

Bifurcations and chaos in discrete-time gonorrhoea model

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Abstract. A deterministic epidemic model for the spread of gonorrhoea is investigated in discrete-time by taking into account the interval between successive clinical cases. It is shown that the discrete-time dynamical system exhibits far more complex dynamics than its continuous analogues. Stability analysis is obtained in order to investigate the local stability properties of the fixed points; it is verified that there are phenomena of Fold and Flip bifurcations. Numerical simulation tools are used in order to illustrate the stability analysis results and find some new qualitative dynamics. We come across the phenomenon of “intermittency route to chaos”. The density of infected individuals goes through quasi-periodicity and a strange attractor appears in the system. Chaos control is obtained in order to see how the male latex condom use during sexual intercourse affects the incidence of gonorrhoea. It is shown that male latex condom use stabilizes the chaotic vibrations of the system to a point where the number of infected individuals remains stable and is significantly small or zero, leading to the control of disease.

Keywords: Gonorrhoea dynamics, Dynamical system, Bifurcations, Chaotic dynamics, Strange attractor, Chaos control, Serial interval, Male latex condom

1 Introduction

Gonorrhoea is one of the oldest known human infections. The organism *Neisseria gonorrhoeae* was first described by Neisser in 1879 and cultivated in 1982 [25]. *Neisseria gonorrhoeae* causes an estimated 62 million cases of gonorrhoea worldwide each year [16]. Furthermore, the financial impact of gonorrhoea is high. Only the direct medical cost for gonorrhoea treatment in the United States is estimated at \$1,051,000,000 annually [9]. Infections due to *Neisseria gonorrhoeae* are a major cause of pelvic inflammatory disease (PID) in the United States. PID can lead to serious outcomes in women such as tubal infertility, ectopic pregnancy, and chronic pelvic pain. In men gonorrhoea can cause a painful condition called epididymitis in the tubes attached to the testicles [7]. Further, if left untreated, gonorrhoea can also spread to the blood and cause disseminated gonococcal infection (DGI), a condition that can be life threatening. Moreover, the organism *Neisseria gonorrhoeae* has the ability to develop resistance against all clinically useful antibiotics. William Smith [31] suggests that we are on the verge of a highly untreatable gonorrhoea epidemic.



Although gonorrhoea was easily cured with antibiotics years ago, however bacteria develop resistance to treatments. Furthermore, the World Health Organization recently put out an alert with regards to the reported cases of resistance to cephalosporin antibiotics - the last treatment option against gonorrhoea - in several countries including Australia, France, Japan, Norway, Sweden and the United Kingdom [40]. According to Dr. Manjula Lusti-Narasimhan from the Department of Reproductive Health and Research at WHO, gonorrhoea is becoming a major public health challenge due to the high incidence of infections accompanied by dwindling treatment options. And after the failure from this last effective treatment, as there are no new therapeutic drugs in development, if gonococcal infections become untreatable the health implications will be significant. Mathematical models have been widely used from epidemiologists over the years to predict epidemics of infectious diseases. Hethcote and Yorke [20] monograph is an excellent work in the context of gonorrhoea transmission dynamics and control. They used nonlinear differential equations to model the transmission dynamics of gonorrhoea in a heterosexually-active population with two distinct levels of sexual activity. Continuous-time epidemic models have been widely used over the years in the investigation of the transmission of infectious diseases due to their mathematical tractability ([5], [22], [26]). However, discrete-time models are often directly applicable to time-series data and may represent contacts, which are restricted to a specific time or time period more accurately [4]. Therefore, it may be easier to compare the output of discrete-time models with statistical real-world data. Several works have based their models on difference equations in order to investigate the gonorrhoea transmission dynamics ([13], [23]). However, discrete-time SIS epidemic models sometimes are capable of generating complex dynamics such as period-doubling and chaotic behavior, in contrast with continuous-time epidemic models. In this paper we study a discrete-time version of Hethcote and Yorke [20] gonorrhoea model using discrete time steps, based on the duration of the serial interval of gonorrhoea infection.

