A computational model of Internet addiction phenomena in social networks

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Abstract. Addiction is a complex phenomenon, stemming from environmental, biological and psychological causes. It is defined as a natural response of the body to external pulses, such as drugs, alcohol, but also job, love and Internet technologies, that become compulsive needs, difficult to remove. At the neurological level, the Dopamine System plays a key role in the addiction process. Mathematical models of the Dopamine System have been proposed to study addiction to nicotine, drugs and gambling. In this paper, we propose a Hybrid Automata model of the Dopaminergic System, based on the mathematical model proposed by Gutkin et al., that allows different kinds of addiction causes to be described. In particular, we consider the problem of Internet addiction and its spread through interaction on social networks. This study is undertaken by performing simulations of virtual social networks by varying the network topology and the interaction propensity of users. We show that scale-free networks favour the emergence of addiction phenomena, in particular when users having a high interaction propensity are present.

Keywords: computational model, Hybrid Automata, simulation, scale-free networks, dopamine system, internet addiction, social networks

1 Introduction

Addiction is a social and complex phenomenon that has had different interpretations over the years. We can define it as a natural response of the body to some substances and external stimuli that, having a strong repercussion on the organism, become a compulsive need. This condition is difficult to remove because dependence produces a false feeling of wellness, which leads to a total loss of control and to repeat the same actions periodically. Many factors contribute to the development of addiction; it can be influenced by biological, psychological and environmental factors.

From the biological point of view, the brain has the central role: many neural circuits and, above all, the Dopamine System, are involved in the addiction process [17]. Dopamine is a neurotransmitter and has many functions in the body. In the brain, it has a key role in the reward system: the level of dopamine

changes after a desired external stimulus is received (the achievement of a goal, the intake of a chemical substance, etc.), producing a sensation of pleasure. Such a sensation may induce the subject to look for a repetition of the stimulus. If repetition happens too frequently, the subject may enter an addiction state in which the effect of dopamine decreases (tolerance). This can cause the addict to look for stronger stimuli and to suffer in case of absence of stimuli.

Environmental aspects of addiction are the subject of recent studies [2], focusing on the impact of age, sex and social background on the spread of addiction, on which the effect of the peer group acts too. Indeed, some habits are easily shared in social aggregation because of the emulation principle for which people in a group tend to imitate each other.

In this paper, we study Internet addiction, namely the excessive Internet (and technology) use that may interfere with daily life, and the way it spreads through the interaction on social networks. In particular, we show that the scale-free topology of social networks could be a favoring factor for the spread of Internet addiction among their users.

To facilitate and improve the comprehension of this particular kind of addiction, we examined it from a computational perspective. Our hypothesis combines the last theories about the use of social networks [9] from the users and the pre-existing computational models of addiction to drugs [16] and, in particular, to cocaine [7], since there aren't other models on Internet addiction.

We start from the mathematical model proposed by Gutkin et al. in [10] for the study of nicotine addiction. Such a model describes the main neurological processes involved in addiction phenomena and it has been validated against experimental data [6]. Moreover, the model describes the interaction between dopamine and neurological receptors that lead to persistent changes in brain structures (due to neuronal plasticity) that really occur in the case of addiction.

In our work, we simplify and, subsequently, extend the model proposed by Gutkin et al. in order to adapt it to different forms of addiction. To this purpose, we define our model in terms of Hybrid Automata [11], that allow us to better describe the different responses of the Dopaminergic System to stimuli of different intensities, and to better separate the description of the neural structures from the description of the external stimuli that are the cause of addiction.

In order to show that our model is a conservative modification and extension of the model in [10], we perform simulations that reproduce the results on nicotine addiction already obtained by Gutkin et al. Subsequently, we move to the problem of Internet addiction by performing simulations of networks of individuals, rather than of a single subject. This means that in each simulation experiment the model is replicated as many times as the number of individuals of the network. Moreover, stimuli for an individual are represented by messages sent to and received from other individuals in the network. Consequently, each individual has his/her own dopamine level and stimuli may cause some of the individuals to become addict.

