Hindawi Publishing Corporation
Journal of Biomedicine and Biotechnology
Volume 2010, Article ID 890672, 10 pages
doi:10.1155/2010/890672

Research Article

Mexican *Trypanosoma cruzi T. cruzi* I Strains with Different Degrees of Virulence Induce Diverse Humoral and Cellular Immune Responses in a Murine Experimental Infection Model

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Received 21 August 2009; Revised 18 November 2009; Accepted 29 December 2009

Academic Editor: Abhay R. Satoskar

It is has been shown that the majority of *T. cruzi* strains isolated from Mexico belong to the *T. cruzi* I (TCI). The immune response produced in response to Mexican *T. cruzi* I strains has not been well characterized. In this study, two Mexican *T. cruzi* I strains were used to infect Balb/c mice. The Queretaro (TBAR/MX/0000/Queretaro)(Qro) strain resulted in 100% mortality. In contrast, no mortality was observed in mice infected with the Ninoa (MHOM/MX/1994/Ninoa) strain. Both strains produced extended lymphocyte infiltrates in cardiac tissue. Ninoa infection induced a diverse humoral response with a higher variety of immunoglobulin isotypes than were found in Qro-infected mice. Also, a stronger inflammatory TH1 response, represented by IL-12p40, IFNγ, RANTES, MIG, MIP-1β, and MCP-1 production was observed in Qro-infected mice when compared with Ninoa-infected mice. We propose that an exacerbated TH1 immune response is a likely cause of pathological damage observed in cardiac tissue and the primary cause of death in Qro-infected mice.

1. Introduction

Chagas' disease is a major endemic disease caused by the protozoan *Trypanosoma cruzi*. This parasitic disease is widely distributed throughout Latin America, affecting 18 million people [1]. In the past, it was believed that Chagas' disease was very rare in the northern part of America, including Mexico, as few human disease cases were reported. More recently, however, it has been estimated that there are as many as two million infected individuals and more than 72 000 cases in Mexico and Central America [2]. Even though these figures are only estimates, other field studies support the notion that the prevalence of *T. cruzi* infection might be higher in some regions of the country than previously thought [3–5]. *T. cruzi* has also been repeatedly found infecting insects and mammals in Mexico and the United States of America [6–8].

T. cruzi strains have been divided into six discrete typing units (DTUs) according to their genetic background. These groups are designed the *T. cruzi* I to VI [9]. The geographical distribution of these groups indicate that *T. cruzi* II to VI are the main causal agent of Chagas' disease in the southern parts of South America, with *T. cruzi* I only present in the sylvatic cycle [9–11]. In contrast, *T. cruzi* I has been reported as the primary parasite present in human cases in Colombia, Venezuela, and Central America [12–14]. In Mexico, most of the *T. cruzi* strains that have been genetically analyzed to date belong to the *T. cruzi* I group [15–17]. We have reported that this Mexican *T. cruzi* I strain possesses different biological characteristics such as growth rates, metaciclogenesis, and infectivity in vitro [15]. However, the pathology and immune response that these strains can induce has largely gone unstudied.

Knowledge of the pathology and immune response to *T. cruzi* infection has been beneficiated by data obtained from murine models. These models have shown that the innate and adaptive immune responses play an important role in parasite control, depending on the combined action