BIOGRAPHICAL SKETCH

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NAME: Kalinski, Ashley

eRA COMMONS USER NAME (credential, e.g., agency login): acurren

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE (if applicable)	END DATE MM/YYYY	FIELD OF STUDY
Adrian College, Biology, Modern Languages, Adrian, MI	BA	05/2010	Biology, Modern Languages
Drexel University, Biology, Philadelphia, PA	PHD	09/2015	Biological Sciences
University of South Carolina, Columbia, SC	Postdoctoral Fellow	12/2015	Molecular Neuroscience
University of Michigan, Ann Arbor, MI	NIH training grant	06/2018	Neuroscience
University of Michigan, Ann Arbor , MI	Postdoctoral Fellow	07/2020	Neuroinflammation

A. Personal Statement

I am an assistant professor of biology at The University of South Carolina with 10 years of experience studying the underlying mechanisms of axonal regeneration in the peripheral and central nervous systems. I have been lead or co-author on 16 peer-reviewed publications on this topic. During my doctoral training with Dr. Jeffery Twiss, I extensively studied the role of injury-induced neuronal intrinsic repair programs in peripheral and central axons. Under normal conditions axons of the central nervous system are unable to regenerate, while axons of the peripheral nervous system are able to spontaneously regenerate. This dichotomy can be addressed by both extrinsic and intrinsic differences. Our lab focused on intrinsic mechanisms that happen locally within injured axons and we were one of the first to show that protein synthesis occurs in axons independently of the neuronal cell body. Through my work I was able to show for the first time, that if axons of the central nervous system are given a permissive substrate to regenerate into, these neurons were capable of up regulating mRNA transport and likely protein synthesis. This work suggested that axons of the central nervous system are able to induce regeneration programs if given the proper extrinsic conditions. I continue regular and ongoing consultation and collaboration with the Twiss lab.

During my postdoctoral training with Dr. Roman Giger, I focused on extrinsic mechanisms that regulate axon regeneration in the peripheral and central nervous system. Utilizing single cell RNA sequencing, my work showed for the first time a characterization of the temporal regulation of immune cell populations following peripheral nerve injury. Further, this novel work showed that efferocytosis plays a significant role in nerve debridement during the injury response. Importantly, I learned several techniques such as peripheral nerve grafting, immune cell injections into the sciatic nerve, parabiosis, flow cytometry, optic nerve crush, and dorsal column lesion during this fellowship. We have now established several of these techniques in my own laboratory. We are continuing to characterize the inflammatory response in the peripheral nerve following injury through a very productive collaboration with the Giger Lab.

I am committed to providing students with authentic and meaningful research experiences, which result in authorship on peer-reviewed publications. All of my first author publications have had undergraduate student mentees as co-authors. In 2021 my lab published a comprehensive review on Neuroinflammation after injury in Neuroscience Research with a graduate student as first author and 3 undergraduate

students as co-authors. Prior to arriving at BSU, I have successfully mentored 6 undergraduate research students for 1-2 years each. Four of these students are currently in medical school or residency programs and two students are research technicians in either industry or in an academic laboratory. Since joining BSU in 2020, I have mentored an additional 8 undergraduate students, 1 masters student, and 3 high school students. Of the 18 total students, 12 are women, 7 are from historically underrepresented racial and ethnic groups and/or students with disabilities. Three students have presented their work at local or regional scientific meetings. One of our high school students competed at the regional, state, and international Regeneron ISEF Science Fair (winning his division at the regional and state level). Two students have graduated from the Kalinski Laboratory and have been either accepted into graduate school or are working in biotech.

In addition to hands on training of the students in the laboratory, I have also mentored three students through the grant-writing process. One of which was awarded a Sigma XI grant in aid of research (she was one of only 21 undergraduates to be awarded), one was a Goldwater Scholarship BSU nominee, and one has received an internal research grant. Additionally, an undergraduate research student was selected as an honorable mention in an imaging competition from the Society for Leukocyte Biology for their work imaging macrophages on a confocal microscope.

While I was an Assistant Professor at Ball State University, we applied our expertise in axonal repair mechanisms to understand the relationship between the nervous system and immune system, and I will continue this work at UofSC. Peripheral nerve injuries impact approximately 20 million people in the United States alone and the long-lasting health impacts to traumatic injuries are equivalent to many neurological diseases. In the long-term, my laboratory is interested in understanding how inflammation impacts axonal regeneration in hopes of finding new therapeutic targets for severe injuries of the central nervous system. Our laboratory, lead by undergraduate research students, has established several assays to unravel these mechanisms including mouse genetics, mouse behavioral assays, flow cytometry, immunostaining, fluorescent in situ hybridization, protein extraction, western blot analysis, PCR, light and confocal microscopy, primary cell culture, and live cell imaging. Our current research funding supports work to address neuron-immune cell interactions. Working primarily in vitro, we are trying to understand how different activation states of immune cells (primarily macrophages) influence sensory neuron outgrowth. This will allow us to elucidate the dynamic growth changes that occur after PNS injury in vivo. Additionally, we are characterizing the inflammatory landscape of the nerve in young, middle aged, and old mice to begin to understand how chronic inflammation impacts regeneration. We are also assessing how impaired injury signaling pathways may impact pain responses in sensory neurons.