We perform simulation experiments of three kinds of network: a 2-nodes network, a star graph and scale-free networks. The first two kinds of network

are used to reason on the model parameters (in particular, on the interaction propensity factor of social network users), while the scale-free networks (that may represent the structure of a real social network) are used to show that such a topology may actually favor the spread of Internet addiction.

2 The Model of the Dopaminergic System

We start from the model proposed by Gutkin et al. [10], which defines the working principles of the Dopaminergic System in the case of constant stimuli.

Mathematical model. The model consists of differential equations describing the dynamics of the Dopaminergic System, of the Action-Selection circuits and of synaptic plasticity. We give a simplified definition of the model by describing the Action-Selection and synaptic plasticity components by means of a simpler differential equation representing, in a abstract way, the "memorization" of the received stimuli. Moreover, we replace the sigmoid functions used in the model to implement threshold-based switches, with simpler differential equations defined by cases. As a result, we obtain a model consisting of two differential equations:

- *Dopamine concentration.* We have an equation describing the dynamics of variable D representing the dopamine concentration in the prefrontal cortex:

$$\frac{dD}{dt} = \alpha \left(-D + k + \begin{cases} 1, & if\ r - M \geq \theta_p \\ 0, & if\ \theta_n \leq r - M \leq \theta_p \\ -\frac{D*M}{2}, & if\ r - M \leq \theta_n \end{cases} \right)$$

  The dynamics of D is calculated by considering the following parameters:
  - k is the basal production rate of dopamine;
  - r is the perceived stimulus;
  - M is the memory of the stimulus, whose value is given by the second differential equation;
  - θ_p is the positive threshold, in the simulation is set to 80;
  - θ_n is the negative threshold, in the simulation is set to -30;
  - $\alpha = 0.3$ is a unique time-scaling parameter.

  Apart from standard decay and basal production, the differential equation describes the dynamics of the dopamine concentration by considering three cases given by the comparison of the current stimulus r with the memory M. When the stimulus is largely greater than the memory, the dopamine concentration increases. When the stimulus and the memory are comparable the dopamine concentration does not increase. Finally, when the stimulus is largely smaller than the memory, the dopamine concentration decreases with a rate that depends both on D and on M.
- *Memory.* The second differential equation describes, in an abstract way, the opponent process (in psychology defined as a contrary emotional reaction to a previous stimulus) that is modeled as a "memorization" process of previous stimuli.

$$\frac{dM}{dt} = \alpha \left(-M + \begin{cases} \frac{r-M}{2}, & if\ r > M \\ 0, & otherwise \end{cases} \right)$$

Dopamine and memory take different times to reach "high" values: Memory requires some time to reach values comparable to the stimulus r, but when it reaches such a level, it contrasts the increase of dopamine concentration in the brain.
To establish if a user became addicted, we considered properly the memory level, because it represents the tolerance and so the phenomenon that better characterizes the addiction. As threshold we selected $M \geq 15$, because at that point in the performed simulations, the users showed peaks and consequently decreases in dopamine trend.

Hybrid Automata model. Hybrid Automata [3,11] are finite state automata in which states are associated to differential equations that describe the dynamics of a set of continuous variables. Transitions of Hybrid Automaton can update the values of the variables in a discrete way. Moreover, by moving to a different state, transitions can also activate a different set of differential equations.

We propose a Hybrid Automata model to analyse the mechanisms of Dopaminergic System in an addiction context. It is an extension of the model by Gutkin et al. because it allows different types of stimuli to be dealt with. The stimulus is no longer a simple parameter of the differential equations, but becomes a continuous variable whose dynamics is governed by a Hybrid Automaton.

Indeed, by exploiting the modularity of Hybrid Automata, we can separate the part of the model describing the Dopaminergic System from the part of the model describing the dynamics of the stimuli. The two parts are described by two different automata that are composed in parallel. In order to consider a different stimulus (or a stimulus with a different dynamics) it will be enough to change the relevant automaton, without changing the automaton of the Dopaminergic System.

