How Much Variability Can an Impulsive Threshold Sustain? Malaria Spraying as an Example

Adélaïde G. Gunn and Stacey R. Smith?

Abstract Impulsive differential equations are a useful tool for assessing timeliness of regular interventions, but the question of stochasticity in the timing and nature of the impulses has not been investigated. We use a previously published model of malaria as a baseline to investigate varying three key parameters: the time of the impulse, the duration between impulses and the degree of effectiveness of the impulse. Surprisingly, the model remains impervious to most biologically reasonable variations. However, we also showed that some extreme theoretical possibilities—such as very small durations or impulses that go backwards in time—can lead to unexpected outcomes. The malaria model can withstand large stochastic variations in the impulse parameters, suggesting that impulsive differential equations are fairly robust, at least when the virulence of the disease is high.

Keywords Malaria · Impulsive differential equations · Stochasticity

1 Introduction

Impulsive differential equations are a useful tool for including semi-discrete effects that describe short interruptions to a continuous process [1, 2]. The duration of such interruptions is assumed to be negligible, which is a reasonable assumption when the cycle time is large compared to the duration of the impulsive approximation [3]. Impulsive differential equations are characterised by two key factors: the time of the

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impulse and the strength of the impulsive effect. Either of these could be subject to variations, perhaps significantly.

Stochastic models, though more complex than their deterministic counterparts, are more realistic and asymptotically tend to their deterministic cousins [4]. Stochastic models lead to varying, non-equilibrium outcomes, can hasten disease extinction and result in resonance oscillations [5]. Stochastic effects have been widely investigated in continuous differential equations, but investigation of the impact of stochasticity on the thresholds of semi-discrete modelling is limited, and very little work has been done on the comparison between continuous and semi-continuous systems in a stochastic context. We use an impulsive malaria model as an example in order to investigate this problem.

Malaria is an infectious disease transmitted to humans primarily by the bite of female mosquitos of the genus *Anopheles* infected by a parasite of the genus *Plasmodium* [6, 7]. The mild symptoms present as fever, chills, headaches and respiratory difficulties, whereas the more severe symptoms include jaundice, kidney failure, impaired consciousness and abnormal bleeding [6, 7, 9]. The resulting infection in humans can be entirely curable and preventable when addressed early and effectively [6]. Nonetheless, the burden of the disease in African countries causes numerous fatalities, particularly in pregnant women and children under five [7, 9]. As of 2021, 50% of the world population was at risk of malaria, but 95% of the 247 million cases and 96% of the 619 000 deaths were in the African region. The predominant microorganism in Africa, *P. falciparum*, causes more severe cases of malaria and thrives in the local climate year-round. Poor access to resources and unstable socioeconomic conditions render preventative strategies hard to implement consistently and effectively. The COVID-19 pandemic significantly increased the malaria burden in 2020 and 2021 in Africa, due to interruptions in control measures [8]. Countries with higher gross domestic product (GDP) benefit from high-quality intervention and infrastructure, which reduces their malaria incidence compared to countries with lower GDPs [10, 11].

Malaria prevention and control strategies that are cost-effective and easily implemented are urgently needed to combat the severe consequences in public health and the economy that have been present for decades already in Africa [12]. Vector-control strategies, such as long-lasting insecticide-treated nets (LLINs) and indoor residual spraying (IRS), are effective against malaria transmission and a manageable and sustainable option for many African communities [13]. IRS has had a significant impact worldwide in the Global Malaria Eradication Campaign [7]. IRS is effective in unstable malaria conditions [14]: a meta-analysis concluded that when IRS is reapplied consistently and includes DDT (dichloro-diphenyl-trichloroethane), it could reduce risk of malaria by 65%, [10]. These strategies show a lowered incidence of malaria despite a minor effect on prevalence, resulting in the need for increased research in vector-control strategies [13].

Mathematical modelling of malaria is a crucial tool to help optimise management strategies by making predictions. The first model for malaria was developed by Ross in 1911, where humans can move from the susceptible to the infected state and back to the susceptible state (SIS), and where mosquitos only move from susceptible to

infected (SI) [15]. In 1957, the Ross model was modified by Macdonald to include the incubation period for the parasite to make a mosquito infectious, thereby following an SEI framework for mosquitos, where an individual must go through the exposed state before reaching the infected state [16]. Anderson and May improved the model by adding an exposed category to the humans, following an SEIS model, in addition to the mosquitos following an SEI model [17]. The increased complexity from Ross's initial model led to a decrease in the reproductive ratio (R_0), which leads to lower prevalence in the long term [18]. R_0 represents the number of cases that result from one infected individual and is an important indicator used to compare interventions [19]. In search of effective eradication techniques, more complex models have been developed based on the Ross–Macdonald and Anderson–May models to include various socio-economic, demographic, geographic and environmental factors, although no model can be exhaustive due to the enormous complexity of the malaria dynamics [18, 20]. The idea that lowering mosquito numbers below a certain threshold can control malaria was first brought up by Ross and is the basis of disease modelling today [15, 18, 20]. The early Ross–Macdonald model focused on adult female mosquitos as the most effective target, which led to the implementation of IRS as a large-scale effort [18, 20]. In order to effectively apply these concepts to dynamic and unpredictable populations where known techniques are insufficient, more recent models have included stochasticity, which was not included in the earlier models and can provide more realistic predictions [20, 21].

