Networks for Smoking Dynamics

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1 Introduction

Over the years, multiple policies have helped considerably decrease the prevalence of smoking worldwide. The majority of these policies have been at the population level. Despite the current progress in tobacco control resulting from these policies, the rate of decline of smoking prevalence is decreasing. Therefore, there is a need for new models that can capture the system's full complexity and act as test-beds to develop better policies and control measures.

Recent network-based studies have shown that smoking is a behavioural contagion. In particular, smoking behaviour can spread through social ties and depending on the social tie, the probability of both smoking initiation and quitting changes [2, 3]. Additionally, relapsing into smoking is more likely when quitters engage with a smoker group [2]. Even though empirical research has established the contagious nature of smoking behaviour, current models do not incorporate this completely. Most of these models are Ordinary Differential Equation (ODE) models and do not account for network topology or consider all possible empirically proven interactions.

We identify the following contributions in this paper. Firstly, we develop an agent-based model (ABM), which considers both spontaneous terms and interactions between agents to study the spread of smoking. This model can be used to develop network-based intervention strategies and policies for tobacco control. Secondly, we explore the effect of the underlying network topology on smoking dynamics. We find that the underlying network structure affects smoking dynamics considerably. The ABM on the real-world network accurately replicates historical data, while the ABM on a Fully-Connected (FC) network performs much worse than other networks. Finally, we show that policymakers can use Lancichinetti-Fortunato-Radicchi (LFR) networks which have community structure embedded in them to model smoking behaviour when the actual network of the local population is unavailable.

2 Methods

Model Each agent in the ABM can be in one of the three states: never-smoker (N), smoker (S) or quitter (Q). Each of these agents forms links based on the predefined network structure. We incorporate smoking initiation ($N \to S$), cessation ($S \to Q$) and relapse ($Q \to S$) into the model. These three state-change processes can occur both spontaneously and through interactions. For example, an N-agent can initiate smoking due to the influence of its S network neighbours. Similarly, an S agent can quit smoking due to interaction with N and Q agents, while Q can relapse into smoking due to interaction with their S neighbours. The calibrated parameters are not time-dependent.

Data We calibrate (both coarse and fine-grained) and validate the model for each network on publicly available yearly S and Q prevalence data in the UK [4], and US [8].

Experiments To study the effect of network topology, we calibrate and validate the model on six different network structures. Namely, the Fully Connected network, the Barabasi–Albert (BA) model for scale-free network [1], the Erdős–Rényi (ER) model for random network [5], the Lancichinetti-Fortunato-Radicchi benchmark network [7], the Watts–Strogatz model for small-world network (SW) [9] and a real-world network generated using configuration model from the Framingham Heart Study (FHS) data [6].

3 Results

Our results suggest that the underlying network structure affects smoking dynamics. The dynamics observed change drastically when we move from an FC network to other networks. We find that the network built from the FHS data (our real-world network) most accurately replicates the empirical data in both the US and UK. At the same time, we see that the FC network consistently performs poorly in both countries.

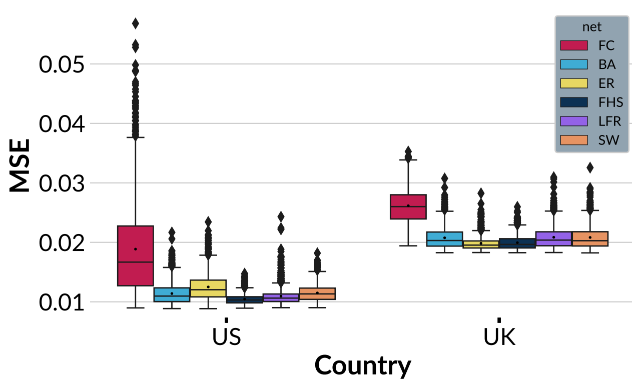

Fig. 1. The figure shows box plots of the MSE values (sum of S and Q) from 1000 runs of the ABM on all six networks for both the US and the UK. We see that the ABM replicates the US data more accurately between the two countries. At the same time, among the six networks, we see that the ABM on the FHS network reproduces the data most accurately in both countries. In contrast, the ABM on the FC network provides the worst fit for the validation data. The ABM on all other networks except FC gives MSE values very close to each other.

For the spread of smoking behaviour, the influence of other individuals is only substantial when they are a close family member or friend [3]. This limits the average degree of the network for the spread of smoking behaviour. This network will have a much lower average degree than a general social network. In such situations, the ABM on networks which have the same average-degree, give similar fits to the data. However, when we compare the best-fit parameters returned by each network, we see that the LFR network returns parameters which are not significantly different from the real-world network (FHS). LFR networks are unique because of the presence of communities embedded in their network generation process. Our results suggest that synthetic networks with communities (LFR networks) can be used to model smoking behaviour when a population's actual network is unavailable.

An ODE analogue of the ABM would demonstrate similar dynamics to the ABM on an FC network. Since the FC network gives the worst fit among all networks, we can conclude that the ODE model would give an equally bad fit. ODE models and ABM on FC networks should not be used to model smoking and similar behavioural contagion.

Summary. We have developed a network-based Agent-based model to study the spread of smoking behaviour. This model considers all possible interactions between agents which can lead to changes in their smoking behaviour. We show that the network structure of the population has an effect on the dynamics observed. Further, the network generated from real-world data best replicates the empirical data. We also show that, given practical difficulties in collecting information on the offline network topology, policymakers can use LFR networks to model smoking behaviour.

Taken together, the ABM can be used to develop network-based intervention strategies, which will prove essential in shaping the policies for tobacco control.

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