In order to validate our model and to show that it is a conservative modification and extension of the one proposed by Gutkin et al., we used it to reproduce the experiments on nicotine addiction presented in [10].

In Figure 1 the two automata constituting our model are depicted. The biggest one is the automaton describing the Dopaminergic System. It essentially corresponds to the already described differential equations of the dopamine concentration D and of the memory M, in which the cases are made explicit as different states of the automaton. The initial values of D and M are 0.2 and 0, which represent, in percentage, the neural activation as in [10]. The initial state of this automaton depends on the initial value of the stimulus r.

The smaller automaton is the one describing the stimulus, i.e. the continuous variable r. In this case, the stimulus dynamics is the same considered in [10]: initially set to 100, then constant for 25 days and then interrupted (by the transition that updates r into 0). The parameter, representing the constant stimulus, is experimentally obtained from the performed simulations, to reproduce

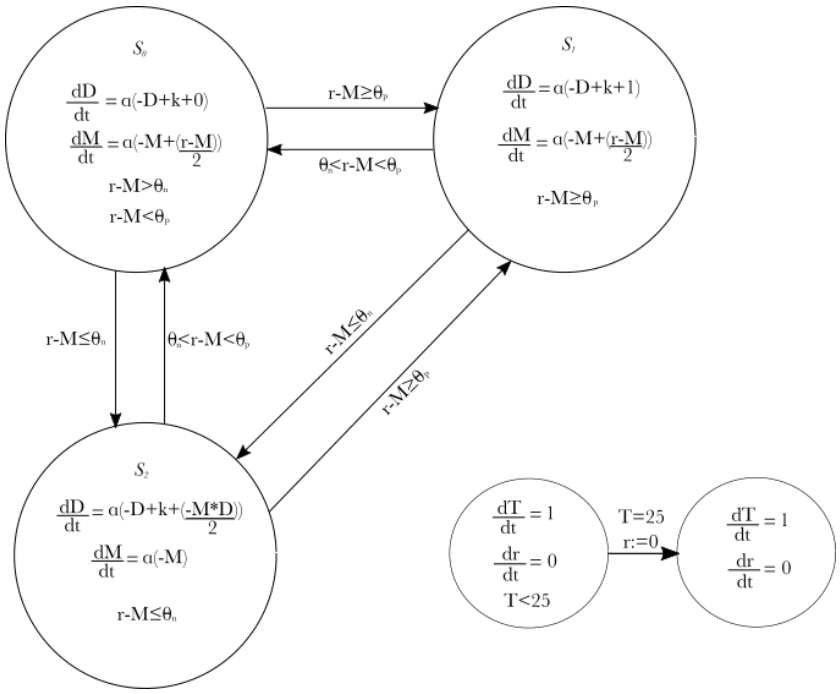


Fig. 1: Hybrid Automata of the Dopaminergic System and of a stimulus that remains constant for 25 days and then is interrupted.

the achieved results in [10]. The continuous variable T and the corresponding differential equations are used only to make it possible to define the guard on the elapsed time (25 days).

Simulation. The simulation and, in particular, the resolution of the differential equations are implemented using GNU Octave [12]. In Octave, the differential equation is written in the form of a vector *xdot* that is passed to the standard ODE solver LSODE. The discrete transitions are implemented as *if-then-else* conditions inside the functions computing *xdot*. The Octave source code of the model is freely available online [1].

Graphical results. From the performed simulations, we get three graphs, representing respectively dopamine and memory trends, in relation to impulses. The dopamine trend, similar to graphs in [10], shows an initial peak resulting in withdrawal symptoms before the stimulus interruption after 25 days. Subsequently, the dopamine concentration is sustained only by the basal production rate. The performance of the memory, however, corresponds to the opponent process, that in the motivational theory has the function to quiet a previous process, which becomes weaker.

The memory ensures the pulse is absorbed and routinized, reducing its perception. It grows slower than the dopamine and shows no peaks, but its growth counteracts the constant dopamine trend, causing a spike down when the impulse is interrupted.