2 Basic Gonorrhoea Model Description

Hethcote and Yorke [20] studied a continuous-time dynamical system for the spread of gonorrhoea. They divided population into two groups, *females at risk* (N_f) and *males at risk* (N_m); and each group into two subgroups, *susceptible females* ($N_f S_f$) & *infective females* ($N_f I_f$) and *susceptible males* ($N_m S_m$) & *infective males* ($N_m I_m$). They modeled the dynamics of the spread of gonorrhoea by the 4-dimensional system:

$$\begin{aligned} N_f \dot{S}_f &= -\lambda_f \cdot S_f \cdot N_m I_m + N_f I_f / d_f & N_m \dot{S}_m &= -\lambda_m \cdot S_m \cdot N_f I_f + N_m I_m / d_m \\ N_f \dot{I}_f &= \lambda_f \cdot S_f \cdot N_m I_m - N_f I_f / d_f & N_m \dot{I}_m &= \lambda_m \cdot S_m \cdot N_f I_f - N_m I_m / d_m \end{aligned} \quad (1)$$

where, the sexually active population N_f and N_m is constant and equals the number of susceptible plus the number of infective individuals ($N_f = S_f + I_f$ and $N_m = S_m + I_m$); λ_f and λ_m indicate the transmission rate of infection of susceptible

females and males respectively; d_f and d_m indicate the average duration of infection for females and males respectively. Since the population is constant, the system (1) reduces to the 2-dimensional dynamical system:

$$\frac{dI_f}{dt} = \frac{\lambda_f}{r} (1 - I_f) I_m - \frac{I_f}{d_f}, \quad \frac{dI_m}{dt} = r \lambda_m (1 - I_m) I_f - \frac{I_m}{d_m} \quad (2)$$

where, $S_f = 1 - I_f$ and $S_m = 1 - I_m$ since the total population size remains constant and $r = N_f / N_m$. The limiting system (2) has two equilibrium points, a trivial and a non-trivial. If the nontrivial equilibrium point exists, it is asymptotically stable and gonorrhoea dies out. If the nontrivial equilibrium point does not exist, then the trivial equilibrium point is asymptotically stable and gonorrhoea remains endemic.

3 Discrete Gonorrhoea Model

According to Ramani et al. [28], discretizing an epidemic model has the following advantages: (a) the epidemic statistics are collected from given time intervals, not continuously, (b) the discrete-time models provide natural simulators for the continuous-time models, (c) with discrete-time models one can use the entire arsenal of methods that have been developed for the study of mappings and lattice equations, either from integrability and/or chaos points of view. There are several ways to discretize a continuous-time model. However, the approach of discretizing the differential equations of a dynamical system has the advantage that can keep track of the known properties of the continuous time system [19].

Thus, in order to discretize Hethcote and Yorke model, we replace dI_f / dt and dI_m / dt by the difference quotients $(I_{n+1}^f - I_n^f) / \delta$ and $(I_{n+1}^m - I_n^m) / \delta$ in the dynamical system (2). Time is measured in “generations”. We obtain a deterministic discrete gonorrhoea model defined by the 2-dimensional map:

$$I_{n+1}^f = I_n^f + \delta \left(\frac{\lambda_f}{r} (1 - I_n^f) I_n^m - \frac{I_n^f}{d_f} \right), \quad I_{n+1}^m = I_n^m + \delta \left(r \lambda_m (1 - I_n^m) I_n^f - \frac{I_n^m}{d_m} \right) \quad (3)$$

where, δ is the length of each discrete-time step, where the number of infective individuals grow by the addition of the newly infective individuals; I_n^f, I_n^m is the number of infective females and males in one time step respectively; I_{n+1}^f, I_{n+1}^m is the number of infective females and males at the next time step respectively; the infection rates of susceptible males and females (λ_f, λ_m) indicate the average number of individuals with whom an infectious individual makes adequate contact¹ during a unit time interval [3]. The map (3) has two fixed points, which are identical to the equilibrium points of the analogous continuous model (2), a disease-free equilibrium E_1 and an endemic equilibrium E_2 :

¹ Adequate contact is a direct or indirect contact that is sufficient for transmission of infection, if the individual contacted is susceptible. The concept of a sufficient contact is necessary since transmission of infection sometimes does not occur during sexual intercourse between an infective and a susceptible [20].

$$E_1 : (I_f^*, I_m^*) = (0, 0), \quad E_2 : (I_f^*, I_m^*) = \left(\frac{l_f l_m d_f d_m - 1}{l_m d_m r + l_f l_m d_f d_m}, \frac{(l_f l_m d_f d_m - 1)r}{l_f l_m d_f d_m r + l_f d_f} \right)$$

In order to study² the local behavior around each of the two fixed points, we calculate the Jacobian matrix at E_1, E_2 . If λ_1, λ_2 are the eigenvalues of the Jacobian matrix at each fixed point, then the fixed point is stable, if $|\lambda_1| < 1$ and $|\lambda_2| < 1$. By using Vieta's equations: $\lambda_1 + \lambda_2 = Tr(J)$, $\lambda_1 \cdot \lambda_2 = Det(J)$ (i.e. $TR(J)$ and $Det(J)$ are the trace and the determinant of the Jacobian matrix) and applying Jury's conditions [21], the fixed point is linearly asymptotically stable if and only if:

$$-TrJ(I_f^*, I_m^*) < 1 + DetJ(I_f^*, I_m^*), \quad TrJ(I_f^*, I_m^*) < 1 + DetJ(I_f^*, I_m^*) \quad \text{and} \quad DetJ(I_f^*, I_m^*) < 1$$

