# The changes in public's terror-to-death influence the dynamics of infectious diseases

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**Article**

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**Data Availability:**

This manuscript is a theoretical work, so it does not have any raw data, but the relative code will be available on Github once the manuscript is accepted.

**Abstract (227):**

Previous studies about vector-borne diseases have emphasized the feedback between human psychology and diseases but neglected the changes in psychological processes. Here I first studied whether and how the two types of psychological dynamics in people's Terror-To-Death (TTD) — periodical terror reinforcement and memory decay of terror — can influence the host-vector-pathogen interactions. Through developing a generic Ross-MacDonald model with TTD dynamics tailored for *Zika* virus transmitted by *Aedes aegypti* mosquito, I found that in general, the increase in initial terror increases control effort, while memory decay of terror decreases disease control. Memory decay also exhibits a threshold effect: when initial terror is below certain level, TTD decay would not influence the system much; once initial terror reaches a threshold, memory decay of TTD can largely reduce the public's control effort, increase mosquito population and disease level in the system under a larger mosquitoes' carrying capacity. Adding periodical terror reinforcement could introduce dynamical oscillation to the system, dampen the peak of human infection, and shorten the time of disease outbreak. If the reinforcement frequency is large enough, system dynamics could approach the scenario with constant TTD in the absence of memory decay. This work significantly advances the theory in disease epidemiology and biopsychology and can provide guidance for disease control by considering the joint effects of initial terror, the public's memory decay, and the frequency of terror reinforcement simultaneously.

## Introduction:

Human activities can largely influence disease dynamics, e.g., modifying hosts' behaviors to influence disease spread among patches[1-3], or changing species diversity to affect pathogens' competence etc. [4,5]. One direct way to shape pathogens' populations comes from disease control[6-9]. Governments often design a variety of disease control strategies to achieve a goal of disease reduction (e.g., expand surveillance network; improve cost-effective vector controls for Zika, malaria, dengue fever etc. [10-14]). One critical strategy is to educate the public about the danger of infectious diseases and the necessity of disease control, e.g., broadcasting the death number caused by infectious diseases via social media, or performing regular visits by local health professionals. [15-18]. Previous studies have demonstrated that disease information conveyed through social media or governments' education can largely affect the public's attitudes to infectious diseases and further influence the public's control behaviors on diseases[11,17,19].

However, those studies often neglected the dynamics in public's perceptions of the risks of infectious diseases. For example, disease education from governments often takes place as a routine (e.g., weekly or monthly), which could create a periodical effect on the public's psychological reactions towards infectious diseases. For example, people's Terror-To-Death (TTD) psychological reactions could increase around the time when governments broadcast how seriously infectious diseases can lead to death[20-22].

Another common change in the public's psychological process towards disease comes from human memory decay. Take people's TTD as an example, after human populations build up their initial terror towards infectious diseases, this terror would gradually decay with time. For example, previous studies demonstrated that people's TTD and panic about infectious diseases could exhibit memory fade[23-25]. This forgetting process of fear plays an important role in brain

functions and human evolution (e.g., "forgetting cells" in brain; memory decay of emotions can serve as a treatment to deal with pathological psychological issues [23,26,27] ) and can be well depicted by mathematical curves[24,28]. The strength of initial terror built towards infectious diseases and the memory fading rate of this terror can together affect the dynamics of public's TTD reactions.

The above two types of changes of TTD, combined with the deaths and human infection, would largely influence the public's perceived risk of infectious diseases. The perceived risk can motivate the public to take action to control infectious diseases. Beyond risk, other factors can also influence the public's attitudes towards disease control. For example, for vector-borne diseases, many vector control strategies involve the usage of chemicals (e.g., pesticides) [9,29], which can cause environmental pollution. The public would tend to reduce disease control efforts due to their concerns about environmental contamination. Hence, public's control efforts on vectors often positively relate with people's TTD, deaths, and infections, but negatively correlate to environmental concerns[11].

In this study, I will first incorporate those two types of temporal changes of public's TTD into a generic vector-borne disease model (i.e., a modified Ross-MacDonald modified with control effort) and explore whether and how TTD dynamics can affect the host-vector-pathogen dynamics. This model is specifically tailored and parameterized for *Zika* virus transmitted by *Aedes aegypti* mosquito. Through this model, I first explored the separate and joint effects of initial terror and memory decay of terror through analytical and numerical solutions. I then added the periodical terror reinforcement into the model and further simulated the joint effects of all three factors on disease prevalence, death cases due to infection, total infected humans, mosquito population, disease control strength, and efficacy in the system.

**Methods:**

Here I assume both human and mosquito populations are well mixed in the system. Initially, the system has $S_H^0$ susceptible human population and $S_M^0$ susceptible mosquitoes. *Zika* virus would be then introduced into this system by first infecting one human in that patch ($I_H^0 = 1$). At any time, human can be in any of the three states: susceptible to infection ($S^H$), infected ($I^H$) or recovered ($R^H$). Certain proportion of the infected humans could have serious symptoms and eventually die from infection ($D^H$). The total human population at that time would be $S^H + I^H + R^H - D^H$ while the human population that can produce offspring is: $N^H = S^H + I^H + R^H$. I also considered the natural birth rate ($b^H$) and death rate ($\mu^H$) for human populations. The mosquito population has three states: stage without infection but susceptible to virus ($S^M$), and adult stage with infection ($I^M$), which can transmit virus to susceptible human ($S^H$). The infection rates from susceptible mosquitoes and human are $\beta^M$ and $\beta^H$, respectively. The natural death rate of adult mosquitoes is $\mu^M$. The birth of mosquitoes is limited by carrying capacity, having the form $f(M,K) = M\left(1 - {}^{M}/_{K}\right)$ (1) where $K$ is the maximum carrying capacity of mosquitoes, and $M = S^M + I^M$, the total number of mosquitoes that produce offsprings.

During disease outbreak, human and mosquitos' dynamics can drive human's control actions on both susceptible and infected mosquitoes through usage of pesticides (control effort as $C$). The control strength is positively correlated to the public Terror-To-Death (TTD) with memory decay ($f(A,B,t) = \frac{1.84\,A}{log\,(t)^B + 1.84}$ (2); details see[23,30]) and the number of death cases ($D^H$), where $A$ and $B$ describe the initial strength of the public fear to death and the forgetting rate of the fear with time $t$ after the introduction of disease. Similarly, control strength would also increase with the public averse to disease infection ($\gamma$) and the number of infected people ($I^H$). In

contrast, control strength also decreases with environmental concerns ($\epsilon$) caused by one unit of control action (e.g., one unit usage of pesticide) and the amount of control action (e.g., control strength $C$).