In order to study the possible scenarios of addiction development, in [14], we modified the automaton describing the stimulus in order to describe non-constant stimuli. In particular, we studied how the dopamine response changes to stimuli provided at regular intervals and to impulsive stimuli in which both the frequency and the intensity change dynamically in response to changes in the dopamine concentration (in order to simulate a subject that looks for more frequent or stronger stimuli when the feeling of satisfaction due to the high dopamine concentration disappears).

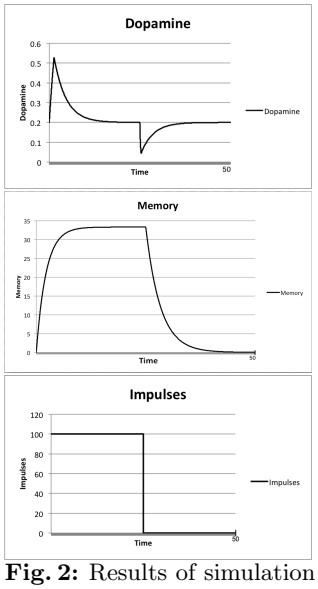


Fig. 2: Results of simulation at constant pulses

3 The Internet addiction

In few years, the impact of Internet and technology has fundamentally changed the way we relate and communicate with each other [21]. People, especially the younger ones, tend to prefer non-verbal communication, choosing text messages to communicate with their peers [9]. Adopting a computer-mediated communication has multiple consequences, such as the loss of empathy and the increase of cortisol, resulting in increased stress and addiction [13]. Usually, users show a different attitude to this communication form, which mainly depends on their level of stress, sense of isolation and inadequacy [15]. To investigate this type of behaviour, we decided to use our model of addiction to study different kinds of scenario. Among all the kinds of Internet addiction, we choose to consider the one due to social network usage since, as reported in some recent studies, it is a very common and increasing phenomenon [19,20].

3.1 The network communication model

Social networks, similarly to many other Internet related networks, have a scale-free topology [8]. Therefore, in order to study them, we consider graphs in which each node corresponds to a user, edges corresponds to social network connections. Our automata-based model is then replicated once for each node of the graph, thus allowing the dopamine and memory levels of each user to be considered and simulated.

Each node of the graph (that is, each user) is also associated to a parameter, which spans the range [0,1], that we call *propensity factor*. Such a factor, governs the approach to the network and influences the probability of such user to send (or reply to) messages through the social network.

In particular, communication on the social network is simulated as follows:

- we assume, for simplicity, that each user can send at most one message each day (apart from replies). Such a single message actually represents, in an abstract way, the involvement of the user in social network interactions during such a day;
- on each day, each user chooses with a probability proportional to his/her propensity factor whether to send a message to his/her neighbors or not. If sent, the message is received by all the neighbors;
- on the same day, all user that have received one or more messages choose whether to reply or not, again with a probability proportional to their propensity factors.

The exchange of messages causes stimuli to be received by users. In normal conditions, when in the model a user receives a message, it receives also a stimulus of intensity 100, that is comparable to the stimulus considered in the model of nicotine addiction presented in the previous section. Moreover, when the user becomes addict to such a stimulus, that is when the memory becomes greater than a threshold level of 15, it receives an additional stimulus of intensity 150 at the time of sending a message. This additional stimulus is due to a particular aspect of the Dopaminergic System, presented by Samson et al. in [18]: in the presence of addiction, the dopamine concentration increases also when the subject waits for a stimulus to whom he is particularly sensitive. Namely, addiction causes the dopamine level to increase as a consequence of *the expectation* of a reward. This phenomenon is often referred to as *prediction error*, as it is related with withdrawal symptoms when a stimulus is expected, but then not obtained.

3.2 The experiments

We study our model on three forms of computer-mediated communication, represented as different graphs: (i) with only two nodes; (ii) with multiple nodes constituting a star-graph; and, (iii) with multiple nodes constituting a scale-free network. The 2-nodes graph allows us to better understand the role of the propensity factor in the dynamics of the Dopaminergic System. The star-graph, instead, allows us to better understand how the number of neighbors of a node influences addiction development and propagation. Finally, the scale-free networks allow us to study the role of topology and of the presence of users with high propensity factors in the spread of addiction through social networks.