5 Numerical Simulations for the Discrete-Time Model

A series of numerical simulations³ (using the numerical simulation tools: *parametric basins of attraction, bifurcation diagrams, phase plots* and *Lyapunov exponent diagrams*) are introduced in order to illustrate the results of the analytical stability analysis and to find some new qualitative dynamics of the discrete-time model (3) as the parameters are varied. The rates of infection λ_m and λ_f should be different, because transmission efficiency is gender dependent. The average probability of transmission of gonococcal infection during a single sexual exposure (a) from an infectious woman to a susceptible man is estimated to be about 0.2 – 0.3, while (b) from an infectious man to a susceptible woman is about 0.5 – 0.7 [37]. Hence, we use the following values for the rate of infection parameters: $\lambda_f = 0.6$ and $\lambda_m = 0.25$. However, the probability of transmission of gonococcal infection is increased for individuals, who have ever had gonorrhea or other STI, for individuals who are street-involved youth and for individuals having sex with many partners, with sex workers, or with a partner coming from a country where gonorrhea is frequent [17]. The average durations of infection d_m and d_f have also to be different, because (a) 90% of all the men who have had gonococcal infection notice symptoms within a few days after exposure and promptly seek medical treatment, while (b) up to 75% of women with gonorrhea fail to have symptoms and remain untreated for some time [27]. In particular, when symptoms occur in men, they usually occur 3-5 days after sexual contact with an infected individual; while women, who develop symptoms, usually experience them within 10 days of sexual contact with an infected individual [38]. So we fix the average duration of infection for females and males at $d_f = 10$ and $d_m = 3$ respectively.

² The results of the stability analysis have been exhibited by using the software package *wxMaxima 12.04.0* (<http://maxima.sourceforge.net/>).

³ Numerical simulations have been exhibited by using the software *E&F Chaos 1.02* [12].

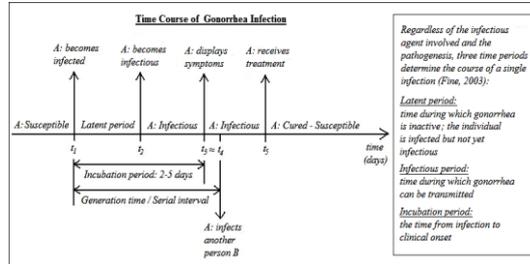


Fig. 1. Time course of a single gonorrhea infection; Individual A becomes infected, transmits the infection to individual B and receives treatment.

Gonorrhea affects males and females almost equally⁴. So we assume that the number of males and females at risk (N_m, N_f) is equal with ratio $r = 1$. We also assume that the initial number of infective individuals is the same both for males and females. Therefore, we use for initial conditions the values $(I^f_0, I^m_0) = (0.5, 0.5)$. Finally, in order to accurately describe the gonorrhea transmission dynamics, the size of the discrete time step should match the epidemiology of the disease [34]; that is, whether the dynamic of infection is a matter of days or hours. Thus, we assume that the discrete time step δ corresponds to the generation time of gonorrhea, that is, the time from the moment one person becomes infected until that person infects another person [29]. This time interval is well-known as the serial interval (Figure 1), that is, the time period between successive clinical cases [14]. In other words, this is the average time between the observation of symptoms of gonorrhea in one person and the observation of symptoms in another person that has been infected from the first. The serial interval is important in the interpretation of infectious disease surveillance and trend data, in the identification of outbreaks and in the optimization of quarantine and contact tracing [14]. Furthermore, the symptoms of gonorrhea usually appear two to five days after infection (i.e. incubation period) [25]. Thus, since an infected individual remains infectious until he/she receives treatment, we assume that infections occur during the infectious period (Figure 1). Moreover, although a range of values for the serial interval is possible, the average serial interval can be estimated as: *(average incubation period) + (half the average infectious period)*, assuming that the maximum infectiousness occurs at the middle of the infectious period [30]. So the serial interval could be estimated by the incubation period. Therefore we define the length of the discrete time step between infection and subsequent transmission as $2 < \delta < 5$ days. Using these values for our parameters, we observe that the dynamics of the basic model (2) alters significantly in discrete time for time interval length between successive clinical cases ($2 < \delta < 4$) days, as the rate of infection of susceptible females increases (Figure 2).

