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Mathematical **Biosciences**

Mathematical Biosciences 211 (2008) 333-341

www.elsevier.com/locate/mbs

A schistosomiasis model with mating structure and time delay

Carlos Castillo-Chavez^a, Zhilan Feng^b, Dashun Xu^{c,*}

^a Department of Mathematics and Statistics, Arizona State University, Tempe, AZ 85287, USA

^b Department of Mathematics, Purdue University, West Lafayette, IN 47907, USA

^c Department of Mathematics (Mailcode 4408), Southern Illinois University, 1245 Lincoln Drive, Carbondale, IL 62901, USA

Received 13 June 2007; received in revised form 6 November 2007; accepted 8 November 2007 Available online 22 November 2007

Abstract

A system of homogeneous equations with a time delay is used to model the population dynamics of schistosomes. The model includes the parasite's mating structure, multiple resistant schistosome strains, and biological complexity associated with the parasite's life cycle. Invasion criteria of resistant strains and coexistence threshold conditions are derived. These results are used to explore the impact of drug treatment on resistant strain survival. Numerical simulations indicate that the dynamical behaviors of the current model are not qualitatively different from those derived from an earlier model that ignores the impact of time delays associated with the multiple stages in parasite's life cycle. However, quantitatively the time delays make it more likely for drug-resistant strains to invade in a parasite population.

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Keywords: Homogeneous equations; Time delay; Schistosome mating structure; Multiple strains; Drug resistance

1. Introduction

The expansion of agricultural and water resources that come hand in hand with population and economic growth in developing nations have facilitated the growth and evolution of schistosomiasis. Recent estimates suggest that there are 200 million individuals are infected worldwide and 600 million at risk worldwide [4]. Children are especially vulnerable to infection, which develops into chronic disease if not treated [17]. Current control programs primarily focus on chemotherapy with praziquantel (PZQ), a chemical that reduces morbidity by killing adult worms and halting the deposition of parasite eggs within treated human hosts. Not surprisingly, systematic efforts to control schistosomiasis in human populations through drug treatment establishes an additional selective force that impacts genetic variation within the parasite population. Current evidence supports the view that natural schistosome strains

Corresponding author.

E-mail address: dxu@math.siu.edu (D. Xu).

exhibit varying resistance to treatment with PZQ [5,7,14]. Nevertheless, the subject of schistosome resistance to chemotherapy has just begun to receive attention. In fact, most mathematical models of schistosomiasis do not consider drug resistance of the parasites. In this article we expand our initial efforts to address the role of treatment on the genetic variation of schistosomiasis.

The impact of alternative host treatment rates can affect, as it was shown in earlier work, the range of strains that may be selected by the schistosome population [8,18]. In these modeling efforts, it was assumed that strains with higher resistance levels pay higher costs which reduce transmission. It was also shown that increasing treatment rates favors not only strains with higher levels of resistance (despite the costs) but also strain variability. The model in [8] considered definitive (human) and intermediate (snail) hosts while allowing for an aggregated distribution of parasites in the definitive host population. In order to keep the model manageable, schistosome mating behaviors were ignored in [8]. Previous studies have suggested that mating structure may play an important role in the study of pop-

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