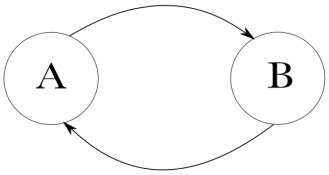
We performed several simulation experiments for each graph topology and by varying the propensity factors of users. In all experiments, the simulated time corresponds to 50 days (as in the model of nicotine addiction described in the previous section and in [10]). All the experiments have been performed by implementing the model in the Python programming language. The source code of the model is available at [1].

User A \ User B	0	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1
0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0
0.1	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=66	A=0; B=97	A=0; B=99
0.2	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=83	A=0; B=100	A=0; B=100	A=0; B=100	A=0; B=100	A=0; B=100	A=0; B=100
0.3	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=0	A=0; B=95	A=1; B=100	A=2; B=100	A=6; B=100	A=6; B=100	A=5; B=100	A=8; B=100
0.4	A=0; B=0	A=0; B=0	A=90; B=0	A=96; B=3	A=95; B=96	A=93; B=100	A=91; B=100	A=96; B=100	A=91; B=100	A=94; B=100	A=93; B=100
0.5	A=0; B=0	A=0; B=0	A=100; B=0	A=100; B=5	A=100; B=94	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100
0.6	A=0; B=0	A=0; B=0	A=100; B=0	A=100; B=6	A=100; B=96	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100
0.7	A=0; B=0	A=0; B=0	A=100; B=0	A=100; B=2	A=100; B=95	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100
0.8	A=0; B=0	A=51; B=0	A=100; B=0	A=100; B=2	A=100; B=99	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100
0.9	A=0; B=0	A=88; B=0	A=100; B=0	A=100; B=5	A=100; B=96	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100
1	A=0; B=0	A=99; B=0	A=100; B=0	A=100; B=2	A=100; B=96	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100	A=100; B=100

None □ One ▒ Both ■

Table 1: Each cell represents a simulation of the communication between two users, with a different combination of propensity factors. On the vertical axis, there are the propensities of User A, and on the horizontal axis those of User B. The reported values and the color intensity, express the number of times each users became addicted.

Communication between two users. The first network topology, depicted below, describes the communication between only two users, A and B.

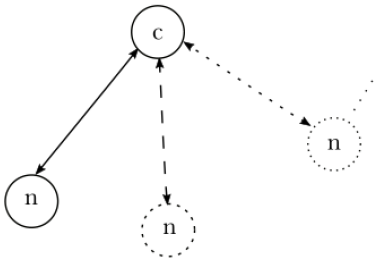


This graph is used to examine the role of the propensity factor. All the possible combinations of users have been tested, in this simple communication, to study the behaviour of the Dopaminergic System in different situations (summarised in Table 1). For each combination, we made 100 simulations and measured the number of times in which one or both users became addicted (i.e. $M \geq 15$).

These experiments allow us to identify three representative values for the propensity factor (low, medium and high). As regards low and high values we choose 0.2 and 0.9, respectively. Indeed, it can be seen that in the case of two users with propensity 0.2, none of the two becomes addict; in the case of two users with propensity 0.9, both of them become addict; finally, in the case of one user with propensity 0.2 and the other with 0.9, only one becomes addict.

To find the medium value, we performed additional simulations (results not shown) by varying the propensity factors of both users in the range [0.3, 0.4] by steps of 0.05. We repeated these simulations 500 times for each combination of parameters and, in the end, we identified 0.35 as medium value.

The star graph. This network is a particular kind of tree, in which every node n is linked to the central one c. Each node interacts only with its neighbours. So, when, for example, the node c sends a message, it is received by all peripheral nodes n; instead, when one of the peripheral nodes sends a message, it is received only by node c.



