



Stability Analysis for Diarrheal Epidemic Models using Routh Hurwitz Method

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Abstract:

Epidemic modelling of infectious diseases improves the understanding of the dynamics displayed by disease outbreaks. Modelling of diarrheal epidemics has been presented in this research work. Since the disease is caused by several pathogens, three major pathogens have been selected as the subjects of discussion in this presentation. Based on their respective pathogenesis, continuous mathematical models have been built for each pathogen. The epidemic theory has been used to determine the presence/absence of an epidemic based on the disease free state and basic reproduction number which have been defined and calculated for all the models. Stability analysis of the disease free-state has been computed using the Routh Hurwitz method. The system presented herewith is aimed towards use in Botswana and has been proved for feasibility.

Keywords: Epidemic modelling, diarrhea, mathematical modelling, stability, Routh Hurwitz

I. INTRODUCTION

Ranging from drinking, bathing, agricultural uses, cooking and cleaning, water widely comes in contact with every individual and thus serves a significant position in transmitting many types of water-related diseases. Diarrhea is one such disease that is very common in the current era. Generally overlooked in terms of treatment and quarantining, this disease produces an alarming global burden of 1.7 billion cases every year, as per World Health Organisation (WHO) records. With approximately 700000 child deaths per year [1], diarrhea is the second leading cause of child mortality in world (pneumonia being the first) [2].

According to the statistics obtained from the International Disease Surveillance and Reporting Centre of Botswana, there were about 17000 cases of diarrhoea and 150 deaths due to diarrhoea reported in 2014 [3]. This shows that necessary prevention and precaution methods need to be employed so as to avoid health hazards in Botswana. These high statistical values are continuously being produced despite numerous efforts of treatments across the globe. A recent study in Botswana confirms the persistence of the disease regardless of providing treatment methods such as Oral Rehydration Salt (ORS) therapies [4].

Apart from high mortality rates, it is worth noting that diseases such as diarrhea consequence in enormous financial loads under the veil of easy treatment. Considering such cases, it is more useful if the disease is prevented rather than giving the treatment. Diarrheal diseases are caused by several pathogens including Escherichia Coli, Rotavirus, norovirus, shigella, campylobacter, cryptosporidium and many more. This work focusses on the causes of diarrhea in Botswana. On a national level, Rotavirus is one of the topmost viruses that causes diarrhea [5]. Bacterial infections follow next. In this context salmonella and Shigella are the most hazardous pathogens in Botswana [6]. This research summarizes the dynamics of these

pathogens into continuous mathematical models which are simulated to ensure fidelity using assumed parameters. Using these models and their analysis, a system is envisaged to be built that will contribute in preventive measures for the disease. Upon completion and confirmation of all analyses, the models presented are to be further scrutinized, simulated and elaborated to be integrated onto a user interface platform that will be easy to be used by any public health official /technician. Simple and straightforward selection of information on the interface will produce direct and understandable output regarding the epidemic situation in a selected area. An alert system is thus envisioned to be produced using these models. It is for these reasons that basic aspects of the environment, population and disease have been considered in the modelling process.

II. BACKGROUND

Epidemic Models

Epidemic models have been widely explored and exploited in the context of infectious diseases beginning with Karmack and Mc Kendrick in 1927, who introduced the initial compartmental epidemic model [7]. This model had three compartments namely the susceptible, infected and recovered and thus got the name SIR model. Each compartment in this model is described using a differential equation. In its simplest form, the model can be shown as follows:

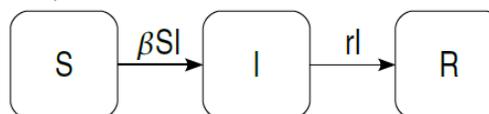


Figure.1. SIR Model

Ordinary differential equations together with some defined parameters are used to illustrate the model above as shown below. Two pre-defined parameters have been used in this model namely β (infectivity rate) and r (recovery rate).

$$dS/dt = -\beta SI \quad (1)$$

$$dI/dt = \beta SI - rI \quad (2)$$

$$dR/dt=rI \quad (3)$$

With the help of these equations, it is not only possible to monitor the changes in the population in each class but one can also scrutinize the movement of the population between the classes. The above epidemic model has been used as a basis for the many following models that have been built for infectious diseases. Infectious diseases have been abundantly described using epidemic models by research centres across the globe. Diseases like hepatitis, HIV infections, Influenza and malaria have been modelled giving significant end results [8,9,10].