Previously, we used a system of impulsive differential equations to determine the ideal intervals to apply IRS, providing valuable insight to better benefit from IRS [22]. However, this model excludes stochastic noise in the impulses, which may alter the results over a long-term period. This variation may come from irregular application of insecticides, the delay in reapplication of IRS and the quality of the sprayed insecticide. Even with optimistic outcomes from modelling, results will be worse than expected when there is a lack of resources and awareness [23]. Furthermore, vector-control strategies are strongly influenced by the local population size and ecology, so an IRS framework may not be compatible from one region to another [24]. Studying the modelling of IRS, including various degrees of randomness within key parameters, will lead to further comprehension that better represents the reality of communities with insufficient infrastructure in order to explore the accuracy of malaria predictions.

2 The Model

We use the model from Smith? and Hove-Musekwa as our baseline [22]. This baseline model is built on the biological interactions between the different classes of humans and mosquitos. The system of differential equations includes susceptible (S), infected (I) and recovered (R) humans, as well as susceptible (M) and infected (N) mosquitos. Humans recover without immunity at rate h or become immune temporarily at rate α before returning to the susceptible state at rate δ. A susceptible

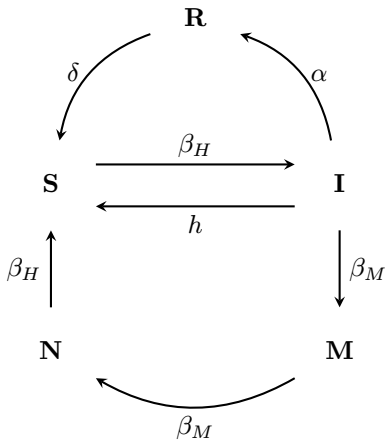


Fig. 1 A schematic representation of the malaria model (1). This illustration excludes the birth and death rates for clarity

individual is infected at rate β_H, and susceptible mosquitos are infected at rate β_M. π is the human birth rate, μ_H is the human background death rate, γ is the human death rate from malaria, Λ is the mosquito birth rate, and μ is the mosquito death rate. The model is illustrated in Fig. 1 and given by the following set of differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \beta_H SN + hI + \delta R - \mu_H S, \\
\frac{dI}{dt} &= \beta_H SN - hI - \alpha I - (\mu_H + \gamma)I, \\
\frac{dR}{dt} &= \alpha I - \delta R - \mu_H R, \\
\frac{dM}{dt} &= \Lambda - \mu M - \beta_M MI, \\
\frac{dN}{dt} &= \beta_M MI - \mu N.
\end{aligned} \tag{1}$$

Finally, we add impulses that represent the addition of insecticide via the IRS strategy. We can do this by adding conditions where the addition of insecticide removes both susceptible and infected mosquitos at a rate r (where $0 \leq r \leq 1$) for a given time of application t_k ($k = 0, 1, 2, 3, ...$). Therefore, immediately following a spraying event, the system undergoes the impulsive conditions [22]:

$$\begin{aligned}
\Delta M &= -rM^-, \\
\Delta N &= -rN^-.
\end{aligned} \tag{2}$$

When $t = t_k$, $\Delta M = M^+ - M^-$, where $M^- \equiv M(t_k^-)$ and $M^+ \equiv M(t_k^+)$. The period between impulses is defined as $\tau \equiv t_{k+1} - t_k$ (assumed constant at baseline).

3 Numerical Simulations

For theoretical analysis of the baseline model (1), see Smith? and Hove-Musekwa [22]. Here, we ran numerical simulations with different degrees of randomness in three relevant parameters in order to analyze the resulting behavioural patterns.

The system of differential equations with the impulses representing regular IRS is represented graphically in Fig. 2. Here, regular spraying is applied four times a year for five years where the recovered humans (in red) stabilise as the majority. Under perfect conditions, this model predicts that with the IRS strategy, malaria is still occurring but is kept at a moderate level. The mosquito populations have considerable overlap during the impulses, but the overall trend is that the infected mosquito population size is larger than the susceptible mosquito population. The human populations have much more distinction, where the recovered human population stabilises as the largest size, followed by the infected humans and then susceptible humans as the smallest group. None of the human populations overlap during the impulses, except in the first instance where the initial susceptible and infected populations trend downwards and the recovered population trends upwards. The range of the impulses in the three human populations is smaller than that observed in the two

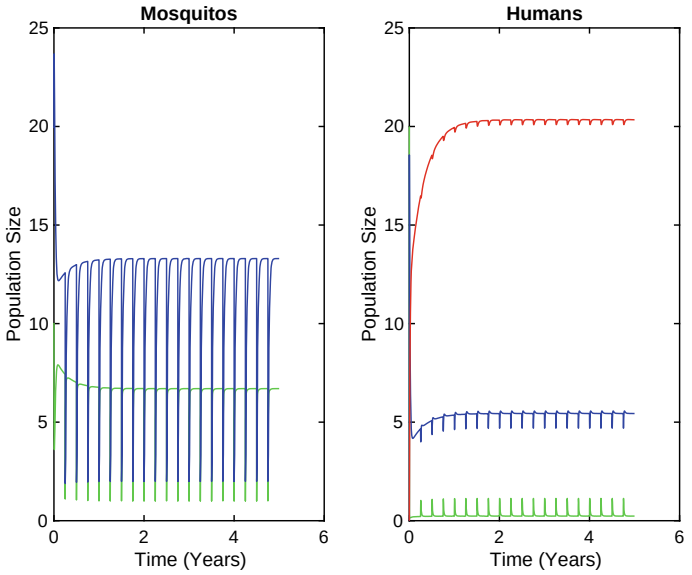


Fig. 2 Regular IRS spraying. Green represents susceptible individuals, blue represents infected individuals, and red represents recovered individuals. The values used are $\Lambda = 1000$ mosquitos$\times$years^{-1}; $\mu = 1/7.3$ days^{-1}; $\beta_M = 0.05$ mosquitos$^{-1}\times$days^{-1}; $\alpha = 1/8$ days^{-1}; $h = 1/9$ days^{-1}; $\beta_H = 0.5$ humans$^{-1}\times$days^{-1}; $\pi = 100$ humans$\times$days^{-1}; $\gamma = 1/20$ days^{-1}; $\delta = 1/30$ days^{-1}; $\mu_H = 1/30$ years^{-1}

mosquito populations. We want to describe the effect of randomness on the model to see whether or not the outcome changes. We will modify three parameters: the impulse period τ, which translates to irregular spraying; t_0, representing the time delay between the application of the spray and when it begins to have an effect; and r, which represents the variability in the effectiveness of the insecticide.

