Modelling Behavioural Preferences in Epidemic Models for Sexually Transmitted Infections on Temporal Networks

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Modelling Behavioural Preferences in Epidemic Models for Sexually Transmitted Infections on Temporal Networks

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***Abstract*— In this paper, we propose a temporal model for the spreading of curable sexually transmitted infections (STIs). The model is developed within the framework of activity-driven networks, which allows to model the time-varying pattern of sexual encounters and the individuals' heterogeneity in their proclivity to initiate them. Our model explicitly includes the delay between infectiousness and symptoms onset, and individuals' behavioural preferences for the use of protection during encounters. Behavioural preferences evolve according to a nontrivial mechanism that accounts for the perceived risks, the cost of adopting protective measures, and the persuasive effect of interactions with individuals who have a different preference. In the limit of large-scale populations, we use a mean-field approach to derive the epidemic threshold and study the effect of two control measures on the spread of STIs: i) routine screening at STI clinics, and ii) condom (social) marketing campaigns. Our results reveal the important effect of routine screening for STIs, which has emerged as a key factor to favour stability of the disease-free equilibrium, while marketing campaigns can be very effective in mitigating endemic diseases.**

I. INTRODUCTION

Without treatment, sexually transmitted infections (STIs) such as chlamydia, gonorrhoea, syphilis and trichomoniasis can cause health problems among which, but not exclusively, infertility, stillbirths, cancer and chronic neurological problems [1]. Since the discovery of the treatment using penicillin in 1928, however, syphilitic facial deformities and death have predominantly become things of the past. After having rid society of such grim visual reminders of the dangers of having "one night with Venus", curable STIs have been underappreciated adversaries for decades [2], and today, they are on the rise. Each day, more than one million people become infected with a common STI [1], and in 2018, the prevalence of combined cases of gonorrhoea, chlamydia and syphilis in the United States reached an all-time high [3]. Furthermore, the current health crisis related to COVID-19 has called for the relocation of STI resources to the COVID-19 response, and additionally caused a disruption in STI services [4], the consequences of which are yet to be seen. Since STIs are often asymptomatic [5], they present a peril as a silent global epidemic, with risk for escalation lurking in the shadows of the ongoing COVID-19 pandemic. The rise in STI cases is concerning, since STIs — in particular gonorrhoea — are developing resistance to antibiotics, vastly reducing treatment options [6]. Although condoms are highly effective in preventing the spreading of STIs [7], their use is prevalent at varying degrees, and not always supported by public health authorities [8]. An understanding of the dynamics behind the current trends of risky sexual behaviour would aid in the development of effective control policies.

Mathematical models of epidemic spreading on networks are potent means to shed light on spreading of infectious diseases [9]–[12]. After analysis, the gained insights can subsequently be used to inform control strategies to impact the evolution of epidemic spreading [10]–[13]. Since real-world networks of interactions are often time-varying, temporal networks have especially emerged as effective paradigms to capture the patterns of human interactions [14]. A valuable paradigm to study real-world time-varying networks can be found in the concept of activity-driven networks (ADNs) [15]–[17]. In ADNs, an activity rate is assigned to each individual, which captures the individual's propensity to have interactions with others. Using such a network construction enables analysis of epidemic spread on heterogeneous temporal networks [13], [15]–[17].

Despite most of the literature on epidemic models on temporal networks focusing on other types of disease, such as flu or recently COVID-19, some efforts have been made to study STIs through the lens of temporal networks. In [18], a multi-layer temporal network model is used to study the impact of casual partners on the spreading of STIs. A framework to handle heterogeneities existing in contact networks can be found in [19]. In [20], a bipartite network is employed to model a heterosexual contact network.

In this paper, we propose a model for the spread of STIs, in which individuals initiate sexual interactions with others according to a stochastic mechanism governed by a continuous-time ADN. The proposed model incorporates individuals' heterogeneity and their behavioural preferences with regard to the use of protective measures when engaging in sexual contacts. We achieve this by adding extra compartments to the well-known susceptible–infected–susceptible (SIS) model, which is standardly used to model STIs such as gonorrhoea [21]. These additional compartments allow the representation of the individual behavioural preferences, and the presence of asymptomatic, unaware — but infectious — individuals. The individual's behavioural preference is determined by a trade-off decision between the perceived risk (associated with the detected prevalence of the disease) and the cost of adopting protective measures, and it may also change after an encounter with an individual who has a different preference. In our model, we implemented two measures to control the spread of infection: (i) routine screening

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for STIs at high-volume sites, which was determined to be the most effective approach to control the spreading of STIs [5]; and (ii) condom (social) marketing campaigns, whose positive effect on the likelihood to adopt protective measures has been proved through systematic studies [22].

Besides the mathematical formalisation of the model, the main contributions of this paper are given by the derivation of the epidemic threshold, by means of a mean-field analysis in the limit of large-scale populations, and the numerical analysis of the system above the epidemic threshold. Our theoretical results allow to shed light on the role of the model parameters and on the effectiveness of the two control measures on the spread of STIs. Specifically, our results suggest that routine screening of STIs is a more effective control measure than the introduction of condom marketing campaigns. The simulation results extend our theoretical findings beyond the limitations of our analysis and suggest that condom marketing campaigns may be critical to mitigating endemic diseases.

The rest of the paper is organised as follows. In Section II, we formulate the network model for STIs. In Section III, we derive the dynamical system. In Section IV, we present our main results. Section V concludes the paper and outlines future research.

