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TITLE

PRAZIQUANTEL EFFECT ON THE GENETIC DIVERSITY OF WILD RODENT-DERIVED SCHISTOSOMA MANSONI IN EXPERIMENTALLY INFECTED MICE

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ABSTRACT

Introduction: Praziquantel (PZQ) is currently the only drug recommended by the World Health Organization (WHO) for treating schistosomiasis, raising concerns about potential resistance. Frequent use of PZQ may reduce the genetic diversity of Schistosoma mansoni, affecting its adaptability and survival. Objective: To evaluate the effects of PZQ treatment and population bottlenecks on the genetic variability of S. mansoni through experimental infections with a naturally derived strain from the rodent Nectomys squamipes. Methods: Experimental infections were conducted in 18 outbred mice individually infected with 120 cercariae via a transcutaneous route through the tail. The mice were divided into three groups of six animals each: 1) the infected control group (IC); 2) the infected group and treated with 3× 150 mg/kg PZQ (IT150), (50% LD); and 3) the infected group and treated with 3× 300 mg/kg PZQ (IT300) (90% LD). The treatment occurred at 50, 51, and 52 days post-exposure, and the mice were necropsied 15 days later. The worms were subsequently washed in saline solution (0.85% NaCl), counted, and stored individually in 70% ethanol at -20°C. We first used the MT-CO1 marker for an initial exploratory analysis according to previous reports that used MT-CO1 in genetic studies of S. mansoni. Microsatellite markers have also been used due to their higher genetic variability, making them more informative for population genetic diversity studies. Results: MT-CO1 analysis revealed two haplotypes differing by one polymorphic site, with one haplotype representing 84.2% of the population. FST demonstrated low genetic differentiation between groups, and AMOVA indicated a greater percentage of variation within the groups than between groups. All seven microsatellite loci studied presented polymorphisms, with 3 to 7 alleles per locus. Praziquantel treatment caused a population bottleneck, reduced genetic variability in both dosage groups: IT150 (RST = 0.14043, p = 0.000) and IT300 (RST= 0.13610, p = 0.005), and eliminated alleles with low initial frequencies. Conclusion: Our analysis of microsatellite markers revealed genetic differentiation and the loss of rare alleles, supporting the hypothesis of a genetic bottleneck caused by PZQ treatment. This study highlights the potential of PZQ treatment to reduce genetic diversity in S. mansoni, underscoring the importance of continuous monitoring of its populations due to the possibility of the emergence of a resistant strain.

KEYWORDS

Schistosomiasis; Cytochrome C Oxidase I; Microsatellites; Bottleneck

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