Here I propose a modified Ross-Macdonald equation[31,32] to capture the above dynamics among human, mosquitoes and control actions with Zika virus:

$$\frac{dS^H}{dt} = b^H N^H - \beta^H I^M S^H - \mu^H S^H \quad (3)$$

$$\frac{dI^H}{dt} = \beta^H I^M S^H - rI^H - \mu^H I^H \quad (4)$$

$$\frac{dR^H}{dt} = rI^H - \mu^H R^H \quad (5)$$

$$\frac{dD^H}{dt} = \delta I^H \quad (6)$$

$$\frac{dS^M}{dt} = f\left(\eta\left(S^M + I^M\right), K\right) - \beta^M I^H S^M - \mu^M S^M - C \quad S^M \quad (7)$$

$$\frac{dI'^M}{dt} = \beta^M I^H S^M - \mu^M I^M - C \quad I^M \quad (8)$$

$$\frac{dC}{dt} = f(A, B, t)D^H + \gamma I^H - \epsilon C \quad (9)$$

where Eq. 3-6 describe the dynamics of human population, Eq. 7-9 are for mosquito dynamics, while Eq. 9 indicates the dynamics of control actions on either susceptible or infected mosquitoes. For simplicity, here I also assumed that per death case would initially produces 100 times control of one infected case (i. e. , $A = 100\gamma$). The details of all the variables in the model are described in Table 1. The parameters and their values are in Table 2. The time *t* has unit as

per day. In the following, I use 1000 time-steps for all simulations, which is enough for the system to reach equilibria under one disease outbreak with the assigned parameters.

Through this above model, I first analytically studied the equilibrium of disease prevalence, control effort as well as the total mosquito population size in the absence of periodical terror reinforcement. Armed with the above analytical calculations, I then explored the separate and combined effects of initial terror *A* and terror decay *B* on system dynamics and equilibria by arranging different combinations of those two factors at a gradient of mosquitoes' carrying capacity. Lastly, I added the periods of terror reinforcement to the simulations and explored the joint effects of the three factors related to TTD temporal changes: initial terror *A*, terror decay *B*, and the number of periodical terror reinforcement on system dynamics and equilibria as well.

Here I focused on six indexes to keep track of the system dynamics or equilibria: disease prevalence $p$, number of deaths in human, number of infected humans, total mosquito population, control strength and efficacy ($\frac{I^H_{Cont-} - I^H_{Cont+}}{C_t}$ (10), where $I^H_{Cont-}$ and $I^H_{Cont+}$ represent the human infected cases at time *t* in the absence and presence of control actions respectively and *C* is the control strength at time *t*). For the case in the presence of periodical terror reinforcement, I also keep the record on the changes of overall sum of deaths in human, total control effort, mosquito population, and control efficacy across the entire time steps of one disease outbreak.

147 **Table 1** All variables and the corresponding initial values in the model

| Variables | Description | Initial values |
|---|---|---|
| $N^H$ | Human population size that can produce offspring | $S^H + I^H + R^H$ |
| $S^H$ | Susceptible humans | Random number ∈ [700, 710] |
| $I^H$ | Infected humans | 1 as initial value |
| $R^H$ | Recovered humans | 0 |
| $D^H$ | Death cases in humans | 0 |
| $S^M$ | Susceptible mosquitoes | Random number ∈ [1000, 1010] |
| $I^M$ | Infected mosquitoes | 0 |
| $C$ | Control on mosquitoes, either adult or larvae stages | 0 |

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149 **Table 2** All parameters and the corresponding values in the model. Some parameter values were
150 chosen from the incidence and mortality in early *Zika* outbreaks in South America (based on
151 daily values; see Reference).

| Parameters | Description | Value | Reference |
|---|---|---|---|
| $\beta^H$ | Transmission rate in humans | $1.5\times 10^{-4}$ | |
| $\beta^M$ | Transmission rate in mosquitoes | $3.0 \times 10^{-4}$ | |

| | | | |
|---|---|---|---|
| $\mu^H$ | Natural mortality in humans | $(8.6/1000)/365$ | [33] |
| $\mu^M$ | Natural mortality in mosquitoes | 1/13 | [34] |
| $b^H$ | Birth rate in humans | $(9/1000)/365$ | [35] |
| $r$ | Recovery rate in humans | 0.037 | [36] |
| $\delta$ | Composite rate | 190/3,474,182 | [35] |
| $\eta$ | Egg laying rate for mosquitoes | 10 | [34] |
| $A$ | Initial control due to terror-to-death | 1000 or vary | |
| $B$ | Memory decay of terror | 2 or vary | |
| $\epsilon$ | Demotivation to control per unit of control action | 100 or vary | |
| $\gamma$ | Control strength per infected case on adult mosquitoes | $e^{-\epsilon/80}$ | |
| $K$ | Carrying capacity for mosquito | 20000 or vary | |

## Results

### *Analytical solutions*

By summing up Eq. 3-5 but deducting Eq. 6, I calculate disease prevalence $p^*$ and control strength $C^*$ at equilibria when the total human population does not change (i.e., $\frac{d(S^H+I^H+R^H-D^H)}{dt}=0$):

$$p^* = \frac{I^{H^*}}{S^{H^*}+I^{H^*}+R^{H^*}} = \frac{b^H-\mu^H}{\delta} \quad (12)$$

Where disease prevalence is fixed given the birth, natural death rate and composite rate from infected to death.

$$\text{and } C^* = \frac{1}{\epsilon}\left(f(A,B,t)D^{H^*} + \gamma p^*\right) \quad (13)$$

in which control action at equilibrium mainly depends on the public perception of Terror-To-Death (TTD). In general, TTD: $f(A,B,t)$ follows the forgetting curve[23]: i.e., the strength of TTD is highest when terror is first built up in public, this initial terror would later decay with memory fade. Therefore, how strongly public's initial TTD caused by Zika virus (*A* parameter) and how fast this fear can decay with memory (*B* parameter) would largely influence the control's strength on mosquitoes at equilibrium in the absence of periodical terror reinforcement.

By summing up Eq. 8-9 and setting up the sum = 0, I can further calculate the equilibrium of total mosquito population ($S^{M^*} + I^{M^*}$):

$$S^{M^*} + I^{M^*} = \frac{1}{C+\mu^M} f\left(\eta\left(S^{M^*} + I^{M^*}\right), K\right) \quad (15)$$

Eq. 15 showed that mosquito population size at equilibria depends on control action ($C$) and mosquitoes' carrying capacity ($K$). Therefore, larger control would lead to smaller mosquito population with the modification from mosquitoes' carrying capacity.

***Numerical simulations***

*In the absence of periodical terror reinforcement*

In general, the increase in initial terror could largely increase control strength on the vector population, which then decreases disease level in the system (compare the solid and dashed lines in black or red in Fig. 1). Memory decay of this terror would offset the effects of elevated terror, exhibiting the opposite effect of the increased initial terror: e.g., reducing control effort, boosting mosquito population and overall disease level (e.g., see the relative locations of the black lines with $B = 0$ and red lines with $B = 2$ in Fig. 1) in the system. Specifically, memory decay of terror would decrease the terror level as well as the public's tendency for control effort, thus, mosquito population size would increase due to less control actions. Increased number of mosquitoes would lead to an increase in human infection and death cases in the human population (see relative locations of the black and red lines in Fig. 1B, C). The increased disease would need a longer time to cease, so the larger rate of memory decay would drive the disease to linger longer time (e.g., the curves with higher peaks would also take a longer time for the disease to drop to 0; see Fig. 1C). However, less disease also corresponds to larger control effort (see the relative locations of the four lines in Fig. 1E). Hence, larger initial terror or small memory decay would lead to a lower peak of control efficacy (Eq. 10; also see relative locations of lines in Fig. 1F).

