

10th ANNUAL DEPARTMENT OF PATHOLOGY YOUNG INVESTIGATORS' DAY
POSTER SESSION

POSTER #

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(for Admin. use)

Thursday, April 17th, 2008
TURNER CONCOURSE
REGISTRATION FORM

Applicant's Name: _____ Geral C. Baldeviano _____ Degree: _____ Ms _____

Applicant's Division: _____ Department of Pathology, Division of Immunology _____

Faculty Preceptor: _____ Noel R. Rose M.D, Ph.D. _____
(Must hold a primary appointment in Pathology)

Appointment Category: _____ House Staff _____ Clin Fellow _____ Research Fellow
_____ Medical Student Graduate Student (Program: MMI)

Register for: _____ Clinical Research _____ Translational Research Basic Research

Full Poster Title **Th17 Differentiation by Dendritic Cells is Dependent on IL-13**

Where has the work been presented?

Meeting Name _____ AAI 2008 _____

Meeting Date _____ April 4- 9 _____

Not Previously Presented _____

Where is this work being published? _____

Journal Name, Volume, Page, Date _____

In Preparation (Y/N) - Where? _____ Y (Journal of Immunology) _____

Author(s) (First & Last) _____ Christian Baldeviano and Daniela Cihakova _____

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***INCLUDE A ONE-PAGE ABSTRACT (including title and all authors) OF THE WORK YOU WILL BE PRESENTING**

**E-mail COMPLETED Registration form and abstract to:
Stacey Morgan (smorgan9@jhmi.edu) on or before
Friday, March 14th, 2008**

If you have questions or problems regarding your submission, please contact Stacey Morgan via e-mail (smorgan9@jhmi.edu)

(Paste abstract here)

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Th17 Differentiation by Dendritic Cells is Dependent on IL-13

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Interleukin-17 (IL-17) drives pathogenesis of a number of autoimmune disease models, including experimental autoimmune myocarditis (EAM). We have previously reported that disruption of IL-13 results in uniquely severe EAM. To assess whether IL-17 drives severe pathogenesis in IL-13 deficient mice (IL-13 KO), we determined the levels of IL-17. Interestingly, IL-17 was significantly decreased in the spleen, heart (R&D System Elisa kit), and draining lymph nodes (by RT-PCR) of IL-13 KO mice at day 21 of EAM. Naïve IL-13 deficient CD4⁺ T cells differentiated into Th-17 cells and CD4⁺ FOXP3⁺ T regulatory cells similar to naïve WT BALB/c T cells. However, bone marrow derived dendritic cells (BMDCs) from IL-13KO mice were deficient in driving OVA₃₂₃₋₃₃₉-specific Th-17 differentiation from naïve DO11.10 CD4⁺ T cells. This defect was partially restored when recombinant IL-13 was added to the bone marrow culture. IL-13 KO BMDCs expressed less MHCII, CD80, and CD40 upon maturation with LPS, curdlan, or α CD40 mAb; as well as produced less pro-inflammatory cytokines IL-6 and TNF α after maturation with LPS. Levels of costimulatory molecules ICOSL and OX40L were also dysregulated in IL-13KO BMDCs. Thus, IL-13 is required for proper DCs maturation and cytokine production, which determines DCs ability to induce Th17 phenotype. In summary, a decrease of IL-17 in hearts of IL-13 KO mice during EAM represents a general defect of IL-13 KO DCs in inducing Th-17.

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