Disease Epidemiology and Pathogenesis

Diarrhea is a condition that involves an increment in the frequency of bowel movements and fluidity of stool. Apart from this major symptom, diarrhea is associated with symptoms such as abdominal pain, fever and most important of all dehydration [11]. Dehydration results in the loss of many necessary salts and chemicals along with water. This condition of loss can become dreadfully harsh resulting in serious circumstances ranging from heavy malnutrition to even death. Rotavirus is one of the leading pathogens worldwide related to diarrhea and results in approximately 527000 deaths annually. Rotavirus involves an incubation period of about two to three days. During this time, the person remains asymptomatic but due to its high contagiousness, rotavirus is spread during this period too. An infected person remains highly contagious up to ten days after the onset of symptoms and may continue to shed the virus at slower rates after this period [12]. The main treatment given to the infected people with rotavirus is rehydration. Out of many other bacteria, salmonella is one of the most common causes of diarrhea in Botswana [13]. Salmonella infections can cause typhoid or can be non-typhoidal. It is the non-typhoidal serotypes of salmonella that cause diarrhea. The global burden of non-typhoidal salmonellosis is estimated to be approximately 93 million cases annually [14]. Symptoms begin to appear after about 24 hours following ingestion of contaminated food or water and may manifest in different forms like gastroenteritis, bacteremia and enteric fever. Out of these, gastroenteritis is associated with diarrhea which may last for about a week. In general, an oral rehydration therapy is carried out for treatment of gastroenteritis to replace the lost fluids and electrolytes. Antibiotics may be used in severe cases or for immune suppressed individuals. Symptoms fade subside after a few days but the patient may remain contagious for even months [15, 16]. In relation to diarrhea, another bacteria causing significant number of cases in Botswana, is shigella. The World Health Organisation records that there are approximately 100000 deaths and 90 million cases of shigellosis per year around the world [17]. Symptoms lasts for about a week and the individual remains slightly contagious for a period of four weeks [18]. Shigellosis usually fades away in a few days. If there is persistence, antibiotic treatment can be opted for [19].

III. METHODOLOGY

Following the deterministic approach for modelling, the first step will be to divide the population into compartments based on the characteristics and pathogenesis of the respective pathogen being considered. Since each of the pathogens has different infection methods and symptom durations, three models will be built separately for each pathogen. As Explained in sections above, this modelling involves the use of Ordinary Differential Equations (ODEs) to define the whole

system. Different models have been formulated for each pathogen depending on their respective characteristics. The Disease-Free Equilibrium (DFE, denoted as E_0 in the calculations) is defined as the point when the disease is absent from the whole population. Infections are hampered and the pathogen is almost extinct at this point. Since there is no disease, the infectious classes of the models have zero population and following this, the recovered class also reaches a zero population. At the DFE, the only active class for all the models described herewith is the susceptible class. For the calculation of the DFE, the left hand side of the equation describing the active compartment is equated to zero. And the value of the population of the active class is then made the subject of the formula. The basic reproduction number (generally denoted as R_0) is defined as the number of secondary cases being caused by a single infection. According to theory, if this number is less than one (i.e. $R_0 < 1$), then there will be no epidemic in the population. On the other hand, if the basic reproduction number has a value greater than one (i.e. $R_0 > 1$), it shows that the disease is being transmitted through the population and an epidemic may break out. Thus, this value has a crucial role as it is used to establish the existence of the epidemic. The Routh Hurwitz stability test examines the system by ensuring that all the roots of a given polynomial are in the left half plane of the axis i.e. negative values (real or imaginary). For any physical system, the variations within are described using equations. In epidemic modelling, ODEs are used to describe the motion of the population in the prescribed compartments. Solutions of these equations are stable and this can be confirmed using the Routh Hurwitz method. **Theorem 1** If the roots have negative values, this will indicate a stable system.