⁴ Global estimated incidence of gonorrhea, occurred in 1999, is 62.35 million infected people annually. Particularly gonorrhea affected 33.65 women and 28.70 men [39].

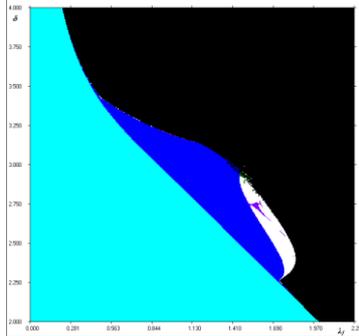


Fig. 2. The basins of attraction diagram for $\delta \in [2, 4]$ and $\lambda_f \in [0, 2.25]$.

For small values of the infection rate parameter ($0 < \lambda_f < 0.4$), the solutions converge either to a disease-free fixed point or to an endemic fixed point (light-blue area) for every value of time interval between clinical cases. For average values of the infection rate parameter ($0.4 < \lambda_f < 1.46$), as λ_f increases, the light-blue area is being replaced with the dark-blue area and the solutions converge to an attracting cycle of period 2. Moreover, for specific step size values ($2.25 < \delta < 3$), further increase in the infection rate parameter λ_f gives rise to non-periodic behavior (white area). For large values of the infection rate parameter ($\lambda_f > 1.85$) as the value of λ_f increases, any periodic and non-periodic behavior is being replaced with divergence to infinity (black area). This abrupt behavior is not meaningful, but it could be taken as some kind of catastrophe causing the extinction of the infected population. Thus, for sufficiently low infection rate of susceptible females, the behavior of solutions of the discrete-time model is qualitatively the same with the basic model. However, as the infection rate increases, the discrete-time model exhibits the same behavior as the continuous-time model only for certain short time interval between successive clinical cases of gonorrhea ($\delta < 2.25$).

Fixing the time period between clinical cases at $\delta = 2.65$ ($2 < \delta < 4$ days) and let the rate of infection of susceptible females parameter increasing in the interval $\lambda_f \in [0, 1.59]$, we observe bifurcations occurring in the system (Figure 3).

The value $\lambda_f \approx 0.1333$ is a bifurcation point at which a “fold” bifurcation occurs: For exceptionally small values of the varying parameter ($\lambda_f < 0.1333$), the disease-free fixed point $E_1: (0, 0)$ is locally asymptotically stable (stable node) and the endemic (negative) fixed point $E_2: (I_f^*, I_m^*)$ is unstable (saddle). Some solutions converge to the attracting disease-free fixed point; there are no infective individuals and gonorrhea dies out. Hence, the initial infective population sizes $(I_f^0, I_m^0) = (0.5, 0.5)$ lead to the extinction of the disease due to the low probability of infection. Near the value $\lambda_f \approx 0.1333$ both fixed points $E_1: (0, 0)$ and $E_2: (-0.0001, -0.0001)$ undergo a “fold” bifurcation and become non-hyperbolic with eigenvalues of the Jacobian matrix $(\lambda_1 = -0.1483, \lambda_2 = 0.9999 \approx +1)$ and $(\lambda_1 = -0.1483, \lambda_2 = 1.0001 \approx +1)$ respectively. Hence, for this critical value, the system has only one non-hyperbolic fixed point $E_1 \approx E_2: (0, 0)$. For $0.1333 < \lambda_f < 1.1035$, the system has again two fixed points, the trivial

and a non-trivial positive fixed point. The fixed points have exchanged their stability. The disease-free fixed point E_1 is now unstable (saddle), while the endemic fixed point E_2 is locally asymptotically stable (stable node). The initial infective population sizes $(I_0^f, I_0^m) = (0.5, 0.5)$ converge to the attracting endemic fixed point, where both infected males and females are fixed in time. Moreover, the number of infective females is larger than the number of infective males ($I_f^* > I_m^*$) likely due to the fact that the infection rate of females is larger than the infection rate of males ($\lambda_f > \lambda_m$) and the duration of infection is larger in females than in males ($d_f > d_m$). As the parameter λ_f increases in this interval, the number of infective individuals (I_f^*, I_m^*) increases continuously and gonorrhea remains endemic. Near the value $\lambda_f \approx 1.1035$ the saddle disease-free fixed point $E_1: (0,0)$ becomes non-hyperbolic ($\lambda_1 = -0.9999, \lambda_2 = 1.8516$) and for $\lambda_f > 1.1035$ is an unstable node. The value $\lambda_f \approx 1.2961$ is a bifurcation point at which a “flip” bifurcation occurs: At $\lambda_f \approx 1.2961$ the endemic fixed point $E_2: (0.8329, 0.3845)$ undergoes a “Flip bifurcation” and becomes non-hyperbolic with eigenvalues of the Jacobian matrix ($\lambda_1 = -0.9999 \approx -1, \lambda_2 = -0.0207$). For $1.2961 < \lambda_f < 1.59$ the endemic fixed point E_2 becomes unstable (saddle) and a stable cycle of period 2 appears in the system. Both infective males and females, now, converge to different 2-period cycles, while both periodic cycles become wider, as the parameter increases at this particular interval.

