LOMA LINDA UNIVERSITY

School of Medicine

Center for Health Disparities and Molecular Medicine

25th Annual Health Disparities Research Symposium

Education – Development – Health Disparities Research – Community

PROGRAM, BIOS, AND ABSTRACTS

Wednesday, July 29, 2026
12:00 pm – 7:30 pm
Centennial Complex 4th Floor 4220
Loma Linda University School of Medicine
Loma Linda, California

LOMA LINDA UNIVERSITY
School of Medicine
Center for Health Disparities and Molecular Medicine

25th Annual Health Disparities Research Symposium
Wednesday, July 29, 2026
12:00 pm - 7:30 pm, Centennial Complex 4th Floor 4220

Agenda

Noon Session

12:00 pm – 1:30pm

Alumni Journeys

Catherine Elix, PhD
Biomedical Research Scientist at Certis Oncology Solutions, Inc.

Greisha Lee Ortíz-Hernández, PhD
Postdoctoral Fellow at City of Hope

Alfonso Duran, MD, PhD
Assistant Professor, Department of Pathology and Anatomy
Loma Linda University School of Medicine

Poster Session

2:00 pm – 4:00 pm **Poster Presentations by Research Fellows**

Apprenticeship Bridge to College Program (ABC)
Undergraduate Training Program (UTP)
Medical Training Program (MTP)
Summer Undergraduate Research Fellowship (SURF)
Student Undergraduate Research Generating Evidence (SURGE)
CHDMM Graduate Students
Macpherson Society Scholars
Lab Assistants and Volunteers

4:00 pm – 4:30 pm **Dinner**

4:30 pm – 5:00pm **Research Snapshots**
Johnny D. Figueroa, PhD
Associate Director, CHDMM

Evening Program

5:00 pm – 7:30 pm

Welcome
Eileen J. Brantley, PhD
Associate Professor, Basic Sciences

Invocation
Frankis Almaguel, MD, PhD
Director, Molecular Imaging & Therapeutics Research Program
Loma Linda University Cancer Center

Remarks

Tamara L. Thomas, MD
Dean, School of Medicine

Anthony Hilliard, MD, FACC
President, Loma Linda University

Paul Herrmann, MD, PhD
Provost, Loma Linda University

Remarks about the Program and Center
Carlos A. Casiano, PhD
Director, CHDMM

Special Recognitions
Johnny D. Figueroa, PhD
Associate Director, CHDMM

Introduction to Keynote Speaker
Carlos A. Casiano, PhD
Director, CHDMM

Keynote Speaker
"*Centering Community in Cancer Disparities Research.*"
Carmen Guerra, MD, MSCE, FACP
Ruth C. and Raymond G. Perelman Professor of Medicine
University of Pennsylvania

Recognition of Research Fellows

Johnny D. Figueroa, PhD
ABC and UTP Trainees

Daisy D. De León, PhD
MTP Trainees

Kylie J. Watts, PhD
SURF Trainees

Lisa Roberts, MD
SURGE Trainees

Salma Khan, PhD
CHDMM Graduate Students

Susanne B. Montgomery, PhD, MPH, MS
Macpherson, Lab Assistants and Volunteers

Final Remarks and Acknowledgements

Carlos A. Casiano, PhD
Director, CHDMM

ACKNOWLEDGEMENTS

We would like to acknowledge the contributions of all who were instrumental in making this 2026 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).

2026 Faculty Research Mentors

Arlin Blood, PhD
Alfonso Duran, MD, PhD
Carlos A. Casiano, PhD
Christian Hurtz, PhD
Christopher Perry, PhD
Christopher Wilson, PhD
Danilo Boskovic, PhD
Eileen Brantley, PhD
Daisy De León, PhD
Johnny Figueroa, PhD
Julia Unternaehrer, PhD
Jiang Zhong, PhD
Lisa Roberts, DrPH, MSN
Rameshwar Patil, PhD
Salma Khan, PhD
Seth Wiafe, PhD
Sean Wilson, PhD
Susanne Montgomery, PhD, MPH, MS
Kylie Watts, PhD
William Langridge, PhD
Ying Nie, MD, PhD

CHDMM Administrative Staff

Amy Barajas, Senior Administrative Assistant
Lorena Salto, Center Manager
Lynn Lingas, Program Manager

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.

