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Pathogen-induced proapoptotic phenotype and high CD95 (Fas) expression accompany a suboptimal CD8+ T-cell response: Reversal by adenoviral vaccine

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MHC class Ia-restricted CD8+ T cells are important mediators of the adaptive immune response against infections caused by intracellular microorganisms. Whereas antigen-specific effector CD8+ T cells can clear infection caused by intracellular pathogens, in some circumstances, the immune response is suboptimal and the microorganisms survive, causing host death or chronic infection. Here, we explored the cellular and molecular mechanisms that could explain why CD8+ T cell mediated immunity during infection with the human protozoan parasite Trypanosoma cruzi is not optimal. For that purpose, we compared the CD8+ T-cell mediated immune responses in mice infected with T. cruzi or vaccinated with a recombinant adenovirus expressing an immunodominant parasite antigen. Several functional and phenotypic characteristics of specific CD8+ T cells overlapped. Among few exceptions was an accelerated expansion of the immune response in adenoviral vaccinated mice when compared to infected ones. Also, there was an upregulated expression of the apoptotic-signaling receptor CD95 on the surface of specific T cells from infected mice, which was not observed in the case of adenoviral-vaccinated mice. Most importantly, adenoviral vaccine provided at the time of infection significantly reduced the upregulation of CD95 expression and the proapoptotic phenotype of pathogenspecific CD8+ cells expanded during infection. In parallel, infected adenovirusvaccinated mice had a stronger CD8 T-cell mediated immune response and survived an otherwise lethal infection. We concluded that a suboptimal CD8+ T-cell response is associated with an upregulation of CD95 expression and a proapoptotic phenotype. Both can be reprogrammed by adenoviral vaccination.

Biography

Dr. Vasconcelos received his PhD at the Federal University of Sao Paulo on 2004, Brazil, where he developed and tested genetic vaccines against experimental infection with the human protozoan parasite Trypanosoma cruzi. He went to Saint Louis University-MO USA, where conducted his first PostDoctorade at Department of infectious diseases. During this time he also worked on developing vaccines against this parasite. Back to Brazil, continued to study immune mechanisms generated by adenivírus recombinant vaccine containing genes of different forms of the parasite. He is currently a postdoc senior in the department of microbiology, immunology and parasitology of the Federal University of Sao Paulo.

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