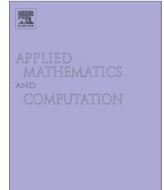




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## Transmission dynamics and optimal control of measles epidemics <sup>☆</sup>



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### ABSTRACT

Based on the mechanism and characteristics of measles transmission, we propose a susceptible-exposed-infectious-recovered (SEIR) measles epidemic model with vaccination and investigate the effect of vaccination in controlling the spread of measles. We obtain two critical threshold values,  $\mu_{c1}$  and  $\mu_{c2}$ , of the vaccine coverage ratio. Measles will be extinct when the vaccination ratio  $\mu > \mu_{c1}$ , endemic when  $\mu_{c2} < \mu < \mu_{c1}$ , and outbreak periodically when  $\mu < \mu_{c2}$ . In addition, we apply the optimal control theory to obtain an optimal vaccination strategy  $\mu^*(t)$  and give some numerical simulations for those theoretical findings. Finally, we use our model to simulate the data of measles cases in the U.S. from 1951 to 1962 and design a control strategy.

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## 1. Introduction

Measles is a highly contagious disease caused by the measles virus. It is spread by coughing and sneezing via close interpersonal contact or direct contact with secretions. Symptoms usually develop 10–12 days after exposure to an infectious person ([28]). There is no specific treatment for measles. Routine measles vaccination for children is the key public health strategy to prevent the disease. Today measles is still a common and often fatal disease in the world.

Data from the U.S. Centers for Disease Control and Prevention (CDC) [10] indicate that there were about 500,000 cases and 500 deaths reported annually in the U.S. before 1963, with epidemic cycles every 2–3 years. Following vaccine licensing in 1963, the incidence of measles decreased by over 98% and the 2–3-year epidemic cycles no longer occurred. But a dramatic increase in cases occurred between 1989 and 1991, during which a total of 55,622 cases was reported (18,193 in 1989; 27,786 in 1990; 9643 in 1991). In fact, the major cause of the resurgence of 1989–1991 was low vaccination coverage. In general, vaccination protects not only those who are vaccinated but also their neighbors. As a result, many others in the community can also be benefited. However, whether or not to vaccinate largely depends on the perceived risk of infection and the vaccination behavior of neighboring individuals [4,5,38]. Hence, the low risk of infection and the free-rider effects maybe be main factors which caused a low measles vaccination coverage rate before 1989. Since the measles vaccination was popularized in 1993, less than 500 cases have been reported per year and fewer than 200 cases annually since 1997. In 2004, a total of 37 cases was reported, mainly U.S. citizens traveling abroad and foreign visitors. Measles was eliminated from U.S. since 2002 (see Fig. 1(A)). In other countries, especially in developing countries, measles vaccination has not been

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extensively popularized. According to the report by the World Health Organization (WHO), in 2012 there were 122,000 measles deaths globally [33]. From available data reported by the Chinese Center for Disease Control and Prevention (CCDC) [11], there were 70,549 measles cases in China in 2004 and 6183 measles cases in 2012 (see Fig. 1(B)). Consequently, it is still very important to model the transmission dynamics of measles and investigate the effect of vaccination on the spread of measles.

Mathematical modeling has become an important and useful tool in studying the spread and control of infection diseases (Anderson and May [1], Zhang and Sun [35], Xia et al. [42], Diekmann and Heesterbeek [14], Zhang et al. [39], Keeling and Rohani [24], Boccaletti et al. [6], Wang and Li [31,32]). Vaccination, is one of the most effective strategies in preventing morbidity and mortality associated with various infectious diseases, has also been included in modeling. Bauch and Earn [4] investigated the impacts of vaccination policy on the level of vaccination coverage and found that voluntary vaccination was unlikely to reach the group-optimal level. Zhang et al. [37] studied the epidemic spreading with voluntary vaccination strategy on both Erdos–Renyi random graphs and Barabasi–Albert scale-free networks and Zhang et al. [36] investigated effects of behavioral response and vaccination policy on epidemic spreading. The transmission dynamics of measles epidemics have been extensively studied. In 1957, Bartlett [2] observed that the number of localized extinctions of measles was related to the population size of the community. In small communities epidemics are often followed by extinction of disease as the chain of transmission breaks down by mass vaccination (Bartlett [3], Bolker and Grenfell [9]). The critical community size above which measles can persist may depend on the spatial structure and connectedness of the regional population (Bolker and Grenfell [7,8], Keeling and Grenfell [23]). Complex dynamics such as oscillations and chaos in measles epidemic models have also attracted attentions of many researchers (Bolker and Grenfell [7], Earn et al. [16], Grenfell [19]) which are believed to be strongly related to the seasonal forcing (Conlan and Grenfell [12]). In 1960, Bartlett [3] gave an estimate of the critical community size for measles for the United States in terms of total population. Since then, various mathematical models have been developed to investigate the transmission dynamics of measles in different countries and regions (Bolker and Grenfell [9], Earn et al. [16], Ferrari et al. [17]).

