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Opinion Article

Prevention of Chagas Disease in Mouse Models by *Trypanosoma* Vaccine Strain

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DESCRIPTION

Trypanosoma cruzi is the causative agent of Chagas disease and there is no preventive vaccine. Cyclophilin 19 is a secretory cistranspeptidyl isomerase expressed at all life stages of *Trypanosoma cruzi*, causing inactivation of insecticidal insect peptides and parasite transformation at the insect stage, and intracellular amastigote is involved in the production of ROS, which promotes the growth of parasites, we generated parasite knockout mutants of Cyp19 that do not replicate in cell culture or mice. This shows that the absence of Cyp19 is important for infectivity. *Trypanosoma cruzi*, a protozoan parasite, is the causative agent of Chagas disease endemic to Mexico and Latin America. People living in the southern United States, especially Arizona, New Mexico, and Texas, also have an increased risk of getting sick. The disease is transmitted in multiple ways, chiefly by the feeding of hematophagous triatomine insects on mammalian hosts. Humans are an incidental hosts and those living rural areas are at greatest risk where poor housing conditions lead to the entrance and feeding of triatomines into houses. Feeding leads to the deposition of fecal material containing infective metacyclic trypomastigotes parasitic forms that end the contamination of mucosae around the feeding sites. Metacyclics bind to and invade host cells wherein they transform into and multiply in the host cell cytosol as intracellular amastigotes. Amastigotes transform thereafter into motile trypomastigotes that exit host cells, which circulate through the body and further infect cells in a variety of target organs. Chronic infections of organs, especially cardiomyocytes and gastrointestinal smooth muscle, cause sequelae of chronic Chagas disease.

The genomic sequence of *T. cruzi* revealed that cyclophilin 19 is encoded by a single copy gene. To determine the role of cyclophilin 19 in the pathogenicity and etiology of parasites, a double allelic homologous recombinant gene knockout strategy was used to remove the cyclophilin 19 gene from the parasite. Cyclophilin 19 depleted double knockout parasite variants were unable to infect mice *in vitro* in host cells or *in vivo*. Repeated infection of mice with these variants increased parasite-specific T and B cell responses and prevented 100% death from acute Chagas disease in mice. This suggests that this is an effective vaccine strain to prevent Chagas disease. Double Knockout (DKO) mutant parasite strains with reduced expression of Cyp19 are viable in culture, but have a reduced growth rate, a defective ability to reach peak densities of wild-type parasites, and replication as epimastigotes. The mutant strain rapidly differentiates into metacyclic tripomastigote, further suggesting a defect in normal cell cycle regulation. Since the metacyclic form is not replicated, we assume that the rapid conversion of epimastigote to the metacyclic form in the early stages of cell culture is the reason why these cells cannot reach high densities. Replication of a small number or epimastigote prior to differentiation into metacyclic morphology is a key to their continued survival in culture. The DKO metacycle is resistant to complement-mediated killing by normal human serum and is morphologically similar to that of wild-type parasites, with reduced Cyp19 expression affecting these properties. It suggests that there isn't. When used to infect mammalian host cells, metacyclic parasites enter the cell at a reduced rate, forming a sparse amastigote-like structure that degenerates and disappears from the host cell.

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