IV. MODEL DEVELOPMENT

Assumptions

For all the modelling processes, a set of assumptions has been used. These are as follows:

1. All individuals are born susceptible to the disease
2. Disease induced mortality is negligible under the temporal scale consideration therefore no individual dies due to infection
3. The infection described in the models combines both direct and indirect infection
4. The population is well mixed i.e. the infected people are homogeneously spread throughout the entire population
5. All individuals are equally susceptible
6. No co-infection cases are involved
7. No cases of multiple pathogen infection are considered
8. No other cases of prevention (like vaccination) are considered herewith because the vaccines are not yet significantly prominent in Botswana as per information from the National Health Laboratory, Gaborone.

Rotavirus – Model and Simulation: Incorporating the pathogenesis of rotavirus diarrhea, the following flow diagram shows the epidemic model for Rotavirus:

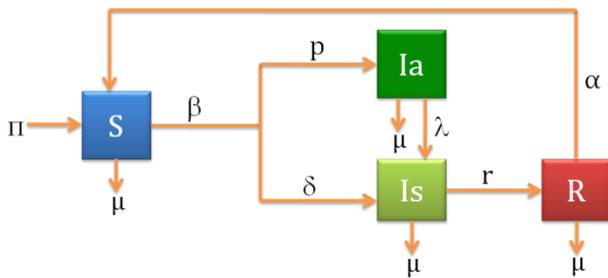


Figure.2. Rotavirus Model

The following table describes the variables that have been used and their values which are used for simulation purposes:

Table.1. Parameters for the Rotavirus Model

Parameter	Description
β	Infectivity rate
α	Immunity loss rate
μ	Natural death rate
$p(=1-\delta)$	Probability of asymptomatic infection
r	Recovery rate
δ	Probability of symptomatic infection
Π	Population renewal
λ	Symptom gaining rate

In a group of susceptible people, a certain proportion can be infected to either become symptomatic or asymptomatic infectious. Both these classes contribute to the infection at different rates. Infectivity rate depicts the rate at which the pathogen is likely to attack an individual. After infection, the individual becomes either symptomatic infected or asymptomatic infected depending on the level of virus outbreak in the body. These two scenarios are governed by the parameters p and δ . After a certain period of time, the asymptomatic individuals may become symptomatic if the virus dose increases in the body. With the help of medication and rehydration therapies or body immunity, the infectious people become recovered. For some time, the body maintains an immunity level but gradually, this level drops and the recovered population again becomes susceptible to the disease. Using the above mentioned parameters and assumptions, the extended compartmental model can be defined using the following equations:

$$\frac{dS}{dt} = \Pi - \mu S - \beta S(pI_A + \delta I_S) + \alpha R \quad (4)$$

$$\frac{dI_A}{dt} = \beta p S I_A - \mu I_A - \lambda I_A \quad (5)$$

$$\frac{dI_S}{dt} = \beta \delta S I_S - \mu I_S - r I_S + \lambda I_A \quad (6)$$

$$\frac{dR}{dt} = r I_S - \mu R - \alpha R \quad (7)$$

At the DFE,

$$\begin{aligned} \Pi - \mu S^* &= 0 \\ S^* &= \frac{\Pi}{\mu} \end{aligned}$$

Thus, the DFE can be described as follows:

$$DFE = \left(\frac{\Pi}{\mu}, 0, 0, 0 \right) \quad (8)$$

Using the next generation matrix, the basic reproduction number of the model is calculated as:

$$R_0 = \frac{\beta p \Pi}{\mu(\mu + \lambda)} \quad (9)$$

To work out the stability analysis of the rotavirus system, firstly we consider the set of equations that describe the model. For this step we assume that the immunity loss rate, α is approximately 0, for calculation purposes.

$$f1 = \Pi - \mu S - \beta S(pI_A + \delta I_S)$$

$$f2 = \beta \delta S I_S - \mu I_S - r I_S + \lambda I_A$$

$$f3 = \beta p S I_A - \mu I_A - \lambda I_A$$

$$f4 = r I_S - \mu R$$

The Jacobian matrix of the system above is evaluated as:

$$J = \begin{bmatrix} -\mu - \beta(pI_A + \delta I_S) & -\beta \delta S & -\beta p S & 0 \\ \beta \delta I_S & \beta S - \mu - r & \lambda & 0 \\ \beta p I_A & 0 & \beta S - \mu - \lambda & 0 \\ 0 & r & 0 & -\mu \end{bmatrix} \quad (10)$$