Here, we define some notation used throughout this paper. The set of real, real nonnegative, and strictly positive real numbers is denoted by $\mathbb{R}$, $\mathbb{R}_{\geq 0}$, and $\mathbb{R}_{>0}$, respectively. Given a function $x(t)$ with $t \in \mathbb{R}_{\geq 0}$, we define $x(t^+) = \lim_{s \searrow t} x(s)$, and $x(t^-) = \lim_{s \nearrow t} x(s)$.

II. Model

We consider a population of n individuals $\mathcal{V} = \{1, \dots, n\}$. Sexual interactions are modelled by a directed temporal network $(\mathcal{V}, \mathcal{E}(t))$, which evolves in continuous time $t \in \mathbb{R}_{\geq 0}$. The nodes represent the individuals and $\mathcal{E}(t)$ is the time-varying set of directed links, in which the directed link $(j,k) \in \mathcal{E}(t)$ if and only if individual j initiates a sexual encounter with player k at time t.

A. Activity Driven Networks

Interactions are generated according to a mechanism inspired by continuous-time ADNs [16]. Specifically, each individual $j \in \mathcal{V}$ is characterised by a parameter $a_j \in \mathbb{R}_{>0}$ called *activity*, which models j's tendency to initiate a sexual encounter. Interactions are generated in a probabilistic fashion. Each individual j is equipped with a Poisson point process (to which we refer as a clock) with the rate a_j, independent of the others, as in [16]. When a clock clicks, the individual associated to it activates and initiates a sexual encounter with another member of the population, selected uniformly at random in the network. The network formation process is summarised in the following algorithm: i) at time $t = 0$, $\mathcal{E}(t) = \emptyset$; ii) if at time $t \in \mathbb{R}_{>0}$, the Poisson clock associated with individual $j \in \mathcal{V}$ ticks, then j activates and selects an individual $k \in \mathcal{V} \setminus \{j\}$ uniformly at random. Instantaneously, the directed link is (j,k) is added to $\mathcal{E}(t)$; iii) Immediately afterwards, (j,k) is removed from $\mathcal{E}(t)$, the Poisson clock associated with individual j is re-initialised, and the algorithm resumes from point ii).

B. Epidemic Model

We consider an extension of the classical network SIS model [23], where we introduce extra compartments to account for behavioural preferences with regard to the use of protective measures during sexual interactions, and for detected infected individuals that are receiving treatment. Each individual $j \in \mathcal{V}$ is characterised by a state $X_j(t) \in \{\mathrm{S_r}, \mathrm{S_p}, \mathrm{I_r}, \mathrm{I_p}, \mathrm{I_t}\}$ at time t, reflecting their behavioural preference and health state. In particular,

$$X_j(t) = \begin{cases} \mathrm{S_r} & \text{if } j \text{ is } \textit{susceptible} \text{ and prefers to not use protective measures } (\textit{risky}), \\ \mathrm{S_p} & \text{if } j \text{ is } \textit{susceptible} \text{ and prefers to use } \textit{protective} \text{ measures}, \\ \mathrm{I_r} & \text{if } j \text{ is } \textit{infected}, \text{ asymptomatic, and prefers to not use protective measures } (\textit{risky}), \\ \mathrm{I_p} & \text{if } j \text{ is } \textit{infected}, \text{ asymptomatic, and prefers to use } \textit{protective} \text{ measures}, \\ \mathrm{I_t} & \text{if } j \text{ is } \textit{infected}, \text{ symptomatic, and receiving } \textit{treatment}, \end{cases}$$

at time t. Here, we make the simplifying assumption that once infected individuals become symptomatic, they are immediately diagnosed and receive treatment. Note that asymptomatic infected individuals are unaware of their health condition, and thus exhibit the same behavioural patterns as susceptible individuals.

The infection spreads through pairwise interactions between susceptible and infected individuals, which occur according to the following mechanism. If $(j,k) \in \mathcal{E}(t)$, and none of the individuals is receiving treatment (i.e. $X_j(t) \neq \mathrm{I_t}$ and $X_k(t) \neq \mathrm{I_t}$), then individual j proposes a sexual encounter to individual k. Depending on individual j's behavioural preference, j proposes to use protective measures (if $X_j(t) \in \{\mathrm{S_p}, \mathrm{I_p}\}$), or not (if $X_j(t) \in \{\mathrm{S_r}, \mathrm{I_r}\}$). If individual k has the same behavioural preference, then we assume that k always accepts. If individual k's behavioural preference differs, then the encounter is accepted with probability $\sigma \in [0,1]$ (see Fig. 1a), where σ is the individuals' degree of amenability.[1] If k accepts, then the behavioural preference of k changes accordingly, consistent with the literature on *decision inertia*, i.e. people's tendency to repeat past decisions [24], [25]. The state of an individual $j \in \mathcal{V}$ evolves over time according to the following processes.

Play safe: Besides the behavioural changes due to an encounter with an individual that wants to use protective measures, an individual may start favouring protective behaviour as a response to the epidemic spreading. To model this, each individual that has a non-protective tendency $(X_j(t^-) \in \{\mathrm{S_r}, \mathrm{I_r}\})$ starts to desire the adoption of protective

[1] Note that different degrees of amenability among the individuals may be defined to capture behavioural heterogeneities in the population.