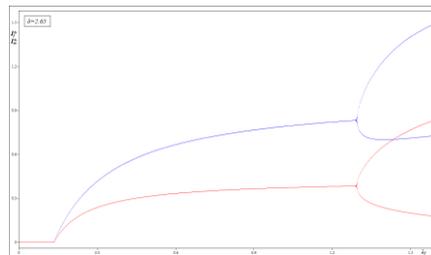
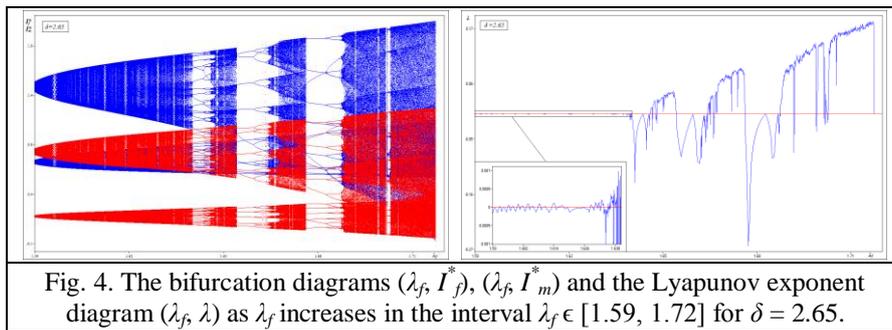


Fig. 3. The bifurcation diagrams (λ_f, I_f^*) and (λ_f, I_m^*) for $\delta = 2.65$ as λ_f increases in the interval $\lambda_f \in [0, 1.59]$.

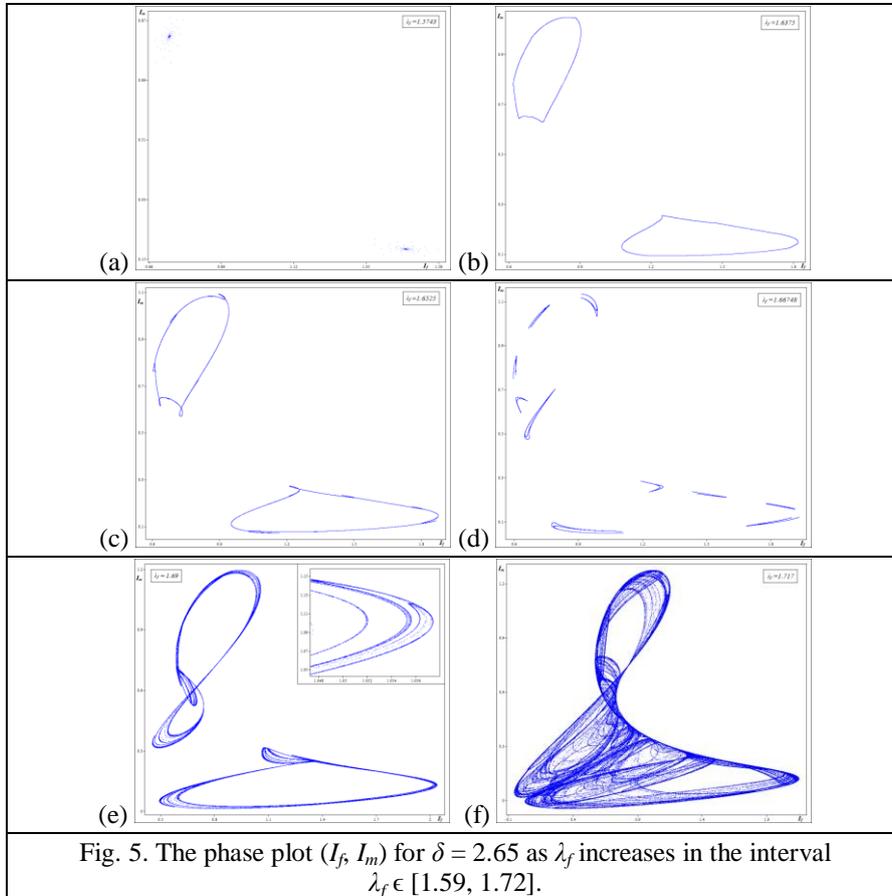
For higher values of the rate of infection of susceptible females $\lambda_f \in [1.59, 1.72]$, we come across the phenomenon of “intermittency route to chaos”, which according to Manneville and Pomeau [24], is characterized by regular (laminar) phases alternating with irregular bursts. In particular, as the varying parameter increases, the endemic fixed point remains unstable (saddle), while periodic behavior of high periods, cascades of period-doubling bifurcations and deterministic chaos appear eventually in both infected males and females (Figure 4). For $1.59 < \lambda_f < 1.6402$ the Lyapunov exponents vary among negative and exceptionally small positive values $\lambda_i^+ < 0.001$ and the behavior of solutions appears to be slightly chaotic. For $1.6402 < \lambda_f < 1.6544$ the map exhibits the familiar infinite sequence of period-doubling bifurcations ($32 \cdot 2^n$): (32-period cycle, 64-period cycle, 128-period cycle, etc.) followed by chaotic oscillations, where the Lyapunov exponents take higher positive values $\lambda_i^+ < 0.05$. At $\lambda_f \approx 1.6544$ a second series of period-doubling bifurcations ($10 \cdot 2^n$): (10-period

