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*Research article*

## Asymptomatic transmission shifts epidemic dynamics

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**Abstract:** Asymptomatic transmission of infectious diseases has been recognized recently in several epidemics or pandemics. There is a great need to incorporate asymptomatic transmissions into traditional modeling of infectious diseases and to study how asymptomatic transmissions shift epidemic dynamics. In this work, we propose a compartmental model with asymptomatic transmissions for waterborne infectious diseases. We conduct a detailed analysis and numerical study with shigellosis data. Two parameters, the proportion  $p$  of asymptomatic infected individuals and the proportion  $k$  of asymptomatic infectious individuals who can asymptotically transmit diseases, play major roles in the epidemic dynamics. The basic reproduction number  $\mathcal{R}_0$  is a decreasing function of parameter  $p$  when parameter  $k$  is smaller than a critical value while  $\mathcal{R}_0$  is an increasing function of  $p$  when  $k$  is greater than the critical value.  $\mathcal{R}_0$  is an increasing function of  $k$  for any value of  $p$ . When  $\mathcal{R}_0$  passes through 1 as  $p$  or  $k$  varies, the dynamics of epidemics is shifted. If asymptomatic transmissions are not counted,  $\mathcal{R}_0$  will be underestimated while the final size may be overestimated or underestimated. Our study provides a theoretical example for investigating other asymptomatic transmissions and useful information for public health measurements in waterborne infectious diseases.

**Keywords:** Asymptomatic transmission, basic reproduction number, final size

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### 1. Introduction

Asymptomatic transmission refers to the transmission of a disease from an individual who does not develop symptoms. It has been recognized in many infectious diseases. Nelson et al. [1] pointed out there was an asymptomatic transmission in cholera epidemics. Lopez et al. [2] reported an asymptomatic transmission occurred in *Giardia lamblia* infections. Okpalanwa et al. [3] showed that there was an asymptomatic population of resistant salmonella in Nigeria. Most importantly, the current pandemic COVID-19 can be transmitted asymptotically. It is necessary now to incorporate asymp-

omatic transmissions into traditional models of infectious diseases. In this study, as proof of principle, we focus on modeling of waterborne infectious disease which has an asymptomatic transmission component.

Pathogens, viruses, parasites, and microorganisms with eutrophication in non-clean drinking water can cause the spread of infectious diseases. After drinking, it may cause diseases such as cholera, typhoid fever, amoebic dysentery, bacillus dysentery, and other diarrheal diseases, and it may cause dengue fever, lymphatic filariasis, malaria, onchocerciasis, yellow fever trachoma, and other waterborne diseases [4]. According to World Health Organization, in 2019, 785 million people still lack basic drinking water, of which 144 million people live on surface water. At least 2 billion people worldwide use drinking water sources that are contaminated with feces. Contaminated drinking water is estimated to cause more than 485,000 deaths [5].

The earliest mathematical model for studying infectious diseases is the SIR model proposed by Kermack and McKendrick [6]. This model has been widely applied in real life, and there have been a lot of related studies. Mathematical models have also been applied to study waterborne infectious diseases. An early models related to waterborne infectious diseases was a system of two ordinary differential equations proposed by Capasso and Fontana [7] in 1979, which considered the evolution of a human infected population in urban communities and marine bacterial population (cholera bacteria). Codeco [8] extended this model to a SIB model with a susceptible population to study the long-term dynamics. We conducted detailed analysis for several early models [9]. Tien and Earn [10] proposed a multi-channel transmission for infectious disease modeling. That is, the model has susceptible individuals, infectious individuals, bacteria in the water, and recovered individuals (SIWR), where the disease can be transmitted from infected individuals to susceptible individuals, and from bacteria in the water to susceptible individuals. In real situations, when knowing viruses in the water, some disinfection and sterilization will be taken. Misra and Singh [11] considered the effectiveness of the disinfection and sterilization and the time lag of disinfection to build the XYZBC (X: susceptible individuals, Y: infected individuals, Z: removed individuals, B: water cholera concentration, C: disinfectant concentration) model. Eisenberg et al. [12] established the SIRSW model for cholera spread, and calculated the basic reproduction number and the global stability of endemic diseases. Wang and Cao [13] established a network epidemic model for waterborne diseases which is a SIWR model.

The presence of asymptomatic individuals and incubation periods in many waterborne infectious diseases can increase the spread of infectious diseases. Okpalanwa et al. [3] demonstrated that the presence of multiple and extensively resistant salmonella in asymptomatic populations had a significant impact on the ecology and epidemiology of antimicrobial resistance in Nigeria. Lopez et al. [2] described that most, about 76%, of *Giardia lamblia* infections that occur during an epidemic are asymptomatic. In his thesis [14], Laveri conducted a detailed data study about traveler's diarrhea and found that the proportion of pathogen-positive stools was highest among asymptomatic travellers who had visited East Africa (80%), followed by Latin America (75%), West and Central Africa (68%), and South Asia (64%), and lowest in Southeast Asia (46%). In a study by Nelson [1] et al, they claimed that the number of asymptomatic cases of cholera reached nearly half. It should be noticed that there is certain incubation period for asymptomatic transmissions. Angelo et al. [15] stated that the incubation period of outbreak-associated listeriosis cases had a median of 11 days and 90% of cases occurred within 28 days. The incubation period varies depending on specific pathogens, viruses, parasites, or microorganisms. In 2014, Chen et al. [16] built a short-term SEIAWR model (also see [17]) based

on the characteristics of Shigella infection, and simulated the effectiveness of combining various measures (treatments, disinfection, isolation, and school suspension). Recently, Chen et al. [18] proposed a long-term SEIAWR model of COVID-19, they calculated the basic reproduction number to assess the transmissibility of COVID-19 according to the reported data in Wuhan city, China. However, it is important to characterize asymptomatic transmissions in these epidemic models.

In this work, we perform some detailed analysis to study the global dynamics of the long-term SEIAWR model. We find the basic reproduction number, prove global stability for the endemic equilibrium state, and show how the basic reproduction number changes as parameters related to asymptomatic transmission vary. To compare the dynamics with short-term SEIAWR model, we also perform analysis for the short-term model, and obtain the basic reproduction number and the final size. We also conduct numerical analysis to demonstrate the dynamic behaviors of both short and long term models, particularly, to show how an asymptomatic transmission shifts their dynamic behaviors.

The rest of the article is organized as follows. In Section 2, the long-term model SEIAWR is proposed and analyzed. In Section 3, the short-term SEIAWR model is analyzed. In Section 4, numerical studies are presented. The article is closed with discussion and conclusions in Section 5.

## 2. Long-term SEIAWR model and its analysis

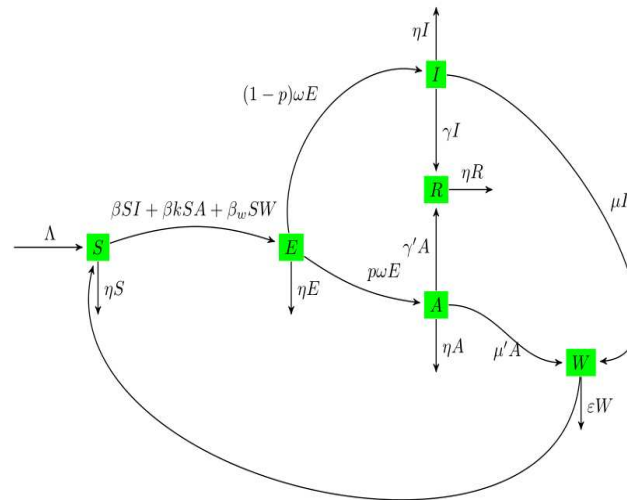
In this section, we propose our long-term model, and compute equilibrium solutions and the basic reproduction number. We analyze how an asymptomatic transmission changes the basic reproduction number. We also prove global stabilities for the equilibria.

### 2.1. Preliminaries

We divide the population into five compartments: susceptible individuals, exposed individuals, infected individuals, asymptomatic individuals who have acquired the disease, and removed individuals. As traditional models, infected individuals can transmit disease to susceptible individuals. However, there is a subpopulation of asymptomatic infectious individuals, and we consider some proportion of those individuals can also transmit to susceptible individuals. For susceptible individuals, there is some period of time to be exposed to both infected and asymptomatic infectious individuals. Both infected and asymptomatic infectious individuals shed viruses or disease pathogens into the water. Susceptible individuals can also acquire the disease from pathogens in the water. The Figure 1 shows the interactions and flows in our model.