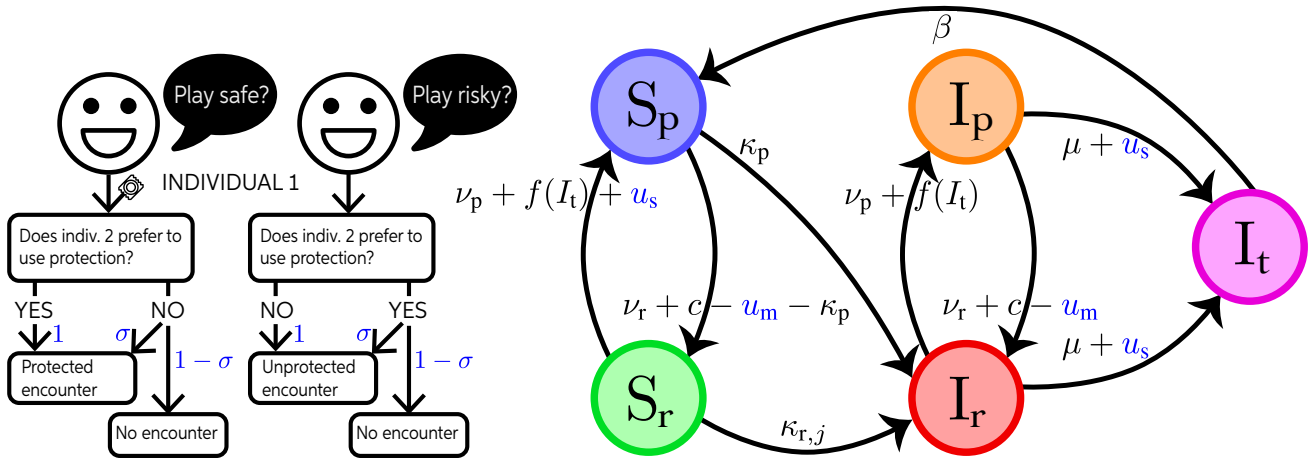


Fig. 1: (a) A flow chart of the process of proposal and acceptance; (b) State transitions of the epidemic model for $j \in \mathcal{V}$.

behaviour ($X_j(t^+) \in \{\mathrm{S_p}, \mathrm{I_p}\}$) according to a Poisson clock with rate equal to $f(I_\mathrm{t})$. Here, $I_\mathrm{t}(t)$ denotes the detected prevalence of the disease at time t, i.e. $I_\mathrm{t}(t) := \frac{1}{n}\big|\{j \in \mathcal{V} \mid X_j(t) = \mathrm{I_t}\}\big|$, and $f(I_\mathrm{t}(t)) : [0,1] \to \mathbb{R}_{\geq 0}$ is a monotonically non-decreasing risk perception function, which represents the incentive to adopt self-protective behaviours due to fear arising when the disease spreads within the population.

Play risky: Apart from the behavioural changes due to an encounter with an individual that does not want to use protective measures, an individual having a protective tendency ($X_j(t^-) \in \{\mathrm{S_p}, \mathrm{I_p}\}$) may spontaneously lose the desire to protect ($X_j(t^+) \in \{\mathrm{S_r}, I_\mathrm{r}\}$) due to the costs of using protections. In the model, we introduce a Poisson clock with rate c_j, associated with each individual that has a protective tendency, where $c_j \in \mathbb{R}_{>0}$ captures the cost of using protection: this includes not only the cost of condoms, but also the potential personal reluctance in using them. For the sake of simplicity, we assume that $c_j = c \in \mathbb{R}_{>0}$ for all $j \in \mathcal{V}$.[2]

Contagion: If a susceptible individual ($X_j(t) \in \{\mathrm{S_r}, \mathrm{S_p}\}$) does not use protective measures during an encounter with an infected individual k ($X_k(t) \in \{\mathrm{I_r}, \mathrm{I_p}\}$), individual j becomes infected with probability $\lambda \in [0,1]$.

Symptoms onset: An asymptomatic infected individual ($X_j(t^-) \in \{\mathrm{I_r}, \mathrm{I_p}\}$) spontaneously develops symptoms according to a Poisson clock with rate $\mu \in \mathbb{R}_{>0}$. We assume that symptomatic individuals become aware and immediately start treatment ($X_j(t^+) = \mathrm{I_t}$).

Recovery: An infected individual undergoing treatment ($X_j(t^-) = \mathrm{I_t}$) spontaneously recovers according to a Poisson clock with rate $\beta \in \mathbb{R}_{>0}$. We assume that after recovery, the individual adopts protective behaviour ($X_j(t^+) = \mathrm{S_p}$).

C. Control

We introduce two measures to control the spreading of STIs.

[2]Note that different costs among individuals may be used to capture heterogeneities in the population with respect to the perceived cost for using protections, which may be influenced by wealth status, social stigmas, religious interdicts, and prevailing moral norms [26].

Routine screening at STI clinics: We examine what happens if the government provides routine screening at clinics, and free condoms after the STI tests. By providing free routine tests, individuals without symptoms are induced to get tested. This is implemented in the modelling framework by introducing an additional Poisson clock with rate $u_\mathrm{s} \in \mathbb{R}_{>0}$, which represents the rate at which STI screening takes place. Hence, asymptomatic infected individuals ($X_j(t^-) \in \{\mathrm{I_r}, \mathrm{I_p}\}$) receive diagnosis and treatment ($X_j(t^+) = \mathrm{I_t}$) according to a Poisson clock with rate u_s. Susceptible individuals ($X_j(t^-) \in \{\mathrm{S_r}, \mathrm{S_p}\}$) get tested according to a Poisson clock with rate u_s. We assume that, as a consequence of the reminder of the dangers of STIs, they adopt a behavioural preference for the use of protective measures after receiving the negative diagnosis ($X_j(t^+) = \mathrm{S_p}$).

Condom marketing campaigns: Due to marketing programmes, individuals are stimulated to use protection during intercourse. The effect of such schemes is incorporated in the model by reducing their cost (in the "play risky" mechanism) by $u_\mathrm{m} \in [0, c]$, that is, from c to $c - u_\mathrm{m}$.

