BIOGRAPHICAL SKETCH

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NAME: Tolunay Beker Aydemir

eRA COMMONS USER NAME (credential, e.g., agency login): BEKERAYDEMIRT

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Ankara University Science Faculty, Ankara, Turkey	B.S.	05/1999	Biology
Ankara University School of Medicine, Ankara, Turkey	M.S.	05/2004	Molecular Hepatology
University of Florida, College of Medicine, Gainesville, FL	Ph.D.	08/2011	Biochemistry and Molecular Biology
University of Florida, Center for Nutritional Sciences, Gainesville, FL	Postdoctoral	07/2016	Nutritional Genomics Laboratory

A. Personal Statement

The Aydemir group overall research interests centered on how transporter-mediated zinc and manganese mobilization function to regulate diverse cellular processes in health and disease. ZIP14/SLC39A14 is a ZIP family transmembrane protein that regulates intracellular levels of zinc (Zn), Mn, and iron (Fe). Although occupational and environmental Mn exposures are known sources of Mn toxicity, genetic mutations in metal transporters, including ZIP14, also cause Mn-induced parkinsonism. Thus, our most recent research project specifically focuses on understanding the mechanisms of both: ZIP14-mediated Mn detoxification, and how ZIP14 dysfunction leads to neuroinflammation and neurodegeneration via Mn overload.

a. Aydemir TB, Kim MH, Kim J, Colon-Perez LM, Banan G, Mareci TH, Febo M Cousins RJ. 2017. Metal transporter ZIP14 (SLC39A14) deletion in mice increases manganese deposition and produces neurotoxic signatures and diminished motor activity. *J Neurosci* 37(25):5996–6006.

B. Positions and Honors

Positions and Employment

2018-Present	Assistant Professor of Molecular Nutrition, Division of Nutritional Sciences, Cornell University, Ithaca, New York
2016-2018	Assistant Research Professor, Center for Nutritional Sciences, Food Science and Human Nutrition Department, University of Florida, Gainesville, Florida
2014-2016	Senior Postdoctoral Associate, Nutritional Genomics Laboratory, Center for Nutritional Sciences, Food Science and Human Nutrition Department, University of Florida, Gainesville, Florida
2011-2014	Postdoctoral Associate, Nutritional Genomics Laboratory, Center for Nutritional Sciences, University of Florida, Gainesville, Florida
2006-2011	Graduate Assistant, University of Florida College of Medicine, Biochemistry and Molecular Biology, Gainesville Florida/ Nutritional Genomics Laboratory, Center for Nutritional Sciences, University of Florida, Gainesville, Florida

2004-2006	Biological Scientist, Nutritional Genomics Laboratory, Center for Nutritional Sciences, University of Florida, Gainesville, Florida.
2002-2004	Training student, Laboratory of Biochemical Pharmacology, Emory University, VA Medical Center, Atlanta, Georgia.
2000-2004	Research Fellow, Institute of Hepatology, Ankara University School of Medicine, Ankara, Turkey.
1997-2000	Volunteer, Institute of Hepatology, Ankara University School of Medicine, Ankara, Turkey.
July-Oct 1997	Visiting Scientist; Medical Biology Department, Ankara University School of Medicine, Ankara, Turkey.

Professional Memberships

2010-	Member, American Society for Nutrition (ASN)
2010-	Member, American Society for Biochemistry and Molecular Biology (ASBMB)
2011-	Member, Sigma Xi

C. Contributions to Science

1. My early publications mainly focused on investigating the role of zinc in immune cells and in searching for a biological marker of zinc responsiveness. Zinc deficiency has dramatic implications for immune function and is of critical importance to human health and disease prevention. Therefore, zinc supplementation became a powerful therapeutic tool in clinical research. Zinc supplementation, however, may not benefit the host in every case since the effectiveness of zinc supplementation depends on both the patients' initial zinc status and the stage of the disease. Addressing these issues, my publications revealed that modest supplementation of zinc is adequate to attain significant biological effects in immune cells. Assessment of zinc status could be done by measurement of zinc-responsive genes from blood as well as from dried blood spots (DBS), which have the advantage of being more easily used as a screening technique for larger populations, a critical factor in medicine. These findings contributed to the understanding of zinc in immune cells and serving as a launching pad for subsequent research.

