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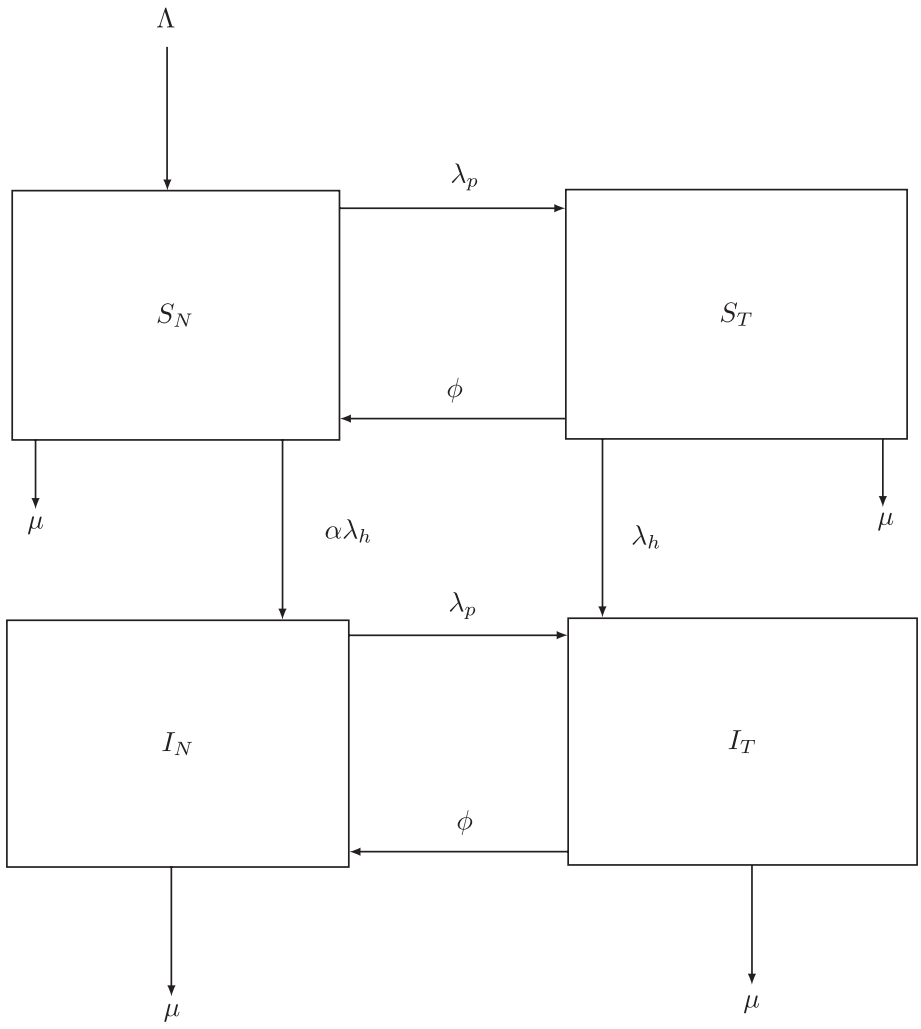

COMPUTATIONAL SCIENCE | RESEARCH ARTICLE

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Modelling the effects of sexting on the transmission dynamics of HSV-2 amongst adolescents

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Abstract: Prior studies have indicated that adolescents who are into sexting are likely to engage in risky sexual behaviours. In this paper, a mathematical model to assess the impact of sexting and peer influence on the spread of HSV-2 amongst adolescents is developed. The threshold parameters of the model are determined and stabilities are analysed. The impact of filtering and awareness campaigns is explored. Results from the study suggest that HSV-2 prevalence is high amongst adolescents who are into sexting as compared to those who do not. Further, we applied optimal control theory to the proposed model. The controls represent filtering and awareness campaigns. The objective is based on minimising the susceptible sexting adolescents, infected non-sexting adolescents and the infected sexting adolescents. The optimal control is characterised and numerically solved. Overall, the application of optimal control theory suggests that more effort should be devoted to both controls, filtering and awareness campaigns.

Subjects: Applied Mathematics; Mathematics & Statistics; Mathematics for Biology & Medicine; Science

Keywords: HSV-2; stability; sexting; reproduction number; sensitivity analysis; optimal control

1. Introduction

Cell phone usage has increased dramatically in the USA, with more than 320 million individual subscriber connections as of 2011 (Cellular Telecommunications and Internet Association, 2011). Adolescents are more likely to be cell phone users, where adolescents are the range of persons who are 10–19 years old (World Health Organisation, 1998) and constituting approximately 20% of the world's population (Denhe & Riedner, 2005). Cell phone use has the potential for other negative

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PUBLIC INTEREST STATEMENT

Sexting has been found to polarise adolescents sexual behaviours, usually leading them to engage in risky sexual behaviours. In this paper, the impact of sexting on HSV-2 is explored through a mathematical model. Results of model analysis suggest that sexting strongly contributes to the rise of HSV-2 cases amongst the adolescents. This result points to the need to put in place mechanisms to monitor how adolescents make use of computer-related gadgets in order to control sexting-related spread of sexually transmitted infections.

outcomes and in recent years, a new trend of sexualised text communication has emerged, which is known as sexting. "Sexting" was listed in Time magazine as the number one word buzzword of 2009 (Stephey, 2009), and was also a finalist for the 2009 word of the year by the New Oxford American Dictionary (Stanglin, 2009). Sexting is a play word on the words "sex-texting", which involves sending, posting or forwarding nude, semi-nude, sexually suggestive or vulgar text, picture or video messages via an electronic device (Siegle, 2010). The major force behind the majority of adolescents joining sexting is because of peer influence (Carter, 2009; National Campaign to Prevent Teen and Unplanned Pregnancy, 2008). Researchers suggest that the adolescents were amongst the group with the highest sexting rate, and they are also the group that reports the greatest use of these devices (Mitchell, Finkelhor, Jones, & Wolak, 2012; National Campaign to Prevent Teen and Unplanned Pregnancy, 2008). Adolescents and people who seek partners online or who are involved in sexting have a higher number of sexual partners, more unprotected sex acts, higher rates of substance use in conjunction with sexual activity and more sexually transmitted infections (STIs) (Benotsch et al., 2011; Bolding, Davis, Hart, Sherr, & Elford, 2006; Bull & McFarlane, 2000; Grov et al., 2007; Horvath, Bowen, & Williams, 2006; McFarlane, Bull, & Rietmeijer, 2000, 2002). With rapidly increasing popularity of smart phones as web platforms, there would appear to exist a potential for cell phone technology to also play a role in sexuality. Since sexting was found to be greatly associated with high-risk sexual behaviour and higher rates of STIs (Benotsch, Snipes, Martin, & Bull, 2013), it would be very crucial to find ways of reducing sexting, especially amongst adolescents.

STIs found in adolescents include gonorrhoea, chlamydial infection, syphilis, trichomoniasis, chancroid, HSV-2, genital warts, HIV infection and hepatitis B infection (World Health Organisation, 2007). Adolescents are the age group that is at a high risk for nearly all STIs (Hill & Biro, 2001). There are overwhelming epidemiological data that demonstrate the heavy burden of STIs on adolescents. In 2007, WHO estimated that one-third of the 333 million cases of curable STIs occurred amongst young people under the age of 25 (World Health Organisation, 2007). More than 40% of adolescents are subsequently infected by at least one STI other than the initial infecting organism (Fortenberry, 2009). One in 20 young individuals contracts a curable STI every year, and one in four active adolescent women is diagnosed with an STI every year (Hill & Biro, 2001; Yarber & Parillo, 1992). Together, HIV and STIs are responsible for the destruction of health on a massive scale worldwide (Mushayabasa, Tchuenche, Bhunu, & Ngarakana-Gwasira, 2011). There are many reasons which drive adolescents to engage in such risky activities, including cognitive development, physiological susceptibility, peer influence, logistic issues and specific sexual behaviours. Sexting plays a major role in polarising someone's sexual behaviour. Of all the STIs that may be prevalent amongst the adolescents, HSV-2 is currently the one mostly advocated for use in evaluating the impact of interventions to reduce sexual risk behaviours (Bastien et al., 2012). Unlike bacterial STIs, which can be cured, HSV-2 cannot be cured and while prevalence for bacterial STIs are on the decline in Sub-Saharan Africa, HSV-2 is on the rise (Abdu-Raddad et al., 2008). Adolescents infected with HSV-2 infection may be at an increased risk for transmission and acquisition of human immunodeficiency virus (HIV) (Corey, Wald, & Celum, 2004). Furthermore, pregnant adolescents infected with HSV-2 can transmit the infection to the neonate, which can lead to serious complications for the neonate, such as neurologic problems and even death (Hutto, 1987; Nahmias et al., 1971; Stagno & Whitely, 1999).