III. DYNAMICS

Since all the mechanisms in the dynamics are triggered by Poisson clocks, each one independent of the others, the system's state $X(t)$ evolves according to a continuous-time n-dimensional Markov process in the state space $\{\mathrm{S_r}, \mathrm{S_p}, \mathrm{I_r}, \mathrm{I_p}, \mathrm{I_t}\}^n$. As illustrated in Fig. 1b, the generic j^{th} entry of the vector $X(t)$ can undergo nine different state transitions. Three of them —namely, the ones from $\mathrm{I_p}$ and $\mathrm{I_r}$ to $\mathrm{I_t}$, and the one from $\mathrm{I_t}$ to $\mathrm{S_p}$— are just triggered by spontaneous mechanisms. Hence, the corresponding transition rates are simply equal to the sum of the rates of the Poisson clocks involved in the process. The other six transitions, however, involve mechanisms that are triggered by mechanisms involving interactions between individuals. These mechanisms are the contagion process, and the behavioural changes due to persuasion. In the following, we derive the expressions for the rates of these mechanisms and, consequently, we compute the six transition rates that involve them.

Contagion: If $X_j(t^-) = \mathrm{S_r}$, j contracts the disease ($X_j(t^+) = \mathrm{I_r}$) according to a Poisson clock with rate

$$\kappa_{\mathrm{r},j} := \frac{\lambda}{n-1}\left[a_j\left[\sum_{k:X_k=\mathrm{I_r}} 1 + \sigma\cdot\sum_{k:X_k=\mathrm{I_p}} 1\right] + \sum_{k:X_k=\mathrm{I_r}} a_k\right].$$

If $X_j(t^-) = \mathrm{S_p}$, individual j becomes infected ($X_j(t^+) = \mathrm{I_r}$) according to a Poisson clock with rate

$$\kappa_{\mathrm{p}} := \frac{\lambda\sigma}{n-1}\sum_{k:X_k=\mathrm{I_r}} a_k.$$

Play safe: If $X_j(t^-) \in \{\mathrm{S_r}, \mathrm{I_r}\}$, j is persuaded by k to start desiring the use of protective measures ($X_j(t^+) \in \{\mathrm{S_p}, \mathrm{I_p}\}$) according to a Poisson clock with rate

$$\nu_{\mathrm{p}} := \frac{\sigma}{n-1}\sum_{k:X_k\in\{\mathrm{S_p},\mathrm{I_p}\}} a_k.$$

Play risky: If $X_j(t) \in \{\mathrm{S_p}, \mathrm{I_p}\}$, j is persuaded by k to no longer covet the use of protective measures ($X_j(t^+) \in \{\mathrm{S_r}, \mathrm{I_r}\}$) according to a Poisson clock with rate

$$\nu_{\mathrm{r}} := \frac{\sigma}{n-1}\sum_{k:X_k\in\{\mathrm{S_r},\mathrm{I_r}\}} a_k.$$

Hence, the overall transition rate from state $\mathrm{S_r}$ to state $\mathrm{S_p}$ is equal to the sum of three contributions: the persuasion effect ν_{p}, the risk perception $f(I_{\mathrm{t}})$, and the control effort placed in routine screening u_{s}. Similarly, the overall transition rate from state $\mathrm{I_r}$ to state $\mathrm{I_p}$ is equal to the sum of the first two contributions, since an infected individual that undergoes a routine screening would test positive and transition to the diagnosed compartment $\mathrm{I_t}$. Next, the overall transition rate from state $\mathrm{I_p}$ to $\mathrm{I_r}$ is equal to the sum of the persuasion effect ν_{r} and the cost of using protective measures c, minus the control effort placed in marketing campaigns u_{m}. The overall transition rate from state $\mathrm{S_p}$ to $\mathrm{S_r}$ equals the latter, but additionally contains the term $-\kappa_{\mathrm{p}}$, due to contagion taking place. The transition rate matrix for the generic i^{th} component of the Markov process $X(t)$ is given by

$$Q_j = \begin{bmatrix} \cdot & \nu_{\mathrm{p}} + f(I_{\mathrm{t}}) + u_{\mathrm{s}} & \kappa_{\mathrm{r},j} & 0 & 0 \\ \nu_{\mathrm{r}} + c - u_{\mathrm{m}} - \kappa_{\mathrm{p}} & \cdot & \kappa_{\mathrm{p}} & 0 & 0 \\ 0 & 0 & \cdot & \nu_{\mathrm{p}} + f(I_{\mathrm{t}}) & \mu + u_{\mathrm{s}} \\ 0 & 0 & \nu_{\mathrm{r}} + c - u_{\mathrm{m}} & \cdot & \mu + u_{\mathrm{s}} \\ 0 & \beta & 0 & 0 & \cdot \end{bmatrix},$$

where the rows (columns) correspond to state $\mathrm{S_r}, \mathrm{S_p}, \mathrm{I_r}, \mathrm{I_p}$, and $\mathrm{I_t}$, respectively. The diagonal elements equal the opposite of the sum of the other row elements, to ensure that each row of Q_j sums to zero. For any $h, k \in \{\mathrm{S_r}, \mathrm{S_p}, \mathrm{I_r}, \mathrm{I_p}, \mathrm{I_t}\}$ with $h \neq k$,

$$\mathbb{P}\left[X_j(t+\Delta t) = k | X_j(t) = h\right] = (Q_j)_{hk}\Delta t + o\left(\Delta t\right),$$