3.1 Individual Randomness

First, we examine each parameter's role in the model. In Fig. 3, the parameters τ, t_0 and r were assigned a random value between zero and one. In Fig. 3a, the effect of irregular spraying is observed. The overall trends of each population remain the same as in Fig. 2, but the impulses occur at irregular intervals. The possible activation delay of the insecticide is shown in Fig. 3b. Once again, each population stabilises similarly to the regular model (Fig. 2), but the time to produce 20 impulses increases

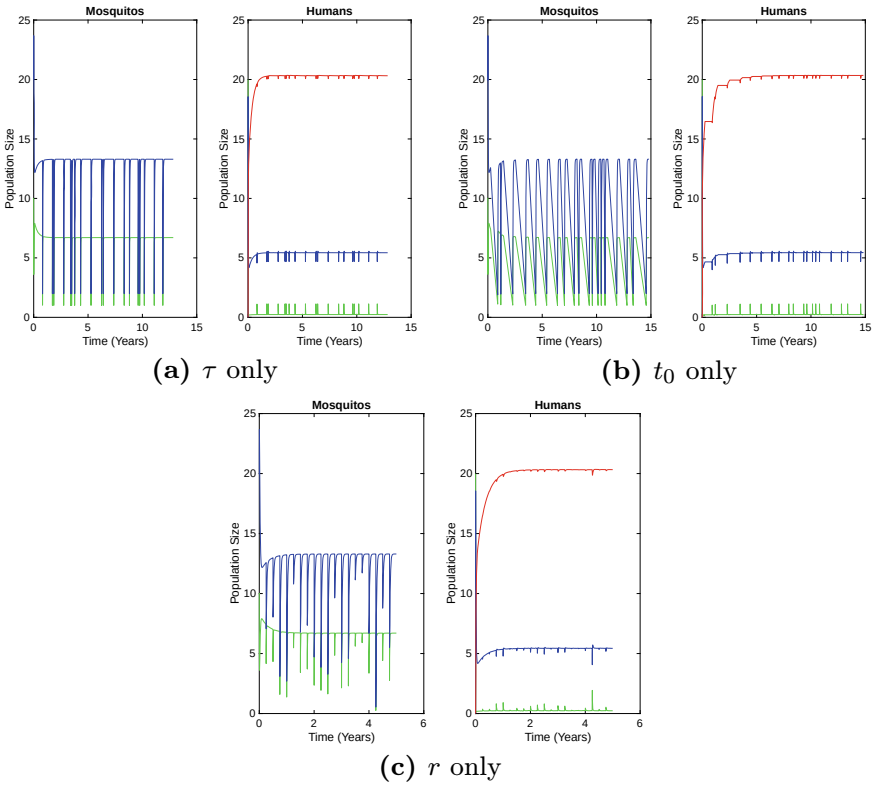


Fig. 3 Isolated stochastic behaviour in τ, t_0 and r between 0 and 1

due to the delay in reaching the maximum at each impulse. In Fig. 3c, the minimum value of each impulse varies, representing the variation in the effectiveness of the insecticide, where impulses with higher minimum values leave a larger proportion of mosquitos alive. Nonetheless, each population stabilises at the same values as predicted by the regular model (Fig. 2).

3.2 Combined Randomness

Next, we want to combine the randomness of the three parameters. Initially, we predict that randomness translates to irregular spraying and that unreliable insecticides will have a long-term negative impact on population survival. However, as we see in Fig. 4, where τ, t_0 and r were assigned a random value between zero and one simultaneously, the outcome repeats the patterns of Fig. 2. Despite some impulses being substantially different from the regular impulses, the population size returns to the same equilibrium value in every case. We see that in some instances, for example in Figs. 4d–e, the mosquito population is close to zero, which we had hypothesised would substantially affect the population dynamics, but, due to the nonzero birth rate, the model regains stability.

3.3 Large Randomness

Randomness between 0 and 1 did not appear to have a significant effect on the overall trends of the impulsive model, so we next tried simulations with a larger range for τ and t_0, where the value can be greater than one. Figures 5 and 6 show that there is some difference in the time scale and some larger deviations from the regular model, but the result remains very similar to Fig. 4. The outstanding trends and stabilities of each group follow the regular model, and we see that the model is very resistant to the randomness.

