



LOMA LINDA UNIVERSITY

School of Medicine

*Center for Health Disparities
and Molecular Medicine*

24th Annual Health Disparities Research Symposium



Education – Development – Health Disparities Research – Community

PROGRAM, BIOS, AND ABSTRACTS

Wednesday, July 30, 2025

2:00 pm – 7:00 pm

Wong Kerlee International Conference Center

Loma Linda University School of Medicine

Loma Linda, California



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Center for Health Disparities and Molecular Medicine

24th Annual Health Disparities Research Symposium

Wednesday, July 30, 2025

2:00 pm - 7:00 pm, Wong Kerlee International Conference Center

Agenda

Poster Session

2:00 pm – 4:00 pm

Poster Presentations by Research Fellows

Apprenticeship Bridge to College Program (ABC)

Undergraduate Training Program (UTP)

LLU-NIH Initiative for Maximizing Student Development Program (IMSD)

Summer Undergraduate Research Fellowship (SURF)

Macpherson Society Scholars

4:00 pm – 4:30 pm

Refreshments

4:30 pm – 5:00pm

Oral Flash Presentations

Johnny D. Figueroa, PhD

Associate Professor, CHDMM

Evening Program

5:00 pm – 7:00 pm

Welcome

Johnny D. Figueroa, PhD

Associate Professor, CHDMM

Invocation

Eileen J. Brantley, PhD

Associate Professor, Basic Sciences

Remarks

Tamara L. Thomas, MD

Dean, School of Medicine

Richard H. Hart, MD, DrPH

President, Loma Linda University

Introduction to Panel Speakers

Marino De León, PhD - Chair
Director, CHDMM

Panel Speakers

Dr. Elaine Vanterpool, PhD
Professor and Chair, Department of Biological Sciences
Oakwood University

Dr. Brian C. Hutchins, M.D.
San Bernardino County Sheriff's Department Coroner Division
Chief Forensic Pathologist
Forensic Pathology Fellowship Program Director

Dr. Wendalee Rivera Pacheco, M.D.
San Bernardino County Physician

Dr. Gabriel R. Linares, PhD
Senior Scientist at VersaPeutics Inc.

Acknowledgement of Research Fellows

Carlos A. Casiano, PhD
Associate Director, CHDMM

Kylie J. Watts, PhD
Associate Professor, Basic Sciences

Tamara L. Thomas, MD
Dean, School of Medicine

Richard H. Hart, MD, DrPH
President, Loma Linda University

Johnny D. Figueroa, PhD
Associate Professor, Basic Sciences, Physiology Division

Daisy D. De León, PhD
Professor of Physiology

Marino De León, PhD - Chair
Director, CHDMM

Final Remarks and Acknowledgements

Marino De Leon, PhD
Director, CHDMM

ACKNOWLEDGEMENTS

We would like to acknowledge the contributions of all who were instrumental in making this 2025 Health Disparities Summer Research successful. Teamwork, cooperation, and flexibility are just a few of the skills necessary to successfully implement such a dynamic research program. We also would like to acknowledge the support of the Loma Linda University School of Medicine, the National Institute of General Medical Sciences, NIH (grant 5R25GM060507-22).

2025 Faculty Research Mentors

Eric Behringer, PhD

Arlin Blood, PhD

Danilo Boskovic, PhD

Eileen Brantley, PhD

Carlos A. Casiano, PhD

Daisy De León, PhD

Marino De León, PhD

Alfonso Duran, MD, PhD

Johnny Figueroa, PhD

Christian Hurtz, PhD

Salma Khan, MD, PhD

William Langridge, PhD

Eugenia Mata-Greenwood, PhD,
PharmD

Subburaman Mohan, PhD

Ann Morcos, PhD Post-Doctoral Fellow

Ying Nie, MD, PhD

Rameshwar Patil, PhD

William Pearce, PhD

Christopher Perry, PhD

Reinhard Schulte, MD

Salvador Soriano, PhD

Nathan Wall, PhD

Seth Wiafe, PhD

Christopher Wilson, PhD

Sean Wilson, PhD

Jiang Zhong, PhD

CHDMM Administrative Staff

Lorena Salto, Center Manager

Lynn Lopez, Program Manager

Amy Barajas, Senior Administrative Assistant

This is by no means an exhaustive list. We wish to acknowledge all of the unsung heroes who contributed in very significant ways, too numerous to mention.

2025 Student Research Fellows

ABC – Apprenticeship Bridge to College

Sharan Bir
Caroline Coronado
Yaniah Gamboa
Ava Gatillo
Bernice Juarez
Khaveen Kabilan
Kaavya Kaka
Clara Lee
Vivian Medrano
Mani Momeni
Kevin Nguyen
Sonia Parande
Sanjana Ramesh
Elizabeth Vivianco
Crystal Wei

Krystal Santiago

Julio Sierra

Francis Zamora

SURF – Summer Undergraduate Research Fellowship

Daniel Baldwin
Kiar-Ra Cameron
Benjamin Chun
Juliana Gruenler
Natalie Holm
Joshua Lohr
Kiera McGivney
Dana Robinson
Richard Ye

UTP – Undergraduate Training Program

Micah Andrews
Azaria Carey
Angela Ceja
Lia Hutchins
Robert Lister
Addie McIver
Kyah Miller
Janani Nagasubramaya
Laila Prentice
Annika Samayoa
Kyla Tucker

Lab Volunteers and Research Assistant

Darine Abu Hilal
Raquel Bendita Larico
Justis Cospers
Jonathan De Anda
Julia Soliz
Hans Westenburg
Arianna Williams

Macpherson Society Scholars

Samuel Chan
Aaren Harewood
Madeline Kim
Brian Nguyen

IMSD – PhD Graduate Fellows

Danielle Malivert
Pedro T. Ochoa
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Kayla Sanchez

Institutional Affiliations of Student Research Fellows

High Schools

Arrowhead Christian
Arroyo Valley HS
Beaumont HS
Centennial HS
Chaparral HS
Hemet HS
Loma Linda Academy
Los Osos HS
Moreno Valley HS
Ontario Christian HS
Rancho Cucamonga
Redlands Adventist Academy
Redlands HS
San Geronimo HS
Sigma Prep Academy

Universities

La Sierra University
Loma Linda University School of School of Allied Health Professions
Loma Linda University School of Medicine
Oakwood University
Southern Adventist University
University California State San Diego
University of Redlands



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LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE

CENTER FOR HEALTH DISPARITIES RESEARCH OFFICE OF STUDENT DEVELOPMENT IN THE BIOMEDICAL PROFESSIONS

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Panel Speakers

DR. ELAINE VANTERPOOL, PhD
PROFESSOR AND CHAIR, DEPARTMENT
OF BIOLOGICAL SCIENCES, OAKWOOD UNIVERSITY

Dr. Elaine Vanterpool is a proud alumna of Oakwood (College) University, where she earned her Bachelor of Science in Biology in 2001. She went on to receive her Ph.D. in Microbiology and Molecular Genetics from Loma Linda University, specializing in Microbial Pathogenesis. After completing a one-year postdoctoral fellowship, Dr. Vanterpool returned to Oakwood in 2006 as an Assistant Professor. She was promoted to Associate Professor in 2011 and attained the rank of full Professor in 2017. In 2019, she was appointed Chair of the Department of Biological Sciences.



As Chair, Dr. Vanterpool spearheaded a comprehensive revamp of the biology curriculum to better prepare students for both the workforce and graduate/professional programs. She has been the recipient of over ten federal and non-federal grants, currently serving as Director or Program Manager on three. She also serves as the Point of Contact for several key partnerships and pipeline programs linking Oakwood STEM departments with esteemed institutions including the Mayo Clinic, the University of Pennsylvania, Loma Linda University, among others.

Throughout her tenure at Oakwood, Dr. Vanterpool serves as a mentor for all students who come through her department, over 300+ undergraduate research students and more than 35 middle and high school students. Many of her mentees have successfully matriculated into medical, dental, graduate, and other professional programs. She is actively involved in campus life, serving on numerous committees and sponsoring student clubs and organizations to enrich student development and networking opportunities.

A key contributor to research infrastructure growth at Oakwood, Dr. Vanterpool played a pivotal role in establishing the department's genetics, tissue culture, and gene expression laboratories. These facilities are used for both faculty-led research and the instruction of undergraduate research-intensive courses.

Deeply passionate about STEM education, particularly among underrepresented youth, Dr. Vanterpool has long been an advocate for K-12 outreach. As the service-learning coordinator for the biology department, she fosters university-community partnerships to elevate STEM learning among local youth. With support from the U.S. Department of Education, she recently launched the STEM Success Academy (SSA); a weekend program offering hands-on science labs and math tutoring to local middle and high school students. The SSA aims to address learning gaps in math and science resulting from the COVID-19 pandemic.

Despite her many academic and professional accomplishments, Dr. Vanterpool considers her greatest achievement to be her family. She is the proud wife of Dr. Jevon Vanterpool (married 24 years) and the devoted mother of four amazing children: Jayden (22), Jasmyn (18), Jordon (17), and Janaia (15). Dr. Vanterpool attributes all of her successes to the grace and guidance of God.

DR. BRIAN C. HUTCHINS, M.D.
SAN BERNARDINO COUNTY SHERIFF'S DEPARTMENT CORONER DIVISION
CHIEF FORENSIC PATHOLOGIST
FORENSIC PATHOLOGY FELLOWSHIP PROGRAM DIRECTOR

Born and raised in the city of Mayagüez, Puerto Rico, to Seventh-day Adventist missionaries in which my father is a Loma Linda University alumnus from the School of Dentistry and my mother an alumnus from the School of Nursing. I earned my Bachelor of Science in Biology from Antillean University in Puerto Rico and two weeks after graduation I married my high school sweetheart, Wendalee Rivera Pacheco. Interestingly, we have shared every grade together since fourth grade, including medical school.



I was admitted to Loma Linda University School of Medicine through the Medical Scholar Early Acceptance Program and graduated in 2008. Before and during medical school, I participated in the CHDMM Summer Research Program which was pivotal for my career. During third year of medical school, we surprisingly welcomed our daughter, Lia Hutchins. After medical school, I completed a four-year anatomic/clinical pathology residency at Loma Linda University Medical Center and a one-year forensic pathology fellowship at the Los Angeles County Coroner Division.

I have worked twelve years at the San Bernardino County Sheriff's Department Coroner Division, including three years as the current Chief Forensic Pathologist. In July 2025, I began serving as Program Director of our newly formed forensic pathology fellowship program.

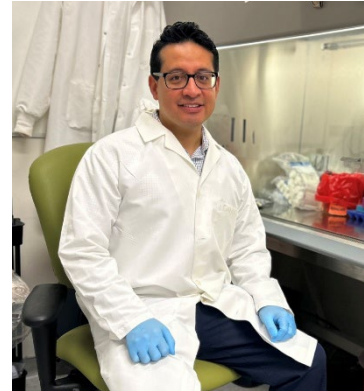
DR. WENDALEE RIVERA PACHECO, M.D.
SAN BERNARDINO COUNTY PHYSICIAN

Born and raised in Mayagüez, Puerto Rico, I initially considered a career in music before deciding that medicine would be a more fulfilling path. I earned my Bachelor of Science in Biology from the University of Antillean and was accepted into medical school through the Loma Linda University School of Medicine's Medical Scholar Early Acceptance Program. Before and during medical school, I participated multiple times in the Center for Health Disparities and Molecular Medicine (CHDMM) Summer Research Program. In the midst of medical school, I welcomed my daughter, Lia Hutchins, an experience that shaped my resilience and deepened my commitment to medicine. I went on to complete my residency in Family Medicine at Kaiser Permanente Fontana. For the past 13 years, I've been serving as a physician at the San Bernardino County jails, providing care to an underserved and often overlooked population.



DR. GABRIEL R. LINARES, PhD
SENIOR SCIENTIST AT VERSAPEUTICS INC.

Dr. Gabriel Linares is a Senior Scientist at VersaPeutics Inc., an early-stage biotechnology company located in San Diego, CA. The research team is pioneering the development of a therapeutic humanized antibody drug for the treatment of spinal cord injury, Alzheimer's disease, and other neurodegenerative indications. Dr. Linares possesses expertise in patient-based drug discovery, target identification and validation, small molecule screening, disease modeling using human inducible pluripotent stem cells (iPSCs), 3D organoid models, interrogating biological mechanisms of action, and testing a range of therapeutic modalities using preclinical models of neurodegeneration. Dr. Linares has authored nearly 20 papers with publications in several high-impact journals such as *Cell*, *Nature Medicine*, *Neuron*, and *Cell Stem Cell*, among others. He is an inventor on a U.S. patent application entitled "Antagonism as a therapy for TDP-43 proteinopathies."



Dr. Linares is a Southern California native of the Inland Empire. He is a proud alumnus of the Loma Linda University Center for Health Disparities and Molecular Medicine pipeline programs where he received outstanding training in biomedical research and professional skills development starting in high school (ABC program) and continuing at the undergraduate (UTP program) and graduate school (IMSD program) levels.

Dr. Linares received his bachelor's degree in Neurobiology, Physiology, and Behavior from the University of California at Davis. As an incoming freshman, he was awarded a prestigious full scholarship from the National Institutes of Health Undergraduate Scholars program (NIH UGSP). The NIH UGSP provides extensive training and mentorship to individuals committed to a career in biomedical research including a summer internship at the NIH intramural research campus and paid employment at NIH after graduation. Dr. Linares obtained his Ph.D. in Physiology from Loma Linda University where he worked with Dr. Subburaman Mohan at the Musculoskeletal Disease Center of the Jerry L. Pettis Memorial VA Medical Center. He investigated the functional role of novel genes that regulate bone formation and bone resorption with implications for understanding fundamental mechanisms relevant to the pathogenesis of osteoporosis. For this work, he received a Young Investigator's Award from the American Society for Bone and Mineral Research and an Outstanding Abstract Award from the Endocrine Society.

Following the completion of his doctoral work, Dr. Linares conducted postdoctoral training at the Molecular Neurobiology Section of the National Institutes of Mental Health, NIH. He demonstrated that preconditioning bone-marrow derived mesenchymal stem cells with mood stabilizers (lithium and valproic acid) prior to transplantation significantly enhanced their biological and therapeutic properties, thereby improving functional recovery and reducing neuropathology in mouse models of Huntington's Disease and traumatic brain injury. These findings have implications for improving the therapeutic efficacy of stem cell-based therapy.

Dr. Linares completed a second postdoctoral fellowship at the Department of Stem Cell Biology and Regenerative Medicine at the Keck School of Medicine of the University of Southern California. At USC, Dr. Linares performed a phenotypic chemical screen and identified broadly acting therapeutic targets that showed efficacy in motor neurons generated from a diverse cohort of human iPSCs comprised of patients with genetic and sporadic forms of

amyotrophic lateral sclerosis (ALS). He showed that the inhibition of *PIKFYVE* rescues neurodegeneration and improves motor function by stimulating the exocytosis of toxic aggregation-prone proteins that accumulate during ALS pathogenesis. Dr. Linares also identified *SYF2*, a spliceosome-associated factor as an unconventional therapeutic target for ALS. He demonstrated that modulation of the spliceosome mitigates neurodegeneration and rescues TDP-43 pathology and dysfunction in models of diverse forms of ALS. Dr. Linares' science was recognized by an Eli and Edythe Broad Fellowship Award, a Barber ALS Research Award from the ALS Network, and a Merit Abstract Award from the International Society for Stem Cell Research. The findings from this work have the high potential to be translated to ALS patients in the clinical setting. Consequently, two companies in the life sciences sector are currently conducting investigational new drug (IND) enabling studies against these novel targets as part of their ALS drug development program.

**Apprenticeship Bridge to
College (ABC)
High School Program**

SHARAN BIR
ABC PARTICIPANT 2025

Since I was young, I have known that I wanted to gain an education and career in the medical field. Whenever I was asked what I wanted to be when I grew up, that was the answer. This decision was driven based on the experiences of others I saw around me, such as the lack of access to affordable healthcare many have been exposed to, and the want to help those in that situation.

As a participant in this program, I strive to make the greatest positive impact on my peers and overall community. Later this year, I will be majoring in biochemistry at UCLA, and eventually hope to pursue a degree in medical school. I hope to be able to do community work as well as learn about the impact that the medical field has on society. As such, I am extremely grateful for this opportunity to research at Loma Linda's ABC Program, not only for their fascinating projects, but also for their focus on prevalent health disparities around the world.

I would like to sincerely thank Dr. Carlos Casiano for welcoming me into his lab and providing me with the opportunity to learn more about the research process and scientific topics at hand, as well as Pedro Ochoa and Adelaide Makamure for their guidance in completing my project.



**NOVEL ROLES OF LEDGF/P75 IN MODULATING PROSTATE CANCER RELATED
INFLAMMATORY PATHWAYS AND CANCER PATIENT RESPONSE TO
IMMUNOTHERAPY**

Janani Nagasubramanya, Sharan Bir, Pedro T. Ochoa, Adelaide V. Makamure, Evelyn S.
Sanchez-Hernandez, Kai Wen Cheng, Zhong Chen, Issac Kremsky, Charles Wang,
Carlos A. Casiano

Center for Health Disparities and Molecular Medicine, Department of Basic Sciences, Loma
Linda University School of Medicine, Loma Linda, California, USA

Prostate cancer (PCa) is the second-leading cause of cancer death among men in the United States, with African American (AA) men experiencing a higher incidence and mortality rate compared to European American men. Moreover, AA men are more frequently diagnosed with aggressive forms of PCa, which reduces treatment options, particularly as tumors develop resistance to therapies. Unraveling the mechanisms behind PCa therapy resistance is vital for the creation of new therapeutic strategies. The lens epithelium derived growth factor p75 (LEDGF/p75) is a stress oncoprotein that contributes to cancer chemoresistance and tumor aggressiveness through its ability to tether oncogenic transcription factors to active chromatin, promote RNA-loop resolution at transcriptionally active sites, enhance DNA repair, and maintain genomic integrity. Our recent studies also demonstrated that LEDGF/p75 is upregulated in docetaxel (DTX)-resistant PCa cells and contributes to chemoresistance by regulating gene pathways associated with stress survival, DNA repair, and cell cycle progression. We hypothesized that its silencing in chemoresistant PCa cells may also alter immune-related gene pathways. Knockdown of LEDGF/p75 in chemoresistant PCa cells followed by RNA-seq analysis led to the identification of 970 differentially expressed genes (DEGs). Gene set enrichment analysis (GSEA) revealed a role

for LEDGF/p75 in modulating gene pathways associated with lymphocyte and inflammatory responses, since its downregulation led to upregulation of several inflammation-related genes including *IL7R*, *IL18*, *SWAP70*, *BMI-1*, *ULBP2*, etc. Protein expression of these genes was validated by Western blotting in PCa cells. Some protein expression levels did not align with the RNA-seq data, highlighting the need for validation through RT-PCR and CRISPR knockout of LEDGF/p75. High expression of most of these inflammatory genes also correlated with better overall survival of cancer patients receiving PD-L1 immunotherapy, as revealed by KM Plotter-Immunotherapy analysis. We conclude that LEDGF/p75 contributes to the negative regulation of inflammatory gene pathways. Understanding LEDGF/p75's role in cancer immunity may provide new insights into its immunomodulatory functions and its influence on cancer patient response to immunotherapy.

CAROLINE CORONADO
ABC PARTICIPANT 2025

When I applied to the ABC Program at Loma Linda University, I did not know what to expect, but I am thankful I took a chance. Participating has opened my eyes to the vast world of science, research, and medicine, and has made me even more excited for the future.

I am a rising homeschooled junior currently working toward completing my associate's degree in biology before graduating high school. Being homeschooled has allowed me to explore many different fields, but I always come back to science.

I serve as Vice President at my local co-op, intern at a Christian nonprofit in Riverside, and have accumulated many volunteer hours serving my community. Outside of academics and service, I love exercising, playing piano, reading, traveling, and learning about diverse cultures. I also play competitive cornhole and am an ACO Western Junior Champion.

In the future, I aspire to become a surgeon who serves both internationally and across hospitals in the United States. My long-term goal is to open my own practice, where I can combine clinical excellence with compassion. I am especially drawn to the transformative impact of surgery and am passionate about exploring innovative ways to improve patient care.

This summer, I am working in Dr. Durán's lab. Our research focuses on breast cancer prevention and mitochondrial health. My favorite aspect of our work this summer has been combining my passion for cellular and molecular biology with real life problems, such as breast cancer, and learning many different laboratory techniques. I am incredibly grateful for Dr. Durán's guidance and support throughout this summer.



INVERSE CORRELATION BETWEEN β 2-ADRENERGIC RECEPTORS AND PARKIN EXPRESSION IN BREAST CANCER

Caroline R. Coronado^{1*}, Lia V. Hutchins^{1*}, Daisy De Leon¹, Alfonso M. Durán^{1,2}

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Breast cancer (BCa) remains the most diagnosed malignancy among women in the United States, with one in eight expected to develop the disease in their lifetime. While genetic risk factors, including BRCA1/2 mutations, have been extensively characterized, the role of non-genomic factors in BCa pathogenesis is less understood. Emerging evidence suggests that overactivation of the sympathetic nervous system (SNS) contributes to BCa initiation, progression, and tumor aggressiveness; however, the precise mechanisms remain elusive. In other conditions, such as type II diabetes, excessive SNS activity has been linked to mitochondrial dysfunction, raising the possibility of a similar mechanism in BCa. Parkin, an E3 ubiquitin ligase, plays a central role in mitochondrial quality control and homeostasis. In this study, we investigated the relationship between β 2-adrenergic receptor (β 2-AR) abundance, a biomarker of SNS overactivation, and Parkin expression in BCa tissues and cell models. Using western blotting, immunohistochemistry (IHC), and cell culture experiments, we identified an

inverse relationship between β 2-AR levels and Parkin expression. These findings suggest that increased SNS signaling may downregulate Parkin, thereby compromising mitochondrial health and potentially promoting BCa tumor progression. If confirmed in larger clinical studies, these results highlight a novel mechanistic link between SNS overactivation and mitochondrial dysfunction in BCa. Targeting SNS signaling or restoring Parkin function could represent promising strategies for preventing BCa, improving recovery, and enhancing treatment interventions.

YANIAH GAMBOA
ABC PARTICIPANT 2025

I am a student at Moreno Valley High School, in which I will be a senior beginning Fall 2026. After graduation, I plan to major in Molecular Biology. I aspire to become a physician working in endocrinology in the future.

Throughout this summer, I have grown a newfound fondness for research. Every day was exciting as I encountered different techniques, protocols, and lab brains. While new and confusing, I was able to appreciate the scenes of research as I realized the greatness that I was able to take part in. I am very grateful to have been a part of a lab that was centered on values of compassion and innovation to bring new discoveries to better the field of physiology. It was much more than simply following the procedure but also understanding the entire picture. While I took part in a pursuit of knowledge, I also saw how the research conducted is a commitment to improving lives. As I learned and observed the incredible individuals in the lab, I came to the understanding that the true essence of research is not the investigation or the experimental design. Rather, it is a contribution to building a future where every human has the opportunity to experience the best that science can offer.

Thank you to Dr. William Pearce, Danielle Lonie Malivert, Desirelys Ortiz Martinez, and James Williams for guidance throughout this entire learning process. It was an honor to take part in this lab.