To study the propagation of addiction in a graph, we start by considering how many peripheral nodes are necessary to cause addiction of the central node c. As depicted in the figure, in the simulations, we added peripheral nodes one by one (all with the same propensity factor) until c became addict, and we counted how many peripheral nodes are necessary in order to reach such a result.

As summarised in Table 2, we used the three previously identified propensity values both for the central node c and the peripheral nodes n.

Nodes n

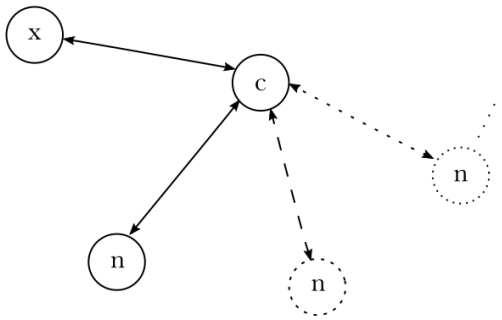
Node c	0.2	0.35	0.9
0.2	9	7	4
0.35	3	1	1
0.9	1	1	1

Few Nodes — More Nodes

Table 2: Each cell represents the number of nodes we have to add in the graph to make susceptible to the dependence the central node c. On the vertical axis and horizontal factors, there are respectively the different propensity factors of the central node and added nodes, used during the simulations.

With 0.2, we add the greater number of nodes, confirming that, with this value, the interaction is really low. After studying how addiction influences the network's central node, we study how one of the peripheral nodes x whose propensity factor is 0.2 (fixed) can be influenced by the others (both c and n).

This time, by changing the propensity factor of the central node c, we count how many nodes n we have to add to the graph until the target node x becomes addicted (as shown in the figure).



Changing the propensity factor of the added nodes n, we are able to examine different situations to notice what influences more between the central node and the added nodes. As shown in Table 3, the number of nodes to add is proportional to the propensity factor and, in particular, it is influenced by the central node, because it has a direct connection with the node x.

Nodes N

Node C	0.2	0.35	0.9
0.2	44	36	17
0.35	17	13	5
0.9	15	7	4

Few Nodes ■ More Nodes ■

Table 3: Each cell represents the number of nodes we have to add in the graph to make susceptible to the dependence the target node x. On the vertical axis and horizontal factors, there are respectively the different propensity factors of the central node and added nodes, used during the simulations.

Scale-free networks. The study of the two previous networks has allowed us to test the model and to delineate the significant values of the propensity factor.

Now, with the aim of understanding what happens in social networks, we start studying scale-free network topology.

The characteristics of scale-free networks is the presence of few nodes with a very high degree. These high-degree nodes are called *hubs* and are responsible for many phenomena in this kind of network. In particular, one of the most relevant is the robustness to random failures: information, as well as phenomena like diseases or computer viruses, spread differently on a scale-free network depending on whether the propagation started from low-degree nodes or from the hubs. In the first case, the nodes propagation is slower than the second case. This is caused by the limited role of low-degree nodes in network integrity.

Experiments. For our experiments, we used scale-free networks of 100 nodes generated in two different ways: with the Bollobás-Riordan (BR) [5] and the Barábasi-Albert (BA) [4] approaches. Both the approaches construct the network by using a preferential attachment algorithm, defined as the probability to attach a new node to one already present. Since both approaches generate scale-free networks, in both cases we have degree distributions that follow a power law, as shown in Figure 3; with the BR approach we usually obtain more hubs than with the BA approach.

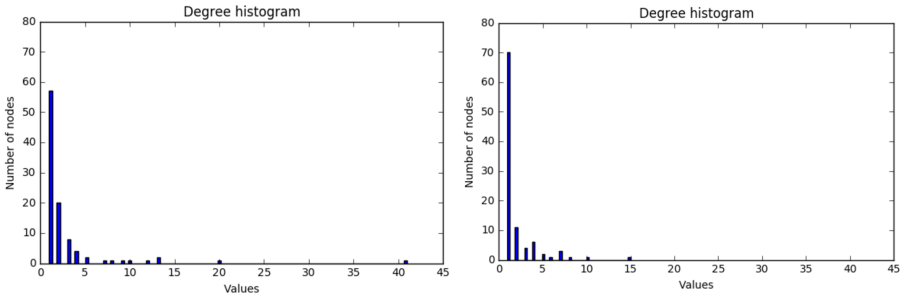


Fig. 3: Example of degree distribution of two scale-free networks generated with Bollobás-Riordan (BR), on the left, and with Barábasi-Albert (BA), on the right.