as $\Delta t \to 0$. Note that Q_j is dependent on the state of the other nodes, and that the dimension of the state space grows exponentially with n. This complicates the direct analysis of the Markov process $X(t)$ for large-scale populations. Hence, as in [13], [27], we study the following continuous-state deterministic mean-field relaxation of the stochastic system. For all $j \in \mathcal{V}$, we consider the probabilities $s_{\mathrm{r},j}(t) := \mathbb{P}\left[X_j(t) = \mathrm{S_r}\right]$, $s_{\mathrm{p},j}(t) := \mathbb{P}\left[X_j(t) = \mathrm{S_p}\right]$, $i_{\mathrm{r},j}(t) := \mathbb{P}\left[X_j(t) = \mathrm{I_r}\right]$, $i_{\mathrm{p},j}(t) := \mathbb{P}\left[X_j(t) = \mathrm{I_p}\right]$, and $i_{\mathrm{t},j}(t) := \mathbb{P}\left[X_j(t) = \mathrm{I_t}\right]$. For all $j \in \mathcal{V}$, in the mean-field relaxation, the probabilities are governed by the set of differential equations $\left(\dot{s}_{\mathrm{r},j}\ \dot{s}_{\mathrm{p},j}\ \dot{i}_{\mathrm{r},j}\ \dot{i}_{\mathrm{p},j}\ \dot{i}_{\mathrm{t},j}\right) = \left(s_{\mathrm{r},j}\ s_{\mathrm{p},j}\ i_{\mathrm{r},j}\ i_{\mathrm{p},j}\ i_{\mathrm{t},j}\right)Q_j$, or equivalently

$$\begin{aligned}
\dot{s}_{\mathrm{r},j} =& -\Big(\gamma_{\mathrm{p},j} + f(I_{\mathrm{t}}) + u_{\mathrm{s}} + \frac{\lambda}{n-1}\sum_{\substack{k\in\mathcal{V}\\k\neq j}} a_k i_{\mathrm{r},k}\Big)s_{\mathrm{r},j} \\
&- \Big(a_j\frac{\lambda}{n-1}\sum_{\substack{k\in\mathcal{V}\\k\neq j}}\left(i_{\mathrm{r},k} + \sigma i_{\mathrm{p},k}\right)\Big)s_{\mathrm{r},j} \\
&+ \Big(\frac{\sigma}{n-1}\sum_{\substack{k\in\mathcal{V}\\k\neq j}} a_k\left(s_{\mathrm{r},k} + (1-\lambda)i_{\mathrm{r},k}\right) + c - u_{\mathrm{m}}\Big)s_{\mathrm{p},j}, \\
\dot{s}_{\mathrm{p},j} =& \left(\gamma_{\mathrm{p},j} + f(I_{\mathrm{t}}) + u_{\mathrm{s}}\right)s_{\mathrm{r},j} - \left(\gamma_{\mathrm{r},j} + c - u_{\mathrm{m}}\right)s_{\mathrm{p},j} + \beta i_{\mathrm{t},j}, \\
\dot{i}_{\mathrm{r},j} =& \frac{\lambda}{n-1}\Big(\sum_{\substack{k\in\mathcal{V}\\k\neq j}} a_k i_{\mathrm{r},k} + a_j\sum_{\substack{k\in\mathcal{V}\\k\neq j}}\left(i_{\mathrm{r},k} + \sigma i_{\mathrm{p},k}\right)\Big)s_{\mathrm{r},j} \\
&+ \frac{\lambda\sigma}{n-1}\Big(\sum_{\substack{k\in\mathcal{V}\\k\neq j}} a_k i_{\mathrm{r},k}\Big)s_{\mathrm{p},j} + \left(\gamma_{\mathrm{r},j} + c - u_{\mathrm{m}}\right)i_{\mathrm{p},j} \\
&- \left(\gamma_{\mathrm{p},j} + f(I_{\mathrm{t}}) + u_{\mathrm{s}} + \mu\right)i_{\mathrm{r},j}, \\
\dot{i}_{\mathrm{p},j} =& \left(\gamma_{\mathrm{p},j} + f(I_{\mathrm{t}})\right)i_{\mathrm{r},j} - \left(\gamma_{\mathrm{r},j} + c - u_{\mathrm{m}} + \mu + u_{\mathrm{s}}\right)i_{\mathrm{p},j}, \\
\dot{i}_{\mathrm{t},j} =& \left(\mu + u_{\mathrm{s}}\right)i_{\mathrm{r},j} + \left(\mu + u_{\mathrm{s}}\right)i_{\mathrm{p},j} - \beta i_{\mathrm{t},j},
\end{aligned} \tag{1}$$

where we use the notation $\gamma_{\mathrm{r},j} := \frac{\sigma}{n-1}\sum_{\substack{k\in\mathcal{V}\\k\neq j}} a_k\left(s_{\mathrm{r},k} + i_{\mathrm{r},k}\right)$, and $\gamma_{\mathrm{p},j} := \frac{\sigma}{n-1}\sum_{\substack{k\in\mathcal{V}\\k\neq j}} a_k\left(s_{\mathrm{p},k} + i_{\mathrm{p},k}\right)$, for the sake of readability.

IV. MAIN RESULTS

In this section, we perform the theoretical and numerical analysis of the dynamical system in (1), to shed light on the effect of the two control actions on the epidemic spreading. We start by showing that $\left(s_{\mathrm{r},j}(t)\ s_{\mathrm{p},j}(t)\ i_{\mathrm{r},j}(t)\ i_{\mathrm{p},j}(t)\ i_{\mathrm{t},j}(t)\right)$ governed by (1) is a probability vector for all $j \in \mathcal{V}$ and for all $t \in \mathbb{R}_{\geq 0}$.