Most of the limited research on sexting to date focused on social and legal consequences of this behaviour (O'Keeffe & Clarke-Pearson, 2011). There is less research that has examined the potential health implications of sexting. Specifically, there has been limited research examining the relationship between sexting and sexual risk behaviour (Benotsch et al., 2013). A number of mathematical models have looked into mathematical modelling of STIs and high-risk sexual behaviour (Garnett, 2002; Garnett & Anderson, 1996; Gorbach & Holmes, 2003; Pinkerton, Chesson, Crosby, & Layde, 2011; Truong et al., 2006; Turner, Garnett, Ghani, Sterne, & Low, 2004; Zenilman, 2007 to mention just a few). Of all the STIs, this paper focuses on HSV-2 in particular since it is on the rise in Sub-Saharan Africa amongst the adolescents and in contrast to other STIs such as chlamydial, gonorrhoea, syphilis and trichomoniasis, infection with HSV-2 is lifelong and once established, there is no treatment to eliminate it (Geretti, 2006; Gupta, Warren, & Wald, 2007). The other problem with HSV-2 in adolescents is that it's

somewhat difficult to determine, given many infections are asymptomatic or they go unrecognised; hence, it differs with most of other STIs in general. HSV-2 can be prevented by behavioural interventions (Hill & Biro, 2001) and increased awareness. Education on HSV-2 is crucial, and can be done at schools, homes and hospitals to make adolescents aware of this infection (Cheng & Lo, 2002). To date also, a number of mathematical models have looked into mathematical modelling of HSV-2 (Alsallaq et al., 2010; Foss et al., 2009; Garnett, Dublin, Slaoui, & Darcis, 2004; Gershengorn, Darby, & Blower, 2003; Lou, Qesmi, Wang, Steben, & Wu, 2007; Mhlanga, Bhunu, & Mushayabasa, 2014; Schwartz, Bodine, & Blower, 2007; Schwartz & Blower, 2005 to name but just a few). To the best of our knowledge as authors, no mathematical model has looked into the effects of sexting and peer influence on the transmission dynamics of any STIs, mainly HSV-2. It is against this background that our study finds relevance and motivation, by formulating a mathematical model to investigate the impact of sexting and peer influence on the transmission dynamics of HSV-2. The model incorporates some key epidemiological features such as the impact of awareness campaigns and filtering. Filtering is a modern technology that prevents individuals from sending or accessing pornographic content and sexting on the internet, mobile phones, ipads, etc. These filters can operate at network and device levels (Carter, 2009; Eneman, 2006; Patlar, Bayrak, Mendi, & Hanna, 2012). The main objective of this study is to forecast future trends in the incidence of HSV-2 epidemic and also to quantify the association between sexting, peer influence and HSV-2 epidemic amongst adolescents in a society.

The paper is structured as follows. The HSV-2 transmission model is formulated in Section 2. Analytic results of the model system are presented in Section 3. Simulation results and projection profiles of HSV-2 are presented in Section 4. In Section 5, optimal control theory has been applied to the model formulation in Section 2. Summary and concluding remarks round-up the paper.

2. Model formulation

The model subdivides the population based on HSV-2 status and also on sexting amongst adolescents. The population is divided into the following classes: susceptible adolescents who are not into sexting S_N, infected adolescents who are not into sexting I_N, susceptible adolescents who are into sexting S_T and infected adolescents who are into sexting I_T. Individuals in all subclasses experience natural death only at a rate μ, which is proportional to the number in each class and neither sexting nor HSV-2 causes death. The total sexually active population at time t is given by $N(t) = N_N(t) + N_T(t)$, $N_N(t) = S_N(t) + I_N(t)$ and $N_T(t) = S_T(t) + I_T(t)$ with $N_T(t)$ and $N_N(t)$ being the total number of adolescents who are into sexting and non-sexting, respectively. The susceptible population is increased by a constant inflow into the population at a rate Λ. Assuming homogeneous mixing of the population, the sexting and non-sexting susceptibles acquire HSV-2 infection at a rate λ_h and $\alpha\lambda_h$, respectively, with

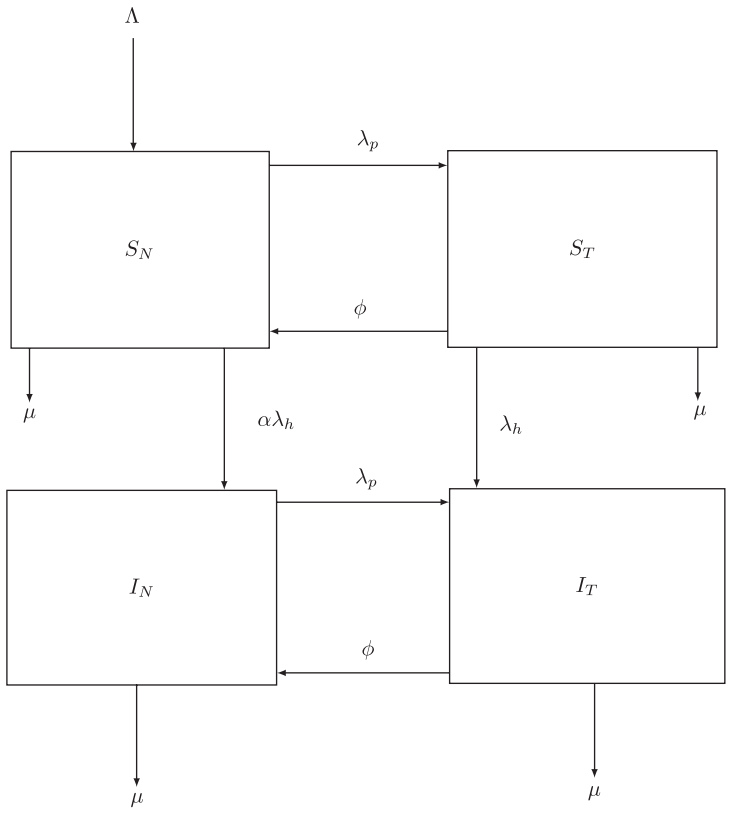
$$\lambda_h = \frac{\beta_h(I_T + (1-\eta)I_N)}{N}, \tag{1}$$

and where β_h is the effective contact rate for HSV-2 infection (contact sufficient to result in HSV-2 infection). The modification parameter η captures the fact that HSV-2 infectives who are into sexting are assumed to have a higher chance, relative to HSV-2 infectives who are not into sexting of generating new infections due to their risky sexual behaviours (Benotsch et al., 2013; Ferguson, 2011). The modification parameter $\alpha \in (0, 1)$ accounts for the reduced chance of being infected with HSV-2 as non-sexting individuals have less risky sexual behaviours.

Susceptible adolescents who are not into sexting and the infectious adolescents who are not into sexting acquire sexting habits via effective contacts with those who are into sexting at a rate λ_p. With many reasons that can lead adolescents into sexting, we shall only focus on peer influence as the main one (Carter, 2009; National Campaign to Prevent Teen and Unplanned Pregnancy, 2008). Thus, the force of peer influence is given by

$$\lambda_p = \frac{\beta_p(S_T + I_T)}{N}, \tag{2}$$

Figure 1. Structure of model.



where β_p is the effective contact rate for peer influence (contact sufficient enough to coerce a fellow adolescent to join sexting through peer influence). We assume that adolescents quit the behaviour of sexting at rate ϕ, and this may be due to some adverse outcomes for those involved, such as embarrassment, mental health problems, public dissemination of sexual photos and legal consequences if the images are of underage individuals, repenting due to various religions who do not encourage it and also through some educational campaigns (Lenhart, 2009; Mitchell et al., 2012; Ryan, 2010; Theodore, 2011). The model flow diagram is depicted in Figure 1.

From the descriptions and assumptions on the dynamics of the epidemic made above, the following are the model equations.

$$\begin{aligned} S_N' &= \Lambda - \alpha\lambda_h S_N - \lambda_p S_N - \mu S_N + \phi S_T, \\ S_T' &= \lambda_p S_N - \lambda_h S_T - (\mu + \phi) S_T, \\ I_N' &= \alpha\lambda_h S_N + \phi I_T - \lambda_p I_N - \mu I_N, \\ I_T' &= \lambda_h S_T + \lambda_p I_N - (\mu + \phi) I_T. \end{aligned} \tag{3}$$

3. Analytic results

3.1. Basic properties of the model

In this section, we study the basic properties of the solutions of model system (Equation 3), which are essential in the proofs of stability.