3.4 Restricted Randomness

We return to the range of biologically relevant values by adding randomness in a more localised manner. The biological ranges of the three variables are not equal, so we want to better reflect how stochasticity is incorporated into communities by adding randomness around a set value. Instead of a random value between 0 and 1, r and t_0 now have a value of 0.5 plus a random value between 0 and 0.5, whereas τ has a value of 0.25 plus a random value between 0 and 0.5. These numbers are chosen based on the model represented in Fig. 2, where $r = 0.85$, $t_0 = t_f$ and $\tau = 0.25$. This simulation will more closely depict a situation that might happen biologically, so we

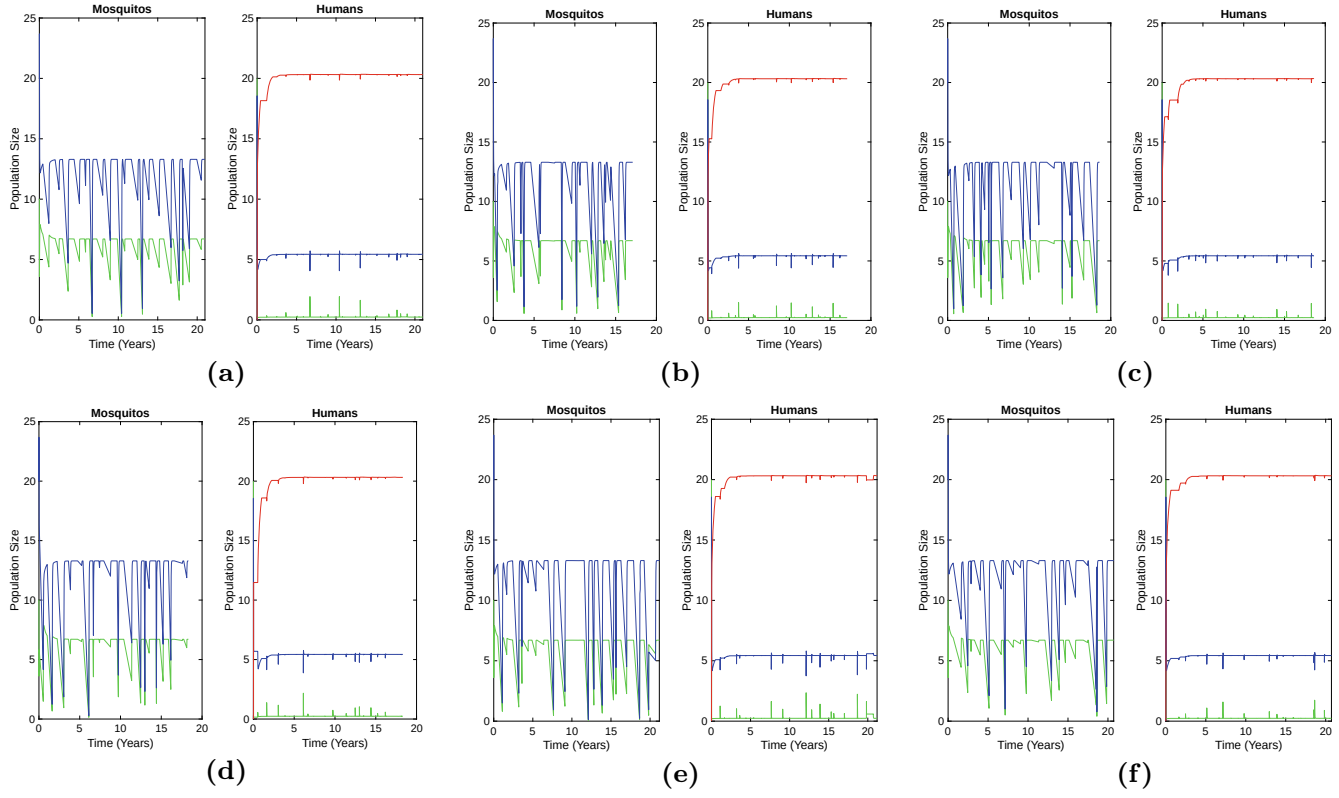


Fig. 4 Combined stochastic behaviour of τ, t_0 and r between 0 and 1

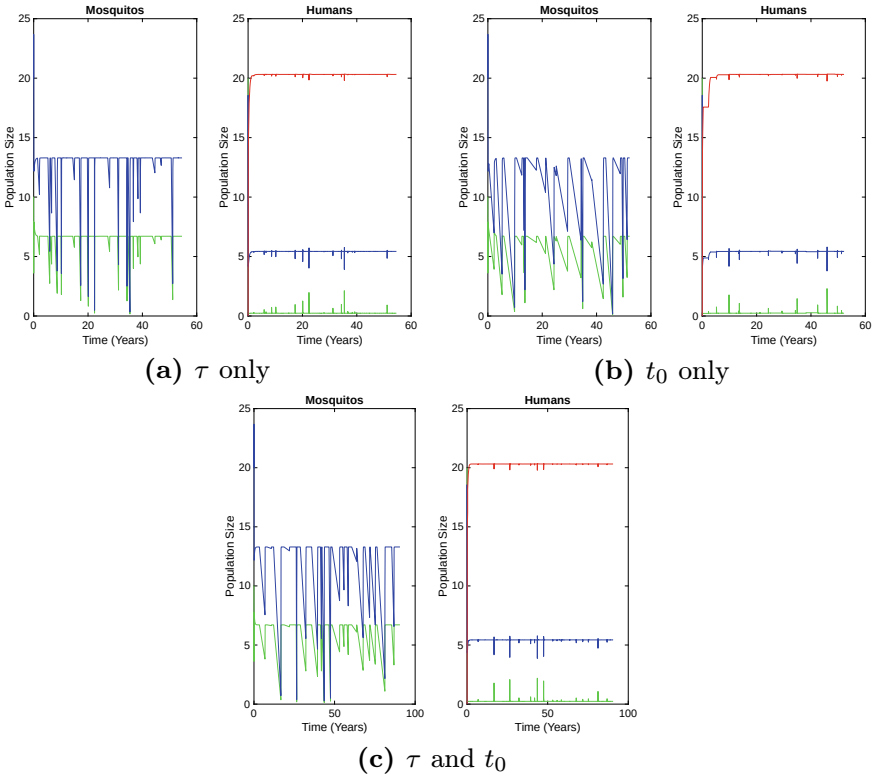
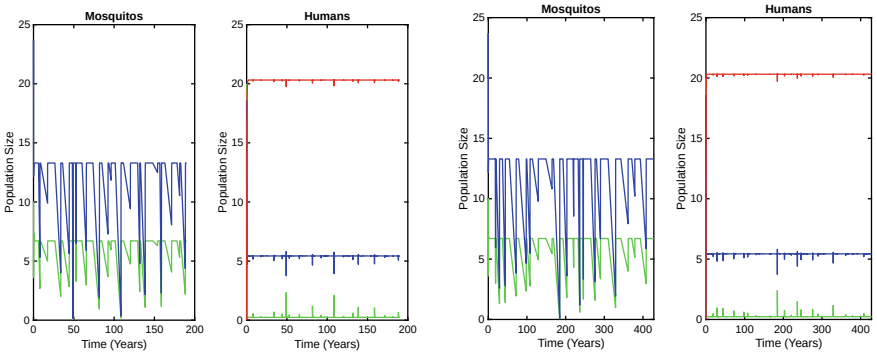


