*Article*

# A generalized discrete dynamic model for human epidemics

**WenJun Zhang**[1], **ZeLiang Chen**[2], **Yi Lu**[3], **ZhongMin Guo**[4], **YanHong Qi**[5], **GuoLing Wang**[2], **JiaHai Lu**[2]

[1]School of Life Sciences, Sun Yat-sen University, Guangzhou, P. R. China

[2]School of Public Health, Sun Yat-sen University, Guangzhou, P. R. China

[3]Department of Health Law, Policy and Management, School of Public Health, Boston University, U.S.A

[4]Animal Experiment Center, Sun Yat-sen University, Guangzhou, P. R. China

[5]Sun Yat-sen University Libraries, Sun Yat-sen University, Guangzhou, P. R. China

E-mail: zhwj@mail.sysu.edu.cn, lujiahai@mail.sysu.edu.cn

*Received 4 March 2020*; *Accepted 8 March 2020*; *Published 1 September 2020*



**Abstract**

A discrete dynamic model for human epidemics was developed in present study. The model included major parameters as transmission strength and its dynamic changes, mean incubation period, hospitalization time (i.e., the time from illness to hospitalization), non-hospitalization (i.e., outside hospitals) daily mortality, non-hospitalization daily recovery rate, and hospitalization proportion (proportion of cases for hospitalization), etc. Sensitivity analysis of the model indicated the total cumulative cases significantly increased with the increase of initial transmission strength and hospitalization time. The total cumulative cases significantly decreased with the increase of transmission strength's dynamic decline and hospitalization proportion, and decreased with the increase of non-hospitalization daily mortality and non-hospitalization daily recovery rate. The total cumulative cases significantly increased with the decrease of mean incubation period. Sensitivity analysis demonstrated that dynamic change of transmission strength is one of the most important and controllable factors. In addition, reducing the delay for hospitalization (i.e., hospitalization time) is much effective in weakening disease epidemic. Enhancing immunity to recover from the disease is of importance for increasing non-hospitalization recovery rate.

**Keywords** discrete dynamic model; difference and differential equations; human epidemics; hospitalization time; hospitalization proportion; incubation period; transmission strength; COVID-19.

**Computational Ecology and Software**
**ISSN 2220-721X**
**URL: http://www.iaees.org/publications/journals/ces/online-version.asp**
**RSS: http://www.iaees.org/publications/journals/ces/rss.xml**
**E-mail: ces@iaees.org**
**Editor-in-Chief: WenJun Zhang**
**Publisher: International Academy of Ecology and Environmental Sciences**

## 1 Introduction

So far a lot of dynamic models have been developed and used in the mechanic analysis and prediction of animal epidemics. Among them the differential equations based models are the mainstream methods, including SIR model (Kermack and McKendrick, 1927), Anderson-May model (Anderson and May, 1981), the models

of Zhang et al. (1997, 2011), etc. Zhang et al. (1997) model was a group of differential-integral equations mainly treating susceptible and infected insect populations. The improved model (Zhang et al., 2011; Zhang, 2016, 2018) was composed of nearly twenty differential equations supplemented by other equations. In these models, both susceptible and infected populations were treated as population density, and susceptible population interacts with infected population mainly through feeding on virus on the leaves spread by insects that died from virus infection. Serving as both explanatory and simulation models, they have demonstrated the better performance. Both SIR model (Kermack and McKendrick, 1927) and Anderson-May model (Anderson and May, 1981) include differential equations (correspondingly, difference equations) for susceptible (*s*) and infected (*i*) populations rather than population density, and the two populations interact with each other through the interaction term, p*s*(*t*)*i*(*t*) (Fuxa and Tanada, 1987). Nevertheless, numerous simulation results showed that both of their models are extremely sensitive to some of the key parameters and initial population sizes, especially the infection coefficient, p. Given true parameters and initial conditions, it was so difficult to synchronously obtain realistic results for population size and key time points (such as the peak time) although both of them are better explanatory models for the epidemic dynamics. Furthermore, many important parameters such as incubation period, hospitalization terms, etc., were not included in such explanatory models and most of the other models (Chen et al., 2020a). For these reasons, in present study we developed a generalized discrete dynamic model for human epidemics, and sensitivity analysis and scenario predictions were made, aiming to provide a generalized simulation tool for future uses (Zhang et al., 2020).

## 2 Methods

### 2.1 Model

Suppose the susceptible population is infinite in terms of the infected population, i.e, the size of susceptible population is approximately an infinite value. The susceptible population can thus be ignored. In addition, the hospital acts as a “black hole”. The hospital accommodates infected cases, and the later recovered from medical treatment and are released to the susceptible population, or die. According to general rules and human actions for disease epidemic, the generalized discrete dynamic model (delay difference equation) for human epidemics is developed as the following

$$
\begin{aligned}
&i(t) = i(t-\Delta t) + \Delta i(t-\Delta t) - p(t-\Delta t)\, i(t-\Delta t)\, \Delta t - a(t-\Delta t)\, i(t-\Delta t)\, \Delta t - h(t-\Delta t)\\
&\Delta i(t-\Delta t) = r(t-\Delta t\text{-c})\, i(t-\Delta t\text{-c})\, \Delta t\\
&h(t-\Delta t) = b(t-\Delta t)\, \Delta i(t-\Delta t\text{-d})\\
&s(t) = s(t-\Delta t) + \Delta i(t)
\end{aligned}
\qquad (1)
$$

where

$i(t)$: non-hospitalization cases (i.e., the existing cases outside hospitals) at time $t$

$\Delta t$: time step

$\Delta i(t)$: non-hospitalization new cases (i.e., the newly occurred cases outside hospitals) at time $t$

$h(t)$: hospitalization cases (i.e., the cases for hospitalization) at time $t$

$s(t)$: cumulative cases at time $t$

$r(t)$: new infection cases infected by a non-hospitalization case at time $t$ in $\Delta t$ (i.e., transmission strength)

