# 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
2023-Present Publications committee chair, SLB
2020-2022 Reviewer Training Task Force chair, SLB
2022-2023 Methods Task Force committee member, SLB
2022-2024 Training Editor-in-Chief, Reviewer Training Program, SLB
2022-2023 SLB 2023 Annual Meeting co-Chair

***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; 2023-2024 Veterans Affairs, Infectious Diseases Scientific Group (INFB)

**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***

The Treasurer is an important position in the Society that demands trust and excellence. I believe that a conservative financial framework is essential for supporting our initiatives, fostering collaboration among members, and sustaining our annual meeting. I am eager to bring my dedication, and collaborative spirit to the Treasurer position and work closely with the executive team and members to advance our shared goals. I look forward to the opportunity to contribute to the continued success and growth of the Society for Leukocyte Biology. If elected as Treasurer, I am dedicated to upholding transparency, accountability, and prudent fiscal decision-making. I will strive to ensure the long-term sustainability of our society's initiatives. Thank you for considering my candidacy. I am excited about the possibility of serving as your Treasurer and contributing to the bright future of our society.