Fig. 5 Combined and isolated stochastic effect for τ and t_0 between 0 and 5 and r between 0 and 1

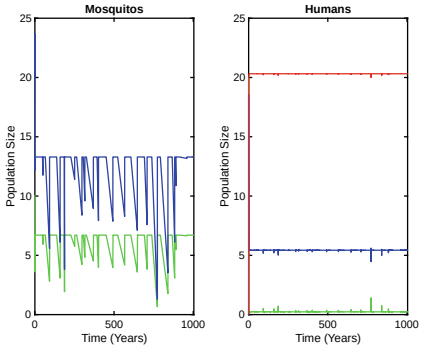
can make some more relevant conclusions. In Fig. 7, we see similarly to the previous simulations that there is little overall effect from the addition of randomness.

3.5 Small Randomness

In order to understand the full scope of the model, we next assigned τ, r and t_0 random values between 0 and 0.2. These values have no biological meaning, but we can learn more about the model by looking at the full range of possible values. In Fig. 8, we see for the first time a large deviation from the regular model. There is a clear distinction between the infected and susceptible mosquito populations instead of an overlap, and the recovered humans take longer to reach equilibrium. Nonetheless, the stability of recovered humans and infected mosquitos mirrors all the previous cases.



(a) $[0, 10]$ **(b)** $[0, 20]$



(c) $[0, 50]$

Fig. 6 Combined stochastic behaviour for τ and t_0 with large intervals and r between 0 and 1

3.6 Extreme Values

Finally, we consider what happens when we set the parameters to zero and infinity. So far we have only seen one possible outcome, so we will look at the range of all possibilities to see if there is a change at any point. First, we set the parameters to infinity (Fig. 9), and we see, as expected, that the model has one impulse and approaches the equilibrium values quickly. In Fig. 10a, we see that r set to zero and τ set to 0.25 provide the same outcome as in Fig. 9. In Fig. 10b, we have a more interesting result with τ. This parameter cannot be set to zero, because that would represent all impulses happening simultaneously, so we arbitrarily choose a value of 10^{-6}. (Note that the impulsive assumptions break down for τ small, so this is merely a theoretical exercise.)

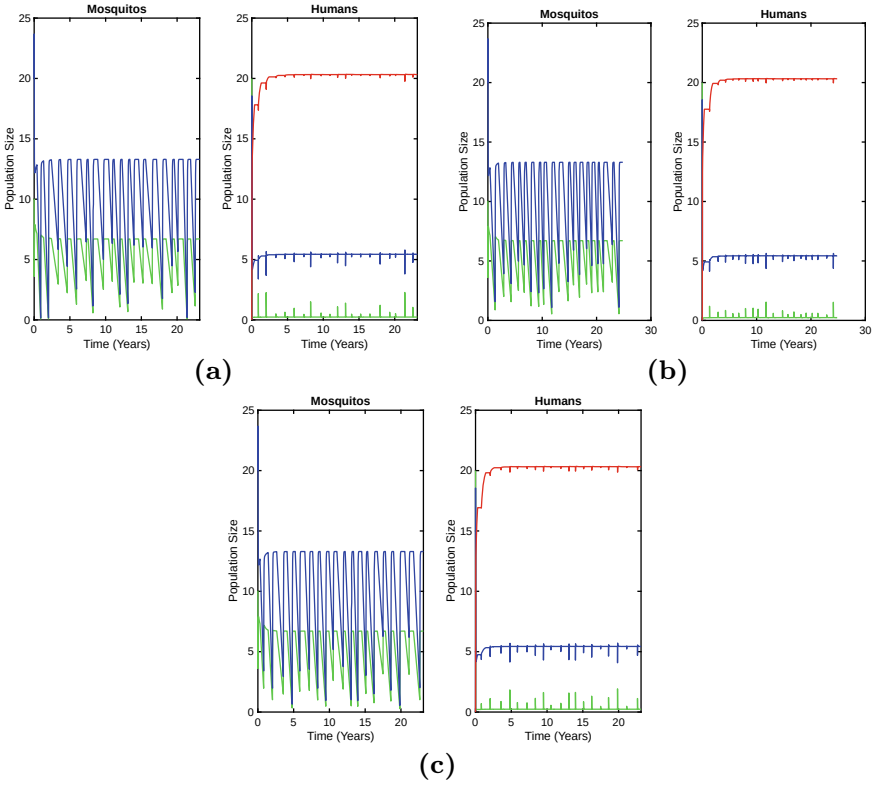


Fig. 7 Combined stochastic behaviour for τ between 0.25 and 0.75 and t_0 and r between 0.5 and 1

The outcome is very different from any of the previous models: the mosquito population quickly dies out, and the susceptible humans are the majority at equilibrium. When we combine the two, in Fig. 10c, we see both the infected mosquitos and humans are the overarching majority, but the model does not seem to reach an equilibrium within 20 impulses.