    a. Liuzzi JP, Lichten LA, Rivera S, Blanchard RK, Aydemir TB, Knutson MD, Ganz T, Cousins RJ. 2005. Interleukin-6 regulates the zinc transporter Zip14 in liver and contributes to the hypozincemia of the acute-phase response. *Proc Natl Acad Sci U S A* 102:6843–8.
    b. Aydemir TB, Blanchard RK, Cousins RJ. 2006. Zinc supplementation of young men alters metallothionein, zinc transporter, and cytokine gene expression in leukocyte populations. *Proc Natl Acad Sci U S A* 103:1699–704.

2. In these publications, the main finding was the necessity of zinc as a signaling molecule in two different biological processes; T cell activation and hepatocyte proliferation. T cell activation is a critical step for immune host defense as is hepatocyte proliferation to liver regeneration. We found that In both cases, zinc acts as a signaling molecule to inhibit the activity of phosphatase enzymes, resulting in modulation of cellular responses. These findings contributed to the development of new therapeutic strategies for improving immune functioning and enhancing liver regeneration.
    a. Aydemir TB, Liuzzi JP, McClellan S, Cousins RJ. 2009. Zinc transporter ZIP8 (SLC39A8) and zinc influence IFN-gamma expression in activated human T cells. *J Leukoc Biol* 86(2):337–48.
    b. Aydemir TB, Sitren HS, Cousins RJ. 2012. The Zinc Transporter Zip14 influences c-Met phosphorylation and hepatocyte proliferation during liver regeneration in mice. *Gastroenterology* 142(7):1536–46.

3. These papers describe the discovery that deletion of zinc transporter *Zip14* gene in mice produces a phenotype resembling diet-induced diabetes (type 2) and obesity, including impaired intestinal barrier function with chronic systemic inflammation, hyperinsulinemia, increased body fat, and insulin resistance in adipose tissue. Unexpectedly, the liver was insulin-sensitive, indicating a tissue-specific differential role for zinc and Zip14 in metabolic tissues.

a. Aydemir TB, Chang SM, Guthrie GJ, Maki AB, Ryu M-S, Robert J. Cousins. 2012 Zinc transporter ZIP14 functions in hepatic zinc, iron and glucose homeostasis during the innate immune response (endotoxemia). *PLoS ONE* 7(10): e48679. doi:10.1371/journal.pone
b. Guthrie GJ, Aydemir TB, Troche C, Martin AB, Chang SM, Cousins RJ. 2015. Influence of ZIP14 (slc39A14) on intestinal zinc processing and barrier function. *Am J Physiol Gastrointest Liver Physiol,* 308(3):G171–8.
c. Troche C, Aydemir TB, Cousins RJ. 2016. Zinc transporter Slc39a14 regulates inflammatory signaling associated with hypertrophic adiposity. *Am J Physiol Endocrinol Metab* 310(4): E258–68
d. Aydemir TB, Troche C, Kim MH, Cousins RJ. 2016. Hepatic ZIP14-mediated Zinc Transport Contributes to Endosomal Insulin Receptor Trafficking and Glucose Metabolism. *J Biol Chem* 291(46):23939–51.

4. My current research has expanded into a new field involving manganese metabolism and manganese-induced parkinsonism. We found that ablation of the *Zip14* gene from the mouse genome caused excessive manganese accumulation in the brain (manganism) with motor dysfunction.
   a. Aydemir TB, Kim MH, Kim J, Colon-Perez LM, Banan G, Mareci TH, Febo M Cousins RJ. 2017. Metal transporter ZIP14 (SLC39A14) deletion in mice increases manganese deposition and produces neurotoxic signatures and diminished motor activity. *J Neurosci* 37(25):5996–6006.

Complete List of Published Work in My Bibliography:
https://www.ncbi.nlm.nih.gov/sites/myncbi/11Zz9zUku5Uth/bibliography/57428595/public/?sort=date&direction=ascending

D. Additional Information: Research Support and/or Scholastic Performance

Ongoing Research Support

None

Completed Research Support

None