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Preservation of Tregs during CD28/CD154 blockade of *ex vivo* antigen-specific activation of diabetogenic T cells

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Developing means to delete or inactivate beta cell-specific T cells while enhancing regulatory T cells is a promising conceptual approach for preventing or reversing Type 1 diabetes (T1D). To better understand the mechanism of tolerance-inducing agents, we evaluated the effect of CD28 and CD154 blockade on both beta cell-specific effector and regulatory T cell responses in a model of autoimmune diabetes.

Diabetes transferred by lymphocytes isolated from BDC2.5.NOD mice could be prevented if cells were antigen-activated in vitro in the presence of CTLA4Ig and antiCD154. Even in the presence of costimulation blockade both non-regulatory (NRT, FoxP3-and regulatory CD4+ T cells (Tregs; FoxP3+) went through many processes occurring with activation (i.e. CD25 upregulation, cell division, and accumulation). Costimulation blockade appeared to selectively suppress activation of NRT versus Tregs. IL2 production was impaired by costimulation blockade, and supplementing exogenous IL2 reversed its protection.

These findings may modify the perception that Tregs are minimally responsive to antigen and require traditional costimulatory signals for activation and function. This study supports the concept that in certain situations there are different activation sequences and requirements for effector and regulatory T cells which can be manipulated to alter the outcome of unwanted immune responses like T1D.

Biography

Mark R. Rigby graduated with a B.S. from Duke University and an M.D. and Ph.D. at the University of Massachusetts, conducting a thesis with Aldo Rossini. Then, at Johns Hopkins he competed pediatric residency and fellowship in pediatric critical care. Then, as faculty at Emory University he was a pediatric intensivists and on faculty at the Emory Transplant Center. He is presently Academic Chief of Pediatric Critical care, the Director of Translations Research Center in Pediatric, conducts basic T1DM research in the Well Diabetes Center, and is the Protocol Chair of the NIH supported intervention trial, the T1DAL study.

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