

# Effective Approaches to Control the Epidemic Based on the Improved SIR Model

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## Abstract

In this paper, we have established a SECADI model on the basis of the traditional epidemic model and under the consideration of factors such as the spread of the disease, the quantity of the medicine in need, the medicine production speed etc. We have improved the crowd classification standard and the spread styledifferential equation model in classical SIR model. We distinguished the crowd into six categories, including the susceptible, the exposed, the curable, the advanced, the dead and the immune, and we established integrated transformation relationships between them after taking control measures through qualitative and quantitative method, and then derive the adequate epidemic differential equation model before taking controls. We applied the method of computer simulation to solve the model, worked out uncertain parameters with the method of parameter identification, and we verified the validity and accuracy of the SECADI model. Meanwhile, we calculated with the actual data of Ebola in the epidemic area in Western Africa, simulated the evolution of the epidemic, analyze and offered effective approaches to control the epidemic situation. We further discussed development directions of this model in the end.

**Keywords:** SECADI model, control the epidemic, SIR model, computer simulation, parameter identification

## 1. Introduction

February 2014, Ebola virus broke out in Western Africa with the trend of spreading rapidly. Up to September 2014, the virus has caused 3,300 deaths. People infected with Ebola virus can cause Ebola hemorrhagic fever. The virus has a 50-90 percent mortality rate. Due to the high infection rate and mortality rate, Ebola virus has caused the attention of international community about the epidemic model.

Existing epidemic models include simple model, SI model, SIS mode and SIR model, of which the most widely used, is the SIR model. Yongwei Zhou and others from Zhengzhou Institute of Aeronautical Industry Management studied the infectious disease management model by using the theory of Markov skeleton process (Zhou & Li, 2011). Aschwanden, C. from Hawaii University focused on simulating how an infectious viral disease spreads by computer and took SARS and common flu as examples (Aschwanden, 2004). Yoshihiro Maki from Kyushu Institute of Technology applied the SIR model to see the behavior of the pandemic with easy computation (Maki & Hirose, 2013). Huaiyu Tian and others from Disease Control Center of Hunan Province made a further study about the epidemic model using the SIR model with differential equations (Xiao, Tian, & Lin et al., 2013). Hua Jiang and her partners from Sichuan Academy of Medical Sciences made a simulation of the spread of the Ebola virus using the SIR model and differential equations, taking China as an example, and finally solved out results about the best time to take actions (Jiang, Pan, & Sun, 2014). Guangzheng Li and others from Shanghai University researched the epidemic spread model with immune failure characteristic on complex networks. They used the mean field theory to analyze the spread of diseases, and compare the result with that of computer simulation, proved that the determination of spreading on a complex network is the network topology (Li & Shi, 2006). Chengyi Xia from Nankai University proposed an SIRS model with direct immune based on traditional SIRS model. Using the mean field theory, he pointed out that epidemic critical threshold is strongly related with the network topology, direct immune speed, immune loss rate and others (Li & Ma, 2006). Jianquan Li from Xi'an Transportation University found out that the input of constant and index will change the progress of disease constitutional through the research of two kinds of SEIS epidemic model (Xia, Liu, & Chen, 2008).

They proved when endemic equilibrium exists, that part is willing to stay at a stable level, and the disease will exist constantly. Qian Huang from Beijing Science and Technology University proposed an improved SIRS epidemic model describing the influence of population and immune based on traditional SIRS model. They found out that the most effective way to cut off the spread of disease is reducing people touching rate and increasing immune rate (Huang & Min, 2014).

This paper analyzes the regulation of spread of the Ebola virus, studies the characteristic of its spread, and researches the statistics published by WHO based on an improved SIR model.

## 2. SECADI Model

### 2.1 Preparation

In the improved SECADI model, total population can be divided into six categories: (1) The susceptible: People who have not been infected but do not have the immunity. If they make effective contacts with the infective, they will be vulnerable to infections. (2) The exposed: People who have made effective contacts with the infective but are still in the incubation period. (3) The curable: People who have been infected but can be cured by medication. (4) The advanced: People who have been infected and cannot be cured by medication, constituting the dead directly. In our model, the total number of the curable and the advanced is the infective number. (5) The dead. (6) The immune: People including the susceptible who have taken vaccine, the curable and the exposed who have been cured.

