of the interaction networks. Previously designed single-layer real-world networks left their place to multilayer networks: a developing social phenomenon finds its reflections on the other layers of social networks as different types of interactions. The spread of contagious disease is an excellent example of this situation. In the contact layer, the interactions of the individuals result in the spread of infection, while in the second layer, the information on the contagiousness of the disease motivates the individuals to take preventive measures. The preventive measures can be in two categories: vaccination and social distancing [4].

The most commonly encountered contagious diseases are suitably modeled by the susceptible-infected-recovered (SIR), susceptible-infected-susceptible (SIS) and susceptible-infected (SI) epidemiology models [5]. Different spreading mechanisms and epidemic control strategies are introduced for all three types of epidemiology models on complex networks [6–9]. The contagious disease has always been very destructive for the societies [10]. Hence, the mathematical models of the diffusion of contagious diseases have a long history, starting as early as the beginning of the 20^{th} century. The early models [11, 12] are aggregate models which rapidly evolved into agent-based models. Initial agent-based models employed fully connected or regular networks for the connectivity of the society. Recently, more realistic network structures are also incorporated along with the underlying dynamics of the spread [6, 9]. The epidemic studies on networks [13–16] are not only useful and limited to the spread of contagious diseases within human societies. A large variety of complex systems, such as physical, engineering, technological, and information networks exhibit similar diffusion of malicious agents [17–22].

The best prevention strategy in fighting the infectious diseases is the immunization. The immunization of the whole population is not a possibly realizable challenge [23]. Hence, various strategies of immunization which may be effective in the prevention of further spreading the infection are introduced. The effectiveness of such immunization methods is studied by using mathematical models of spreading phenomena. Random immunization and targeted (selected) immunization are the methods which aim to block the spreading paths of the contagion. The efficiency of the immunization is greater if one can select highly connected nodes. Such a selection requires prior knowledge of the whole network. Another immunization strategy is the acquaintance immunization in which the selection of highly connected nodes is naturally realized [7, 8, 24–26]. Another approach to the efficient immunization is awareness-motivated immunization: the informed individuals decide to take precautions. The most effective element of the decision-making process is the word-of-mouth. The word-of-mouth immediately recalls one-to-one interaction. In the real-life, the contact networks are only a small part of the interaction network. In modern societies, most of the information comes from the virtual-communication networks. In this sense, the word-of-mouth is all trustable one-to-one correspondences. The human element of the immunization strategies constitutes the spread of information and decision-making processes. The multiple networks widen the understanding of epidemic and epidemic control methods by introducing multiple spread mechanisms. The best example is the disease spreading on the contact network during the diffusion of information on another. The information creates the awareness of what is essential to control the epidemic spread [27–29], hence, the competition between the awareness and the disease spreading may rise to an epidemic threshold [30–32].

Complex networks are potent tools to describe spreading phenomena in both human societies and the other real-life problems. Nevertheless, the spreading phenomena among human societies have more elements than a single complex network. As a simple example, the traveling individuals change the dynamics of spreading infections. Similarly, information networks and social networks affect the dynamics of spreading. Hence, recently the models of spreading the infections are extended from a single complex network to multilayer networks [33, 34]. Multilayer networks [35–37] are composed of several layers of complex structures in which the same node may have multiple channels of interactions. The multilayer networks capture correct interaction structures between the nodes since an individual in a society may have different kinds of interactions such as business relations, social environment, connections through social media. Hence, each is in direct contact with some members of the society while communicating with some others on a virtual network of friends. In online social networks, the information propagates between the nodes through friendship connections which may be entirely different from the contact network of the nodes.

The most common examples of epidemics are respiratory infectious diseases. They also remain the most severe health risks as well as economical problem [38] facing the population. The importance of non-pharmaceutical interventions in the prevention of infectious diseases is the subject of protection motivation theory (PMT) [39]. In social distancing which may be merely hand-washing, wearing face masks, the increased tendency to reduce social contacts, may have a significant impact on the spread of the infectious disease. Some simple, low-cost, non-pharmaceutical prevention methods can minimize the transmission and impact of acute respiratory infections. The information coming from a trusted friend or colleague may break the social barriers such as rejection of personal risk of infection. In the present work, the focus is on the topic of spreading of contagious disease, while the intelligent nodes communicate on the severeness of the epidemic. Two interacting networks constitute the interaction base of the artificial society. The first network is the contact network in which contact interactions result in the spread of contagious disease. At this layer, SIR model governs the dynamics of the diffusion of contagion. The second layer, information spread layer, connects the same nodes with a different connectivity pattern. The individual gain awareness by the information gathering from the information network. It is a sociological fact that not all of the informed individuals act upon the received knowledge [40, 41]. There is an adoption process after which the individual reacts. The Bass model [42] governs the information adoption process which is the first step for social distancing or pharmaceutical interventions. Initially, the Bass model was introduced to describe the adoption process of a new product or opinion. Despite its simplicity, the model is still thriving to explain the diffusion of new ideas, information and it is commonly used in marketing studies. The main success of the Bass model is due to the well-represented social behavior of the individuals. The parametrization of the social behavior of the individuals is based on Roger's seminal work [40, 41] on the diffusion of innovation. The Bass model assumes two types of individuals. The first type, a minority group, accepts the new idea as soon as it is introduced. This group is also called innovators. The second group which is the majority of the population, likes to see the benefit of adaption of the new information. This group is called imitators. The originality of the proposed model is that no other model assumes a dynamics for the information adoption or awareness. In this work, the Bass model [42] sets the dynamics of information spread which results in the immunization or social distancing [4].

The work is organized as follows: The following section is devoted to the details of the multilayer network and the models of the disease and information spread dynamics. The third section presents the results of the proposed model and the effects of the proposed awareness dynamics on the epidemics. Finally, the conclusions are the subject of the last section.

2. The model

The proposed model is based on two well-known aggregate models, namely SIR and Bass models. The SIR model [5] was proposed to explain the dynamics of the infection spread for the illnesses spread by direct contact, while the Bass model is a well-established model of diffusion of information or innovations. In the proposed model, SIR and Bass models will be used interactively as the underlying dynamics of an agent-based simulation model. Before entering the details of the agent-based simulation model, some basic notation on the aggregate SIR and Bass models will be reviewed for completeness.

