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Auto-presentation of Staphylococcal enterotoxin A by mouse CD4+ T cells without a safety mechanism requires cellular interaction between two different types of cells

Reuven Rasooly

United States Department of Agriculture, USA

The currently accepted model for superantigen (SAg) induced T cell activation suggests that SAg, without being processed, cross link both MHC class II, from Antigen Presenting Cells (APC), and V-beta, from T-cell receptor (TCR), initiating nonspecific T-cell activation. This T-cell proliferation induces a massive cytokine release associated with several human diseases. It is thought that murine CD4+ T cells do not express

MHC class-II molecules. However, we discovered that a subtype of mouse naïve CD4+ T cells expresses MHC class II on their cell surface and that these CD4+ T cells can perform the role of both APC and T cells, able to present Staphylococcal enterotoxin A (SEA) to itself or neighboring CD4+ T cells via MHC class II, thus inducing massive CD4+ T cell proliferation. Treatment with neutralizing anti MHC class II antibody inhibits this CD4+ T cell proliferation response. The fact that murine CD4+ T cells express MHC class II offers new insight about SAg activity. Based on our findings, we propose revising and extending previous models for SAg induced T cell activation, altering previous models of MHC class II restriction of T cell responses to SEA as well as the requirement for SAg processing.

Biography

In the last 6 years Reuven Rasooly has been working as a research chemist for the U.S. Department of Agriculture focusing on developing sensitive alternative assay to the *in vivo* bioassay for toxin detection in food matrices. As a post doctoral researcher he studied the effects of Vitamin A on T helper-cell development. His Ph.D. thesis research was developing a vaccine against trypanosome microtubule associated protein for prevention of African sleeping sickness. His M.Sc. research was determining the agent and vector for transmission of citrus stubborn disease. He has published more than 30 scientific papers.

reuven.rasooly@ars.usda.gov