The transformation relationships between six kinds of people are shown in Figure 1:

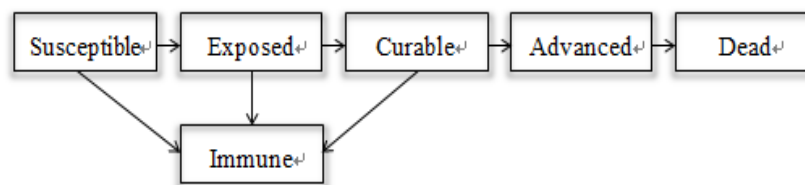


Figure 1. Transformation relationship between six kinds of people

In Figure 1, the susceptible will carry virus through effective contacts with the infective and convert into the exposed, at the same time they can also become the immune through medication. If the exposed did not take medication in time, they would become the curable and can also become the immune by timely medication. Similarly, if the curable did not take medication in time, they would convert into the advanced, or become the immune by timely medication. Assumed that the advanced cannot be cured. They constitute the death toll in our model directly. And assumed that the total population is fixed, without considering the birth rate and natural mortality rate in this period. The immune will no longer be infected by Ebola viruses. The advanced cannot be cured by medication, meaning that the advanced convert into the death toll directly. The exposed do not have the infectious ability. Medications will be distributed to the curable firstly, and the rest of medications will be distributed to the exposed and the susceptible randomly.

First, define the relationship between number of cases and time as the function 1 below:

$$N = N_0(1 + K)^t \quad (1)$$

In this function,  $N$  is the total population,  $N_0$  is the initial value of cases number,  $K$  is a dominant parameter, showing that the probability of people infected by someone in a certain geographic range. During the entire process of the outbreak,  $K$  determines the evolution of the epidemic trend. Its value depends on the strength of preventions taken by the Health Department, public awareness and other factors. Furthermore, its value varies at the beginning of the epidemic and the late period. With the improvement in public awareness, the value of  $K$  will decrease continuously.

(1) According to conversion relations in Figure 1 and previous effective hypothesis, we can draw the daily variation of the susceptible, as following equation 2:

$$\frac{dS(t)}{dt} = -\lambda(t)S(t)[C(t) + A(t)] - P_1S(t)\frac{m(t) - NC(t)}{N[S(t) + E(t)]} \quad (2)$$

where  $S(t)$  represents the proportion of the susceptible to the total population at  $t$  moment,  $C(t)$  is the proportion of the curable to the total population at  $t$  moment,  $A(t)$  is the proportion of the advanced to the total population at

$t$  moment,  $E(t)$  is the proportion of the exposed to the total population at  $t$  moment,  $m(t)$  is total quantity of medication people can obtain at  $t$  moment,  $\lambda(t)$  is daily infection rate,  $P_1$  is the probability of the susceptible changing into the immune. Assume the susceptible have great possibilities changing into the immune by taking medication, so we set  $P_1=0.99$  here.

Daily variation of the exposed equals to the transferred-in number from the susceptible minus the transferred-out number of the immune and the curable, that is,

$$\frac{dE(t)}{dt} = \lambda(t)S(t)[C(t) + A(t)] - P_2E(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} - \varepsilon E(t) \tag{3}$$

where  $P_2$  is the probability of the exposed changing into the immune, we take  $P_2=0.6$  here through selecting related information.  $\varepsilon$  is the daily morbidity rate during the incubation period.

Daily variation of the curable equals to the transferred-in number from the exposed minus the transferred-out number of the immune and the advanced, that is

$$\frac{dC(t)}{dt} = E(t)\varepsilon - P_3C(t) - C(t)\varphi \tag{4}$$

where  $P_3$  is the probability of the curable changing into the immune, and set  $P_3=0.6$  after analyzing.  $\varphi$  is the daily deterioration rate.

Daily variation of the advanced equals to the transferred-in number from the curable minus the transferred-out number of the dead, that is,

$$\frac{dA(t)}{dt} = C(t)\varphi - A(t)\xi \tag{5}$$

where  $\xi$  is the daily death rate.