**MITOCHONDRIAL CHARACTERISTICS AND CONTRACTILE FUNCTION ARE
SIGNIFICANTLY DEPENDENT ON OXYGEN CONCENTRATION AND ARTERY TYPE
IN NEONATAL CEREBROVASCULAR SMOOTH MUSCLE**

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Neonatal hypoxia induces vascular maladaptation that includes impaired functional maturation, leading to significant complications, particularly in the cerebral circulation. Despite the well-established causes of neonatal hypoxia, the mechanisms underlying cerebrovascular responses to neonatal hypoxia remain poorly understood. Accumulating evidence indicates that mitochondria are essential regulators of vascular development and function. However, their role in hypoxia-induced neonatal cerebrovascular adaptation remains unclear. Recognizing the essential role of mitochondria in vascular maturation, this study examines the effects of graded hypoxia on mitochondrial characteristics and contractility in ovine neonatal middle cerebral arteries (MCA) and posterior cerebral arteries (PCA). To determine the effects of graded hypoxia on mitochondria, mitochondrial DNA (mtDNA) copy number per cell and Succinate Dehydrogenase A (SDHA) per mtDNA copy number (a measure of mitochondrial size), were measured via PCR and immunoblotting, respectively. Additionally, vascular stress-strain relations were characterized using measurements of passive wall stress, myogenic wall stress, and depolarization-induced stress. Relative to 18%O₂, the greatest reduction in mtDNA copy number

per cell was 43% at 5%O₂ in MCA and 44% at 2%O₂ in PCA. SDHa per mtDNA copy number increased from 7.6 to 43.5 in MCA at 18%O₂ and 5%O₂, respectively and 21.3 to 72.7 at 18%O₂ and 2%O₂, respectively in PCA. Correspondingly, reducing O₂ from 6% to 5% increased depolarization-induced active stress by 21% and 261% in MCA and PCA respectively. Overall, our results suggest that graded hypoxia increases contractility while simultaneously decreasing mtDNA copy number per cell and increasing SDHa per mtDNA copy number in the neonatal cerebral circulation in an artery dependent manner. This study offers new insights into the mechanisms that mediate neonatal cerebrovascular adaptation to hypoxia.

AVA GATILLO
ABC PARTICIPANT 2025

As a rising senior at Loma Linda Academy High School, I've developed a deeper understanding of what it truly means to help others. Through extracurricular activities like serving in class office and volunteering with children, I've learned how to lead, contribute my ideas, and apply my creativity to inform and support those around me. I especially enjoy babysitting and reading to kids at the library. In my free time, I love drawing, creating crafts for my friends, and spending quality time with my family.



Ever since I was little I've dreamed of becoming a doctor because of the meaningful impact they have on people's lives. My goal is to become a Dermatologist, someone who not only treats skin conditions but also supports patients emotionally, recognizing how closely skin health is tied to mental well-being. I am committed to keeping God at the center of my journey, trusting Him to guide me as I follow His purpose for my life.

This summer, I had the incredible opportunity to work in Dr. Wiafe's Health Geoinformatics Laboratory at Loma Linda University. There, I explored the field of public health and learned how spatial data and mapping tools can be used to understand and improve population health. I focused specifically on access to mental health care across California. I'm truly grateful to Dr. Wiafe for this experience, which not only expanded my knowledge, but also deepened my passion for addressing health challenges with compassion and creativity.

**ADDRESSING MENTAL HEALTH NEEDS AND PROVIDER
SHORTAGES ACROSS CALIFORNIA**

Ava Gatillo, Seth Wiafe

Center for Health Disparities and Molecular Medicine, Health Geoinformatics Lab, School of
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Disparities in the distribution of mental health care providers continue to pose a significant challenge across California. Access to mental health services remains uneven, particularly in rural, low-income, and underserved urban communities, many of which experience disproportionately high rates of mental illness. This project utilizes ArcGIS Pro 3.3 to examine the spatial relationship between the prevalence of depression and federally designated Mental Health Professional Shortage Areas (MHPSAs) across the state. Data on MHPSAs were obtained from the Health Resources and Services Administration (HRSA), while depression prevalence estimates were sourced from the Centers for Disease Control and Prevention (CDC). Depression rates were mapped at the census tract level using choropleth symbology, and MHPSAs were delineated using polygon feature classes. These two spatial layers were then overlaid to assess geographic convergence. To identify statistically significant spatial clusters, the Getis-Ord Gi* hot spot analysis tool was applied, revealing areas with unusually high (hot spots) and low (cold spots) rates of depression. Results indicated substantial overlap between depression hot spots and MHPSAs, particularly in Northern California, the Central Valley, and the Inland Empire, highlighting regions of compounded vulnerability. These findings underscore the urgent need for targeted policy interventions. Specifically, state and local policymakers should prioritize funding for training and workforce development programs that prepare mental

health professionals, including therapists, psychologists, and psychiatrists, to serve in high-need areas. By directing resources to regions with both elevated mental health burdens and provider shortages, California can advance efforts to improve access, equity, and outcomes in mental health care delivery.

BERNICE JUAREZ
ABC PARTICIPANT 2025

Attending Arroyo Valley High School in San Bernardino and being enrolled in the International Baccalaureate program, I learned firsthand how health and economic disparities affected different communities. My mother, being the first in her family to pursue higher education, pushed me to work hard in school. Her passion and determination in the nursing field influenced me to learn deeper into the different fields of medicine. Not only this, but the lifelong questions I've held about neurons and how the body can possibly turn against you have also influenced me in choosing to spend my summer in the Apprenticeship Bridge to College program here at Loma Linda University.



At my high school, I spend most of my time learning about social, economic, educational, and health disparities in different communities through my courses. I am a multi-sport athlete who has dedicated their time and commitment to track and field, cross country, and cheerleading. I am also an advocate for youth participation in voting as well as being a part of conversations regarding political decisions. This is why I am also dedicated to a youth club called Students for Change/ICUC, our mission being to target youth and get them involved in decision making regarding our community and schools.

I give my greatest appreciation to Doctor Marino De Leon as well as Francis Zamora for taking the time to teach me patiently and giving me the opportunity to research what I am passionate about.

**INTERPLAY OF FATTY ACID BINDING PROTEIN 5 AND AUTOPHAGY
DURING PALMITIC ACID-INDUCED LIPOTOXICITY**

Bernice Juarez, Francis Zamora, Addie McIver, Viet Hoang Dinh, Jo-Wen Liu, Marino De Leon

Center for Health Disparities and Molecular Medicine, Basic Sciences,
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Fatty acid binding protein 5 (FABP5), an intracellular lipid chaperone, is upregulated during cellular stress, including palmitic acid-induced LTx (PA-LTx) and nerve injury. Previously, we showed FABP5 functions as an antioxidant in neuronal cells, supporting axonal growth and nerve regeneration. Polyunsaturated fatty acid Docosahexaenoic acid (DHA) also regulates FABP5, which is associated with improved functional recovery following injury. Autophagy, a key process for degrading damaged cellular components, is disrupted during PA-LTx and nerve injury, with enhanced autophagy promoting cell survival. Emerging research suggests interactions between FABP5 and autophagy-related proteins. Therefore, this study investigates the functional role of FABP5 in autophagy during PA-LTx injury in immortalized Schwann cells (ISC). PA-LTx was induced by treating ISCs with 300 μ M PA:150 μ M BSA for 24–48 hours, and 50 μ M DHA co-treatment inhibited LTx. Chloroquine (CQ) and rapamycin served as controls for autophagy flux inhibition or induction, respectively. Prior to treatments, FABP5 was silenced by siRNA. Western blot and RT-qPCR were used to confirm knockdown efficiency and to monitor autophagy by measuring LC3-II and p62/SQSTM1 protein levels, and expression of autophagy-related genes. WST-1 assay assessed cell viability. At 24 hours, PA and CQ increased FABP5

expression and increased LC3-II and p62, which is consistent with increased oxidative stress and impaired autophagy flux. DHA co-treatment normalized these markers, suggesting enhanced autophagic clearance and reduced stress. Interestingly, FABP5 knockdown reduced LC3-II and p62 accumulation in response to PA and CQ, suggesting that FABP5 levels may affect autophagosome formation or cargo degradation. FABP5 silencing also decreased cell viability in PA/DHA and CQ groups. These findings suggest a potential role for FABP5 in autophagy dynamics, which regulates cellular homeostasis during lipotoxic stress.

KHAVEEN KABILAN
ABC PARTICIPANT 2025

My love of the brain and my willingness to research such a mysterious organ have led me to seek mentoring through this profound opportunity. This program has taught me the essentials of research, whether it involves attending seminars that ignite my curiosity or working in a lab where breakthroughs happen. Not only did it teach me research, but it also gave me a basic understanding of using programming in science. As an ABC student, I can confidently say, "My passion for neurosurgery and neuro-research has gained a new level of understanding."

As I graduate from Redlands Adventist Academy next year, I plan to major in neuroscience both in Pre-med and Medical school. Although I aspire to become a neurosurgeon, I plan to pursue a PhD later in my career. With that PhD, I plan to research brain waves and Deep Brain Stimulation.

Our research was on calcium oscillations in the basilar artery in hypoxic and normoxic sheep. Our goal is to educate mothers in high-elevation environments about the risks of giving birth in such areas. This research was more than just a job; it was a way to make a difference in many lives. I cherished every moment I spent at the lab as it brought me immense joy. This feeling, mixed with curiosity, is the reason I strive to do more in my field.

I'm deeply grateful to Dr. Sean Wilson for welcoming me into his lab and supplementing my curiosity. His magnificent mentoring has brought me a step closer to making a significant impact in neuroscience!



**IMPACT OF LONG-TERM HYPOXIA ON BASILAR ARTERIAL MYOCYTES: CALCIUM
OSCILLATIONS FROM NEWBORN SHEEP**

Khaveen Kabilan, Grant Thomas, Devin Wilson, Eris Albert-Minkler, Arlin B Blood, Lubo
Zhang, Christopher G Wilson, and Sean M Wilson
Center for Perinatal Biology, AIM Core Facility, School of Medicine, Loma Linda University,
Loma Linda, CA

Many studies have shown that giving birth in a high elevation environment result in the newborn having neurodevelopmental disorders from a dysregulation in cerebral blood flow (CBF) due to the long-term hypoxia in such an environment. Our goal is to learn more about why this event happens. We hypothesize that the dysregulation of L-type calcium channels (Ca_L) in vascular myocytes contributes to impaired CBF. We chose to focus on Ca_L because these channels contribute to calcium oscillations in various myocytes, and they are known to be developmentally regulated and affected by long-term hypoxia before birth. To study the oscillatory events, we examined the basilar arteries of hypoxic and normoxic newborn sheep. Arteries were placed in a physiological buffer (control) and then treated with 30 mM K⁺ (30K), with or without 1 μmol nifedipine (NIF). Full frame (512X512) fluorescence of Fluo-4, representing calcium signals, was recorded over 5 minutes at 1 Hz using confocal microscopy techniques. Pipeline with region of interest (ROI) detection had an average specificity of 97% and a sensitivity of 81%. The results show that myocytes from hypoxic sheep had fewer oscillatory events compared to the normoxic

sheep. We also noticed that NIF increased oscillatory activity in myocytes from normoxic animals. This discovery was highly unexpected and suggests that hypoxia may lead to modifications in the structure and function of the channels. Future studies will focus on determining the mechanisms underlying the agonist-like properties of NIF towards the calcium oscillatory activity observed in the current series of studies.

KAAVYA KAKA
ABC PARTICIPANT 2025

I attend Rancho Cucamonga High School, and I am an incoming senior in the class of 2026. I plan to major in biology, and I aspire to become a neonatologist. I was born and raised in California. In my free time, I volunteer at Pomona Valley Hospital Medical Center, hike, play with my dog Oreo, and draw.

I have spent this past summer working in Dr. Mata-Greenwood's lab, alongside PhD student Nana Anti. The research that we have undertaken is about glucocorticoid homeostasis (GR) in pregnant women, ranging from gestational diabetics to women who were diabetic prior to pregnancy. The most interesting part of research and science to me is how interconnected everything is, and the importance of looking at the bigger picture in order to understand how everything works together.

Through this program, I have gained hands-on lab experience and a deep understanding of glucocorticoid receptors and how glucocorticoid (cortisol) levels affect multiple aspects of our daily lives because of their prevalence in the human body. I have learned that mistakes in the lab are opportunities to learn, grow, and improve with every experiment.

Thank you to Dr. Eugenia Mata-Greenwood's lab for welcoming me and providing me with this incredible opportunity.



**PLACENTAL GLUCOCORTICOID HOMEOSTASIS DYSREGULATION IN
PREGNANCIES COMPLICATED WITH MATERNAL DIABETES**

Kaavya Kaka, Nana A. O. Anti, Ciprian Gheorghe, Eugenia Mata-Greenwood

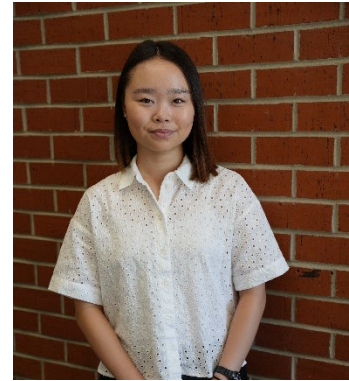
Center for Health Disparities and Molecular Medicine, Lawrence D. Longo Center for Perinatal

Maternal diabetes complicates approximately 5-9% of all USA pregnancies and is associated with fetal and neonatal complications such as macrosomia and respiratory distress. It has been hypothesized that cortisol resistance partially mediates the effects of maternal diabetes on the fetus and neonate. Therefore, the aim of this study was to investigate the levels of cortisol and cortisol-relevant gene expression in placentas of diabetic (n=16 gestational diabetes, and n=10 pre-diabetic, 50% female) and obese-control pregnancies (n=16, 50% female). Umbilical cord serum was studied for cortisol levels by ELISA, while placental biopsies were used to investigate the expression of the glucocorticoid receptor isoforms alpha, gamma, P, and beta, and that of cortisol metabolizing enzymes 11-beta-hydroxysteroid dehydrogenase 1 (activating), and 2 (inactivating) by qPCR and western blotting. Our results confirmed that all placentas present a higher ratio of the cortisol inactivating enzyme 11beta-hydroxysteroid dehydrogenase 2 than the activating counterpart 11beta-hydroxysteroid dehydrogenase 1. Diabetic pregnancies were characterized by higher birthweights and birthweight percentiles, with pre-diabetic pregnancies showing the worse outcomes. Interestingly, there were no differences in placental expression of cortisol metabolizing enzymes or umbilical cord blood cortisol levels between gestational diabetes and obese non-diabetic controls. However, there was a trend on pre-diabetic pregnancies to show lower cortisol levels and lower ratios of cortisol – birthweight percentiles. Lastly, there was a significant upregulation of placental 11-beta-hydroxysteroid dehydrogenase 1 expression in pre-diabetic pregnancies. Altogether, these data suggest that

pre-diabetes dysregulates fetal/placental glucocorticoid homeostasis characteristic of reduced glucocorticoid sensitivity.

CLARA LEE
ABC PARTICIPANT 2025

Every morning on weekdays, I drive 25 minutes from my home in Rancho Cucamonga, California to Loma Linda University to participate in the ABC summer program. On the weekends, I am active in my community through volunteering at a local senior home, and teaching as a certified instructor at a swim academy. This autumn, I will return to Los Osos High School as a senior to continue to be a part of the varsity speech and debate team, and to serve as the president of the Medical Opportunities club. Exploring my various interests has helped me to understand that my passion and dedication are geared toward service, and after seven weeks of conducting immersive research in an accelerated academic environment, my vision for the future has been clarified. My goal is to major in molecular and cellular biology in college. Then, I am hoping to apply to an MD/PhD program in order to improve healthcare by working with patients in the clinical setting, and through carrying out innovative research in the laboratory.



After receiving an unexpected appendectomy at Loma Linda Children's Hospital in 2018, I never thought I would return to the University campus in 2025 to conduct biomedical research in a lab just a ten-minute walk away from my former hospital room. I send my sincerest gratitude to Dr. Langridge and Miss Valencia Green for providing me with continuous guidance when working on my project to construct a novel vaccine to fight the SARS-CoV-2 virus.

A NOVEL VACCINE FOR ARRESTING RESPIRATORY VIRUS TRANSMISSION

Clara Lee, Valencia Green, Nathan Wall, Bela Denes, Ryan Fuller,
Anthony Firek, and William Langridge

Center for Health Disparities and Molecular Medicine, Division of Biochemistry,
School of Medicine, Loma Linda University, Loma Linda, CA.

Current mRNA vaccines fail to block the transmission of respiratory virus SARS-CoV-2. The CDC estimates that in the United States from October of 2024 to July of 2025, over 10 million cases of infection occurred, more than 300,000 patients were hospitalized, and over 35,000 people died (2025). To address this issue, we constructed a DNA-based vaccine in plants that can block viral infection of the mucosa, the site of virus entry into the human body. Leaf and stem tissues from tobacco, tomato, and potato plants were genetically modified with a vector containing the CTB-SARS-CoV-2-ACE-2-RBD vaccine gene through transformation by *Agrobacterium tumefaciens* (*A.t*). The bacteria-infected explants were then transferred to growth-inducing medium containing Kanamycin to select for transformed tissue expressing the NPT II gene marker, which is located in the transferred DNA (T-DNA). After several weeks of incubation, tobacco was the first of the three species of Solanaceae plants to be transformed and to produce shoots. The shoots from the explants were excised and moved onto root induction medium for regeneration of intact plants. In the future, the intact plants will be transferred to soil for growth to maturity before examination of the leaves for the presence of vaccine proteins. If the proteins are detected, they will be isolated and purified for assessment of protection efficacy against viral infection by SARS-CoV-2 variants in cell cultures and in animal models. This research supports the long-term potential of utilizing plant-derived vaccine proteins in humans to generate mucosal immunity

against respiratory viruses, such as SARS-CoV-2, through the intake of genetically engineered plant material.

VIVIAN MEDRANO
ABC PARTICIPANT 2025

Biomedical research is something that is truly intriguing to me because of the amount of time and effort put into it. Biomedical research requires such a rigorous and altruistic heart from the people who decide to put in all of their effort to research various medical situations people are struggling with in order to hopefully find a result that can change lives. Biomedical research, to me, is something that is beyond an interest, research is something that has been applied to my everyday life. As someone who has a sibling with a neurological disorder, I've seen how much biomedical research has done to contribute to improving my sibling's life with his disorder.



I am an upcoming senior at Hemet High School. At HHS I dedicate a lot of my time to academics, but I am also very involved in the band program. I love performing music to create a fun environment for the community around me. After high school I plan to pursue a career as a neurologist. I plan to accomplish this by attending a 4-year college, majoring in the field of cognitive science with a specialization in neuroscience. Throughout college, I hope to participate in research and other opportunities around me to strengthen my understanding in neurology and research. Once I complete college, I hope to return to LLU to pursue a degree.

Thank you to Dr. Christopher G. Wilson's lab for taking the time to teach me, and provide me with amazing research experience.

THE EXPRESSION OF CASPASE-9 IN THE HIPPOCAMPUS OF PRETERM HYPOXIC-ISCHEMIC ENCEPHALOPATHY (PHIE) MICE

Vivian Medrano, Nicholas Iwakoshi, Tyler C. Hillman, and Christopher G. Wilson

Center for Health Disparities and Molecular Medicine, Lawrence D. Longo MD, Center for Perinatal Biology, Loma Linda University, Loma Linda, CA

Hypoxic-Ischemic encephalopathy (HIE), a form of early-life brain injury in babies, is caused by oxygen deprivation and reduced blood flow to the brain. HIE can result in chronic inflammation in the brain, leading to possible long-term injuries, including developmental delays and cerebral palsy. HIE can also appear in premature infants, referred to as preterm HIE (pHIE). Because this disease is difficult to detect before injury occurs in neonates, we used a pHIE mouse model to assess brain injury. We quantified the expression of caspase-9, a marker of apoptosis, in the hippocampus of pHIE mice, to observe how HIE hippocampal neurons. We looked at the hippocampus because it's crucial for long-term memory formation, and normal cognitive function. Previous research has shown that HIE affects the cortex and can cause injury to the hippocampus, but no studies have focused on caspase-9 as a marker of injury in the hippocampus of pHIE mice. In our study we compare the expression of caspase-9 in the hippocampus of pHIE and control mice. In our model a pregnant mouse is injected with lipopolysaccharide (LPS) to create an inflammatory response. Then dam and pups are exposed to chronic hypoxia for 6 days. The controls are Saline hypoxia (instead of LPS the mother is injected with saline) and naive mice. To quantify caspase-9, we used unbiased stereology for counting cells expressing caspase-9. **We hypothesized that pHIE preferentially affects the**

most hypoxia sensitive region of the brain, the hippocampus. We expect to see increased caspase-9 expression in the hippocampus of pHIE mice. Our work will help advance understanding of pHIE's effects on hippocampal development in neonates for further research and clinical practice.

MANI MOMENI
ABC PARTICIPANT 2025

I am currently a rising junior at Redlands High School. After high school, I hope to earn a bachelor's degree in a science-related field, with a long-term goal of obtaining an MD/PhD. My interest in this path was ignited in 2020 while visiting my family in Iran. During my time there, I witnessed the profound challenges of Alzheimer's disease. Although I knew my grandfather had been diagnosed with Alzheimer's, I didn't quite know all that it would entail. It was heartbreaking to see him greet my mother without recognizing her as his own daughter. From that moment on, I became motivated to make a difference in the lives of individuals and families affected by this disease.



This motivation led me to gain previous lab experience working on Alzheimer's and L.A.T.E disease at UCI. To deepen my understanding in this field, I am spending the summer working in the neurodegenerative diseases laboratory at Loma Linda University under the guidance of my amazing mentors, Dr. Soriano, Kayla Sanchez, and Jacob White.

My current project involves bioinformatics analysis focused on inherited prion diseases, such as Creutzfeldt-Jakob Disease and Fatal Familial Insomnia. My goal is to understand the early mechanistic pathways that precede clinical symptoms. I cannot thank Soriano Lab enough for welcoming me and teaching me what it means to work in this important field.

**CHARACTERIZING EARLY PATHWAYS IN HEREDITARY PRION DISEASES VIA
TRIOSIG: A NOVEL FERROPTOSIS DATABASE**

Mani Momeni ¹, Kayla Sanchez ¹, Jacob White ¹, Antonio Currais ², Pamela Maher ², Salvador Soriano ¹

¹Department of Pathology and Human Anatomy, Loma Linda University, School of Medicine,
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Prion diseases remain among the most understudied and challenging to investigate due to the unique nature of their infectious model. These diseases are characterized by progressive neurodegeneration and are always fatal. Although prion diseases can be acquired through various routes, the hereditary form enables us to understand the genetic mechanism of pathogenesis. Hereditary prion diseases are caused by mutations in the PRNP gene, resulting in the misfolding of the normal prion protein (PrP^C) into the disease-associated form, known as prion-scrapie PrP^{Sc}. PRNP mutations result in different prion disease variants, each with distinct cellular and regional variabilities. Two examples include Creutzfeldt-Jakob disease (CJD), which has over 40 associated variants, and Fatal Familial Insomnia (FFI), which has two variants. In both conditions, survival rates are approximately one year after the onset of symptoms. Although the function of PrP^C remains under investigation, studies have shown that mutations lead to metal ion imbalance and increased levels of lipid peroxidation, hallmarks of ferroptosis. However, the link between prion disorders and ferroptosis has not been thoroughly explored. One reason we believe this connection remains unclear is the lack of neuron-specific ferroptosis signatures. To address this gap, our lab utilized a novel ferroptotic database called TrioSig, developed by the Maher lab at the Salk Institute. As such, we hypothesize that PRNP mutations increase vulnerability to ferroptosis and these signatures may be uncovered using the TrioSig database. To test this, we performed a bioinformatic analysis of various neuronal cell types in pre-

symptomatic CJD and FFI mouse models. We found that ferroptosis was significantly upregulated within somatostatin-expressing (SST) cells, a subtype of inhibitory neuron found in the cortex, in FFI. These findings provide the first evidence that ferroptosis is differentially activated across prion diseases, suggesting a potential therapeutic target for slowing neurodegeneration in the future.

KEVIN NGUYEN
ABC PARTICIPANT 2025

Growing up, my parents rarely discussed their immigration story and the Vietnam War. However, there was always a sense of pride combined with sorrow when they would open up about their childhood. For example, my dad loves to “shower in the rain”, or had to “collect food left in the trashcan” to fill up his belly. These stories, though told jokingly, made me empathetic to their childhood. As an American, I do not have to sacrifice my health to survive. And because of this, I have become passionate about providing accessible and affordable public health for all.



I am currently a rising junior at San Geronio High School in San Bernardino. I’ve seen firsthand how health disparities affect underserved populations through my education and in real-life experiences. Because of this impact, I carry these experiences in my actions. For example, I serve as the president of two community-service-based organizations, help distribute food for the hungry at local food drives, and teach children at my church about the benefits of public health. These examples show my passion for advocacy and my commitment to serving the underserved. With trust in God, I will pursue a higher education in public health and build a career in the medical field.

