Fatemeh Talebian
PhD Candidate

CD200-CD200R Interaction in Tumor Immunity

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Ross Auditorium, Room H1213
10 a.m.
VITA

September 1979. . . . . . . . . . . . Born in Kansas City, Kansas

Bachelor’s of Art: Major 1: Biochemistry
Major 2: English Literature

1998-2002: Tehran University, College of Sciences, Tehran Iran
Department of Cellular and Molecular Biology;
Bachelor’s of Science: Cellular & Molecular Biology

2003-2006: Tehran University of Medical Sciences(TUMS), Iran
Department of Immunology
Master’s of Science: Medical Immunology
No.1 in MSc Entrance Exam

2007-2012: Ohio State University, Columbus Ohio USA
Department of Pathology
Doctor of Philosophy: Integrated Biomedical Sciences

COMMITTEE MEMBERS

Professor Xue-Feng Bai, Advisor
Professor Amy Lovett-Racke
Professor Ramish Ganju
Professor Sujit Basu
FUTURE PLANS

After my graduation I will be traveling to Iran where I will be part of the editorial team of two scientific Journals. I am also looking into an adjunct professor position at the Medical School at Tehran University. I will return to the states for my post doctoral position in 1-2 years.

ABSTRACT

CD200 is a member of the Ig super family (IgSF) of proteins, expressed on cell surface of lymphoid cells and some lineages of cancer cells such as melanoma cells. CD200 functions through engaging its specific receptor, CD200R. CD200R has an inhibitory intracellular NPXY signaling motif and is mainly detected on cells of the myeloid lineage. CD200-CD200R interaction inhibits the function(s) of myeloid cells. Myeloid cells are the first cells recruited by tumors and are essential in the regulation of tumor initiation, establishment, progression and metastasis. Tumor associated myeloid cells (TAMCs) are also known to inhibit activation and effector function of T cells. Therefore, we hypothesized that CD200-CD200R interaction affects tumor formation, metastasis and tumor immunity via inhibiting TAMC functions.

The goals of this dissertation thesis are three fold:

1) To investigate CD200/CD200R expression in the tumor compartments
In the tumor microenvironment, myeloid cells express high levels of both CD200 and CD200R. Tumor infiltrating T cells express high levels of CD200, while their expression of CD200R is barely detectable.

2) To determine the role of CD200-CD200R interaction in tumor formation and metastasis
Gr1⁺ cells dramatically inhibited tumor foci formation in the lungs. Treatment with tumor antigen specific CD4 and CD8 T cells or their combination yielded a survival advantage for CD200 positive tumor bearing mice over mice bearing CD200-negative tumors. Analysis of microarray data from human cancer patients revealed that patients with CD200hi tumors have better prognosis and longer survival time.

3) To determine if targeting CD200R is a feasible approach for cancer immunotherapy
CD200-positive melanoma cells grow and metastasize progressively in CD200R-/- mice but not in WT mice. Stimulation of CD200R with an agonistic antibody dramatically inhibited lung metastasis of CD200-negative melanoma. Use of monoclonal
agonistic CD200R antibodies inhibited tumor growth and improved survival time in established tumor models.

**Conclusion**

Taken together, we have found that in the tumor microenvironment there are highly significant expressions of CD200 and CD200R. CD200-CD200R interaction is broadly involved in regulating tumor formation, metastasis and tumor immunity. Targeting CD200R may be a novel approach for the immunotherapy of human cancer.

**Publications**

- J.Q. Liu, Z. Liu, X. Zhang, Y. Shi, **F. Talebian** et al. Enhanced Th17 and Treg responses in EBI3-deficient mice lead to marginally enhanced development of autoimmune encephalomyelitis *Journal of Immunology* 2012 In Press.