cycle, 20-period cycle, 40-period cycle, etc.) route to chaos once again, while the Lyapunov exponents at this parameter interval ($1.6544 < \lambda_f < 1.6763$) vary among larger positive values $\lambda_i^+ < 0.1$. At $\lambda_f \approx 1.6763$ another series of period-doubling bifurcations ($12 \cdot 2^n$): (12-period cycle, 24-period cycle, 48-period cycle, etc.) lead to even more chaotic behavior, where the oscillations in the density of infected individuals can be hard to predict. The Lyapunov exponents take even larger positive values and reach the maximum value $\lambda_{\max} \approx 0.1849$ for the parameter value $\lambda_f \approx 1.7172$ for which the variations in the number of gonorrhea cases are the less predictable (fully developed chaos). At this point, the exceptionally high infection rate of susceptible females leads the number of infected individuals sometimes close to extinction and other times close to overgrowth (i.e. gonorrhea outbreaks).



Furthermore, as the rate of infection of susceptible females increases in the interval $\lambda_f \in [1.59, 1.72]$ for the same time interval between clinical cases ($\delta = 2.65$), the system goes through quasi-periodicity and a strange attractor appears in the system (Figure 5). The stable period-2 orbit (Figure 5.a) near the value $\lambda_f \approx 1.59$ loses stability via a supercritical Neimark-Sacker bifurcation, giving rise to two attracting closed invariant curves. At this point the number of infected males and females oscillates between all the states of the two invariant curves. The invariant curves grow in size (i.e. the amplitudes of oscillations in the number of infected individuals are increasing), interact with the saddle non-trivial fixed point $(I_f^*, I_m^*) \approx (0.86, 0.39)$ and near the value $\lambda_f \approx 1.6375$ become noticeably kinked (Figure 5.b). The kinked curves have a split, lock into a stable periodic orbit due to the first sequence of period-doubling occurring in the system ($32 \cdot 2^n$) and reappear slightly deformed (Figure 5.c). They have another split, due to the second series of period-doubling ($10 \cdot 2^n$), which gives rise to a motion of period-10 (Figure 5.d). The motion of period-10 forms into two weakly chaotic contiguous bands (Figure 5.e), while successive enlargements of the attractor can show its fine structure, which looks identical in all scales (i.e. self-similarity). The chaotic contiguous bands become more and more complicated, merging to form a strange attractor (Figure 5.f) for a value of the varying parameter ($\lambda_f \approx 1.717$) in the chaotic domain (Figure 4). For higher values of the infection rate parameter, the successive iterates diverge to infinity

(i.e. both infected males and females become extinct through some kind of catastrophe) and the attractor disappears.



So, we observe that the behavior of the discrete-time gonorrhea model (3) differs significantly from its continuous counterpart (2). Particularly, a time period $2 < \delta < 4$ days between successive clinical cases of gonorrhea and a sufficiently large infection rate of susceptible females allow for infinite sequences of period-doubling and chaotic behavior in the density of infected individuals.