Thank you to Dr. Erik Behringer’s lab for welcoming me in, patiently guiding me, and answering my never-ending questions. I’m also grateful to the ABC Program for allowing me to learn, grow, and showcase my capabilities.

EXPRESSION OF K⁺ CHANNELS IN THE HIPPOCAMPUS DURING AGING & ALZHEIMER’S DISEASE

Robert Lister³, Kevin Nguyen², Fritz E. L. Miot¹, Zion Shih¹, Phoebe Chum¹, Erik Behringer¹

Department of Basic Science, School of Medicine, Loma Linda University¹; San Geronio High School²; Oakwood University³

Alzheimer's disease (AD) is a neurodegenerative illness that impairs memory and motor skills, primarily due to damage to the hippocampus and cerebral cortex. With a global impact on 1 in 10 men and 1 in 5 women, AD is a significant health concern. Emerging evidence demonstrates AD as a “channelopathy”, whereby normal regulation of K⁺ channels may be impacted to influence plasma membrane excitability and energy transport. In particular, the inward rectifying potassium (KIR) channel and small conductance calcium-activated potassium (SKCa) channels may coordinate blood flow with metabolic rate in the tissue parenchyma to ensure optimal perfusion of the brain. The expression, localization, and function of SKCa and KIR ion channels in the hippocampus during AD pathology is unclear. Thus, we worked on the hypothesis that hippocampal K⁺ channels may show differential expression among experimental AD mice relative to wild-type controls. Using the 3xTg-AD animal model (8 months, male & female), brains were perfused, dissected, and flash-frozen to create 20 µm coronal sections of the hippocampus. Immunohistochemistry (IHC) procedure was used, and sections were stained with hematoxylin to illustrate individual cell nuclei within the brain tissue. Images are captured with a light microscope and quantitatively assessed. If expression and localization patterns are significantly different among AD and wild-type mice, the results

may help explain the deterioration of long-term potentiation and memory in the hippocampus. If similar, then we would have to consider other possibilities such as select post-translational modifications of channel activity (e.g., phosphorylation, oxidation) rather than expression *per se* or perhaps other signaling mechanisms and/or protein markers altogether. Regardless, we expect that our results will enhance insight into novel therapeutic strategies to combat illnesses such as stroke and AD.

SONIA PARANDE
ABC PARTICIPANT 2025

Growing up, summers in India meant time with my grandfather, a doctor who served rural communities. Although I was young, he let me help in small ways, organizing cabinets, comforting children receiving vaccines. What stayed with me most was that no matter their financial situation, my grandpa made sure no one left without receiving proper treatment. One of the biggest lessons I learned from him was that it is often the individuals that are the most hesitant to reach out for help that need it the most.

These experiences guided me towards volunteering at hospitals such as Temecula Valley Hospital and Kaiser Permanente. Being in clinical settings, I observed how there was a consistent flow of individuals into the ER. Some young, some homeless, some with family and others alone. Volunteering at the hospital showed me that everyone has a story which deserves to be heard. I truly believe that the cause of suffering is the lack of love. My goal is to become an Emergency Medicine Doctor and be in the front-line treating patients with deserved love and care.

I am a senior at Chaparral High School in Temecula. This summer, I am honored and grateful to work under Dr. Wiafe in the LLU Health Geoinformatics Laboratory to track patterns within healthcare to aid in effective public health policy development. Understanding spatial relations within medicine has deepened my interest in how communities experience illness and will aid in providing effective healthcare in the future.



AVOIDABLE HOSPITALIZATIONS AND PRIMARY CARE ACCESSIBILITY IN CALIFORNIA: A SPATIAL ANALYSIS OF TYPE 2 DIABETES PREVALENCE

Sonia Parande, Seth Wiafe
Center for Health Disparities & Molecular Medicine, Geoinformatics Laboratory,
School of Public Health, Loma Linda University, Loma Linda, CA

As of 2023, diabetes remains the seventh leading cause of death in the United States. Type 2 diabetes, a chronic condition, occurs when the body fails to produce enough insulin or cannot effectively utilize the insulin it produces. When left unmanaged, it significantly increases the risk of cardiovascular disease, stroke, and neuropathy. Poor diet and sedentary lifestyles are major contributors to the growing prevalence of this disease. Recent trends show persistently high rates of avoidable hospitalizations for diabetes, often signaling limited access to primary care, inefficiencies within the healthcare system, and broader structural inequities. This study used Health Professional Shortage Area (HPSA) data from the Health Resources and Services Administration (HRSA) and diabetes prevalence estimates from the CDC's PLACES project to conduct spatial analysis at the census tract level using ArcGIS Pro 3.3. Getis-Ord Gi* Hot Spot Analysis identified statistically significant clusters of elevated diabetes prevalence in regions such as the Inland Empire and California's Central Valley. Overlay analysis revealed strong spatial convergence, at a 99% confidence level, between these high-prevalence areas and primary care provider shortages, highlighting regions of compounded vulnerability. Furthermore, many of the most affected communities are located more than 20 miles from the nearest primary care facility, underscoring geographic barriers to timely and preventive care. To address these disparities, policy interventions should prioritize expanding the healthcare

workforce in underserved areas and integrating comprehensive nutrition and healthy lifestyle education into school curriculum. Together, these strategies can help reduce the burden of Type 2 diabetes and improve long-term health outcomes in high-need communities.

SANJANA RAMESH
ABC PARTICIPANT 2025

I am a rising senior at Arrowhead Christian Academy. From an early age, I developed an ardent desire to be a pediatrician, driven by compassion and empathy for little kids. John 15:12-13 revealed to me the depth of Jesus' sacrificial love, which inspired me to positively impact the lives of children in my community, bringing purpose and meaning to my own life. These dreams urged me to pursue a growth mindset, higher-order thinking, and lifelong learning.

I participated in a research internship focused on stem cell therapies in regenerative medicine. This experience evoked my interest in conducting a systematic literature review on the effect of a low FODMAP diet on gastrointestinal symptoms and inflammatory markers in adult patients with IBD, awaiting to be published in a scholarly, peer-reviewed high school journal. As the director of events management at the Youth STEM learning leadership club and as the VP of the medical club, I raised health and wellness awareness events, supporting peers. Volunteering at LLU's UReach ministry, "Meals on Wheels," was a fulfilling experience in serving senior citizens in my community.

Through the ABC program, my pursuit of research and service grew stronger. I'm sincerely grateful to Dr. Salma Khan for welcoming me into her lab and mentoring me on my project on "double staining fluorescent immunohistochemistry to identify TGF-beta and PDL-1 in preserved anaplastic thyroid cancer tissue slides." I am extremely thankful to Romi Yamauchi, Joseph Cruz, and my peers for their continued support and guidance in this journey.



**CORRELATION OF SURFACE MARKERS AND DOWNSTREAM PATHWAYS IN
ANAPLASTIC THYROID CANCER TISSUE SAMPLES BY MULTIPLEX
IMMUNOHISTOCHEMISTRY**

Sanjana Ramesh, Joseph Cruz, Janice Pakkianthan, Andrea Shield, Alfred Simental, Salma
Khan

Center for Health Disparities and Molecular Medicine, Biochemistry, School of Medicine, Loma
Linda University, Loma Linda, CA

Anaplastic thyroid cancer (ATC) is a highly aggressive and undifferentiated malignancy, accounting for just 1–2% of all thyroid cancer diagnoses but nearly 50% of thyroid cancer-related deaths. Its exceptional resistance to conventional therapies, including radioiodine therapy, is primarily due to the absence of the sodium-iodide symporter. Our laboratory aims to identify overexpressed surface markers in ATC that can be exploited for radioligand-based theranostics, a dual diagnostic and therapeutic strategy wherein a radioisotope-bound ligand targets and destroys cancer cells while sparing normal tissues. Ideal targets are significantly overexpressed in ATC but minimally present in normal tissue. We previously identified programmed death-ligand 1 (PD-L1), L-type amino acid transporter 1 (LAT-1), and the vitamin D receptor (VDR) as promising markers overexpressed in ATC. PD-L1 enables immune evasion by binding to PD-1 on T cells. LAT-1 increases amino acid uptake by tumor cells. Vitamin D

binding protein (DBP) delivers metabolically active vitamin D to tissues, allowing VDR-modulated antitumorigenic activity. Although, resistance to vitamin D and non-genomic VDR signaling may contribute to tumor progression. Other molecules of interest include transforming growth factor-beta 1 (TGF- β 1), which can suppress tumor growth in early stages but promote cancer progression later; c-Myc, a transcription factor frequently upregulated in cancer; and AKT (protein kinase B), a key regulator of cell survival and proliferation. This study investigates the expression and potential interactions among PD-L1, VDR, LAT-1, c-Myc, TGF- β 1, AKT, and DBP in ATC tissue. Using multiplex immunohistochemistry on formalin-fixed paraffin-embedded ATC samples, we observed notable expression of PD-L1 and TGF- β 1 in the tumor microenvironment. All evaluated surface markers were consistently overexpressed in malignant regions compared to benign tissue, supporting their potential utility in radioligand-based theranostic applications.

ELIZABETH VIVANCO
ABC PARTICIPANT 2025

As a first-generation student, I have come to value hard work, faith, and compassion as pillars of both my education and personal growth. These principles have guided me through three years of rigorous coursework, where my curiosity for science continually exceeded what I had learned, leading me to the ABC Program, where my passion for science grew tremendously.

Now entering my final year at Beaumont High School, I serve as secretary of the Donor, Organ, and Tissue Club, where I lead public health advocacy focused on organ donation and Parkinson's disease. Over my junior year, I completed more than 190 hours of clinical internships at a medical weight loss clinic and a family clinic, applying skills from my three-year medical pathways program. I also engage in community outreach by distributing Christmas, Valentine's, and Easter gifts to underserved neighborhoods, as well as providing school supplies to families in need. Additionally, I support my church through Kids Ministry teaching and Praise Team worship.

These experiences have shaped my goal to make a meaningful impact in healthcare and become one of the first in my family to earn a bachelor's degree or MD. With a strong interest in pediatrics, I hope to become either a pediatric nurse (BSN) or a pediatrician, specializing in biology or public health. I express great gratitude to the program, my mentor Ann Morcos, and the various seminars for their patient mentorship and for educating me on the minuscule, yet intricate abilities of the body.



**FL-118: A NOVEL IAP INHIBITOR WITH POTENTIAL THERAPEUTIC EFFICACY IN
GEMCITABINE RESISTANT AND SENSITIVE PDAC CELL LINES**

Elizabeth Vivanco, Ann Morcos, Ryan Fuller, Joab Bustillos, Yeonkyu Jung, Nathan Wall
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Pancreatic Ductal Adenocarcinoma (PDAC) is the most prevalent and fatal type of pancreatic cancer, distinguished by its poor 5-year survival rate of only 13%. Despite advances in treatment, PDAC remains highly lethal, largely due to late diagnosis and its aggressive behavior, resulting in local invasion and widespread metastasis. This study evaluates the therapeutic potential of FL-118, a small-molecule inhibitor targeting several anti-apoptotic proteins, including survivin, XIAP, and cIAP2. We hypothesize that FL-118 will enhance proton radiation-induced cell death in the Gemcitabine-resistant MIA PaCa-2 cell line commensurate with that of the sensitive cell line and we will analyze this sensitivity by using colony formation assays combined with those that measure apoptosis (Annexin IV/7AAD) and cell cycle (PI). Gemcitabine-sensitive (MIA PaCa-2) and gemcitabine-resistant (MIA PaCa-2 GR) PDAC cell lines were assessed using FL-118. The proliferation assays revealed similar IC50 values in both MP2 and MP2-GR cell lines, indicating consistent potency despite Gemcitabine-associated chemoresistance. While FL-118 induced a non-significant increase in apoptosis in the GR cell line, it stimulated significant apoptosis in the sensitive parent cell. In addition, FL-118 induced significant levels of necrosis in both cell lines. Cell cycle analysis revealed a significant arrest in the G2/M phase and colony formation assays confirmed that FL-118 inhibits long-term colony formation. Importantly, its

combination with proton radiation further amplified its cell killing effects. These findings suggest that FL-118 overcomes gemcitabine resistance in PDAC by targeting key inhibitor of apoptosis proteins (IAPs), including survivin, XIAP, and cIAP2. Its efficacy in both gemcitabine-sensitive and -resistant cell lines, along with enhanced cytotoxicity in combination with proton radiation, supports its continued development as a promising therapeutic strategy for PDAC.

CRYSTAL WEI
ABC PARTICIPANT 2025

During my freshman year, I served on the medical team on a mission trip to Nicaragua with my school, Ontario Christian High School. Through that trip, I learned why the Lord calls us to be servant-hearted and realized my love for medicine. The realm of medicine has always been fascinating to me, and as a rising senior now, my love and fascination for medicine has only grown. I hope to continue to make modern medicine more accessible to everyone around the world, and contribute to the advancement of treatment, too.



Being part of the ABC program, this summer has allowed me to continue learning more about molecular biology, and the significant contributions research has to the future of medicine and health. I am helping out in Dr. Zhong's lab this summer, and I contributed to investigating the role gene fusions have in acute myeloid leukemia and brain tumors, specifically their role in treatment resistance. The specific mechanisms I learned and used include PCR (polymerase chain reaction), gel electrophoresis, and analyzing gene sequences- looking for fusions, gaps, breakpoints, and other features. I am so grateful to Dr. Zhong, those I work with in the lab (lab technician Fu Yingjie and graduate student Michel Irumva), and the staff of the ABC program for such an enlightening experience that has solidified my desire to pursue a career in healthcare and inspired me to dream big and be hopeful for the future of medicine.

IDENTIFICATION OF A NOVEL ISG20L- P2RY8 FUSION GENE IN ACUTE MYELOID LEUKEMIA

Crystal Wei, Yingjie Fu, Henry Trinh, Jiang F. Zhong

Center for Health Disparities and Molecular Medicine, Microbiology, School of Medicine, Loma Linda University, Loma Linda CA

Fusion genes are present in nearly one-third of acute myeloid leukemia (AML) cases and often play critical roles in prognosis, disease classification, and therapeutic development. In this study, we identified the presence of a novel gene fusion between ISG20L2 and P2RY8 through RNA sequencing of AML specimens. This gene fusion was further analyzed with reverse-transcriptase PCR and validated with Sanger sequencing. The PCR amplified product precisely matched the expected fusion junction, and fusion boundaries of the genes and confirmed via NCBI BLAST analysis. Alignment of ISG20L2 was confirmed to a predicted transcript region on chromosome 1p33. P2RY8 is involved in multiple fusion genes in AML. ISG20L2 plays a significant role in multiple myeloma (MM) by mediating resistance to proteasome inhibitors (PIs) such as bortezomib, specifically by suppressing MM cells' sensitivity to PIs. Patients with high ISG20L2 expression demonstrate treatment resistance and poorer clinical outcomes, whereas patients with low ISG20L2 expression are associated with enhanced therapeutic response and better overall survival. The fusion gene combination we found, ISG20L2-P2RY8, may have a similar function in AML treatment with PIs. P2RY8 is a known fusion partner in the P2RY8-CRLF2 gene fusion found in acute lymphoblastic leukemia (ALL). The P2RY8-CRLF2 fusion is associated with poor prognosis and higher relapse rates. Our results confirmed the presence of the ISG20L2-P2RY8

gene fusion in AML samples. This novel gene fusion may contribute to chemotherapy resistance and leukemogenesis in AML. We will be continuing our study to determine ISG20L2-P2RY8's functional significance, prevalence, and potential as a molecular therapeutic target.

Undergraduate Training Program (UTP)

MICAH ANDREWS
UTP PARTICIPANT 2025

The opportunity to participate in the UTP is something that I am very thankful for. I was given the amazing opportunity to experience multiple areas of research. This is very important for my undergraduate journey since it helps not only in gaining knowledge and research experience, but also exposure into what a future career path in research and medicine could look like. I had the opportunity to learn how valuable the impact of clinical research is in the development and growth of the medical field.

I am entering my third year at Oakwood University, located in Huntsville AL, where I major in Biology (Pre-Med) and serve as the TA for both the Microbiology and Genetics lab. After graduation from Oakwood University, I plan to attend medical school at Loma Linda University. My future goal is to become a physician who serves and makes a difference in the health and health outcomes of underserved communities. The UTP has helped strengthen my research abilities and mentorship experience, which will help me become better equipped to serve others.

During my experience in the UTP, I was given the opportunity to research with Dr. Sean Wilson. What stood out to me the most about this experience was observing and understanding the thought process that a Ph.D/graduate student follows when approaching a problem. I also am grateful for the connections I created during my time here. I want to thank Dr. Sean Wilson and Justis Cosper for their help in this valuable experience.



EFFECTS OF GESTATIONAL LONG-TERM HYPOXIA ON LOCAL Ca^{2+} SIGNALS IN BASILAR ARTERIAL MYOCYTES OF NEWBORN SHEEP

Micah Andrews, Justis Cosper, Eris Albert-Minkler, Jose L Puglisi,
Lubo Zhang, Arlin B Blood, Ellie Pettijohn, Sean M Wilson

Center for Health Disparities and Molecular Medicine,
Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA

Cerebral vasoreactivity is crucial for regulation of cerebral blood flow. This includes the dilation and constriction of arteries, which is dependent on changes in the membrane potential. Membrane depolarization is important for contraction while hyperpolarization causes dilation. Calcium (Ca^{2+}) sparks play an important role in these mechanisms, as they are activated due to membrane depolarization and cause membrane hyperpolarization. In arterial myocytes, a Ca^{2+} spark represents a localized release of Ca^{2+} ions across the sarcoplasmic membrane through activation of ryanodine receptors (RyRs). Previous evidence has shown that gestational long-term hypoxia (LTH) reduces Ca^{2+} spark activity in arterial myocytes of various vascular beds from newborn sheep. This study aimed to further investigate the effects of gestational long-term hypoxia on Ca^{2+} spark activity in basilar arterial myocytes in newborn sheep. We hypothesize that gestational long-term hypoxia will reduce the activity of Ca^{2+} sparks in basilar arterial myocytes due to membrane depolarization in cerebral arteries. To test this, normoxic pregnant sheep were kept at low altitude while hypoxic pregnant sheep were kept at high altitude for >110 days, then were moved to hypoxic chambers at Loma Linda after birth

(mimicking altitude of 3801m.). Basilar arteries were then extracted and loaded with Flou-4, a Ca^{2+} ion indicator. Arteries were placed in a physiological buffer (control), and then treated with 30mM K^+ (30K) which causes membrane depolarization in the absence and then presence of 1 μM nifedipine (NIF) which is an L-type calcium channel (LTCC) blocker. Membrane depolarization was expected to increase Ca^{2+} spark activity while NIF was expected to decrease Ca^{2+} spark activity. Ca^{2+} spark activity was recorded using confocal fluorescence imaging techniques and analyzed with a customized program (SparkLAB). Gestational long-term hypoxia decreased Ca^{2+} spark activity in basilar arterial myocytes and impaired depolarization mediated increases in spark activity while nifedipine failed to reduce spark activity among either group. Future research aims to determine the cellular mechanisms responsible for the impairment in depolarization mediated spark activity and lack of an influence of nifedipine on spark activity.

AZARIA THÉA CAREY
UTP PARTICIPANT 2025

Growing up in a Caribbean household, I learned the importance of perseverance. As I grow and mature in a world filled with hardship and unrest, my determination, curiosity, and creativity guide me forward in life and science. The scientific field reflects these qualities in me and provides a space where I can thrive. Research is not just a career path — it is a part of who I am, opening avenues toward my future.

Motivated by these ideals, I am pursuing a career focused on one of the body's most vital and expressive organs: the skin. I am a rising junior at Oakwood University, one of the best HBCUs, where I am pursuing a degree in biology. My academic goal is to become a dermatologist. My professional goal is to advance our understanding and contribute to the development of long-term treatments for incurable skin conditions, such as Hidradenitis Suppurativa, with a focus on individuals of color and those from diverse ethnic backgrounds.

With a new and deepening passion for neuroscience, I plan to apply the knowledge and skills I have gained during the Undergraduate Training Program to complete my degree and forge my path forward. I want to thank my mentor, Dr. Johnny Figueroa, and my incredible peer mentors — Julio Sierra, Arianna Williams, Venjaminne Fua, and Darine Abu Hilal — for recognizing my potential, affirming that I belong in this remarkable field, and encouraging me to grow as a researcher. As a member of a family of innovative, passionate professionals, I am proud to carry on their legacy of compassion, excellence, and faith through science, research, and service. Just as I was to persevere through adversity, I now carry that same spirit into the laboratory, where each challenge becomes an opportunity to grow and contribute to something greater.



**INVESTIGATING HORMONAL AND NEURAL MECHANISMS IN MALADAPTIVE
EATING BEHAVIORS**

Azaria Carey¹, Darine Abu Hilal¹, Venjaminne Fua^{1,2}, Julio Sierra¹, and Johnny D. Figueroa¹

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²Department of Biological Sciences, College of Science, California State Polytechnic University, Pomona, CA, United States

Binge eating disorder (BED) is a serious and common psychiatric condition affecting over 2.8 million people in the United States. It is characterized by episodes of overeating and a loss of control, often occurring alongside environmental stressors such as traumatic stress and exposure to a high-fat Western diet (WD). Evidence increasingly suggests that sex hormones significantly influence the risk of developing maladaptive eating behaviors, especially during adolescence, a critical period for brain development and hormonal shifts. Notably, females are more frequently affected, highlighting the importance of sex-specific research. However, the interaction between adversity, diet, and fluctuations in sex hormones in shaping neural circuits involved in reward, stress, and appetite remains poorly understood. The hippocampus and ventral tegmental area (VTA) are key regions involved in these processes. This study aimed to clarify how early-life exposure to a WD and traumatic stress affects hormone levels and neural activity in a sex-

dependent manner. We used a novel adolescent rat model of trauma-induced binge eating and measured circulating steroid and sex hormones, including progesterone, estradiol, and testosterone, using ELISAs. Immunofluorescence was employed to evaluate excitatory and inhibitory neuronal markers. Trauma and WD exposure significantly altered progesterone and estradiol levels in males and increased testosterone in both sexes. These changes indicate that adolescent stress disrupts neuroendocrine pathways that regulate food intake and reproductive health. Our findings reveal a complex relationship between diet, stress, and sex hormones that may impact the gut-brain connection and neuronal control of eating behavior. Understanding these processes is crucial for creating targeted interventions that address sex-specific vulnerabilities in BED and related conditions.

ANGELA CEJA
UTP PARTICIPANT 2025

My curiosity has sparked my interest in science and medicine from an early age. It is a privilege to study the complexity of God's creation and use that knowledge to serve my community. Whether that means serving locally in the Inland Empire or serving on mission trips to Baja California, I believe my purpose in life is to use my God given talents to address the health disparities that our communities endure.

This fall, I will begin my senior year in the Clinical Laboratory Science program at Loma Linda University. Here at LLU, I am involved in leadership as an officer for the CLS program and for the School of Allied Health Professions. My experience in research and clinical care inspire me to pursue a career as a Medical Scientist to investigate diseases and find solutions to the unanswered questions that remain in the field of medicine.

This summer, I have the honor and privilege to be working with Dr. Subburaman Mohan on a project that elucidates the role and mechanism of actions of Tumor Necrosis Factor (TNF) family of proteins (RANKL and Osteoprotegerin) in regulating marrow adiposity during aging and obesity. I would like to thank Dr. Mohan, Oasis Perez, and Sheila Pourteymoor for their support and guidance throughout this project.



TISSUE CONTEXT-DEPENDENT EFFECTS OF RANKL ON DIFFERENTIATION OF MESENCHYMAL STEM CELLS INTO OSTEOBLASTS AND ADIPOCYTES

Angela Ceja, Sheila Pourteymoor, Oasis Perez and Subburaman Mohan

Musculoskeletal Disease Center, VA Loma Linda Healthcare System and Center for Health Disparities and Molecular Medicine, Loma Linda University, Loma Linda, CA.