To work out the stability of the DFE, we substitute the above Jacobian matrix with the DFE expression for rotavirus. Doing this we get,

$$E_0 = \left(\frac{\Pi}{\mu}, 0, 0, 0 \right)$$

$$J(E_0) = \begin{bmatrix} -\mu & \frac{-\beta \Pi \delta}{\mu} & \frac{-\beta \Pi p}{\mu} & 0 \\ 0 & -\mu - r & \frac{\beta \Pi}{\mu} & 0 \\ 0 & 0 & \frac{\beta \Pi}{\mu} - \mu - k & 0 \\ 0 & r & 0 & -\mu \end{bmatrix}$$

Subtracting the index matrix multiplied by the operating parameter (denoted as γ), we get:

$$J(E_0) - \gamma I = \begin{bmatrix} -\mu - \gamma & \frac{-\beta \Pi \delta}{\mu} & \frac{-\beta \Pi p}{\mu} & 0 \\ 0 & \frac{\beta \Pi}{\mu} - \mu - r - \gamma & \frac{\beta \Pi}{\mu} & 0 \\ 0 & 0 & \frac{\beta \Pi}{\mu} - \mu - k - \gamma & 0 \\ 0 & r & 0 & -\mu - \gamma \end{bmatrix}$$

We use this matrix to derive the characteristic equation which is defined as:

$$\begin{aligned} E_\gamma &= (-\mu - \gamma) \times (-\mu - \gamma) \times \left(\frac{\beta \Pi}{\mu} - \mu - r - \gamma \right) \\ &\quad \times \left(\frac{\beta \Pi}{\mu} - \mu - k - \gamma \right) \quad (11) \end{aligned}$$

The roots of this equation are:

$$\gamma_1 = -\mu$$

$$\gamma_2 = -\mu$$

$$\gamma_3 = \frac{\beta \Pi}{\mu} - \mu - r = R_0 \left(\frac{\mu + k}{\mu} \right) - \mu - r$$

$$\gamma_4 = \frac{\beta \Pi}{\mu} - \mu - k = R_0 \left(\frac{\mu + k}{\mu} \right) - \mu - k$$

$$= \left(\frac{R_0}{\mu} - 1 \right) (\mu + k)$$

Using theorem 1, the results deduced are describe as:

The DFE of the rotavirus system is locally asymptotically stable when $R_0 < 1$ and is instable if $R_0 > 1$.

Salmonella – Model and Simulation

The model for salmonella will also have four compartments according to the pathogenesis [29].

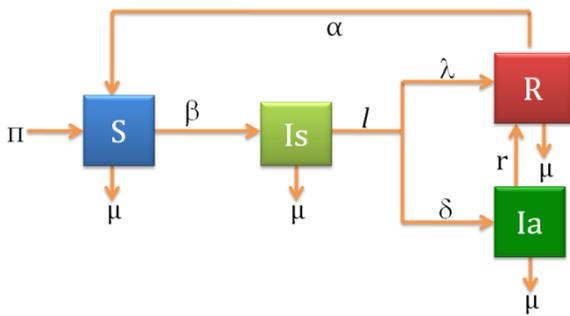


Figure.3. Salmonella Model

The table below describes the parameters and their values that have been used in the above model.

Table.2. Salmonella Parameter Description

Parameter	Description
Π	Population renewal rate
β	Infectivity rate
μ	Natural death rate
l	Rate of losing symptoms
$\lambda(=1-\delta)$	Direct recovery rate
δ	Rate of pathogen prevalence in body
r	Indirect recovery rate
α	Immunity loss rate

The susceptible population, as the name suggests, is non-resistant to the disease and gets in contact with the pathogen through the infected population. Both the infected classes contribute to the pathogen population and hence transmit the disease. Resolution of the symptoms could mean that one has become a bacteria carrier or he has recovered. Therefore, once a symptomatic infected individual reduces the pathogen concentration in the body at a rate l , he could either flush out the total pathogen amount at a direct recovery rate λ , or retain a certain pathogen amount in the body at the rate δ . The asymptomatic carrier also recovers progressively as the pathogen clears from his body. For some time, the body maintains an immunity level and the individual remains in the recovered class but gradually, this immunity level can drop rendering the recovered population to become susceptible to the disease again. Using the above mentioned parameters and assumptions, the extended compartmental model can be defined using the following equations:

