Subject: Dissertation Defense - Lama Elzohary, PhD in Biosciences
Date: Monday, April 6, 2026 at 11:22:10 AM Eastern Daylight Time
From: SSB Faculty List on behalf of Diane St. Germain
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Dissertation Defense Announcement
To: The George Mason University Community

Candidate: Lama Elzohary

Program: PhD in Biosciences

Date: April 20, 2026

Time: 12:00 PM Eastern Time (US and Canada)

Location: Via Zoom
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Committee Chair: Dr. Ancha Baranova

Committee Members: Dr. Alessandra Luchini, Dr. Hongbao Cao, Dr. Vikas Chandhoke

Title: Network Analysis of Genetic Susceptibility in Pulmonary Diseases

Abstract:

Pulmonary diseases are characterized by chronic inflammation, immune dysregulation, and progressive loss of lung function. Although environmental exposures contribute significantly to disease development, genetic factors are central to individual susceptibility, immune response, and disease severity. Elucidating the genetic architecture of pulmonary diseases is therefore critical for understanding their pathophysiological underpinnings and informing therapeutic strategies.

This dissertation investigated the genetic and immunological foundations of major pulmonary diseases by integrating Mendelian randomization, transcriptomic evidence synthesis, and immunopeptidome analysis across three studies.

The first study examined the relationship between chronic obstructive pulmonary disease (COPD) and coronavirus disease 2019 (COVID-19). Two-sample Mendelian randomization analyses were performed to assess potential causal relationships between genetic liability to COPD and three COVID-19 related outcomes: SARS-CoV-2 infection, hospitalization, and development of critical illness. These analyses were supplemented by literature for mining-driven reconstruction of molecular, cellular, and tissue-level interactions between the two diseases. Our Mendelian randomization analyses did not support a causal genetic relationship between COPD and COVID-19 outcomes at the genetic level; however, we found that COPD-associated molecular features including elevated levels of soluble ACE2, TMPRSS2, IL-6, TNF-α, and dysregulated interferon signaling may amplify inflammatory and immune responses during SARS-CoV-2 infection.

The second study addressed the reported association between allergic atopy, Th2-high asthma, and reduced COVID-19 severity. Curated gene signatures representing Th2-high upregulated and interferon-related downregulated pathways were derived from published transcriptomic studies of atopic asthma. Pathway enrichment analysis across Gene Ontology, KEGG, and WikiPathways databases revealed strong enrichment for cytokine signaling, eosinophil recruitment, and IL-4/IL-13-mediated immune pathways in the Th2-high signature, alongside suppression of antiviral interferon signaling. Integration with genetic association data identified key regulatory nodes including STAT6, TSLP, IL33, and interferon-related genes at the intersection of atopy-associated immune responses and SARS-CoV-2 susceptibility pathways.

The third study investigated antigen presentation and immune recognition in non-small cell lung cancer through analysis of the human leukocyte antigen (HLA) class I immunopeptidome. A novel anchor-based clustering framework, termed Dual Anchor Cluster (DAC) and Triple Anchor Cluster (TRAC), was developed to organize HLA-bound peptides according to conserved anchor residue constraints. Application of this framework to immunopeptidome datasets from EGFR-mutant lung cancer cell lines demonstrated that anchor-based peptide

clustering can effectively prioritize candidate neoantigens with high potential for stable HLA presentation and T-cell recognition.

Together, these three studies provide an integrated perspective on how genetic susceptibility, immune polarization, and antigen presentation interact across pulmonary diseases. By combining population-level genetic inference with pathway analysis and immunopeptidome characterization, this dissertation reveals convergent molecular mechanisms linking airway inflammation, respiratory infection susceptibility, and tumor immune recognition, providing a multi-scale framework linking immune regulation to antigen presentation and disease susceptibility to pulmonary diseases.

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