$a(t)$: non-hospitalization disease mortality at time $t$ in $\Delta t$ ($0 \le a(t) \le 1$)

$b(t)$: proportion of non-hospitalization cases for hospitalization at time $t$ ($0 \le b(t) \le 1$) (i.e., hospitalization proportion)

$p(t)$: non-hospitalization recovery rate at time $t$ in $\Delta t$ ($0 \le p(t) \le 1$)
c: mean incubation period
d: mean time from illness to hospitalization (i.e., hospitalization time)

Equation (1) can be represented by a delay differential equation and an integral

$$di(t) / dt = r(t\text{-c})\, i(t\text{-c}) - (p(t) + a(t))\, i(t) - b(t)\, r(t\text{-d-c})\, i(t\text{-d-c})$$
$$s(t) = \int r(t)\, i(t)\, dt \qquad (2)$$

Without losing generality, let $\Delta t$=1 (e.g., one day), we have the following dynamic model corresponding to equation (1)

$$i(t) = i(t\text{-}1) + \Delta i(t\text{-}1) - p(t\text{-}1)\, i(t\text{-}1) - a(t\text{-}1)\, i(t\text{-}1) - h(t\text{-}1)$$
$$\Delta i(t\text{-}1) = r(t\text{-}1\text{-c})\, i(t\text{-}1\text{-c})$$
$$h(t\text{-}1) = b(t\text{-}1)\, \Delta i(t\text{-}1\text{-d})$$
$$s(t) = s(t\text{-}1) + \Delta i(t) \qquad (3)$$

The parametrical functions $r(t)$, $p(t)$, $a(t)$, and $b(t)$ are partially controllable functions. In addition, the parameter, d, is a controllable parameter also, i.e., we can use $d(t)$ to replace d. Epidemic dynamics are diverse, which is dependent upon the specific functions, $r(t)$, $p(t)$, $a(t)$, $b(t)$, and $d(t)$ (or d). For example, the periodic oscillation may occur in certain conditions, etc.

**2.2 Problem of *r*(*t*)**

The transmission strength of non-hospitalization cases, $r(t)$, is a function of time $t$, dependent upon the type of dynamics of transmission strength of non-hospitalization cases.

As the most occurred type, $r(t)$ may decline with time for the reasons such as the natural attenuation of pathogenicity and transmission strength, the increase of people's self-protection, and other transmission-reducing measures and behaviors used by governments and individuals, etc. In this situation, it can be expressed as the linear approximation

$$r(t) = \mathrm{w} - \mathrm{v}\, t \qquad (4)$$

$r(t)$ can also be represented by other functions. Some of the representative functions include

$$r(t) = \mathrm{u} \sin(\omega t + \theta) + \mathrm{q} \qquad \text{(periodic function)}$$
$$r(t) = \mathrm{u}_n t^n + \mathrm{u}_{n-1} t^{n-1} + \ldots + \mathrm{u}_1 t + \mathrm{q} \qquad \text{(polynomial function)}$$

A representative dynamic type of the model is illustrated in Fig. 1.

**2.3 Peak time and earliest termination time**

The maximum cumulative cases and peak daily new cases in the epidemic period $[1, t_m]$ are as the follows

$$s_{max} = max\, \{s(t) \mid t \in [1, t_m]\}$$
$$\Delta i_{max} = max\, \{\Delta i(t) \mid t \in [1, t_m]\}$$

and the peak time, $t_{max}$, is the time point meeting with $\Delta i(t) = \Delta i_{max}$. For S-shape dynamics of cumulative cases, the cumulative cases increases and tends to its upper limit, i.e., $s_{max}$, and the dynamics of $\Delta i(t)$ is a unimodal curve with peak daily new cases $\Delta i_{max}$ and peak time, $t_{max}$ (Fig. 1)

If there exists a minimum $t_{end}$ that meets $\Delta i(t)=0$ ($t \geq t_{max}$, $t_{end} \leq t \leq t_{end}+c_{max}$), $t_{end}$ is the earliest termination time of epidemic (Fig. 1), where $c_{max}$ is the maximum incubation period.