Lemma 1. *For all $j \in \mathcal{V}$, the set $\{(s_{\mathrm{r},j}\ s_{\mathrm{p},j}\ i_{\mathrm{r},j}\ i_{\mathrm{p},j}\ i_{\mathrm{t},j}) : s_{\mathrm{r},j}, s_{\mathrm{p},j}, i_{\mathrm{r},j}, i_{\mathrm{p},j}, i_{\mathrm{t},j} \geq 0, s_{\mathrm{r},j} + s_{\mathrm{p},j} + i_{\mathrm{r},j} + i_{\mathrm{p},j} + i_{\mathrm{t},j} = 1\}$ is positive invariant under* (1).

Proof. Note that $\dot{s}_{\mathrm{r},j} + \dot{s}_{\mathrm{p},j} + \dot{i}_{\mathrm{r},j} + \dot{i}_{\mathrm{p},j} + \dot{i}_{\mathrm{t},j} = 0$, for all $j \in \mathcal{V}$, so $s_{\mathrm{r},j} + s_{\mathrm{p},j} + i_{\mathrm{r},j} + i_{\mathrm{p},j} + i_{\mathrm{t},j} = 1$ for all $t \in \mathbb{R}_{\geq 0}$. Next, note that if one of the variables governed by (1) equals zero, then its respective time-derivative is non-negative. This implies that $s_{\mathrm{r},j}, s_{\mathrm{p},j}, i_{\mathrm{r},j}, i_{\mathrm{p},j}, i_{\mathrm{t},j} \geq 0$ for all $t \in \mathbb{R}_{\geq 0}$. □

Lemma 1 entails that system (1) consists of $4n$ linearly independent differential equations. Before stating our results, let us introduce some notation. We define $\alpha_1 := \frac{1}{n}\sum_{j\in\mathcal{V}} a_j$, and $\alpha_2 := \frac{1}{n}\sum_{j\in\mathcal{V}} a_j^2$, that is, the mean and the second moment of the activity distribution, respectively. Next, the average probability for a randomly selected individual to be in state $\mathrm{S_r}$, $\mathrm{S_p}$, $\mathrm{I_r}$, $\mathrm{I_p}$, and $\mathrm{I_t}$ is given by $y_{\mathrm{s,r}} := \frac{1}{n}\sum_{j\in\mathcal{V}} s_{\mathrm{r},j}$, $y_{\mathrm{s,p}} := \frac{1}{n}\sum_{j\in\mathcal{V}} s_{\mathrm{p},j}$, $y_{\mathrm{i,r}} := \frac{1}{n}\sum_{j\in\mathcal{V}} i_{\mathrm{r},j}$,

$y_{\mathrm{i,p}} := \frac{1}{n}\sum_{j\in\mathcal{V}} i_{\mathrm{p},j}$, and $y_{\mathrm{i,t}} := \frac{1}{n}\sum_{j\in\mathcal{V}} i_{\mathrm{t},j}$, respectively. The prevalence of the states can be arbitrarily closely approximated by the average probabilities for any finite time-horizon [17], [28] by considering a sufficiently large population, i.e. $I_{\mathrm{t}}(t) \approx y_{\mathrm{i,t}}$, $S_{\mathrm{r}}(t) := \frac{1}{n}|\{j \in \mathcal{V} | X_j(t) = \mathrm{S_r}\}| \approx y_{\mathrm{s,r}}$, $S_{\mathrm{p}}(t) := \frac{1}{n}|\{j \in \mathcal{V} | X_j(t) = \mathrm{S_p}\}| \approx y_{\mathrm{s,p}}$, $I_{\mathrm{p}}(t) := \frac{1}{n}|\{j \in \mathcal{V} | X_j(t) = \mathrm{I_p}\}| \approx y_{\mathrm{i,p}}$, and $I_{\mathrm{r}}(t) := \frac{1}{n}|\{j \in \mathcal{V} | X_j(t) = \mathrm{I_r}\}| \approx y_{\mathrm{i,r}}$ if n is sufficiently large (see Fig. 2). In view of the above, we will henceforth study the behaviour of large populations using the average probabilities. Specifically, we wish to investigate whether there exist conditions that prevent the escalation of a local outbreak of the disease into a pandemic. Formally, we desire to acquire the conditions necessary for a (local) asymptotically stable disease-free equilibrium. Close to the disease-free equilibrium, it is reasonable to assume that the risk perception function is linear in the detected prevalence.

Assumption 1. *We assume that $f(I_{\mathrm{t}}) = \zeta I_{\mathrm{t}}$, with $\zeta \in \mathbb{R}_{>0}$.*

The following theorem presents conditions required for (local) asymptotic stability of the disease-free equilibrium, i.e. the *epidemic threshold*, for system (1). In our results, we present the threshold as a critical value for the parameter μ. If μ is larger than the critical value μ^*, then the epidemic is quickly eradicated; otherwise, it becomes endemic. Note that a negative value of μ^* implies that the epidemic is quickly eradicated, since $\mu \in \mathbb{R}_{>0}$.

Theorem 1. *Consider the behavioural SIS model* (1) *under Assumption 1. Then, in the thermodynamic limit of large scale systems $n \to \infty$, the following hold:*

(i) For $u_{\mathrm{s}} \ll c - u_{\mathrm{m}}$, the epidemic threshold is equal to

$$\mu_1^* := \lambda\left(\alpha_1 + \sqrt{\alpha_2}\right) - u_{\mathrm{s}}. \tag{2}$$

If $\mu > \mu_1^*$, the disease-free equilibrium (with $y_{\mathrm{i,r}} = y_{\mathrm{i,p}} = y_{\mathrm{i,t}} = 0$) is locally asymptotically stable.