The SIR model dynamics [5] explains the infection spread among the members of a society which are susceptible to the infection. The susceptible individuals S meet a minimal number of infected individuals I and become infected. Infected individuals recover after a certain period or with a given probability. The recovered individuals R gain immunity until the end of the spread of infection. Infection spread stops when there exist no infected individuals

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = -\beta\, I(t)\, S(t)\,, \tag{1}$$

$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta\, I(t)\, S(t) - \gamma\, I(t)\,, \tag{2}$$

$$\frac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma\, I(t)\,, \tag{3}$$

where $S(t)$, $I(t)$, and $R(t)$ are the number of susceptible, infected and removed individuals at time t. The total population is the sum of all individuals regardless of their health state, $N = S + I + R$. The SIR model has two free parameters, β and γ. The parameter β represents an average rate of encounters between the infected and susceptible individuals, while the second parameter, γ, is the rate of recovery per unit time.

In the original form of the SIR model, every individual interacts with every other (fully connected system). There exists no underlying social network structure for the interacting neighbors. Moreover, individuals, as well as the interactions, are uniform. In order to accommodate individual behavior in the model, a new dynamics which controls the spread of awareness must be incorporated.

The Bass model well represents the dynamics of the information spread. The original form of the Bass model assumes two different types of individuals. The first group is the innovators who adopt a new idea immediately after having been informed. The second group is the imitators who want to see the results of the adoption of the new idea by observing the adopters.

A new idea starts to diffuse through innovators. After a certain number of initial adopters, imitators are the main driving force of the spread of information. The Bass equation is as follows:

$$\frac{\mathrm{dAW}(t)}{\mathrm{d}t} = \left(p + \frac{q}{N}\mathrm{AW}(t)\right)(N - \mathrm{AW}(t)), \tag{4}$$

where p and q are innovation and imitation parameters, N and AW are the total number and the number of aware individuals. Here, the innovation parameter, p, is the best understood as the probability of adoption of new information immediately after having been informed. The innovators are open to new information; they adopt the information as soon as a piece of new information arrives and respond accordingly. The imitation parameter q is related with the probability of adoption after observing the experiences of the neighbors (word-of-mouth). Imitators are the majority in any society.

The proposed model is an agent-based model inspired by the above-mentioned two very successful aggregate models. The main goal of the proposed model is to incorporate the infection and information spreading processes on a multiplex real-world network connectivity structure. The model consists of N nodes which accommodate N interacting individuals. A two-layer multiplex network has common nodes but different connectivity patterns. The first layer is the contact layer where the infection spreads, while the information spreads on the second layer. The state of each individual is given by two parameters, S_{layer_1}, and S_{layer_2}. Hence, $X_i[S_{\mathrm{layer}_1}, S_{\mathrm{layer}_2}]$, represents the state of the i^{th} node. layer_1 and layer_2 are the representative labels of contact and information layers of the network respectively. The contact layer state parameter, S_{layer_1} has five separate values: susceptible S, infected I, recovered R, vaccinated V and, finally, P which identify the state of the individual who has taken precautionary measures. In addition to the three states of the aggregate SIR model, two new states V and P are introduced which is a result of the awareness spread. The introduced five states connect the SIR and the Bass dynamics. Susceptible individuals obtain information through their virtual connections and take precautionary measures. The precautionary measures reduce the probability of interactions with their neighbors and hence the spread of illness. The awareness parameter, S_{layer_2} takes only two values aware (informed) AW and non-aware (uninformed) NA $= N -$ AW. Initially, all individuals are uninformed, $S_{\mathrm{layer}_2} = \mathrm{NA}$. The first infected individual is set as infected and informed. Both infection and the information spread start from the first infected node. Since direct contact and information spread are on different layers with different connectivities, the speeds of infection and information spreads are different.

2.1. Interactions

Initially, all nodes are initialized as susceptible, $S_{\text{layer}_1} = S$, and non-aware, $S_{\text{layer}_2} = \text{NA}$. The infection spread from only one randomly chosen node, i, $S_{\text{layer}_1} = I$. The first infected individual automatically becomes aware, $S_{\text{layer}_2} = \text{AW}$. Both contamination and information spread start from this single node. The infection spreads in the first layer by the contact interactions.

2.1.1. Spread of information

The information layer serves for two purposes: spread and adoption of the information on the disease. For both of these processes, the Bass model is suitable. The informed individuals transmit the information to their neighbors through their connections on the second layer. When an individual receives the information, they evaluate it. According to the dynamics determined by the Bass equation, the information is adopted or not. In the agent-based approach, the information adoption takes the following form:

$$\begin{array}{ll} \text{if } \ p \ > \ r & X_i[S,\text{NA}] \to X_i[S,\text{AW}] \\ \text{else if } \ q \times \frac{\text{NN}_{\text{AW}}}{\text{NN}} \ > r & X_i[S,\text{NA}] \to X_i[S,\text{AW}]\,, \end{array} \tag{5}$$

where $X_i[S,\text{AW}]$ and $X_i[S,\text{NA}]$ indicates the state of a susceptible, S, individual at the site i, in informed and non-informed state, respectively. p indicates the probability of the chosen individual being an innovator. The probability of an individual being an imitator is given by $q \times \frac{\text{NN}_{\text{AW}}}{\text{NN}}$, here, NN and NN_{AW} are the number of nearest neighbors and number of aware neighbors, respectively. The uniform random number r takes values between $0 \ \leq \ r \ < \ 1$.

If the information is adopted, the awareness state is set to aware, $S_{\text{layer}_2} = \text{AW}$. Once an individual is informed and adopted the information, sends messages to all neighbors to advise them about the severity of the infectious disease. Each individual sends the message only once just after having been informed. The individual remains informed, take precursory measures, until the end of the epidemic. The individuals take precursory measures according to their attitudes. Two precursory measures are vaccination and reducing the probability of interactions. If the susceptible individual is vaccinated (V), they gain immunity. If an individual takes a precursory measure of reducing the number of interactions, they do not gain immunity. They remain susceptible, but their interaction probability is reduced. The interaction at the information layer leads to the adoption of information and decision of a precursory action.