To compare the spread of addiction in the two kinds of scale-free networks, we test our model by associating different propensity factors to the users. In the first experiment, each user in the networks has a propensity factor equal to 0.2. As we can notice in the Figure 4, the propagation of the addiction is influenced by the topology of the network. Indeed, in the scale-free network generated by the BR model, there are more hubs and as a consequence we obtain more addicted nodes. To explore the difference between networks, we associated medium and high propensity factors, to particular nodes. In the first case, we made more susceptible to addiction some random nodes, to study how changes the communication and the messages sharing; in the second case we

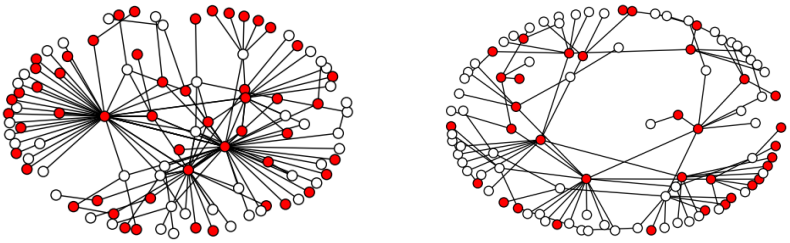


Fig. 4: On the left, there is the graph of scale-free network generated by BR; on the right, the graph of scale-free generated by BA. When all nodes have propensity equal to 0.2, 52 nodes become addicted (red color) in the first graph, 35 in the second one.

made susceptible an increasing number of hubs, to compare the result with the first experiment. How shown in Figure 5, in line with the scale-free network features, we notice that in the second case we obtain in the end a greater number of addicted users. In Figure 6 we present different scale-free networks, generated

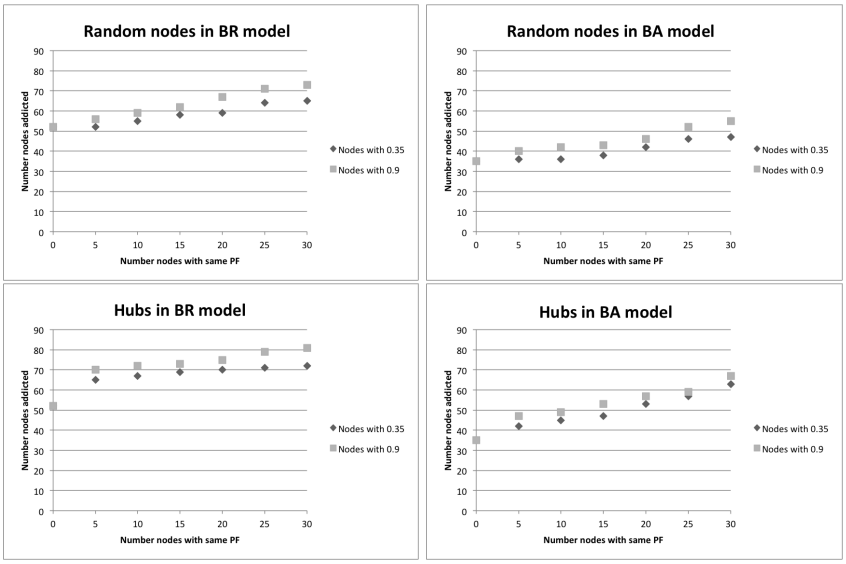


Fig. 5: The difference between the number of the addicted users in BR and BA networks, when we change the propensity factor of some of the nodes (randomly chosen on the top, chosen among hubs on the bottom). The size of the nodes in the graphs represents their propensity factors.

