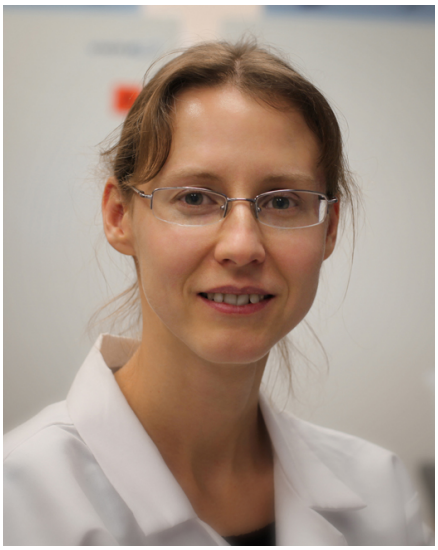

The 11th Annual Exhibition of Undergraduate Research and Creative Activities - EXPO 2024

GUEST SPEAKER

Natasha Kirienko, Ph.D.

Associate Professor of BioSciences

CPRIT Scholar in Cancer Research

Rice University

April 18, 2024 - 9:15 to 10:00 a.m.

Live Oak Ballroom - Setzer Center

SHORT BIOGRAPHY:

Dr. Kirienko is a CPRIT Scholar in Cancer and an Associate Professor at BioSciences at Rice University, where she started her independent lab in 2015. She is the President-Elect of Texas Branch ASM and the Chair of the Antimicrobial Resistance Cluster of the Gulf Coast Consortia. Dr. Kirienko began working on cancer research using a *Caenorhabditis elegans* model to characterize Retinoblastoma and its interactors during her PhD studies at the University of Wyoming (advisor: Dr. David Fay). During her postdoc at Massachusetts General Hospital / Harvard Medical School under Dr. Fred Ausubel and Dr. Gary Ruvkun, she was involved in several drug-screening projects using *C. elegans*, as well as host-pathogen interactions research. She has continued these projects in her own lab, augmenting them with collaborations with clinical researchers, facilitating access to patient samples and pre-clinical mammalian models. During her time at Rice, she has trained 9 graduate and over 50 undergraduate students in her lab. She also published over 50 peer-reviewed publications.

LECTURE: Harnessing Mitochondrial Dysfunction as a Target for Leukemia Therapy

Acute myeloid leukemia (AML) is one of the most common hematological malignancies. It has rapid progression and startling mortality in untreated patients. This is particularly true for elderly patients (i.e., > 65 years of age), where up to 70% of newly diagnosed patients succumb within a year. Using bioinformatic and wet-lab approaches, we found that leukemic cells show striking sensitivity to mitochondrial damage. Our subsequent research identified lower coupling efficiency in the mitochondria of AML cells as a cause for this sensitivity. We showed strong synergy between multiple combinations of mitochondria-targeting molecules and known anti-cancer agents in AML. To leverage these findings, we performed a high-throughput screen and identified several compounds that increased levels of the kinase PINK1, which licenses mitophagic activity and drives mitochondrial turnover. Iterative structure-activity relationship studies yielded analogs that killed AML cells at nanomolar concentrations. These compounds also synergized with known AML chemotherapeutics, were effective against leukemia stem cells, and reduced tumor burden and extended survival in mice engrafted with human leukemia cells.

Office of Undergraduate Research
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