If an individual is susceptible and informed, $X_i[S, \mathrm{AW}]$, may take precaution (social distancing) or may prefer vaccination,

$$X_i[S, \mathrm{AW}] \rightarrow \begin{cases} X_i[P, \mathrm{AW}] & \text{if } \mathrm{Prb} > r\,, \\ X_i[V, \mathrm{AW}] & \text{if } (1-\mathrm{Prb}) > r\,, \end{cases} \tag{6}$$

where $X_i[S, \mathrm{AW}]$, $X_i[P, \mathrm{AW}]$, and $X_i[V, \mathrm{AW}]$ indicates the state of an informed AW individual at the site i, as susceptible, precautioned, and vaccinated, respectively. Here, Prb and $(1 - \mathrm{Prb})$ are the probabilities of an individual to take precaution or get vaccinated; r is a uniform random number, $0 \leq r < 1$. In this work, it is assumed that only 20% of the population prefers vaccination. The individuals who prefer social distancing are assumed to avoid three out of four social contacts. When contact is established between the susceptible who try to avoid contacts and infected the infection spreads with the usual rules given by Eq. (7).

2.1.2. Spread of infection

For the agent-based simulation model, SIR model dynamics is implemented as probabilistic interactions among the members of the society. At each time step, randomly selected nodes interact with the neighbors at the contact layer and spread information on the virtual network. The uninfected and aware individuals may be in two states: vaccinated, V, or in the precaution state, P in which the probability of interactions of the individual changes. At each time step, a randomly chosen individual interacts with a randomly chosen neighbor. The individual and its neighbor can be in any of the five states. Unless the interaction is between a susceptible and contaminated one, interacting individuals do not change the state. There are two types of susceptibles: S- and P-state individuals. For S state, each interaction with infective individual spreads contamination. For the individuals who are in the P state, the individual does not interact at every time step even if they are chosen. Their interactions are limited with a probability $p_{\mathrm{interaction}}$ which represent the prevention effort of the P-state individuals.

The equation gives the transition rules for the individual living at the i^{th} node which is in interaction with an infected neighbor, $X_j[I, \mathrm{AW}]$,

$$\begin{aligned} X_i[S, \mathrm{AW}, \mathrm{NA}] &\rightarrow X_i[I, \mathrm{AW}] \quad \text{if } \beta > r\,, \\ X_i[P, \mathrm{AW}] &\rightarrow X_i[I, \mathrm{AW}] \quad \text{if } p_{\mathrm{interaction}} > r \text{ and } \beta > r\,, \\ X_i[V, \mathrm{AW}] &\rightarrow X_i[V, \mathrm{AW}]\,, \\ X_i[I, \mathrm{AW}] &\rightarrow X_i[R, \mathrm{AW}] \quad \text{if } \gamma > r\,, \\ X_i[R, \mathrm{AW}] &\rightarrow X_i[R, \mathrm{AW}]\,, \end{aligned} \tag{7}$$

where $X_i[S, \mathrm{AW}]$, $X_i[I, \mathrm{AW}]$, $X_i[V, \mathrm{AW}]$, $X_i[P, \mathrm{AW}]$, and $X_i[R, \mathrm{AW}]$ indicates the state of an informed AW individual at the site i, as susceptible, infected, vaccinated, precautioned, and recovered, respectively. β, γ and $p_{\text{interaction}}$ are infection recovery and interaction avoiding probabilities, respectively. Probability of interaction correspond to the social distancing [4]. When the information of severity of the results of infection reaches an individual, the individual tries to avoid interaction with the neighbors.

The contact layer interaction rules are: if a susceptible individual interacts with an infected neighbor, they become infected ($S \to I$) with probability β. An infected individual becomes recovered ($I \to R$) with probability γ. Recovered, R, and vaccinated, V, individuals are not affected by an infected member of the society. The precautioned individuals, P avoid interaction with individuals in any state with probability $p_{\text{interaction}}$. Interaction probability is kept constant as $p_{\text{interaction}} = 0.25$, only one-fourth of the encounters ends with physical contact.

2.2. The multiplex network

Two interconnected networks, one for contact interactions and the second one for the spread of information carry the social interactions. Both systems share the same nodes with different intra-layer connectivity structures. The proliferation of contagious disease progresses on the contact network. The contact network layer has two alternative network structures: regular two-dimensional lattices with periodic boundary conditions and scale-free networks. The underlying network structure is scale-free for the information layer. This choice is due to the similarities between the scale-free and the real-world social network structures.

Both regular and scale-free networks are used as the contact layer. In the regular network case, periodic boundary condition with simple square ($k = 4$) and triangular ($k = 6$) lattices are used to test the effects of connectivity. Two different scale-free networks with the same average connectivity ($\langle k \rangle = 4$ and 6) per node are tested on the contact interaction layer. The Barabási–Albert network algorithm is used to generate the scale-free networks [43]. In this algorithm, the number of seed nodes, m, guarantees the average number of undirected edges, $\langle k \rangle = 2 \times m$ [43]. Changing the number of seed nodes controls the density of the number of connections, the degree of the node. The degree distribution of the nodes affects the spread of the information and the contagious disease. On the information layer, only scale-free networks are used. The networks with a wide range of average degree distributions are obtained by using the Barabási–Albert algorithm for the information layer. The effects of information spread on the spread of contagious disease are tested by using lattices in the range of $\langle k \rangle = 4$ to 20. The relation between the connectivity structure of two layers and the speed of the disease spread is the subject of the next section.

In the next section, simulation results, obtained by applying the proposed model, are presented with figures.