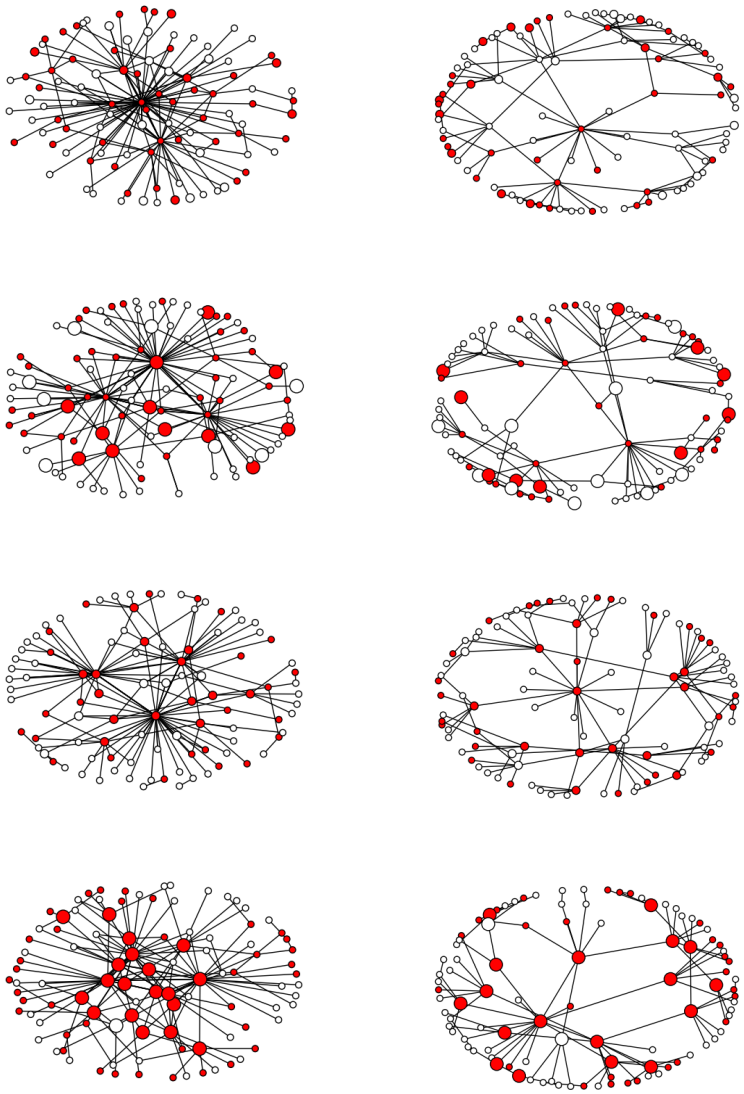


Fig. 6: We made susceptible to addiction 20 random nodes and 20 hubs, in the two scale-free models, setting their propensity factors before to 0.35 and after 0.9. On the left, BR models, on the right the BA model.

by the two algorithms, in which we change the propensity factor of 20 nodes, first randomly chosen and then chosen among hubs. In the BR model, we obtain the greater number of addicted users.

4 Conclusions

The aim of this work is to propose a computational framework for the study of addiction, taking into account both neurological and sociological characteristics. To achieve this, we decided to proceed gradually, to understand the complexity and dynamics of the phenomenon. Starting from the model proposed by Gutkin et al., we developed a simplified modular model of the Dopaminergic System, using the theory of Hybrid Automata. This allowed us to develop a hybrid system consisting of two components, the Dopaminergic System and a generator of stimuli, in order to dynamically grasp the trend of neurological activity, in relation to many environmental factors. After a general overview we decided to focus mainly on Internet Addiction because it is an unexplored phenomenon, that have many sociological and psychological implications. Linked to this, we decided to study how addiction is correlated to network topologies, testing how people in a social network can influence each other.

Our model can be further developed: in the future, we plan to explore other kinds of communication, using networks with a greater number of nodes, to better investigate different stimuli (like for example non-deterministic stimuli) and to consider non-constant propensity factors.

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