In Fig. 11, we set r to zero and $t_0 = t_f$ to look at the effect of τ over 5000 repetitions. We can see that a switch occurs between $\tau = 10^{-6}$ (Fig. 11c) and $\tau = 10^{-5}$ (Fig. 11d), where the equilibrium changes from a majority of infected individuals remaining to a majority of recovered individuals remaining. If we increase the value of τ further, the model increasingly resembles Fig. 9. We can also see that decreasing the value of τ, as in Fig. 11a, leads to recovered and infected humans at zero and susceptible individuals remaining; the opposite occurs for mosquitos, with susceptible individuals at zero and only infected individuals remaining.

So far, we have set $t_0 = t_f$, but we can continue our analysis of the parameters by including randomness in t_{k+1}. In this case, time can go backwards because the impulse at t_{k+1} can occur before the impulse at time t_k. In Fig. 12, we can see that

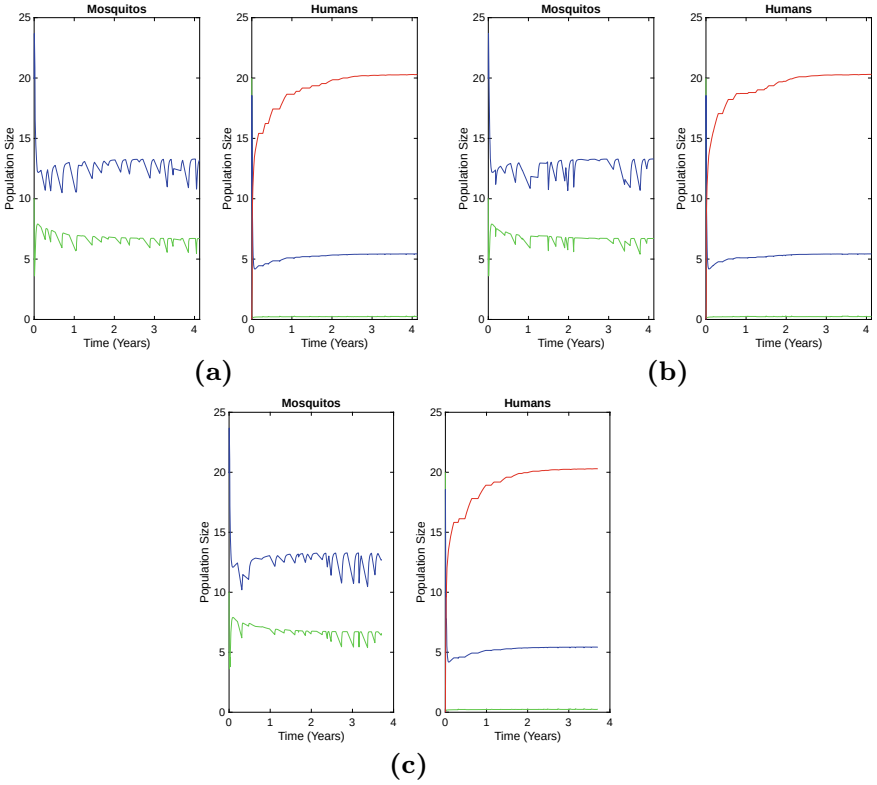


Fig. 8 Combined stochastic behaviour for τ, t_0 and r between 0 and 0.2

at zero (10^{-10} for τ), infected and recovered humans are at zero and susceptible humans are at the initial values. In Fig. 12b–f, we see the model converge towards the regular model as we increase the value of the parameters. In this scenario, time can move backwards (due to the nature of the impulses), so this is only performed as a theoretical exercise.

4 Discussion

Models using impulsive differential equations often exclude stochasticity for conciseness or simplicity. However, these small disruptions have the potential to change the outcome entirely when considering whether a disease will die out or become endemic. The results of the various degrees of randomness in the malaria model presented in Figs. 3, 4, 5, 6 and 7, regardless of the amount of randomness added, maintain the same overall trends, and the different populations consistently stabilise

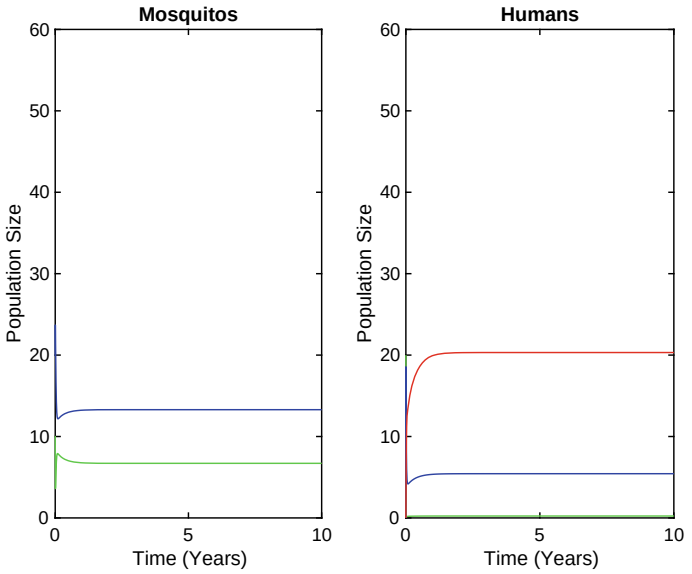


Fig. 9 Parameters set to infinity

around the same equilibrium values. The main difference observed in Figs. 5c and 6 is the increase in time required to produce 20 impulses. This change is due to higher values in t_{k+1}. This correlates to the insecticides requiring multiple years to take effect, which is not a realistic scenario, but we can still conclude that the model can withstand large variations and make the same predictions as the regular model (Fig. 2). Also, it can be concluded that the mosquito populations are more affected by the variation in the parameters. Figure 8 shows a significant disturbance in the outcomes; however, this is due to the values of the parameters t_{k+1}, τ and r being small, not due to the randomness. In Fig. 8, the parameters are constrained to values between 0 and 0.2, which changes the meaning of the model. We can conclude that the small values have local effects on the model that can be counteracted by higher values when the range of randomness is larger, but when constrained to a small interval, the model loses its stability. Figures 3, 4, 5, 6 and 7 demonstrate the resilience in the model.