I also engage undergraduate students in research in the classroom. At Ball State I taught a novel research experience course, Methods in Cell Biology. In this course students performed novel cell biology research that is related to the instructor's research interests. Following the course, validation of the findings occurs within the instructor or other biology department faculty's lab. We then were able to publish these results in small research journals such as micropubs. So far, in collaboration with Drs. Rubenstein, Smaldino, and True we have published 2 micropublications based on protein degradation in yeast in 2022 and 2024. Importantly, this course provided an additional 32 undergraduates a novel research experience every year.

Ongoing Research Support: University of South Carolina Start-up Funds. 08/2024-07/2027. Kalinski, Ashley Lauren (PI) Immune cell mediated neural repair Role: PI The goal of these funds is to restablish the Kalinski Laboratory at University of South Carolina and continue our work on SARM1 and inflammation post injury.

NINDS R15. 05/2023-05/2026. Kalinski, Ashley Lauren (PI) Regulation of Neuroinflammation after Peripheral Nerve Injury. Role: PI The goal of these funds is to establish a role for SARM1 during inflammation mediated during the injury response.

John's Hopkins Merkin Peripheral Neuropathy and Nerve Repair Center Research Grant. 12/2022-11/2024. Kalinski, Ashley Lauren (PI) Elucidating the cell-autonomy of SARM1 for injury induced axon regeneration, nerve inflammation, and Schwann cell reprogramming Role: PI The goal of these funds is to establish 3 new SARM1 conditional knockout mouse lines to determine if SARM1 has a non-neuronal role in peripheral nerve injury.

ASPiRE Junior Faculty Research Grant. 05/2023-04/2024. Kalinski, Ashley Lauren (PI) Establishing a co-culture system to examine the impacts of injury-activated immune cells on axonal dynamics Role: PI The goal of these funds is to further establish macrophage-neuron co-cultures to understand how neurons respond to injury activated macrophages

Completed Research Support: Ball State University Provost Start-up Funds. 08/2020-07/2023 Kalinski, Ashley Lauren (PI) Unraveling Signaling Pathways between Immune Cells, Schwann Cells, Astrocytes, and Neurons Role: PI The goal of these funds is to establish the Kalinski Laboratory at Ball State University, and to begin to identify outcomes of interactions between neuronal and non-neuronal cells in the context of nerve injury.

Indiana Academy of Science. 11/2021-10/2022. Kalinski, Ashley Lauren (PI) Impact of injury-activated immune cells on axonal dynamics Role: PI The goal of these funds is to examine the effects of immune cells on sensory neuron axonal dynamics in vitro

1. Schmitd LB, Hafner H, Ward A, Asghari Adib E, Biscola NP, Kohen R, Patel M, Williamson RE, Desai E, Bennett J, Saxman G, Athaiya M, Wilborn D, Shumpert J, Zhao XF, Kawaguchi R, Geschwind DH, Hoke A, Shrager P, Collins CA, Havton LA, Kalinski AL, Giger RJ. Sarm1 is not necessary for activation of neuron-intrinsic growth programs yet required for the Schwann cell repair response and peripheral nerve regeneration. bioRxiv. 2024 Apr 17; PubMed Central PMCID: PMC10942360.
2. Zhao,Xiao-Feng,, Huffman,Lucas D.,, Hafner,Hannah,, Athaiya,Mitre,, Finneran,Matthew,, Kalinski,Ashley L.,, Kohen,Rafi,, Flynn,Corey,, Passino,Ryan,, Johnson,Craig,, Kohrman,David,, Kawaguchi,Riki,, Yang,Lynda,, Twiss,Jeff,, Geschwind,Daniel H.,, Corfas,Gabriel,, Giger,Roman J.,. The Injured Sciatic Nerve Atlas (iSNAT), Insights into the Cellular and Molecular Basis of Neural Tissue Degeneration and Regeneration. [Preprint]. 2022 June 29. DOI: 10.1101/2022.06.26.497651
3. Kalinski AL, Yoon C, Huffman LD, Duncker PC, Kohen R, Passino R, Hafner H, Johnson C, Kawaguchi R, Carbajal KS, Jara JS, Hollis E, Geschwind DH, Segal BM, Giger RJ. Analysis of the immune response to sciatic nerve injury identifies efferocytosis as a key mechanism of nerve debridement. Elife. 2020 Dec 2;9 PubMed Central PMCID: PMC7735761.
4. Sas AR, Carbajal KS, Jerome AD, Menon R, Yoon C, Kalinski AL, Giger RJ, Segal BM. A new neutrophil subset promotes CNS neuron survival and axon regeneration. Nat Immunol. 2020 Dec;21(12):1496-1505. PubMed Central PMCID: PMC7677206.