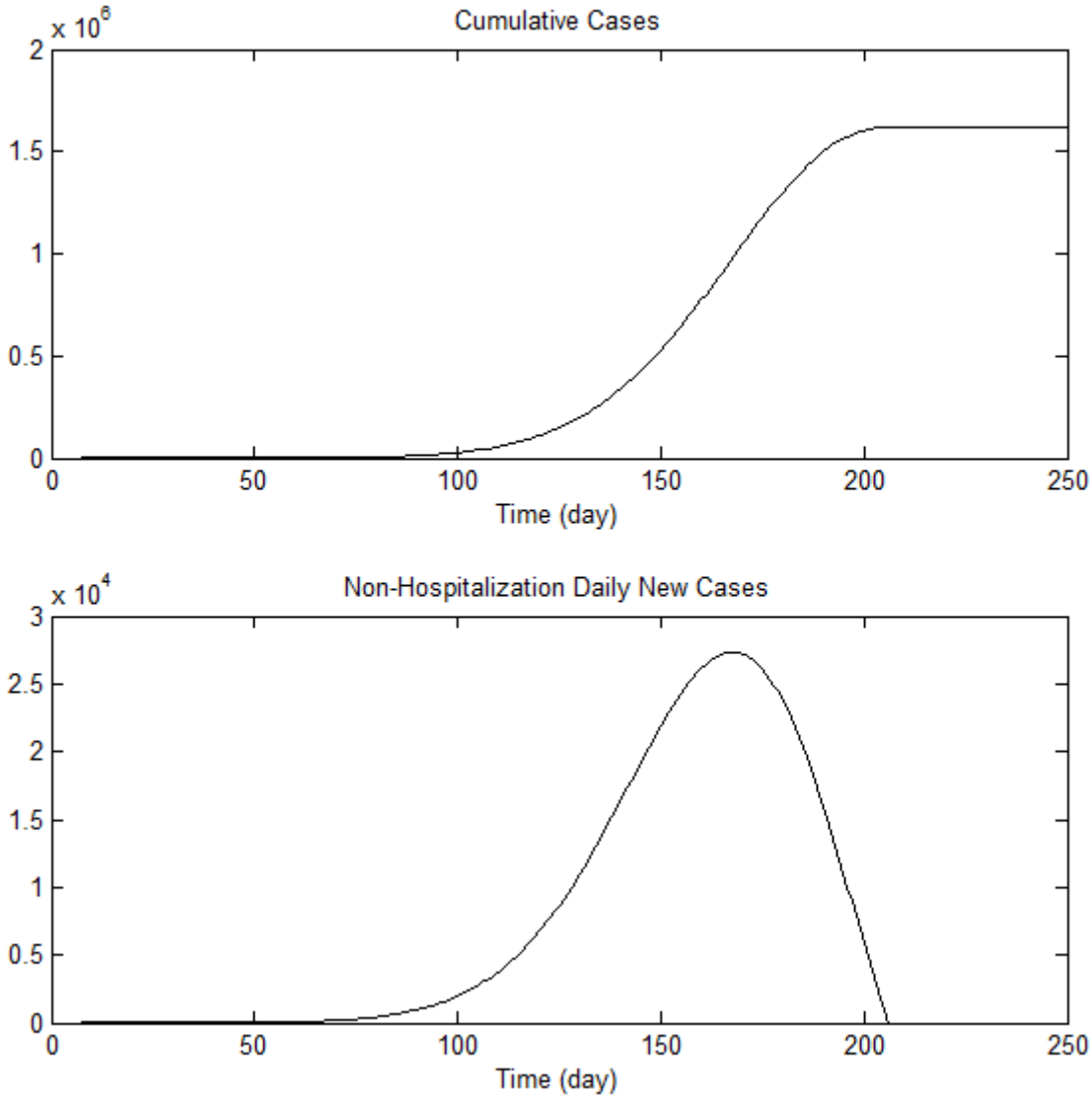


**Fig. 1** Illustration of an epidemic dynamics produced by equation (3) (based on equation (4)).

## 3 Sensitivity analysis

As the most occurred type, based on equation (4) and some parameters of the epidemic disease (Chen et al., 2020; Guan et al., 2020; Guo et al., 2020; Liu et al., 2020; Riou and Althaus, 2020), we assume a set of parametrical values of (c, d, p, a, b, w, v) for sensitivity analysis (Zhang, 2016b). The initial infected population, $i(0)=1$.

### 3.1 Transmission strength

(1) Effect of the change of initial transmission strength (w)

Total cumulative cases (TCC) increases exponentially with initial transmission strength (w) (Fig. 2). Based on w=0.3, the 0.02 of increase in initial transmission strength (w) will result in an increase of TCC by 183%, while the 29679% increase of TCC is expected by an increase of 0.1 in w.

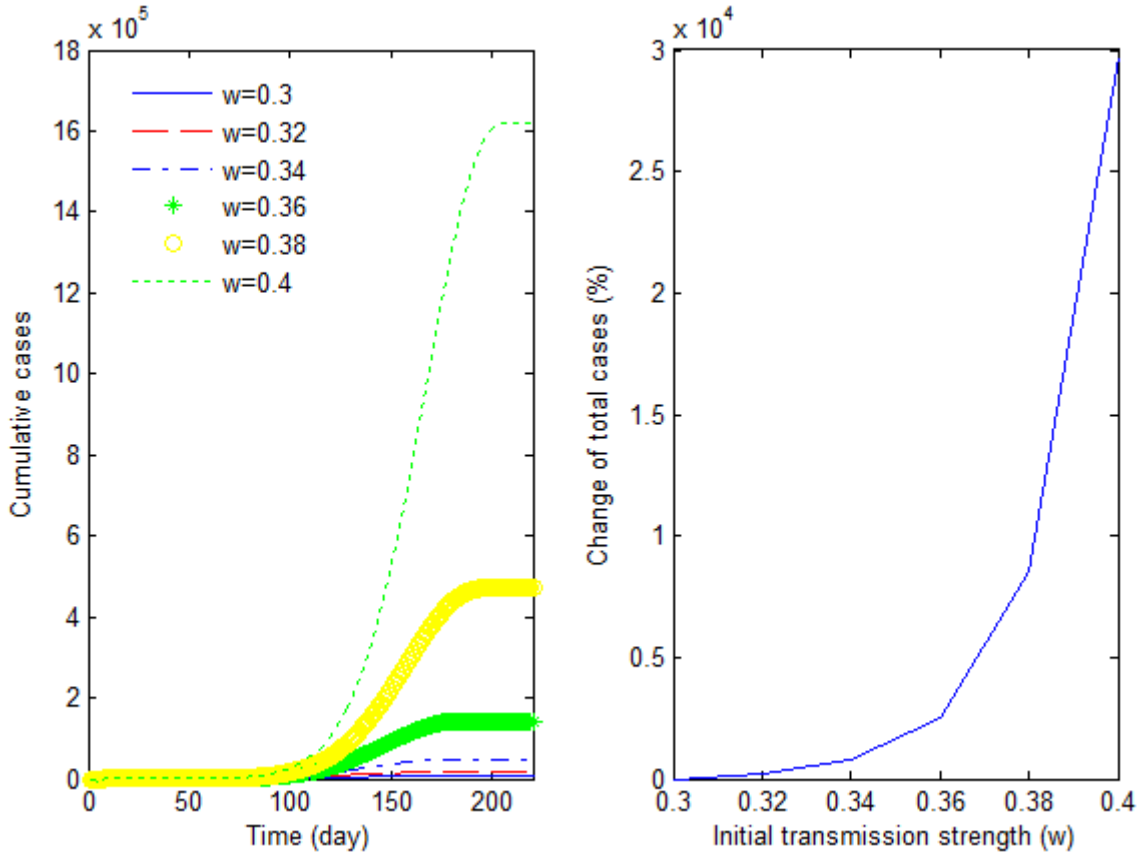


**Fig. 2** Effect of the change of initial transmission strength (w). (c, d, p, a, b, w, v) = (5, 3, 0.01, 0.01, 0.4, w, 0.002).

