

GENE EXPRESSION OF TOLL-LIKE 2 AND 4 RECEPTORS AND T $\gamma\delta$ WC1 CELLS RECEPTORS RELATED TO THE LEVELS OF INFECTION IN CATTLES NATURALLY INFECTED BY AGENTS OF TICK-BORNE DISEASES

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ABSTRACT

Bovine babesiosis is an infectious disease caused by protozoa *Babesia bovis* and *B. bigemina*, intra-erythrocyte parasites that produce intravascular hemolytic anemia. *Anaplasma marginale* is a bacterium that also causes extensive hemolysis in infected animals. These infections accompany the spread of the tick *Rhipicephalus microplus* and, together, form the complex of diseases recognized for causing severe damage to cattle producers in Brazil. Gene expression studies to evaluate the immune response are an alternative to determine the genetic basis for selecting more resistant cattle to these diseases. Thus, this study aimed to evaluate the variation in gene expression of toll-like receptors 2 and 4 (TRL 2 and TRL 4), T cells $\gamma\delta$ WC1 and the level of hemoparasite infection in blood samples from 46 Canchim calves, from birth to four and a half months of age, totaling ten collections. Infection levels were measured separately for each hemoparasite using absolute quantification (qPCR). The expression of cellular receptor genes was evaluated using Real-Time Quantitative Reverse Transcription (qRT-PCR). The data obtained showed variations in gene expression over time. Associations were also found between these results and the degree of infection by hemoparasites, which showed significant effects of collection and precedence classes. Thus, this study presents the behavior of these genes against infections and suggests that, despite the oscillations of expression throughout the study, there is the participation of these genes during the confrontation to infections in the studied period. We conclude that this work is an initial basis for studies that enable the use of TLR2, TLR4, and WC1 genes as genetic markers of immune response in cattle infected by tick-borne disease agents.

KEYWORDS: tick-borne diseases; gene expression; immune response

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