LEMMA 1 *The equations preserve positivity of solutions.*

Proof The vector field given by the right-hand side of model system (Equation 3) points inward on the boundary of $\mathbb{R}^4_+ \setminus \{0\}$. For example, if $S_N = 0$, then, $S_N' = \Lambda\pi_0 + \phi S_T \geq 0$. In an analogous manner,

the same result can be shown for the other model components (variables). □

LEMMA 2 *All solutions of model system (Equation 3) are bounded.*

Proof Using model system (Equation 3), we have

$$\begin{aligned} N' &= S_N' + S_T' + I_N' + I_T', \\ N' &= \Lambda - \mu N. \end{aligned} \tag{4}$$

Therefore, all feasible solutions of model system (Equation 3) enter the region

$$\Gamma = \left\{ (S_N, S_T, I_N, I_T) \in \mathbb{R}_+^4 : N \leq \frac{\Lambda}{\mu} \right\}. \tag{5}$$

Thus, Γ is positively invariant and it is sufficient to consider solutions of model system (Equation 3) in Γ. Existence, uniqueness and continuation results for model system (Equation 3) hold in this region, and all solutions of model system (Equation 3) starting in Γ remain in Γ for all (time) $t \geq 0$. All parameters and state variables for model system (Equation 3) are assumed to be non-negative (for biological relevance) $\forall\, t \geq 0$ since it monitors human population. □

THEOREM 1 *For every non-zero, non-negative initial value, solutions of model system (Equation 3) exist for all times.*

Proof Local existence of solutions follows from standard arguments since the right-hand side of (Equation 3) is locally Lipschitz. Global existence follows from the a-priori bounds. □

3.2. Disease-free equilibrium and its stability analysis

Model system (Equation 3) has an evident DFE given by,

$$\mathcal{V}^0 = (S_N^0, S_T^0, I_N^0, I_T^0) = \left(\frac{\Lambda(\mu+\phi)}{\mu\beta_p}, \frac{\Lambda}{\mu} - \frac{\Lambda(\mu+\phi)}{\mu\beta_p}, 0, 0 \right). \tag{6}$$

Following Van den Driessche and Watmough (2002), the non-negative matrix F, of the new infection terms and the M matrix V, of the transition terms associated with model system (Equation 3), are given by

$$F = \begin{bmatrix} \dfrac{\alpha(1-\eta)(\mu+\phi)\beta_h}{\beta_p} & \dfrac{\alpha(\mu+\phi)\beta_h}{\beta_p} \\ \dfrac{(1-\eta)(\beta_p-(\mu+\phi))}{\beta_p} & \dfrac{(\beta_p-(\mu+\phi))}{\beta_p} \end{bmatrix} \text{ and } V = \begin{bmatrix} \beta_p - \phi & -\phi \\ (\mu+\phi) - \beta_p & \mu+\phi \end{bmatrix}, \tag{7}$$

and the effective reproduction number of model system (Equation 3), denoted by $\mathcal{R}_{HP}$, is thus given by

$$\mathcal{R}_{HP} = \rho(FV^{-1}) = \frac{\beta_h[\psi^2 + \alpha(1-\eta)(\mu+\phi)^2 + \psi(\mu(1+\alpha)+\phi(1-\eta)+\alpha\phi)]}{\mu\beta_p^2}. \tag{8}$$

$\psi = \beta_p - (\mu+\phi) > 0$, by Lemma 1, throughout the manuscript. The reproduction number $\mathcal{R}_{HP}$ is defined as the number of secondary HSV-2 infections produced by one HSV-2-infected individual during his/her entire infectious period in a mixed population of susceptible adolescents, who perform sexting and those who do not.

Theorem 2 follows from Van den Driessche and Watmough (2002).

THEOREM 2 *The disease-free equilibrium $\mathcal{U}^0$ of model system (Equation 3) is locally asymptotically stable if $\mathcal{R}_{HP} \leq 1$ and unstable otherwise.*

3.2.1. Analysis of the reproduction number, $\mathcal{R}_{HP}$
The reproduction number is differentiated into categories:

Case 1: Every adolescent is joining sexting in the society.

In this case, $(\beta_p, \phi) \rightarrow (\infty, 0)$, so that $\mathcal{R}_{HP}$ becomes $\mathcal{R}_{0P}$ which is given by

$$\mathcal{R}_{0P} = \frac{\beta_h}{\mu}. \tag{9}$$

Here, $\mathcal{R}_{0P}$ denotes the number of secondary HSV-2 cases generated by one infected HSV-2 adolescent during his/her entire life as an adolescent in the presence of sexting and other adolescents who are also sexting.

Case 2: No sexting in the society amongst adolescents.

In this case, we set $S_T = I_T = 0$ and $\mathcal{R}_{HP}$ becomes $\mathcal{R}_{H0}$, given by

$$\mathcal{R}_{H0} = \frac{\alpha(1-\eta)\beta_h}{\mu} = \alpha(1-\eta)\mathcal{R}_{0P}. \tag{10}$$

Since $\eta \in (0, 1)$, it follows that $R_{H0} < R_{0P}$. Here, $\mathcal{R}_{H0}$ denotes the number of secondary HSV-2 cases generated by one infected HSV-2 adolescent during his/her entire life as an adolescent in the absence of sexting amongst other susceptible adolescents who are also not into sexting.

Case 3: The general case.

It is given and defined in Equation 8.

Since we have deduced that $R_{H0} < R_{0P}$, we now use a numerical illustration to explore the relationship between R_{HP} and R_{H0}.

Figure 2 depicts that in a society where all adolescents would be sexting, the prevalence of HSV-2 is higher as shown by the graph of $\mathcal{R}_{0P}$. In the absence of sexting, the reproduction number would be very low, hence HSV-2 can be easier to curtail. Thus, $\mathcal{R}_{0P} > \mathcal{R}_{HP} > \mathcal{R}_{H0}$, as illustrated in Figure 2.

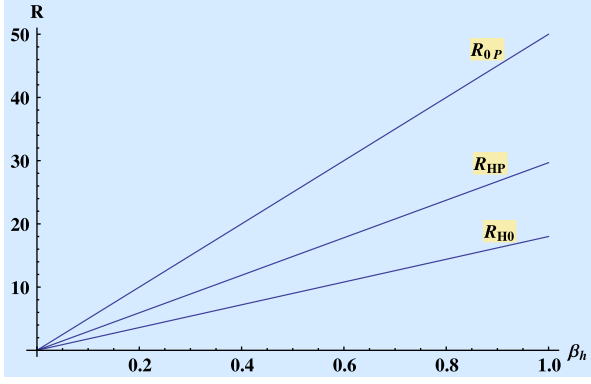


Figure 2. Effects of varying the effective contact rate for HSV-2 infection (β_h) on the reproduction numbers.

3.3. Endemic equilibrium

Model system (Equation 3) has two possible endemic equilibria: sexting-free endemic equilibrium and the endemic equilibrium where the adolescents who are sexting and those who are not sexting coexist, "interior equilibrium point".

3.3.1. Sexting-free endemic equilibrium

This occurs when there are no adolescents who are involved in sexting, that is, $(S_T = 0, I_T = 0)$. Model system (Equation 3) reduces to

$$\begin{aligned} S_N' &= \Lambda - \alpha\lambda_h S_N - \mu S_N, \\ I_N' &= \alpha\lambda_h S_N - \mu I_N. \end{aligned} \tag{11}$$

For model system (Equation 11), it can be shown that the region

$$\Gamma_1 = \left\{ (S_N, I_N) \in \mathbb{R}_+^2 : N_N \leq \frac{\Lambda}{\mu} \right\}, \tag{12}$$

is invariant and attracting. The basic reproduction number for model system (Equation 11) is

$$\mathcal{R}_{H0} = \frac{\alpha(1-\eta)\beta_H}{\mu}, \tag{13}$$

as given by equation 10. The dynamics of sexting-free endemic equilibrium will be considered in Γ_1, and it is given by,

$$\mathcal{U}_{H0}^* = (S_N^*, 0, I_N^*, 0) = \left(\frac{\Lambda}{\mu\mathcal{R}_{HO}}, 0, \frac{\Lambda}{\mu}\left(1 - \frac{1}{\mathcal{R}_{H0}}\right), 0 \right). \tag{14}$$

Thus, the sexting-free endemic equilibrium point exists and is unique for $\mathcal{R}_{H0} > 1$.

Definition 1 The disease is said to persist if there exists some $\varepsilon > 0$, such that $\lim_{t\to\infty} \sup \frac{I_N}{N_N} \geq \varepsilon$ for all non-negative solutions of (Equation 11), with $I_N(0) > 0$ and $S_N(0) >> 0$.

