UNIVERSITY of MARYLAND
SCHOOL OF MEDICINE

Update Your Profile

# Erik P. Lillehoj, PhD


Academic Title:

Associate Professor

Primary Appointment:

Pediatrics

Email:

elillehoj@peds.umaryland.edu

Location:

655 W. Baltimore St., BRB 13-029

Phone (Primary):

(410) 706-3872

Fax:

(410) 706-0020


Download CV

## Education and Training

- 1972-1976 - B.S., Microbiology/Chemistry, University of Illinois, Urbana, IL
- 1976-1981 - Ph.D., Immunology. Wayne State University, Detroit, MI
- 1981-1986 - Post-doctoral Fellow, Immunogenetics, NIAID, NIH, Bethesda, MD

## Biosketch

I have a broad research background in protein/peptide chemistry, microbiology, and immunology. As a predoctoral student at Wayne State University School of Medicine, I purified and characterized the $\beta_2$-microglobulin subunit of the major histocompatibility complex (MHC) class I molecules that are responsible for host immune recognition of microbial pathogens. As a postdoctoral fellow at the NIH, I continued this line of research, defining the structure-function relationship of mouse MHC class I proteins.

As a member of the faculty at George Washington University School of Medicine and the University of Maryland School of Medicine, I continued studies of host-pathogen interactions at mucosal surfaces, receiving a Dalsemer Award from the American Lung Association to pursue these investigations. In collaboration with Dr. K. Chul Kim, we discovered that the MUC1 membrane-

tethered mucin is an epithelial cell surface receptor for Pseudomonas aeruginosa and provided the first credible evidence at the molecular level of this host-pathogen interaction that is becoming the paradigm for ligand-receptor studies in airway epithelial cell biology.

These investigations were significant since P. aeruginosa infection impacts the morbidity and mortality of patients with a variety of lung diseases, including cystic fibrosis, ventilator-associated pneumonia, and chronic obstructive pulmonary disease. Subsequently, Dr. Kim and I collaborated on a series of studies that characterized the effect of neutrophil elastase and TNF-α on MUC1 expression and identified an anti-inflammatory role for MUC1 expressed by airway epithelial cells.

More recently, in collaboration with Dr. Simeon E. Goldblum and Dr. Avelino C. Verceles at the University of Maryland School of Medicine, we have focused on the ability of host sialidases to regulate the airway epithelial cell response to environmental cues and danger signals, including the ability of the NEU1 sialidase to desialylate the MUC1 ectodomain in response to P. aeruginosa. We have established that among the 4 mammalian sialidases (NEU1-4), NEU1 is expressed at the greatest levels in human airway epithelia, and have identified preformed pools of NEU1 and its chaperone/transport protein, PPCA, that associate with and desialyate MUC1 in response to its cognate ligand, P. aeruginosa flagellin. At the same time, NEU1-mediated desialylation of MUC1 unmasks a protease recognition site to increase its shedding, and the shed MUC1 ectodomain acts as a decoy receptor that competitively block P. aeruginosa adhesion to cell-associated MUC1. We envision that MUC1 has the potential to be developed into a therapeutic agent against, and/or diagnostic test for, invasive P. aeruginosa lung infection.

## Research/Clinical Keywords

Pseudomonas aeruginosa, Helicobacter pylori, Mucins, MUC1, Sialidase/Neuraminidase, NEU1

## Highlighted Publications

For a complete list of published work in MyBibliography, click here.

Update Your Profile

×