Daily variation of the dead equals to the transferred-in number from the advanced, that is,

$$\frac{dD(t)}{dt} = A(t)\xi \tag{6}$$

Diurnal variation of the immune equals to the transferred-in number from the susceptible plus the transferred-in number of the exposed and the curable, that is,

$$\frac{dI(t)}{dt} = P_1S(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} + P_2E(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} + P_3C(t) \tag{7}$$

where  $I(t)$  is the proportion of the immune to the total population at moment  $t$ .

### 2.2 Epidemic Model in the Post-Controlled Stage

The spread of the disease can be expressed by the daily infection rate  $\lambda(t)$ . There are different factors influencing  $\lambda(t)$  at different stage. In the post-controlled stage, daily infection rate  $\lambda(t)$  decreased as the increasing precautionary measures, medicine transportation and production. Reasons that cause increasing precautionary measures can be mainly cited as the following two aspects, one is the fear of the disease, and it forces people to strengthen their awareness. We use inherent awareness index  $a(t)$  to depict, the other is the policy and laws promulgated by relevant departments, which strengthen public awareness as well. We use control strength of relevant departments,  $s(t)$ , to depict. Their relations are shown in Figure 2 below.

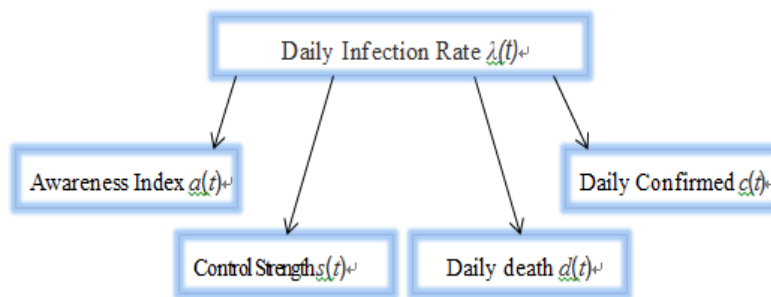


Figure 2. Relations of factors influencing  $\lambda(t)$

Before making quantitative analysis and calculating, we can use the qualitative analysis method to determine the function relationships between various factors and employ the method of parameter identification in the process of solving parameters (He & Tang, 2013).

### 2.2.1 Determination of Epidemic Development $l(t)$

The main indicators influencing the development are daily newly-added death toll  $d(t)$  and daily newly-added confirmed cases  $c(t)$ . We can find the weighted sum after dealing with these two factors by normalization and obtain the following equation,

$$l(t) = q_1 \frac{d(t)}{\max d(t)} + q_2 \frac{c(t)}{\max c(t)} \quad (8)$$

where  $q_1$  and  $q_2$  are the respective relative influencing weight of  $d(t)$  and  $c(t)$  to the development of epidemic.

### 2.2.2 Determination of Control Strength from Relevant Departments, $s(t)$

It relates to the following factors: (1) Epidemic development. (2) When  $t=t_0$ ,  $s(t)$  has an initial value, that is the control strength from relevant departments  $k_0$  ( $0 < k_0 < 1$ ). (3)  $s(t)$  increases with the epidemic growing severe. Due to the delay in realizing the urgency of epidemic and the insufficient of taking measures in the early days,  $s(t)$  increases in a slow speed, but when the epidemic develops to a certain extent, control strength is increases, leading the growth speed of  $s(t)$  increasing. (4) When the epidemic aggravates to a certain degree,  $s(t)$  tends to its largest value, that is, 1. And the expression of  $s(t)$  is,

$$s(t) = k_0 + k_1 \left(1 - e^{-\frac{l(t)}{\sigma_1}}\right) \quad (9)$$

where  $k_0 + k_1 = 1$ .

### 2.2.3 Determination of the Public Awareness $a(t)$

Public awareness to Ebola varies with the development of the epidemic situation. When the epidemic is announced in the early days, changes in epidemic cause great public attention, and public awareness fluctuates great with a tiny variation in epidemic. Gradually, fluctuations of  $a(t)$  become gently till smooth. The following expression can depict the relationship between  $a(t)$  and  $l(t)$  quantitatively.

$$a(t) = k_2 - k_3 e^{-l(t)} \quad (10)$$

When  $l(t)=0$ ,  $a(t)=0.2$ , which is the inherent awareness indicator. When  $l(t)$  tends to be infinite,  $a(t)$  tends to be 1.