Obesity affects 4 in 10 Americans and is a risk factor for type 2 diabetes, hypertension, and heart disease. Increased bone marrow adiposity is linked to deteriorations in bone quality and strength, and, thereby, increased risk of fragility fractures. Mesenchymal stem cells (MSCs) derived from bone marrow or white adipose tissue serve as common precursors for osteoblasts, which form bone, and adipocytes, which form fat. Receptor activator of nuclear factor kappa-B ligand (RANKL) is expressed in osteoblasts and adipocytes and regulates bone health by promoting osteoclast functions. Based on the finding that the expression of RANKL is increased in the bones of high fat diet fed obese mice, we evaluated the role of RANKL and its inhibitor, osteoprotegerin (OPG) on differentiation of MSCs-derived from white adipose tissue (WAT) and bone marrow (BM) into osteoblasts and adipocytes.

LIA HUTCHINS
UTP PARTICIPANT 2025

From an early age, my family instilled in me the importance of caring for others and meeting their needs. At Loma Linda Academy, I took on roles in student leadership and participated in athletics; these experiences helped me develop a strong sense of responsibility for those often overlooked in my community. My curiosity about how the human body works led me to discover a passion for anatomy, which ultimately inspired me to pursue a degree in Biomedical Sciences at La Sierra University, where I am currently in my second year.



As I explored science, I looked for ways to connect it with my passions for music and art. I discovered that all three disciplines require resilience—each demands persistence, creativity, and the ability to learn from failure. I began my research journey through the SEA-PHAGES program, where I studied bacteriophages and experienced the excitement of scientific discovery. When I found the opportunity to conduct translational research through the UTP program at Loma Linda University, which focuses on health disparities, I eagerly applied. This focus deeply resonated with me, as I have witnessed the impact of health disparities on my community.

Participating in UTP strengthened my goal of becoming a family medicine physician, which ideally combines medical care with advocacy for underserved populations. Research is the foundation of compassionate, evidence-based medicine, and UTP has equipped me with the tools to pursue both clinical and research excellence.

I sincerely thank Dr. Duran for his patience, mentorship, and encouragement. This summer has significantly shaped my academic journey and clarified the path I aspire to follow.

**INVERSE CORRELATION BETWEEN B2-ADRENERGIC RECEPTORS AND
PARKIN EXPRESSION IN BREAST CANCER**

Caroline R. Coronado^{1*}, Lia V. Hutchins^{1*}, Daisy De Leon¹, Alfonso M. Durán^{1,2}

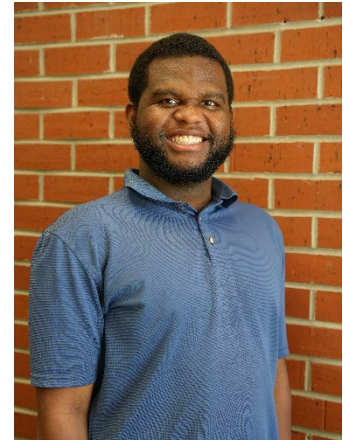
¹Center for Health Disparities and Molecular Medicine, School of Medicine, Loma Linda University, Loma Linda, CA; ²Department of Pathology and Anatomy, School of Medicine, Loma Linda University, Loma Linda, CA; *These two authors contributed equally to this project

Breast cancer (BCa) remains the most diagnosed malignancy among women in the United States, with one in eight expected to develop the disease in their lifetime. While genetic risk factors, including BRCA1/2 mutations, have been extensively characterized, the role of non-genomic factors in BCa pathogenesis is less understood. Emerging evidence suggests that overactivation of the sympathetic nervous system (SNS) contributes to BCa initiation, progression, and tumor aggressiveness; however, the precise mechanisms remain elusive. In other conditions, such as type II diabetes, excessive SNS activity has been linked to mitochondrial dysfunction, raising the possibility of a similar mechanism in BCa. Parkin, an E3 ubiquitin ligase, plays a central role in mitochondrial quality control and homeostasis. In this study, we investigated the relationship between β 2-adrenergic receptor (β 2-AR) abundance, a biomarker of SNS overactivation, and Parkin expression in BCa tissues and cell models. Using western blotting, immunohistochemistry (IHC), and cell culture experiments, we identified an

inverse relationship between β 2-AR levels and Parkin expression. These findings suggest that increased SNS signaling may downregulate Parkin, thereby compromising mitochondrial health and potentially promoting BCa tumor progression. If confirmed in larger clinical studies, these results highlight a novel mechanistic link between SNS overactivation and mitochondrial dysfunction in BCa. Targeting SNS signaling or restoring Parkin function could represent promising strategies for preventing BCa, improving recovery, and enhancing treatment interventions.

ROBERT LISTER
UTP PARTICIPANT 2025

During the spring of 2025, I was accepted into the Loma Linda University School of Medicine Early Assurance Program (LLU EAP) based on my academic achievements and strong GPA. One of the requirements was to attend the UTP program at Loma Linda and gain firsthand research experience on campus. This was a huge growth opportunity for me because, although I had conducted research at school and during an internship, the complex research conducted at Dr. Erik Behringer's lab was challenging and provided a valuable learning experience. The study examines the impact of calcium and potassium channels within blood vessels and the brain to investigate how Alzheimer's disease affects these channels. Future goals would enable this lab to manipulate these channels, preventing the degradation of the hippocampus and cortex, reducing the effects of Alzheimer's, and potentially leading to the discovery of a cure.



I desire to become a MD/MPH who educates the community around me. People should experience God's love through my every word and action as I provide them with quality care. However, that is not enough for me. I want quality care from physicians to be a choice, not a requirement. There are multiple lifestyle options to improve overall health and diminish severe disease and issues within numerous communities. I want my patients to leave me with the knowledge to help themselves and others within their community, thereby improving health literacy for those around them. With these aspirations, I will work diligently to impact others in every way possible.

EXPRESSION OF K⁺ CHANNELS IN THE HIPPOCAMPUS DURING AGING & ALZHEIMER'S DISEASE

Robert Lister³, Kevin Nguyen², Fritz E. L. Miot¹, Zion Shih¹, Phoebe Chum¹, Erik Behringer¹

Department of Basic Science, School of Medicine, Loma Linda University¹; San Geronio High School²; Oakwood University³

Alzheimer's disease (AD) is a neurodegenerative illness that impairs memory and motor skills, primarily due to damage to the hippocampus and cerebral cortex. With a global impact on 1 in 10 men and 1 in 5 women, AD is a significant health concern. Emerging evidence demonstrates AD as a "channelopathy", whereby normal regulation of K⁺ channels may be impacted to influence plasma membrane excitability and energy transport. In particular, the inward rectifying potassium (KIR) channel and small conductance calcium-activated potassium (SKCa) channels may coordinate blood flow with metabolic rate in the tissue parenchyma to ensure optimal perfusion of the brain. The expression, localization, and function of SKCa and KIR ion channels in the hippocampus during AD pathology is unclear. Thus, we worked on the hypothesis that hippocampal K⁺ channels may show differential expression among experimental AD mice relative to wild-type controls. Using the 3xTg-AD animal model (8 months, male & female), brains were perfused, dissected, and flash-frozen to create 20 µm coronal sections of the hippocampus. Immunohistochemistry (IHC) procedure was used, and sections were stained with hematoxylin to illustrate individual cell nuclei within the brain

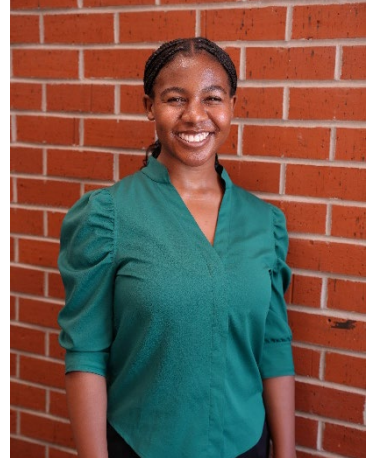
tissue. Images are captured with a light microscope and quantitatively assessed. If expression and localization patterns are significantly different among AD and wild-type mice, the results may help explain the deterioration of long-term potentiation and memory in the hippocampus. If similar, then we would have to consider other possibilities such as select post-translational modifications of channel activity (e.g., phosphorylation, oxidation) rather than expression *per se* or perhaps other signaling mechanisms and/or protein markers altogether. Regardless, we expect that our results will enhance insight into novel therapeutic strategies to combat illnesses such as stroke and AD.

ADDIE MCIVER
UTP PARTICIPANT 2025

During my first two years at Oakwood University in Huntsville, Alabama, I had the privilege of doing research under Dr. Elaine Vanterpool through the iSTEM- CARE program. This experience reinforced my idea of helping patients and those more susceptible to disease through innovative methods. Currently as a Biochemistry major, I seek to continue finding creative ways to lead and equip others with the skills to overcome challenges, as I have begun doing as a tutor and as concert master of our orchestra.

With the goal of improving health outcomes, this summer experience at Loma Linda University involves more of a translational approach. My mentor, Dr. Marino De Leon, my supervisor, Dr. Viet Hoang Dinh, Francis Zamora, and Dr. Jo-Wan Liu have been instrumental in emphasizing the value of research and its application in helping individuals burdened by health disparities. Studying socioeconomic factors, contributors, and potential treatments of neuropathic pain has opened my eyes to how research and preventative medicine might alleviate some of the suffering of my community.

In 2027, I plan to attend Loma Linda University Medical School and pursue a career as a physician, bringing novel treatments to vulnerable patients and improving health outcomes. Thank you, Dr. De Leon, for challenging my thinking, making research exciting, and teaching me the value of mentorship.



**N-3 POLYUNSATURATED FATTY ACIDS MODULATE NOCICEPTIVE SIGNALING
PATHWAYS AND IMPROVE PAIN OUTCOMES IN A PERIPHERAL NERVE INJURY
MODEL**

Addie McIver, Viet Hoang Dinh, Magda Descorbeth, Johnny D Figueroa, and Marino De Leon

Center for Health Disparities and Molecular Medicine, Basic Sciences, School of Medicine, Loma
Linda University, Loma Linda, CA

Neuropathic pain (NP) affects approximately 7-10% of the population worldwide and is driven by complex mechanisms including neuroinflammation, ion channel dysfunction, and oxidative stress. Despite available treatments, many patients experience inadequate pain relief, necessitating alternative therapeutic strategies. N-3 polyunsaturated fatty acids (n-3 PUFAs) have emerged as potential modulators of NP through their effects on pathological pathways. This study aimed to evaluate the therapeutic effects and molecular mechanisms of dietary n-3 PUFAs in NP through a rat model. Forty Sprague-Dawley rats received either a control diet or an n-3 PUFAs -enriched diet for four weeks prior to Chronic constriction injury (CCI) of the sciatic nerve. Pain behaviors were assessed weekly up to 4 weeks after CCI inducement using the Hargreaves and Catwalk tests. Additionally, dorsal root ganglia tissues were collected at day 7 post-injury for transcriptomic analysis using RNA sequencing, followed by RT-PCR for gene expression validation. Results showed that preventative administration of OMG3 significantly reduced thermal hyperalgesia and mechanical allodynia, along with improving locomotor function. Transcriptomics analysis revealed enrichment of key pathways including

activation of selenoamino acid metabolism and response of EIF2AK4 to the amino acid deficiency pathway. These mechanisms were coupled with inhibition of the mitochondrial dysfunction pathway, leading to promoted redox homeostasis, attenuated inflammatory response, and limited neuronal hyperexcitability, thereby alleviating pain signaling and improving motor function. Our study identified the expression of several NP linked transcripts that were significantly downregulated following treatment. In conclusion, preventative n-3 PUFAs supplementation alleviates NP and promotes functional recovery in the CCI model, primarily through modulation of selenoamino acid metabolism and suppression of pain-related signaling pathways.

KYAH MILLER
UTP PARTICIPANT 2025

I believe that knowledge directly equates to power. Although this statement can have a negative connotation, I believe that knowledge or education in this sense arms one with the power to serve on a greater scale.

I am a rising junior at Oakwood University in Huntsville, AL, majoring in Biology with a concentration in Pre-Medicine. Additionally, I am pursuing a minor in music with piano being my instrument of study.

The primary reason I have chosen to study biology is due to my heart for others. My heart for service has led to my various involvement in service positions including, but not limited to, serving as my Freshman Class President for the 2023-2024 school year, co-director for a freshman worship program on campus, and an assistant highschool volleyball for Oakwood Adventist Academy. In my free time I enjoy singing in my choir, working out, and journaling. I am currently interested in pediatric orthopedic surgery. This stems from my personal connection to both children and rehabilitation medicine.

As a participant of the Undergraduate Training Program, I have been given the wonderful opportunity to investigate differential rates of BRAF mutations in African Americans vs European Americans and its expression in ovarian cancer. I would like to acknowledge and thank Dr. Salma Khan, Romi Yamauchi, Alena McQuarter, Samuel Chan, and all the individuals in Dr. Khan's lab for their kindness and knowledge that has impacted me far beyond this summer.



**DISTINCT PROGNOSTIC GENE EXPRESSIONS IN OVARIAN CANCER AMONG
AFRICAN WOMEN**

Kyah Miller, Alena McQuarter, Jane Munide, Cody A. Carter, Saied Mirshahidi, Celena R. Yamauchi, Salma Khan

Center for Health Disparities & Molecular Medicine, School of Medicine, Loma Linda
University, Loma Linda, CA

Ovarian cancer is a leading cause of cancer-related death among women, with African American women experiencing disproportionately higher mortality and lower survival despite lower incidence compared to European American women. Contributing factors include tumor heterogeneity, chemoresistance, and an immunosuppressive tumor microenvironment. In our prior cBioPortal analysis, we identified BRAF, ITGB1, and TIMP3 as key prognostic genes. The BRAF gene, a proto-oncogene, encodes a cytoplasmic serine/threonine kinase involved in the MAPK signaling pathway. Mutations in BRAF are observed in approximately 5–9% of high-grade serous ovarian cancers. ITGB1 (Integrin beta-1), frequently overexpressed in ovarian cancer, encodes a cell surface receptor that promotes tumor invasion via the FAK/AKT signaling pathway. TIMP3 (Tissue Inhibitor of Metalloproteinases 3), commonly downregulated in ovarian cancer, functions as a tumor suppressor by inhibiting matrix metalloproteinases, suppressing metastasis, and inducing apoptosis. In this study, we assessed their expression in

fresh and archival ovarian cancer tissues from African American and European American patients using The Cancer Genome Atlas (TCGA) dataset, and findings were validated via quantitative polymerase chain reaction (qPCR) using gene-specific primers using LLU-cohorts. BRAF, a proto-oncogene involved in MAPK signaling, is mutated in ~5–9% of high-grade serous ovarian cancers. ITGB1, a cell surface receptor promoting invasion via the FAK/AKT pathway, is often overexpressed. TIMP3, a tumor suppressor, inhibits metastasis and promotes apoptosis but is typically downregulated. We found all three genes-BRAF, ITGB1, and TIMP3-significantly upregulated in samples from African American patients. These findings suggest potential prognostic biomarkers and therapeutic targets to address racial disparities in ovarian cancer outcomes. Ongoing studies aim to further investigate their functional roles and therapeutic implications.

JANANI NAGASUBRAMANYA
UTP PARTICIPANT 2025

Through my inner calling of treating and curing cancer, I have progressed by volunteering, learning, and now researching.

I began fundraising for cancer research at American Cancer Society's Relay For Life in 10th grade by initiating Santa Clara Relay's first ever talent-show fundraiser and performing Bharatanatyam Indian classical dance.

This summer, I am grateful that, by God's grace, I got the opportunity to conduct prostate cancer research under the mentorship of Dr. Carlos Casiano and his graduate students, Pedro Ochoa and Adelaide Makamure, in the Center for Health Disparities and Molecular Medicine. With classroom knowledge in genetics, I see this research in new light, with an interesting aspect being how cancer cells undergo mutations and gene pathway activation to develop resistance to therapy. For example, inhibiting the androgen receptor upregulates the glucocorticoid receptor in prostate cancer cells, which in turn increases LEDGF/p75 expression. This enhances the survival of cancer cells in the presence of stressors like radiation and chemotherapy.

This fall, I will return to UC San Diego as a sophomore majoring in Molecular and Cell Biology and conducting cancer research. In the future, I envision pursuing an MD/PhD degree.

Participating in UTP, I understood the need to ensure equal access to healthcare for everyone. Volunteering at Loma Linda's Fusion Homeless Ministry, I observed the positive impact of street medicine being provided as a service beyond business. I pledge to continue serving society by helping overcome health disparities and healing in God's abundant love.



**NOVEL ROLES OF LEDGF/P75 IN MODULATING PROSTATE CANCER RELATED
INFLAMMATORY PATHWAYS AND CANCER PATIENT RESPONSE TO
IMMUNOTHERAPY**

Janani Nagasubramanya, Sharan Bir, Pedro T. Ochoa, Adelaide Makamure, Evelyn S. Sanchez-Hernandez, Kai Wen Cheng, Zhong Chen, Issac Kremsky, Charles Wang, Carlos A. Casiano

Center for Health Disparities and Molecular Medicine, Department of Basic Sciences, Loma Linda University School of Medicine, Loma Linda, California, USA

Prostate cancer (PCa) is the second-leading cause of cancer death among men in the United States, with African American (AA) men experiencing a higher incidence and mortality rate compared to European American men. Moreover, AA men are more frequently diagnosed with aggressive forms of PCa, which reduces treatment options, particularly as tumors develop resistance to therapies. Unraveling the mechanisms behind PCa therapy resistance is vital for the creation of new therapeutic strategies. The lens epithelium derived growth factor p75 (LEDGF/p75) is a stress oncoprotein that contributes to cancer chemoresistance and tumor aggressiveness through its ability to tether oncogenic transcription factors to active chromatin, promote RNA-loop resolution at transcriptionally active sites, enhance DNA repair, and maintain genomic integrity. Our recent studies also demonstrated that LEDGF/p75 is upregulated in docetaxel (DTX)-resistant PCa cells and contributes to chemoresistance by

regulating gene pathways associated with stress survival, DNA repair, and cell cycle progression. We hypothesized that its silencing in chemoresistant PCa cells may also alter immune-related gene pathways. Knockdown of LEDGF/p75 in chemoresistant PCa cells followed by RNA-seq analysis led to the identification of 970 differentially expressed genes (DEGs). Gene set enrichment analysis (GSEA) revealed a role for LEDGF/p75 in modulating gene pathways associated with lymphocyte and inflammatory responses, since its downregulation led to upregulation of several inflammation-related genes including IL7R, IL18, SWAP70, BMI-1, ULBP2, etc. Protein expression of these genes was validated by Western blotting in PCa cells. Some protein expression levels did not align with the RNA-seq data, highlighting the need for validation through RT-PCR and CRISPR knockout of LEDGF/p75. High expression of most of these inflammatory genes also correlated with better overall survival of cancer patients receiving PD-L1 immunotherapy, as revealed by KM Plotter-Immunotherapy analysis. We conclude that LEDGF/p75 contributes to the negative regulation of inflammatory gene pathways. Understanding LEDGF/p75's role in cancer immunity may provide new insights into its immunomodulatory functions and its influence on cancer patient response to immunotherapy.

LAILA PRENTICE
UTP PARTICIPANT 2025

From a young age, I became familiar with the challenges of managing skin conditions like eczema and keloids. While these experiences were difficult, these experiences are ultimately what led me to discover my passion and future career in dermatology. I want to help others embrace their beauty, heal their pain, and rediscover their self-worth. This is why I am committed to pursuing medicine not only with skill, but also with compassion.

I am currently a biology major on the pre-med track attending Oakwood University in Huntsville, Alabama, with an expected graduation date of May 2027. Though my passion is science, I love to sing with my choir, Voices of Triumph, stay active through exercise, help tutor 9th-grade biology, and spend time with my family and friends.

This summer, I had the privilege of conducting research under the mentorship of Dr. Julia Unternaehrer and the guidance of my supervisor, Brigitte Vasquez. Our work focuses on the role of PD-L1 in ovarian cancer and its impact on cisplatin sensitivity. Through this internship, I have developed critical thinking, research planning, and lab skills, while gaining confidence in my scientific abilities.

I am deeply grateful for the opportunity to be a part of the undergraduate training program and for the mentorship and encouragement I've received. I am glad to be taking part in such an amazing program that is helping me take the next steps into healthcare.



THE EFFECT OF PD-L1 KNOCKDOWN IN HIGH-GRADE SEROUS OVARIAN CANCER CELLS ON CISPLATIN RESISTANCE AND PROLIFERATION

Laila Prentice, Brigitte Vazquez, Jay Deng, Ashlyn Conant, Kiera McGivney, Julia Unternaehrer
Division of Biochemistry, Department of Basic Sciences. School of Medicine, Loma Linda
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Ovarian cancer refers to a spectrum of gynecologic malignancies arising from the ovaries or fallopian tubes. Among its various forms, high-grade serous ovarian carcinoma (HGSOC) is the most prevalent and aggressive subtype, accounting for approximately 75% of epithelial ovarian cancer cases. HGSOC is often diagnosed at an advanced stage and is initially responsive to chemotherapy, however, it frequently recurs with acquired resistance to treatment, posing a significant clinical challenge. Recent studies have shown resistant cells to express higher levels of Programmed Death-Ligand 1 (PD-L1). The objective of our research is to investigate the role of PD-L1 in contributing to cisplatin resistance and proliferation in HGSOC. We hypothesize that knocking down the PD-L1 gene in cisplatin-resistant PD-X4 ovarian cancer cells will increase their sensitivity to cisplatin and inhibit the proliferation of PD-L1-expressing ovarian cancer cells.

To test this hypothesis, we utilized bacterial transformation and transfection techniques to generate lentiviral plasmids encoding PD-L1 shRNA constructs (PD-L1 14 and 15) along with a non-targeting control (Non). Lentiviral transduction was used to introduce these constructs into PD-X4 CR and SE ovarian cancer cells. Cisplatin sensitivity was assessed using the MTT assay

to evaluate cell viability following drug treatment. In parallel, a proliferation assay was conducted to determine the effect of PD-L1 knockdown on cell growth independent of drug treatment.

While data analysis is ongoing, the experimental design aims to uncover a potential intrinsic link between PD-L1 expression and cisplatin resistance in ovarian cancer. These findings may contribute to the development of novel therapeutic strategies targeting PD-L1 to improve treatment outcomes in patients with recurrent, chemoresistant HGSOC.

ANNIKA SAMAYOA
UTP PARTICIPANT 2025

My interest in research started during my first year of college at La Sierra University. I decided to get involved in three different research areas (ecology, virology, and astrophysics) to learn about how research is done in different fields. Oftentimes, these research endeavors left me in awe of all that God has created, as said by the psalmist, "Great are the works of the Lord, studied by all who delight in them." This led me to be an author for an oral presentation for the nationwide SEA Symposium in 2024. I have also been able to be an author and present 3 posters at La Sierra University's Research Emphasis Week (REW) in 2024, one of which won first prize. This year, I have been able to present posters at the SEA Symposium, La Sierra University's REW, and the International Worm Meeting at UC Davis. As my interest in research grew, I felt it was important to get more involved in biomedical research since I want to apply to the MD/PhD program at Loma Linda University. I believe that getting accepted to the UTP program has helped me greatly in achieving this and understanding more about what biomedical research can be like. In Dr. Danilo Boskovic's lab, I worked primarily with PhD candidate Lidia Malina on determining if the presence of lipopolysaccharides (LPS) from bacteria such as *Porphyromonas gingivalis* in the blood affects platelet activation time. Thank you to Dr. Danilo Boskovic for allowing me to work in your lab this summer, and thank you to Lidia Malina for your unwavering kindness and patience in teaching me this summer.