Using the Lyapunov's direct stability method, we have

$$\begin{aligned} &\frac{dI_N}{dt} < \frac{\alpha\beta_h(1-\eta)I_N S_N}{N} - \mu I_N \\ \Leftrightarrow\ &\frac{dI_N}{dt} < \mu\left[\frac{\alpha\beta_h(1-\eta)}{\mu} - 1\right] I_N \\ \Rightarrow\ &\frac{dI_N}{dt} < \mu\left(\mathcal{R}_{H0} - 1\right) \end{aligned} \tag{15}$$

The linearised differential inequality system (Equation 15) holds for $\mathcal{R}_{H0} > 1$. We summarise the result in Theorem 3, below.

THEOREM 3 *If $\mathcal{R}_{H0} > 1$, the endemic equilibrium point exists.*

Now, we have to explore the stability of this endemic equilibrium following an approach by Korobeinikov (2006). *From Theorem 3, $\mathcal{R}_{H0} > 1$, assume that $\mathcal{U}_{H0}^*$ exists for all $S_N, I_N > \epsilon$ for some ϵ.*

Let $\frac{(1-\eta)\beta I_N S_N}{S_N + I_N} = g(S_N, I_N)$ (a monotonic and positive function) and define the following continuous function $\mathbb{R}_2^+$ (for more details, see Korobeinikov, 2006). If a function is such that

$$V(S_N, I_N) = S_N - \int_{\epsilon}^{S_N} \frac{g(S_N^*, I_N^*)}{g(\tau, I_N^*)} d\tau + I_N - \int_{\epsilon}^{I_N} \frac{g(S_N^*, I_N^*)}{g(S_N^*, \tau)} d\tau. \tag{16}$$

Given $g(S_N, I_N)$ is monotonic with respect to its variables, then the endemic $\mathcal{U}_{H0}^*$ is the only extremum and the global minimum of this function. Indeed,

$$\frac{\partial V}{\partial S_N} = 1 - \frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)} = \frac{I_N^*(S_N - S_N^*)}{S_N(S_N^* + I_N^*)}, \ \frac{\partial V}{\partial I_N} = 1 - \frac{g(S_N^*, I_N^*)}{g(S_N^*, I_N)} = \frac{S_N^*(I_N - I_N^*)}{I_N(S_N^* + I_N^*)}. \tag{17}$$

Furthermore,

$$\frac{\partial^2 V}{\partial S_N^2} = \frac{S_N^* I_N^*}{S_N^2(S_N^* + I_N^*)}, \ \frac{\partial^2 V}{\partial I_N^2} = \frac{S_N^* I_N^*}{I_N^2(S_N^* + I_N^*)}, \tag{18}$$

are non-negative, then $\mathcal{U}_{H0}^*$ is a minimum. That is, $V(S_N, I_N) \geq V(S_N^*, I_N^*)$, and hence V is a Lyapunov function. At endemic equilibrium point, model system (Equation 11) has the following identities,

$$\begin{aligned} \Lambda &= g(S_N^*, I_N^*) + \mu S_N^*, \\ \mu I_N^* &= g(S_N^*, I_N^*). \end{aligned} \tag{19}$$

The Lyapunov function (Equation 16) satisfies

$$\begin{aligned} \frac{dV}{dt} &= S_N' - S_N'\left(\frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)}\right) + I_N' - I_N'\left(\frac{g(S_N^*, I_N^*)}{g(S_N^*, I_N)}\right) \\ &= (\Lambda - g(S_N^*, I_N^*) - \mu S_N^*)\left(1 - \frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)}\right) + (g(S_N^*, I_N^*) - \mu I_N^*)\left(1 - \frac{g(S_N^*, I_N^*)}{g(S_N^*, I_N)}\right) \\ &= \mu S_N^*\left(1 - \frac{S_N}{S_N^*}\right)\left(1 - \frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)}\right) + g(S_N^*, I_N^*)\left(1 - \frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)}\right)\left(1 - \frac{g(S_N, I_N)}{g(S_N^*, I_N)}\right) \\ &\quad + g(S_N^*, I_N^*)\left(\frac{I_N}{I_N^*} - \frac{g(S_N, I_N)}{g(S_N, I_N^*)}\right)\left(\frac{g(S_N^*, I_N^*)}{g(S_N^*, I_N)} - 1\right) \end{aligned} \tag{20}$$

Since $\mathcal{U}_{H0}^* > 0$, the function $g(S_N, I_N)$ is concave with respect to I_N and $\frac{\partial^2 g(S_N, I_N)}{\partial I_N^2} \leq 0$, then $(dV/dt) \leq 0$ for all $S_N, I_N > 0$. Also, the monotonicity of $g(S_N, I_N)$ with respect to S_N and I_N ensures that

$$\left(1 - \frac{S_N}{S_N^*}\right)\left(1 - \frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)}\right) \leq 0 \quad \textit{and} \quad \left(1 - \frac{g(S_N^*, I_N^*)}{g(S_N, I_N^*)}\right)\left(1 - \frac{g(S_N, I_N)}{g(S_N^*, I_N)}\right) \leq 0 \tag{21}$$

holds for all $S_N, I_N > 0$. Furthermore,

$$\left(\frac{I_N}{I_N^*} - \frac{g(S_N, I_N)}{g(S_N, I_N^*)}\right)\left(\frac{g(S_N^*, I_N^*)}{g(S_N^*, I_N)} - 1\right) \leq 0. \tag{22}$$

If

$$\begin{aligned} &\frac{g(S_N, I_N)}{g(S_N, I_N^*)} \geq \frac{I_N}{I_N^*} \quad \textit{when} \quad g(S_N^*, I_N^*) \geq g(S_N^*, I_N) \quad \textit{and} \\ &\frac{g(S_N, I_N)}{g(S_N, I_N^*)} \leq \frac{I_N}{I_N^*} \quad \textit{when} \quad g(S_N^*, I_N^*) \leq g(S_N^*, I_N) \end{aligned} \tag{23}$$

holds for all $S_N, I_N > 0$. *Since* $g(S_N, I_N)$ *monotonic* $g(S_N^*, I_N) \geq g(S_N^*, I_N^*) \Rightarrow I_N \geq I_N^*$. *Inequalities (Equation 23) which hold for any concave function are sufficient to ensure that* $dV/dt \leq 0$. *Thus, we have established the following result.*

THEOREM 4 *The unique endemic equilibrium* $\mathcal{U}_{H0}^*$ *is globally asymptotically stable whenever conditions (Equation 23) are satisfied.*

3.3.2. Interior equilibrium

This equilibrium occurs when the non-sexting adolescents and the adolescents who are into sexting both exist in the presence of HSV-2, denoted by

$$\mathcal{U}_{HP}^{**} = (S_N^{**}, I_N^{**}, S_N^{**}, I_N^{**}). \tag{24}$$

Expressing the expressions for each variable in terms of λ_p^{**} and λ_h^{**} and solving the problem is a daunting task; but, we claim that a biologically feasible interior equilibrium exists. The permanence of the disease destabilises the disease-free equilibrium $\mathcal{U}_0$ since $\mathcal{R}_{HP} > 1$, and the endemic equilibrium $\mathcal{U}_{HP}^{**}$ exists.

LEMMA 3 *System (Equation 3) is uniformly persistent on* Γ.

Proof If $\mathcal{R}_{HP} > 1$, then the disease is uniformly persistent, where uniform persistence of a system implies that there exists $\zeta > 0$, such that

$$\liminf_{t\to\infty} I_N(t) > \zeta, \quad \liminf_{t\to\infty} I_T(t) > \zeta, \tag{25}$$

for all solutions (S_N, S_T, I_N, I_T) of (Equation 3) with $I_N(0) > 0$ and $I_T(0) > 0$.