Valuable information on how to more effectively prevent the outbreaks of measles and accordingly adopt appropriate vaccination policies are very important. In this article, we study the effect of vaccination by mathematical modeling and analysis and determine the level of vaccination coverage that can the most effectively prevent the spread of measles. Note that when an individual becomes infected with the measles virus, the virus begins to multiply within the cells. After an incubation period about 8 to 12 days, early measles symptoms begin. The exposed individual is not contagious during the incubation period and has life time immunity after recovery from the disease. To set up the model, we divide the total population size  $N(t)$  into four distinct categories which are the susceptible, the exposed, the infectious and the recovered, with size denoted by  $S(t)$ ,  $E(t)$ ,  $I(t)$  and  $R(t)$  at time  $t$ , respectively. We assume that the growth of the susceptible population admits a logistic process (Kar and Batabyal [21], Wang et al. [30], Zhang and Chen [40]) in the absence of infection. The incidence rate is bilinear, i.e., proportional to the product of the number of infective individuals and the number of susceptible individuals. The model takes the following form:

$$\begin{cases} \frac{dS}{dt} = rS(1 - bS) - \beta SI - \mu S, \\ \frac{dE}{dt} = \beta SI - (d + \alpha)E, \\ \frac{dI}{dt} = \alpha E - (d + \delta)I, \\ \frac{dR}{dt} = \delta I + \mu S - dR, \end{cases} \quad (1.1)$$

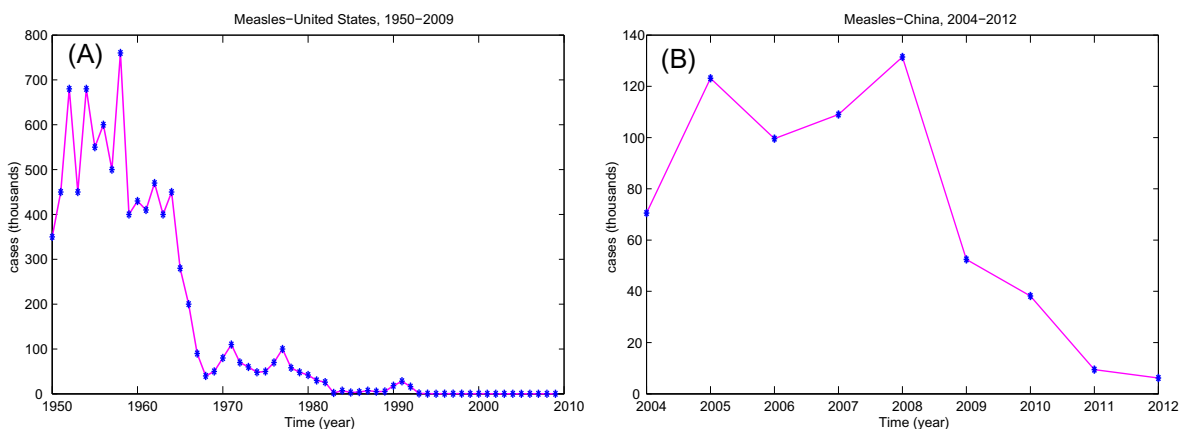


Fig. 1. Reported measles cases in the United States from 1950 to 2010 (CDC [10]) and in China from 2004 to 2012 (CCDC [11]).

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