**PLATELET FUNCTIONS ARE ALTERED IN THE PRESENCE OF LIPOPOLYSACCHARIDE
FROM *PORPHYROMONAS GINGIVALIS***

Annika Samayoa¹, Lidia M. M. Wells², Emely Murillo-Vega³, Anton Zivanovic², Danilo S. Boskovic^{2,3}

¹Center for Health Disparities and Molecular Medicine, ²Department of Earth and Biological Sciences, and ³Division of Biochemistry, Department of Basic Sciences, School of Medicine, Loma Linda University, Loma Linda, CA

Platelets are anucleate fragments of mature megakaryocytes that play key roles in hemostasis, inflammation, and infection. Platelets can be directly or indirectly affected by bacteria via their surface interactions or through their excreted substances. In this context, lipopolysaccharides (LPS), found on the outer membrane of gram-negative bacteria, can interact with platelet Toll-like Receptor 4 (TLR4). *Porphyromonas gingivalis*, a gram-negative oral pathogen, is known for its role in periodontal disease and as a contributor to increased risk of atherosclerosis. Human whole blood was exposed to varying levels of *P. gingivalis* LPS for specific pre-incubation durations. Platelet plug formation was measured utilizing the Platelet Function Analyzer (PFA-100), by Siemens. The *P. gingivalis* LPS alters platelet plug formation in a concentration and incubation time-dependent manner, with evidence of inhibition. To better understand the changes in platelet function in response to LPS exposure, a flow cytometric approach is being developed. The analysis of whole blood (with or without ADP) was conducted over the course of 3 hours after blood draw to ensure platelet function stability within the experimental context.

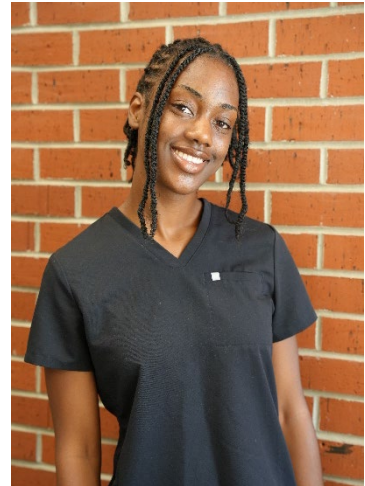
Antibodies targeting CD16 (neutrophils), CD61 (platelets), CD62p (α granules), and CD63 (dense granules), were used to assess platelet-neutrophil aggregate (PNA) formation and degranulation over time. The optimal time to analyze platelet functions using whole blood was found to be between 20 and 60 minutes after blood collection, before artifactual changes in PNA and granule secretion occur. This methodology will be implemented in future analyses of platelets in whole blood following exposure to *P. gingivalis* LPS.

KYLA TUCKER
UTP PARTICIPANT 2025

From an early age, I have always wanted to become a physician. As I grew older, my passion for aiding others continued and I concluded that my main reason for wanting to become a physician was due to that sense of fulfillment I felt after have helping others.

As a junior at Oakwood University, I am majoring in Biology with a concentration in PreMed/PreOsteopathic. This fall, I plan to continue participating in research as well as volunteering at Oakwood's Stem Success Academy and OU Reach while participating in Oakwood University's chapter of Minority Association of Pre-Medical Students, and more. Upon graduating from Oakwood University, I aspire to attend Loma Linda School of Medicine to obtain a medical degree. After graduation, I plan to specialize in orthopedic surgery focusing on sport medicine related injuries. My passion for orthopedics developed due to a few family members experiencing orthopedic related injuries and myself having ACL reconstruction surgery. My drive and passion to become a physician significantly grew after personally experiencing injury to surgery to recovery. I plan to continue to walk in God's plan for my life and soon become one of the very few black, female orthopedic surgeons treating each of my patients with holistic care.

This summer, I am working in Neurology Oncology department under the guidance of Dr. Rameshwar Patil. This experience has been nothing short of amazing, and I continue to learn daily.



**SYNTHESIS AND CHARACTERIZATION OF EGFR-TARGETED GOLD
NANOPARTICLES FOR RADIOSENSITIZATION OF HEAD AND NECK CANCERS**

Kyla Tucker, Cedric Lansangan, Serge Rudensky, Sanjay Yadav, Kevin Nick, Rameshwar Patil

Department of Basic Sciences and Neurosurgery, Division of Cancer Sciences, School of
Medicine, Loma Linda University, Loma Linda, CA

Head and neck squamous cell carcinoma (HNSCC) is a common form of cancer found in various regions of the head and neck. Annually, over half a million individuals are diagnosed with HNSCC, accounting for 300,000 deaths worldwide. Radiotherapy is part of the standard of care for HNSCC. However, radioresistance is prevalent in recurrent HNSCC. Epidermal Growth Factor Receptor (EGFR) is highly overexpressed in HNSCC and plays a crucial role in its growth, development, and acquisition of radioresistance. EGFRvIII is a mutant of the EGFR prevalent in HNSCC and plays a crucial role in radioresistance. Nanoparticles with higher atomic numbers, such as gold nanoparticles (GNP), increase the radiation deposit in tumors and thus potentially act as radiosensitizers in the treatment of HNSCC.

In this study, we synthesized and characterized EGFR-targeted GNPs designed to specifically target HNSCC for radiosensitization. The GNP core (~12 nm) was synthesized by standard citrate reduction of Au(3+) precursor with modification. An EGFRvIII-specific targeting peptide was attached to the GNP surface via a stable covalent bond for effective tumor targeting

and accumulation in HNSCC. GNP surface was coated by polyethylene glycol to increase colloidal stability. GNPs were characterized by various analytical methods such as UV-vis spectrometry, Dynamic Light Scattering, and wet chemical assays well established in Dr. Patil's lab. In vitro evaluation is ongoing to quantify the cellular uptake of targeted vs nontargeted GNPs in EGFR expressing cell lines. Cellular uptake of GNPs will be studied by quantifying the internalized gold using Microwave Plasma Atomic Emission Spectroscopy (MP-AES) in Dr. Kevin Nick's Lab. This study will pave the way towards clinical radiosensitization of HNSCC tumors treated with EGFR-targeted GNPs.

Initiative to Maximize Student Development (IMSD)

DANIELLE MALIVERT
IMSD PARTICIPANT 2025

Born to Haitian parents Jean Daniel and Esther Malivert, I am the eldest of three children. I, along with my parents, my younger sister, Daina, and my younger brother, Emmanuel, live in Massachusetts. I matriculated through South Lancaster Academy and Oakwood University where I received a Bachelor of Science in Biomedical Sciences.

After I graduated from Oakwood in 2022, I have had the privilege of not only being in the Ph.D. program at Loma Linda University but also being in the IMSD program. I have learned a lot these past two years both in and out of school and lab.



I would like to thank the directors of both programs including Dr. Fletcher, Dr. De Leon, and Dr. Casiano along with Keenan. In addition, I would like to thank my PI, Dr. Pearce, along with my lab members Desirelys Ortiz Martinez, James Williams, and Jose Garcia who has been with us in Dr. Pearce's lab this summer. I would also like to thank Lorena, Amy, and Lynn along with my classmates and colleagues in the PhD and IMSD programs. It was a great summer, and I look forward to having more experiences as I continue my journey through this program.

**MITOCHONDRIAL CHARACTERISTICS AND CONTRACTILE FUNCTION ARE
SIGNIFICANTLY DEPENDENT ON OXYGEN CONCENTRATION AND ARTERY TYPE
IN NEONATAL CEREBROVASCULAR SMOOTH MUSCLE**

Yaniah X Gamboa, Danielle Lonie Malivert, Desirelys Ortiz Martinez, James Williams, William Pearce

Center for Health Disparities and Molecular Medicine, Perinatal Biology, School of Medicine,
Loma Linda University, Loma Linda, CA

Neonatal hypoxia induces vascular maladaptation that includes impaired functional maturation, leading to significant complications, particularly in the cerebral circulation. Despite the well-established causes of neonatal hypoxia, the mechanisms underlying cerebrovascular responses to neonatal hypoxia remain poorly understood. Accumulating evidence indicates that mitochondria are essential regulators of vascular development and function. However, their role in hypoxia-induced neonatal cerebrovascular adaptation remains unclear. Recognizing the essential role of mitochondria in vascular maturation, this study examines the effects of graded hypoxia on mitochondrial characteristics and contractility in ovine neonatal middle cerebral arteries (MCA) and posterior cerebral arteries (PCA). To determine the effects of graded hypoxia on mitochondria, mitochondrial DNA (mtDNA) copy number per cell and Succinate Dehydrogenase A (SDHa) per mtDNA copy number (a measure of mitochondrial size), were measured via PCR and immunoblotting, respectively. Additionally, vascular stress-strain relations were characterized using measurements of passive wall stress, myogenic wall stress, and depolarization-induced stress. Relative to 18%O₂, the greatest reduction in mtDNA copy number per cell was 43% at 5%O₂ in MCA and 44% at 2%O₂ in PCA. SDHa per mtDNA copy number increased from 7.6 to 43.5 in MCA at 18%O₂ and 5%O₂, respectively and 21.3 to 72.7 at 18%O₂ and 2%O₂, respectively in PCA. Correspondingly, reducing O₂ from 6% to 5% increased depolarization-induced active stress by 21% and 261% in MCA and PCA respectively. Overall, our results suggest that graded hypoxia increases contractility while simultaneously decreasing mtDNA copy number per cell and increasing SDHa per mtDNA copy number in the neonatal

cerebral circulation in an artery dependent manner. This study offers new insights into the mechanisms that mediate neonatal cerebrovascular adaptation to hypoxia.

PEDRO T. OCHOA
IMSD PARTICIPANT 2025

As a child I was always intrigued by how things operate which cultivated my passion to learn. I discovered my calling in life until a close family member of mine was diagnosed with cancer. I saw my family member go from a healthy individual to a completely different person. Thanks to the hard work of the medical staff and cancer researchers who strive to provide the best treatment for cancer patients, my family member was able to beat cancer. Although this experience was heart wrenching, it ultimately helped in defining my future.



I attended the University of California, Irvine (UCI) where I obtained a Bachelors degree in Biology and Sociology. During my time at UCI, I was fortunate enough to have an opportunity to perform undergraduate research. It was this experience that reminded me of how crucial research is for identifying new treatments. The combination of my previous experience, thirst for knowledge and, passion for cancer biology drove me to pursue my PhD. I am a fifth year PhD student in the Cancer Developmental, and Regenerative Biology Division in Dr. Carlos Casiano's laboratory. My project aims to further explore the role of the GR-LEDGF/p75-HSP27 axis in PCa therapy cross-resistance and identify inhibitors targeting this network as a potential treatment option.

**NOVEL ROLES OF LEDGF/P75 IN MODULATING PROSTATE CANCER RELATED
INFLAMMATORY PATHWAYS AND CANCER PATIENT RESPONSE TO
IMMUNOTHERAPY**

Janani Nagasubramanya, Sharan Bir, Pedro T. Ochoa, Adelaide Makamure, Evelyn S. Sanchez-Hernandez, Kai Wen Cheng, Zhong Chen, Issac Kremsky, Charles Wang, Carlos A. Casiano

Center for Health Disparities and Molecular Medicine, Department of Basic Sciences, Loma Linda University School of Medicine, Loma Linda, California, USA

Prostate cancer (PCa) is the second-leading cause of cancer death among men in the United States, with African American (AA) men experiencing a higher incidence and mortality rate compared to European American men. Moreover, AA men are more frequently diagnosed with aggressive forms of PCa, which reduces treatment options, particularly as tumors develop resistance to therapies. Unraveling the mechanisms behind PCa therapy resistance is vital for the creation of new therapeutic strategies. The lens epithelium derived growth factor p75 (LEDGF/p75) is a stress oncoprotein that contributes to cancer chemoresistance and tumor aggressiveness through its ability to tether oncogenic transcription factors to active chromatin, promote RNA-loop resolution at transcriptionally active sites, enhance DNA repair, and maintain genomic integrity. Our recent studies also demonstrated that LEDGF/p75 is upregulated in docetaxel (DTX)-resistant PCa cells and contributes to chemoresistance by regulating gene pathways associated with stress survival, DNA repair, and cell cycle progression. We hypothesized that its silencing in chemoresistant PCa cells may also alter immune-related gene pathways. Knockdown of LEDGF/p75 in chemoresistant PCa cells followed by RNA-seq analysis led to the identification of 970 differentially expressed genes (DEGs). Gene set enrichment analysis (GSEA) revealed a role for LEDGF/p75 in modulating gene pathways associated with lymphocyte and inflammatory responses, since its downregulation led to upregulation of several inflammation-related genes including *IL7R*, *IL18*, *SWAP70*, *BMI-1*, *ULBP2*, etc. Protein expression of these genes was

validated by Western blotting in PCa cells. Some protein expression levels did not align with the RNA-seq data, highlighting the need for validation through RT-PCR and CRISPR knockout of LEDGF/p75. High expression of most of these inflammatory genes also correlated with better overall survival of cancer patients receiving PD-L1 immunotherapy, as revealed by KM Plotter-Immunotherapy analysis. We conclude that LEDGF/p75 contributes to the negative regulation of inflammatory gene pathways. Understanding LEDGF/p75's role in cancer immunity may provide new insights into its immunomodulatory functions and its influence on cancer patient response to immunotherapy.

OASIS PEREZ
IMSD PARTICIPANT 2025

I have been extremely fortunate to have the support of Loma Linda University's Center for Health Disparities and Molecular Medicine for almost 6 years now. The early hands-on research experience I gained during the ABC program was truly transformative, shaping my academic journey. The opportunity for summer research further enhanced this during my undergraduate degree within UTP. At the same time, I received my Bachelor's degree in Biological Science from the University of California, Irvine. I am entering my third year of my PhD within the Cancer, Development, and Regenerative Biology track here at Loma Linda University. All of which would not have been possible without the careful time and dedication Loma Linda University faculty and staff have bestowed upon me. I am grateful to Dr. Kylie Watts for introducing me to the research world through the ABC program. I am forever grateful to Dr. Subburaman Mohan for welcoming me into his lab and guiding me throughout both my undergraduate and graduate studies. Within Dr. Mohan's lab, we focus on identifying the causes and proposing therapeutic approaches for different musculoskeletal diseases, including osteoporosis and osteoarthritis induced by external factors such as obesity or Type 2 diabetes.



THYROID HORMONE RECEPTOR BETA AGONIST, MGL3196, EXERTS OPPOSITE EFFECTS IN PREVENTING THE NEGATIVE EFFECTS OF HIGH FAT DIET ON BONE AND CARTILAGE IN AGED MALE MICE.

Oasis Perez, Gustavo Gomez, Sheila Pourteymoor, Bouchra Edderkaoui, Subburaman Mohan

Musculoskeletal Disease Center, VA Loma Linda Healthcare Systems and
Center for Health Disparities and Molecular Medicine, Loma Linda University, Loma Linda,
CA

Common risk factors that lead to increased prevalence of osteoporotic fractures and osteoarthritis are conditions such as obesity and Type 2 Diabetes (T2D). To investigate a potential intervention to ameliorate the bone and cartilage degradation in elderly obese populations, we evaluated the effects of thyroid hormone receptor beta agonist, MGL3196, in aged high-fat diet (HFD) fed C57BL/6J mice. 18-month-old C57BL/6J mice of both sexes were fed a control diet (CD) or HFD for 12 weeks and treated daily with MGL (10 mg/kg) or vehicle. The HFD increased fat mass in both sexes while treatment with MGL only alleviated adiposity in male mice. By contrast, MGL treatment exacerbated HFD-induced cortical bone loss measured by microCT in male mice, resulting in significantly weaker bone (maximum load at which bone breaks) by the 3-point bending test in MGL-treated vs vehicle-treated HFD male mice. Transcriptome analyses revealed that expression levels of Wnt10b and Wnt16 were decreased while Wnt inhibitor, Sost, expression was increased by HFD in the bones of male mice. Articular cartilage measurements by histology showed that HFD promoted loss of articular cartilage in both male and female mice and that MGL treatment ameliorated the HFD-induced articular cartilage loss in both sexes. Our findings suggest that tailored therapies are needed to alleviate adverse HFD effects on fat, bone and cartilage metabolism according to age and sex.

KAYLA SANCHEZ
IMSD PARTICIPANT 2025

I received my Bachelor of Science in Biochemistry and Molecular Biology from California Baptist University. After graduation, my curiosity for neuroscience evolved into a passion, leading me to pursue a PhD in Neuroscience, Systems Biology, and Bioengineering at Loma Linda University.

Alongside my studies, I have had the privilege of participating in the IMSD program. This experience has allowed me to interact with scientists from diverse fields, provided valuable professional development opportunities, and given me the chance to serve as a mentor myself.

Currently, I am completing my degree in Dr. Soriano's laboratory, where I investigate underlying pathogenic mechanisms in Niemann-Pick Disease Type C. My research focuses on whether oxytosis/ferroptosis serves as an early driver of cellular pathology before clinical symptoms appear. Additionally, I am examining the therapeutic potential of J147, a novel anti-ferroptotic drug developed by our collaborators at the Salk Institute.

I am on track to graduate this year and intend to continue my journey by pursuing a postdoctoral fellowship in the field of prion diseases. I am deeply grateful to my PI, Dr. Soriano, whose exceptional mentorship has not only guided my scientific development but also served as an inspiration for the kind of scientist and leader I aspire to become.



**CHARACTERIZING EARLY PATHWAYS IN HEREDITARY PRION DISEASES VIA
TRIOSIG: A NOVEL FERROPTOSIS DATABASE**

Mani Momeni ¹, Kayla Sanchez ¹, Jacob White ¹, Antonio Currais ², Pamela Maher ², Salvador Soriano ¹

¹Department of Pathology and Human Anatomy, Loma Linda University, School of Medicine,
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Prion diseases remain among the most understudied and challenging to investigate due to the unique nature of their infectious model. These diseases are characterized by progressive neurodegeneration and are always fatal. Although prion diseases can be acquired through various routes, the hereditary form enables us to understand the genetic mechanism of pathogenesis. Hereditary prion diseases are caused by mutations in the PRNP gene, resulting in the misfolding of the normal prion protein (PrP^C) into the disease-associated form, known as prion-scrapie PrP^{Sc}. PRNP mutations result in different prion disease variants, each with distinct cellular and regional variabilities. Two examples include Creutzfeldt-Jakob disease (CJD), which has over 40 associated variants, and Fatal Familial Insomnia (FFI), which has two variants. In both conditions, survival rates are approximately one year after the onset of symptoms. Although the function of PrP^C remains under investigation, studies have shown that mutations lead to metal ion imbalance and increased levels of lipid peroxidation, hallmarks of ferroptosis. However, the link between prion disorders and ferroptosis has not been thoroughly explored. One reason we believe this connection remains unclear is the lack of neuron-specific ferroptosis signatures. To address this gap, our lab utilized a novel ferroptotic database called TrioSig, developed by the Maher lab at the Salk Institute. As such, we hypothesize that PRNP mutations increase vulnerability to ferroptosis and these signatures may be uncovered using the TrioSig database. To test this, we performed a bioinformatic analysis of various neuronal cell

types in pre-symptomatic CJD and FFI mouse models. We found that ferroptosis was significantly upregulated within somatostatin-expressing (SST) cells, a subtype of inhibitory neuron found in the cortex, in FFI. These findings provide the first evidence that ferroptosis is differentially activated across prion diseases, suggesting a potential therapeutic target for slowing neurodegeneration in the future.

KRYSTAL SANTIAGO
IMSD PARTICIPANT 2025

I was born in Mayaguez, PR, where my parents taught me that even though success was hard, if I set my mind to it and worked for it, I could achieve it. With this lesson in mind, aiming to obtain academic excellence with the help of God is one of my priorities. Because of this, I have put a lot of effort into becoming the best student I can be. I graduated from the University of Puerto Rico with a BS in Industrial Microbiology and I am now part of the IMSD program as a fourth-year student, where I will further my education by earning a PhD. I learned to play the flute



and I also trained my voice which allowed me to be the recipient of different scholarships throughout my undergraduate studies. After Puerto Rico suffered from hurricane Maria, my friends and I helped rebuild houses and feed the homeless. In order to be a force for positive change, I selected Dr. Casiano and Dr. Almaguel, experts in health disparities, to be my co-advisors. This way I could focus my research on diseases that affect underrepresented communities. For my research, I am studying the role of Enolase, a cytoplasmic enzyme, and its effect on the proliferation, migration, invasion and metastasis of Prostate Cancer.

**INVESTIGATING THE ROLE OF ENO1 IN NEUROENDOCRINE PROSTATE CANCER
CELL GROWTH AND SURVIVAL AS A TARGET FOR ANTI-CANCER THERAPY**

Krystal R. Santiago, Alfonso Duran, Kristen Whitley, Lemti Nyirendah, Julia Soliz, Frankis Almaguel, Carlos Casiano

Center for Health Disparities and Molecular Medicine, School of Medicine, Cancer Center,
Loma Linda University, Loma Linda, CA

Prostate cancer (PCa) is the second most commonly diagnosed cancer and the leading cause of cancer-related death among men in the United States. Taxane-based chemotherapy with docetaxel (DTX) and cabazitaxel is used as last-line treatment for metastatic castration-resistant prostate cancer (mCRPC). However, these drugs often fail due to the development of chemoresistance. The prostate-specific membrane antigen (PSMA) has emerged as a critical target for theranostics (imaging + therapy) in mCRPC. Nonetheless, around 30% of patients show a limited response, particularly those with low or absent PSMA expression. This highlights the need for alternative theranostic targets in PSMA-negative or low-PSMA-expressing tumors. Given these limitations, exploring molecular targets such as Enolase (isoforms ENO1 and ENO2) could enhance treatment strategies for mCRPC. ENO1, a glycolytic enzyme, shows promise as it is found on the surface of various tumors. We hypothesized that ENO1 is abundant on the surface of neuroendocrine-like prostate cancer (NEPC-L) cell lines, and that targeting this protein could disrupt key metabolic pathways, leading to anti-cancer effects and tumor growth inhibition. Western blot analysis revealed that DTX-sensitive PCa cell lines expressing NEPC markers also express both ENO1 and ENO2. In contrast, DTX-resistant NEPC-L cells expressed ENO1 with reduced ENO2, suggesting a metabolic vulnerability. Additionally, ENO1 surface localization in DTX-resistant NEPC-L cells was influenced by glucose levels. High glucose, as found in metabolically active tumors, increased surface ENO1, while normal glucose reduced it, likely due to c-MYC inhibition and MBP1 upregulation. We also assessed whether ENO1

silencing impacts NEPC-L cell proliferation and clonogenicity. MTT and clonogenic assays showed that ENO1 knockdown significantly reduced proliferation, colony formation, and altered morphology, while maintaining viability. These findings suggest ENO1 inhibition could impair tumor expansion. Ongoing studies will assess whether ENO1 knockdown re-sensitizes cells to DTX and affects stemness, supporting its potential as a therapeutic target in drug-resistant NEPC.

JULIO SIERRA
IMSD PARTICIPANT 2025

My parents immigrated from Mexico looking for opportunities to better their lives, and consequently, my life. As they struggled to acculturate, they instilled in me the importance of education and hard work so that I could successfully overcome the barriers they faced. I enjoy learning, problem-solving, and challenging myself, which motivated me to pursue undergraduate studies in biomedical engineering. During that time, I gained experience developing systems for use in research.



I am a second-year Ph.D. student in the Neuroscience, Systems Biology, and Bioengineering program at Loma Linda University School of Medicine. As a first-generation college student, I understand the difficulties of navigating post-secondary education without a support system. Being involved with the IMSD program in the Center for Health Disparities and Molecular Medicine provides a sense of community and allows me to play a role in mentoring and encouraging students to pursue higher education.

I am grateful for the mentorship and support of my PI, Dr. Johnny Figueroa, my peer mentor, Timothy Simon, and many others for helping me develop as a neuroscientist. I expect to continue contributing to the field by investigating the impact of environmental factors, such as high-fat diet consumption, on neurobiological pathways that confer resiliency to stressors during adolescence. I am excited to continue our research, driving the field forward, and helping to address a significant health disparities issue.