However, the change of disease in response to memory decay largely depends on the levels of initial terror. When initial terror (i.e., parameter $A$) is relatively large, the disease is

more sensitive to memory decay (i.e., a small memory decay can drive a larger disease change (see the larger differences between the dashed red and black lines in Fig. 1). When initial terror is relatively small (e.g., $A = 100$), even large memory decay (e.g., $B = 2$) would not change disease level much (e.g., small difference between the solid red and black lines in Fig. 1). Hence, once initial terror is below a certain threshold value, memory decay would not influence disease much (extreme case is when $A = 0$, TTD would be 0 no matter what $B$ is; see Eq. 2). In other words, if the public has very low initial terror to death caused by infectious diseases, there would be no sufficient motivation for them to control the disease at the beginning, no matter how fast they forget the terror after their initial exposure to this disease. In other words, keeping up the same strength of control (i.e., with constant TTD with time; black lines in Fig. 1) would make a bigger difference for disease levels in the system when people initially have a larger terror and control action.

At a given initial terror, the effect of memory decay on the system would also interact with mosquitoes' carrying capacity. In general, the mosquito population size is larger under a larger carrying capacity (see more red areas at the top of Fig. 2C), corresponding to a larger disease prevalence (Fig. 2A). In general, memory decay of terror would produce more significant influences on control effort and mosquito population when the system has a larger mosquitoes' carrying capacity (see the color changes between white and red in Fig. 2B, C when carrying capacity is > 10000). When the mosquito population is bounded under a smaller carrying capacity (e.g., $K < 5000$ along the y-axis in Fig. 2), the decrease in control effect due to larger memory decay (see the color trend from red to white along the x-axis in Fig. 2B) may not increase mosquito population much (see the general blue colors at the bottom of Fig. 2C). Similarly, under a smaller mosquitoes' carrying capacity, the increase in initial terror, which

largely increases total control effort, may not lead to the reduction in mosquito population (see the general blue colors at the bottom between Fig. S1B and C along x-axis). Total control efficacy ($\frac{I^H_{Cont-} - I^H_{Cont+}}{Ct}$) is mainly determined by the total control effort ($Ct$): larger control effort usually leads to smaller control efficacy (see the opposite color trends between Fig. 2B and D, Fig. S1B and D). Therefore, total control efficacy is larger at smaller carrying capacity due to the overall smaller control effort (see more red colors at the bottom of Fig. 2 and S1).

*In the presence of periodical terror reinforcement*

Introducing periodical terror reinforcement to the public could balance out the effects of memory decay on disease dynamics to a certain degree (see the decreasing trend of either introducing period or decreasing *B* in disease; both dashed and dotted lines are below the solid black line in Fig. 3A-C; the color trend in Fig. 5C). This periodically reinforced terror would act as a repeated reminder for the public of the initial terror they perceived at their first exposure to infectious disease; thus, more frequent terror reinforcement could slow down the public's memory decay of TTD, reduce mosquito population (Fig. S2, 3C in Appendix), infected human (Fig. S2, 3A in Appendix), and deaths (Fig. S2, 3B) caused by infectious diseases (compare the solid and dashed lines in Fig. 3). The influences on the system from periodical terror reinforcement are similar as elevated initial terror (see the almost overlapped dynamics of the dashed and dotted lines in Fig. 4). The basic difference between periodical terror reinforcement and elevated initial terror comes from how those factors can shape TTD dynamics: at certain interval between periods, TTD could first decrease due to memory decay, but then increase due to the enhanced terror from the next period reinforcement, producing an oscillation in TTD along time (see oscillation curves in Fig. 3, 4, 6D, E); elevated initial terror would bring up the initial TTD to balance out the later TTD decay due to memory loss, creating a decreasing dynamic of

TTD even both factors would rise up the average TTD over time (see Eq. 2). The extreme case is when period number is very large, TTD would be reinforced faster before memory decay really lower the terror. In that way, TTD would almost become one constant value, approaching the effects from a constant initial terror in the absence of memory decay (see the gradually approaching trends from the black to the blue, to the green lines towards the red lines in Fig. 6). The frequency and magnitude of oscillation in disease dynamics and mosquito population size would also decrease with the increase of the terror-reinforcement frequency (compare the blue, green and red curves in Fig. 6A-E). In addition, the oscillation of TTD due to periodical terror reinforcement would first enter the system through disease control (see Eq. 9), which would also lead to stronger oscillations in control effort as well as mosquito population accordingly (see the lines with periodical cycling in Fig.s 3, 4, and 6D, E). The oscillations in mosquito population would then influence the dynamics of disease level in the system (Eq. 3-6; see also a relatively weaker cycling in Fig.s 3, 4 and 6A, C). The death due to infection, which is the least related to TTD oscillations, exhibits almost no oscillation (see the lines without much cycling in Fig.s 3, 4 and 6B).

Given that periodical terror reinforcement can dampen the peak of infected humans as well as shorten the time of disease outbreak (see the relative locations of human infected curves at different period numbers in Fig. 6C), periodical terror reinforcement could largely reduce average disease prevalence (Fig. 5A, C), total mosquito population (Fig. S2, S3C in Appendix) and total infected humans (Fig. S2, S3A). Hence, although periods of terror reinforcement could increase total control effort (see Fig. S2, S3D), overall speaking, disease control efficacy still increases with the increase of terror-reinforcement frequency (Fig. 5B, D).

**Discussion:**

This study systematically studied how the temporal changes in public's psychology can shape their behaviors such as control actions accordingly, which further affect vectors and diseases' dynamics in the entire system. In general, memory decay of Terror-To-Death (TTD) would weaken people's motivation for disease control, and boost mosquito population and disease levels when the environment has relatively larger mosquitoes' carrying capacity (see upper areas across panels in Fig. 2). In contrast, TTD's periodical changes due to repeated terror reinforcement (e.g., governments' education about infectious diseases through social media or professionals' home visitations on a regular basis etc.) could enhance public's control effort on vectors and may significantly reduce disease levels (Fig. 5).

The findings of this work can be broadly applied to the arrangement of different disease control strategies in real systems. For example, if a government has a general knowledge of people's memory decay and the total resource that can be used for the education of infectious diseases, this study can help to calculate a balance point between the strategies of how frequently (x-axis of Fig. 5A, B) and strongly per education (y-axis of Fig. 5A, B) the government should carry out to maximize disease reduction under a reasonable range of control efficacy. Or, with a fixed frequency of disease education and general knowledge of memory decay, this study can also help governments calculate how effective each education program needs to be to achieve a goal of certain disease reduction. Future studies can be done about what types of social media, what kind of education program can produce larger effects on the public's TTD as well as generating better motivation for control actions.

In addition, this frequent terror reinforcement for disease control can be compared to a wax-and-wane process of organisms' immune systems. The reintroduction of terror to the public is similar to vaccination to (re)build up the immune system's memory of antigens[37,38]. When the

public has gradually forgotten the initial TTD (similar as the wane process), terror reintroduction (e.g., through educational messages about the risks of infectious diseases on social media) would allow the public to regain their TTD. The strength of terror reinforcement (e.g., people's perception about disease risks at each reinforcement; compared to the doses of vaccination[39]) as well as the frequency of reinforcement [16-18] would produce a balanced effect on the overall disease control effort and efficacy. Therefore, our models and results can be easily modified and applied to the design of effective vaccination, adding to the body of knowledge in related medical areas[40-42].