(2) Effect of the dynamic decline of transmission strength (v)

TCC decreases exponentially with the increase of transmission strength's dynamic decline (v) (Fig. 3). Based on v=0.0028, the 0.0002 of decrease in v will result in an increase in TCC by 112%, while the 0.001 of decrease in v will lead to the increase in TCC by 22354%.

**3.2 Effect of the change of mean incubation period (c)**

TCC decreases dramatically with the increase of mean incubation period (c) (Fig. 4).

**3.3 Effect of the change of hospitalization time (d)**

TCC increases exponentially with the increase of hospitalization time (d) (Fig. 5). Based on d=7, five days decrease in hospitalization time will lead to the decrease of 99% in TCC.

**3.4 Effect of the change of non-hospitalization daily mortality (a) and daily recovery rate (p)**

TCC decreases exponentially with the increase of non-hospitalization daily mortality (a) and non-hospitalization daily recovery rate (p) (Fig. 6 and 7).

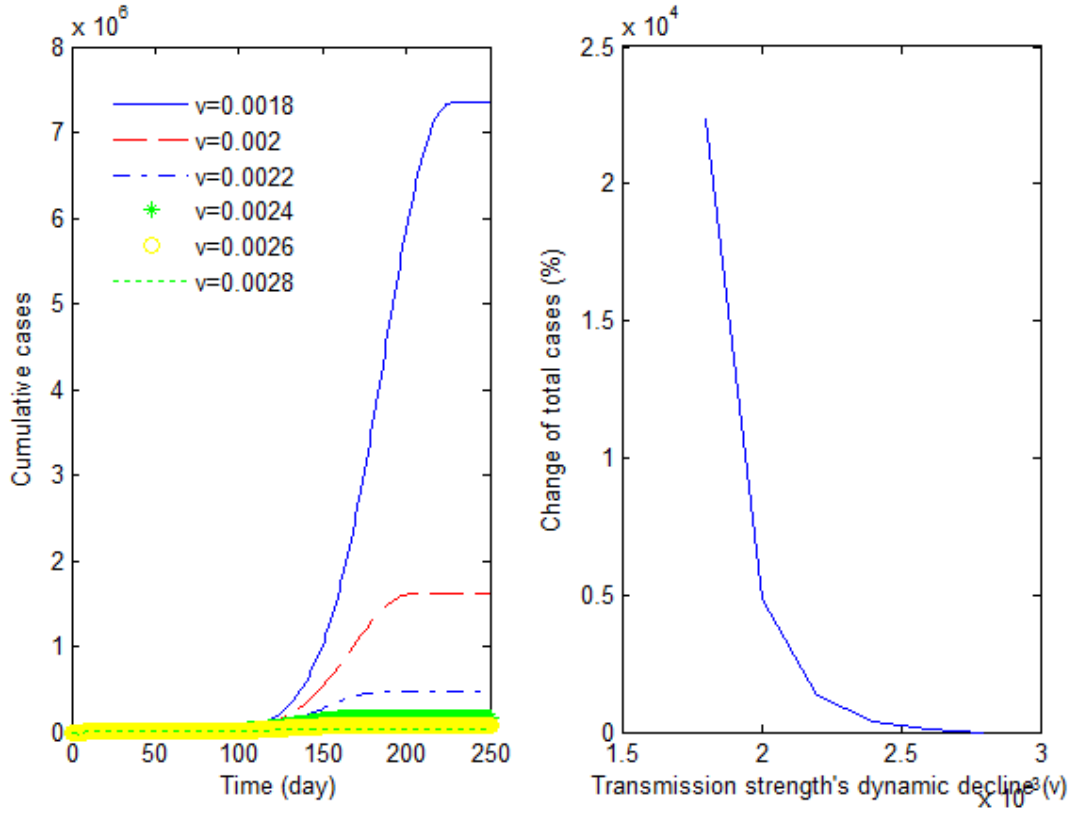


**Fig. 3** Effect of the change of transmission strength' dynamic decline (v). (c, d, p, a, b, w, v) = (5, 3, 0.01, 0.01, 0.4, 0.4, v).

