Provide the following information for the Senior/key personnel and other sign Td[i).S EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Start Date MM/YYYY	Completion Date MM/YYYY	FIELD OF STUDY
Rhodes College	BS	08/2015	05/2019	Biochemistry & Molecular Biology
University of South Alabama	PhD	08/2019	08/2024	Basic Medical Sciences (Lung Biology focus)

My interest in the pulmonary endothelium and its role in cardiopulmonary

Balczon R, Morrow K, Stevens TC, , Agwaramgbo E, Langham G, Francis C, and Stevens T. Cystatin C regulates the cytotoxiity of infection-induced endothelial-derived beta amyloid. 10: 2464-2477, 2020.

Lee J, , Kash M, Zhou C, Koloteva A, Renema P, Paudel S, & Stevens T. KD025 shifts pulmonary endothelial cell bioenergetics and decreases baseline lung permeability. , 63: 519-530, 2020.

Fall 2017, Fall 2018	Foundations of Chemistry Lab Teaching Assistant, Rhodes College
2019 – present	Graduate Research Assistant, University of South Alabama

2018 – present	Member, Sigma Epsilon Honor Society
2018 – present	Member, TriBeta Biology Honor Society
2018 – present	Member, Mortar Board National College Senior Honor Society
2019 – present	Member, American Thoracic Society
2020 – present	Member, American Heart Association

Fall 2015, Spring 2018, Spring 2019	Honor Roll, Rhodes College
Spring 2016	Dean's List, Rhodes College
2015, 2018	Cross Country Academic All-American, Rhodes College
2015 – 2019	Southern Athletic Association Academic Honor Roll, Rhodes College
2019 – 2020	for best academic performance in first year basic medical science courses, University of South Alabama

During my time in high school, I had the opportunity to train in the lab under Dr. Adam Morrow at the University of South Alabama, supported by a NIH-sponsored high school research fellowship. Dr. Morrow investigated the mechanisms for inhibiting repair in pulmonary microvascular endothelial cells (PMVECs) following infection. PMVECs utilize aerobic glycolysis to meet their bioenergetic demands during proliferation, therefore my research project was to determine if bacterial infection impairs aerobic glycolysis in PMVECs. I infected PMVECs with , treated the cells with antibiotics, and then subjected the cells to single cell cloning. Aerobic glycolysis acidifies the medium and can be detected by a medium color shift. Two weeks after single cell growth, I analyzed cell colony media color and found bacterial infection reduces the number of PMVECs utilizing aerobic glycolysis. My results suggested that PMVECs have impaired bioenergetics post infection, contributing to reduced vascular repair.

, Hartman L, Balczon R, Morrow A, and Stevens T. ExoY impairs the rapid growth of pulmonary microvascular endothelial cells. Research day, summer medical research program, University of South Alabama College of Medicine, July 31, 2015.

During my undergraduate studies, I joined Dr. Balczon's lab at the University of South Alabama to continue investigating endothelial dysfunction after bacterial infection. Dr. Balczon and collaborators had

project focused on characterizing the chemical properties and fluorescent signatures of cytotoxic amyloids derived from the lung endothelium I isolated lung endothelial amyloids and then treated the proteins with 1,1,1,3,3,3-hexofluoro-2-propanol (HFIP), a chemical known to disrupt amyloid protein structure. Amyloids treated with HFIP did not display any toxicity to naïve PMVECs and had reduced fluorescence when exposed to Thioflavin T (ThT), a fluorescent dye that specifically binds to amyloid proteins. Thus, my results demonstrated HFIP disrupts the complex structure of cytotoxic amyloid variants that are derived from the lung endothelium, and in doing so, eliminates their cytotoxicity. These results proved fruitful as they were incorporated into Dr. Balczon's recent publication (2020).

The following summer, I worked with Dr. Balczon to determine if ThT could be repurposed as a clinical test to detect cytotoxic amyloids in pneumonia patients. I treated pneumonia patient blood samples with ThT and then scanned for fluorescence over a range of excitation and emission wavelengths. Next, I immunoprecipitated cytotoxic amyloid proteins from pneumonia blood samples, treated them with ThT, and repeated the fluorescent scan. Points of overlap in fluorescence between the pneumonia blood samples and isolated amyloid proteins were detected. Thus, my results identified unique fluorescent signatures of amyloid proteins in pneumonia blood samples, suggesting that ThT has the potential to be developed into a rapid bedside diagnostic test.

Balczon R, Morrow K, Stevens TC, , Agwaramgbo E, Langham G, Francis C, and Stevens T. Cystatin C regulates the cytotoxiity of infection-induced endothelial-derived beta amyloid. 10: 2464-2477, 2020.

Berrou M, , Voth S, Williams C, Balczon R, and Stevens, T. induced lung endothelial amyloid proteinopathy: characteristics and inhibitors. , 197: A5724, 2018.

, Francis M, and Balczon R. Chemical properties of endothelial cytotoxic amyloids. Research day, summer medical research program, University of South Alabama College of Medicine, July 28, 2017.

, Cioffi E, Voth S, and Balczon R. Analysis of cytotoxic amyloids from human pneumonia patients. Research day, summer medical research program, University of South Alabama College of Medicine, July 27, 2018.

studies may also provide evidence that CA IX acts as a receptor, regulating the PMVEC response to injury and pH changes.