Spring 2026

Biochemistry and Molecular Biology Brown Bag Series

Diksha Singh

"Zinc: A small ion, with big responsibilities"

Tuesday, March 10, 2026

11:00 AM

103 Biological Sciences Building

Lab: Clintoria Williams, Ph.D.

Boonshoft School of Medicine
WRIGHT STATE UNIVERSITY

WRIGHT STATE UNIVERSITY

https://science-math.wright.edu/biochemistry-and-molecular-biology

Abstract

Background: Renal inflammation is a crucial factor in the progression of Chronic Kidney Disease (CKD), significantly contributing to kidney damage and dysfunction. Individuals with CKD often exhibit low serum zinc (Zn) levels. Our preclinical models demonstrated that Zn deficiency alone is sufficient to promote kidney damage. Since renal inflammation is implicated in kidney damage, this project seeks to investigate if Zn deficiency also promotes renal inflammation.

Experimental Design: To investigate if disrupted Zn homeostasis induces renal inflammation, mice were administered a Zn adequate- or Zn deficient-diet for six weeks. Kidney damage was examined by assessing glomerular (morphological and urinary protein) and tubular (urinary KIM-1) changes. Renal inflammation was evaluated by measuring the expression of a proinflammatory cytokine (urinary IL-6) and presence of renal macrophages (CD68+, F4/80+, and CD206+).

Results: Compared to Zn adequate mice, glomerular histological changes were observed in Zn deficient mice, including loss of endothelial cells, increased urinary space, and mesangial cell expansion. Also, KIM-1 and IL-6 expressions were elevated with Zn deficiency. Consistently, increased abundance of renal macrophages was observed in Zn deficient mice.

Summary: These data demonstrate that Zn deficiency-induced renal damage is accompanied by renal inflammation. Taken together, these novel findings highlight renal inflammation as a possible driving factor in the progressive kidney damage and dysfunction associated with Zn deficiency.

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Maitreyee Chavan

"Allelic Variation in the hs1.2 Enhancer Modulates AhR-Mediated Suppression of Human Antibody Secretion"

Tuesday, March 10, 2026

11:00 AM

103 Biological Sciences Building

Lab: Courtney Sulentic, Ph.D.

Boonshoft School of Medicine
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Abstract

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), a high-affinity ligand of the aryl hydrocarbon receptor (AhR), is a well-established immunotoxicant that alters antibody production in B cells. Previous studies in our laboratory using the CL-01 human Burkitt lymphoma B-cell line showed that TCDD inhibits stimulation-induced IgG secretion, leaves IgM largely resistant, and exerts a stimulation-dependent inhibition on IgA. However, how genetic variation in the immunoglobulin heavy chain (IgH), which encodes each antibody isotype (i.e. IgM, IgG, IgA), these effects remain unknown. The hs1.2 enhancer within a large regulatory region at the 3′ end of the *IgH* gene (3′*IgH*RR) exhibits allelic variations that have been linked to altered antibody expression and immune-related disorders.

This project proposes to investigate if genetic variations in the hs1.2 enhancer modulate the response of human B cells to TCDD. Using a panel of cell lines and CRISPR/Cas9-edited clones exhibiting different hs1.2 alleles, the effects of TCDD on IgM, IgG, and IgA secretion will be evaluated. Antibody secretion will be measured by ELISA, and corresponding *IgH* constant region transcripts will be quantified by RT-qPCR.

The outcomes of this work are expected to clarify whether hs1.2 enhancer variants result in differential sensitivity of antibody isotype production to environmental AhR ligands. Establishing this connection could inform how genetic variation contributes to differential immune response and may help assess individual susceptibility to immunotoxic exposures.