6 Chaos Control

Chaos may be undesirable, as the chaotic oscillations in the density of infected individuals can make the disease uncontrollable and, consequently, harmful to the people's health throughout the world. Therefore, the number of infected individuals needs to be under control. A method of controlling chaos has been

proposed by Güémez and Matias [18], known as the G.M. algorithm⁵, which performs changes in the system variables allowing the stabilization of chaotic behavior. In addition, Codreanu and Danca [11] applied the G.M. method to a prey-predator model supporting its use in biological systems. The G.M. control algorithm consists of the application of a proportional feedback⁶ (γ) to the variables of the system in the form of pulses [18]. We apply the G.M. control algorithm to the discrete map (3) by modifying the system variables I_n^f, I_n^m in the following form: $I_n^f \rightarrow I_n^f(1+g_1)$, $I_n^m \rightarrow I_n^m(1+g_2)$. Hence, our discrete-time gonorrhoea model (3) becomes:

$$\begin{cases} I_{n+1}^f = I_n^f(1+g_1) + d \left(\left(I_f/r \right) \left(1 - I_n^f(1+g_1) \right) I_n^m(1+g_2) - \left(I_n^f(1+g_1)/d_f \right) \right) \\ I_{n+1}^m = I_n^m(1+g_2) + d \left(r I_m \left(1 - I_n^m(1+g_2) \right) I_n^f(1+g_1) - \left(I_n^m(1+g_2)/d_m \right) \right) \end{cases} \quad (4)$$

where, γ_1, γ_2 represent the strength of the feedback for I_f, I_m . For sexually active persons, male latex condoms are the most commonly used contraceptive method to prevent⁷ sexually transmitted infections [10]. So from a practical point of view, the modification in the system variables could be interpreted as the use of male latex condoms during each sexual intercourse. Hence, the new terms $\gamma_1 I_n^f, \gamma_2 I_n^m$ are associated with condom use during sexual intercourse protecting males and females from gonorrhoea transmission and reducing the number of infected individuals ($-1 \leq \gamma_1, \gamma_2 < 0$), while the terms I_n^f, I_n^m are associated with sexual intercourse without condom use. Furthermore, for the sake of simplicity, we assume that the protection from gonorrhoea transmission by condom use from female to male and vice versa is the same ($\gamma_1 = \gamma_2 = \gamma$). The condition $\gamma = -1$ corresponds to an ideal situation where all sexually-active individuals use latex condoms during sexual intercourse consistently and correctly.

Thus, in order to see how the condom use affects the incidence of gonorrhoea, we apply the G.M. method for the parameter values $\delta = 2.65$ and $\lambda_f = 1.717$ (the other parameters remain unchanged) for which the system's behavior is chaotic (Figure 5.f). Let the control parameter (γ) to vary. We illustrate the results by plotting the bifurcation diagram (Figure 6) along with the time series before ($\gamma = 0$) and after ($\gamma < 0$) the action of chaos control algorithm (Figure 7). Without condom use during sexual intercourse ($\gamma = 0$) the number of infective males and females appears irregular oscillations (Figure 7). As the intensity of pulses increases (i.e. condom use increases), the control parameter (γ) is taking smaller and smaller negative values, some part of I_f or I_m is injected from the map depending on the value of I_n^f or I_n^m at that moment and through sequences of reverse period-doubling bifurcations, the chaotic domains give rise to regular behavior (Figure 6), where the oscillations in the density of infected individuals become predictable. Particularly, near the value $\gamma \approx -0.066$ the behavior of

⁵ Güémez and Matias [18] considered the logistic map and the exponential map.

⁶ Depending on the sign of γ , in particular, some part of the system variables is injected or withdrawn depending on the value of the variables at the moment n [18].

⁷ In vitro studies indicate that latex condoms provide an effective mechanical barrier to passage of infectious agents comparable in size to or smaller than STI pathogens [8].

solutions becomes periodic (cycle of period-2). In the parameter interval $-0.5432 < \gamma < -0.2091$ solutions converge to endemic equilibrium (Figure 7).

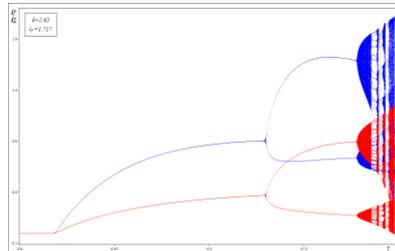


Fig. 6. The bifurcation diagrams $(\gamma, I_f^*), (\gamma, I_m^*)$ as γ decreases in the interval $\gamma \in [-0.6, 0]$, for $\delta = 2.65$ and $\lambda_f = 1.717$.