Let $\Delta = \{(S_N, S_T, I_N, I_T) \in \mathbb{R}_+^4 : I_N = I_T = 0\}$. Thus, Δ is the set of all disease-free states of model system (Equation 3) and it can be verified that Δ is positively invariant. Let $M = \Delta \cap \Gamma$, then, since Δ is positively invariant and Γ is also positively invariant, thus M is also positively invariant. Also note that $\mathcal{U}_0 \in M$ and $\mathcal{U}_0$ attract all solutions in Δ. So, $\Omega(M) = \{\mathcal{U}_0\}$. The equations for the infected components of model system (Equation 3) can be written as

$$x'(t) = Y(x)x(t) \tag{26}$$

where $x(t) = (I_N(t), I_T(t))^T$, $Y(x) = [\mathcal{G} - V]$,

with

$$\mathcal{G} = \begin{bmatrix} \dfrac{\alpha(1-\eta)\beta_h S_N}{N} & \dfrac{\alpha\beta_h S_N}{N} \\ \dfrac{(1-\eta)\beta_h S_T}{N} & \dfrac{\beta_h S_T}{N} \end{bmatrix}. \tag{27}$$

It is clear that $Y(\mathcal{U}_0) = F - V$. Also, it is very easy to check that $Y(\mathcal{U}_0)$ is irreducible. To show that M is a uniformly weak repeller, we will apply Lemma A.4 from Ackleha, Maa and Salceanua (2011), stated below for elucidation. □

LEMMA 4 *Assume that* $\Omega(M)$ *is a union of periodic orbits and the following hold:*

(1) $\forall\ \mathcal{P} \subseteq \Omega(M)$ *a periodic orbit of period T,* $\exists\ x \in \mathcal{P}$ *such that* $P(T, x)$ *is primitive.*

(2) $r(P) > 1$, *for each periodic orbit* $\mathcal{P} \subseteq \Omega(M)$.

Then, M is a uniformly weak repeller.

Since the spectral radius of $Y(\mathcal{U}_0^*) = \mathcal{R}_{HP} > 1$, *the spectral radius of* $e^{Y(\mathcal{U}_0^*)} > 1$. *So, condition 2 of Lemma A.4 is satisfied. Taking* $x = \mathcal{U}_0^*$, *we get* $P(T, \mathcal{U}_0^*) = e^{Y(\mathcal{U}_0^*)}$ *which is a primitive matrix because* $Y(\mathcal{U}_0^*)$ *is irreducible, as mentioned in Theorem A.12(i)* (Smith & Waltman, 1995*). This satisfies the condition 1 of Lemma A.4. Thus, M is a uniformly weak repeller and the disease is weakly persistent. M is trivially closed and bounded relative to* Γ *and hence compact. Therefore, by Theorem 1.3* (Thieme, 1993), *we have that M is a uniformly strong repeller and the disease is uniformly persistent.*

The epidemiological implication of Lemma 3 is that the disease will persist in the population whenever $\mathcal{R}_{HP} > 1$.

4. Numerical results

In order to illustrate the results of the foregoing analysis, we have simulated model system (Equation 3) using the parameters in Table 1. Unfortunately, the scarcity of the data on HSV-2 and sexting correlation with a focus on adolescents limits our ability to calibrate; nevertheless, we assume some of the parameters in the realistic range for illustrative purposes. These parsimonious assumptions reflect the lack of information currently available on HSV-2 and sexting with a focus on adolescents. Reliable data on the risk of transmission of HSV-2 amongst adolescents would enhance our understanding and aid in the possible intervention strategies to be implemented.

From Figure 3(a), we observe that increase in the effective contact rate for peer influence results in an increase of the cumulative HSV-2 cases. It is worth noting that the disease can be reduced to very low levels after 20 years, if the effective contact rate for sexting is zero ($\beta_p = 0$). Figure 3(b) shows that when the sexting quitting rate is significantly high, the reproduction number reduces significantly. The same scenario happens when we have low values of the sexting quitting rate and the reproduction number becomes significantly very high. Thus, it would be very crucial for us to have high sexting quitting rate coupled with low peer influence to be able to curtail the prevalence of the STI amongst adolescents.

Sensitivity analysis investigates the type of change and the amount inherent in the model as captured by the terms that define the reproduction number. If the reproduction number is very sensitive to a particular parameter, then a perturbation of the conditions that connects the dynamics to such a parameter may prove useful in identifying policies or intervention strategies that reduce the epidemic prevalence. In this section, the partial rank correlation coefficients (PRCCs) were calculated to estimate the correlation between values of the reproduction number and the six model parameters across 1,000 random draws from the empirical distribution of $\mathcal{R}_{HP}$ and its associated parameters.

Table 1. Model parameters and their interpretations

Definition	Symbol	Baseline values (range)	Source
Recruitment rate	Λ	10,000 yr^{-1}	Mhlanga et al. (2014)
Natural death rate	μ	0.02 (0.015–0.02) yr^{-1}	Bhunu and Mushayabasa (2011)
Effective contact rate for HSV-2	β_h	0.01 (0.001–0.03)yr^{-1}	Bryson et al. (1993), Corey et al. (2004), Mertz et al. (1988), Wald et al. (2001)
Effective contact rate for peer influence	β_p	0.4	Bhunu, Mushayabasa, and Tchuenche (2011)
Rate of quitting sexting	ϕ	variable	Assumed
Modification parameter	α	0.6 (0–1)	Assumed
Modification parameter	η	0.6 (0–1)	Assumed

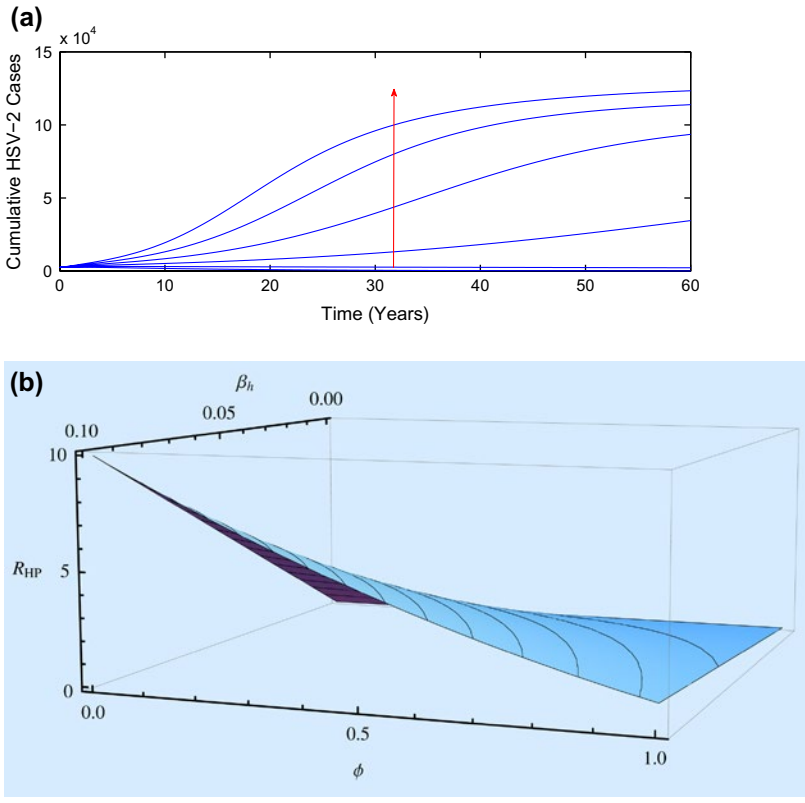


Figure 3. (a) The influence of β_p on cumulative HSV-2 cases over a period of 60 years, with β_p varying from 0 to 1 with a step size of 0.2 and the rest of the parameters being fixed on their baseline values from Table 1. The direction of the arrow shows an increase in β_p. (b) The effects of the effective contact rate for HSV-2 infection β_h and the sexting quitting rate ϕ on $\mathcal{R}_{HP}$.

Figure 4 illustrates the PRCCs using $\mathcal{R}_{HP}$ as an output variable. Results here suggest that peer influence effective contact rate is the most sensitive parameter in increasing the magnitude of the reproduction number, followed by the effective contact rate for HSV-2. The rate of quitting sexting is the most sensitive in reducing the magnitude of the reproduction number. Thus, it would be very advisable to find ways of increasing the number of adolescents quitting sexting and at the same time, finding ways of reducing the number of adolescents who are falling into the behaviour of sexting. Introducing methods such as filtering, with the help of the parents, may be very helpful in reducing the number of adolescents who would be joining sexting, and at the same time minimising sexting.

Since peer influence effective contact rate and the rate of quitting sexting have a significant impact on $\mathcal{R}_{HP}$, we examined their dependence in more detail. We used Latin Hypercube Sampling and Monte Carlo simulations to run 1,000 simulations, where all parameters were simultaneously drawn from across their ranges.

Figure 5 illustrates that varying two sample parameters will have an effect on $\mathcal{R}_{HP}$. If the rate at which the adolescents quitting sexting is sufficiently high, then $\mathcal{R}_{HP} < 1$ and the disease can be controlled. However, if the rate of quitting sexting is low, then $\mathcal{R}_{HP} > 1$ and the disease will persist.