Denote the susceptible compartment by  $S$ , the exposed compartment  $E$ , the infected compartment  $I$ , the asymptomatic compartment  $A$ , the removed compartment  $R$ , and the concentration of pathogens in water  $W$ . This model is given as follows:

$$\begin{cases} \dot{S}(t) = \Lambda - \eta S(t) - \beta S(t)(I(t) + kA(t)) - \beta_w S(t)W(t), \\ \dot{E}(t) = \beta S(t)(I(t) + kA(t)) + \beta_w S(t)W(t) - \omega E(t) - \eta E(t), \\ \dot{I}(t) = (1 - p)\omega E(t) - \gamma I(t) - \eta I(t), \\ \dot{A}(t) = p\omega E(t) - \gamma' A(t) - \eta A(t), \\ \dot{R}(t) = \gamma I(t) + \gamma' A(t) - \eta R(t), \\ \dot{W}(t) = \mu I(t) + \mu' A(t) - \varepsilon W(t), \end{cases} \quad (2.1)$$



**Figure 1.** Flow chart of mathematical model

with initial values  $S(0) = S_0$ ,  $E(0) = E_0$ ,  $I(0) = I_0$ ,  $A(0) = A_0$ ,  $R(0) = 0$ , and  $W(0) = W_0$ . The parameters and their values are given in the Table 1. We briefly explain them here. The parameter  $\Lambda$  is the birth rate of the susceptible individuals while  $\eta$  is the death rate. The parameter  $\beta$  is the individual to individual infection rate. The parameter  $k$  is the proportion of asymptomatic infectious individuals who can transmit the disease to susceptible individuals. The parameter  $\beta_w$  is the infection rate from pathogens in water to susceptible individuals. The parameter  $\omega$  is the incubation rate. The parameter  $p$  is the proportion of asymptomatic infected individuals. The parameter  $\gamma$  is the recovery rate of infected individuals while  $\gamma'$  is the recovery rate of asymptomatic infected individuals. The parameter  $\mu$  is the pathogen shedding rate from infected individuals while  $\mu'$  is the pathogen shedding rate from asymptomatic infected individuals. The parameter  $\varepsilon$  is the pathogen degenerate rate, and  $1/\varepsilon$  is the average lifetime of the pathogens in the water.

If we let  $M(t) = S(t) + E(t) + I(t) + A(t) + R(t)$ , and add first five equations together, we get  $\dot{M}(t) = \Lambda - \eta M(t)$ . Therefore, we can delete one equation from the first five equations. We consider the following model in the rest of the paper,

$$\begin{cases} \dot{S}(t) = \Lambda - \eta S(t) - \beta S(t)(I(t) + kA(t)) - \beta_w S(t)W(t), \\ \dot{E}(t) = \beta S(t)(I(t) + kA(t)) + \beta_w S(t)W(t) - \omega E(t) - \eta E(t), \\ \dot{I}(t) = (1-p)\omega E(t) - \gamma I(t) - \eta I(t), \\ \dot{A}(t) = p\omega E(t) - \gamma' A(t) - \eta A(t), \\ \dot{W}(t) = \mu I(t) + \mu' A(t) - \varepsilon W(t). \end{cases} \quad (2.2)$$

It is easy to show  $S(t) \geq 0$ ,  $E(t) \geq 0$ ,  $I(t) \geq 0$ ,  $A(t) \geq 0$ ,  $W(t) \geq 0$  if the initial values are nonnegative. Hence, we can have that the nonnegative cone  $\mathbb{R}_+^5$  is positively invariant for model (2.2) where our model is well-posed. According to the model (2.2), let  $S(t) + E(t) + I(t) + A(t) = N(t)$ , then  $\dot{N}(t) = \Lambda - \eta N(t) - \gamma I(t) - \gamma' A(t) \leq \Lambda - \eta N(t)$ . We have  $N(t) \leq \Lambda/\eta + (N(0) - \Lambda/\eta)e^{-\eta t}$  and then  $\limsup_{t \rightarrow \infty} N(t) \leq \Lambda/\eta$ . Further, we can have that  $\limsup_{t \rightarrow \infty} W(t) \leq \Lambda(\mu + \mu')/\varepsilon\eta$ . Therefore, all solutions of model (2.2) are bounded in  $\mathbb{R}_+^5$ .

Next, we compute equilibrium solutions for model (2.2). We set the right-hand side of each equation in the model (2.2) to be zero,

$$\begin{cases} \Lambda - \eta S^* - \beta S^*(I^* + kA^*) - \beta_w S^* W^* = 0 \\ \beta S^*(I^* + kA^*) + \beta_w S^* W^* - \omega E^* - \eta E^* = 0 \\ (1-p)\omega E^* - \gamma I^* - \eta I^* = 0 \\ p\omega E^* - \gamma' A^* - \eta A^* = 0 \\ \mu I^* + \mu' A^* - \varepsilon W^* = 0. \end{cases}$$

The equilibrium points are solved as follows.

The disease-free equilibrium point of the model is  $E^0 = (S_0, 0, 0, 0, 0)$ , the endemic equilibrium point is  $E^1 = (S^*, E^*, I^*, A^*, W^*)$ , where  $S_0 = \frac{\Lambda}{\eta}$ ,

$$S^* = \frac{\Lambda}{\eta + \beta(I^* + kA^*) + \beta_w W^*}, \quad E^* = \frac{\Lambda}{\omega + \eta} \left(1 - \frac{1}{\mathcal{R}_0}\right), \quad I^* = \frac{(1-p)\omega\Lambda}{(\gamma + \eta)(\omega + \eta)} \left(1 - \frac{1}{\mathcal{R}_0}\right),$$

$$A^* = \frac{p\omega\Lambda}{(\gamma' + \eta)(\omega + \eta)} \left(1 - \frac{1}{\mathcal{R}_0}\right), \quad W^* = \left[ \frac{(1-p)\mu\omega\Lambda}{(\gamma + \eta)(\omega + \eta)\varepsilon} + \frac{p\omega\mu'\Lambda}{(\gamma' + \eta)(\omega + \eta)\varepsilon} \right] \left(1 - \frac{1}{\mathcal{R}_0}\right).$$

$\mathcal{R}_0$  is the basic reproduction number, which is calculated next.

Now, we can calculate the basic reproduction number of the model (2.2) according to the next generation matrix methods [19]. Then the basic reproduction number  $\mathcal{R}_0$  is given by

$$\begin{aligned} \mathcal{R}_0 &= \frac{(1-p)\omega\beta S_0}{(\gamma + \eta)(\omega + \eta)} + \frac{\mu(1-p)\omega\beta_w S_0}{(\gamma + \eta)(\omega + \eta)\varepsilon} \\ &\quad + \frac{\mu' p\omega\beta_w S_0}{(\gamma' + \eta)(\omega + \eta)\varepsilon} + \frac{pk\omega\beta S_0}{(\gamma' + \eta)(\omega + \eta)} \\ &= (1-p) \left[ \frac{\omega\beta S_0}{(\gamma + \eta)(\omega + \eta)} + \frac{\mu\omega\beta_w S_0}{(\gamma + \eta)(\omega + \eta)\varepsilon} \right] \\ &\quad + p \left[ \frac{\mu'\omega\beta_w S_0}{(\gamma' + \eta)(\omega + \eta)\varepsilon} + \frac{k\omega\beta S_0}{(\gamma' + \eta)(\omega + \eta)} \right]. \end{aligned} \tag{2.3}$$

In order to understand how an asymptomatic transmission changes the dynamics of epidemics, we may consider the basic reproduction number  $\mathcal{R}_0$  is a function of the parameter  $p$  and  $k$ . The partial derivatives with respect to those parameters will reveal the information. It is easy to obtain these derivatives.

$$\frac{\partial \mathcal{R}_0}{\partial p} = \frac{\omega\beta S_0}{\omega + \eta} \left( \frac{k}{\gamma' + \eta} - \frac{1}{\gamma + \eta} \right) + \frac{\omega\beta_w S_0}{(\omega + \eta)\varepsilon} \left( \frac{\mu'}{\gamma' + \eta} - \frac{\mu}{\gamma + \eta} \right),$$

$$\frac{\partial \mathcal{R}_0}{\partial p} = \frac{\omega S_0}{\omega + \eta} \left[ \frac{\beta k}{\gamma' + \eta} - \frac{\beta}{\gamma + \eta} + \frac{\beta_w \mu'}{(\gamma' + \eta)\varepsilon} - \frac{\beta_w \mu}{\varepsilon(\gamma + \eta)} \right],$$

$$\frac{\partial \mathcal{R}_0}{\partial k} = \frac{\omega\beta S_0 p}{(\gamma' + \eta)(\omega + \eta)}.$$

**Remark 1.** The basic reproduction number has two parts, one part is from infected transmission and other one from asymptomatic transmission.

The basic reproduction number  $\mathcal{R}_0$  is an increasing function of the parameter  $k$  which is the proportion of asymptomatic infectious individuals who can transmit the disease to susceptible individuals. If all other parameters are fixed, when the parameter  $k$  is increasing, the dynamics of model (2.1) can be dramatically changed once  $\mathcal{R}_0$  increases from the value smaller than 1 to the value greater than 1.

Now, we fixed all parameters except  $p$  and  $k$ . There exists a critical value of  $k$  under a simple condition, the basic reproduction number  $\mathcal{R}_0$  is an increasing function of  $p$  when  $k$  is greater than the critical value, and  $\mathcal{R}_0$  is a decreasing function of  $p$  when  $k$  is smaller than the critical value. This means that, the proportion of asymptomatic infected individuals can also change the dynamics of the model (2.1) in two different ways depending on the proportion of asymptomatic infectious individuals.

## 2.2. Global stability

In this subsection, we present two theorems about global stability of equilibrium solutions.

**Theorem 1.** When  $\mathcal{R}_0 \leq 1$ , the disease-free equilibrium  $E^0$  is globally asymptotically stable in the region  $\mathbb{R}_+^5$ .