### 2.2.4 Determination of the Strength Index of Measures Taken by People $w(t)$

The strength index of measures taken by people  $w(t)$  is influenced by the control strength from relevant departments  $s(t)$  and the public awareness  $a(t)$ . Their influence degree to  $w(t)$  is roughly identical. So we can determine the following equation.

$$w(t) = 0.5s(t) + 0.5a(t) \quad (11)$$

### 2.2.5 The Relationship between $w(t)$ and $\lambda(t)$

It should satisfy: (1) When  $w(t)=0$ ,  $\lambda(t)$  will achieve its maximum, that is, the daily infection rate in the pre-controlled stage. (2) With the incensement of  $w(t)$ ,  $\lambda(t)$  will decrease. If the value of  $w(t)$  is not big, its impact on the variation of  $\lambda(t)$  will be relatively small. If  $w(t)$  is more than a certain value, its impact will be relatively strong. (3) When  $w(t)$  approaches one (it can never achieve one),  $\lambda(t)$  approaches zero.

According to the above three points, we can determine the shape of the changing curve between  $\lambda(t)$  and  $w(t)$ , and the function can be expressed as,

$$\lambda(t) = k_4 \left(1 - e^{-\frac{(1-w(t))^2}{\sigma_2}}\right) \quad (12)$$

Where  $\sigma_2$  is an undetermined constant.

Then we take influence to the epidemic of the speed of medicine development into consider, so we introduce the growth model of  $M(t)$ , which means the growth of medicine. The growth of medicine against Ebola has the following features: (1) In prophase of the epidemic, the speed of new medicine development is rather slow, as well as the growth of production. (2) As the growing attention among various countries, invest capital will

increase, which makes it possible for rapid speed of medicine production. (3) Due to the restrictions of complicated production process, scarce production equipment and decreasing invest after the control of epidemic situation, the growth of medicine against Ebola virus will slow down. In conclusion, the formula is,

$$\frac{dM(t)}{dt} = rM(t)\left(1 - \frac{M(t)}{M_{\max}(t)}\right) \tag{13}$$

Where  $rM(t)$  stands for the new medicine production growth trend, due to large amounts of money into the process. The factor  $1 - M(t)/M_{\max}(t)$  stands for medicine production process and production equipment complex number retardation of production speed.

Medicine arose from the production of finished product in the user’s process can be thought of as a cycle, the cycle of each day, the medicine distribution to the number of users can be approximately regarded as average, namely:

$$m(t) = \frac{M(t)}{t_{total}} \tag{14}$$

The  $t_{total}$  for time from the medicine production and arrival on users, and according to the actual situation, it can be divided into from origin to receive and two stages from accept to distribute to users, set the time needed for two phase for  $t_1, t_2$ , is available  $t_{total} = t_1 + t_2$ .

From what has been discussed above, the spread model of Ebola in the post-controlled stage is:

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = -\lambda(t)S(t)[C(t) + A(t)] - P_1S(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} \\ \frac{dE(t)}{dt} = \lambda(t)S(t)[C(t) + A(t)] - P_2E(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} - \varepsilon E(t) \\ \frac{dC(t)}{dt} = E(t)\varepsilon - P_3C(t) - C(t)\varphi \\ \frac{dA(t)}{dt} = C(t)\varphi - A(t)\xi \\ \frac{dD(t)}{dt} = A(t)\xi \\ \frac{dI(t)}{dt} = P_1S(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} + P_2E(t) \frac{m(t) - NC(t)}{N[S(t) + E(t)]} + P_3C(t) \\ \lambda(t) = k_4 \left(1 - e^{-\frac{(1-w(t))^2}{\sigma_2}}\right) \\ S + E + C + A + D + I = 1 \\ S(0) = S_0, E(0) = E_0, C(0) = C_0 \\ A(0) = A_0, D(0) = D_0, I(0) = I_0 \end{array} \right. \tag{15}$$

where  $S_0, E_0, C_0, A_0, D_0, I_0$  is the original value of various people in the system.

