

# SIR Model for Ebola Outbreak in Liberia

Roshan Kumar <sup>#1</sup>, Smita Dey <sup>\*2</sup>

<sup>#,\*</sup> University Department of Mathematic, Ranchi University  
Ranchi, India-834008

**Abstract** — *Ebola is the dangerous virus to warrant global fears over a pandemic which infects large numbers of people in West Africa. In order to have a better understanding of the dynamics of its spreading in Liberia 2014, we described an epidemiological (Susceptible- Infected- Recovered) SIR model in which we simulated and compared it with the actual data from world health organization.*

**Keywords** — *SIR model, Ebola virus disease, Reproduction number.*

## I. INTRODUCTION

The severe Ebola virus disease (EVD), also known as haemorrhagic fever. The disease has a high risk of death; killing between 25 and 90 percent of those infected with an average of about 50 percent, and has caused immense sorrow especially for the African people [1]. Ebola spreads through human-to-human transmission via direct contact with the blood, secretions, organs or other body fluids of infected people. The incubation period is 2 to 21 days [2]. And what should be taken into concern is that humans are not infectious until they develop symptoms [3]. Ebola is caused by infection with a virus of the family Filoviridae, genus Ebolavirus [4]. The virus spreads through direct contact with infected blood or body fluids (i.e., urine, saliva, feces, vomit, and semen), contaminated objects (e.g., needles) and infected animals [5]. According to WHO, men who have recovered from the disease can still transmit the virus through their semen for up to 7 weeks after recovery from illness.

The change in travel patterns may also have contributed to the rapid spread of the disease [6]. This outbreak was declared as a Public Health Emergency of International Concern in August 2014 [7]. The first case of Ebola in Liberia occurred in Lofa County in March 2014. The virus spread to the capital, Monrovia, by the end of May and to 10 of 15 countries by August 2014 [8]. During July–December 2014, several Ebola outbreaks were detected in remote rural areas of Liberia, largely initiated by patients traveling from Monrovia [9].

Mathematical modelling has emerged as an important tool for understanding the dynamics of the spreading infectious diseases. Using mathematical modelling for further understanding and predicting infectious diseases has been an established practice for many years and often informs public health practices globally [10]. Incorporating mathematical modelling into epidemiology can aid in forming hypothesis on

disease spread mechanisms, refining data-collecting strategies, and clarifying optimal sample sizes [11]. With even the simplest models, such as the classical susceptible-infected-recovered (SIR) model, the choice of data to which the model is fit can have significant implications for science and policy. In order to model this behaviour, we will use Kermack & McKendrick's classical SIR model which use Ordinary Differential Equations (ODEs) as an appropriate modelling formalism [12]. As of August 29, 2014, the WHO reported cases of 694 EVD deaths, with most cases occurring in Liberia. [13].

The purpose of our study is to determine SIR model in which we will compare and simulate it with actual data from the WHO for the period of August 29 , 2014 to October 28 , 2014 in Liberia at reproductive ratio ( $R_0$ ) = 1.60.

## II. STUDY SITE AND AVAILABLE DATA

Our study site is Liberia (6°25'41.00''N, 9°25'46.20''W) which is situated in West Africa, bordering the North Atlantic Ocean to the country's southwest (Fig 1). The climate of Liberia is tropical and humid. For research purpose, total reported case data have been taken from the WHO for the duration of August 29, 2014 to October 28, 2014.



Fig. 1 Location of study site

## III. KERMACK & MCKENDRICK'S CLASSICAL SIR MODEL

In order to understand the behaviour of infected by EVD, we will use Kermack & McKendrick's classical SIR model which use Ordinary Differential Equations (ODEs) as an appropriate modelling formalism in

which interaction happens in below three variables in a short period of time:

$S$ = number of people that are susceptible to EVD

$I$ = number of people infected with EVD

$R$ = number of people recovered from EVD

Kermack & McKendrick (1927) imagined a fixed population  $N(t)=S(t)+I(t)+R(t)$  and derived three equations that can be used to simulate the deterministic spread of disease through this fixed population (Eq. 1-3).

$$\frac{dS}{dt} = -\frac{\beta SI}{N} \quad (1)$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I \quad (2)$$

$$\frac{dR}{dt} = \gamma I \quad (3)$$

Where  $\beta$  is the rate of infection and  $\gamma$  is the rate of recovery. The initial values of the SIR model must satisfy the following conditions:

$$S(0) = S_0 > 0,$$

$$I(0) = I_0 > 0,$$

$$R(0) = 0,$$

This form of the model assumes a closed population, such that these three categories together make up the entire population and there are no births, deaths (from any cause other than EVD), immigrations and emigrations. It is possible to include these factors in a model, but for a disease like EVD which spreads quickly over a short time frame; these assumptions have little effect on the spread of infection beyond the initial infection in a community.