*Proof.* We prove the global stability of  $E_0$  by constructing a Lyapunov function. Let

$$U(t) = (S(t), E(t), I(t), A(t), W(t))$$

be the solution of model (2.2) through any  $\varphi := (\varphi_1, \varphi_2, \varphi_3, \varphi_4, \varphi_5) \in \mathbb{R}_+^5$ . It is not to find that  $U(t)$  is bounded and further  $S(t) > 0$  for all  $t > 0$ . Now, let us define a function  $V$  on  $D_0 = \{\varphi \in \mathbb{R}_+^5 : \varphi_1 > 0\}$ ,

$$\begin{aligned} V(\varphi) = & \varphi_1 - S_0 - S_0 \ln \frac{\varphi_1}{S_0} \\ & + \mathcal{R}_0 \varphi_2 + \frac{\beta S_0 \varepsilon + \mu \beta_w S_0}{(\gamma + \eta) \varepsilon} \varphi_3 \\ & + \frac{\mu' \beta_w S_0 + k \beta S_0 \varepsilon}{(\gamma' + \eta) \varepsilon} \varphi_4 + \frac{\beta_w S_0}{\varepsilon} \varphi_5. \end{aligned} \quad (2.4)$$

Clearly,  $V$  is continuous on  $D_0 \subset \mathbb{R}_+^5$  and an infinite positive definite function with respect to  $E^0$ .

Taking the derivative of (2.4) along the solution  $U(t) \in D_0$  of model (2.2) for  $t \geq 1$ ,

$$\dot{V}(U(t)) = \left(1 - \frac{S_0}{S(t)}\right) \dot{S}(t) + \mathcal{R}_0 \dot{E}(t) + \frac{\beta S_0 \varepsilon + \mu \beta_w S_0}{(\gamma + \eta) \varepsilon} \dot{I}(t) + \frac{\mu' \beta_w S_0 + k \beta S_0 \varepsilon}{(\gamma' + \eta) \varepsilon} \dot{A}(t) + \frac{\beta_w S_0}{\varepsilon} \dot{W}(t).$$

Then it follows

$$\begin{aligned} \dot{V}(U(t)) = & \left(1 - \frac{S_0}{S(t)}\right) [\Lambda - \eta S(t) - \beta S(t)(I(t) + kA(t)) - \beta_w S(t)W(t)] \\ & + \left[ \frac{(1-p)\omega\beta S_0}{(\gamma + \eta)(\omega + \eta)} + \frac{pk\omega\beta S_0}{(\gamma' + \eta)(\omega + \eta)} \right] [\beta S(t)(I(t) + kA(t)) + \beta_w S(t)W(t) - \omega E(t) - \eta E(t)] \\ & + \left[ \frac{\mu(1-p)\omega\beta_w S_0}{(\gamma + \eta)(\omega + \eta)\varepsilon} + \frac{\mu' p\omega\beta_w S_0}{(\gamma' + \eta)(\omega + \eta)\varepsilon} \right] [\beta S(t)(I(t) + kA(t)) + \beta_w S(t)W(t) - \omega E(t) - \eta E(t)] \\ & + \frac{\beta S_0 \varepsilon + \mu\beta_w S_0}{(\gamma + \eta)\varepsilon} [(1-p)\omega E(t) - \gamma I(t) - \eta I(t)] \\ & + \frac{\mu'\beta_w S_0 + k\beta S_0 \varepsilon}{(\gamma' + \eta)\varepsilon} (p\omega E(t) - \gamma' A(t) - \eta A(t)) \\ & + \frac{\beta_w S_0}{\varepsilon} (\mu I(t) + \mu' A(t) - \varepsilon W(t)). \end{aligned}$$

Simplified the above expression, we have

$$\dot{V}(U(t)) = -\eta \frac{(S(t) - S_0)^2}{S(t)} + (\mathcal{R}_0 - 1) [\beta S(t)(I(t) + kA(t)) + \beta_w S(t)W(t)] \leq 0 \quad (2.5)$$

for  $\mathcal{R}_0 \leq 1$ . By (2.4) and (2.5), there is an  $\epsilon = \epsilon(\varphi) > 0$  such that  $\liminf_{t \rightarrow \infty} S(t) > \epsilon$ . Thus, we have that  $V$  is continuous on  $\bar{\Gamma}_0 \subset D_0$ , where  $\bar{\Gamma}_0$  is the closure of  $\Gamma_0 = \{U(t) : t \geq 1\}$ . Consequently, [20, Corollary 3.3 and Remark 3.2] implies that  $E^0$  is uniformly stable. Further,  $V$  is also a Lyapunov function on  $\Gamma_1$ . From [20, corollary 2.1], we have that  $\dot{V}(\phi) = 0$  for any  $\phi \in \omega(\varphi)$ , where  $\omega(\varphi)$  is the  $\omega$ -limit set of  $\varphi$  for model (2.2).

Next, we only need to prove that  $\omega(\varphi) = \{E^0\}$ . Let  $U(t)$  be the solution of model (2.2) with any  $\phi \in \omega(\varphi)$ . The invariance of  $\omega(\varphi)$  yields  $U(t) \in \omega(\varphi)$  for all  $t \in \mathbb{R}$ . We thus have  $\dot{V}(U(t)) = 0$  for all  $t \in \mathbb{R}$ . Considering (2.5), it holds  $S(t) = S_0$  for all  $t \in \mathbb{R}$ . From the first equation of model (2.2), it follows  $I(t) = A(t) = W(t) = 0$  for all  $t \in \mathbb{R}$ . By the second equation of model (2.2) and the invariance of  $\omega(\varphi)$ , we have  $E(t) = 0$  for all  $t \in \mathbb{R}$ . Therefore,  $\omega(\varphi) = \{E^0\}$ .  $\square$

**Theorem 2.** When  $\mathcal{R}_0 > 1$ , the endemic equilibrium  $E^1$  is globally asymptotically stable in the region  $D = \{\varphi \in \mathbb{R}_+^5 : \varphi_2 > 0\}$ .

*Proof.* Firstly, it is not difficult to obtain the set  $D \subset \mathbb{R}_+^5$  is a positive invariant set of model (2.2). Let  $U(t) = (S(t), E(t), I(t), A(t), W(t))$  be the solution of model (2.2) with any  $\varphi \in D$ . It follows  $U(t)$  is bounded and  $U(t) \gg \mathbf{0}$  for  $t > 0$ . Now, let us define a function  $V$  on  $D_1 = \{\varphi \in \mathbb{R}_+^5 : \varphi \gg \mathbf{0}\}$ ,

$$\begin{aligned} V(\varphi) = & \varphi_1 - S^* - S^* \ln \frac{\varphi_1}{S^*} + \varphi_2 - E^* - E^* \ln \frac{\varphi_2}{E^*} \\ & + \left[ \frac{\beta S^*}{\gamma + \eta} + \frac{\beta_w S^* \mu}{\varepsilon(\gamma + \eta)} \right] \left( \varphi_3 - I^* - I^* \ln \frac{\varphi_3}{I^*} \right) \\ & + \left[ \frac{\beta k S^*}{\gamma' + \eta} + \frac{\beta_w S^* \mu'}{\varepsilon(\gamma' + \eta)} \right] \left( \varphi_4 - A^* - A^* \ln \frac{\varphi_4}{A^*} \right) \\ & + \frac{\beta_w S^*}{\varepsilon} \left( \varphi_5 - W^* - W^* \ln \frac{\varphi_5}{W^*} \right). \end{aligned} \quad (2.6)$$

It is easy to see that  $V$  is continuous on  $D_1 \subset D$  and an infinite positive definite function with respect to  $E^1$ .

We take the derivative of (2.6) along the solution  $U(t) \in D_1$  of the model (2.2) for  $t \geq 1$  as follows,

$$\begin{aligned} \dot{V}(U(t)) = & -\eta \frac{[S(t) - S^*]^2}{S(t)} + \beta S^* I^* + \beta S^* k A^* + \beta_w S^* W^* - \beta S(t) I(t) - \beta k S(t) A(t) - \beta_w S(t) W(t) \\ & - \frac{(S^*)^2}{S(t)} \beta I^* - \frac{(S^*)^2}{S(t)} \beta k A^* - \frac{(S^*)^2}{S(t)} \beta_w W^* + \beta S^* I(t) + \beta k S^* A(t) + \beta_w S^* W(t) \\ & + \beta S(t) I(t) + \beta k S(t) A(t) + \beta_w S(t) W(t) - (\omega + \eta) E(t) - \frac{E^*}{E(t)} \beta S(t) I(t) - \frac{E^*}{E(t)} \beta k A(t) \\ & - \frac{E^*}{E(t)} \beta_w S(t) W(t) + (\omega + \eta) E^* + \frac{\beta S^*}{\gamma + \eta} (1 - p) \omega E(t) - \frac{\beta S^*}{\gamma + \eta} (\gamma + \eta) I(t) \\ & - \frac{\beta S^*}{\gamma + \eta} \frac{I^*}{I(t)} (1 - p) \omega E(t) + \frac{\beta S^*}{\gamma + \eta} (\gamma + \eta) I^* + \frac{\beta_w S^* \mu}{\varepsilon (\gamma + \eta)} (1 - p) \omega E(t) - \frac{\beta_w S^* \mu}{\varepsilon (\gamma + \eta)} (\gamma + \eta) I(t) \\ & - \frac{\beta_w S^* \mu}{\varepsilon (\gamma + \eta)} \frac{I^*}{I(t)} (1 - p) \omega E(t) + \frac{\beta_w S^* \mu}{\varepsilon (\gamma + \eta)} (\gamma + \eta) I^* + \frac{\beta k S^*}{\gamma' + \eta} p \omega E(t) \\ & - \frac{\beta k S^*}{\gamma' + \eta} (\gamma' + \eta) A(t) - \frac{\beta k S^*}{\gamma' + \eta} \frac{A^*}{A(t)} p \omega E(t) + \frac{\beta k S^*}{\gamma' + \eta} (\gamma' + \eta) A^* + \frac{\beta_w S^* \mu'}{\varepsilon (\gamma' + \eta)} p \omega E(t) \\ & - \frac{\beta_w S^* \mu'}{\varepsilon (\gamma' + \eta)} (\gamma' + \eta) A(t) - \frac{\beta_w S^* \mu'}{\varepsilon (\gamma' + \eta)} \frac{A^*}{A(t)} p \omega E(t) + \frac{\beta_w S^* \mu'}{\varepsilon (\gamma' + \eta)} (\gamma' + \eta) A^* \\ & + \frac{\beta_w S^*}{\varepsilon} \mu I(t) + \frac{\beta_w S^*}{\varepsilon} \mu' A(t) - \beta_w S^* W(t) \\ & - \frac{\beta_w S^*}{\varepsilon} \frac{W^*}{W(t)} \mu I(t) - \frac{\beta_w S^*}{\varepsilon} \frac{W^*}{W(t)} \mu' A(t) + \beta_w S^* W^*, \end{aligned}$$