**INVESTIGATING HORMONAL AND NEURAL MECHANISMS IN MALADAPTIVE
EATING BEHAVIORS**

Azaria Carey¹, Darine Abu Hilal¹, Venjaminne Fua^{1,2}, Julio Sierra¹, and Johnny D. Figueroa¹

¹Center for Health Disparities and Molecular Medicine, Department of Basic Sciences, School of Medicine, Loma Linda University, Loma Linda, CA, United States

²Department of Biological Sciences, College of Science, California State Polytechnic University, Pomona, CA, United States

Binge eating disorder (BED) is a serious and common psychiatric condition affecting over 2.8 million people in the United States. It is characterized by episodes of overeating and a loss of control, often occurring alongside environmental stressors such as traumatic stress and exposure to a high-fat Western diet (WD). Evidence increasingly suggests that sex hormones significantly influence the risk of developing maladaptive eating behaviors, especially during adolescence, a critical period for brain development and hormonal shifts. Notably, females are more frequently affected, highlighting the importance of sex-specific research. However, the interaction between adversity, diet, and fluctuations in sex hormones in shaping neural circuits involved in reward, stress, and appetite remains poorly understood. The hippocampus and ventral tegmental area (VTA) are key regions involved in these processes. This study aimed to clarify how early-life exposure to a WD and traumatic stress affects hormone levels and neural activity in a sex-dependent manner. We used a novel adolescent rat model of trauma-induced binge eating and measured circulating steroid and sex hormones, including progesterone, estradiol, and testosterone, using ELISAs. Immunofluorescence was employed to evaluate excitatory and inhibitory neuronal markers. Trauma and WD exposure significantly altered progesterone and estradiol levels in males and increased testosterone in both sexes. These changes indicate that adolescent stress disrupts neuroendocrine pathways that regulate food intake and reproductive

health. Our findings reveal a complex relationship between diet, stress, and sex hormones that may impact the gut-brain connection and neuronal control of eating behavior. Understanding these processes is crucial for creating targeted interventions that address sex-specific vulnerabilities in BED and related conditions.

FRANCIS ZAMORA
IMSD PARTICIPANT 2025

Before coming to Loma Linda University, I attained my Masters in Anatomy & Neurobiology from Boston University, where I discovered my curiosity for the neuroscience field. I yearned for the opportunity to continue developing a thorough understanding of the nervous system and to develop the skills needed to make my own inquiries, investigate biomedical questions, and contribute to closing the gaps in knowledge in the scientific literature. Thus, pursuing a career as a biomedical researcher and professor became a clear goal for me.



I am grateful to be part of the IMSD program, as it has provided me with the resources and tools to earn my PhD and fulfill my dream of becoming a neuroscientist. Additionally, as a first-generation Guatemalan American woman I am proud to represent underrepresented minorities in science and hope to inspire and encourage younger minority students through mentorship.

I am currently a 5th Year PhD candidate in Dr. Marino De Leon's laboratory at the Center for Health Disparities and Molecular Medicine. My research focuses on investigating the role of autophagy and endoplasmic reticulum dynamics during lipotoxicity and docosahexaenoic acid (DHA) protective mechanisms in Schwann cells. Through this research I ultimately hope to contribute to and expand our understanding of how to prevent/treat nerve injury and alleviate neuropathic pain. I am thankful to Dr. Marino De Leon and Dr. Jo-wen Liu for their mentorship and guidance as I continue my academic journey.

**INTERPLAY OF FATTY ACID BINDING PROTEIN 5 AND AUTOPHAGY
DURING PALMITIC ACID-INDUCED LIPOTOXICITY**

Francis Zamora, Bernice Juarez, Addie McIver, Viet Hoang Dinh, Jo-Wen Liu, Marino De Leon

Center for Health Disparities and Molecular Medicine, Basic Sciences,
Loma Linda University School of Medicine, Loma Linda, CA

Fatty acid binding protein 5 (FABP5), an intracellular lipid chaperone, is upregulated during cellular stress, including palmitic acid-induced LTx (PA-LTx) and nerve injury. Previously, we showed FABP5 functions as an antioxidant in neuronal cells, supporting axonal growth and nerve regeneration. Polyunsaturated fatty acid Docosahexaenoic acid (DHA) also regulates FABP5, which is associated with improved functional recovery following injury. Autophagy, a key process for degrading damaged cellular components, is disrupted during PA-LTx and nerve injury, with enhanced autophagy promoting cell survival. Emerging research suggests interactions between FABP5 and autophagy-related proteins. Therefore, this study investigates the functional role of FABP5 in autophagy during PA-LTx injury in immortalized Schwann cells (ISC). PA-LTx was induced by treating ISCs with 300 μ M PA:150 μ M BSA for 24–48 hours, and 50 μ M DHA co-treatment inhibited LTx. Chloroquine (CQ) and rapamycin served as controls for autophagy flux inhibition or induction, respectively. Prior to treatments, FABP5 was silenced by siRNA. Western blot and RT-qPCR were used to confirm knockdown efficiency and to monitor

autophagy by measuring LC3-II and p62/SQSTM1 protein levels, and expression of autophagy-related genes. WST-1 assay assessed cell viability. At 24 hours, PA and CQ increased FABP5 expression and increased LC3-II and p62, which is consistent with increased oxidative stress and impaired autophagy flux. DHA co-treatment normalized these markers, suggesting enhanced autophagic clearance and reduced stress. Interestingly, FABP5 knockdown reduced LC3-II and p62 accumulation in response to PA and CQ, suggesting that FABP5 levels may affect autophagosome formation or cargo degradation. FABP5 silencing also decreased cell viability in PA/DHA and CQ groups. These findings suggest a potential role for FABP5 in autophagy dynamics, which regulates cellular homeostasis during lipotoxic stress.

Lab Volunteers and Research Assistant

DARINE ABU HILAL
DR. FIGUEROA LAB VOLUNTEER 2025

This past spring, I finished my first year at the University of California, Los Angeles pursuing a degree in neuroscience with a minor in public health. My career goal is to become a physician focusing on holistic medicine and working with underserved communities, both in the United States and overseas. My time as a research volunteer at Loma Linda University has changed how I approach problems in the world. I used to fixate on a single piece of the puzzle, but now I consider the larger picture to gain a better understanding of the issues at hand. I have learned the importance of looking past mundane details and prioritizing the contextual context.



Under the guidance of Dr. Johnny Figueroa and my peer mentor, Julio Sierra, I have been working on a research project to investigate the effects of psychosocial trauma on maladaptive eating behaviors. I have learned to consider the broader scope of our research and recognize the real-world implications, particularly as a health disparities issue disproportionately affecting underserved communities, which is something that resonates deeply with me. During my time in the Fig Neurolab, I have not only gained research experience but also acquired several fundamental skills necessary for an aspiring scientist, including critical thinking and scientific communication. I would like to thank Dr. Figueroa and his lab for welcoming me with open arms and providing the resources for me to develop into a well-rounded and skilled researcher.

**INVESTIGATING HORMONAL AND NEURAL MECHANISMS IN MALADAPTIVE
EATING BEHAVIORS**

Azaria Carey¹, Darine Abu Hilal¹, Venjaminne Fua^{1,2}, Julio Sierra¹, and Johnny D. Figueroa¹

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Binge eating disorder (BED) is a serious and common psychiatric condition affecting over 2.8 million people in the United States. It is characterized by episodes of overeating and a loss of control, often occurring alongside environmental stressors such as traumatic stress and exposure to a high-fat Western diet (WD). Evidence increasingly suggests that sex hormones significantly influence the risk of developing maladaptive eating behaviors, especially during adolescence, a critical period for brain development and hormonal shifts. Notably, females are more frequently affected, highlighting the importance of sex-specific research. However, the interaction between adversity, diet, and fluctuations in sex hormones in shaping neural circuits involved in reward, stress, and appetite remains poorly understood. The hippocampus and ventral tegmental area (VTA) are key regions involved in these processes. This study aimed to clarify how early-life exposure to a WD and traumatic stress affects hormone levels and neural activity in a sex-dependent manner. We used a novel adolescent rat model of trauma-induced binge eating and measured circulating steroid and sex hormones, including progesterone, estradiol, and testosterone, using ELISAs. Immunofluorescence was employed to evaluate excitatory and inhibitory neuronal markers. Trauma and WD exposure significantly altered progesterone and

estradiol levels in males and increased testosterone in both sexes. These changes indicate that adolescent stress disrupts neuroendocrine pathways that regulate food intake and reproductive health. Our findings reveal a complex relationship between diet, stress, and sex hormones that may impact the gut-brain connection and neuronal control of eating behavior. Understanding these processes is crucial for creating targeted interventions that address sex-specific vulnerabilities in BED and related conditions.

RAQUEL BENDITA LARICO
DR. FIGUEROA LAB VOLUNTEER 2025

My interest in science began in high school when I visited a petroleum factory. I was fascinated by and desired to understand how chemical reactions occur. Due to my interest in chemistry and mathematics, I pursued a degree in chemical engineering at the National University of Callao, Peru. After graduation, I worked in industry for a couple of years as a chemical engineer, but I felt something was missing. My passion for science led me to volunteer as a lab assistant in a chemistry lab and teach organic and inorganic chemistry courses. During this time, I was advised to apply to a master's program in the Earth and Biological Sciences department at Loma Linda University, eventually graduating with a research focus in geochemistry.



I am currently a research volunteer investigating the impact of high fat diet on neurodevelopment during adolescence. My project is focused on diet-induced alterations in gut metabolite profiles to identify potential biomarkers for aberrant brain maturation. I aspire to pursue a Ph.D. in the Integrated Biomedical Graduate Program in Basic Sciences. My long-term goal is to continue exploring diet-induced differences in metabolomic signatures to further our understanding of how the gut influences metabolic health and brain function. I would like to thank Dr. Johnny Figueroa for giving me the opportunity to be a part of his lab and contribute to his research while developing my skills as a scientist. I hope to conduct research with the purpose to serve and help others and to be hands of God.

**UNTARGETED METABOLOMICS REVEALS DISRUPTED STEROID HORMONE
BALANCE FOLLOWING ADOLESCENT HIGH-FAT DIET CONSUMPTION IN MALE
LEWIS RATS**

Reubin Kim, Raquel Bendita, Julio Sierra, Guangyu Zhang, and Johnny D. Figueroa

Center for Health Disparities and Molecular Medicine, Mass Spectrometry Core, Department of
Basic Sciences, School of Medicine, Loma Linda University, Loma Linda, CA, United States

Adolescent obesity is a global health concern impacting 20% of adolescents in the United States. This issue is exacerbated by the increased consumption of Western-like-high-saturated-fat diets (WD). Adolescent exposure to a WD has been linked to metabolic disruption, hormone imbalance, and altered eating behaviors in adulthood. Building on our previous work, which demonstrated that adolescent exposure to a Western diet induces lasting alterations in gut microbiota, we now investigate how such dietary patterns reshape fecal metabolite profiles and influence hormone-related metabolic pathways. This area remains underexplored using untargeted metabolomics. In this study, we examined the effects of WD consumption during adolescence on fecal metabolite profiles and hormone-associated metabolic pathways in male Lewis rats. Adolescent male Lewis rats were fed either a WD (41% fat) or a control diet (16% fat) for four weeks. Fecal samples were collected, processed for metabolite extraction, and analyzed using the Orbitrap Exploris 240 mass spectrometer and Compound Discoverer 3.3 software. Differential analysis revealed 631 upregulated and 115 downregulated metabolites in WD-fed rats. Pathway analysis revealed alterations in key metabolic pathways, including those involved

in steroid hormone biosynthesis. These findings support prior observations that adolescent WD exposure elevates testosterone and alters steroidogenic activity. Our data validates this model for studying diet-induced hormonal shifts during adolescence. Because steroid hormone imbalances can influence fat deposition, energy expenditure, and appetite regulation, these shifts may increase obesity risk. Future work will incorporate psychosocial stress to explore how environmental factors contribute to long-lasting adverse health outcomes.

JULIA SOLIZ
DR. CASIANO LAB VOLUNTEER 2025

I have always been drawn to science and the challenge of problem-solving, which is part of what led me to pursue a career in medicine. I love the relational side of patient care, but I have also found my curiosity extending beyond the bedside to what happens behind the scenes in the lab, where so many of today's treatments begin.

This summer, I have had the privilege of volunteering in Dr. Carlos Casiano's lab, working with Krystal Santiago and Lemti Nyirendah investigating the role of enolase and anti-enolase antibodies in prostate cancer. From this experience, I have gained hands-on skills in flow cytometry, cell culturing, immunoblotting, and immunofluorescence microscopy. More than that, I have developed a deeper appreciation for the scientific process through seeing firsthand how ideas take shape, how data is built through collaboration, and how discoveries in the lab lay the foundation for future clinical breakthroughs.

I am entering my second year at Loma Linda University School of Medicine, where I will serve as Class President after serving as Vice President last year. Outside of academics, I help lead Fusion Ministries, a local outreach in San Bernardino focused on building relationships with individuals experiencing homelessness. Although I have not yet chosen a specialty, this summer has opened my mind to the possibility of a future in academic medicine. I am grateful for the guidance and mentorship I have received from Dr. Casiano and for the chance to be part of a project that could one day impact patient care.



**ANALYSIS OF ENO1 AND ENO2 CELL SURFACE LOCALIZATION AND EXPRESSION
IN NEUROENDOCRINE PROSTATE CANCER**

Julia Soliz, Krystal R. Santiago, Lemti Nyirendah, Pedro T. Ochoa, Kai Weng Chen,
and Carlos A. Casiano

Center for Health Disparities and Molecular Medicine, Departments of Basic Sciences and
Surgical Urology, School of Medicine, LLUH Cancer Center, Loma Linda University, Loma
Linda, CA

Prostate cancer (PCa) is the second leading cause of cancer-related death in men in the United States, with African American (AA) men exhibiting disproportionately higher mortality rates. Treatment of metastatic castration-resistant prostate cancer (mCRPC) includes androgen-targeted therapies, taxane-based chemotherapy, and PSMA-targeted theranostic approaches. However, resistance to these therapies and limited PSMA expression in a subset of patients, particularly those with neuroendocrine features, highlight the need for alternative therapeutic and theranostic targets. Enolase-1 (ENO1), a glycolytic enzyme with emerging non-glycolytic roles, has been detected on the surface of various tumor types, where it exerts plasminogen receptor roles contributing to tumor cell metastasis. ENO1 is also the target of circulating autoantibodies in patients with cancer, including PCa, highlighting its potential as a tumor biomarker, tumor-associated antigen, and therapeutic target. Enolase-2 (ENO2) is a neuronal isoform of ENO1 that is expressed in neuroendocrine prostate cancer (NEPC), a highly

aggressive and incurable form of the disease. Previous flow cytometry experiments in our lab using specific commercial antibodies against ENO1 and ENO2 showed the presence of ENO1 but not ENO2 on the surface of NEPC-like (NEPC-L) cell lines. We aimed to reproduce these results with a different set of commercial antibodies to these two proteins to confirm their cell surface abundance and intracellular expression in NEPC-L cell lines. In addition, we hypothesized that PCa patient autoantibodies to these proteins recognized both the intracellular and surface variants. To test this hypothesis, we used PCa patient sera known to contain circulating autoantibodies to ENO1 and/or ENO2, as previously determined in our lab by enzyme-linked immunosorbent assay (ELISA) and immunoblotting. Docetaxel-sensitive and -resistant NEPC-L cell lines were incubated with commercial and patient antibodies to ENO1 and ENO2 and assessed for intracellular staining (permeabilized cells) and surface binding (non-permeabilized cells) via flow cytometric analysis. Analysis of the flow cytometry data is currently in progress, with initial findings suggesting stronger intracellular and surface staining for ENO1 compared to ENO2. Additionally, we confirmed by immunoblotting that ENO2 protein is expressed in NEPC-L cell lines (e.g. PC3, DU145, NCI-660 and LASCPC-01), but not in non-NEPC cell lines (e.g. LNCaP), while ENO1 protein was expressed in all the examined cell lines. Consistent with this, analysis of publicly available gene expression databases (GEPIA, TCGA, Protein Atlas) showed that ENO1 transcript and protein are expressed at higher levels in primary prostate adenocarcinoma tumor samples compared to ENO2. Demonstrating that ENO1 and/or ENO2 localize to the surface of prostate tumor cells is critical for understanding the autoantibody response to these proteins in PCa patients, and for the development of novel therapeutic approaches, including antibody-based theranostics, for the treatment of NEPC.

JAY DENG
DR. UNTERNAEHRER LAB VOLUNTEER 2025

I'm a rising senior currently attending the University of Redlands majoring in Biology with a minor in Psychology. I found a passion for molecular biology and genetics during my time at the UofR. As a lifelong athlete, I have always been fascinated by the inner workings and complexities of the human body. My life's dream is to help others and I hope to attend medical school following my undergraduate years, eventually becoming a practicing physician (though which field is yet undecided).



Massive thanks to Loma Linda University's SURF program, the Unternaehrer lab, and Dr. Unternaehrer herself for teaching me so much and for giving me this amazing opportunity and incredible experience this summer. I would also like to specifically thank the wonderful fellows who work/have worked under her: Vishwa Shah, Ashlyn Conant, and Brigitte Vazquez for their unwavering mentorship and guidance. I look forward to continuing my research with Dr. Unternaehrer's lab in the following academic year, and to furthering both my knowledge of and contributions to the field.

EFFECT OF BMI-1 OVEREXPRESSION ON OVARIAN CANCER CELL STEMNESS

Jay Deng, Vishwa Shah, Ashlyn Conant, Juli Unternaehrer

Department of Biology, University of Redlands, Redlands, CA, USA; Division of Biochemistry, Department of Basic Sciences, Loma Linda University, Loma Linda, CA, USA; Department of Gynecology and Obstetrics, Loma University, Loma Linda, CA, USA

Despite being the 11th most common cancer in women, ovarian cancer is the 4th leading cause of cancer death in women worldwide as the most fatal of all gynecologic cancers. High-grade serous ovarian cancer (HGSOC) accounts for approximately 70-80% of all ovarian cancer cases and deaths, with its poor prognosis and high fatality rate largely due to late-stage diagnosis, high recurrence rate, and acquired chemotherapy resistance. There are currently little to no effective methods for early-stage screening, and 80% of HGSOC cases are not diagnosed until the cancer has already reached an advanced stage. Most HGSOC cases initially present platinum-sensitive in which the tumor is responsive to chemotherapy treatment, but upon recurrence present as platinum-resistant. Platinum-resistant cases show severely reduced sensitivity to chemotherapy drugs, such as cisplatin, and the high prevalence of this occurrence further contributes to the high fatality rate. Our lab's work focuses on oncogenes that are implicated in stemness, the EMT process, and chemotherapy resistance to cisplatin. RNA sequencing of a cisplatin-resistant patient-derived xenograft cell line PDX4 CR and its cisplatin-sensitive sister cell line PDX4 SE revealed upregulation of multiple genes implicated in all three criteria. Among those identified is BMI-1, a stem cell factor oncogene. Previous literature has demonstrated a role for BMI-1 in cisplatin resistance and stemness; however, there are little to no known studies examining the effect of BMI-1 overexpression on either. This study aims to test whether BMI1 overexpression induces resistance and/or stemness in the PDX4 SE cells and other sensitive cell lines. We hypothesize that overexpressing BMI-1 induces both resistance and stemness in ovarian cancer cells. Overexpression was achieved through lentiviral transduction

of ovarian cancer cell lines OVCAR8 and patient-derived xenograft PDX4 SE/CR, and confirmed with RT-qPCR. Chemosensitivity of transduced cells compared to controls will be assessed with MTT assays to measure viability with serial dilutions of cisplatin.

HANS WESTENBURG
DR. SEAN WILSON LAB VOLUNTEER 2025

Engaging in laboratory research at the Loma Linda Center for Perinatal Biology during my high school and undergraduate years introduced me to the investigative side of medicine and sparked a lasting interest in understanding the underlying mechanisms of disease. Now, as an incoming medical student to Loma Linda University, I am proud to see how this early exposure to biology research continues to shape my approach to both learning and patient care.



Currently, I work as an EMT for American Medical Response, where I respond to 911 calls across San Bernardino County. These frontline experiences have shown me firsthand how chronic health conditions can shape acute medical emergencies. Through caring for patients suffering from advanced respiratory diseases, I became motivated to pursue research on long-term hypoxia in the Wilson Lab, where I aim to contribute to a deeper understanding of the physiological toll of chronic respiratory conditions.

I am grateful to be a part of this research, and would like to sincerely thank Dr. Sean Wilson for his mentorship and support. His guidance continues to inspire me as I prepare to integrate research and clinical practice in my journey toward becoming a physician.

METABOLIC REPROGRAMMING OF SHEEP PULMONARY ARTERIES IN RESPONSE TO LONG TERM HYPOXIA

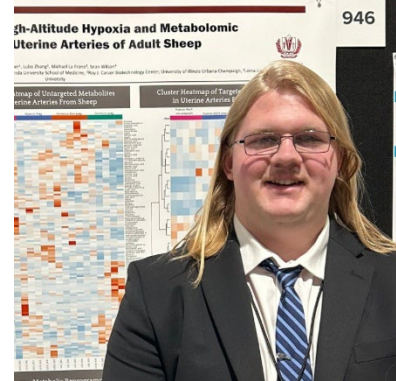
Hans C. A. Westenburg, Alyssa Sorrell, Justis Cosper, Michael La Frano, Sean M. Wilson

Salk Institute for Biology Studies, La Jolla, CA; Lawrence D. Longo, MD Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA

Long-term hypoxia (LTH) triggers structural and functional changes in the pulmonary vasculature, contributing to pulmonary vascular remodeling and the development of pulmonary hypertension (PH). PH has significant clinical implications, including right ventricular hypertrophy and, in severe cases, right-sided heart failure. We hypothesize that LTH drives metabolic reprogramming in pulmonary arteries, contributing to these pathological changes. This study aims to further identify biochemical pathways that may drive structural and functional changes associated with LTH in the pulmonary vasculature. To investigate this, we performed untargeted as well as targeted analysis for small metabolites and lipid mediators in pulmonary arteries from adult sheep exposed to normoxic (700 m, $n = 6$) or hypoxic (3801 m, $n = 7$) environments. Following arterial excision, metabolomic profiling (UPLC-MS/MS) was performed, with analysis performed using MetaboAnalyst. Enrichment analysis of significantly altered metabolites revealed disruptions in key metabolic pathways, including glycolysis, sugar metabolism, and amino acid metabolism. Targeted metabolomic analysis identified significant reductions in specific lipid mediators after LTH exposure. Notably, levels of oleoyl glycine, LTB₅, 9-HOTE, and 19(20)-EpDPE were decreased. These lipid mediators have been previously linked to pulmonary vasoconstriction, vascular remodeling, and oxidative stress. Our findings support the role of metabolic reprogramming in hypoxia-induced pulmonary vascular pathology—which remains an active area of investigation.

JUSTIS LESTAT ASHTON COSPER
DR. SEAN WILSON LAB VOLUNTEER 2025

Upon entering my junior year of undergraduate education at the University of Redlands, I have been so graciously eligible to further our understanding of perinatal biology by working alongside Dr. Sean Wilson. In my freshman year of university, I developed a personal desire to pursue scientific research in any capacity, inspired by the drive to further humanity's knowledge, as my undergraduate professors inspired me. This shortly led me to Dr. Bryce Ryan, who then introduced me to my aforementioned Principal Investigator, Dr. Sean Wilson.



Working alongside Dr. Wilson has been a phenomenal experience, allowing me to further my goals of pursuing research, which has only further cemented my internal desire to pursue an MD/PhD program. The captivating aspect of research is its long-term goal: that eventually, someone will greatly benefit from the combined tens, if not hundreds, of thousands of hours of intensive collaborative effort uniquely seen within laboratories. This idea instills in me a burning desire to pursue research not simply for the sake of enhancing a résumé, but because it will ultimately benefit others.

Being graciously allowed to present the collaborative research efforts on alterations in calcium signaling under specific stimuli, such as long-term high-altitude hypoxia, is always a memorable experience, as it allows me to share our combined work with those who listen.