The Fig 2 shows the compartment and transition of the SIR model.

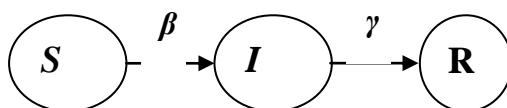


Fig. 2 SIR compartmental diagram

#### IV. SIMULATION OF KERMACK & MCKENDRICK'S CLASSICAL SIR MODEL

SIR compartment modelling is a powerful tool. The key to effective modelling is maximizing simplicity, while taking into account enough realism so that the findings still stand in the real context of interest. So in order to examine the behaviour of EVD in Liberia we have taken  $\beta=0.80$ ,  $\gamma=0.50$ ,  $S(0)=0.80$ ,  $I(0)=0.20$  and  $R(0)=0$ . From MATLAB code on (Eq.1-3) SIR model created (Fig 3).

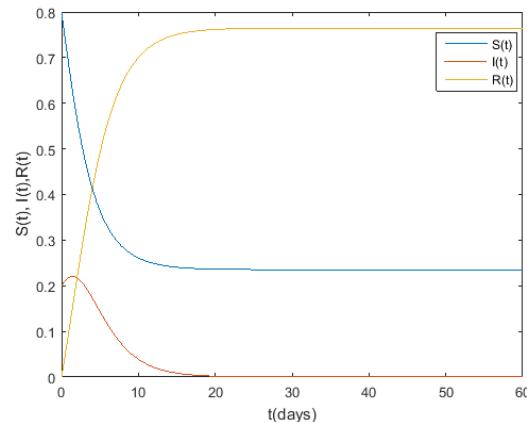


Fig. 3 Relationship between the number of  $S(t)$ ,  $I(t)$  and  $R(t)$  for an EVD governed by an SIR model

In Liberia the EVD Basic reproduction number ( $R_0$ ) varies from 1.59 to 1.60 [14]. And in our model reproduction number ( $R_0$ ) is 1.60.

The 'S' decreases till 12 days because of introduction EVD in the system. After 12 days 'S' became constant and due to no vaccination it never reaches zero. 'I' increases till 2 days after that it decreases because of 'R'. But 'R' increases till 14 days then it became constant. 'R' increases concurrently as 'S' decreases because 'S' and 'R' are inversely proportional to each other.

The graph also signifies that 'N' is always constant for two months via a linear correlation (Eq. 1-3).

$$(S + I + R)' = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \left(-\frac{\beta SI}{N}\right) + \left(\frac{\beta SI}{N} - \gamma I\right) + (\gamma I) = 0$$

#### V. COMPARISON OF KERMACK & MCKENDRICK'S CLASSICAL SIR MODEL WITH ACTUAL DATA

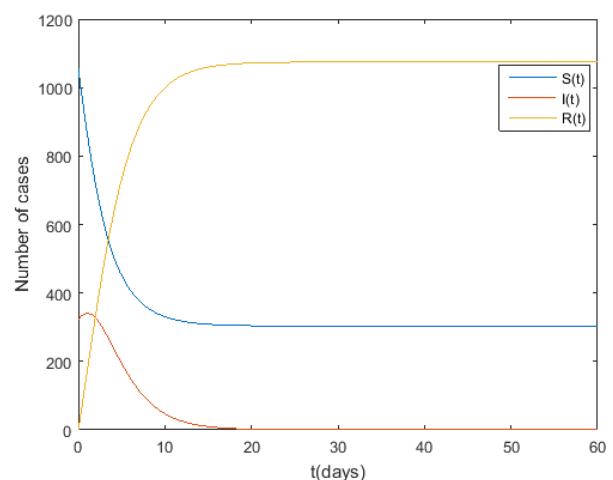


Fig. 4 Relationship between the number of  $S(t)$ ,  $I(t)$  and  $R(t)$  for an EVD governed by an SIR model through actual initial data

So in order to examine the actual behaviour of EVD in Liberia we have taken  $\beta=0.000580$ ,  $\gamma=0.50$ ,  $S(0)=1056$ ,  $I(0)=322$ ,  $R(0)=0$  with reproduction number( $R_0$ ) is 1.60. From MATLAB code on (Eq. 1-3) SIR model created (Fig 4).