where  $(1 - p)\omega = (\gamma + \eta)I^*/E^*$ ,  $p\omega = (\gamma' + \eta)A^*/E^*$ ,  $\omega + \eta = (\beta S^* I^* + \beta k S^* A^* + \beta_w S^* W^*)/E^*$ . Finally, we have that

$$\begin{aligned} \dot{V}(U(t)) = & -\eta \frac{(S(t) - S^*)^2}{S(t)} + \beta S^* I^* \left[ 3 - \frac{S^*}{S(t)} - \frac{E^* S(t) I(t)}{E(t) S^* I^*} - \frac{I^* E(t)}{I(t) E^*} \right] \\ & + \frac{\beta_w S^* A^* \mu'}{\varepsilon} \left[ 4 - \frac{S^*}{S(t)} - \frac{E^* S(t) W(t)}{E(t) S^* W^*} - \frac{A^* E(t)}{A(t) E^*} - \frac{W^* A(t)}{W(t) A^*} \right] \\ & + \frac{\beta_w S^* I^* \mu}{\varepsilon} \left[ 4 - \frac{S^*}{S(t)} - \frac{E^* S(t) W(t)}{E(t) S^* W^*} - \frac{I^* E(t)}{I(t) E^*} - \frac{W^* I(t)}{W(t) I^*} \right] \\ & + \beta k S^* A^* \left[ 3 - \frac{S^*}{S(t)} - \frac{E^* S(t) A(t)}{E(t) S^* A^*} - \frac{A^* E(t)}{A(t) E^*} \right] \leq 0, \end{aligned} \tag{2.7}$$

where the average value inequalities are used.

From (2.6) and (2.7), it follows  $\omega(\varphi) \subseteq D_1$ . Hence by [20, Corollary 3.3 and Remark 3.2], we can obtain that  $E^1$  is uniformly stable. And  $V$  is a Lyapunov function on  $\{U(t) : t \geq 1\} \subset D_1$  for  $\mathcal{R}_0 > 1$ . It follows from [20, corollary 2.1] that  $\dot{V}(\phi) = 0$  for any  $\phi \in \omega(\varphi)$ .

Let  $U(t) = (S(t), E(t), I(t), A(t), W(t))$  be the solution of the model (2.2) with any  $\phi \in \omega(\varphi)$ . Then by the invariance of  $\omega(\varphi)$ , we have  $U(t) \in \omega(\varphi)$  for all  $t \geq 0$ . Consequently, it follow from (2.7) that  $S(t) = S^*$ ,  $I(t)E^* = I^*E(t)$ ,  $A^*E(t) = A(t)E^*$  and  $W^*I(t) = W(t)I^*$  for all  $t \geq 0$ . Hence, we have

$$E^* \dot{I}(t) = E^* (1 - p) \omega E(t) - (\gamma + \eta) E^* I(t) = (E^* (1 - p) \omega - (\gamma + \eta) I^*) E(t) = 0,$$



which yields that  $I(t)$  is a constant function on  $\mathbb{R}^+$ . Further, it holds that  $U(t) (\gg \mathbf{0})$  is a constant vector function, i.e., a positive equilibrium of the model (2.2). By the uniqueness of the positive equilibrium, we have  $\omega(\varphi) = \{E^1\}$  for any  $\varphi \in D$  and then  $E^1$  is globally attractive.  $\square$

### 3. Short-term SEIAWR model and its analysis

Chen et al. studied about shigella outbreaks in a local area in China [16]. They employed a short-term SEIAWR model, where the birth and death of individuals were no longer considered. In this section, we will carry out a basic analysis for the model. Particularly, we compute the basic reproduction number, and the final size.

The model is as follows.

$$\begin{cases} \dot{S}(t) = -\beta S(t)(I(t) + kA(t)) - \beta_w S(t)W(t), \\ \dot{E}(t) = \beta S(t)(I(t) + kA(t)) + \beta_w S(t)W(t) - \omega E(t), \\ \dot{I}(t) = (1-p)\omega E(t) - \gamma I(t), \\ \dot{A}(t) = p\omega E(t) - \gamma' A(t), \\ \dot{R}(t) = \gamma I(t) + \gamma' A(t), \\ \dot{W}(t) = \mu I(t) + \mu' A(t) - \varepsilon W(t), \end{cases} \quad (3.1)$$

with initial conditions  $S(0) = N - E_0 - I_0 - A_0$ ,  $E(0) = E_0$ ,  $I(0) = I_0$ ,  $A(0) = A_0$ ,  $R(0) = 0$ , and  $W(0) = W_0$ .

When the birth rate and death rate of the population are not taken into account, the initial population number is constant  $N$ ,  $S(0) = N$ . It is easy to compute the basic reproduction number, or to derive from the long-term model, which is given by

$$\mathcal{R}_0 = \frac{(1-p)N}{\gamma} \left( \beta + \frac{\mu\beta_w}{\varepsilon} \right) + \frac{pN}{\gamma'} \left( k\beta + \frac{\mu'\beta_w}{\varepsilon} \right). \quad (3.2)$$

If we take partial derivative of  $\mathcal{R}_0$  with respect to parameter  $p$  and  $k$ , we have

$$\frac{\partial \mathcal{R}_0}{\partial p} = \beta N \left( \frac{k}{\gamma'} - \frac{1}{\gamma} \right) + \frac{N\beta_w}{\varepsilon} \left( \frac{\mu'}{\gamma'} - \frac{\mu}{\gamma} \right), \quad \frac{\partial \mathcal{R}_0}{\partial k} = \frac{pN\beta}{\gamma'}.$$

We have a similar remark.

**Remark 2.** *The basic reproduction number has two parts, one part is from infected transmission and other one from asymptomatic transmission.*

*The basic reproduction number  $\mathcal{R}_0$  is an increasing function of the parameter  $k$  which is the proportion of asymptomatic infectious individuals who can transmit the disease to susceptible individuals. If all other parameters are fixed, when the parameter  $k$  is increasing, the dynamics of the system 2.1 can be dramatically changed once  $\mathcal{R}_0$  increases from the value smaller than 1 to the value greater than 1.*

*Now, we fixed all parameters except  $p$  and  $k$ . There exists a critical value of  $k$ , the basic reproduction number  $\mathcal{R}_0$  is an increasing function of  $p$  when  $k$  is greater than the critical value, and  $\mathcal{R}_0$  is a decreasing function of  $p$  when  $k$  is smaller than the critical value. This means that, the proportion of asymptomatic infected individuals can also change the dynamics of the model (2.1) in two different ways depending on the proportion of asymptomatic infectious individuals. Of course, the critical value of  $k$  is different from that in the long-term model.*

For short-term outbreaks of infectious diseases, we can find the final size of the susceptible individuals, which can give us some information about the reproduction number. We now compute the final size. Adding the first two equations of model (3.1), we get

$$\dot{S}(t) + \dot{E}(t) = -\omega E(t). \quad (3.3)$$

We claim that  $\dot{S}(t) + \dot{E}(t)$  is uniformly continuous and then  $\lim_{t \rightarrow \infty} (\dot{S}(t) + \dot{E}(t)) = 0$ . In fact, for any  $\epsilon > 0$ , there exists a  $\delta = \delta(\epsilon) > 0$ , it holds

$$|(\dot{S} + \dot{E})(t_1) - (\dot{S} + \dot{E})(t_2)| < \frac{\epsilon}{2}, \text{ whenever } |t_1 - t_2| < \delta.$$

It is easy to know the limit of  $S(t) + E(t)$  exists. By Cauchy convergence principle, there is a  $T = T(\epsilon) > 0$  such that when  $t > T$ , we have

$$|(S + E)(t + \delta) - (S + E)(t)| < \frac{\epsilon \delta}{2}.$$

Hence it follows

$$\begin{aligned} |\dot{S}(t) + \dot{E}(t)| &= \left| \dot{S}(t) + \dot{E}(t) - \frac{(S + E)(t + \delta) - (S + E)(t)}{\delta} + \frac{(S + E)(t + \delta) - (S + E)(t)}{\delta} \right| \\ &\leq |(\dot{S} + \dot{E})(t) - (\dot{S} + \dot{E})(\alpha)| + \frac{1}{\delta} |(S + E)(t + \delta) - (S + E)(t)| \\ &< \frac{\epsilon}{2} + \frac{1}{\delta} \frac{\epsilon \delta}{2} = \epsilon, \end{aligned}$$

where  $\alpha \in (t, t + \delta)$ . Consequently,

$$\lim_{t \rightarrow \infty} E(t) = E(\infty) = 0.$$

From the third and the fourth equations of model (3.1), we can obtain that  $\limsup_{t \rightarrow \infty} I(t) \leq 0$  and  $\limsup_{t \rightarrow \infty} A(t) \leq 0$ . Since  $I(t) \geq 0$ ,  $W(t) \geq 0$ , it holds  $\lim_{t \rightarrow \infty} I(t) = \lim_{t \rightarrow \infty} A(t) = 0$ . By the sixth equation of model (3.1), similarly, we have  $\lim_{t \rightarrow \infty} W(t) = 0$ . It is easy to conclude that  $S(\infty)$  exists.