$$\frac{dS}{dt} = \Pi - \mu S - \beta S I_S (1 + \delta) + \alpha R \quad (12)$$

$$\frac{dI_S}{dt} = \beta S I_S (1 + \delta) - \mu I_S - l I_S \quad (13)$$

$$\frac{dI_A}{dt} = l I_S - \mu I_A - r I_A \quad (14)$$

$$\frac{dR}{dt} = \lambda I_S + r I_A - \mu R - \alpha R \quad (15)$$

Thus, the DFE can be described as follows:

$$DFE = \left(\frac{\Pi}{\mu}, 0, 0, 0 \right) \quad (16)$$

The basic reproduction number is calculated as:

$$R_0 = \frac{\beta \Pi (1 + \delta)}{\mu (\mu + l)} \quad (17)$$

Considering the equations of the model:

$$f1 = \Pi - \mu S - \beta S I_S (1 + \delta)$$

$$f2 = \beta S I_S (1 + \delta) - \mu I_S - l I_S$$

$$f3 = l \delta I_S - \mu I_A - r I_A$$

$$f4 = \lambda I_S + r I_A - \mu R$$

The Jacobian matrix of the above system is evaluated as:

$$J = \begin{bmatrix} -\mu - \beta I_S (1 + \delta) & -\beta S (1 + \delta) & 0 & 0 \\ \beta I_S (1 + \delta) & \beta S (1 + \delta) - \mu - l & 0 & 0 \\ 0 & l \delta & -\mu - r & 0 \\ 0 & l (1 - \delta) & r & -\mu \end{bmatrix} \quad (18)$$

At the DFE of Salmonella, the Jacobian matrix becomes:

$$J(E_0) = \begin{pmatrix} \frac{\Pi}{\mu}, 0, 0, 0 \end{pmatrix}$$

$$J(E_0) = \begin{bmatrix} -\mu & \frac{-\beta \Pi}{\mu} (1 + \delta) & 0 & 0 \\ 0 & \frac{-\beta \Pi}{\mu} (1 + \delta) - \mu - l & 0 & 0 \\ 0 & l \delta & -\mu - r & 0 \\ 0 & l (1 - \delta) & r & -\mu \end{bmatrix}$$

$$J(E_0) - \gamma I = \begin{bmatrix} -\mu - \gamma & \frac{-\beta \Pi}{\mu} (1 + \delta) & 0 & 0 \\ 0 & \frac{-\beta \Pi}{\mu} (1 + \delta) - \mu - l - \gamma & 0 & 0 \\ 0 & l \delta & -\mu - r - \gamma & 0 \\ 0 & l (1 - \delta) & r & -\mu - \gamma \end{bmatrix}$$

The characteristic equation of the above matrix is expressed as:

$$E_\gamma = (-\mu - \gamma) \times (-\mu - \gamma) \times (-\mu - r - \gamma) \times \left(\frac{\beta \Pi}{\mu} (1 + \delta) - \mu - l - \gamma \right) \quad (19)$$

The roots of this equation are:

$$\gamma_1 = -\mu$$

$$\gamma_2 = -\mu$$

$$\gamma_3 = -\mu - r$$

$$\gamma_4 = \frac{\beta \Pi}{\mu} (1 + \delta) - \mu - l = R_0 \left(\frac{\mu + l}{1 + \delta} \right) - \mu - l$$

$$= \left(\frac{R_0}{1 + \delta} - 1 \right) (\mu + l)$$

Using theorem 1, it is confirmed that the DFE of the salmonella system is stable as long as $R_0 < 1$. If the value of R_0 goes above 1, the system becomes unstable.

Shigella – Model and Simulation

The model for shigella will consist of only three compartments and it is shown in the figure below:

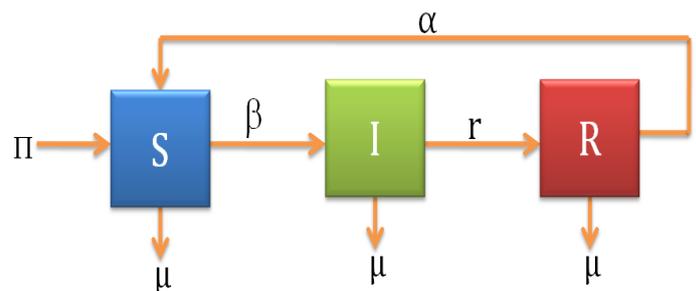


Figure. 4. Shigella Model

Parameters that are used for defining the model above are described in table 5 below.