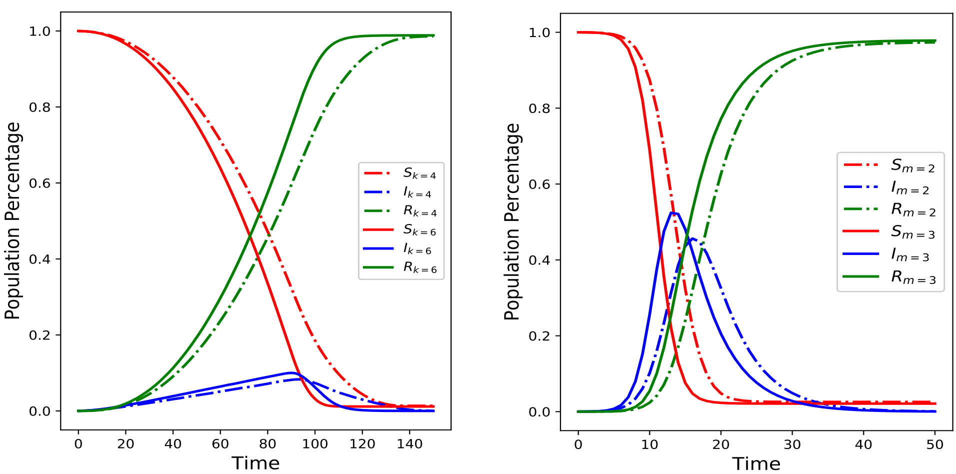
3. Results and discussion

An artificial society of $N = 10\,000$ inhabitants, each occupying a node on a multiplex network, is the simulation system. The connectivity of the nodes is two-fold. The first layer of the multiplex network is the contact network where individuals interact with each other through direct contact interactions. Hence, the contact layer provides a media for the transmission of contagion disease. The second layer is the information layer, through which the information spreads via virtual contacts. The conditions and the speed of the spread of news and infection are a function of both topology and the average degree of nodes. The contact layer consists of both regular lattice and scale-free networks, while for the information spread layer, of only scale-free networks with varying average degree per node.

The presented results are the averages of 100 simulations each starting from a statistically-independent initial configuration. The creation of an initial configuration consists of the creation of a multiplex network, initializing both contact and information layer state parameters for each node. An iteration is one discrete time step. One discrete time step consists of N interactions in the average; one interaction takes time, $\Delta t = 1/N$. In one time step, in the average, each individual interacts at least with one neighbor at each layer. Iterations are continued until the stationary configurations are reached. The stable configuration is reached when no longer state change occurs in either of the layers. The required time duration for the stable configuration varies according to the topology and the density of the links of the contact layer. For regular lattices, approximately 250 time steps are observed to be sufficient. The Barabási–Albert network provides a faster-transmitting media. The system reaches the stable configurations after only 50 time steps. During the simulation, all parameters, apart from the lattice parameters, are kept fixed to compare the effects of the lattice topology. For a fixed number of nodes, changing an average number of connections changes the interaction pattern and hence the speed of the spread. The contact layer parameters which control the spread of contagious disease, the infection transmission, β, and recovery, γ, parameters of the SIR model are kept constant for all networks. The transmission and recovery parameters are $\beta = 1$ and $\gamma = 0.2$, respectively. The information adoption is controlled by the Bass equation parameters p and q. Individuals who adopt information immediately after being informed are rare. The majority adopts it after observing the results of first-hand experiences. The values of innovation and imitation parameters are assumed to be similar to those of the average values obtained from the marketing studies. From marketing, the average ranges are $0.001 < p < 0.1$ and $0.1 < q < 0.5$ for innovation and imitation parameters, respectively. In this work, the fixed values of $p = 0.05$ and $q = 0.35$ are employed for all lattices.

In the societies, the direct contact networks usually have relatively small average degree per node. Hence, in the contact layer, the average degree per node is limited to $\langle k \rangle = 4$ and $\langle k \rangle = 6$. The regular networks are 2-dimensional simple square ($k = 4$) and triangular ($k = 6$) lattices with periodic boundary conditions, while the scale-free networks are generated by using the Barabási–Albert algorithm with initial sites of 2 and 3 which correspond to the average degree, $\langle k \rangle = 4$ and 6. The information spread layer is expected to have denser connections between the nodes. Hence, undirected scale-free networks with increasing density of the edges are generated by using the Barabási–Albert algorithm. The average degree, $\langle k \rangle$, per node is the control parameter of the spreads on the different information networks.

Figure 1 shows the spread of contagious disease on regular and scale-free networks without the contribution of the information layer. Two different topologies, with the same average degree per node, are square and triangular lattices and $m = 2$ and $m = 3$ Barabási–Albert networks, respectively. The comparison between Figs. 1 (a) and 1 (b) shows that the spread of infection on the contact layer is almost three times faster on the scale-free network than the corresponding regular network for the same transmission and recovery parameters. Since the transmission parameter is high, disease spreads among the population, but the peak values of the number of infected individuals are different depending on the topology. In the scale-free network case, almost half of the population is contaminated at the peak of the infection spread. In the regular lattice case, the peak value of the number of infected individuals remains around 10% of the total population. The increasing number of average connections per node pronounce the difference. Hence,