From (3.3), it follows

$$\int_0^{\infty} (\dot{S}(t) + \dot{E}(t)) dt = -\omega \int_0^{\infty} E(t) dt,$$

Further, we have

$$\int_0^{\infty} E(t) dt = \frac{S(0) + E(0) - S(\infty) - E(\infty)}{\omega} = \frac{S(0) + E(0) - S(\infty)}{\omega}. \quad (3.4)$$

The first three equations of the model (3.1) are added up, we have

$$\dot{S}(t) + \dot{E}(t) + \dot{I}(t) = -\omega p E(t) - \gamma I(t),$$

which yields

$$S(0) + E(0) + I(0) - S(\infty) - E(\infty) - I(\infty) = \omega p \int_0^{\infty} E(t) dt + \gamma \int_0^{\infty} I(t) dt.$$

Substituting (3.3), we get

$$\int_0^{\infty} I(t)dt = \frac{(1-p)S(0) + (1-p)E(0) + I(0) - (1-p)S(\infty)}{\gamma}. \quad (3.5)$$

The first four equations of the model (3.1) are added up, we have,

$$\dot{S}(t) + \dot{E}(t) + \dot{I}(t) + \dot{A}(t) = -\gamma'A(t) - \gamma I(t),$$

which leads

$$S(0) + E(0) + I(0) + A(0) - S(\infty) - E(\infty) - I(\infty) - A(\infty) = \gamma \int_0^{\infty} I(t)dt + \gamma' \int_0^{\infty} A(t)dt.$$

Substituting (3.5), which gives

$$\int_0^{\infty} A(t)dt = \frac{pS(0) + pE(0) + A(0) - pS(\infty)}{\gamma'}. \quad (3.6)$$

Integrating the last equation of the model (3.1), we have

$$\int_0^{\infty} \dot{W}(t)dt = \mu \int_0^{\infty} I(t)dt + \mu' \int_0^{\infty} A(t)dt - \varepsilon \int_0^{\infty} W(t)dt. \quad (3.7)$$

Substituting (3.5) and (3.6), it follows

$$\int_0^{\infty} W(t)dt = \frac{W(0)}{\varepsilon} + \frac{\mu(1-p)S(0) + \mu(1-p)E(0) + \mu I(0) - \mu(1-p)S(\infty)}{\gamma\varepsilon} + \frac{\mu'pS(0) + \mu'pE(0) + \mu'A(0) - \mu'pS(\infty)}{\gamma'\varepsilon}. \quad (3.8)$$

Next, we claim that  $S(\infty) > 0$ . In fact, (3.5), (3.6) and (3.8) hint that

$$\int_0^{\infty} (\beta(I(t) + kA(t)) + \beta_w W(t)) dt$$

is convergent. Therefore,

$$S(\infty) = S(0)e^{-\int_0^{\infty} [\beta(I(t)+kA(t))+\beta_w W(t)]dt} > 0.$$

Integrate the first equation of the model (3.1), we have

$$\ln \frac{S(0)}{S(\infty)} = \beta \int_0^{\infty} I(t)dt + \beta k \int_0^{\infty} A(t)dt + \beta_w \int_0^{\infty} W(t)dt, \quad (3.9)$$

Substituting (3.5), (3.6) and (3.8), which yields,

$$\begin{aligned} \ln \frac{S(0)}{S(\infty)} &= \beta \frac{(1-p)S(0) + (1-p)E(0) + I(0) + (p-1)S(\infty)}{\gamma} \\ &\quad + \beta k \frac{pS(0) + pE(0) + A(0) - pS(\infty)}{\gamma'} + \beta_w \frac{W(0)}{\varepsilon} \\ &\quad + \beta_w \frac{\mu(1-p)S(0) + \mu(1-p)E(0) + \mu I(0) - \mu(1-p)S(\infty)}{\gamma\varepsilon} \\ &\quad + \beta_w \frac{\mu'pS(0) + \mu'pE(0) + \mu'A(0) - \mu'pS(\infty)}{\gamma'\varepsilon}. \end{aligned}$$

Further, we have

$$\begin{aligned} \ln \frac{S(0)}{S(\infty)} = \mathcal{R}_0 \left( 1 - \frac{I(0)}{N} - \frac{A(0)}{N} - \frac{S(\infty)}{N} \right) + \left( \frac{\beta}{\gamma} + \frac{\mu\beta_w}{\gamma\varepsilon} \right) I(0) \\ + \left( \frac{\beta k}{\gamma'} + \frac{\mu'\beta_w}{\gamma'\varepsilon} \right) A(0) + \beta_w \frac{W(0)}{\varepsilon}. \end{aligned}$$

We take  $I(0) = A(0) = W(0) = 0$  along this line in [21]. Thus, we have

$$\ln \frac{S(0)}{S(\infty)} = \mathcal{R}_0 \left( 1 - \frac{S(\infty)}{N} \right).$$

If we consider the final size is a function of the basic reproduction number  $\mathcal{R}_0$ , we can get some understanding about how  $\mathcal{R}_0$  affect the final size. We take the derivative of the final size with respect to  $\mathcal{R}_0$ , and have

$$\frac{dS(\infty)}{d\mathcal{R}_0} = \frac{N - S(\infty)}{\mathcal{R}_0 - N/S(\infty)}.$$

Because the total population size  $N$  is always greater than the final size,  $N > S(\infty)$ , the sign of  $dS(\infty)/d\mathcal{R}_0$  is determined by the denominator  $\mathcal{R}_0 - N/S(\infty)$ . If  $\mathcal{R}_0 < 1$ , then the final size is a decreasing function of  $\mathcal{R}_0$ . There exists a critical value of  $\mathcal{R}_0$  which is greater than 1, the final size will be an increasing function of the basic reproduction number  $\mathcal{R}_0$  when  $\mathcal{R}_0$  is greater than this critical value.

We may combine the information in Remark 2 to infer how parameters  $k$  and  $p$  influence the final size. Because  $\mathcal{R}_0$  is an increasing function of  $k$ , we may infer that there exists a critical value of  $k$  which corresponds to the critical value of  $\mathcal{R}_0$ . When  $k$  is greater than this critical value, the final size  $S(\infty)$  is an increasing function of  $k$ ; when  $k$  is smaller than this critical value, the final size  $S(\infty)$  is a decreasing function of  $k$ .

However,  $\mathcal{R}_0$  can increase or decrease as  $p$  varies depending on  $k$ , we may infer that there exists three critical values of  $k$  which divide the interval  $[0, 1]$  into four subintervals. In each subinterval, the final size is a monotonic function of  $p$ .

**Remark 3.** *The proportion of asymptomatic infected individuals and the proportion of asymptomatic infectious individuals both influence on the final size. There exists a critical value of  $k$ , the final size will increase as  $k$  increases when  $k$  is greater than the critical value, while the final size will decrease as  $k$  increase when  $k$  is smaller than the critical value. The way of  $p$  influencing the final size depends on the proportion of asymptomatic infectious individuals.*

## 4. Numerical studies

In this section, we perform several numerical demonstrations for both short and long term models.

### 4.1. Numerical study of the short-term model

Most of parameter values are taken from research [16, 17], two parameter values are taken from [22]. We also assume two parameter values. We are interesting in how  $p$  and  $k$  influence the dynamics

**Table 1.** Description of parameters in the model

Parameter	Description	The parameter value	source
$\beta$	Person-to-person infection rate	-	-
$b$	$b = \beta N$	0.0898	[17]
$\beta_w$	Reservoir-to-person infection rate	-	-
$b_w$	$\mu\beta_w N/\varepsilon$	$1.1264 \times 10^{-9}$	[17]
$k$	Proportion of asymptomatic infectious	0.3125	[17]
$\omega$	Incubation rate	1	[17]
$p$	Proportion of asymptomatic infected	0.1	[17]
$\gamma$	Recovery rate of the infected	0.0741	[17]
$\gamma'$	Recovery rate of the asymptomatic	0.0286	[17]
$\mu$	Pathogen shedding rate from the infected	0.63	Assumed
$\mu'$	Pathogen shedding rate from the asymptomatic infected	0.71	Assumed
$\varepsilon$	Pathogen degenerate rate	0.6931	[17]
$\Lambda$	Birth rate of the population	0.01048	[22]
$\eta$	Natural mortality rate of the population	0.00714	[22]
$N$	The total population	1	Assumed

of the models, therefore as long as all parameter values are in their natural ranges, we are not interested in any particular parameter values for our numerical study. The parameters and their values are summarized in the Table 1.

