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Analysis of a Kind of Quitting Smoking Model with Beddington-DeAngelis Function*

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Abstract In this paper, we discuss the dynamics of quitting smoking with Beddington-Deangelis function. Firstly, the basic reproduction number of the model is obtained by establishing the basic reproduction matrix. Then, by using the Routh-Hurwitz criterion and Lyapunov functionals and LaSalle's Invariant Principle and the second additive compound matrix, local and global dynamics of the model are analyzed. Based on the partial rank correlation coefficients (PRCCs), we discuss some biological implications and focus on the impact of some key model parameters. Finally, the numerical simulations show the theoretical analysis more intuitively, and we give some strategies to control the spread of smokers.

Keywords Beddington-DeAngelis function, Quitting smoking model, Local stability, Global stability.

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1. Introduction

Smoking is called "the fifth threat" by the World Health Organization (WHO) (The others are war, famine, plague and pollution). Among smoking-related deaths, chronic lung diseases accounted for 45%, lung cancer 15%, esophageal cancer, gastric cancer, liver cancer, stroke, heart disease and tuberculosis accounted for 40%. If the epidemic trend of smoking patterns is uncontrolled, it is expected that 3 million Chinese will die from tobacco-related diseases by 2050 [1, 2]. On October 27, 2017, World Health Organization International Agency for Research on Cancer lists smoking as a list of primary carcinogens. Epidemiological investigation showed that smoking is one of the important pathogenic factors of lung cancer. Smokers are 13 times more likely to develop lung cancer than non-smokers. About 85% of lung cancer deaths are caused by smoking. Smokers who are exposed to chemical carcinogens such as asbestos, nickel, uranium and arsenic at the same time are at higher risk of lung cancer. Smoking can reduce the activity of natural killer cells,

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thereby impairing the body's surveillance, killing and scavenging functions of tumor cell growth, which further explains that smoking is a high risk factor for multiple cancers. In China, the smoking rate of minors is on the rise, the age at which minors start to smoke is declining, and about 80,000 young people become long-term smokers every day. This situation not only affects the healthy growth of children, but also seriously affects the improvement of the physical fitness of our country as a whole. Therefore, the problem of smoking among minors has attracted more and more attention of the society [3].

Many scholars have done a lot of works on quitting smoking models [4–10]. In [9], Erturk et al. studied the dynamics of a quitting smoking model containing fractional derivatives. The unique positive solution for the fractional order model is presented. In [10], Zeb et al. studied a new quitting smoking model with square root of potential and occasional smokers of model. The local and global stability of the model and its general solution are discussed. Both non-negativity and conservative law for differential equations system are discussed.

Scholars have studied the predator-prey model in depth. Based on the predator-prey model, A.J.Lotka and V.Volterra proposed the famous Lotka-Volterra model [11] $x' = a_1x - b_1xy, y' = -a_2y + b_2xy$. This model is reasonable to some extent, but it ignores the factors of digestive saturation. Holling [12] proposed three functional response functions for different biological types in 1965:

(1) For filter predators, Holling I type functional response function with saturation is given.

$$\Phi(x) = \begin{cases} cx, & x \leqslant x_0, \\ cx_0, & x < x_0. \end{cases}$$

(2) For invertebrates, the Holling II type functional response function $\Phi(x) = \frac{\alpha x}{1+\omega x}$ is given. This functional response function reflects that when the amount of prey increases, the predator's prey will also increase until the number of predators reaches a saturated level.

(3) For vertebrates with complex behavior, the Holling III type functional response function $\Phi(x) = \frac{\alpha x^2}{1+\beta x^2}$ is given. When the number of prey is small, the predator learns to catch. When the number of prey increases, the predation rate increases accordingly. When the food is very full, the degree of hunger decreases and negative acceleration occurs to reach saturation. If the prey has defensive strategies, the predation behavior also belongs to this kind of functional response. The more general form of Holling III type functional response function is $\Phi(x) = \frac{\alpha x^2}{1+\omega x+\beta x^2}$.

In the study of biodynamics, when the amount of food increases to a certain extent, the population growth will be inhibited. In order to describe this inhibiting phenomenon, Andrews gave Holling IV type functional function: $\Phi(x) = \frac{\alpha x}{a+\omega x+\beta x^2}$. Later, Howell simplified it to $\Phi(x) = \frac{\alpha x}{a+x^2}$. It is unexpectedly found that the simplified function is more in line with the experimental data, and it also reduced the difficulty of research. Based on these practical factors and experimental results, some biologists and scholars realized that the predator's role should be added to the functional response function, so they established a new kind of functional response function, namely predator-dependent functional response. In 1975, the functional response function proposed by Beddington and DeAngelis is called Beddington-DeAngelis [13] functional response function, which is more in line with the actual situation. At present, the Beddington-DeAngelis function is used to study the dynamics model of infectious diseases [14, 15]. Based on population dynamics, this