### 2.3 The Spread Model in the Pre-Controlled Stage

In the early days of the epidemic, Ebola spreads in accordance with the natural law, daily infectious rate  $\lambda(t)$  remains unchanged, that is, an undetermined constant  $\lambda_0$ . Its definite value can use parameter identification to confirm in the following pages.

In the early days of epidemic, public awareness is weak. There is every chance that Super Infection Events (SIE) would happen in some areas coupled with the own characteristics of the Ebola virus spreading. SIE is a sudden outbreak of infectious epidemic events.

The characteristic of SIE is that it can make the number of the infected increase rapidly in a short time. So the effect of SIE can be treated as a pulse transient behavior, which can be described by an impulsive differential equation. We define the pulse function as

$$\delta_\varepsilon(x - x_0) = \begin{cases} \frac{1}{2\varepsilon}, & x_0 - \varepsilon < x < x_0 + \varepsilon \\ 0, & \text{other value} \end{cases} \tag{16}$$

In the function,  $\delta$  is defined as  $\delta(x - x_0) = \lim_{\varepsilon \rightarrow 0} \delta_\varepsilon(x - x_0)$ .

Thus, we can get the natural spread model in the pre-controlled stage as follow:

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = -\lambda(t)S(t)[C(t) + A(t)] - N \sum_{j=1}^m \alpha_j \delta(t - t_j) \\ \frac{dE(t)}{dt} = \lambda(t)S(t)[C(t) + A(t)] - \varepsilon E(t) + N \sum_{j=1}^m \alpha_j \delta(t - t_j) \\ \frac{dC(t)}{dt} = E(t)\varepsilon - C(t)\varphi \\ \frac{dA(t)}{dt} = C(t)\varphi - A(t)\xi \\ \frac{dD(t)}{dt} = A(t)\xi \\ S + E + C + A + D + I = 1 \\ S(0) = S_0, E(0) = E_0, C(0) = C_0 \\ A(0) = A_0, D(0) = D_0, I(0) = I_0 \end{array} \right. \quad (17)$$

In the model,  $S_0, E_0, C_0, A_0, D_0, I_0$  is the original value of various people in the system.  $\varepsilon$  is the daily morbidity rate during the incubation period,  $\varphi$  is the daily deterioration rate,  $\xi$  is the daily death rate.

### 3. Numerical Simulation and Interpretation of Results

In this part, we use the data of Ebola in Western-Africa, and take 45 cases of newly-infected in Western Africa on June 2, 2014 as the initial value of the epidemic. We apply the method of computer simulation to solve the differential equation model and solve the undetermined parameters by parameter identification. Assume that the susceptible have great possibilities changing into the immune by taking medication, so  $P_1=0.99$ , and we can get that  $N=24389197, P_2=0.6, \varepsilon=1/7, P_3=0.6, \varphi=1/3, \xi=1/2, q_1=0.6, q_2=0.4$ , and the average number of infection per SIE cases is 20.

According to the analysis of the main production data of new medicines, can get parameters:  $r=2.714, M_{max}=300000$ .

According to the computer simulation and parameter identification, we can get that  $k_2=1, k_3=0.8, \lambda_0=0.589, \sigma_0=27.1637, \sigma_1=0.2437$ .

And the comparison of the results in the pre-controlled and the post-controlled stage can be seen in the following Figure 3.

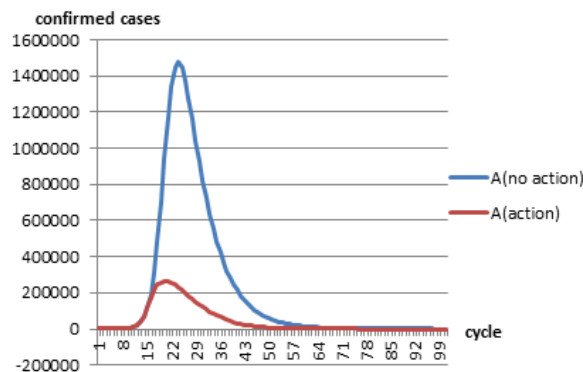


Figure 3. Results in the pre-controlled and the post-controlled stage

From Figure 3, we can intuitively tell that the number of cases before and after taking control measures have obvious differences, indicating that a good effect can be made to optimize the eradication of Ebola through effective medication. That means the SECADI model is productive.