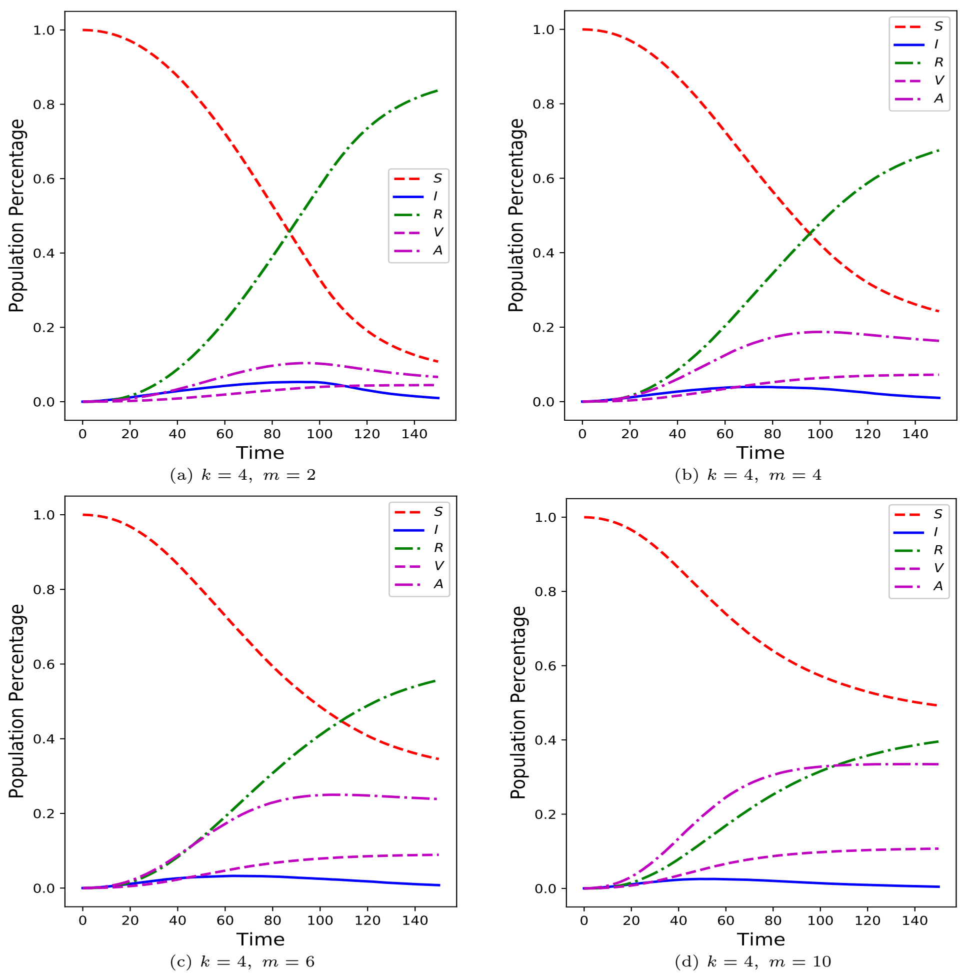
(a) SIR model on regular networks network

(b) SIR model on scale-free network

Fig. 1. Spread of contagious disease in a society with regular and scale-free contact layer network topologies. No information propagation is considered.

reducing the peak value of the number of infected individuals through mass media and social media plays a crucial role in the continuity of the social systems.

Constructing multiplex networks to study the effects of the information spread requires comparing multiple information networks which have the same contact layer network. As the first set of examples, a simple square lattice and a set of scale-free networks with the progressively increasing number of edges are taken as the topologies of contact and information networks, respectively. Figure 2 shows the effects of an increasing number of communication links. Usually, in the social systems, the contact networks are local interactions. Hence, diffusion takes more time than in the real-



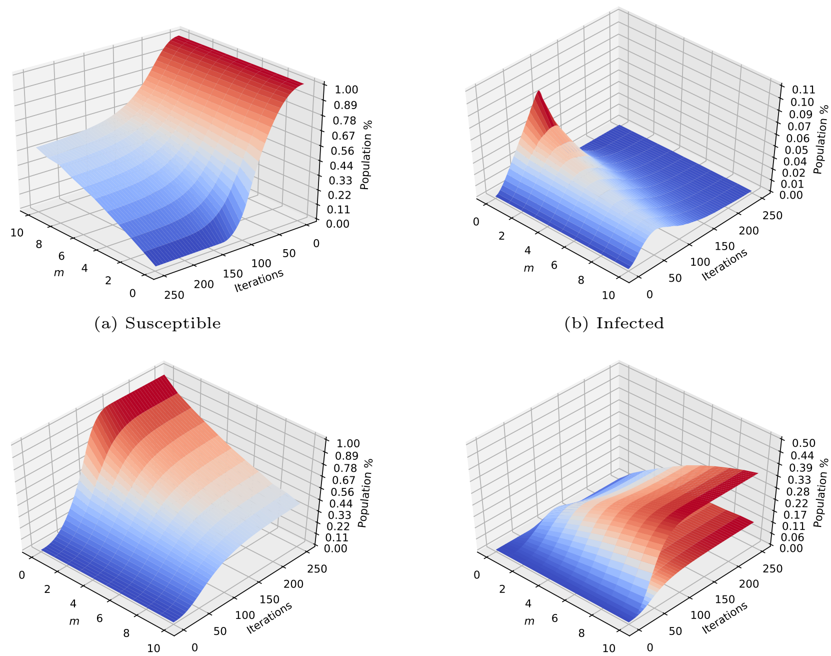
(a) $k = 4,\ m = 2$ (b) $k = 4,\ m = 4$ (c) $k = 4,\ m = 6$ (d) $k = 4,\ m = 10$

Fig. 2. Contagious disease spread on simple square lattice, $k = 4$, while the information spreads on scale-free network topology with increasing connectivity.

world networks. In this first model, an increasing number of second layer links speeds up the spread of information. If the individuals absorb and use the information in the correct way by taking precautions or getting vaccinated, the disappearance or at least control of the contamination is possible. Figures 2 (a), 2 (b), 2 (c) and 2 (d) show the effects of the increasing number of links on the spread of contamination. As explained in Section 2.1, the contamination spread according to the dynamics of the SIR model with an infection transmission rate, $\beta = 1$. If a susceptible contacts with an infective, the susceptible gets contaminated. Each informs all neighbors when they are infected. Adoption of the information is a process governed by the dynamics provided by the Bass equation. A small percentage of the individuals (innovators) immediately adopt the information and take a precaution. Others, collect information from the neighboring nodes before making a decision. Both, innovators and imitators have two choices as far as the precautions are concerned: getting vaccinated and avoiding contacts with the neighbors. In this work, the first assumption is that only 20% of the informed individuals choose vaccination. The rest prefers to keep away from any contact interaction. A second assumption is that on the 75% of the occasions susceptibles can save themselves from contamination by avoiding direct contact.

Figure 3 shows the changes in the number of susceptibles, infected, recovered, vaccinated and precautioned for a constant speed of contamination spread, the peak of the number of infected individuals decreases with increasing density of the information links. Figure 3 (a) shows the changes in the number of susceptibles as a function of time and number of initial sites m (average degree, $\langle k \rangle = 2 \times m$). For small m, all individuals get infected. As the number of initial sites approaches $m = 10$, over 40% of all susceptibles remain unaffected from the contamination which reduces the number of recovered (Fig. 3 (c)). Similarly, the peak of the number of infected individuals decreases rapidly with the increasing number of information links (Fig. 3 (b)). The number of vaccinated remains rather small compared with the number of precaution. Figure 3 (d) shows the changes in the number of vaccinated (below) and the number of precautioned (above) with respect to the changes in the number of connections in the information layer.