Furthermore, the connection between control effort and vector population size should also be paid attention to beyond people's psychological reactions. The logic chain for effective disease control is that the public's psychology affects control efforts, which could modify vector population size and disease levels in the system. The key to this logical process comes from the effect of control efforts on mosquito populations[43], similar to the idea of a top-down effect in ecology. Mosquitoes' carrying capacity would diminish the control effect on mosquito population. At smaller carrying capacity, mosquitoes experience larger density-dependent mortality; thus, larger control, which causes larger mortality, may not necessarily reduce mosquito population size[11,48-51] (see the little or no correlation between control effort and mosquito sizes at lower carrying capacity in Fig. 2 and S1). This density-dependent effect in vector population could largely weaken the negative correlation between control strength and disease levels, which may void the effect of the public's psychological reactions on disease levels. Hence, other control strategies may need to be introduced to reduce mosquito population size (e.g., more effective controls on mosquito population: arranging mosquitoes' life stages to reduce the density-dependent effect [44,45]). Beyond the public's Terror-To-Death (TTD), other

310 psychological processes such as rebellious mentality (e.g., people may take little or no control
311 when they see similar social media information too frequently; [46-48]) could lead to opposite
312 control behaviors. In addition, TTD may be different from person to person. Some people may
313 be more prone to experience a heightened fear of infectious diseases than others[49-51]. This TTD
314 tendency could exhibit diversity across locations and cultures[51-53]. People who are in different
315 age groups, or have different pre-existing health conditions, can also have different fear levels
316 (e.g., individuals experiencing serious diseases tend to have a heightened tendency to TTD[54,55]).
317 This individual heterogeneity to TTD has not been included in this work, future studies and
318 surveys can explore this direction.

319 Since repeated terror reinforcement can introduce periodical dynamics in disease and
320 vectors (see oscillations in Fig.s 3, 4 and 6), this factor can be well added to a large body of
321 studies related to diseases' cycling under metapopulation/metacommunity structures[1,56,57]. In that
322 way, the public's psychological reactions can be added on the top of the layer of disease
323 transmission and host migration among patches, which would advance the current theory of
324 disease epidemiology by incorporating biopsychology.

325 **Reference:**


326 1 Jiao, J. & Fefferman, N. The dynamics of evolutionary rescue from a novel pathogen
327 threat in a host metapopulation. *Scientific reports* **11**, 10932 (2021).
328 2 Wood, C. L. *et al.* Marine protected areas facilitate parasite populations among four
329 fished host species of central Chile. *Journal of Animal Ecology* **82**, 1276-1287 (2013).
330 3 Jiao, J., Gilchrist, M. A. & Fefferman, N. H. The impact of host metapopulation structure
331 on short-term evolutionary rescue in the face of a novel pathogenic threat. *Global*
332 *Ecology and Conservation* **23**, e01174 (2020).

4 Keesing, F., Holt, R. D. & Ostfeld, R. S. Effects of species diversity on disease risk. *Ecology letters* **9**, 485-498 (2006).

5 Cortez, M. H. Using sensitivity analysis to identify factors promoting higher versus lower infection prevalence in multi-host communities. *Journal of Theoretical Biology* **526**, 110766 (2021).

6 Lourenço-de-Oliveira, R. & Failloux, A.-B. Lessons learned on Zika virus vectors. *PLoS neglected tropical diseases* **11**, e0005511 (2017).

7 Manore, C. A., Ostfeld, R. S., Agusto, F. B., Gaff, H. & LaDeau, S. L. Defining the risk of Zika and chikungunya virus transmission in human population centers of the eastern United States. *PLoS neglected tropical diseases* **11**, e0005255 (2017).

8 Sikka, V. *et al.* The emergence of Zika virus as a global health security threat: a review and a consensus statement of the INDUSEM Joint Working Group (JWG). *Journal of global infectious diseases* **8**, 3 (2016).

9 Benelli, G. Spread of Zika virus: the key role of mosquito vector control. *Asian Pacific Journal of Tropical Biomedicine* **6**, 468-471 (2016).

10 Sharma, A. & Lal, S. K. Zika virus: transmission, detection, control, and prevention. *Frontiers in microbiology* **8**, 233402 (2017).

11 Jiao, J., Suarez, G. P. & Fefferman, N. H. How public reaction to disease information across scales and the impacts of vector control methods influence disease prevalence and control efficacy. *PLoS Computational Biology* **17**, e1008762 (2021).

12 Kamya, M. R. *et al.* Malaria transmission, infection, and disease at three sites with varied transmission intensity in Uganda: implications for malaria control. *The American journal of tropical medicine and hygiene* **92**, 903 (2015).

13 Atkinson, J.-A., Vallely, A., Fitzgerald, L., Whittaker, M. & Tanner, M. The architecture and effect of participation: a systematic review of community participation for communicable disease control and elimination. Implications for malaria elimination. *Malaria journal* **10**, 1-33 (2011).

14 Guha-Sapir, D. & Schimmer, B. Dengue fever: new paradigms for a changing epidemiology. *Emerging themes in epidemiology* **2**, 1-10 (2005).

15 Najafi Ghezeljeh, T., Sharifian, S., Nasr Isfahani, M. & Haghani, H. Comparing the effects of education using telephone follow-up and smartphone-based social networking follow-up on self-management behaviors among patients with hypertension. *Contemporary Nurse* **54**, 362-373 (2018).

16 Tang, L., Bie, B., Park, S.-E. & Zhi, D. Social media and outbreaks of emerging infectious diseases: A systematic review of literature. *American journal of infection control* **46**, 962-972 (2018).

17 Yang, E., Kim, J. & Pennington-Gray, L. Social media information and peer-to-peer accommodation during an infectious disease outbreak. *Journal of Destination Marketing & Management* **19**, 100538 (2021).

18 Ye, X., Li, S., Yang, X. & Qin, C. Use of social media for the detection and analysis of infectious diseases in China. *ISPRS International Journal of Geo-Information* **5**, 156 (2016).

19 Sooknanan, J. & Comissiong, D. Trending on social media: integrating social media into infectious disease dynamics. *Bulletin of Mathematical Biology* **82**, 86 (2020).

20 Lerouge, R., Lema, M. D. & Arnaboldi, M. The role played by government communication on the level of public fear in social media: An investigation into the Covid-19 crisis in Italy. *Government Information Quarterly* **40**, 101798 (2023).

21 Sell, T. K., Hosangadi, D. & Trotochaud, M. Misinformation and the US Ebola communication crisis: analyzing the veracity and content of social media messages related to a fear-inducing infectious disease outbreak. *BMC Public Health* **20**, 1-10 (2020).

22 Izquierdo, I., Furini, C. R. & Myskiw, J. C. Fear memory. *Physiological reviews* **96**, 695-750 (2016).

23 Mertens, G., Lodder, P., Smeets, T. & Duijndam, S. Pandemic panic? Results of a 14-month longitudinal study on fear of COVID-19. *Journal of Affective Disorders* **322**, 15-23 (2023).

24 Hardt, O., Nader, K. & Nadel, L. Decay happens: the role of active forgetting in memory. *Trends in cognitive sciences* **17**, 111-120 (2013).

25 Myers, K. M., Ressler, K. J. & Davis, M. Different mechanisms of fear extinction dependent on length of time since fear acquisition. *Learning & memory* **13**, 216-223 (2006).

26 Thomasy, H. The science of forgetting. *Neuroscience* (2023).

27 Davis, R. L. & Zhong, Y. The biology of forgetting—a perspective. *Neuron* **95**, 490-503 (2017).

28 Bi, K., Chen, Y., Zhao, S., Ben-Arieh, D. & Wu, C.-H. J. Modeling learning and forgetting processes with the corresponding impacts on human behaviors in infectious disease epidemics. *Computers & Industrial Engineering* **129**, 563-577 (2019).