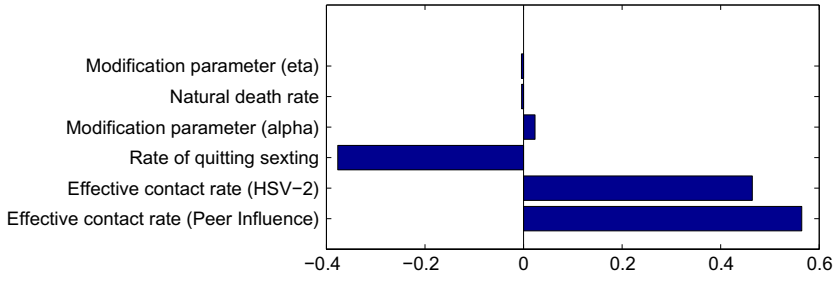


Figure 4. PRCCs showing the effects of parameter variation on $\mathcal{R}_{HP}$ using ranges from Table 1. Parameters with positive PRCCs will increase $\mathcal{R}_{HP}$ when they are increased, whereas parameters with negative PRCCs will decrease $\mathcal{R}_{HP}$ when they are increased.

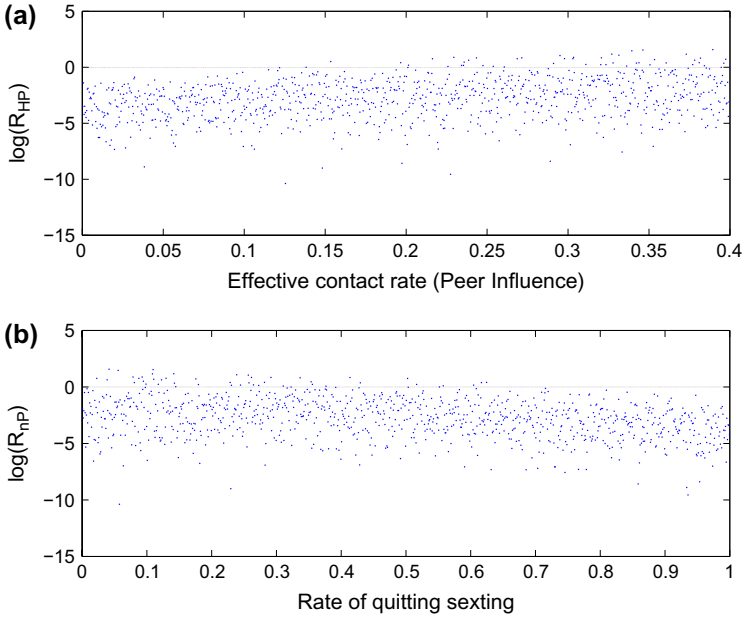


Figure 5. Latin Hypercube Sampling for varying of (a) peer influence effective contact rate and (b) the rate of quitting sexting.

We can also see that the disease persists for higher values of the effective contact rate for peer influence and may be controlled for lower values of the peer influence effective contact rate. Figure 5 results are in agreement with our earlier findings that suggested low peer influence effective contact rate and high sexting quitting rate are crucial in reducing the prevalence of HSV-2.

5. Optimal control

In order to assess an effective campaign to control sexting and HSV-2 in a society which pursues the goals of minimising the HSV-2-infected adolescents and sexting adolescents, and having more susceptible non-sexting and non-HSV-2-infected adolescents. To achieve this goal, we will reconsider model system (Equation 3) and introduce two control variables $u_1(t)$ and $u_2(t)$, so as to try and reduce the number of HSV-2-infected adolescents and the sexting adolescents. The control variable $u_1(t)$ is used to control the number of adolescents who are joining the trade of sexting, usually through peer influence (Carter, 2009; Tanya, 2010). Internet service providers and mobile phone operators with the help of the parents can protect adolescents by installing filters on their devices and networks. These filters that would be fitted to deal with all explicit material can be at network or device level (Carter, 2009). The control variable $u_2(t)$ is used to control the number of adolescents who can become infected with HSV-2, usually through unsafe sex. Measures such as counselling and awareness campaigns can be employed to fulfil control $u_2(t)$. However, there will be a lot of costs generated during the control process. So, it would be advisable to balance between the costs and the effects of HSV-2 prevalence and sexting. In view of this, our optimal control problem is to minimise the objective functional given by

$$J(u_1,u_2)=\int_0^{t_f}[\Phi_1 S_T+\Phi_2 I_N+\Phi_3 I_T+\frac{1}{2}(\Psi_1 u_1^2(t)+\Psi_2 u_2^2(t))]dt, \tag{28}$$

subject to

$$\begin{aligned}
S_N' &= \Lambda-(1-u_2(t))\alpha\lambda_h S_N-(1-u_1(t))\lambda_p S_N-\mu S_N+\phi S_T,\\
S_T' &= (1-u_1(t))\lambda_p S_N-(1-u_2(t))\lambda_h S_T-(\mu+\phi)S_T,\\
I_N' &= (1-u_2(t))\alpha\lambda_h S_N+\phi I_T-(1-u_1(t))\lambda_p I_N-\mu I_N,\\
I_T' &= (1-u_2(t))\lambda_h S_T+(1-u_1(t))\lambda_p I_N-(\mu+\phi)I_T,
\end{aligned} \tag{29}$$

with initial conditions,

$$S_N(0) = S_N^0,\ S_T(0) = S_T^0,\ I_N(0) = I_N^0,\ I_T(0) = I_T^0. \tag{30}$$

Here, $u_i(t) \in U \triangleq \{(u_1, u_2) | u_i(t)$ is measurable and $0 \le u_i(t) \le 1$, for all $t \in [0, t_f]\}, t_f$ is the end time to be controlled, U is an admissible control set, Ψ_i and for $i = 1, 2$ are the weights and cost of awareness campaigns and filtering that adjust the intensity of the two different control measures, (Ψ_1 and Ψ_2 are positive constants). Also, Φ_1, Φ_2 and Φ_3 represent the weight constants of the susceptible adolescents who are into sexting, infected adolescents who are not into sexting and the infected adolescents who are into sexting, respectively. The terms u_1^2 and u_2^2 reflect the effectiveness of filtering and awareness campaigns in the control of the disease. The values $u_1 = u_2 = 1$, represent maximum effectiveness of filtering and awareness campaigns.

5.1. Existence of the optimal control

Now, we investigate the existence of the optimal control of the above-mentioned problem.

THEOREM 5 *There exists an optimal control pair $u^* = (u_1^*, u_2^*) \in U$, such that*

$$J(u_1^*, u_2^*) = \min J(u_1, u_2), \quad u_1, u_2 \in U \tag{31}$$

subjects to the control system (Equation 29) with initial conditions (Equation 30).

Proof To prove the existence of an optimal control, we shall make use of some results from Fleming and Rishel (1975), and we have to show the following;

(1) The control state variables are non-negative values.
(2) The control set U is convex and closed.
(3) The right side of the state system is bounded by linear function in the state and control variables.
(4) The integrand of the objective functional is concave on U.
(5) There exists constants $\kappa_1,\ \kappa_2 > 0$ and $\alpha > 1$, such that the integrand $L(t, u_1, u_2) \triangleq \Phi_1 S_T + \Phi_2 I_N + \Phi_3 I_T + \frac{1}{2}(\Psi_1 u_1^2 + \Psi_2 u_2^2)$ of the objective functional satisfies

Statements 1, 2 and 3 are obviously satisfied; we only need to verify and check the last two. Since the four state variables have all been proved to be bounded above by Λ/μ, we will get the following inequalities:

$$L(t, u_1, u_2) \ge \kappa_1(|u_1| + |u_2|)^{\alpha/2} - \kappa_2 \tag{32}$$

$$\begin{aligned} &S_N' \le \Lambda + \phi S_T, \quad S_T' \le (1 - u_2(t))\lambda_h S_T, \\ &I_N' \le (1 - u_2(t))\alpha\lambda_h S_N + \phi I_T, \quad I_T' \le (1 - u_2(t))\lambda_h S_T + (1 - u_1(t))\lambda_p I_N, \end{aligned} \tag{33}$$

so the fourth condition is set up. As for the last condition,

$$L(t, u_1, u_2) \ge \kappa_1(|u_1| + |u_2|)^{\alpha/2} - \kappa_2 \tag{34}$$

is also true, when we choose $\kappa_1 = \min(\Psi_1/2, \Psi_2/2)$, and for all $\kappa_2 \in \mathbb{R}^+$, $\alpha = 2$, and that completes the proof. □