This effect manifests itself more profoundly in scale-free networks in the contact layer case. When the contact layer is in scale-free topology, the speed of transmission of infection is high comparing with the regular networks. Therefore, the peak of the number of infected is higher in the scale-free contact network concerning regular networks. Figures 1 (a) and 1 (b) show the differences in speed and the scale of the contamination between lattices and scale-free networks with equal average degree per node. The topology of the information layer plays a significant role in reducing both the total number of infected individuals and the peak in the number of infected individuals. Figure 4 shows the effect of the increasing density of information links while

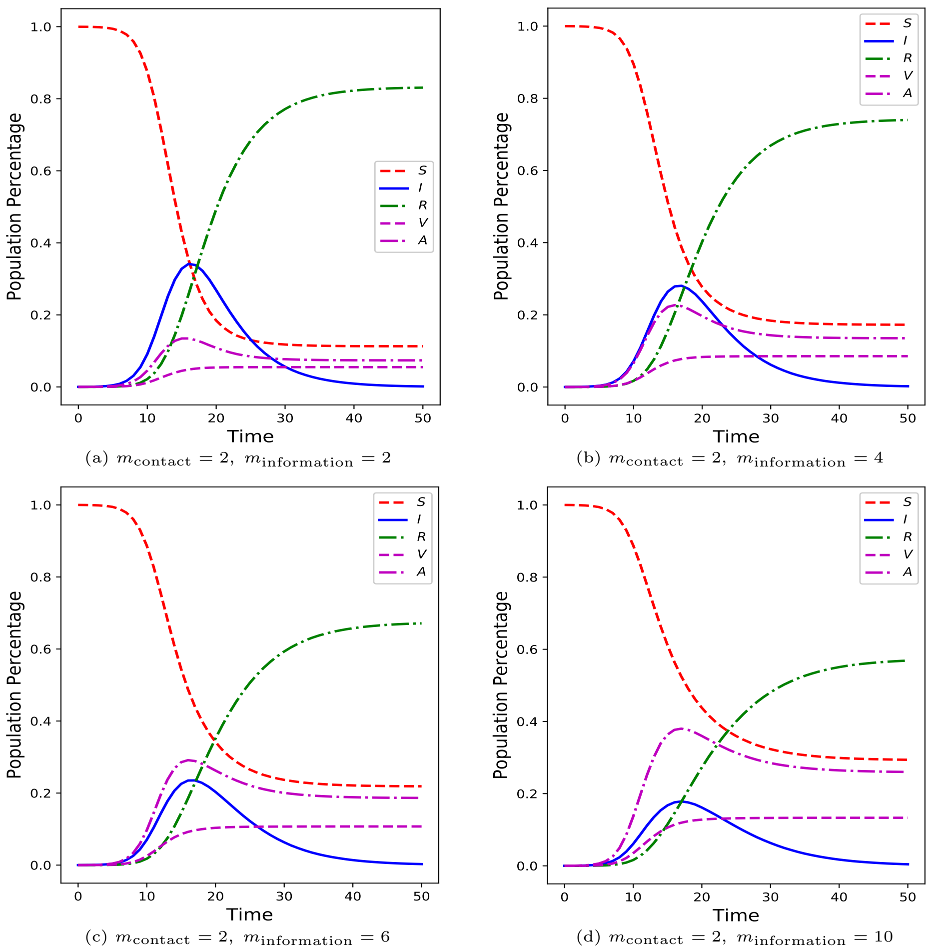


(a) Susceptible (b) Infected

(c) Recovered (d) Precautioned and vaccinated

Fig. 3. The effect of increasing connectivity of the information network on the infection spread. The number of initial sites of the information layer changes from 0 (network consists of only the contact layer) to 10.

the contact network is also scale-free with an average degree of 4 per node. The real-world networks, due to the complex connectivity structure, speed up the spreading phenomena. Figures 4 (a), 4 (b), 4 (c), 4 (d) show the effect of the density of information layer connections for fixed average degree in the contact layer. Increasing the number of average degrees decreases the peak of the number of infected. Two effects contribute to the decrease in the infections, vaccination, and awareness. Informed individuals either get vaccinated and gain immunity or avoid direct contacts with neighbors. As the number of communication links increases, the number of aware individuals increase which results in reducing the number of infected. Comparison of Figs. 4 (a), 4 (b), 4 (c), 4 (d) indicates that the main contribution in the prevention of the epidemic spread comes from the group of individuals who try to avoid direct contacts with the neighbors. This group of individuals increases as the number of infected individuals increase. Their peak is just before the peak of the number of infected individuals which prevent further contamination. As the number of links to the information layer increase, the peak of informed individuals increases with a further suppression on the spread of infection. The contribution to the prevention of the vaccinated does not grow at the same rate.