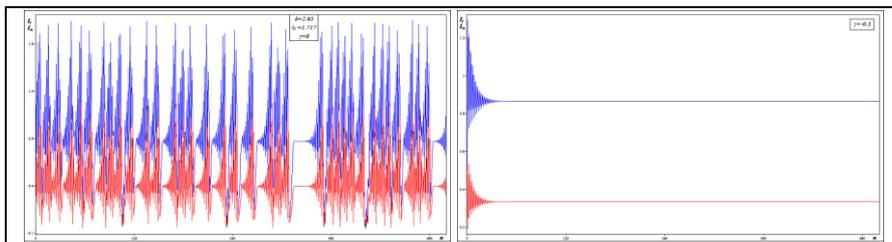


Fig. 7: The time series $(n, I_f), (n, I_m)$ for the first $n \in [0, 500]$ iterations; for $\gamma = 0$ (without control) and $\gamma = -0.3$ (with control), for $\delta = 2.65$ and $\lambda_f = 1.717$.

Finally, the decline in the number of infective individuals leads to the end of the disease (gonorrhoea-free equilibrium) for $-1 < \gamma < -0.5432$ (Figure 6). Hence, for exceptionally high infection rate of susceptible females and time interval between clinical cases $2 < \delta < 4$ days, as the condom use during sexual intercourse increases slowly among individuals, the oscillations in the number of infective males and females decrease rapidly, leading to the reduction of gonorrhoea incidence and the control of disease. So, we observe that condom use, just by a fraction of the population 50%, can reduce substantially the risk of gonorrhoea transmission even for exceptionally high infection rates.

Concluding Remarks and Discussion

In this paper we have discretized the gonorrhoea model of Hethcote and Yorke [20] and studied its dynamical characteristics using as a discrete-time step the interval between successive clinical cases (i.e. serial interval). We showed that the discrete-time model could result in a much richer set of patterns than the corresponding continuous-time model. The analytical stability analysis and the numerical simulation results showed that the discrete-time model undergoes: “fold”, “flip” bifurcations and the number of infective males and females can behave chaotically. In particular, we showed that: (a) for low rate of infection of susceptible females gonorrhoea remains endemic or dies out, while (b) for high

rate of infection of susceptible females chaotic oscillations and gonorrhoea outbreaks appear in the system.

The results of our study reflect the real-world large fluctuations, which appear in the number of gonorrhoea cases throughout the years. For instance, in Sweden from 2007 to 2011 the number of gonorrhoea cases increased by 48% (from 642 to 951 cases) [35]. The factors that might have been contributed to this increase in gonorrhoea incidence seem to be: (a) the increased number of sexual partners over time, (b) the increased number of new casual sexual partners and (c) the low level of condom use with casual sexual partners [33]. Moreover, Alaska's outbreak of *Neisseria gonorrhoeae* (GC) infection began in 2008 and peaked in 2010 with a total 1,273 GC cases reported to Alaska Section of Epidemiology [2]. The 2009 case rate demonstrated a 69% increase from the 2008 rate, representing the greatest single-year increase in reported GC infection in Alaska since the 1970s; the rate increased in both sexes, among all races, in all age groups and in nearly all regions of the state [1]. Another example is the variations in the number of gonorrhoea cases in the Onondaga County, NY, USA. According to the Onondaga County Health Department (OCHD) the number of cases of gonorrhoea more than doubled in the county between 2011 and 2012. There were 602 cases in the first nine months up from 253 cases for the same period of 2011 [32]. Hence, the proposed discrete-time model seems to be more effective in practice and gonorrhoea, despite the fact that it is a non-fatal disease, is likely to have a strong negative effect on life history evolution. Moreover, the results of our study show that the time interval between successive clinical cases is important in case of gonorrhoea emergency situations. However, few are known about clinical onset serial intervals of gonorrhoea. Therefore, collected data on serial intervals of gonorrhoea could provide useful information to guide any public health action.

Moreover we have stabilized the unstable periodic orbits, existing within the strange attractor and the unstable steady states (both endemic and disease-free), using a series of proportional feedbacks on the system's variables. The chaos control results could be definitely regarded as those, which are obtained with the use of male latex condom during sexual intercourse. Our chaos control results show that condom use reduces the risk of gonorrhoea transmission to a point where the number of infected individuals remains stable and is significantly small or zero. This reflects what many studies have shown, such as Barlow's study [6], which showed that a 71% reduction in gonorrhoea was associated with consistent and correct condom use (i.e., for every 100 cases of gonorrhoea infection that would happen without condom use, only 29 would happen when condoms are used consistently). However, one of the paradoxes in modeling infectious diseases is that, despite their quantitative nature, the best that we can often expect is qualitative insights [15]. Quantifying the relation between the number of condoms used and the incidence of gonorrhoea is often difficult. For instance, Warner et al. [36] reviewed studies, published from 1966 to 2004, to assess risk reduction for gonorrhoea associated with male condom use. They found that, although most studies showed that condom use was associated with reduced risk for gonorrhoea among men and women, however the exact

magnitude of risk reduction is difficult to quantify because of limitations and variations in the methods and design of these studies.

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