B. Positions, Scientific Appointments and Honors

Positions and Scientific Appointments

2024 -	Assistant Professor, University of South Carolina, Biological Sciences
2020 - 2024	Assistant Professor, Ball State University, Biology, Muncie, IN
2019 - 2020	Adjunct Professor, Adrian College, Adrian, MI
2015 - 2020	Postdoctoral Research Fellow, University of Michigan, Cell and Developmental Biology, Ann Arbor, MI

Honors

2017 - 2018	Best Research Fellow Poster, University of Michigan
2016 - 2017	T32 NIH Training Grant, University of Michigan/NINDS-NIH

2013 - 2014	Travel Award, Drexel University
2020	Outstanding Professor, Chi Omega Phi Epsilon at Ball State University
2019	Travel Award, University of Michigan
2019	Postdoc 180 Finalist, University of Michigan
2019	RNA Salon Grant Awardee, RNA Society and Lexogen
2019	3rd place Scientific Image Competition, University of Michigan Center for RNA Biomedicine
2016	Young Alumni Achievement Award, Adrian College
2013	International Travel Award, United States-Israel Binational Science Foundation
2012	Outstanding STAR Mentor, Drexel University

C. Contribution to Science

1. As a faculty member at Ball State University, my lab established a role for SARM1 during the inflammatory response following peripheral nerve injury. Through a collaborative project with Roman Giger at the University of Michigan we found that SARM1 is required for inflammation and Schwann cell responses following injury, but not for neuronal transcriptional responses. Further, we generated 2 new SARM1 conditional knockout lines that support SARM1's requirement in both macrophages and neurons. This work is under review and is posted as a preprint on BioRxiv.

    a. Schmitd LB, Hafner H, Ward A, Asghari Adib E, Biscola NP, Kohen R, Patel M, Williamson RE, Desai E, Bennett J, Saxman G, Athaiya M, Wilborn D, Shumpert J, Zhao XF, Kawaguchi R, Geschwind DH, Hoke A, Shrager P, Collins CA, Havton LA, Kalinski AL, Giger RJ. Sarm1 is not necessary for activation of neuron-intrinsic growth programs yet required for the Schwann cell repair response and peripheral nerve regeneration. bioRxiv. 2024 Apr 17; PubMed Central PMCID: PMC10942360.

2. Efferocytosis as a key mechanism in nerve debridement: As a postdoctoral fellow, I investigated the role of inflammation during axonal injury. I showed for the first time a detailed temporal characterization of the cellular population of the sciatic nerve after injury and that efferocytosis is a key mechanism to clear the degenerating nerve stump during axon regeneration. This was the first complete single cell RNA sequencing data set of the sciatic nerve to be generated following injury. Further, in collaboration with the Segal laboratory, I established a neuron-immune cell co-culture system to assess neuronal dynamics in the presence of differentially activated immune cells. This work lead to the novel finding that an alternatively activated neutrophil can promote regeneration in both the central and peripheral nervous system.

    a. Zhao,Xiao-Feng,, Huffman,Lucas D.,, Hafner,Hannah,, Athaiya,Mitre,, Finneran,Matthew,, Kalinski,Ashley L.,, Kohen,Rafi,, Flynn,Corey,, Passino,Ryan,, Johnson,Craig,, Kohrman,David,, Kawaguchi,Riki,, Yang,Lynda,, Twiss,Jeff,, Geschwind,Daniel H.,, Corfas,Gabriel,, Giger,Roman J.,. The Injured Sciatic Nerve Atlas (iSNAT), Insights into the Cellular and Molecular Basis of Neural Tissue Degeneration and Regeneration. [Preprint]. 2022 June 29. DOI: 10.1101/2022.06.26.497651

    b. Kalinski AL, Yoon C, Huffman LD, Duncker PC, Kohen R, Passino R, Hafner H, Johnson C, Kawaguchi R, Carbajal KS, Jara JS, Hollis E, Geschwind DH, Segal BM, Giger RJ. Analysis of the immune response to sciatic nerve injury identifies efferocytosis as a key mechanism of nerve debridement. Elife. 2020 Dec 2;9 PubMed Central PMCID: PMC7735761.

    c. Sas AR, Carbajal KS, Jerome AD, Menon R, Yoon C, Kalinski AL, Giger RJ, Segal BM. A new neutrophil subset promotes CNS neuron survival and axon regeneration. Nat Immunol. 2020 Dec;21(12):1496-1505. PubMed Central PMCID: PMC7677206.