2026 Student Research Fellows

ABC – Apprenticeship Bridge to College
Alana Dominguez-Johnson
Alexandra Michkov
Ayobola Ibitoye
Carlos Silveyra
Caroline Coronado
Catherine Li
Langyue (Diana) Hu
Linda Martinez
Miley Miranda

UTP – Undergraduate Training Program
Adetayo Adeogun
Andrea Latorre Marrero
Brigitte Fonseca Marroquin
Cameron Scott
Darine Abu Hilal
James Mitchell
Jasmyn Vanterpool
Javan Charles
Mia Pierce
Shania McKelvey
Zachary Chang

MTP – Medical Training Program
Allison Rhee
Jocelyn Chow
Leanne Mercado Then
Melissa Lee

CHDMM Graduate Students
Adelaide Makamure
Alena McQuarter
Julio Sierra
Kathleen Escobar Acuña
Krystal Santiago
Nathaly Manrique

SURGE – Student Undergraduate Research Generating Evidence
Abel Massey
Ashra Tugung
Crystal Lee
Emma Karpov

SURF – Summer Undergraduate Research Fellowship
Aiden Riley
Fatimah Ahmed
Gabrielle Kim
Ian Forncrook
Jorge Hiroshi Boyas Homma
Melanie Morales
Sarah Lawrence
Sydney Hanson

Macpherson Society Scholars
Anella Fischer
Emily Rojas
Harel Sims
Matthew Rittenbach

Lab Assistant and Volunteers
Alyssa Sorrell
Dan Celestin
Delaney Werner
Fariba Mamun
Jacqueline Monteza
James Kuo
Katherine Granados
Linh Nguyen
Samantha Najarro
Terence Killebrew
Timothy Kim
Xaria Clark

Institutional Affiliations of Student Research Fellows

High Schools

Canyon Springs High School
Eisenhower High School
Eleanor Roosevelt High School
Linfield Christian School
Loma Linda Academy
Patriot High School
Riverside Poly High School
Sigma Preparatory Academy
Western Center Academy

Universities

Azusa Pacific University
Baylor University
Biola University
California Baptist University
Loma Linda University School of Medicine
Loma Linda University School of Public Health
Loma Linda University School of Nursing
Oakwood University
Pacific Union College
Southern Adventist University
Stony Brook University
Texas Tech University
UC Los Angeles
Universidad Adventista de las Antillas
Universidad Central del Caribe
University of California Berkeley
University of California Davis
University of California Los Angeles
University of California Riverside
University of Redlands
Walla Walla University
Western University of Health Sciences

LOMA LINDA UNIVERSITY

School of Medicine

Center for Health Disparities & Molecular Medicine

LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE

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

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Keynote Speaker

CARMEN E. GUERRA, M.D., M.S.C.E., F.A.C.P.
RUTH C. AND RAYMOND G. PERELMAN PROFESSOR OF MEDICINE
UNIVERSITY OF PENNSYLVANIA

Dr. Carmen Guerra is the Ruth C. and Raymond G. Perelman Professor of Medicine, and Professor of Biostatistics and Epidemiology at the Perelman School of Medicine at the University of Pennsylvania. She is the Associate Director of Community Outreach and Engagement for the Abramson Cancer Center.

Dr. Guerra's research is focused on developing and evaluating interventions to increase the participation of populations in cancer screening and cancer clinical trials. Dr. Guerra developed and oversees the Penn Medicine Colorectal Cancer Screening Navigation Program and the Breast and Cervical Cancer Early Detection Program which have screened thousands of individuals and provided patient navigation to diagnosis and treatment. Dr. Guerra is also the site PI of the NCI-funded Self-collection for HPV Testing to Improve Cervical Cancer Prevention (SHIP) Trial, a platform trial which compares the accuracy of self- and clinician-collected samples for HPV of multiple HPV collection kits and assays. The results of the SHIP trial are being submitted to the FDA and led to the approval of the first at home HPV test for cervical cancer screening. Dr. Guerra also is member of the American Cancer Society (ACS) Clinical Guidelines Development group and an author of the ACS colorectal, cervical, and lung clinical practice guidelines and the ACS guidance to clinicians for Multi-cancer Early Detection Tests, all which widely influence clinical practice.

Apprenticeship Bridge to College (ABC)
High School Program

ALANA DOMINGUEZ-JOHNSON
ABC PARTICIPANT 2026

I am a sophomore at Riverside Polytechnic High School in Riverside, California and I plan to major in Molecular Neuroscience before attending medical school and pursuing a career as a pediatric neurosurgeon. My passion for neuroscience and medicine stems from a desire to better understand the human brain and contribute to improving the lives of children affected by neurological disorders. I have earned 2nd Place at both the district and regional science fairs in Environmental Engineering and 1st Place in both the STEM LEAPS and STEM PULL programs. I am also dedicated to serving my community through my church, where I sing in the choir, assist with catechism classes, and volunteer with different Social Concerns ministries.