5.2. The characterisation of the optimal control

We use Pontryagin's Maximum Principle (Pontryagin, Boltyanskii, Gamkrelidze, & Mishchevko, 1985) to derive the necessary conditions for this optimal control since there exists an optimal control for

minimising the functional (Equation 35) subject to (Equation 29). This is done by first defining an augmented Hamiltonian H, with penalty terms for the control constraints as follows

$$\begin{aligned} H = {} & \Phi_1 S_T + \Phi_2 I_N + \Phi_3 I_T + \frac{\Psi_1}{2} u_1^2(t) + \frac{\Psi_2}{2} u_2^2(t) + \sum_{i=1}^{4} \lambda_i g_i \\ & - w_{11} u_1(t) - w_{12}(1 - u_1(t)) \\ & - w_{21} u_2(t) - w_{22}(1 - u_2(t)), \end{aligned} \tag{35}$$

where $w_{ij}(t) \geq 0$ are penalty multipliers satisfying,

$$\begin{aligned} & w_{11}(t) u_1(t) = w_{12}(t)(1 - u_1(t)) = 0 \text{ at optimal control } u_1^*, \\ & w_{21}(t) u_2(t) = w_{22}(t)(1 - u_2(t)) = 0 \text{ at optimal control } u_2^*. \end{aligned} \tag{36}$$

Also, where g_i is the right hand side of the differential equation of the ith-state variable. By applying Pontryagin's Maximum Principle (Pontryagin et al., 1985) and the optimal control existence result from Fleming and Rishel (1975), we obtain

THEOREM 6 *Given optimal control pairs (u_1^*, u_2^*) and solutions $S_N(t)$, $S_T(t)$, $I_N(t)$, $I_T(t)$ of the corresponding state system (Equation 29), there exist adjoint variables λ_i, $i = 1, 2, 3, 4$, satisfying*

$$\begin{aligned} \frac{\partial \lambda_1}{\partial t} &= \frac{\alpha \beta_h (I_N(1-\eta) + I_T)}{N}(1 - u_2(t))[\lambda_1 - \lambda_3] + \frac{\beta_p (S_T + I_T)}{N}(1 - u_1(t))[\lambda_1 - \lambda_2] + \mu \lambda_1 \\ \frac{\partial \lambda_2}{\partial t} &= -\Phi_1 + \frac{\beta_h (I_N(1-\eta) + I_T)}{N}(1 - u_2(t))[\lambda_2 - \lambda_4] + \frac{\beta_p I_N}{N}(1 - u_1(t))[\lambda_1 - \lambda_2] \\ &\quad + \frac{\beta_p (S_T + I_T) I_N}{N}(1 - u_1(t))[\lambda_3 - \lambda_4] + \phi[\lambda_2 - \lambda_1] + \mu \lambda_2 \\ \frac{\partial \lambda_3}{\partial t} &= -\Phi_2 + \frac{\beta_p (S_T + I_T)}{N}(1 - u_1(t))[\lambda_3 - \lambda_4] + \frac{(1-\eta)\beta_h S_T}{N}(1 - u_2(t))[\lambda_2 - \lambda_4] \\ &\quad + \frac{\alpha(1-\eta)\beta_h S_N}{N}(1 - u_2(t))[\lambda_1 - \lambda_3] + \mu \lambda_3 \\ \frac{\partial \lambda_4}{\partial t} &= -\Phi_3 + \frac{\alpha S_N \beta_h}{N}(1 - u_2(t))[\lambda_1 - \lambda_3] + \frac{\beta_p S_N}{N}(1 - u_1(t))[\lambda_1 - \lambda_2] \\ &\quad + \frac{\beta_h S_T}{N}(1 - u_2(t))[\lambda_2 - \lambda_4] + \frac{\beta_p I_N}{N}(1 - u_1(t))[\lambda_3 - \lambda_4] + \phi[\lambda_4 - \lambda_3] + \mu \lambda_4 \end{aligned} \tag{37}$$

with the terminal conditions

$$\lambda_i = 0, \quad i = 1, 2, 3, 4. \tag{38}$$

Furthermore, (u_1^*, u_2^*) are represented by

$$u_1^* = \min\left(1, \max\left(0, \frac{\beta_p (S_T + I_T) S_N}{N \Psi_1}[\lambda_2 - \lambda_1] + \frac{\beta_p (S_T + I_T) S_N}{N \Psi_1}[\lambda_4 - \lambda_3]\right)\right), \tag{39}$$

$$u_2^* = \min\left(1, \max\left(0, \frac{\alpha \beta_h (I_N(1-\eta) + I_T) S_N}{N \Psi_2}[\lambda_3 - \lambda_1] + \frac{\alpha \beta_h (I_N(1-\eta) + I_T) S_T}{N \Psi_2}[\lambda_4 - \lambda_2]\right)\right). \tag{40}$$

Proof According to Pontryagin's Maximum Principle (Fleming & Rishel, 1975; Lukes, 1982; Pontryagin et al., 1985), we first differentiate the Hamiltonian operator H, with respect to states. Then, the adjoint system can be written as,

$$\frac{\partial \lambda_1}{\partial t} = -\frac{\partial H}{\partial S_N}, \quad \frac{\partial \lambda_2}{\partial t} = -\frac{\partial H}{\partial S_T}, \quad \frac{\partial \lambda_3}{\partial t} = -\frac{\partial H}{\partial I_N}, \quad \frac{\partial \lambda_4}{\partial t} = -\frac{\partial H}{\partial I_T}. \tag{41}$$

The terminal condition of the adjoint equations is given by $\lambda_i(t_f) = 0, \quad i = 1, 2, 3, 4$.

To obtain the necessary conditions of optimality, we differentiate the Hamiltonian operator H with respect to $u_1(t)$ and $u_2(t)$ and set them equal to zero. Thus,

Making $u_1^*(t)$ subject of formulae,

$$u_1^*(t) = \frac{\beta_p(S_T + I_T)S_N}{N\Psi_1}[\lambda_2 - \lambda_1] + \frac{\beta_p(S_T + I_T)I_N}{N\Psi_1}[\lambda_4 - \lambda_3] + \left(\frac{-w_{11} + w_{12}}{\Psi_1}\right). \tag{42}$$

Making $u_2^*(t)$ subject of formulae,

$$u_2^*(t) = \frac{(\alpha\beta_h(I_N(1-\eta) + I_T))S_N}{N\Psi_2}[\lambda_3 - \lambda_1] + \frac{(\beta_h(I_N(1-\eta) + I_T))S_T}{N\Psi_2}[\lambda_4 - \lambda_2] + \left(\frac{-w_{21} + w_{22}}{\Psi_2}\right). \tag{43}$$

To determine the explicit expression for the optimal control without $w_{11}(t), w_{12}(t), w_{21}(t)$ and $w_{22}(t)$, a standard optimality system is utilised (Fleming & Rishel, 1975). The specific characterisation of the optimal controls $u_1^*(t)$ and $u_2^*(t)$.

$$\begin{aligned} u_1^*(t) &= \min\left\{1, \max\left\{0, \frac{\beta_p(S_T + I_T)S_N}{N\Psi_1}[\lambda_2 - \lambda_1] + \frac{\beta_p(S_T + I_T)I_N}{N\Psi_1}[\lambda_4 - \lambda_3]\right\}\right\} \\ u_2^*(t) &= \min\left\{1, \max\left\{0, \frac{(\alpha\beta_h(I_N(1-\eta) + I_T))S_N}{N\Psi_2}[\lambda_3 - \lambda_1] + \frac{(\beta_h(I_N(1-\eta) + I_T))S_T}{N\Psi_2}[\lambda_4 - \lambda_2]\right\}\right\} \end{aligned} \tag{44}$$

The optimality system consists of the state system coupled with the adjoint system with the initial conditions, the transversality conditions and the characterisation of the optimal control. Substituting $u_1^*(t)$ and $u_2^*(t)$ for $u_1(t)$ and $u_2(t)$ in equations (Equation 37) gives the optimality system. The state system and adjoint system have finite upper bounds. These bounds are needed in the uniqueness proof of the optimality system. Due to a priori boundedness of the state and adjoint functions and the resulting Lipschitz structure of the ordinary differential equations, we obtain the uniqueness of the optimal control for small t_f (Magombedze, Garira, Mwenje, & Bhunu, 2011). The uniqueness of the optimal control follows from the uniqueness of the optimality system. □

5.3. Numerical simulations

The optimality system is solved using an iterative method with Runge–Kutta fourth-order scheme. Starting with a guess for the adjoint variables, the state equations are solved forward in time. Then, these state values are used to solve the adjoint equations backward in time, and the iterations continue until convergence. The simulations were carried out using parameter values in Table 1 and the following values $\Phi_1 = 1.5, \Phi_2 = 0.35, \Phi_3 = 0.01, \Psi_1 = 0.0002, \Psi_2 = 0.00005$. The assumed initial conditions for the differential equations are $S_N = 0.4, S_T = 0.3, I_N = 0.2$ and $I_T = 0.1$.