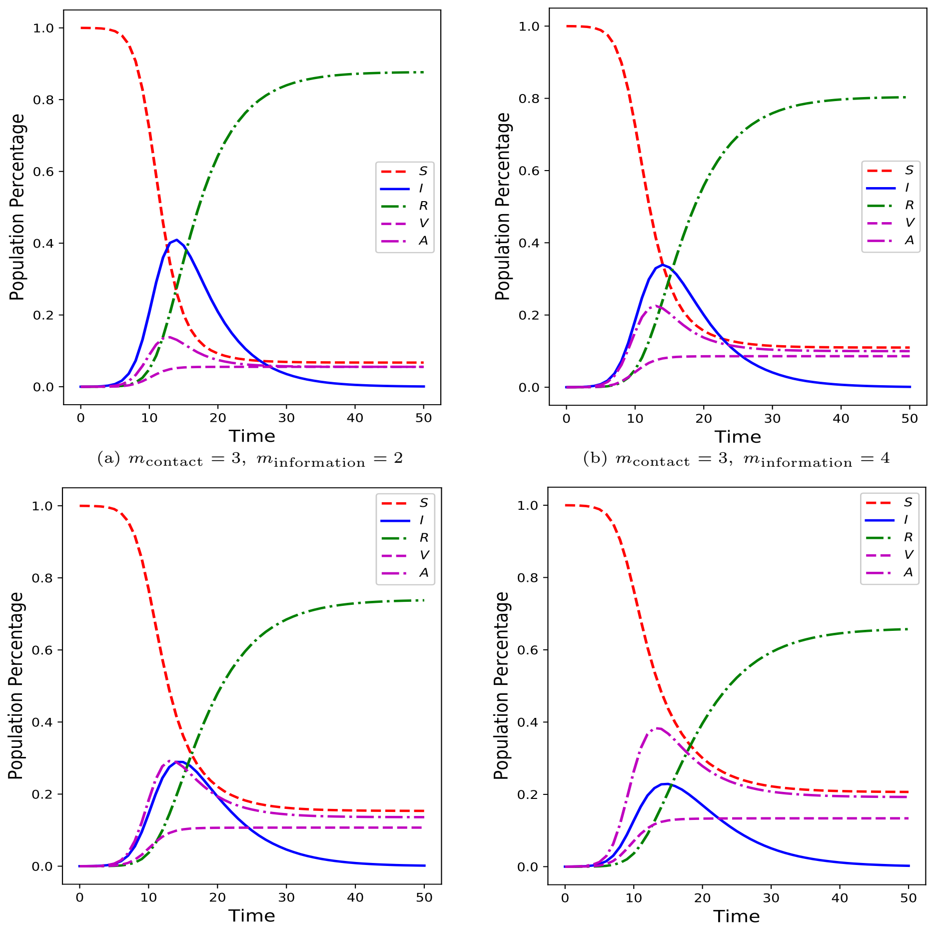
(a) $m_{\text{contact}} = 2,\ m_{\text{information}} = 2$ (b) $m_{\text{contact}} = 2,\ m_{\text{information}} = 4$

(c) $m_{\text{contact}} = 2,\ m_{\text{information}} = 6$ (d) $m_{\text{contact}} = 2,\ m_{\text{information}} = 10$

Fig. 4. Spread of contagious disease in a society with scale-free multilayer network topology. Contact layer has single initial sites configuration, $m = 2$, while the number of initial sites of the information layer changes, $m = 2, 4, 6$ and 10.

When the contact layer becomes denser, the propagation of the infection is very fast. Hence, spread of information to prevent further spread of the illness is less effective. Figure 5 shows the effect of information spread while the contact layer has scale-free topology with average degree per node equals 6.

Figure 6 summarizes the results of the model. The effect of the density of links on the information layer is observed on two different contact network topologies, regular and scale-free networks with equal average degrees per node. Figures 6 (a) and 6 (b) show the percentages of recovered (dash-dotted triangles), susceptible (solid dots), precautioned (solid triangles) and vaccinated (dashed triangles) individuals after the ending the spread of infection.

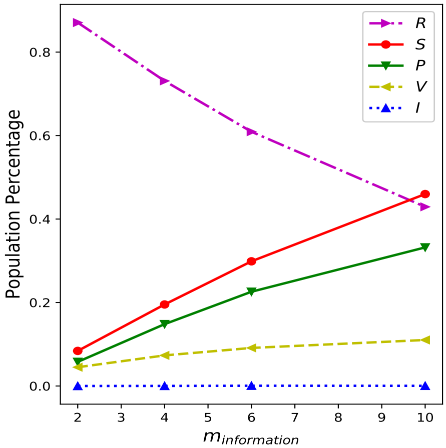


(a) $m_{\text{contact}} = 3,\ m_{\text{information}} = 2$ (b) $m_{\text{contact}} = 3,\ m_{\text{information}} = 4$

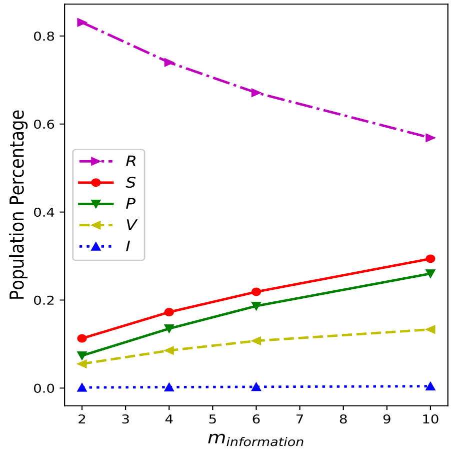
(c) $m_{\text{contact}} = 3,\ m_{\text{information}} = 6$ (d) $m_{\text{contact}} = 3,\ m_{\text{information}} = 10$

Fig. 5. Spread of contagious disease in multi-layer network with scale-free network topology. The same as Fig. 4 only the contact layer has denser connectivity structure, $m = 3$.

The bottom line shows no infected individuals. Figure 6 (a) indicates that as the average number of links to the information layer increases the number of infected individuals (R) decreases to almost 40% of the population which indicates that the total number of healthy (vaccinated or uninfected) reaches up to 60%. The situation changes slightly in the case of scale-free contact layer with $\langle 4 \rangle = 4$. Figure 6 (b) show that with increasing information spread, the percentage of the recovered individuals goes down to only 60% of the population. Total percentage of the non-affected individuals is almost 40%. The difference in the spread speed of the infection and information can explain this drastic difference between two layer topologies on the scale-free networks.



(a) Contact layer: Square lattice ($k = 4$)



(b) Contact layer: Scale-free network ($m = 2$)

Fig. 6. Aftermath of the epidemic. The susceptible, infected, recovered, vaccinated and precautioned population *versus* the number of initial sites of the information layer. Constant contact layer parameter is fixed to $k = 4$ for regular network (a) and $m = 2$ for scale-free network (b).