The various numerical simulations exploring the effects of variability in the malaria model retained the same general trends as the original model. When we added increasing degrees of randomness to the parameters τ, t_{k+1} and r, the equilibrium of the different proportions within the mosquito and human populations remained similar. Infected mosquitos stabilise higher than susceptible mosquitos, with large overlapping impulses. Recovered humans stabilise at the highest value, followed by infected and susceptible humans; all three categories are distinct and do not overlap.

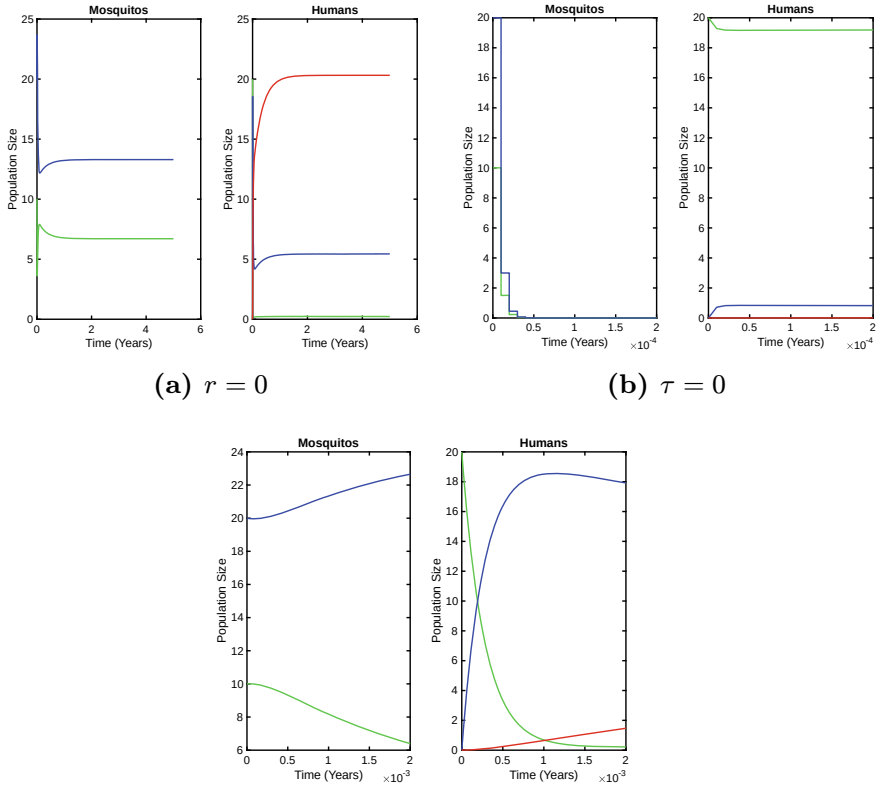
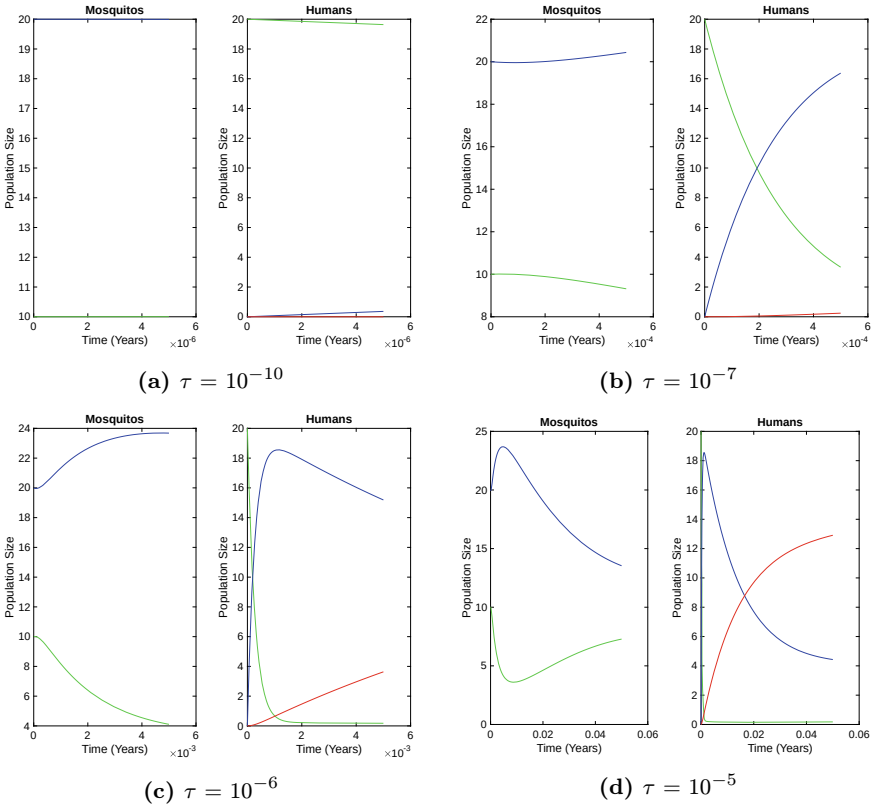


Fig. 10 Individual and combined behaviour for r equal to 0 and τ approaching 0

It should be noted that despite large variation in randomness for the impulsive effect, the mosquitos largely persist, even in extreme cases, with the exception of Fig. 10b. This exception is due to the fact that $\tau \approx 0$, so that mosquitos are eradicated instantaneously. In all other cases, the mosquito population persists. This is due to the virulence of malaria-infected mosquitos in this model, with an R_0 value of 12×10^6. (Note that these data match those in the previously published paper [22].) This is an extreme case for an exceptionally virulent disease, whereas diseases with smaller R_0 values may be more susceptible to stochastic effects from varying the impulses.

