Peter A Keyel, PhD

EDUCATION

BS (chemistry and biochemistry/molecular biology), University of Minnesota Duluth
PhD (cell biology & molecular physiology), University of Pittsburgh
Postdoctoral (immunology), Howard Hughes Medical Institute/Washington University in St Louis, University of Pittsburgh

PROFESSIONAL EXPERIENCE

2020 – Present Associate Professor (Biological Sciences), Texas Tech University
2013-2020 Assistant Professor (Biological Sciences), Texas Tech University

PROFESSIONAL ACTIVITIES

Academia: 2015-Present Associate Director, College of Arts & Sciences Microscopy, Texas Tech University

Society: 2019-Present Publications committee member, SLB
2020-Present Reviewer Training Task Force chair, SLB

Grant Review: 2018 – Present, American Heart Association, Fellowships; 2016 Department of Defense Threat Reduction Agency; 2018 Czech Science Foundation; 2019, Medical Research Council, UK; 2019 Deutsche Forschungsgemeinschaft (German Research Foundation/DFG)/Arts and Humanities Research Council (AHRC); 2020 Swiss National Science Foundation; Biotechnology and Biological Sciences Research Council, UK; 2020 Center for Scientific Review, NIH

RESEARCH INTERESTS

OVERALL (CONTROL OF INFLAMMATION BY MACROPHAGES)

1) *Cellular responses to pore-forming toxins*. Pore-forming toxins are used by immune cells to execute programmed cell death, while bacterial pathogens use pore-forming toxins to promote infection and immune evasion. Macrophages resist bacterial toxins, while executing programmed cell death when needed, and have a ~30-fold difference in toxin sensitivity. Toxin sensitivity is primarily driven by membrane repair. One aspect of my research focuses on understanding the membrane repair mechanisms used to prolong macrophage survival and how pore-forming toxins subvert these responses to alter pro-inflammatory signaling.

2) *Macrophage-derived Dnase1L3 in lupus*. Complete deficiency in the serum endonuclease Dnase1L3, which is secreted by macrophages and dendritic cells, causes pediatric-onset lupus, while reduction in Dnase1L3 activity is associated with sporadic lupus and hypocomplementemic urticarial vasculitis syndrome. The mechanism of Dnase1L3 activity is poorly understood. The other aspect of my research focuses on understanding the structure/function of Dnase1 family members to develop Dnase1L3 replacement therapy as a treatment for lupus.

STATEMENT OF INTEREST

Scientific societies collectively face the challenges of public skepticism of scientific experts, declining participation, and the declining impact of society-led journals. To survive, scientific societies need successfully navigate these challenges, which requires adapting to demographic and cultural changes. This presents a tremendous opportunity for SLB to be the premier scientific society of the future, and set the standard that other scientific societies will follow. For example, the Reviewer Training Task Force that I chair is innovating on reviewer preparation to improve the numbers and quality of scientific reviewers. This training series will provide unique, new value to society members, the *Journal of Leukocyte Biology*, and the scientific community as a whole. As a member of the SLB Council, I would advocate pursuing new approaches to public engagement, finding new ways to bring value to members, especially early career members and trainees, and help SLB position itself to model scientific societies in the 21st century. While many opportunities for SLB exist, three that I think will help SLB thrive in the face of current challenges are:

1) Lay membership options to empower and engage the public
2) Increasing professional development modules
3) Adopting cutting-edge technologies to connect members and accelerate science

I believe SLB has the unique opportunity to forge a new path forward for scientific societies.