29 McAllister, J. C. *et al.* Mosquito control activities during local transmission of Zika virus, Miami-Dade County, Florida, USA, 2016. *Emerging infectious diseases* **26**, 881 (2020).

30 Ebbinghaus, H. Memory: A contribution to experimental psychology, trans. *HA Ruger & CE Bussenius. Teachers College* (1885).

31 Ross, R. Some quantitative studies in epidemiology. *Nature* **87**, 466-467 (1911).

32 Macdonald, G. The analysis of infection rates in diseases in which super infection occurs. *Tropical diseases bulletin* **47**, 907-915 (1950).

33 Agency, C. I. *The world factbook 2009*. (Central Intelligence Agency, 2009).

34 Stone, C. M., Schwab, S. R., Fonseca, D. M. & Fefferman, N. H. Human movement, cooperation and the effectiveness of coordinated vector control strategies. *Journal of the Royal Society Interface* **14**, 20170336 (2017).

35 Ellington, S. R. *et al.* Estimating the number of pregnant women infected with Zika virus and expected infants with microcephaly following the Zika virus outbreak in Puerto Rico, 2016. *JAMA pediatrics* **170**, 940-945 (2016).

36 Gao, D. *et al.* Prevention and control of Zika as a mosquito-borne and sexually transmitted disease: a mathematical modeling analysis. *Scientific reports* **6**, 28070 (2016).

37 Woolsey, C., & Geisbert, T. W. Current state of Ebola virus vaccines: A snapshot. *PLoS pathogens* ***17***, e1010078.38 (2021).

38 Carlsson, R.-M. *et al.* Modeling the waning and boosting of immunity from infection or vaccination. *Journal of theoretical biology* **497**, 110265 (2020).

39 Plotkin, S. A. & Plotkin, S. A. Correlates of vaccine-induced immunity. *Clinical infectious diseases* **47**, 401-409 (2008).

40 Stoddard, M. *et al.* The impact of vaccination frequency on COVID-19 public health outcomes: A model-based analysis. *medRxiv*, 2023.2001. 2026.23285076 (2023).

41 Tokars, J. I. *et al.* Waning of measured influenza vaccine effectiveness over time: the potential contribution of leaky vaccine effect. *Clinical Infectious Diseases* **71**, e633-e641 (2020).

42 Saad-Roy, C. M. *et al.* Immune life history, vaccination, and the dynamics of SARS-CoV-2 over the next 5 years. *Science* **370**, 811-818 (2020).

43 Chandrasegaran, K., Lahondère, C., Escobar, L. E. & Vinauger, C. Linking mosquito ecology, traits, behavior, and disease transmission. *Trends in parasitology* **36**, 393-403 (2020).

44 Abrams, P. A. When does greater mortality increase population size? The long history and diverse mechanisms underlying the hydra effect. *Ecology letters* **12**, 462-474 (2009).

45 de Roos, A. M. Effects of life history and individual development on community dynamics: A review of counterintuitive consequences. *Ecological research* **35**, 930-946 (2020).

46 Billow, R. M. Rebellion in group. *International journal of group psychotherapy* **53**, 331-351 (2003).

47 Billow, R. *Relational group psychotherapy: From basic assumptions to passion*. (Jessica Kingsley Publishers, 2003).

48 Kolbe, M. *et al.* Managing psychological safety in debriefings: a dynamic balancing act. *BMJ simulation & technology enhanced learning* **6**, 164 (2020).

49 Mikulincer, M. & Florian, V. Exploring individual differences in reactions to mortality salience: Does attachment style regulate terror management mechanisms? *Journal of personality and social psychology* **79**, 260 (2000).

50 Landau, M. J., Sullivan, D. & King, L. A. Terror management and personality: Variations in the psychological defense against the awareness of mortality. *Social and Personality Psychology Compass* **4**, 906-917 (2010).

51 Burnham, J. J., Mills, J. D. & Choi, Y.-J. The influence of race, gender, age, and geographic location on children's fears. *Educational Research Quarterly* **43**, 3-25 (2019).

52 Garmany, J. Drugs, violence, fear, and death: The necro-and narco-geographies of contemporary urban space. *Urban Geography* **32**, 1148-1166 (2011).

53 Ellis, L., Wahab, E. A. & Ratnasingan, M. Religiosity and fear of death: a three-nation comparison. *Mental Health, Religion & Culture* **16**, 179-199 (2013).

54 Asmundson, G. J. & Taylor, S. How health anxiety influences responses to viral outbreaks like COVID-19: What all decision-makers, health authorities, and health care professionals need to know. *Journal of anxiety disorders* **71**, 102211 (2020).

55 Salkovskis, P. M., Rimes, K. A., Warwick, H. & Clark, D. The Health Anxiety Inventory: development and validation of scales for the measurement of health anxiety and hypochondriasis. *Psychological medicine* **32**, 843-853 (2002).

56 Lloyd, A. L. & Sattenspiel, L. in *Spatial ecology* 277-298 (Chapman and Hall/CRC, 2009).

57 Han, D., Shao, Q. & Li, D. Exploring the epidemic spreading in a multilayer metapopulation network by considering Individuals' periodic travelling. *Complexity* **2020**, 1-9 (2020).

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**Figures in "The changes in public's terror-to-death influence the dynamics of infectious diseases"**

*Jing Jiao*; The Biology Department, College of Science & Engineering, Texas Christian University*