Figure 6(a) represents the population of the susceptible non-sexting adolescents in the presence and absence of controls over a period of 20 years. The susceptible non-sexting adolescents have a slightly sharp increase during the interval 0–6 years, for both cases (with or without the control). Further, we note that the population of the susceptible non-sexting adolescents is higher when there is a control compared to a situation when there is no control.

In Figure 6(b), the population of the susceptible sexting adolescents in the presence and absence of the controls over a period of 20 years is presented. The susceptible sexting adolescents have a slightly sharp decrease over a period of 0–6 years, for both cases (with or without the control). Further, we note that the population of the susceptible sexting adolescents is higher when there is no control compared to a situation where there is a control. It is also worth noting that the population of the sexting susceptible adolescents goes to zero after 17 years of implementation of the controls.

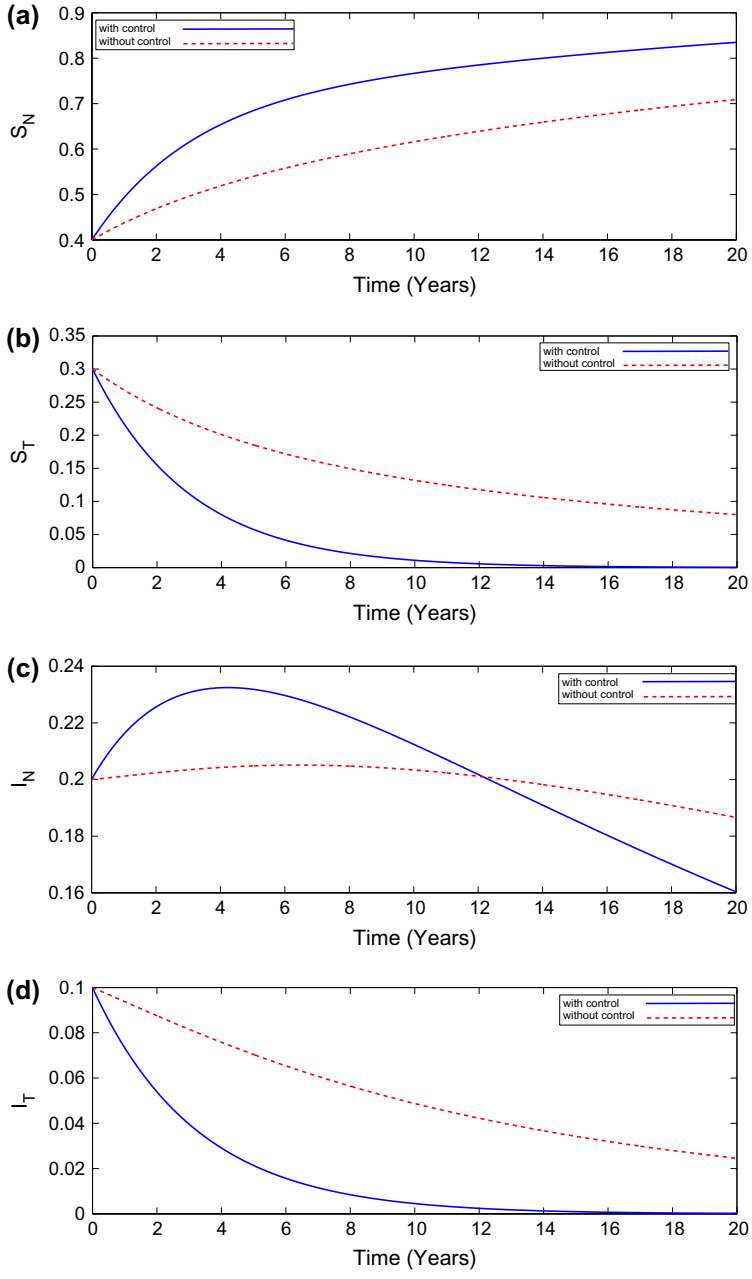


Figure 6. Dynamics of model system (Equation 29) showing the impact of filtering and awareness campaigns on HSV-2 dynamics and sexting.

Figure 6(c) represents the population of the infected non-sexting adolescents in the presence and absence of controls over a period of 20 years. The infected non-sexting adolescents have a sharp increase for the first 4 years for the case when there are controls, and then have a sharp decrease for the remaining period, that is 4–20 years. The infected non sexting adolescents have a moderate increase for the first 7 years for the case were there are no controls, then also have a slight decrease for the remaining period under review, that is 7–20 years. Further, we note that the population of the infected non-sexting adolescents is higher when there is a control compared to a situation where there is no control for the period 0–12 years. After 12 years, the population of the non-sexting adolescents in the presence of the controls begins to decrease sharply and also lesser than the case were there are no controls. Thus, change becomes to be significant in the society after 12 years amongst the adolescents who are infected, yet not into sexting.

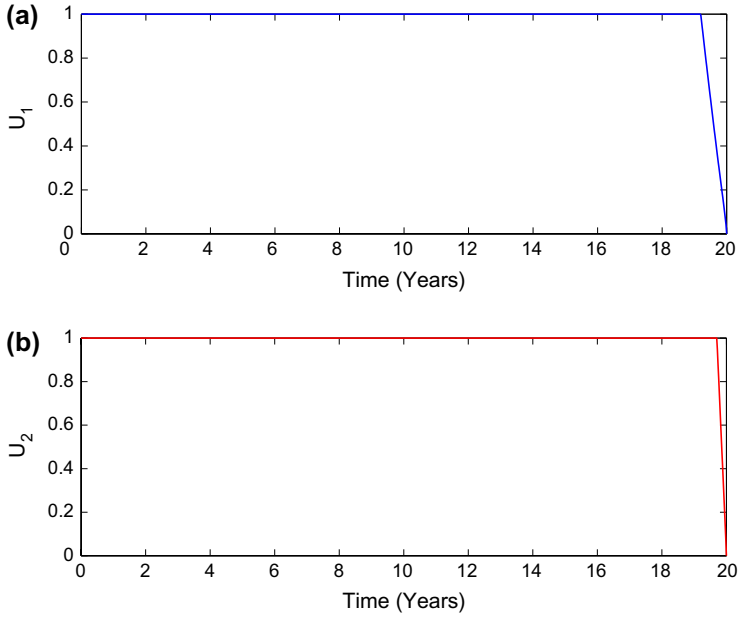


Figure 7. Control profiles for control functions (a) $u_1(t)$ and (b) $u_2(t)$.

In Figure 6(d), the population of the infected sexting adolescents in the presence and absence of the controls over a period of 20 years is presented. The infected sexting adolescents have a slightly sharp decrease over a period of 0–6 years, for both cases (with or without the control). Further, we note that the population of the infected sexting adolescents is higher when there is no control compared to a situation where there is a control, showing the effectiveness of the controls. It is also worth noting that the population of the sexting-infected adolescents goes to zero after 18 years of implementation of the controls.

Figure 7 represents the controls u_1 and u_2. These results suggests that more effort should be devoted to both strategies: "filtering" control u_1 and "awareness campaigns" u_2.

6. Discussion

Sexting is associated with health-jeopardising behaviours including sex with multiple partners, unprotected sex and STIs. It was found that adolescents are amongst the groups who are heavily involved in sexting, and they mostly join this bad behaviour due to peer influence. A mathematical model for assessing the effects of sexting and peer influence on the spread of HSV-2 amongst adolescents is developed. Comprehensive and robust mathematical techniques have been used to analyse the model's steady states. It has been established that the model has a disease-free equilibrium which is locally asymptotically stable when the associated reproduction number is less than unity. Sensitivity analysis of the reproduction number has been carried out. Results from the sensitivity analysis of the reproduction number suggest that an increase in the effective contact rate for HSV-2 infection and an increase in the effective contact rate for peer influence will have a very large influence on increasing the magnitude of the associated reproduction number. We also noted that quitting sexting by the adolescents would be very crucial in reducing the magnitude of the reproduction number. Numerical results have suggested that an increase in the effective contact rate for sexting results in an increase in the cumulative HSV-2 cases. Furthermore, the numerical simulations suggest that for us to be able to curtail the prevalence of HSV-2, we need to have a combination of high sexting quitting rate and low peer influence effective contact rate.

Our optimal control results show that the effectiveness of the combination of filtering and awareness campaigns within a population may influence HSV-2-infected and sexting cases of adolescents over a period of 20 years. Pontryagin's Maximum Principle is used to characterise the optimal levels of the two

controls and then numerically solved them. Overall, the application of the optimal control theory suggests that more effort should be devoted to both controls, filtering and awareness campaigns.

However, just like any other model, we cannot say the model is complete; it can be extended to include the issue of gender in terms of HSV-2 acquisition and infectiousness.

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