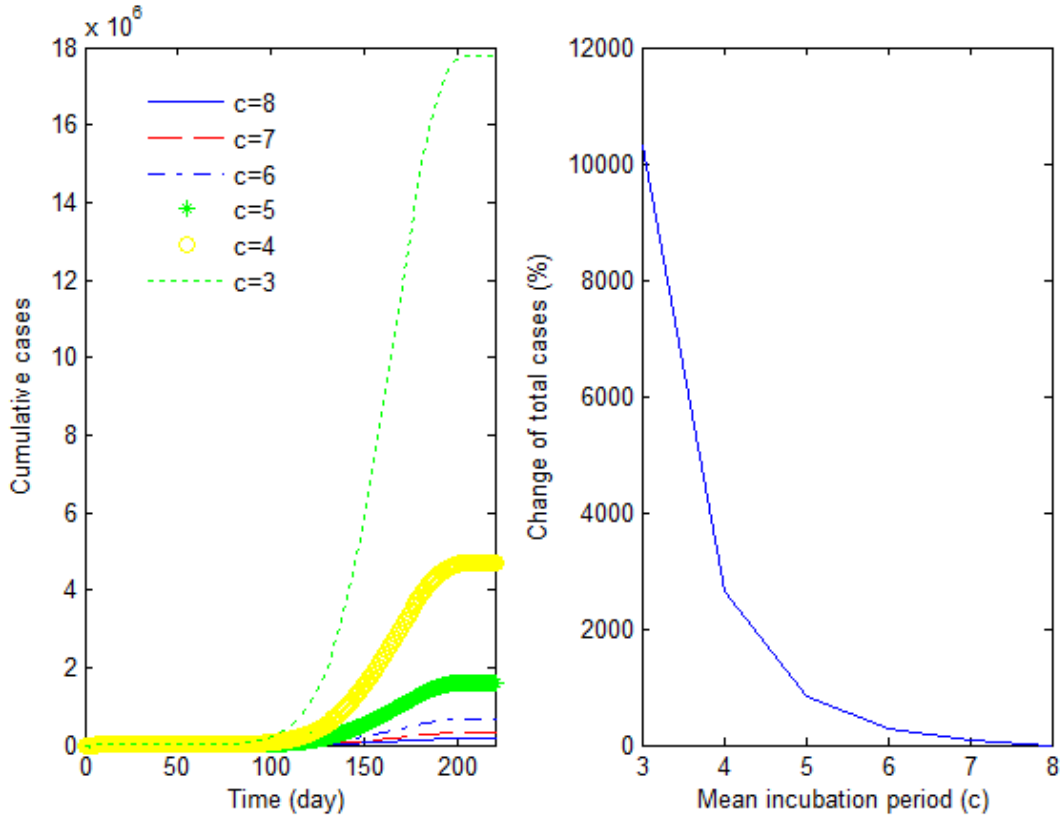


**Fig. 4** Effect of the change of mean incubation period (c). (c, d, p, a, b, w, v) = (c, 3, 0.01, 0.01, 0.4, 0.4, 0.002).

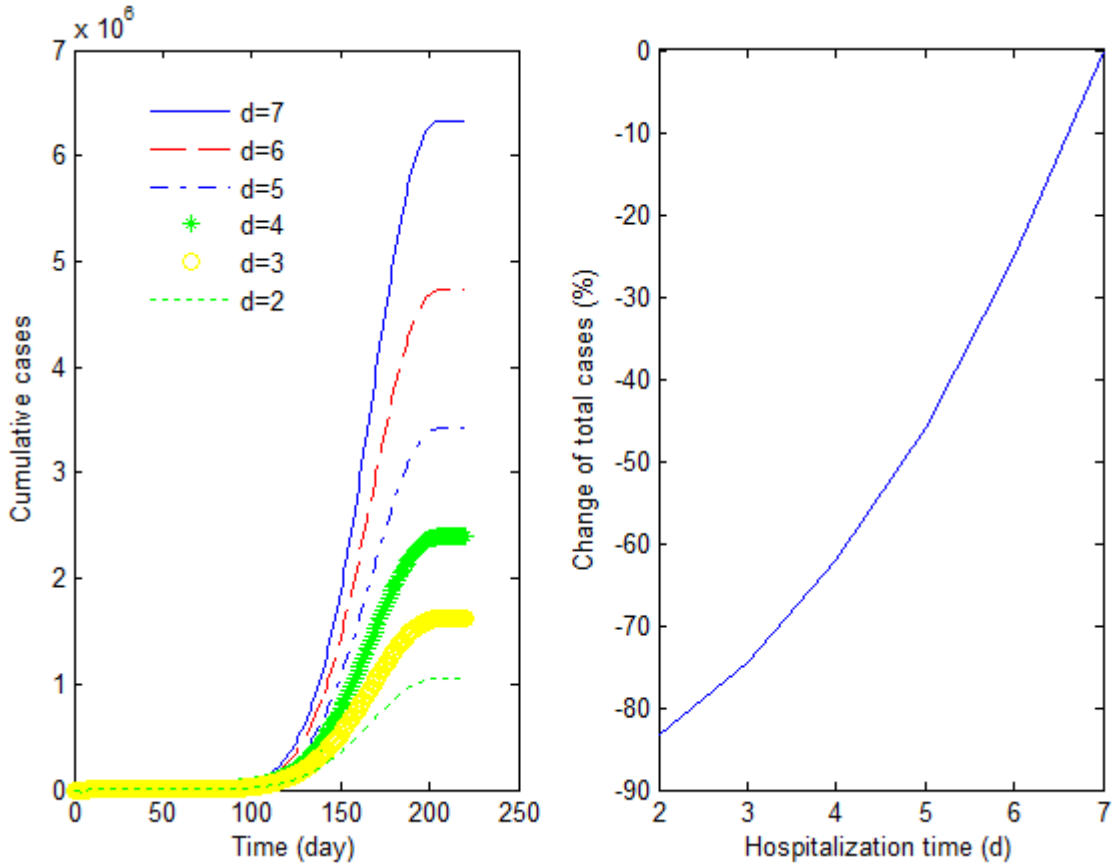


**Fig. 5** Effect of the change of hospitalization time (d). (c, d, p, a, b, w, v) = (5, d, 0.01, 0.01, 0.4, 0.4, 0.002).