These findings can be useful in further studies on vector-control strategies for malaria, where the model excluding the variations can be used and still be applicable in communities where perfect conditions may not occur. This justifies the lack of stochasticity, because the model output is not easily disturbed by variability in the τ, t_{k+1} and r parameters.

Although our results imply stability, it is difficult to say how the local disturbances in models translate biologically. For example, the iteration in Fig. 4b has a local

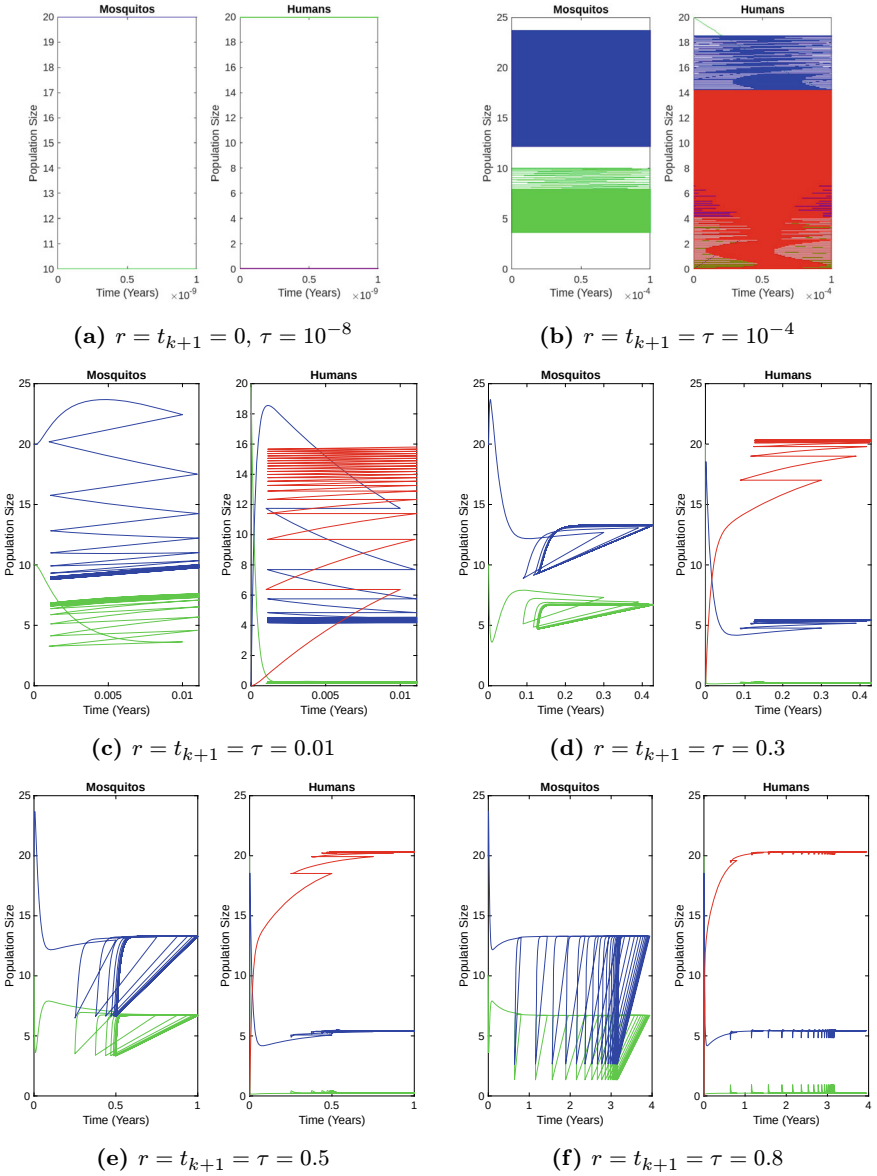


(a) $\tau = 10^{-10}$ **(b)** $\tau = 10^{-7}$

(c) $\tau = 10^{-6}$ **(d)** $\tau = 10^{-5}$

Fig. 11 Small values of τ

plateau in the infected mosquito population, where none of the mosquitos have been eliminated by the insecticide after multiple years. Although the associated human population graph is not affected, the outcome may not reflect biological reality. There may be additional cultural or economic factors that influence malaria dynamics that are not included in the model. We also explored strategies that disturb the impulsive assumptions and involve backward time, which are not realistic but nevertheless provide further insights into the issue of randomness in the key parameters. Further studies on the effect of randomness on other malaria control and prevention schemes would be useful to understand how our results could apply to different strategies and where randomness might play a critical role in disease outcomes.



(a) $r = t_{k+1} = 0,\ \tau = 10^{-8}$ **(b)** $r = t_{k+1} = \tau = 10^{-4}$

(c) $r = t_{k+1} = \tau = 0.01$ **(d)** $r = t_{k+1} = \tau = 0.3$

(e) $r = t_{k+1} = \tau = 0.5$ **(f)** $r = t_{k+1} = \tau = 0.8$

Fig. 12 Increasing the value of the parameters from zero

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