If we completely use the data about a local outbreak of shigellosis from Chen [17] to compute the basic reproduction number, we get

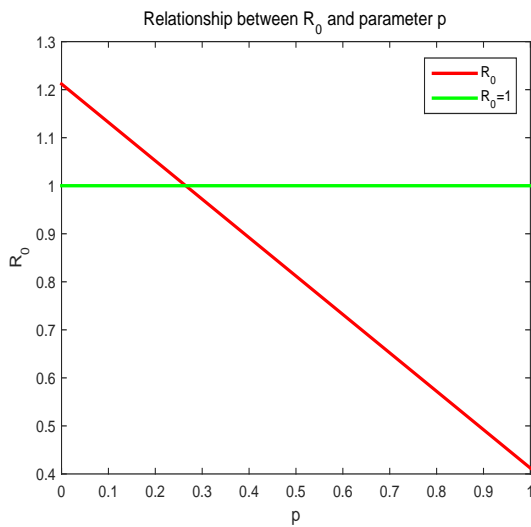
$$\mathcal{R}_0 = 1.188808$$

by simply substituting parameter values into

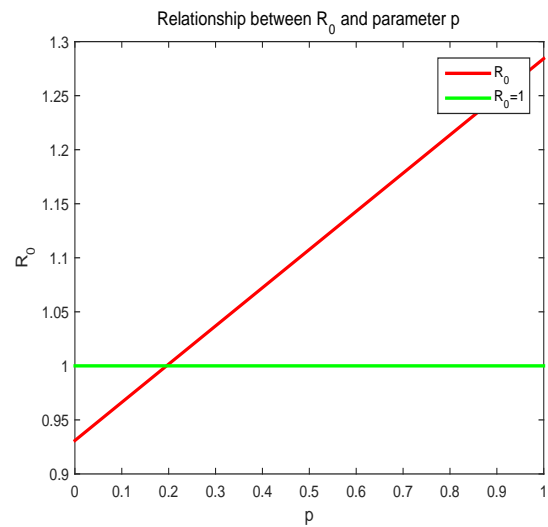
$$\mathcal{R}_0 = \frac{(1-p)b}{\gamma} + \frac{pkb}{\gamma'} + \frac{(1-p)b_w}{\gamma} + \frac{cpb_w}{\gamma'}.$$

According to the relationship between  $\mathcal{R}_0$  and final size, we can calculate

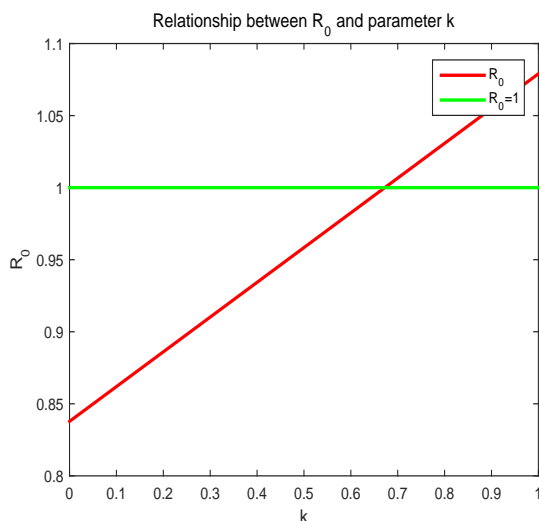
$$S(\infty) = 0.700135.$$



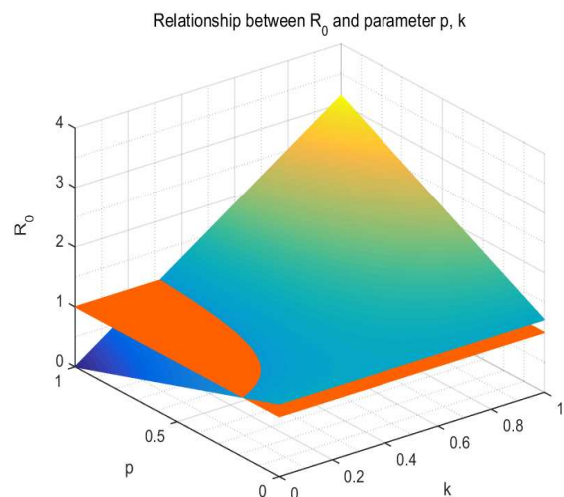
**Figure 2.** Basic reproduction number



**Figure 3.** Basic reproduction number



**Figure 4.** Basic reproduction number



**Figure 5.** Basic reproduction number

We numerically demonstrate how the proportion of asymptomatic infected individuals  $p$  and the proportion of asymptomatic infectious individuals  $k$  influence the basic reproduction number. The parameter values are,  $b = 0.0898$ ,  $b_w = 1.1264 \times 10^{-9}$ ,  $\omega=1$ ,  $\gamma=0.0741$ ,  $\gamma'=0.0286$ ,  $\varepsilon=0.6931$ ,  $\mu'=0.3125$ . We can find that when  $0 \leq k < 0.386$ , the derivative of  $\mathcal{R}_0$  with respect to  $p$  is less than 0, so  $\mathcal{R}_0$  decreases with the increase of  $p$ . When  $0.386 \leq k \leq 1$ , the derivative is greater than 0, so  $R_0$  increases as  $p$  increases. We can see that the partial derivatives of  $\mathcal{R}_0$  with respect to  $k$  are greater than 0 (for any value of  $p$ ), so  $\mathcal{R}_0$  increases monotonically with respect to  $k$ .

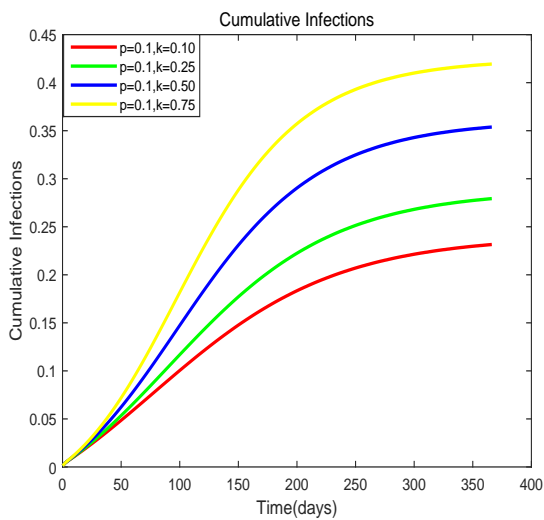
Figure 2 shows that the value of  $\mathcal{R}_0$  changes from greater than 1 to less than 1 as  $p$  increases when  $0 \leq k < 0.386$ . This indicates that the large proportion of asymptomatic infected individuals may prevent outbreaks of the epidemic when the proportion of asymptomatic infectious individuals is

smaller than the critical value.

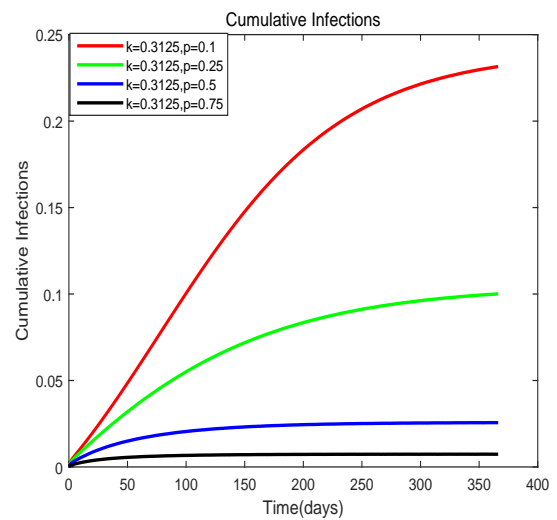
Figure 3 shows that the value of  $\mathcal{R}_0$  changes from less than 1 to greater than 1 as  $p$  increases, when  $0.386 \leq k \leq 1$ . This indicates that the large proportion of asymptomatic infected individuals may promote outbreaks of the epidemic when the proportion of asymptomatic infectious individuals is greater than the critical value.

Figure 4 shows that the value of  $\mathcal{R}_0$  increases as  $k$  increases, and  $\mathcal{R}_0$  changes from less than 1 to greater than 1, which is independent of  $p$ . This indicates that the large proportion of asymptomatic infectious individuals always promote outbreaks of the epidemic.