This summer, I am conducting research in Dr. Christopher Wilson's Perinatal Biology Lab under the mentorship of Dr. Christopher Wilson, Joycelyn Williams, and Nick Iwakoshi. My research focuses on characterizing how maternal inflammation affects neurodevelopment and the resulting gross physiological changes. The most fascinating aspect of this work has been learning immunohistochemistry and immunofluorescent staining techniques to visualize and identify proteins within brain tissue.

Through the ABC Program, I have gained experience in protein identification, identifying antibody-antigen relationships, different technical skills including cryosectioning, and adapting to laboratory protocols in a fast-paced research environment. I enjoy hiking and exploring state parks, playing the violin, singing, and watching films. I am grateful to my mentors for their patience and guidance towards my growth as both a researcher and future healthcare professional.

MATERNAL MTDNA-INDUCED INFLAMMATION IS ASSOCIATED WITH INCREASED GLIAL ACTIVATION AND ALTERED HIPPOCAMPAL NEURONAL MORPHOLOGY IN NEONATAL RATS

Alana D. Dominguez-Johnson, Joycelyn H. Williams, Nicholas Iwakoshi, Christopher G. Wilson

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

Maternal inflammation has been linked to disruptions in central nervous system development, particularly hippocampal neurodevelopment, by promoting pro-inflammatory upregulation and modification of the neural circuitry of the hippocampus. The hippocampus is necessary for learning and memory and can be subject to disruption in early life through inflammatory or hypoxic events. These pathophysiological conditions can alter neuronal morphology in the fetus and cause disruptions during early postnatal brain development. Other investigators have shown that mtDNA-induced maternal inflammation alters neuron morphology and likely contribute to abnormal brain development in offspring resulting in cortical impairment. The objective of this study was to investigate how maternal inflammation induced by mitochondrial DNA (mtDNA) affects hippocampal morphology in neonatal rat pups. We hypothesized that maternal inflammation would increase activation of microglia, resulting in elevated ionized calcium-binding adapter molecule 1(IBA1) accompanied by alterations in neuronal morphology. We analyzed brain tissue from experimental pups born to dams injected with mtDNA and control pups born to untreated dams. We used immunofluorescence staining for neurofilament markers, DAPI, and IBA1. An abundance of IBA1 positive microglia indicates a pro-inflammatory state suggesting that microglia are releasing cytokines and chemokines which have an impact on neuronal function and hippocampal organization during development. Therefore, we expect the group affected by maternal inflammation to exhibit greater glial activation and abnormal neuronal architecture compared with the control pups. These findings will expand our understanding of how maternal inflammation influences early brain development in neonates and allow us to target cellular mechanisms that may contribute to later neurodevelopmental disorders.

ALEXANDRA MICHKOV
ABC PARTICIPANT 2026

I am a rising senior at Linfield Christian High School in Temecula, planning to major in regenerative and developmental medicine as I work toward an MD/PhD. Ultimately, my goal is to lead a research center focused on disease prevention. My interests include chess, reading widely, writing fiction, and collecting pins.

My high school involvement includes serving as a certified food handler and scholarship volunteer leader for the 2025 Lunch at the Library program, which provided over 1,000 meals to children; being the 2025–2026 youth fellow for the Cortico Teen Dialogue Accelerator in partnership with Massachusetts Institute of Technology (MIT) CCC and PBS Frontline; and serving as the 2026–2027 Science National Honor Society president and Mu Alpha Theta vice president.

Under the mentorship of Dr. Arlin Blood, I worked alongside Desirelys Ortiz Martinez and Daylan Ward on research examining the role of the vagus nerve, central to the parasympathetic nervous system, contributes to the development of the fetal lung. Through this experience, I gained skills in cell counting, antibody-targeted staining of lung tissue slices, and data analysis. The experience strengthened my curiosity, adaptability, and initiative, and provided me with a better understanding of birth-transition physiology. I am deeply grateful to Dr. Blood for fostering an open, supportive environment for learning and discovery.