4. Discussion and conclusions

Recent analytical and simulation models indicate that the epidemic spreading on physical contact networks ignites the spread of awareness. The awareness of the individuals, in turn, suppresses the disease spreading. In this work, the relation between the epidemic spread and the effect of individual awareness was discussed. In the proposed model of society, the individual interacts through a two-layer multiplex network, physical contact and information spreading layers. The common nodes are affected by both the infection and information spreading in different layers. The dynamics of infection and information spreads are controlled by the SIR and Bass models, respectively. Adoption of information changes the attitude of the individuals; awareness diffusion creates a group of self-protected individuals. In this model, two types of self-protection are considered. Vaccination is the ultimate immunization method for most of the viral infections. Nevertheless, vaccination requires some effort, time and expenditure. Hence, the first assumption is that only 20% of the population considers vaccination. The rest try to avoid contacts with neighbors by social distancing. The price of not being vaccinated is that the precautions, apart from vaccination, provide only partial protection. As a second assumption, protection level of 75% is used to change the characteristics of the infection spread. Different topologies of contact and information networks embed different diffusion dynamics. Even the same topology with an increasing number of an average degree changes the spread rates of information and contamination. The effect of awareness on suppressing the infection spread makes its impact if the contact network diffusion speed is less than the spreading speed of information. The individual responses to an epidemic situation exhibit similarities but also vary from the adoption of an innovation. In the epidemic case, there exists an immediate danger to the well being of the individual. Identification of the individual response parameters may improve the epidemic prevention efforts considerably.

REFERENCES

[1] M.E.J. Newman, *SIAM Rev.* **45**, 167 (2003).

[2] M. Newman, *Networks: An Introduction*, Oxford University Press, 2010.

[3] C. Castellano, S. Fortunato, V. Loreto, *Rev. Mod. Phys.* **81**, 591 (2009).

[4] L. Williams *et al.*, *Psychol. Health Med.* **20**, 832 (2015).

[5] W.O. Kermack, A.G. McKendrick, *Proc. R. Soc. A* **115**, 700 (1927).

[6] R. Pastor-Satorras, A. Vespignani, *Phys. Rev. Lett.* **86**, 3200 (2001).

[7] R. Pastor-Satorras, A. Vespignani, *Phys. Rev. E* **65**, 036104 (2002).

[8] N. Madar *et al.*, *Eur. Phys. J.* **38**, 269 (2004).

[9] R. Pastor-Satorras, C. Castellano, P. Mieghem, A. Vespignani, *Rev. Mod. Phys.* **87**, 925 (2015).

[10] P.S. Brachman, *Int. J. Epidemiol.* **32**, 684 (2003).

[11] N.T.J. Bailley, *The Mathematical Theory of Infectious Diseases and Its Applications*, London: Charles Griffin & Company Ltd, 1975.

[12] R.M. Anderson, R.M. May, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, 1992.

[13] Y. Moreno, R. Pastor-Satorras, A. Vespignani, *Eur. Phys. J. B* **26**, 521 (2002).

[14] M.E.J. Newman, *Phys. Rev. E* **66**, 1 (2002).

[15] D. Chakrabarti *et al.*, *ACM Trans. Inform. Syst. Security* **10**, 1 (2008).

[16] P. Van Mieghem, J. Omic, R. Kooij, *IEEE/ACM Trans. Networking* **17**, 1 (2009).

[17] Y. Moreno, M. Nekovee, A. Pacheco, *Phys. Rev. E* **69**, 066130 (2004).

[18] J. Kleinberg, *Nature* **449**, 287 (2007).

[19] P. Wang, M.C. González, C.A. Hidalgo, A.-L. Barabási, *Science* **324**, 1071 (2009).

[20] M. Meisel, V. Pappas, L. Zhang, *Comput. Networks* **54**, 901 (2010).

[21] A. Guille, H. Hacid, C. Favre, D.A. Zighed, *SIGMOD Rec.* **42**, 17 (2013).

[22] J. Borge-Holthoefer, R.A. Banos, S. Gonzalez-Bailon, Y. Moreno, *J. Complex Networks* **1**, 3 (2013).

[23] C. Buono, L.G. Alvarez-Zuzek, P. Macri, L. Braunstein, *PloS ONE* **9**, e92200 (2014).

[24] R. Cohen, S. Havlin, D. Ben-Avraham, *Phys. Rev. Lett.* **91**, 247901 (2003).

[25] Y. Chen *et al.*, *Phys. Rev. Lett.* **101**, 058701 (2008).

[26] C. Gao, J. Liu, N. Zhong, *Knowledge Inform. Syst.* **27**, 253 (2011).

[27] C. Granell, S. Gómez, A. Arenas, *Phys. Rev. Lett.* **111**, 128701 (2013).

[28] C. Granell, S. Gómez, A. Arenas, *Phys. Rev. E* **90**, 012808 (2014).

[29] X. Wei *et al.*, *Europhys. Lett.* **114**, 26006 (2016).

[30] W. Wang *et al.*, *Sci. Rep.* **4**, 5097 (2014).

[31] Z. Wang *et al.*, *Europhys. Lett.* **112**, 48002 (2015).

[32] W. Wang *et al.*, *Sci. Rep.* **6**, 29259 (2016).

[33] L.G. Alvarez-Zuzek, C. Buono, L. Braunstein, *J. Phys.: Conf. Ser.* **640**, 012007 (2015).

[34] D. Zhao *et al.*, *PloS ONE* **11**, e112018 (2014).

[35] M. Kivelä *et al.*, *J. Complex Networks* **2**, 203 (2014).

[36] S. Boccaletti *et al.*, *Phys. Rep.* **544**, 1 (2014).

[37] J. Shao, Y.S. Buldyrev, S. Havlin, H.E. Stanley, *Phys. Rev. E* **83**, 036116 (2011).

[38] WHO Guide to Identifying the Economic Consequences of Disease and Injury, World Health Organization, 2009, ISBN 978 92 4 159829 3.

[39] H. Boer, E.R. Seydel, *Protection Motivation Theory*, in: *Predicting Health Behaviour: Research and Practice with Social Cognition Models*, M. Conner, P. Norman (Eds.), Maidenhead, BRK, England: Open University Press, 1996, pp. 95–120.

[40] M.R. Everett, *Diffusion of Innovations*, 1^{st} ed., New York: Free Press of Glencoe, 1962.

[41] M.R. Everett, *Diffusion of Innovations*, 5^{th} ed., New York: Free Press, 2003.

[42] F. Bass, *Manage. Sci.* **15**, 215 (1969).

[43] A.-L. Barabási, R. Albert, *Science* **286**, 509 (1999).