The 'S' decreases till 12 days because of introduction of EVD in the system. After 12 days 'S' became constant and due to no vaccination it never reaches zero. 'I' increases till 2 days after that it decreases because of 'R'. But 'R' increases till 14 days then it became constant. 'R' increases concurrently as 'S' decreases because 'S' and 'R' are inversely proportional to each other.

The graph also signifies that 'N' is always constant for two months via a linear correlation (Eq. 1-3).

$$(S + I + R)' = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \left(-\frac{\beta SI}{N}\right) + \left(\frac{\beta SI}{N} - \gamma I\right) + (\gamma I) = 0$$

## VI. CONCLUSION

In this study, Kermack & McKendrick's classical SIR epidemiological model has emerged as an important tool for understanding the dynamics of the spreading of Ebola virus disease. It's noted that the population is constant in the SIR model by adding together the number of people in all compartments and taking the derivative with respect to time, indicating that the population does not increase or decrease in this model. Because of no vaccination the susceptible never reaches zero (here, we have discussed only SIR model). For future work, we have left the extension of SIR model). On taking 80% population as susceptible and 20% as infected then model fits the actual data. The prepared model is very realistic to the Ebola virus disease in Liberia with reproduction number ( $R_0$ ) 1.60 that indicates the epidemic nature of Ebola virus disease.

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## REFERENCES

- [1] Birmingham, K and Cooney, S. Ebola: small, but real progress (news feature). *Nature Med* 2002;8:313.
- [2] Heinz Feldmann, Thomas W Geisbert. Ebola haemorrhagic fever. *The Lancet* 2011 (9768).
- [3] F. Chiappelli and A. Bakhordarian: Ebola translational science considerations. *Journal of Translational Medicine*. January 2015.
- [4] T. Leslie. "Ebola: what is it and how does it spread?" ABC News, <http://www.abc.net.au/news/2014-07-30/ebola-virusexplainer/5635028>, 2014.
- [5] World Health Organization (WHO), "Ebola virus disease", <http://www.who.int/mediacentre/factsheets/fs103/en/>, 2014.
- [6] Marta C. Gonzalez Ruben Juanes Christos Nicolaides, Luis Cueto- Felgueroso, A metric of influential spreading during contagion dynamics through the air transportation network, *PLoS ONE* 7 (2012), 1-10.
- [7] Pandey, A., Atkins, K. E., Medlock, J., Wenzel, N., Townsend, J. P., Childs, J. E., ...vani, A. P. (2014). Strategies for containing Ebola in West Africa. *Science (New York, N.Y.)*, 346(6212), 991–5. doi:10.1126/science.1260612.
- [8] Arwady MA, Bawo L, Hunter JC, Massaquoi M, Matanock A, Dahn B, et al. Evolution of Ebola virus disease from exotic infection to global health priority, Liberia, mid-2014. *Emerg Infect Dis*. 2015;21:578–84. <http://dx.doi.org/10.3201/eid2104.141940>.
- [9] Kateh F, Nagbe T, Kieta A, Barskey A, Gasasira AN, Driscoll A, et al. Rapid response to Ebola outbreaks in remote areas—Liberia, July–November 2014. *MMWR Morb Mortal Wkly Rep*. 2015;64:188–92.
- [10] Glasser, J., Meltzer, M., & Levin, B. (2004). Mathematical modeling and public policy: responding to health crises. *Emerging Infectious Diseases*, 10(11), 2050–1. doi:10.3201/eid1011.040797\_08.
- [11] Grassly, N. C., & Fraser, C. (2008). Mathematical models of infectious disease transmission. *Nature Reviews Microbiology*, 6(6), 477–87. doi:10.1038/nrmicro1845.
- [12] W. Kermack and A. McKendrick. Contribution to the Mathematical Theory of Epidemics. *Proceedings of the Royal Society of London. Series A*, 115(772):700–721, 1927.
- [13] <http://apps.who.int/ebola/ebola-situation-reports>
- [14] Althaus, Christian L. (2014). "Estimating the Reproduction Number of Ebola Virus (EBOV) During the 2014 Outbreak in West Africa".