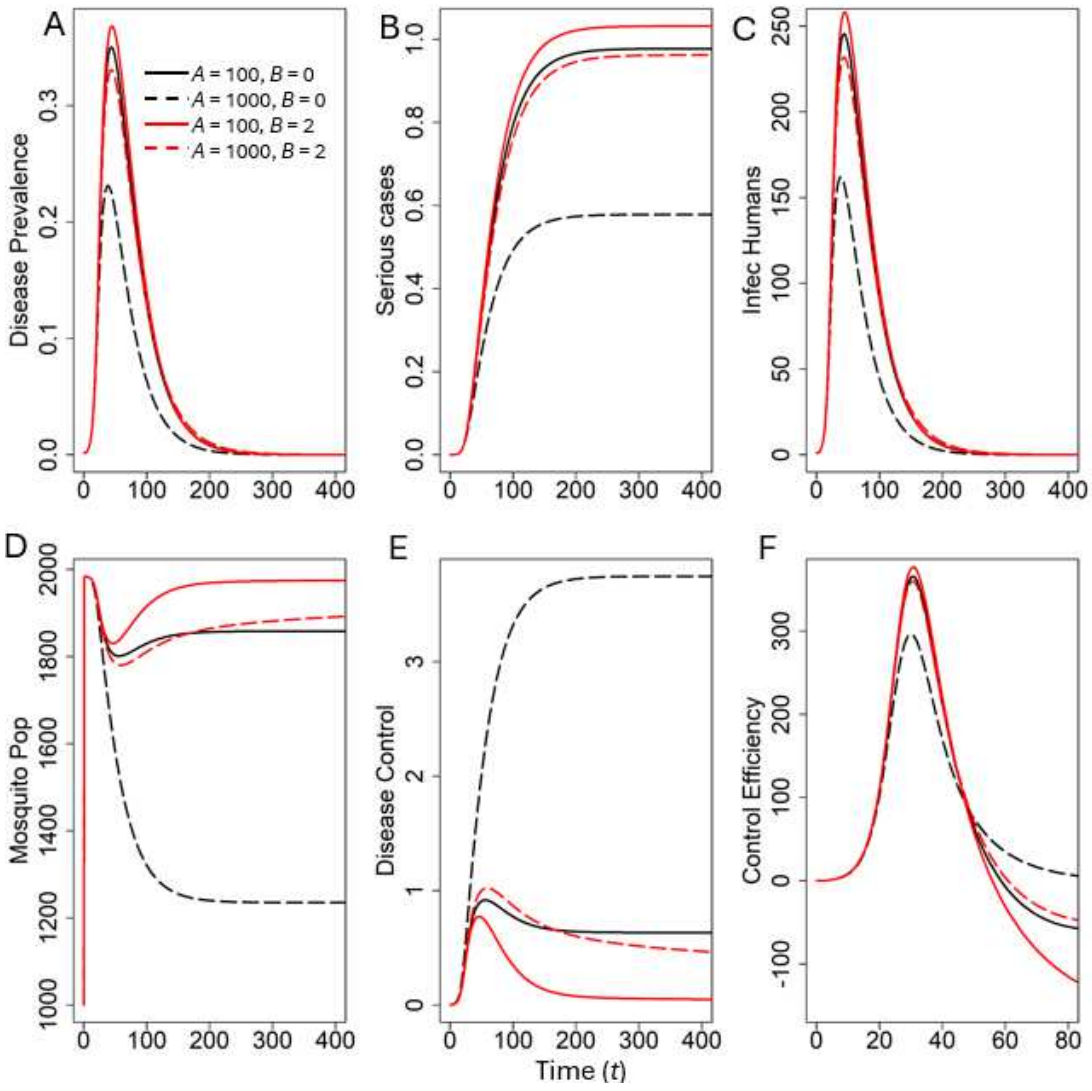


**Fig. 1** System dynamics under four combinations of initial TTD and memory decay: $A = 100$, $B = 0$; $A = 1000$, $B = 0$; $A = 100$, $B = 2$; $A = 1000$, $B = 2$, corresponding to the solid black, dashed black, solid red and dashed red lines, respectively. All other parameters are listed in Table 2.

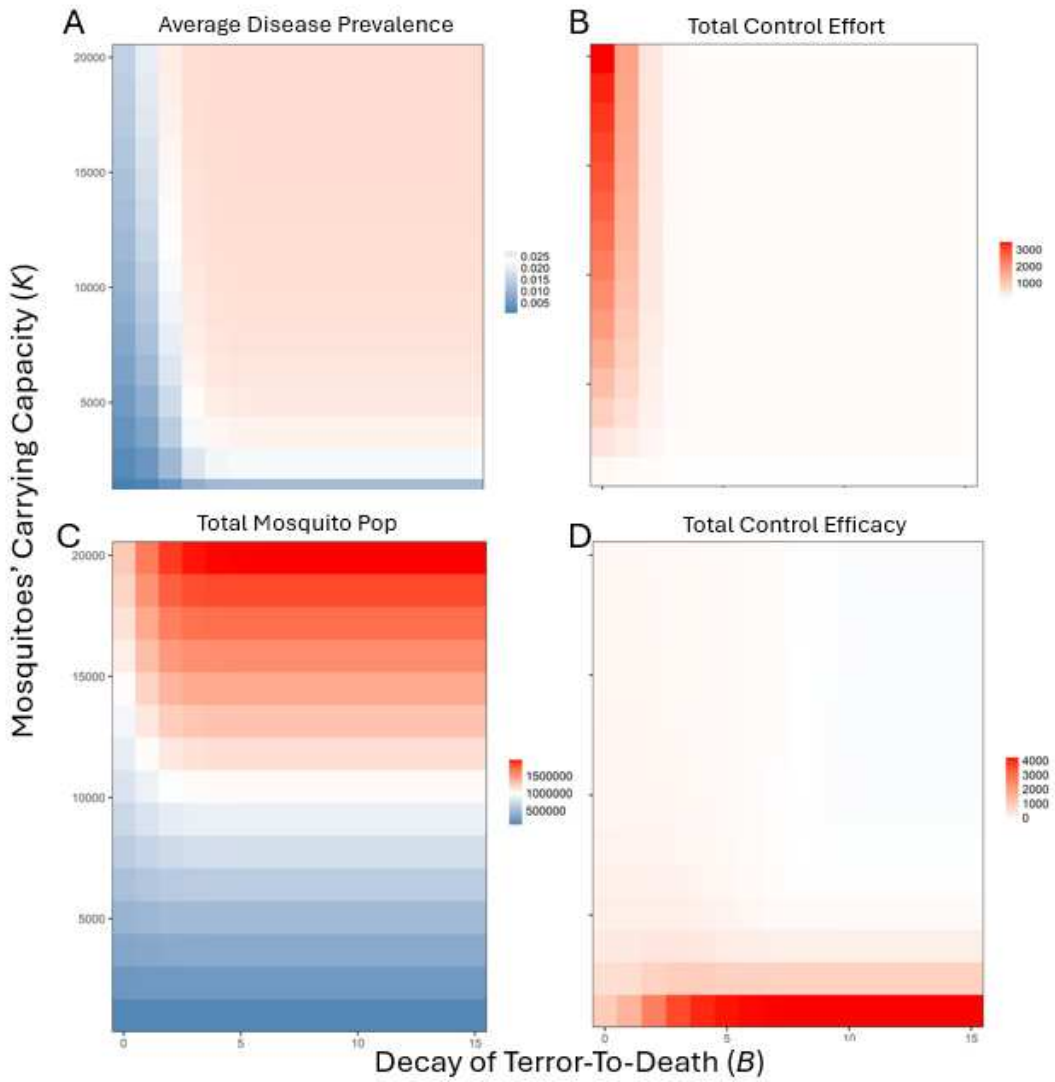


**Fig. 2** The overall system's average disease prevalence (A), total control effort (B), total mosquito population size (C) and total control efficacy (D) across the entire simulation time-steps under the combined influences of mosquitoes' carrying capacity *K* and the memory decay rate *B* of the public's Terror-To-Death (TTD) with constant initial terror as 1000. The more red-areas indicate the larger values while the more blue -areas mean smaller values of the above four indexes. All other simulation parameters are listed in Table 2.