### 3.1 The Influence to the Epidemic of the Changing in Control Strength

The relevant departments' control strength  $s(t)$  has a great effect on epidemic. We take its initial value  $s(t_0)$  as 0.9, 0.7 and 0.4 into the model. After simulation, we can see the results as Figure 4.

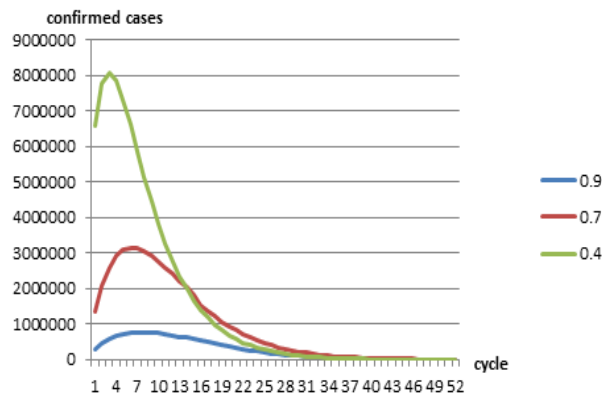


Figure 4. Changing of relevant departments' control strength

From Figure 4, we can see that the weaker the control strength is, the unstable the curve is in the later stage. There might even be a small peak of epidemic situation. On the contrary, the stronger the control strength is, the better the epidemic condition is. Therefore, relevant departments' control strength should keep stronger. And they should not let their guard down when the number of confirmed cases drop.

### 3.2 The Influence of the Changing in Public Awareness

For such a sudden disease, people have an inherent awareness index  $a(t)$ . We take  $a(t)$  as 0.1, 0.2 and 0.3 into the model. After simulation, we can see the results as Figure 5.

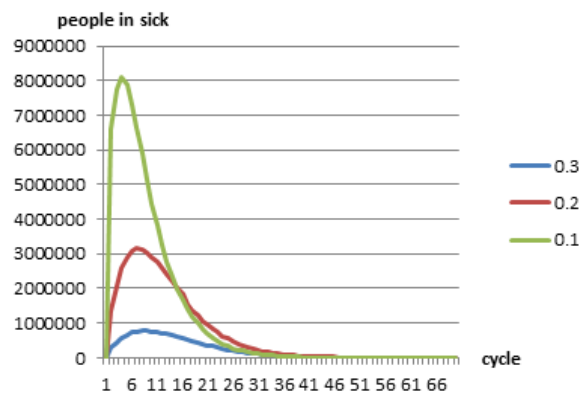


Figure 5. Changing of the public awareness

The figure reflects that the weaker the public awareness is, the unstable the curve is in the later stage. There might even be a small peak of epidemic situation. On the contrary, the stronger the public awareness is, the better the epidemic condition is. Hence, the bigger the public awareness is, the better the situation is. In real life, relevant departments should strengthen propaganda, call on the public to change bad habits and raise vigilance. Let public obtain more knowledge of the epidemic to strengthen the inherent public awareness, shorten the epidemic cycle and reduce the possibility of appearing peak.

### 3.3 The Influence of the Changing in the Medicine Production Speed

In this paper, we set the initial supply of medicines 5000 doses according to the actual situation. As time passing by, the medicine supply increases with a promotion in the production. Using the logistic growth model, we can compare the changing situation of the epidemic before and after expanding production, as is shown in Figure 6.