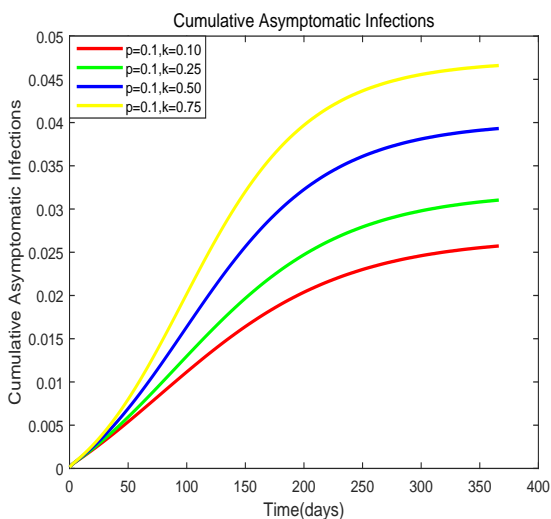
Figure 5 plots  $\mathcal{R}_0$  as a function of  $k$  and  $p$  in the three-dimensional space, where the orange plane is  $\mathcal{R}_0=1$ , the value of  $\mathcal{R}_0$  is greater than 1 above the plane while smaller than 1 below the plane.



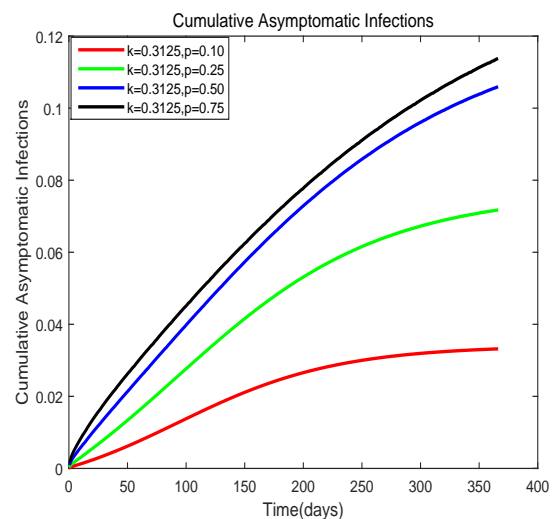
**Figure 6.** Cumulative infections



**Figure 7.** Cumulative infections



**Figure 8.** Asymptomatic infections



**Figure 9.** Asymptomatic infections

We numerically demonstrate how infected and asymptomatic infected individuals changes over time

course with different  $p$  and  $k$  values. Taking  $p = 0.1, k = 0.1, 0.25, 0.5, 0.75$ , the curves of infected and asymptomatic infected individuals over time are calculated and drawn, as shown in Figure 6 and 8. Taking  $k = 0.3125, p = 0.1, 0.25, 0.5, 0.75$ , the curves of infected and asymptomatic infected individuals over time are calculated and drawn, as shown in Figure 7 and 9.

Figure 6 shows how cumulative infections change in time with different proportion  $k$  of asymptomatic infectious individuals when the proportion  $p$  of asymptomatic infected individuals is fixed. This indicates that large  $k$  will make cumulative infections increasing fast. Figure 8 shows how cumulative asymptomatic infections change in time with different proportion  $k$  of asymptomatic infectious individuals when the proportion  $p$  of asymptomatic infected individuals is fixed. This indicates that large  $k$  will make cumulative asymptomatic infections increasing fast.

Figure 7 shows how cumulative infections change in time with different proportion  $p$  of asymptomatic infected individuals when the proportion  $k$  of asymptomatic infectious individuals is fixed. This indicates that small  $p$  will make cumulative infections increasing fast. Figure 9 shows how cumulative asymptomatic infections change in time with different proportion  $p$  of asymptomatic infected individuals when the proportion  $k$  of asymptomatic infectious individuals is fixed. This indicates that large  $p$  will make cumulative asymptomatic infections increasing fast.

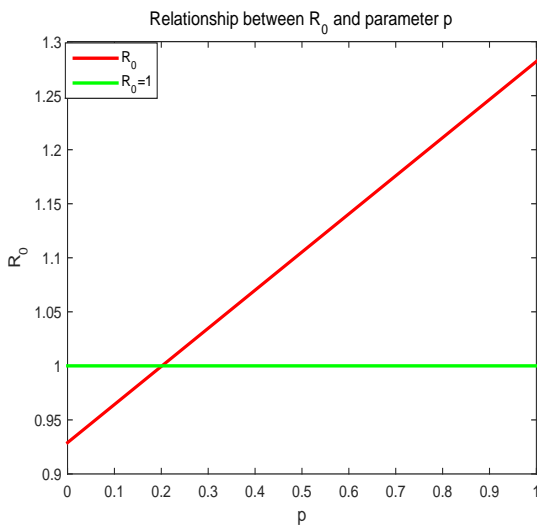
#### 4.2. Numerical study of the long-term model

We numerically demonstrate how the proportion of asymptomatic infected individuals  $p$  and the proportion of asymptomatic infectious individuals  $k$  influence the basic reproduction number. This follows a similar pattern as short-term model.

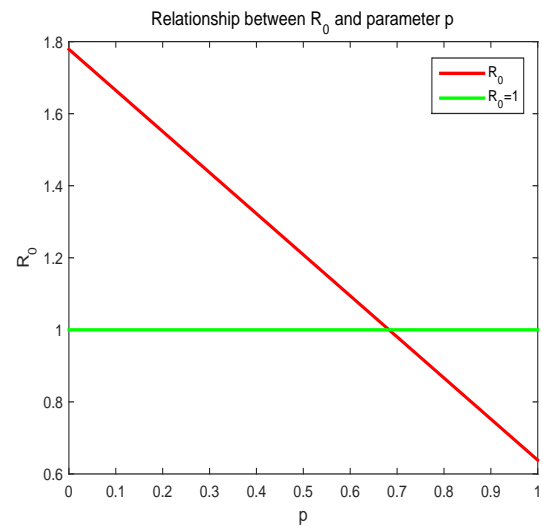
For demonstration, parameter values are  $\mu = 0.63, \mu' = 0.71, \Lambda = 0.01048, \eta = 0.00714, b=0.0898, b_w = 1.1264 \times 10^{-9}, \omega = 1, \gamma = 0.0741, \gamma' = 0.0286, \varepsilon = 0.6931, c = 0.3125$ .

Figure 10 shows that the value of  $\mathcal{R}_0$  changes from less than 1 to greater than 1 as  $p$  increases when  $k$  is greater than a critical value which is 0.3815 under other chosen parameter values. Figure 11 shows that the value of  $\mathcal{R}_0$  changes from greater than 1 to less than 1 as  $p$  increases when  $k$  is smaller than the critical value. Figure 12 shows that the value of  $\mathcal{R}_0$  changes from less than 1 to greater than 1 as  $k$  increases. These indicates that, in various ways, asymptomatic infected individuals including asymptomatic infectious individuals can shift the epidemic dynamics in term of the basic reproduction number. Figure 13 plots  $\mathcal{R}_0$  as a function of  $k$  and  $p$  in the three-dimensional space, where the orange plane is  $\mathcal{R}_0=1$ , the value of  $\mathcal{R}_0$  is greater than 1 above the plane while smaller than 1 below the plane. It shows the combined influence of the two parameters  $p$  and  $k$  on the basic reproduction number.

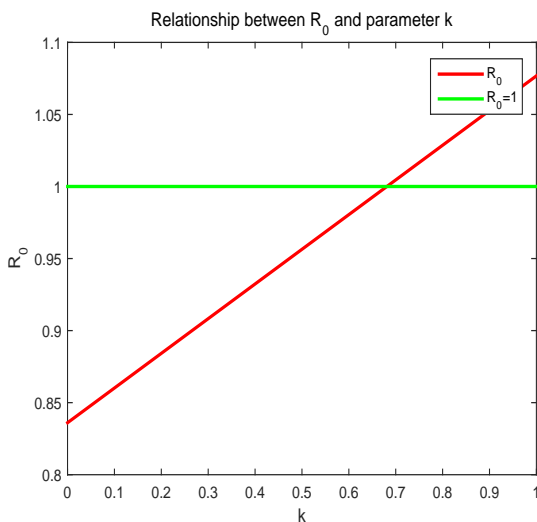




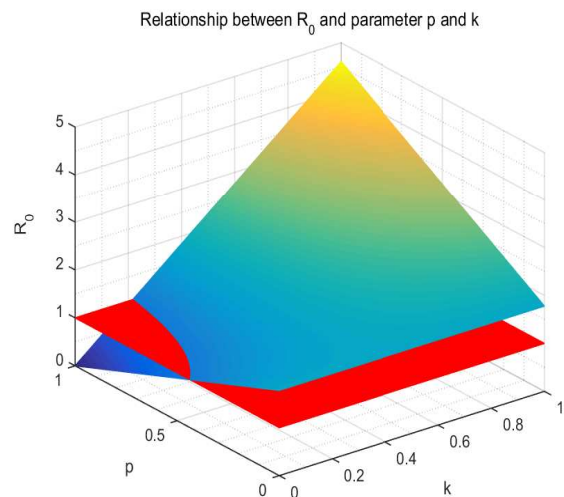
**Figure 10.** Basic reproduction number



**Figure 11.** Basic reproduction number



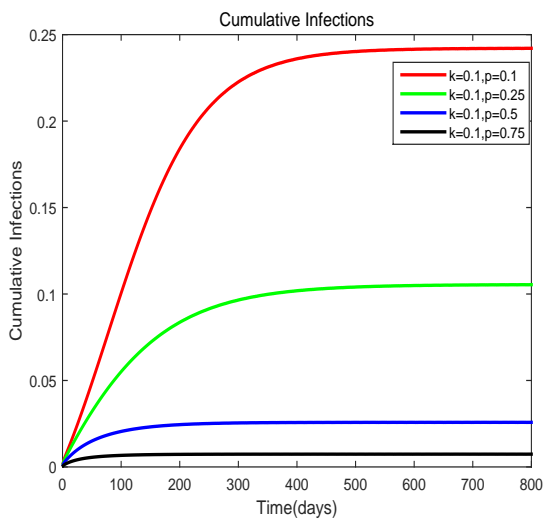
**Figure 12.** Basic reproduction number