(ii) For $u_{\mathrm{s}} \gg c - u_{\mathrm{m}}$, the epidemic threshold is equal to

$$\mu_2^* := -u_{\mathrm{s}} - \tfrac{1}{2}\left(\sigma\alpha_1(1-\lambda) + c - u_{\mathrm{m}}\right) + \tfrac{1}{2}\sqrt{(\sigma\alpha_1(1-\lambda) + c - u_{\mathrm{m}})^2 + 4\lambda\sigma\alpha_1(c - u_{\mathrm{m}})}. \tag{3}$$

If $\mu > \mu_2^*$, the disease-free equilibrium (with $y_{\mathrm{i,r}} = y_{\mathrm{i,p}} = y_{\mathrm{i,t}} = 0$) is locally asymptotically stable.

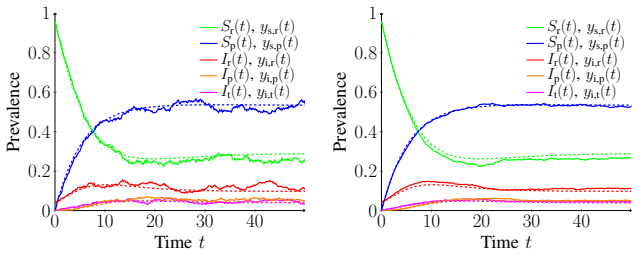
Proof. It can be observed from (1) that the unique disease-free equilibrium ($y_{\mathrm{i,r}} = y_{\mathrm{i,p}} = y_{\mathrm{i,t}} = 0$) is given by

$$(s_{\mathrm{r},j}, s_{\mathrm{p},j}, i_{\mathrm{r},j}, i_{\mathrm{p},j}, i_{\mathrm{t},j}) = \left(\frac{c - u_{\mathrm{m}}}{u_{\mathrm{s}} + c - u_{\mathrm{m}}}, \frac{u_{\mathrm{s}}}{u_{\mathrm{s}} + c - u_{\mathrm{m}}}, 0, 0, 0\right), \tag{4}$$

for all $j \in \mathcal{V}$. Equilibrium (4) is always globally asymptotically stable on the disease-free manifold $i_{\mathrm{r},j} = i_{\mathrm{p},j} = i_{\mathrm{t},j} = 0$ for all $j \in \mathcal{V}$. In order to study its local stability, we define $q_j := s_{\mathrm{p},j} - \frac{u_{\mathrm{s}}}{u_{\mathrm{s}}+c-u_{\mathrm{m}}}$, $y_{\mathrm{q}} := \frac{1}{n}\sum_{j\in\mathcal{V}} q_j$, $z_{\mathrm{q}} := \frac{1}{n}\sum_{j\in\mathcal{V}} a_j q_j$, $z_{\mathrm{i,r}} := \frac{1}{n}\sum_{j\in\mathcal{V}} a_j i_{\mathrm{r},j}$, $z_{\mathrm{i,p}} := \frac{1}{n}\sum_{j\in\mathcal{V}} a_j i_{\mathrm{p},j}$, and $z_{\mathrm{i,t}} := \frac{1}{n}\sum_{j\in\mathcal{V}} a_j i_{\mathrm{t},j}$. Let $x = \begin{pmatrix} y_{\mathrm{q}} & y_{\mathrm{i,r}} & y_{\mathrm{i,p}} & y_{\mathrm{i,t}} & z_{\mathrm{q}} & z_{\mathrm{i,r}} & z_{\mathrm{i,p}} & z_{\mathrm{i,t}} \end{pmatrix}^\top$. Linearizing system (1) about the origin yields a set of differential equations, which can be written as $\dot{x}(t) = Ax(t)$, with $A \in \mathbb{R}^{8\times 8}$. Equilibrium (4) is (locally) asymptotically stable if and only if all eigenvalues λ of A satisfy $\mathrm{Re}(\lambda) < 0$.

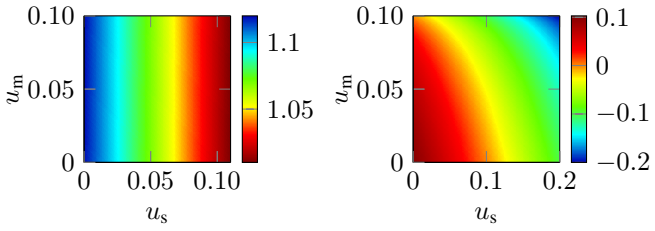
(i) Consider the case $u_{\mathrm{s}} \ll c - u_{\mathrm{m}}$, for which $\frac{u_{\mathrm{s}}}{u_{\mathrm{s}}+c-u_{\mathrm{m}}} \approx 0$. The eigenvalues of A are approximated by $-(\sigma\alpha_1 + c - u_{\mathrm{m}} + u_{\mathrm{s}}) < 0$, $-(\sigma\alpha_1 + c - u_{\mathrm{m}} + u_{\mathrm{s}} + \mu) < 0$ (with multiplicity 2), $-(c - u_{\mathrm{m}} + u_{\mathrm{s}}) < 0$, $-\beta < 0$ (with multiplicity 2), and $\lambda\left(\alpha_1 \pm \sqrt{\alpha_2}\right) - u_{\mathrm{s}} - \mu$. From the latter, we conclude that the largest eigenvalue is negative if and only if $\mu > \mu_1^*$.