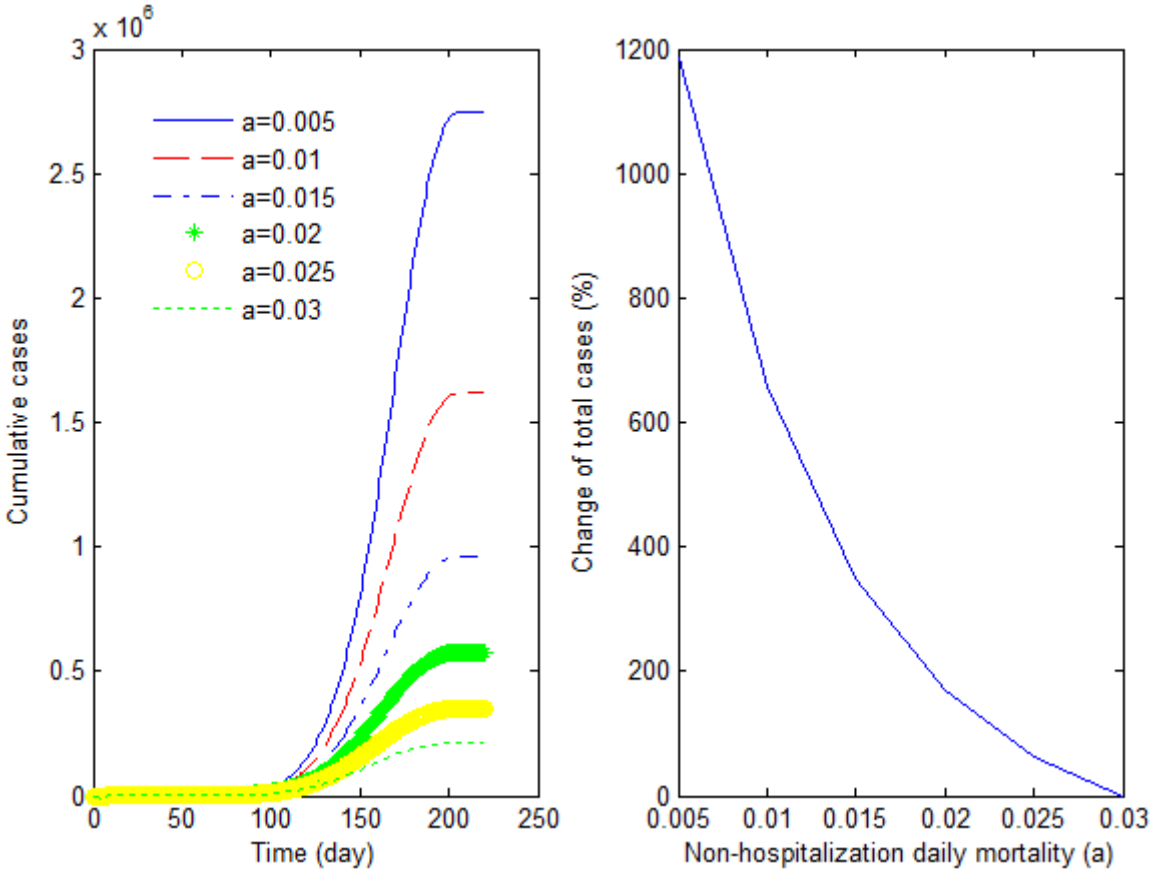


**Fig. 6** Effect of the change of non-hospitalization daily mortality (a). (c, d, p, a, b, w, v) = (5, 3, 0.01, a, 0.4, 0.4, 0.002).

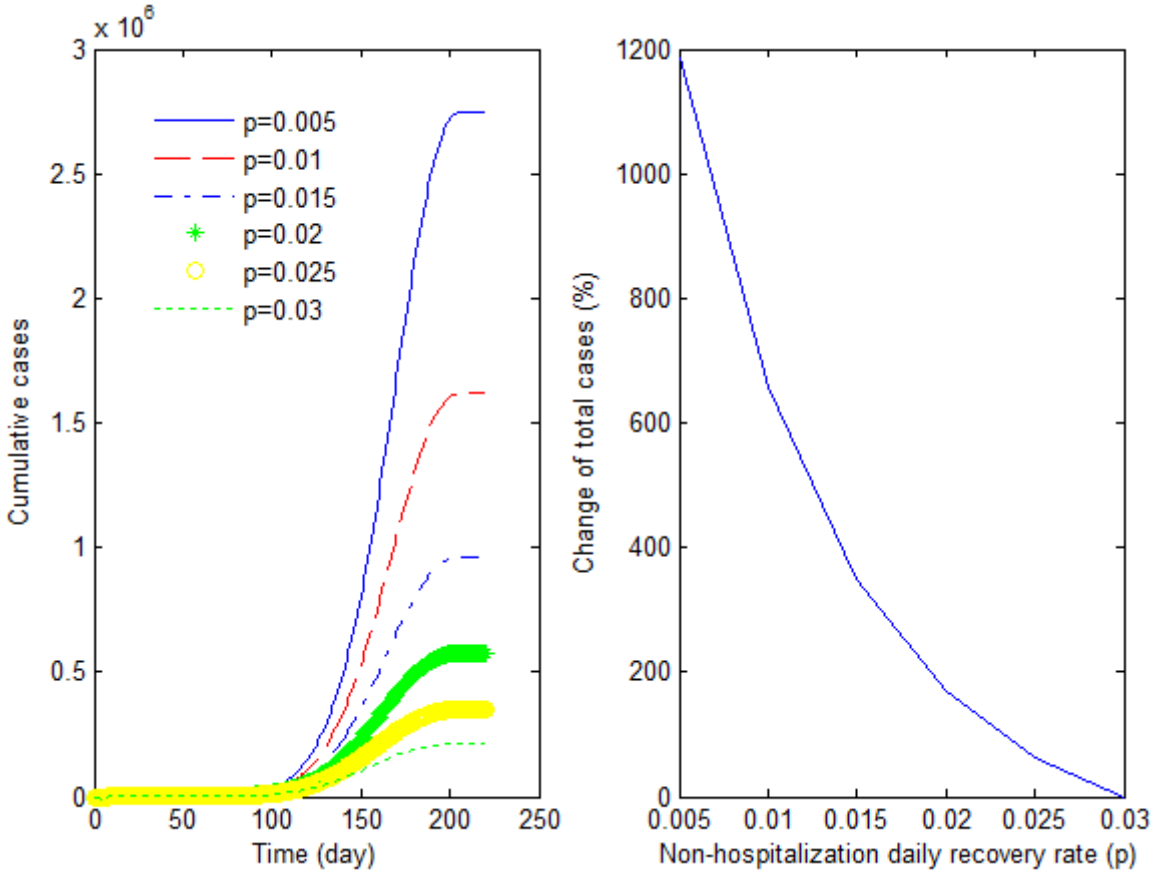


**Fig. 7** Effect of the change of non-hospitalization daily recovery rate (p). (c, d, p, a, b, w, v) = (5, 3, p, 0.01, 0.4, 0.4, 0.002).