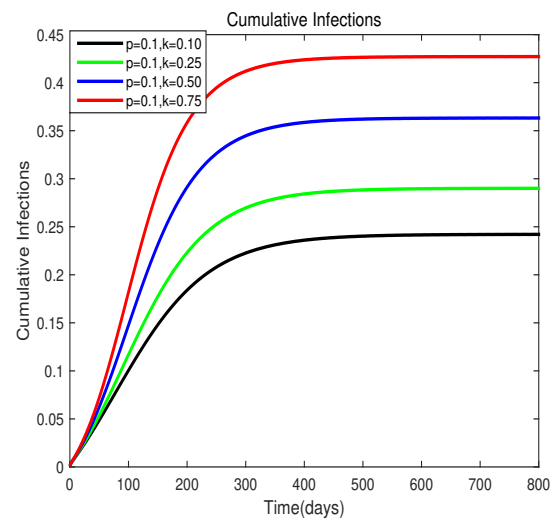
**Figure 13.** Basic reproduction number

In the long-term model, we numerically demonstrate how infected and asymptomatic infected individuals changes over time course with different  $p$  and  $k$  values. Taking  $k = 0.1, p = 0.1, 0.25, 0.50, 0.75$ , the curves of infected and asymptomatic infected individuals over time are calculated and drawn, which are shown in Figures 14 and 16. Taking  $p = 0.1, k = 0.1, 0.25, 0.50, 0.75$ , the curves of infected and asymptomatic infected individuals over time are calculated and drawn, which are shown in Figures 15 and 17.

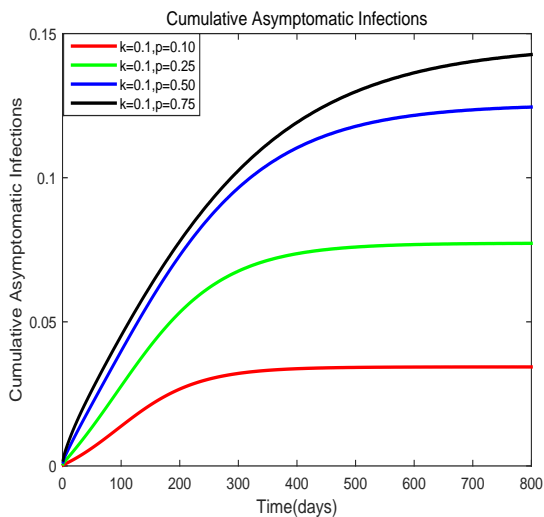
As in the short-term model, if we fix  $p$ , large  $k$  will make both cumulative infections and cumulative asymptomatic infections increasing fast as showed in Figure 15 and Figure 17; if we fix  $k$ , small  $p$  will make cumulative infections increasing fast while large  $p$  will make cumulative asymptomatic infections increasing fast.



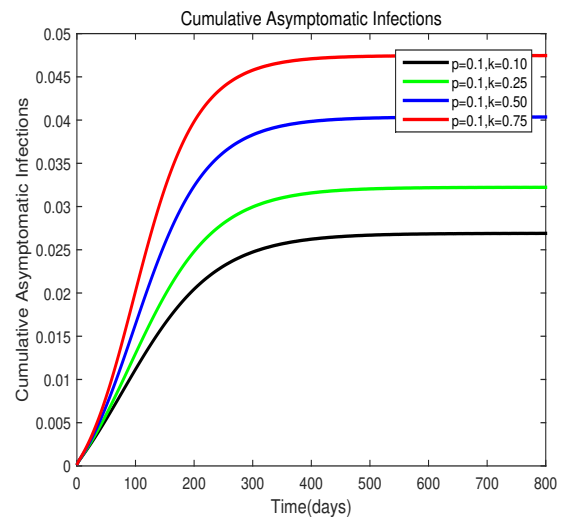
**Figure 14.** Cumulative infections



**Figure 15.** Cumulative infections



**Figure 16.** Asymptomatic infections



**Figure 17.** Asymptomatic infections

## 5. Discussion and conclusions

Asymptomatic transmission is a way to transmit a disease from an individual who does not develop symptoms but has been infected to other susceptible individuals. It has been recognized in many infectious diseases recently. The purpose of this study was to understand how asymptomatic transmissions change the dynamics of epidemics in the frame of traditional mathematical modeling of epidemiology. Based on a short-term model for waterborne infectious diseases, we propose a long-term model for waterborne infectious diseases. Within the traditional modeling framework SEIAWR, we divide the infected compartment into two groups or sub-compartments, infected group and asymptomatic infected group. We attach a proportion or percentage to each of these two group, say  $p$  is for asymptomatic infected group, then  $1 - p$  is for infected group. Individuals in the infected group have symptoms and

are infectious. Individuals in asymptomatic infected group have no symptoms and may be infectious. We then assume there a proportion or percentage  $k$  of the asymptomatic infected group who are infectious, which means they can asymptotically transmit the disease to susceptible individuals. Our study was focusing on these two parameters, and on understanding how these two parameters influence the basic reproduction number and the final size.

The basic reproduction number  $\mathcal{R}_0$  is an increasing function of  $k$  for any value of  $p$ . This is reasonable because of the more asymptomatic infectious individuals in the population, the more possibilities for susceptible individuals to get infected. The parameter  $k$  alone can shift the dynamics of the model.  $\mathcal{R}_0$  can change from a value smaller than 1 to a value greater than 1 as  $k$  increases. However, there is a critical value for  $k$  which characterizes the influence of  $p$ . The basic reproduction number  $\mathcal{R}_0$  is a decreasing function of the parameter  $p$  when parameter  $k$  is smaller than the critical value while  $\mathcal{R}_0$  is an increasing function of  $p$  when  $k$  is greater than the critical value. Of course, in both cases,  $\mathcal{R}_0$  can pass through 1 as  $p$  varies although these dynamic changes are in different ways. We may interpret that, if the infectious portion of the asymptomatic infected group is too low, and even we increase the portion of the asymptomatic infected group, this cannot increase  $\mathcal{R}_0$ , in fact, decreases  $\mathcal{R}_0$ . It should be noticed that the product  $pk$  is the percentage of asymptomatic infectious individuals in the population. The portion of  $pk + (1 - p)$  infected individuals has ability to transmit the disease.  $pk + (1 - p)$  decreases as  $p$  increases. This may explain why there is a critical value for  $k$  which characterizes the influence of  $p$ .

We consider the final size as a function of the basic reproduction number  $\mathcal{R}_0$  in order to understand how two parameters  $p$  and  $k$  influence the final size. If  $\mathcal{R}_0 < 1$ , then the final size is a decreasing function of  $\mathcal{R}_0$ . There exists a critical value of  $\mathcal{R}_0$  which is greater than 1, the final size will be an increasing function of the basic reproduction number  $\mathcal{R}_0$  when  $\mathcal{R}_0$  is greater than this critical value. We combine this with the information about relations between  $\mathcal{R}_0$  and  $k$  and  $p$ . We may infer that there exists a critical value of  $k$  which corresponding to the critical value of  $\mathcal{R}_0$ . When  $k$  is greater than this critical value, the final size  $S(\infty)$  is an increasing function of  $k$ ; when  $k$  is smaller than this critical value, the final size  $S(\infty)$  is a decreasing function of  $k$ . However,  $\mathcal{R}_0$  can increase or decrease as  $p$  varies depending on  $k$ , we may infer that there exists three critical values of  $k$  which divide the interval  $[0, 1]$  into four subintervals. In each subinterval, the final size is a monotonic function of  $p$ . For given data about any outbreak of waterborne infectious disease, we should be able to compute these subintervals and determine how  $p$  influence the final size.

We may conclude as follows. Two parameters  $p$  and  $k$  can characterize influences of asymptomatic transmissions in spreading of infectious diseases. An asymptomatic transmission can shift the dynamics of epidemics. If asymptomatic transmissions are not counted, the basic reproduction number  $\mathcal{R}_0$  will be underestimated while the final size may be overestimated or underestimated.

It is clear there are other ways to incorporate asymptomatic transmission into modeling of epidemiology. For example, asymptomatic transmissions may happen from asymptomatic infectious individuals to both susceptible and infected individuals, and asymptomatic transmissions may happen from asymptomatic infectious individuals to recovered individuals. In addition, it is important to apply our modeling ideas to study current pandemic COVID-19. We plan to carry out some of these ideas in the future.

## 6. Acknowledgements

This work was supported in part by the National Natural Science Foundation of China (Nos. 11871093 and 11901027), the General Program of Science and Technology Development Project of Beijing Municipal Education Commission (No. KM201910016001), the Pyramid Talent Training Project of BUCEA (JDYC20200327), and the BUCEA Post Graduate Innovation Project (PG2020010).

## 7. Conflict of interest

The authors declare there is no conflict of interest in this paper.

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