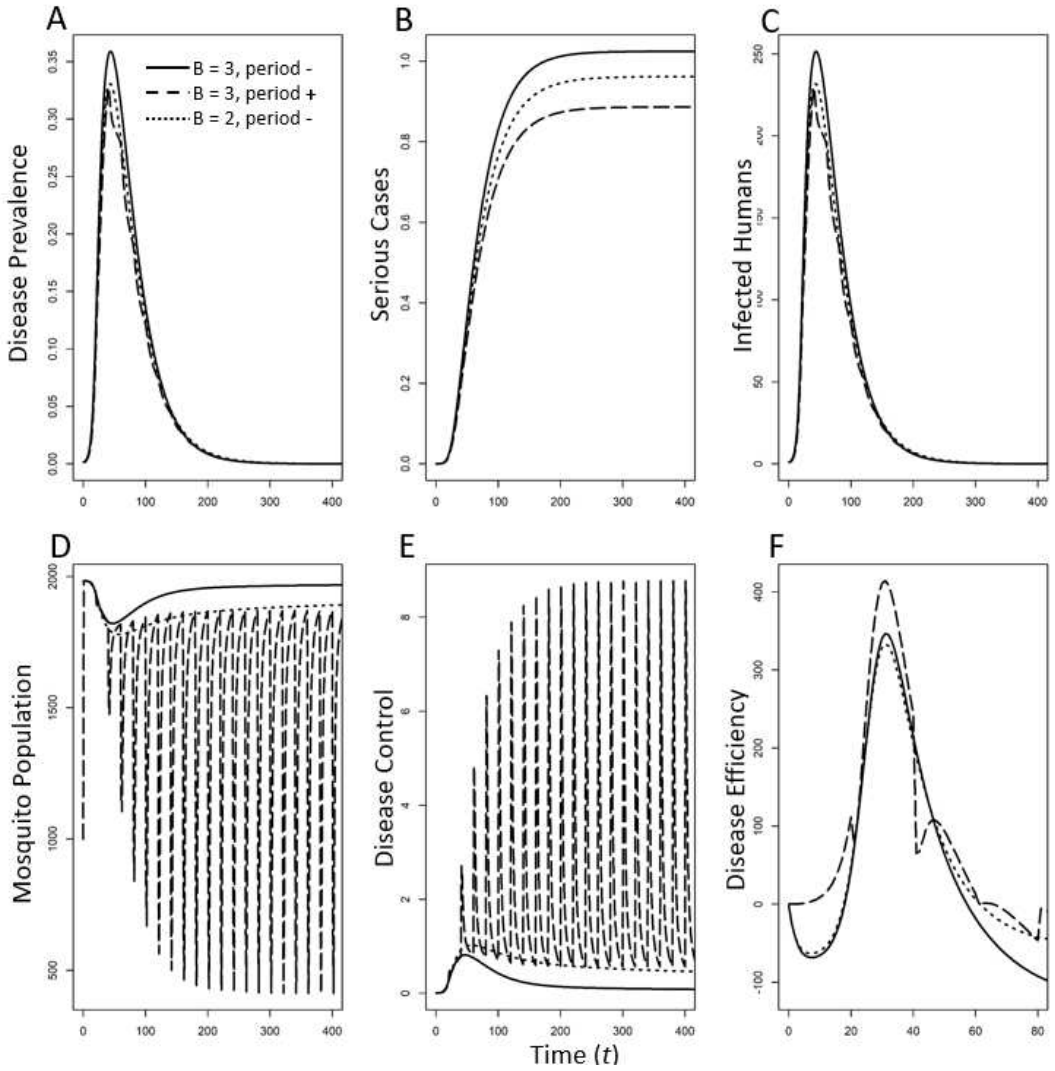


**Fig. 3** System dynamics under the combined influences of memory decay and periodical terror reinforcement with the fixed initial terror $A = 1000$. Here I show the situations under two levels of terror decay with and without periodical terror reinforcement: $B = 3$ without periodical terror decay (black curves), $B = 3$ with periodical reinforcement (dashed lines), and $B = 2$ without periodical terror decay (dotted curves). Here the period is set up as every 20 days for one terror reinforcement. All other parameters are listed in Table 2.

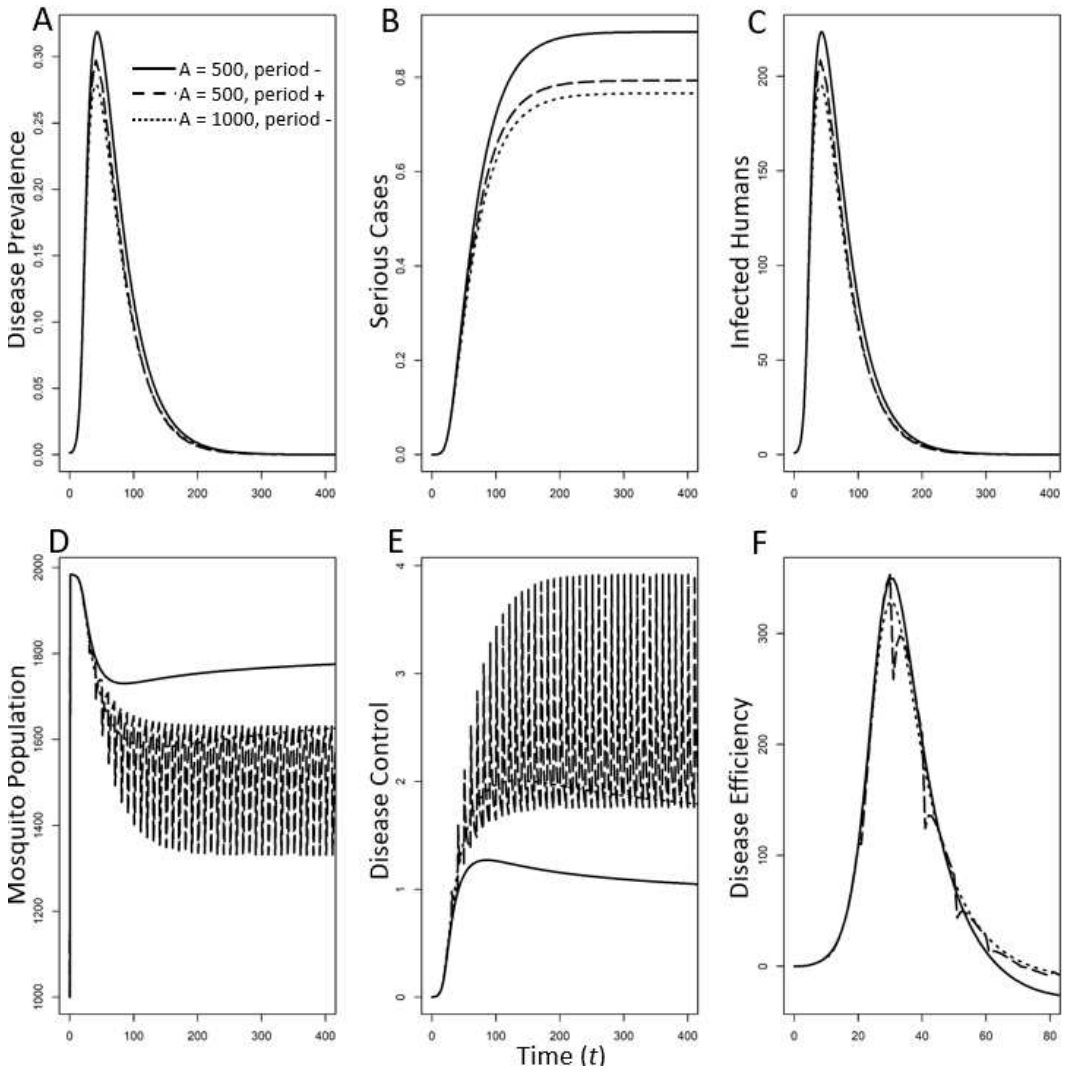


**Fig. 4** System dynamics under the combined influences of initial terror and periodical terror reinforcement with the constant terror decay $B = 1$. Here I show the situations under two levels of initial terror with and without periodical terror reinforcement: $A = 500$ without periodical terror decay (black curves), $A = 500$ with periodical reinforcement (dashed lines), and $A = 1000$ without periodical terror decay (dotted curves). Here the period is set up as every 10 days for one terror reinforcement. All other parameters are listed in Table 2.