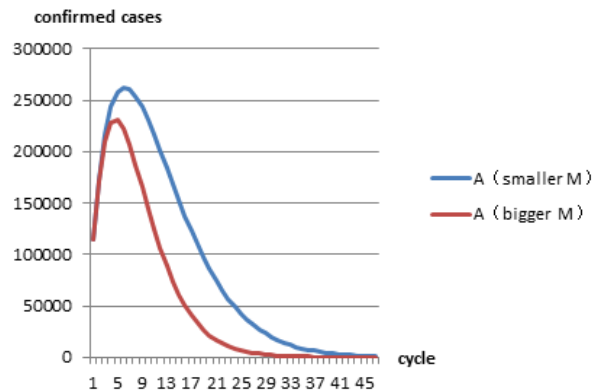


Figure 6. Results of the changing in  $M$

We can tell that a bigger value of  $M$  can lead to a better situation. So, relevant departments should accelerate manufacturing speed and expand production to optimize the eradication of Ebola.

#### 4. Conclusion

In this paper, we establish an improved SECADI model based on the classical SIR model and analyze the impact on the development of the epidemic under the consideration of factors such as the spread of the disease, the quantity of the medicine needed, possible feasible delivery systems, etc. In considering the factor of the disease spreading, we depict the inner-relationship among control strength of relevant departments, public awareness and the spread of the disease qualitatively and quantitatively in combination with reality and establish a basic differential equation model. Then we append the demand for medication and delivery locations and systems into our model, employ the analytic hierarchy process and introduce a series of aggregate evaluation index. We find that these three factors affect epidemic indirectly by influencing the supply of medication first. Additionally, speed of manufacturing of the medicine makes a big difference to the epidemic. We describe its changing rule through the Logistic growth model and analyze its relationship with the distribution time of the medicine which affects the spreading of epidemic. Eventually we establish an improved SECADI model in the post-controlled model.

We also establish a natural propagation model of Ebola in the pre-controlled stage via simplifying the SECADI model, compare the simulation results with the actual historical data and verify the accuracy of our model. Furthermore, we compare the forecast result of the natural propagation model with the results of the SECADI model in the post-controlled stage to verify the effectiveness of the SECADI model. By analyzing the sensitivity of various factors in our model, we draw conclusion that the greater the control strength of relevant departments is and the stronger the public awareness is, the better effect of controlling epidemic we can make.

#### References

- Aschwanden, C. (2004). Spatial simulation model for infectious viral diseases with focus on SARS and the common flu. *Proceedings of the 37th Annual Hawaii International Conference on System Sciences*. <http://dx.doi.org/10.1109/HICSS.2004.1265357>
- He, Y. H., & Tang, S. Y. (2013). Identification and parameter estimation of classical SIR model. *Applied Mathematics and Mechanics*, 34(3), 252-258.
- Huang, Q., & Min, L. Q. (2014). SIRS Epidemic model with immune. *Computer Simulation*, 3, 274-278.
- Jiang, H., Pan, H. X., & Sun, M. W. (2014). Simulating the Ebola virus disease transmission and outbreak in China by using computational epidemiological model. *Chinese Journal of Emergency Medicine*, 5.
- Li, G. Z., & Shi, D. H. (2006). Analyze of SIRS disease Epidemic model on complex network. *Progress in Natural Science*, 4, 508-512.
- Li, J. Q., & Ma, Z. (2006). Analyze of two kinds of SEIS Epidemic model with certain latent period. *Journal of Systems Science and Mathematical Sciences*, 2, 228-236.
- Xia, C. Y., Liu, Z. X., & Chen, Z. Q. (2008). Research of SIRS Epidemic model with immune on complex network. *Journal of Control and Decision*, 4, 468-472.
- Xiao, H., Tian, H. Y., & Lin, X. L. et al. (2013). Influence of extreme weather and meteorological anomalies on



- outbreaks of influenza A (H1N1). *Chin Sci Bull*, 58, 741-749. <http://dx.doi.org/10.1007/s11434-012-5571-7>
- Yoshihiro, M., & Hideo, H. (2013). Infectious Disease Spread Analysis Using Stochastic Differential Equations for SIR Model. *4th International Conference on Intelligent Systems, Modeling and Simulation* (pp. 152-156). <http://dx.doi.org/10.1109/ISMS.2013.13>
- Zhou, Y. W., & Li, Z. Q. (2011). The Application of Markov Skeleton Process on the Infectious Disease Management Model. *2nd International Conference on Artificial Intelligence, Management Science and Electronic Commerce* (pp. 2477-2480). <http://dx.doi.org/10.1109/AIMSEC.2011.6010975>

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