3. mRNA transport and translation in CNS axons: During my doctoral work I asked whether spinal cord

axons contain mRNAs and translational machinery as they regenerate into a permissive environment. I began this work by optimizing fluorescent in situ hybridization (FISH) in CNS tissues. This optimized FISH approach coupled with high-resolution confocal imaging sequences that I developed for quantitation across large regions of tissues to regenerating spinal cord axons. For this, I collaborated with John Houle's laboratory to study ascending spinal axons regenerating into a segment of peripheral nerve grafted into transected spinal cord. I showed, for the first time, that these CNS axons contain mRNAs encoding injury-associated and growth-associated gene products, with several at levels comparable to regenerating peripheral nerve. Presence of ribosomes and translation factors in these axons by immunofluorescence strongly suggests that regenerating sensory axon can locally synthesize proteins. Further, through the advances I have made in detection of axonal RNAs has lead to an increase in collaborations with other labs, which are reflected in subsequent co-authored publications.

a. Terenzio M, Koley S, Samra N, Rishal I, Zhao Q, Sahoo PK, Urisman A, Marvaldi L, Oses-Prieto JA, Forester C, Gomes C, Kalinski AL, Di Pizio A, Doron-Mandel E, Perry RB, Koppel I, Twiss JL, Burlingame AL, Fainzilber M. Locally translated mTOR controls axonal local translation in nerve injury. Science. 2018 Mar 23;359(6382):1416-1421. PubMed Central PMCID: PMC6501578.
b. Merianda TT, Jin Y, Kalinski AL, Sahoo PK, Fischer I, Twiss JL. Neural Progenitor Cells Promote Axonal Growth and Alter Axonal mRNA Localization in Adult Neurons. eNeuro. 2017 Jan-Feb;4(1) PubMed Central PMCID: PMC5291088.
c. Perry RB, Rishal I, Doron-Mandel E, Kalinski AL, Medzihradszky KF, Terenzio M, Alber S, Koley S, Lin A, Rozenbaum M, Yudin D, Sahoo PK, Gomes C, Shinder V, Geraisy W, Huebner EA, Woolf CJ, Yaron A, Burlingame AL, Twiss JL, Fainzilber M. Nucleolin-Mediated RNA Localization Regulates Neuron Growth and Cycling Cell Size. Cell Rep. 2016 Aug 9;16(6):1664-1676. PubMed Central PMCID: PMC4978702.
d. Kalinski AL, Sachdeva R, Gomes C, Lee SJ, Shah Z, Houle JD, Twiss JL. mRNAs and Protein Synthetic Machinery Localize into Regenerating Spinal Cord Axons When They Are Provided a Substrate That Supports Growth. J Neurosci. 2015 Jul 15;35(28):10357-70. PubMed Central PMCID: PMC4502271.

4. Identification of a novel role for HDAC6 in axons: During my doctoral and postdoctoral work I investigated the role of HDAC6 in sensory axons. We discovered a novel interaction between HDAC6 and Miro1 that regulates mitochondrial function and transport in axons in response to extracellular growth cues. Non-permissive substrates, such as chondroitin sulfate proteoglycans normally prevent axonal growth, however, in the presence of an HDAC6 inhibitor growth can be rescued. We were able to show that this is likely due to a calcium and Rho/Rock dependent cascade mediated through Miro1. The loss of HDAC6 improved mitochondrial function, transport, and growth cone dynamics in the presence of these inhibitory substrates. Importantly, through this work I was able to establish live cell imaging techniques and methods to measure axonal dynamics and health that are now being implemented in my own laboratory.

    a. Kalinski AL, Kar AN, Craver J, Tosolini AP, Sleigh JN, Lee SJ, Hawthorne A, Brito-Vargas P, Miller-Randolph S, Passino R, Shi L, Wong VSC, Picci C, Smith DS, Willis DE, Havton LA, Schiavo G, Giger RJ, Langley B, Twiss JL. Deacetylation of Miro1 by HDAC6 blocks mitochondrial transport and mediates axon growth inhibition. J Cell Biol. 2019 Jun 3;218(6):1871-1890. PubMed Central PMCID: PMC6548128.

Complete List of Published Work in My Bibliography:
https://www.ncbi.nlm.nih.gov/myncbi/ashley.kalinski.1/bibliography/public/