(ii) Next, consider the case $u_{\mathrm{s}} \gg c - u_{\mathrm{m}}$. Since $\frac{u_{\mathrm{s}}}{u_{\mathrm{s}}+c-u_{\mathrm{m}}} \approx 1$, the eigenvalues of A are approximated by $-(\sigma\alpha_1 + c - u_{\mathrm{m}} + u_{\mathrm{s}}) < 0$, $-(c - u_{\mathrm{m}} + u_{\mathrm{s}}) < 0$, $-\beta < 0$ (with multiplicity 2), $-(\mu + u_{\mathrm{s}}) < 0$, $-(c - u_{\mathrm{m}} + \mu + u_{\mathrm{s}} + \sigma\alpha_1) < 0$, and $-\left[\mu + u_{\mathrm{s}} + \frac{1}{2}\left(\sigma\alpha_1(1-\lambda) + c - u_{\mathrm{m}}\right)\right] \pm \frac{1}{2}\sqrt{\left(\sigma\alpha_1(1-\lambda) + c - u_{\mathrm{m}}\right)^2 + 4\lambda\sigma\alpha_1(c - u_{\mathrm{m}})}$. We conclude that the largest eigenvalue is negative if and only if $\mu > \mu_2^*$. □



(a) $n = 1000$ (b) $n = 10000$

Fig. 2: Simulations of the extended SIS-model (solid curves) and its deterministic approximation (dashed curves). The model parameters are $\lambda = 0.8$, $\sigma = 0.5$, $u_{\mathrm{s}} = 0.1$, $c = 0.4$, $u_{\mathrm{m}} = 0.3$, $\mu = 0.04$, $\beta = 0.5$, and $a_j = 0.3$ for all $j \in \mathcal{V}$.



(a) $u_{\mathrm{s}} \ll c - u_{\mathrm{m}}$ (b) $u_{\mathrm{s}} \gg c - u_{\mathrm{m}}$

Fig. 3: The epidemic threshold for different values of the control effort put in routine screening u_{s} and the control effort put in marketing campaigns u_{m} in the two limit cases considered in Theorem 1. The model parameters are $\lambda = 0.8$, $\sigma = 0.5$, $c = 0.1$, and $a_j = 0.7$ for all $j \in \mathcal{V}$.

Remark 1. *Note that for $u_{\mathrm{s}} = 0$, the threshold* (2) *reduces to the threshold for an uncontrolled SIS model on ADNs, which is given by $\frac{\lambda}{\mu} < \frac{1}{\alpha_1 + \sqrt{\alpha_2}}$ [16].*

In a scenario in which the effective cost of using protection is dominant, i.e. when $c - u_{\mathrm{m}} \gg u_{\mathrm{s}}$, the epidemic threshold is monotonically decreasing in u_{s}, as shown in Fig. 3a. In this scenario, it is possible to establish a critical value of control effort placed in screening practices $u_{\mathrm{s}}^* = \lambda\left(\alpha_1 + \sqrt{\alpha_2}\right) - \mu$, such that, if $u_{\mathrm{s}} > u_{\mathrm{s}}^*$, then possible STIs outbreaks are always

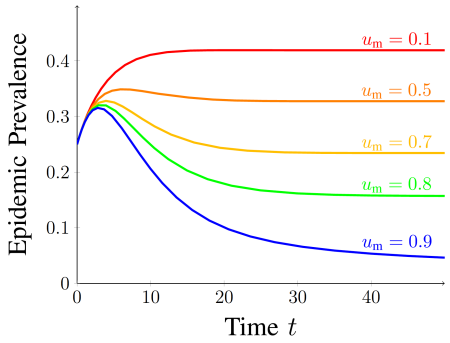


Fig. 4: Simulation of the deterministic approximation of the endemic prevalence $y_{i,r}(t)+y_{i,p}(t)+y_{i,t}(t)$ for different values of u_m. The model parameters are $\lambda = 0.8$, $\sigma = 0.5$, $u_s = 0.15$, $c = 1$, $\mu = 0.04$, $\beta = 0.5$, and $a_j = 0.3$ for all $j \in \mathcal{V}$.

eradicated at their inception. We observe from (2) that the control action u_m has no effect on the epidemic threshold. When the efforts put in the routine screenings are sensibly larger than the effective cost of using self protection, that is, when $u_s \gg c - u_m$, the epidemic threshold is monotonically decreasing with respect to the control actions u_s and u_m, as shown in Fig. 3b.

Our results suggest that during the initial phase of the epidemics, routine screening is the most effective control measure, capable of stopping the disease from becoming endemic. Numerical simulations suggest, however, that condom marketing campaigns are key to mitigating endemic STIs, sensibly reducing their prevalence (see Fig. 4).

V. CONCLUSION

We proposed a model on ADNs for the spread of STIs, which incorporates behavioural preferences for the use of protective measures during sexual intercourse, and a risk perception function based on the detected infection prevalence. By means of a mean-field analysis in the limit of large-scale populations, we established some preliminary results on the epidemic threshold, and studied the impact of routine screening at STI clinics, and condom (social) marketing programmes. Our preliminary findings suggest that, whereas routine screening is key to avoid outbreaks, condom marketing campaigns become very important in managing and mitigating endemic diseases, allowing to effectively decrease the endemic prevalence of STIs. As a next step, we plan to expand the analysis of the epidemic threshold for homogeneous ADNs, and characterise the system behaviour above the epidemic threshold. For future research, one can consider incorporating social networks, to model real-world interaction patterns more accurately, and subsequently study the effect of contact tracing as a control measure. A third research avenue would be to validate the model against real-world data on STIs.

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