### 3.5 Effect of the change of hospitalization proportion (b)

TCC decreases exponentially with the increase of hospitalization proportion (b) (Fig. 8). Based on b=1, the 0.25 days' decrease in hospitalization proportion will lead to the increase in TCC by 385%.

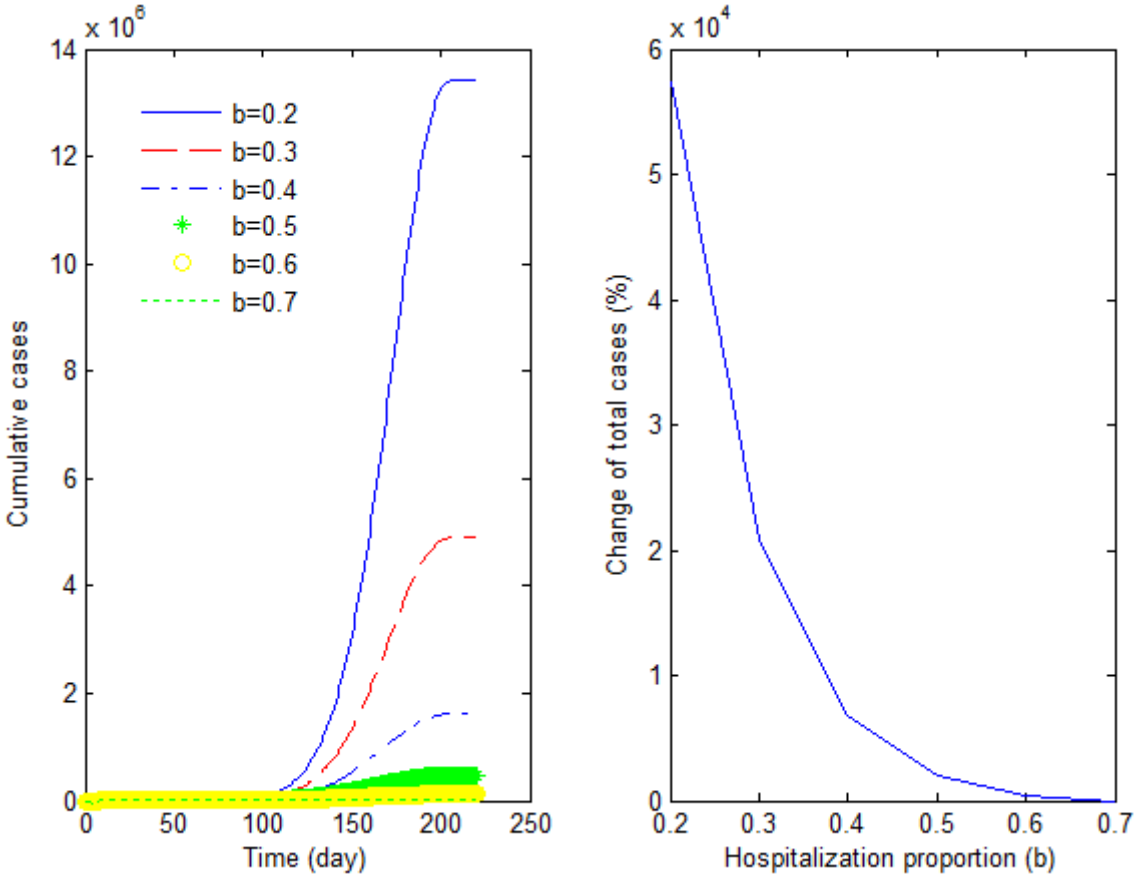


**Fig. 8** Effect of the change of hospitalization proportion (b). (c, d, p, a, b, w, v) = (5, 3, 0.01, 0.01, b, 0.4, 0.002).

## 4 Discussion

Sensitivity analysis indicated that almost all parameters have significant influence on the dynamics and final outcome of human epidemic. Among them, the dynamic change of transmission strength is one of the most important and controllable factors. The human epidemic can be substantially weakened by reducing transmission strength. In addition, the results demonstrated that reducing the delay for hospitalization is much effective in weakening disease epidemic. Sensitivity analysis on non-hospitalization recovery rate reminds us the importance for enhancing immunity to recover from the disease. Mean incubation period is another key factor in determining disease epidemic. The shorter incubation period may lead to the rapid development and serious outcome of epidemic disease.

As shown in sensitivity analysis, the model performance depends upon the exact parametrical values. How to obtain the exact parametrical values for the disease is the basis for the better simulation and prediction of model performance. On the other hand, some parameter, e.g., incubation period, falls in an interval rather than a deterministic value. Therefore, a comprehensive analysis based on scenario prediction is necessary to obtain the most reliable prediction on epidemic dynamics.

As mentioned earlier, the present multi-parametrical model will exhibit various behaviors and it is thus a generalized model. In some situations, the model will produce smooth dynamics, as indicated in Fig. 1. However, sometimes the stepwise or wavy dynamics may occur due to the constant parameterization (e.g., constant incubation period c, constant hospitalization time d, etc.) or specific parametrical values in the model, as exhibited in scenario predictions and some situations in sensitivity analysis. To produce more smooth

dynamics, dynamic parameters, as indicated in the model (1) to (3), can be used.

In present study, the concept, *hospitalization*, can be further defined to include two parts, being sent to the hospital (i.e., hospitalization) and being isolated at home or other places (i.e., self-isolation). In the situation of self-isolation, the infection case will finally recover from infection, or die, or is sent to the hospital for medical treatment. For such an extended concept of hospitalization (hospitalization time, hospitalization proportion, etc.), the models above hold also.