Table.3. Shigella Model Parameter Description

Parameter	Description
Π	Population renewal rate
β	Infectivity rate
μ	Natural death rate
r	Recovery rate
α	Immunity loss rate

The susceptible population, as the name suggests, is non-resistant to the disease and gets in contact with the pathogen through the infected population. The infected class contributes to the pathogen population in the environment and hence transmits the disease. With the help of medication and rehydration therapies or body immunity, the infectious people become recovered. For some time, the body maintains an immunity level but gradually, this level drops and the recovered population again becomes susceptible to the disease. Using the above mentioned parameters and assumptions, the extended compartmental model can be defined using the following equations:

$$\frac{dS}{dt} = \Pi - \mu S - \beta SI + \alpha R \quad (20)$$

$$\frac{dI}{dt} = \beta SI - \mu I - rI \quad (21)$$

$$\frac{dR}{dt} = rI - \mu R - \alpha R \quad (22)$$

Thus, the DFE can be described using equation 8 as follows:

$$DFE = \left(\frac{\Pi}{\mu}, 0, 0 \right) \quad (23)$$

The basic reproduction number of this model is worked out as:

$$R_0 = \frac{\beta \Pi}{\mu(\mu + r)} \quad (24)$$

To formulate the Jacobian matrix for the Shigella model, the set of ODEs are considered:

$$\begin{aligned} f1 &= \Pi - \mu S - \beta SI \\ f2 &= \beta SI - \mu I - rI \\ f3 &= rI - \mu R \end{aligned}$$

The Jacobian matrix of the above system is evaluated as:

$$J = \begin{bmatrix} -\mu - \beta I & -\beta S & 0 \\ \beta I & \beta S - \mu - r & 0 \\ 0 & r & -\mu \end{bmatrix} \quad (25)$$

Substituting the DFE of Shigella into the matrix, we get:

$$\begin{aligned} E_0 &= \left(\frac{\Pi}{\mu}, 0, 0 \right) \\ J(E_0) &= \begin{bmatrix} -\mu & -\beta \frac{\Pi}{\mu} & 0 \\ 0 & \beta \frac{\Pi}{\mu} - \mu - r & 0 \\ 0 & r & -\mu \end{bmatrix} \\ J(E_0) - \gamma I &= \begin{bmatrix} -\mu - \gamma & -\beta \frac{\Pi}{\mu} & 0 \\ 0 & \beta \frac{\Pi}{\mu} - \mu - r - \gamma & 0 \\ 0 & r & -\mu - \gamma \end{bmatrix} \end{aligned}$$

The characteristic equation of the above matrix is expressed as:

$$E_\lambda = (-\mu - \gamma) \times (-\mu - \gamma) \times \left(\frac{\beta \Pi}{\mu} - \mu - r - \gamma \right) \quad (26)$$

The roots of this equation are:

$$\gamma_1 = -\mu$$

$$\gamma_2 = -\mu$$

$$\gamma_3 = \frac{\beta \Pi}{\mu} - \mu - r = R_0(\mu + r) - \mu - r = (R_0 - 1)(\mu + r)$$

Using theorem 1, it can be stated that the DFE of the Shigella system is stable as long as $R_0 < 1$ and the system becomes unstable if the value of R_0 goes above 1.

V. CONCLUSION

This paper presented continuous models on diarrhea caused by rotavirus, salmonella and shigella thus provided an increased understanding of the dynamics of the disease in a country like Botswana. Analysis of the system was done by evaluating the disease Free State and basic reproduction number for all the models. The disease Free states for each model were tested for stability using the Routh Hurwitz method. It was proved that as long as the value of R_0 is kept minimal, the disease can be eradicated from the population. The model shows that the higher the value of R_0 , the more likely an epidemic will spread at higher rates. R_0 can be kept low by employing various policies such as increasing knowledge of public in terms of prevention and treatment, increased hygiene conditions at work places and better water treatment facilities. By combining the study of three major diarrheas causing agents, this study presents a unique combination of three models that have been tested for stability and feasibility by simulation.

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