IMPACT OF GESTATIONAL LONG-TERM HYPOXIA AND MICRORNA-210 ON LOCAL CA²⁺ SIGNALS AND PULMONARY ARTERIAL FUNCTION IN NEWBORN SHEEP

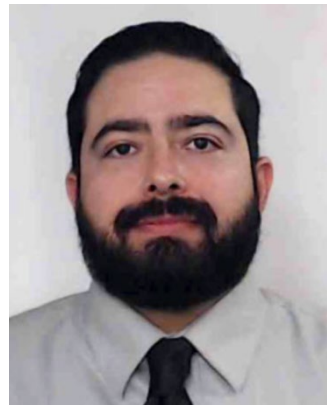
Justis Cosper, Ashley Thompson, Eris Albert-Minkler, Lubo Zhang, Arlin B Blood, Jose L Puglisi, Sean M Wilson

Center for Health Disparities and Molecular Medicine, Perinatal Biology, School of Medicine,
Loma Linda University, Loma Linda, CA

Pulmonary hypertension in newborns is driven by hypoxic disruption of pulmonary arterial vasoreactivity, a process influenced by subcellular Ca²⁺ signaling. This study investigates the impact of gestational long-term hypoxia (LTH) and microRNA-210 (miR-210) on local Ca²⁺ spark activity and pulmonary arterial function in newborn sheep. Pregnant ewes were maintained either under normoxic conditions (350 meters above sea level) or within hypoxic chambers that mimicked elevations of 3,801 meters. Pulmonary arteries were isolated from their newborn lambs. Isolated arteries were exposed to various treatments, including 30 mM K⁺ (to induce depolarization) and ryanodine (to block ryanodine receptors), with or without 30 mM K⁺. Additional animals were transfected with a vector containing miR-210, or with other constructs including an empty vector, antagomir, or LNA-scrambled controls. Ca²⁺ sparks were visualized in ovine pulmonary myocytes using line-scan imaging of Fluo-4 with a laser scanning confocal microscope and quantitatively analyzed with custom line-scan analysis software (SparkLab). Results demonstrated that LTH increased Ca²⁺ spark activity in newborn pulmonary arterial myocytes relative to controls. Both hypoxia and miR-210 overexpression

altered the spatiotemporal characteristics of Ca^{2+} sparks, suggesting distinct mechanisms of disruption. Ryanodine reduced Ca^{2+} spark activity in specific groups. Notably, miR-210 downregulation differentially modulated Ca^{2+} spark activity compared to LTH, implicating separate influences on RyR function. Control and antagomir treatments also had unique effects, further complicating interpretation. These findings indicate that both gestational hypoxia and miR-210 independently modify Ca^{2+} spark dynamics in newborn pulmonary arterial myocytes, likely through differential regulation of the ryanodine receptor, with varied consequences for vasoregulation. Understanding these mechanisms may inform strategies to address pulmonary hypertension in at-risk neonates.

Curiosity and the potential to gain new experiences was the driving force behind my decision to join the CHDMM summer research program this year. This experience will serve as an incredible opportunity in my pharmacy career journey. I was warmly welcomed into Dr. Eileen Brantley's laboratory where I am engaged in a project to investigate the potential for rutin to inhibit DNA methyltransferase 1 to confer anticancer activity against triple negative breast cancer cells derived from patients of African and European ancestries. This project has ignited my passion for future research. I have acquired invaluable laboratory skills and a greater understanding of cancer biology. This experience has highlighted the immense and urgent need for ongoing oncology research.



This fall I will begin my second year as a student at Loma Linda University School of Pharmacy as a Hispanic Center of Excellence in Pharmacy (HCEP) scholar. God willing, I will receive my PharmD degree in 2028 and pursue a residency to advance my career as a pharmacist specializing in oncology. I will aim to improve patient care and cancer therapeutic outcomes. My goal is to make a meaningful impact on underserved communities in the Inland Empire. I thank Dr. Eileen Brantley, Michael Hall, and Ozichi Amobi for the warm welcome into the lab and the incredible experience and learning opportunity thus far.

RUTIN CONFERS ANTICANCER ACTIONS IN BREAST CANCER CELLS DERIVED FROM PATIENTS OF EUROPEAN AND AFRICAN ANCESTRY

Jonathan DeAnda, Elyssa Fraser, Michael Hall, Ozichi Amobi, Victoria Chinedu, John Khalaf,
Eileen Brantley

Center for Health Disparities and Molecular Medicine, Cancer Developmental and Regenerative
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While breast cancer survival has increased overall, women of African Ancestry experience a disproportionate risk for breast cancer mortality. Emerging data suggest that differences in tumor biology influence patient responses to therapies, contributing to this disparity. Triple negative breast cancer (TNBC), which lacks expression of hormone receptors and human epidermal growth factor receptor 2, is the most aggressive breast cancer subtype and is more commonly diagnosed among women of African ancestry. Patients with TNBC often receive chemotherapy, which is associated with numerous adverse effects. The aim of our study was to assess the anticancer activity of rutin, a flavonoid derived from plants, in TNBC cells derived from patients of African and European ancestries. Using the Alamar Blue assay, we discovered that rutin decreased the viability of select TNBC cells. The colony-forming, mammosphere-forming, and wound-healing assays revealed that rutin exhibits antiproliferative, anti-stemness, and antimigration properties, respectively. TNBC cells derived from patients of African ancestry were particularly susceptible to the anticancer actions of rutin. Our data suggest that rutin has the potential to improve clinical outcomes among patients with TNBC. Studies are underway to define the mechanism of anticancer action for rutin and to elucidate its ability to enhance chemotherapeutic efficacy in preclinical models derived from patients of diverse ancestries.

ARIANNA WILLIAMS
CHDMM RESEARCH ASSISTANT

Growing up in an environment full of inquiring minds inspired me to question the relationship between science, faith, and human purpose. Motivated by such a rich backdrop, I now seek to pursue a career in studying the intricacies that drive human behavior alongside treatments and strategies to serve underrepresented communities. My career goal is to obtain a Doctorate in Clinical Psychology, which will lead me to establish my private practice that offers a range of short-term solutions and long-term plans for chronic psychiatric disorders.

Extracurricular activities that add to my goals and purpose include indoor and outdoor rock climbing and holding multiple leadership positions in clubs that pursue Diversity, Equality and Inclusion through community activities and open discussions, thus guiding me to try new things and opening new doors around me.

I will be a Junior at La Sierra University with focus and dedication to the summer experience for the CHDMM program. I want to thank the mentorship team under Dr. Johnny D. Figueroa, especially IMSD student Julio for helping me recognize my strengths, enhancing my understanding of the role of research in neuropsychology, and showing me how I can contribute to a growing scientific community. Coming from a line of pioneer scientists, I seek to continue their legacy of care and compassion through research and service.



LEVETIRACETAM ATTENUATES BINGE-LIKE EATING IN SOCIALLY ISOLATED FEMALE RATS

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¹Department of Psychology, La Sierra University, Riverside, CA

²Center for Health Disparities and Molecular Medicine, Loma Linda University, Loma Linda, CA

Social deprivation, particularly during the critical developmental window of adolescence, has been shown to disrupt emotional regulation and increase vulnerability to binge eating behaviors. However, the precise neural mechanisms linking social deprivation stress to binge eating remain poorly understood. Emerging evidence suggests that adolescent social deprivation impairs synaptic maturation and disrupts the excitatory/inhibitory (E/I) balance in corticolimbic brain circuits involved in emotion and reward processing, thereby promoting binge eating. Synaptic Vesicle Protein 2A (SV2A), a key modulator of synaptic transmission and E/I homeostasis, is reduced in individuals with obesity and those exposed to stress, potentially linking social deprivation stress with bingeing. Yet, SV2A's role in stress-induced bingeing remains undefined. We hypothesized that the SV2A modulator, Levetiracetam (LEV, Keppra®), would attenuate binge-like behavior following social isolation (SI). Adolescent Lewis rats (n = 64; 50% female) were either pair-housed or subjected to SI and exposed to a three-week intermittent access model of high-fat diet-induced binge eating. In the third week, rats received either LEV (10 mg/kg) or vehicle before reintroduction to the high-fat diet. Behavioral tests and brain scans were conducted thereafter. SI led to increased weight gain, binge-like eating, heightened startle responses, and reduced sociability, particularly in females. Notably, LEV significantly reduced binge-like eating in isolated female rats but not in males. Consistent with prior findings, binge-eating females exhibited reduced hippocampal SV2A

mRNA levels, suggesting a molecular signature of stress-induced vulnerability. These results implicate SV2A-mediated E/I imbalance as a potential mechanism linking social stress to binge eating and highlight SV2A-targeting drugs as promising treatments for binge eating disorder.

**Summer Undergraduate
Research Fellowship
(SURF)**

DANIEL BALWIN
SURF PARTICIPANT 2025

My foray into academic research started a decade ago when my father asked me to assist on a research project concerning noise pollution in the operating room. This experience opened my eyes to the exciting world of medical research. Since then, I have been able to build off this experience to have a diversity of research projects including studying overactive bladder symptoms and their relation to patient personality, the role of macrophages in cervical ripening, and catalysts of limb loss in invasive spiders in the American Southeast.

I currently attend Southern Adventist University in Collegedale, Tennessee. I am a biology – research major with a minor in chemistry. After graduation, I hope to enroll in a medical scientist training program and pursue an MD/PhD. This will give me a rich background with the methods of a scientific researcher and clinical experience of a medical doctor providing me a unique framework to tackle the problems which still face us regarding human health. This line of work is personally fulfilling, as I can see the impact of my research in the lives of my patients.

This summer, I have had the distinct pleasure of working under my principal investigator Dr. Kerby Oberg and senior research associate Charmaine Pira. They have been incredibly kind and informative, patiently teaching me relevant techniques and information.



**FIBROBLAST GROWTH FACTORS (FGFS) OF THE APICAL ECTODERMAL RIDGE
UPREGULATE *LIM HOMEBOX 2 (LHX2)* DURING LIMB OUTGROWTH AND
PATTERNING**

D. Daniel Baldwin, Charmaine U. Pira, Kerby C. Oberg
Department of Pathology and Human Anatomy, Loma Linda University, Loma Linda, CA

During development, fibroblast growth factor 10 (Fgf10) in the presumptive limb mesoderm induces thickening of the distal ectoderm to form the apical ectodermal ridge (AER). The AER produces additional Fgfs to drive progressive outgrowth and limb patterning. These AER-related Fgfs also maintain Fgf10 in the underlying distal mesoderm, forming a positive feedback loop. *Lim homeobox 2 (Lhx2)* is expressed in the distal limb bud mesoderm maintaining a population of undifferentiated cells at the distal tip during this progressive outgrowth. *Lhx2* is also thought to coordinate patterning along the three asymmetrical axes (proximodistal, anteroposterior, and dorsoventral) during limb development. Functional loss of *Lhx2* leads to limb truncations and digit loss. The regulation of *Lhx2* has not been well characterized. However, removal of the AER results in the loss of *Lhx2* expression and limb truncation, suggesting a role for AER-Fgfs in the regulation of *Lhx2*. We hypothesized that the AER-secreted Fgfs (Fgf2, 4, 8, 9, and 17) would upregulate the expression of *Lhx2*. To interrogate this hypothesis, we implanted Fgf-soaked heparin acrylic beads into central limb mesoderm adjacent to the *LHX2* expression domain of a chicken embryo. These embryos were incubated for 4 hours, harvested, and evaluated by whole mount *in situ* hybridization for *LHX2* expression. Our results demonstrated increased expression in response to several of the Fgfs examined. Thus, our work documents that AER-Fgfs upregulate *LHX2* in the distal limb. This data contributes to our understanding of the mechanisms by which the AER regulates *Lhx2* and the underlying mesoderm during limb development, providing critical insights into how limb truncation birth defects might occur.

KIAR-RA CAMERON
SURF PARTICIPANT 2025

Building a sense of community is of utmost importance on my journey to becoming a physician-scientist. Throughout my future practice, I aim to deliver the best care possible by combining science and medicine to find long-term solutions. I am a rising Junior Biology Pre-Medicine student at Oakwood University in Huntsville, Alabama. Additionally, I enjoy working alongside students at STEM Success Academy and organizing tutoring programs for the Oakwood iLearn Initiative.



In the 2025 Summer Undergraduate Research Fellowship (SURF) program, I had the opportunity to investigate the use of Mesenchymal Stem Cells to ameliorate the effects of Bronchopulmonary dysplasia (BPD). Ultimately, this experience led me to be a part of something larger than myself. Moreover, participating in research team meetings and daily communication with my team helped to develop a sense of togetherness. A Bible verse used as my guide was “Do nothing out of selfish ambition or vain conceit...” (Philippians 2:3, NIV).

While working alongside my counterparts, I learned to look beyond textbook answers and seek more practical experiences. I also learned to look at the broader picture of an issue, then narrow down the specifics. As I enter a new phase of becoming a Junior, I am looking forward to future experiences. At Loma Linda University, I learned to embrace the unknown, for it is during this time that the most growth occurs.

A heartfelt thanks to the SURF program coordinators, Dr. Sean Wilson’s lab, and Dr. Ciprian Gheorghe.

**MORPHOMETRIC ANALYSIS OF MESENCHYMAL STEM CELL-DERIVED EXOSOME
THERAPY IN A MURINE MODEL OF BRONCHOPULMONARY DYSPLASIA**

Kiar-Ra Cameron, Aditi Bhatnagar, Cassidy Webb, Ciprian Gheorghe, and Sean M. Wilson

Center for Perinatal Biology, Loma Linda University School of Medicine, Loma Linda, CA

Preterm neonates are susceptible to Bronchopulmonary Dysplasia (BPD), which is caused by using ventilators to aid in lung ventilation. Ultimately, the high-oxygen and pressure environment disrupts the neonatal alveoli, reducing surface area and increasing wall thickness. The focus of this study was to determine if treatment with exosomes derived from mesenchymal stem cells (MSC-exos) isolated from Wharton’s Jelly in human umbilical cord (hUC-WJ) would mitigate the effects of hyperoxia on lung structure in a murine model. Following exposure to normoxia (21% O₂) or hyperoxia (95% O₂) from postnatal (PN) day 1 to PN5, on PN1 and PN4, mouse pups (n=3) were injected intraperitoneally with MSC-exos. Lungs were excised on PN14 and stained with hematoxylin and eosin, which enables visualization of the septal walls and alveoli. High-resolution digital images were then captured at 20x magnification using brightfield microscopy. Using ImageJ, obtained images were resized using the “cropPNG.ijm” macro to 680 by 680 pixels and semi-automated morphometric analysis was conducted using the “Morphometry_v4.0” plugin. The plugin uses stereological techniques to determine values of the

septal volume (V_{vsep}), mean linear intercept (Lm) and other parameters. Analysis is ongoing, where the Lm values for the normoxia, hyperoxia, hyperoxia+exos and normoxia+exos are being measured and compared using ANOVA. We expect the Lm value for the hyperoxic group to be significantly higher than the normoxic control, while treatment with MSC-exos will reduce the value towards that of normoxic animals. This study aims to verify the therapeutic effect of MSC-exos in the BPD murine model through morphometric analysis to support future studies on characterization of exosomal content and determine the mechanism of therapeutic action.

BENJAMIN CHUN
SURF PARTICIPANT 2025

I learned the true meaning of compassion from my grandmother. Through supporting local efforts, delivering meals, and babysitting, she left a lasting, positive mark on her community. Her long struggle with dementia inspired me to bridge my passion for engineering with the field of medicine to improve healthcare. Her unwavering selflessness and tenacious battle against dementia continue to inspire my path toward an MD-PhD physician-scientist.

I am a junior attending Southern Adventist University in Collegedale, Tennessee, where I am pursuing a double-major in Biophysics and Mechanical Engineering. In honor of my grandmother's heart of service, I've sought to give back to my community. From gaining clinical experience at an Alzheimer's Care Center, attending medical mission trips, and leading worship at my church, I've learned the true meaning of service. I've also held leadership roles as Vice President for the Pre-Med club and Religious Vice President for the Physics and Engineering clubs.

Prior to SURF, I was able to assist Dr. Vola Adrianarijaona in constructing an alkaline oven apparatus for ion collision experiments and co-write a successful grant proposal that earned national recognition from the Society of Physics Students. I am currently conducting research under Dr. Christian Hurtz to standardize electroporation protocols for various leukemia cell lines. This process enables efficient delivery of CRISPR-Cas9 ribonucleoproteins into cells for future gene editing applications.

I'm grateful to Dr. Hurtz for this opportunity, which has given me hands-on experience with wet lab techniques and critical problem-solving, opportunities unavailable at my small institution.



SMARCA4'S ROLE IN ACUTE LYMPHOBLASTIC LEUKEMIA

Benjamin Chun, Ria Perencsik, V.S.S. Abhinav Ayyadevara, Shreya Patil, Christian Hurtz

Department of Basic Sciences, Cancer Sciences Division, School of Medicine, Loma Linda University, Loma Linda, CA

The SMARCA4 gene encodes a key ATPase that is part of the SWI-SNF chromatin remodeling complex. Although SMARCA4 is most known for its role in solid tumors, researchers have underpinned its potential contribution to leukemogenesis in blood cancers like acute lymphoblastic leukemia (ALL). Patients with high-risk subtypes of ALL, such as Philadelphia-like (Ph-like) or KMT2a-rearranged (KMT2a-r), do not respond well to conventional cancer treatments like chemotherapy or radiation, indicating the need for novel therapies. Previously, we found that Ph-like ALL patients with higher SMARCA4 expression levels were associated with poorer outcomes. This suggests that SMARCA4 may serve as a potential therapeutic target in Ph-like ALL patients specifically.

To determine why higher SMARCA4 expression levels are associated with poorer outcomes in Ph-like ALL, we have used the small molecule inhibitors BRM014 and FHD-286 to assess the

sensitivity of Ph-like and KMT2A-R ALL to SMARCA4 inhibition. To identify mechanistically why Ph-like ALL cells are dependent on SMARCA4, we are currently establishing a standardized electroporation protocol to enable CRISPR-mediated knockout of SMARCA4. Given the off-target effects of the small-molecule inhibitors BRM014 and FHD-286, which also affect SMARCA2, CRISPR-mediated deletion of SMARCA4 will enable us to achieve a clean, specific knockout and facilitate more accurate mechanistic validation in the future.

Although inhibiting SMARCA4 appears to be an effective monotherapy for Ph-like ALL, it is not known whether these drugs can be combined with standard chemotherapy agents to provide a synergistic effect. We are now testing if combining SMARCA4 inhibitors with chemotherapy drugs will help evaluate whether SMARCA4 inhibitors can be included in induction therapy regimens to enhance treatment outcomes. We are specifically testing combinations of FHD-286 with vincristine, dexamethasone, L-asparaginase, and doxorubicin. Identifying synergistic effects may allow for the use of lower doses of chemotherapy drugs, which are associated with severe long-term side effects. This approach could be especially beneficial for adults, who generally tolerate chemotherapy less well than children, and may lead to improved outcomes in the future.

JULIANA GRUENLER
SURF PARTICIPANT 2025

It has been such an honor to participate in the SURF program this summer. It has given me hands-on experience, enabling me to apply concepts I have learned in my classes in real research scenarios. I am a Biochemistry major with a Bible minor at Biola University in La Mirada, California. Working at Loma Linda has been an opportunity to use both my major and minor as I do research in a Christian environment. I feel empowered by the ideals of LLU to serve all people as Christ's hands and feet.

As I approach my junior year in college, I am beginning to formulate my plans for after graduation. This program has introduced me to what the life of a PhD student is like and helped me to consider whether it is the right path for me. I have also gotten the chance to dive deeper into cancer research, specifically in the field of nanomedicine.

I would like to thank everyone in Dr. Rameshwar Patil's lab for teaching me so much and being willing to answer all my questions. I'm so grateful for the space they have given me to learn and grow through both my successful and unsuccessful moments.



LRP1-TARGETED GOLD NANOPARTICLES FOR RADIOSENSITIZATION OF GLIOBLASTOMA

Juliana Gruenler, Kyla Tucker, Cedric Lansangan, Serge Rudensky, Sanjay Yadav, Kevin Nick,
Rameshwar Patil

Department of Basic Sciences and Neurosurgery, Division of Cancer Sciences, School of
Medicine, Loma Linda University, Loma Linda, CA

Glioblastoma (GBM) is a primary brain tumor with high levels of malignancy. The rate of recurrence is so high that less than 5% of patients are expected to survive more than 5 years post-diagnosis. One of the main treatment options for GBM is radiation therapy. However, with recurrence of the tumor, the efficacy of radiation is limited by the sensitivity of surrounding healthy cells to the radiation and resistance to radiation in the tumor cells. High-Z nanoparticles, such as gold nanoparticles (GNPs), can be used as radiosensitizers to enhance the efficacy of radiation, specifically in the cancer cells.

To target the GNPs, Angiopep-2 (AP-2), a synthetic peptide related to low-density lipoprotein receptor-related protein-1 (LRP-1), was conjugated to the nanoparticles to allow the particles to cross the blood-brain barrier. We used Polyethylene glycol (PEG) to stabilize the particles. Various concentrations of PEG were added to the particles along with 5% surface coverage of AP-2. The stability of the particles was evaluated under physiological conditions (PBS) and a gel electrophoresis. GNPs were characterized by various analytical methods such as UV-vis spectrometry, Dynamic Light Scattering, and wet chemical assays well-established in Dr. Patil's lab. GNPs with lower surface coating (<50%) showed significantly higher levels of aggregation, when tested by 2-mercaptobenzothiazole (2-MBT) assay. GNPs with higher surface coating showed superior stability in PBS. In vitro evaluation is ongoing to quantify the cellular uptake of targeted vs nontargeted GNPs in LRP-1 expressing GMB cell lines. Cellular uptake of GNP will be studied by quantifying the internalized gold using Microwave Plasma Atomic Emission

Spectroscopy (MP-AES) in Dr. Kevin Nick's Lab. Results from this study will open new avenues for improved radiation therapy for glioblastoma.

NATALIE HOLM
SURF PARTICIPANT 2025

The challenge of cancer research became deeply personal this past year as I witnessed a close friend's battle with cancer, cementing my dedication to a career in bioengineering research. As a rising senior studying bioengineering at Walla Walla University, I have had the opportunity to apply engineering principles to complex biological challenges. This summer the SURF program has allowed me to translate my bioengineering skills into direct application.

I have had the privilege of working in the lab of Dr. Reinhard Schulte, contributing to a project that studies tumor metabolism and radiation oncology. Under the guidance of my mentor, Ph.D. student Kristian Holgersson, I am investigating tumor-derived metabolites and their effect on cardiomyocytes. We also have an interest in the anti-glycolytic drug TEPP-46 and its potential in suppressing tumor metabolism. This experience has been invaluable, as it has led to an increased confidence in designing experiments and lab skills. When not in the lab, I enjoy paint-by-numbers, skiing, and playing board games.

Ultimately, my goal is to earn a Ph.D. in bioengineering and become a professor, in which I can combine my passions for research and teaching. This love for teaching was discovered after serving as a student missionary teaching high school math and history in Pohnpei, Micronesia.

I extend my sincere gratitude to Kristian Holgersson for his patient and knowledgeable mentorship, which has made this an unforgettable summer of growth.



TOXIC EFFECTS OF TUMOR-DERIVED METABOLITES ON HL-1 CARDIOMYOCYTES

Natalie Holm, Kristian Holgersson, Elva Garcia, Ying Nie, Guangyu Zhang, Reinhard Schulte

Basic Sciences, Loma Linda University School of Medicine, Loma Linda, CA

Purpose/Objective: Cardiac toxicity is a serious, dose-limiting complication in patients undergoing chemoradiation for inoperable stage III non-small cell lung cancer (NSCLC). Recent findings suggest that cardiac toxicity does not originate from radiation exposure alone, but also from tumor-derived metabolites in the pulmonary circulation. As part of a larger study, this project evaluated the cytotoxic impact of lactic acid on murine cardiomyocytes and explored whether the anti-glycolytic drug TEPP-46 can mitigate these effects.

Methods: Murine Lewis lung carcinoma (LLC1) cells were cultured and, using a 2x2 factorial design, were exposed to TEPP-46 (0 μ M vs 200 μ M) and radiation (0 Gy vs 4 Gy). Metabolic profiling of LLC1 cell medium was conducted to quantify treatment-induced changes. Murine HL-1 cardiomyocytes were treated with increasing concentrations of L-(+)-lactic acid and proliferation was assessed with MTT assay and Ki67 immunocytochemistry. A co-culture insert-well system was implemented to study the direct interaction between LLC1 cells and HL-1 cardiomyocytes.

Results: Preliminary analysis revealed a significant downregulation of many tumor metabolites following TEPP-46 treatment, indicating suppressed tumor metabolic activity. HL-1 cardiomyocytes exhibited threshold-dependent proliferation inhibition after lactic acid exposure. The pattern observed with the co-culture system supported these findings, further implicating tumor metabolic-driven cardiac toxicity.