To improve prediction or simulation performance of the model, Monte Carlo or randomization method can be used (Zhang, 2010, 2011). The parameters $r(t)$, $p(t)$, $a(t)$, $b(t)$, and $d(t)$ (or d) can be treated as interval variables. The incubation period c, can be treated as an interval variable also (in this situation, c is changeable and in a sense, $c(t)$=c). Randomly assigning values fallen in the corresponding intervals to these parameters and obtaining the modeled infection cases. Repeating the procedure many times, e.g., 100 times, and calculating the averaged infection cases, the finally modeled infection cases are thus achieved.

How to evaluate the goodness of a dynamic model in both mechanism explanation and prediction? We argue that a realized model should meet these criteria: (1) as many as epidemiological parameters with explicit meaning should be included in the model; (2) as many as practical behaviors should be theoretically produced by the model; (3) major time points (peak time, termination time, etc.) and corresponding population sizes can be better predicted, and (4) the dynamic trajectoy produced by the model exhibits a good fitness with the practical one. Generally, the present model meets all of the criteria above, superior to conventional SIR models (Anderson and May, 1981; Kermack and McKendrick, 1927).

In future applications of the present model, we suggest that the exact function, $r(t)$, should be carefully estimated and used in the model (e.g., an approximate estimation of $r$ is $r$=(R0-1)/Tg, where R0 is the basic reproduction number, and Tg is the disease generation time). In addition, the asymptomatic infection was ignored in present model due to its general insignificance in most epidemic diseases (Verma et al., 2018). For some diseases, these infections may play an important role and should be included in the model. Further, more complex models that include other factors or processes, e.g., network models, can be developed for specific uses (Zhang, 2012, 2015, 2016a, 2018; Banerjee, 2017; Shams and Khansari, 2019; Chen et al., 2020a).

**Acknowledgment**

WenJun Zhang, ZeLiang Chen, Yi Lu, ZhongMin Guo, YanHong Qi, and GuoLing Wang contributed equally to this work. This work is supported by The National Key Research and Development Program of China (2017YFD0201204), and Guangzhou Science and Technology Project (No. 201707020003).

**References**

Anderson RM, May RM. 1981. The population dynamics of microparasites and their invertebrate hosts. Journal of Animal Ecology, 291: 451-524

Banerjee S. 2017. A stage structured hybrid model for within-host emerging infectious disease modelling. Network Biology, 7(4): 94-97

Chen TM, Rui J, Wang QP, et al. 2020a. A mathematical model for simulating the transmission of Wuhan novel Coronavirus. bioRxiv (https://doi.org/10.1101/2020.01.19.911669)

Chen ZL, Zhang WJ, Lu Y, et al. 2020b. From severe acute respiratory syndrome-associated coronavirus to 2019 novel coronavirus outbreak: similarities in the early epidemics and prediction of future trends. Chinese Medical Journal, 133(9): 1112-1114

Fuxa JR, Tanada Y. 1987. Epizootiology of Insect Diseases. 534, John Wiley & Sons, New York, USA

Guan WJ, Ni ZY, Hu Y, et al. 2020. Clinical characteristics of 2019 novel coronavirus infection in China. medRxiv (doi: 10.1101/2020.02.06.20020974)

Guo Q, Li M, Wang CH, et al. 2020. Host and infectivity prediction of Wuhan 2019 novel coronavirus using deep learning algorithm. bioRxiv (https://doi.org/10.1101/2020.01.21.914044)

Kermack WO, McKendrick AG. 1927. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society A, 115(772): 700-721

Liu T, Hu JX, Kang M, et al. 2020. Transmission dynamics of 2019 novel coronavirus (2019-nCoV). bioRxiv (https://doi.org/10.1101/2020.01.25.919787)

Riou J, Althaus CL. 2020. Pattern of early human-to-human transmission of Wuhan 2019-nCoV. bioRxiv (https://doi.org/10.1101/2020.01.23.917351)

Shams B, Khansari M. 2019. Average reachability: A new metric to estimate epidemic growth considering the network structure and epidemic severity. Network Biology, 9(3): 42-57

Verma R, Tiwari SP, Upadhyay RK. 2018. Fuzzy modeling for the spread of influenza virus and its possible control. Computational Ecology and Software, 8(1): 32-45

Zhang WJ. 2010. Computational Ecology: Artificial Neural Networks and Their Applications. World Scientific, Singapore

Zhang WJ. 2011. A Java algorithm for non-parametric statistic comparison of network structure. Network Biology, 1(2): 130-133

Zhang WJ. 2012. Computational Ecology: Graphs, Networks and Agent-based Modeling. World Scientific, Singapore

Zhang WJ. 2015. A generalized network evolution model and self-organization theory on community assembly. Selforganizology, 2(3): 55-64

Zhang WJ. 2016a. Selforganizology: The Science of Self-Organization. World Scientific, Singapore

Zhang WJ. 2016b. Some methods for sensitivity analysis of systems / networks. Network Pharmacology, 1(3): 74-81

Zhang WJ. 2018. Fundamentals of Network Biology. World Scientific, Singapore

Zhang WJ, Chen ZL, Lu Y, et al. 2020. A generalized discrete dynamic model for human epidemics. bioRxiv (https://doi.org/10.1101/2020.02.11.944728)

Zhang WJ, Pang Y, Qi YH, Chen QJ. 1997. Simulation model for epizootic disease of *Spodoptera litura* F. baculovirus. Acta Scientiarum Naturalium Universitatis Sunyatseni, 36(1): 54-59

Zhang WJ, van der Werf W, Pang Y. 2011. A simulation model for vegetable-insect pest-insect nucleopolyhedrovirus epidemic system. Journal of Environmental Entomology, 33(3): 283-301