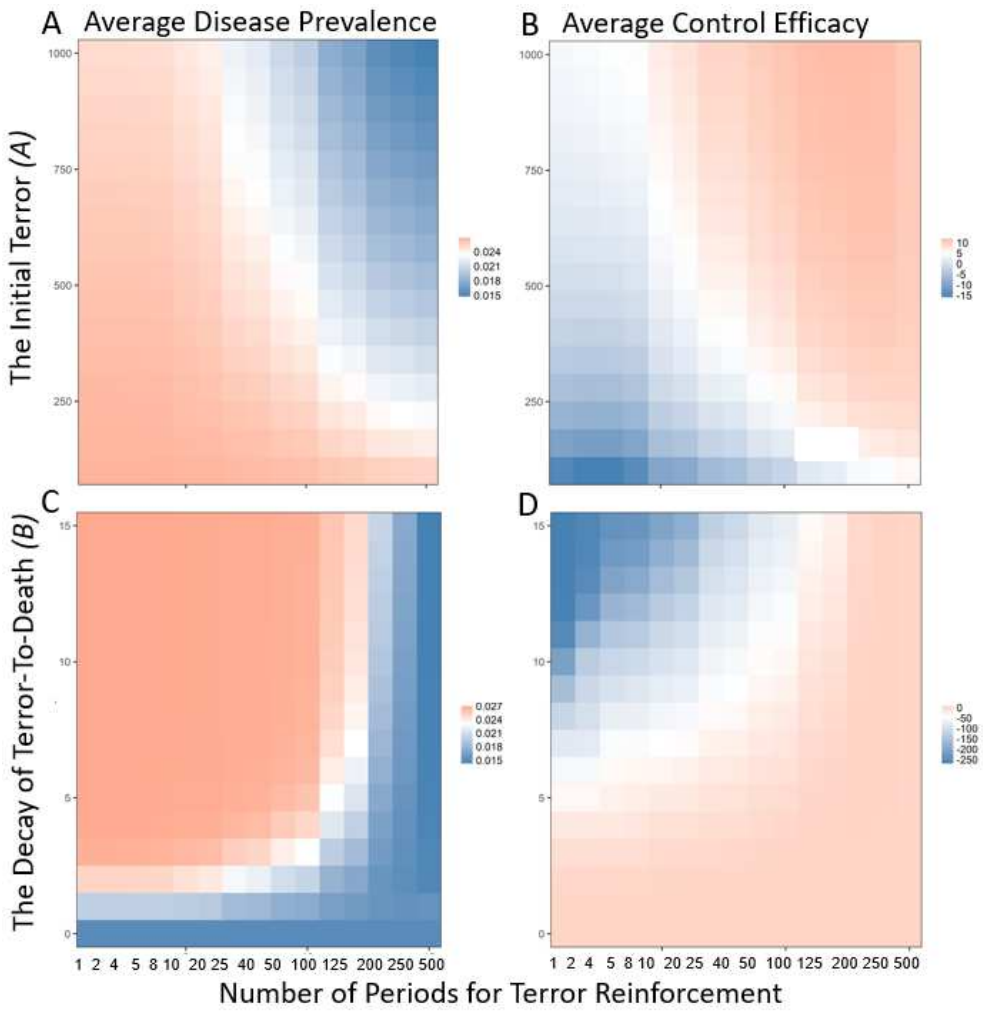


**Fig. 5** The overall system's average disease prevalence and control efficacy across the entire simulation time-steps under the combined effects between the number of periods for TTD reinforcement and initial terror strength (Panel A, B with constant memory decay $B = 2$), or the memory decay of terror (Panel C, D with constant initial terror $A = 1000$). The more red-areas indicate the larger values while the more blue -areas mean smaller values of either average disease prevalence or control efficacy. All other simulation parameters are listed in Table 2.

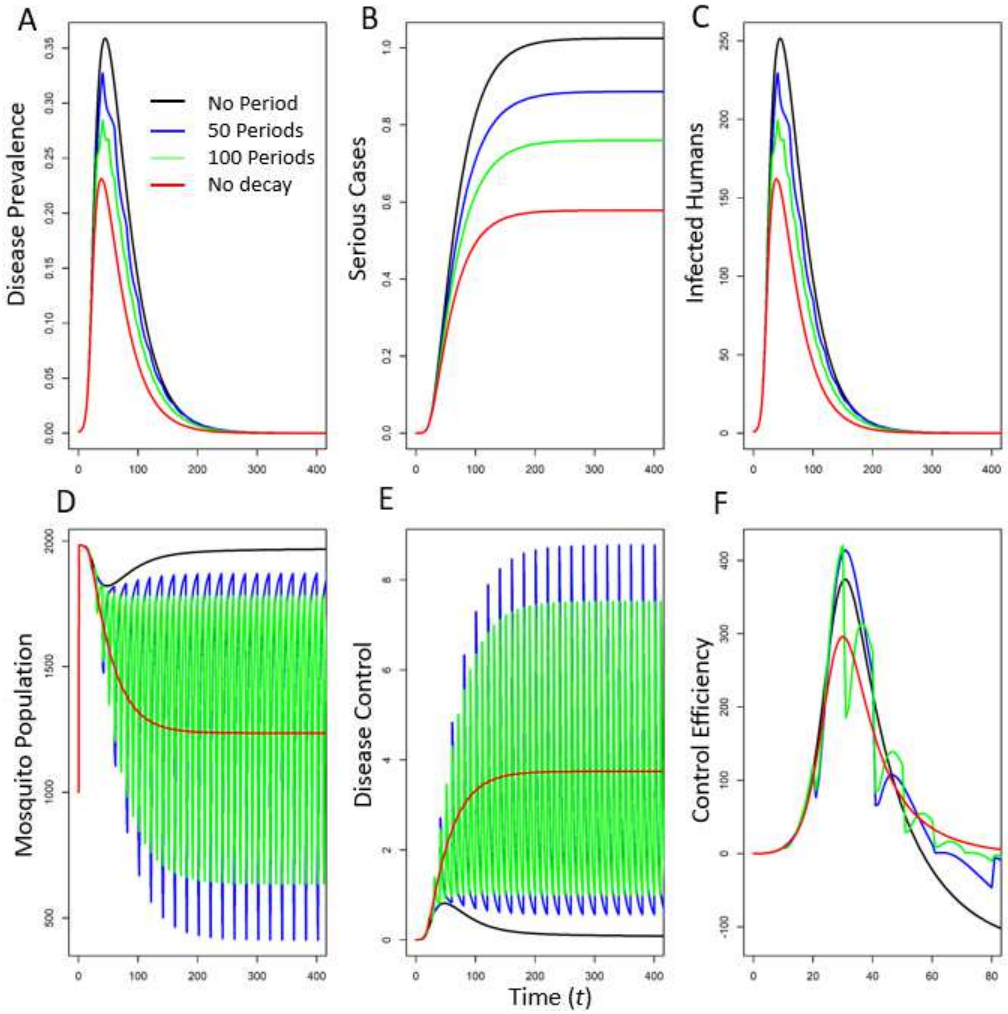


**Fig. 6** The system dynamics in disease prevalence, serious human cases, infected human, mosquito population, control effort and control efficacy under three levels of frequency in terror reinforcement with memory decay and the case without memory decay in terror: no terror reinforcement (solid black lines), 50 periods of terror reinforcement (blue lines) and 100 periods of terror reinforcement (green lines) and the case without memory decay (red lines). Here total time-steps = 1000, $A = 1000$ and $B = 3$ of TTD for all four lines. All other parameters are listed in Table 2.

# Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- AppendixofTTDmanuscriptJiao.pdf