Conclusion: This study indicates that lactic acid exerts dose-dependent cytotoxic effects on murine cardiomyocytes, supporting the hypothesis that tumor metabolites contribute to the observed cardiac complications. It further indicates that TEPP-46 reduces many tumor metabolites, which could prevent these complications. Future experiments should test whether lactic acid disrupts excitation-contraction coupling in HL-1 cardiomyocytes which can be tested with real-time confocal imaging staining for calcium release. Additional experiments will characterize circulating metabolites in tumor-bearing mice and their cardiotoxic effects.

JOSHUA CHRISTIAN LOHR
SURF PARTICIPANT 2025

As far as I know, I have always been curious about the world around me. This curiosity cultivated within me a yearning for knowledge that drove me towards uncovering the inner workings of the visible universe. The physical sciences captivated my interest, developing within me a passion towards making sense of the world through mathematically-derived physical laws.

I am currently attending Southern Adventist University, on the verge of completing my final year with degrees in chemistry, physics and math. I hope to return to Loma Linda University for their MD/PhD program.

Although my primary passion belongs to physics, I also hold passions in other areas. Growing up with travel-oriented missionary parents, I had the opportunity of visiting over 20 countries. Since commencing college, I aided in mission trips to Nigeria, Belize, and Guam. I was also lucky enough to travel to Italy for a semester through Adventist Colleges Abroad. I also enjoy playing piano and reading.

I see my walk with Christ as integral to my journey as a scientist, and I seek to reflect His character in everything I do. Science to me is a path to understanding that which is created. The hope is it brings me closer to the Creator.

I would like to thank Dr. Christopher Perry and Dr. Rameshwar Patil for allowing me into their labs, as well as Cedric Langansan, Kyla Tucker, and Juliana Gruenler for providing me with advice and camaraderie in this beautiful, complex collaboration of knowledge known commonly as science.



**PROBING THE CATALYTIC EFFECTS OF GOLD NANOPARTICLE SURFACES FOR A
PROPOSED CANCER TUMOR RADIOSENSITIZATION MECHANISM**

Joshua Lohr, Juliana Gruenler, Kyla Tucker, Cedric Lansangan, Dr. Sanjay Yadav, Dr. Serge Rudensky, Dr. Jamie R. Milligan, Dr. Rameshwar Patil, Dr. Christopher Perry

Department of Basic Sciences, School of Medicine, Loma Linda University, Loma Linda, CA

Radiotherapy is used to treat malignant neoplasms. Ionizing sources range from photons (gamma rays or x-rays) to particles (alpha particles, protons, or neutrons). A significant challenge with conventional cancer treatments is the collateral damage to surrounding healthy tissue. Therefore, the goal of optimizing therapies is minimizing the dose received while maximizing tumor shrinkage. This is accomplished with radiosensitizers. Chemicals have been used as sensitizers for years; however, gold nanoparticles have recently shown promise as alternatives. In both in vivo and in vitro studies, the radiosensitization effects of gold nanoparticles have been shown to mainly be independent of the ionizing radiation source, resulting in effective radiosensitization at most doses. However, multiple mechanisms behind radiosensitization have been proposed. One explanation involves surface catalysis, where the nanoparticle surface enables the cycling of reactive oxygen species (ROS). In this model, nanoparticles act as enzyme mimetics, serving as electron relays in redox reactions between an electron donor (typically an antioxidant biomolecule) and an electron acceptor (usually an oxygen-containing species). This mechanism

depends on the nanoparticle's surface properties and availability of catalytic centers. My research focuses on ascorbic acid (vitamin C) as a model antioxidant, investigating how the kinetics of this catalytic process vary with nanoparticle size, shape, and surface coating. Surface characterization was performed using UV-Vis to monitor the catalyzed O₂-driven oxidation of ascorbic acid. This work aims to deepen our understanding of the mechanisms underlying the radiosensitization of gold nanoparticles.

KIERA MCGIVNEY
SURF PARTICIPANT 2025

I am originally from Big Bear City, California, but I attend Whitworth University in Washington state. I currently study biochemistry and am going into my junior year this fall. I have previously been interested in organic chemistry, and earlier this year I was given the opportunity to begin research with Dr. Kraig Wheeler studying quasiracemate crystal structures. This marked the beginning of my love for research, inspiring me to apply for the LLU SURF program.

After college, I hope to attend a PhD program with the goal of teaching higher education and eventually leading a laboratory of my own. Spending the summer researching in Dr. Julia Unternaehrer's lab and being mentored by PhD student Ashlyn Conant has deepened my love for the natural world around me. My curiosity for molecular biology has grown, and I can't wait to see the career I land and all the things I will learn along the way.

I am so thankful for the connections and relationships I've made here at LLU. Every day was challenging, yet fulfilling, and I loved every minute of it. I can only hope to become half the scientist my mentors are, and I cannot wait to spend my life pushing the limits of what we know and teaching students that they can achieve anything if they work hard enough, just as I have been taught.

Thank you, Dr. Julia Unternaehrer and Ashlyn Conant, for encouraging me and teaching me more than I have ever learned in my life. It's been a blast!



**VALIDATION OF TYPE-1 INTERFERON PRODUCTION AND RESPONSE IN OVARIAN
CANCER AS A MECHANISM FOR THERAPEUTIC RESISTANCE**

Kiera McGivney, Ashlyn Conant, Julia Unternaehrer

Summer Undergraduate Research Fellowship, Division of Biochemistry, Department of Basic Sciences, Loma Linda University, Loma Linda, CA.

Ovarian Cancer (OVC) is a leading cause of cancer-related deaths among women worldwide, partly due to heterogeneous therapeutic resistant mechanisms. Type 1 interferons (IFN-1) are cytokines that most cells produce as an innate immune response to infection. IFN-1 may play a unique role in cancer, promoting expression of interferon stimulated genes (ISGs) that drive cancer aggression and therapy resistance. Previously, a patient-derived cisplatin sensitive OVC cell line was continuously treated with cisplatin until established as resistant. To examine the mechanism of acquired resistance, RNA sequencing was performed on the sensitive (SE) and resistant (CR) pair, and genes involved in IFN-1 production were found to be upregulated in CR cells. The purpose of this study was to quantify IFN-1 protein expression and validate the mRNA expression of IFN-1 in SE and CR cell lines, and OVCAR8 cell line. It was hypothesized that CR cells produce more IFN-1 than their SE counterpart. To test this, HEK-Blue IFN α/β reporter cells were incubated with the supernatant of OVC samples under various conditions, producing a reporter gene detectable by QUANTI-Blue detection reagent. IFN-1 concentrations were determined by measuring absorbance at 620 nm and interpolating unknown concentrations from a standard hyperbolic curve. The reporter cells successfully detect IFN-1

near the lower limit of detection in OVC cells, but neither unstimulated CR and SE cells, nor virus-stimulated CR and SE cells, show a significant difference in IFN-1 production. These data suggest that although mRNA expression of IFN-1 is upregulated in CR cells, more sensitive tests are required to validate IFN-1 protein expression. Other protein detection methods may be examined to further validate expression of IFN-1.

DANA ROBINSON
SURF PARTICIPANT 2025

I am a student at Southern Adventist University in Collegedale, TN, majoring in biochemistry and international studies. I hope to enroll in a PhD program after earning my bachelor's degree to continue to learn and make meaningful contributions to my field through research. I am grateful for the opportunity to participate in the SURF program, as it has challenged me in new ways, allowed me to explore my interest in research, and helped me grow as an aspiring scientist.

I especially enjoyed learning about the different methods and techniques used in a microbiology/biochemistry lab, and I was fascinated by the projects we worked on this summer. Our research focused on the Aer2 chemoreceptor in *Pseudomonas*, which are bacteria that can cause plant and animal disease. Our goal was to determine the Aer2 receptor response to independent signals through two input domains and to show how the receptors process these signals that are integral to regulating receptor function.

Thank you to Dr. Kylie Watts and Dr. Suzie Phillips for welcoming me into the lab, for their patient guidance, and for showing me what it takes to be an excellent researcher. I appreciate the incredible mentorship I have received this summer.



**DELINEATING THE SENSORY REPERTOIRE OF *PSEUDOMONAS* AER2 RECEPTORS
WITH DUAL, INDEPENDENT SENSING DOMAINS**

Dana Robinson and Kylie Watts

Microbiology and Molecular Genetics,
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Pseudomonas are versatile environmental bacteria that cause plant, animal, and human disease. The human pathogen *Pseudomonas aeruginosa* contains a chemoreceptor called Aer2, which is a heme-based O₂ sensor that regulates stress responses and virulence. In addition to *P. aeruginosa*, Aer2 receptors have been identified in 43 other *Pseudomonas* species. Sequence homologies predict that some of these receptors are unique because they have dual, independent sensing domains. This provides us with an exceptional opportunity to assess signal integration within the same receptor. We chose Aer2 receptors with dual sensing domains from three species: *Pseudomonas corrugata* (PcAer2), *Pseudomonas* sp. HMWF032 (PHAer2), and *Pseudomonas fluvialis* (PfAer2). We hypothesize that these three receptors not only sense O₂ via a PAS-heme domain, but two of them also bind ligands using a four-helix-bundle ligand-binding domain (4HB-LBD; PcAer2 and PHAer2), and the third receptor senses redox potential via an FAD-binding PAS domain (PfAer2). Thus, heme- and FAD-binding in PfAer2 ostensibly integrate direct and indirect O₂ sensing within the same protein. We suggest that PfAer2 is an unusual hybrid between a classic FAD-binding Aer receptor and a heme-binding Aer2 receptor. In this study, we provide initial characterization of the various sensing domains and attempt to identify native ligands. We focused on 1) the oligomeric state of the 4HB-LBDs (which must be dimeric for ligand-binding), and the feasibility of using thermal shift assays to identify ligands, 2) the solubility of purified PAS-heme domains and their heme and gas binding properties, and 3) the

ability of chimeric chemoreceptors containing the N-terminal “Aer-like” region to behave like Aer, rather than like Aer2. Overall, these studies are vital early steps before testing synergistic signaling models and clarifying the reasons for dual decision-making in the environments of these organisms.

RICHARD YE
SURF PARTICIPANT 2025

I am an upcoming fourth year student from the University of California Riverside studying biology. After graduating, I will continue my work in research, advancing the discoveries we have already made in the field of oncology and cancer sciences.

Throughout the previous year, I have been a volunteer at Dr. Hurtz's laboratory. I am very grateful to be welcomed back as a SURF participant during the summer of 2025. My research this summer is based around B-cell specific nanoparticle delivery.

Thank you to Dr. Christian Hurtz and Abhinav Ayyadevara for mentoring me and providing me with the opportunities learn and grow.



DEVELOPMENT OF B CELL SPECIFIC RNA DELIVERY STRATEGY

Richard Ye, V.S.S . Abhinav Ayyadevara
Department of Basic Sciences, Loma Linda University, CA

Despite therapeutic advances, high-risk subtypes of B-cell acute lymphoblastic leukemia (B-ALL) - notably Philadelphia chromosome-like (Ph-like) and *KMT2A*-rearranged (*KMT2A*-R) leukemias - remain a major clinical challenge due to poor prognosis and limited treatment options. Recent studies by our group implicate BRG1 (gene name *SMARCA4*), the ATPase subunit of the SWI/SNF chromatin remodeling complex, as a novel, subtype-specific vulnerability in Ph-like B-ALL. While BRG1-inactivating mutations are rare, elevated BRG1 expression is significantly associated with inferior survival in Ph-like cases and appears essential for leukemic cell proliferation. Pharmacologic inhibition of BRG1 with agents such as FHD-286 leads to cell cycle arrest and prolonged survival in xenograft models, positioning BRG1 as a compelling therapeutic target.

To enable BRG1-targeted gene therapy, and to circumvent the general disadvantages of pharmacologic inhibitors such as off-target effects, limited bioavailability, systemic toxicity, drug resistance, and short circulation half-life, we are developing a **CD22-directed RNA delivery platform** using polyplexes prepared using **poly (β -amino ester)** (PBAE) and 25kDa Polyethyleneimine (PEI). These nanoparticles are engineered for high specificity, efficient endosomal escape, and low immunogenicity, and are surface-coated with anti-CD22 antibodies via Streptavidin to enable selective uptake by B-ALL cells. As a proof of concept, we successfully delivered siRNA via PEI targeting the model gene **DYRK1A**, which is essential for survival in *KMT2A*-rearranged ALL, to B-ALL cells via CD22-mediated endocytosis. DYRK1A expression was silenced to 33% of control levels, resulting in increased apoptosis, demonstrating the platform's functional delivery capability. Additionally, we have made PBAE and PEI-based short-interfering RNA (siRNA) or guide RNA (gRNA) polyplexes with a consistent diametrical size around 100nm or 150nm, respectively, both with a net neutral surface charge.

These preliminary findings establish a foundation for future therapeutic knockdown or knockout of *SMARCA4* in Ph-like B-ALL. Our targeted RNA delivery approach offers a promising, translation silencing strategy for inhibiting BRG1 function and suppressing the proliferation of leukemic cells. By combining subtype-specific biological insights with rationally engineered

delivery vehicles, this work advances a novel treatment paradigm for improving outcomes in high-risk B-ALL.

Macpherson Society Scholars

SAMUEL CHAN
MACPHERSON PARTICIPANT 2025

My name is Samuel Chan, and I am a second year medical student at Loma Linda University. This summer, I am doing research in Dr. Salma Khan's lab where—together with Alena McQuarter and Romi Yamauchi—I am investigating the expression of HYOU1, PPFIA2, and PDK1 in anaplastic thyroid cancer. Before medical school, I earned a bachelor's degree in Neuroscience from Claremont Mckenna College in Los Angeles. During my time in college, I participated in ALS stem cell research, led a Bible study small group in my Christian club, and served a various underserved communities—ranging from tutoring low-income background youth to working as a camp counselor for individuals with Down Syndrome. In my free time, I enjoy building Legos, trying new foods, and spending time with my partner and family. I would like to extend a special thank you to Dr. Salma Khan and all the members of her lab for their mentorship, guidance, and support throughout this research experience.



**TRANSCRIPTOMIC ANALYSIS REVEALS RACIAL DIFFERENCES IN SEVERAL GENE
EXPRESSION IN PAPILLARY THYROID CARCINOMA**

Samuel Chan, Romi Yamauchi, Luiza Barseghyan, Alfred Simental, Andrea Shield, Salma Khan

School of Medicine, Loma Linda University, Loma Linda, CA

Papillary thyroid carcinoma (PTC) is the most common form of thyroid cancer, characterized by a generally favorable prognosis yet significant molecular heterogeneity. Notably, PTC exhibits significant racial and ethnic disparities in incidence, recurrence, and survival outcomes. In our recent study, *Unraveling Racial Disparities in Papillary Thyroid Cancer* (Barseghyan et al., Curr Oncol, 2025), we identified differential expression of key genes—including HYOU1, PPFIA2, and PDK1—across White, Hispanic, Black, and Asian patient samples using bulk RNA-sequencing and CARIS molecular profiling. HYOU1 encodes an endoplasmic reticulum chaperone protein that stabilizes mRNA of glycolytic enzymes, promoting tumor proliferation, invasion and aerobic glycolysis in PTC cells. PPFIA2 encodes scaffold proteins involved in cell adhesion and migration, and have been implicated to contribute towards PTC invasion and metastasis. PDK1 encodes a mitochondrial enzyme that inactivates pyruvate dehydrogenase complex, serving as a central metabolic regulator in aggressive PTC. To validate these findings and assess their potential as biomarkers or therapeutic targets, we analyzed gene expression patterns of HYOU1, PPFIA2, and PDK1 in PTC samples from four racial groups using quantitative PCR (qPCR) and TCGA datasets on UALCAN software (full form). qPCR confirmed differential expression of the three genes across White, Hispanic, Black, and Asian samples, consistent with the results from RNA sequencing and CARIS profiling. TCGA analysis via UALCAN showed significant downregulation of all three genes in tumors from White, Black, and Asian patients. Moreover, elevated expression of any of the three genes was associated with lower overall survival in patients with thyroid carcinoma. Together, these

findings highlight HYOU1, PPFIA2, and PDK1 as promising molecular markers in PTC, warranting further investigation into their functional roles and potential utility in diagnostic and prognostic applications.

AAREN HAREWOOD
MACPHERSON PARTICIPANT 2025

This summer, I worked under the mentorship of Dr. Sean Wilson to evaluate the Ride 4 focus (R4F) lifestyle medicine program, a cycling program designed to promote mental well-being among middle school students. My research focus centered on examining screen time and its correlation with students' Nature Scores, a measure of their connectedness to natural environments. This work has helped me appreciate how modern habits can influence emotional health in subtle, measurable ways.

I am a second-year medical student (Class of 2028) at Loma Linda University School of Medicine, following the completion of my B.S. in Biomedical Sciences at Oakwood University in Huntsville, Alabama. During my undergraduate years, I was honored with awards including Biology Senior and Junior of the Year and participated in research projects involving both molecular medicine and quantum materials, including a summer as a UTP student in the CHDMM program. These experiences have shaped my long-term goal of becoming a physician-scientist focused on integrative approaches to health and healing. Beyond academics, I enjoy calisthenics, piano, and exploring strategy-based games, outlets that challenge me to stay disciplined, curious, and creative. I've also had the privilege of participating in service-oriented initiatives such as tutoring, student leadership, and international mission work, each reinforcing my value of compassion for others.

I am grateful to Dr. Sean Wilson and the CHDMM team for their support, mentorship, and for fostering an environment where research and purpose meet meaningfully.



EFFECTS OF A SCHOOL-BASED CYCLING PROGRAM ON ADOLESCENT MENTAL HEALTH: INTERACTIONS WITH SCREEN TIME AND NATURE EXPOSURE

Aaren Harewood, Isaac Joo, Enlai Xu, Julia Ivana, Lauren Schunk, Esther Walker, Sean M. Wilson

Center for Perinatal Biology, Loma Linda University, Loma Linda, CA, Outride, Morgan Hill CA

Adolescent mental health has declined significantly, with rising rates of psychosocial distress. This study evaluates the impact of the Riding for Focus (R4F) middle school cycling program, a 6-8 week curriculum designed to promote physical and emotional wellness on adolescent mental health. We examined how environmental exposure (Nature Score) and adherence to CDC screen time recommendations (≤ 2 hours/day) modified program outcomes. Anonymous pre- and post-program surveys were collected from 17,180 students across 75 schools in North America. Psychosocial well-being was assessed using the World Health Organization-Five Well-Being Index (WHO-5) and the Pediatric Symptom Checklist (PSC-17). WHO-5 scores range from 0–100, with higher scores indicating better well-being; PSC-17 scores ≥ 15 suggest greater risk for psychosocial issues. Our findings demonstrate that students experienced improved well-being following R4F participation, regardless of screen time habits. However, students who met screen time recommendations consistently showed higher WHO-5 and lower PSC-17 scores both before and after the program, while access to nature did not systematically moderate outcomes. These

results suggest that school-based cycling programs like R4F are beneficial to adolescent mental health while access to nature may not enhance these outcomes.

MADELINE KIM
MACPHERSON PARTICIPANT 2025

I am currently a second-year medical student at Loma Linda University School of Medicine and a research trainee in Dr. Salma Khan's laboratory. The research I'm involved in focuses on investigating potential therapeutic strategies for late-stage high-grade serous ovarian carcinoma.

Prior to medical school, I earned a Bachelor of Science in Biology from Southern Adventist University in Tennessee. I also gained clinical experience as a medical transcriber at the Loma Linda University Eye Institute, where I worked for a year before beginning my medical training.

Outside of academics, I enjoy playing golf, spending time with family and friends, and cooking. My experience in the lab has deepened my appreciation for the scientific process and its role in advancing patient care. I am sincerely grateful to Dr. Khan and her research team for the opportunity to contribute to this important work in ovarian cancer therapeutics.



**INVESTIGATING KEY IMMUNE AND SIGNALING PROTEIN INTERACTIONS IN
OVARIAN CANCER USING IMMUNOPRECIPITATION**

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Ovarian cancer, particularly high-grade serous ovarian carcinoma (HGSOC), remains one of the deadliest gynecological malignancies due to challenges in early detection and the immunosuppressive tumor microenvironment. The vitamin D receptor (VDR), a nuclear hormone receptor regulating cell growth, apoptosis, and immune responses, has been identified as a potential tumor suppressor in HGSOC. VDR expression is often reduced in advanced tumors, and its activation has been shown to inhibit proliferation, induce apoptosis, and limit metastasis. VDR also interacts with critical oncogenic and immune-regulatory pathways. MDM2, a negative regulator of p53, can inhibit VDR function; TGF- β may act cooperatively with VDR in early-stage tumors; and p38 MAPK signaling, which upregulates PD-L1, may intersect with VDR pathways. This study examines protein-protein interactions between VDR and MDM2, TGF- β , p38 MAPK, and PD-L1 using immunoprecipitation and Western blotting in 11 HGSOC tissue samples. Results showed VDR-TGF- β interactions in 83.2% of samples (low to moderate intensity), VDR-MDM2 with high binding in 66.7%, VDR-p38 MAPK with low to moderate binding in all samples, and VDR-PD-L1 with high binding in 50% of cases. These findings support VDR's role as a central regulator of tumor-immune interactions and highlight its potential as a prognostic biomarker and therapeutic target. Ongoing studies aim to explore VDR-targeted strategies, including the use of vitamin D analogs in combination with immunotherapy, to overcome treatment resistance and improve outcomes in ovarian cancer.

BRIAN NGUYEN
MACPHERSON PARTICIPANT 2025

I attended La Sierra University, where I completed my bachelor's degree in biomedical sciences. During my undergraduate years, I was part of a virology research lab, conducting molecular biology research and analysis on bacteriophages. I have also had the opportunity to volunteer for local homeless organizations such as Operation: Safehouse, as well as participate in medical mission trips in Guatemala. These experiences helped cultivate my interest in medical science and enhanced my medical knowledge and understanding, which has proven beneficial as I am now entering my second year at Loma Linda University School of Medicine. Other interests and hobbies of mine include playing badminton and spending time with my family.



I am currently doing research with Dr. Salma Khan on genetic analysis of papillary thyroid cancer amongst different ethnic groups. For me, the most interesting part of participating in research is the chance to discover new information about the world around us, revealing more pieces of the puzzle of life. I also enjoy the collaborative aspect of research, being able to work with others to consolidate our findings. I am grateful to Dr. Khan and her lab for teaching and guiding me through my research project, giving me the opportunity to learn and grow as a physician scientist.

ETHNIC VARIATION IN THYROID CANCER MUTATIONS REVEALED BY NEXT-GENERATION SEQUENCING

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Thyroid cancer is the most common endocrine malignancy in the United States, with papillary thyroid carcinoma (PTC) being the predominant subtype. The rising incidence of PTC is attributed to improved diagnostic tools and changing environmental and lifestyle factors. Prior studies have noted significant racial and ethnic disparities in PTC incidence, presentation, and survival. For example, Asians show the highest incidence and recurrence, while Black and Hispanic populations experience the lowest survival rates, suggesting contributions from genetic, environmental, and healthcare-related factors in the course of thyroid malignancy. Using next-generation sequencing data from cBioPortal, TCGA, and the LLU cohort, we identified distinct gene mutation patterns across four ethnic groups. BRAF mutations were the most common overall, with the highest frequency in Hispanics. Caucasians showed the widest variety of gene mutations. African Americans and Hispanics had higher TERT mutation rates and alterations in genes regulating proliferation and survival. Asians more frequently harbored mutations in genes associated with DNA repair and cell cycle arrest, such as ATM, BRCA1, and CHEK2. TERT mutations had the strongest negative impact on survival across all groups. TCGA analysis confirmed that gene expression differences were more pronounced between mutated and non-

mutated genes than among ethnicities, though higher expression levels in Caucasians and lower levels in African Americans were generally associated with poorer outcomes. This study highlights the genetic underpinnings of racial and ethnic disparities in papillary thyroid cancer, emphasizing the importance of molecular profiling to develop not only a deeper understanding of the molecular mechanisms of such a common malignancy, but also advance personalized therapies and improve survival outcomes across diverse populations.



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