THE FUNCTION OF THE VAGUS NERVE IN THE DEVELOPMENT OF FETAL LUNGS

Alexandra Michkov[1], Daylan Ward[2], Desirelys Ortiz-Martinez[2], MS, Karina Mayagoitia[2], PhD, Arlin Blood[2], PhD

[1]Center for Health Disparities and Molecular Medicine, [2]Lawrence D. Longo, MD Center for Perinatal Biology, School of Medicine, Loma Linda University, CA

Newborn survival, especially in premature infants, relies on the development of the connection between the central nervous system (CNS) and the lungs, which includes the vagus nerve. The vagus is central to the parasympathetic system and innervates the fetal lung from the first trimester onward. In adults, the parasympathetic system exerts anti-inflammatory effects. However, in the fetus, the function of these nerves is relatively unstudied. It is possible that the parasympathetic nerves in the fetal lung release growth factors that direct lung development. We hypothesized that the removal of the vagus nerve in the 2nd trimester would hinder lung development and promote dysregulation of the parasympathetic system. At 110 days of gestation (term = 147 days), preterm lambs were randomly selected to either have sham surgeries or bilateral vagotomies. After their surgeries, the lambs were returned to the uterus and continued their gestation until 135 days, when they had a C-section delivery. At birth, both the sham group and the vagotomized group were mechanically ventilated, and arterial PO_2, FiO_2, and mean airway pressure were measured and used to calculate the oxygen index (OI), a clinically useful index of lung function. After 90 minutes of ventilation, including a hypoxia challenge, the lambs were euthanized, the lungs removed, and histology slides prepared. Macrophages (M0) were counted relative to total cell number. The results demonstrated no significant differences between the groups in PO_2, FiO_2, mean airway pressure, or M0 abundance. However, OI was significantly elevated in the vagotomized lambs compared to the sham lambs. Thus, the vagus nerve contributes to post-birth oxygenation, which expands its role in fetal lung development research.

AYOBOLA IBITOYE
ABC PARTICIPANT 2026

Before joining the ABC Program this summer, I knew I was interested in biology and microbiology, but I had never experienced what research was really like. Working in a laboratory has shown me how much time, patience, and teamwork go into every experiment. It has also shown me how research can improve people's lives.

I am a student at Eisenhower High School and a member of the Class of 2028. After high school, I plan to study biology and attend medical school to become a physician. My interest in medicine comes from seeing the health challenges that exist in my community. Those experiences inspired me to pursue a career where I can care for patients and help improve access to quality healthcare.

This summer, I am working in the microbiology laboratory of Eileen J. Brantley, PhD. My research focuses on breast cancer and examines whether a specific protein may improve survival outcomes for people of African ancestry. One of my favorite parts of the program has been learning hands-on lab skills, like treating and harvesting cells, while seeing how each experiment contributes to a larger research question.

I am grateful to Dr. Brantley and the ABC Program for their mentorship and support throughout the summer. Outside of school, I enjoy staying active, spending time with my family, and watching movies together. This experience has strengthened my interest in medicine and confirmed that this is the path I want to pursue.

RUTIN SUPPRESSES PROLIFERATION AND STEMNESS IN TRIPLE-NEGATIVE BREAST CANCER CELLS FROM PATIENTS OF WEST AFRICAN ANCESTRY

Ayobola Ibitoye, Elyssa Vazquez, John Khalaf, Jonathan De Anda, Michael Hall, and Eileen Brantley

Department of Basic Sciences, Division of Pharmacology, Center for Health Disparities and Molecular Medicine, Loma Linda University, Loma Linda, CA

Triple-negative breast cancer (TNBC) lacks expression of the estrogen receptor, progesterone receptor, and human epidermal growth factor receptor-2 and thus has few targeted treatment options. TNBC is the most aggressive breast cancer subtype and is diagnosed more frequently among patients of West African ancestry than patients of other ancestries. This increased incidence of TNBC contributes to the survival disparity observed among patients of West African ancestry. We investigated whether rutin, a naturally occurring flavonoid with reported anticancer properties, suppresses proliferation and stemness in TNBC cells from patients of West African ancestry. We performed colony-forming and mammosphere-forming assays to determine the impact of rutin on TNBC cell proliferation and stemness, respectively. Using quantitative PCR analysis, we assessed changes in alpha6-integrin (ITGA6), DNA methyltransferase 1 (DNMT1), vimentin (VIM), and epithelial cadherin (CDH1) mRNA expression in TNBC cells following rutin treatment. Our data show that rutin suppresses proliferation of CRL2335, MDA-MB-468, and MDA-MB-157 TNBC cells. Rutin also diminished mammosphere-forming capacity in these cells. In CRL2335 cells, rutin significantly decreased ITGA6 and VIM mRNA expression, and these genes are known to promote breast cancer cell stemness and epithelial-to-mesenchymal transition (EMT) respectively. In contrast, rutin displayed no appreciable impact on DNMT1 or CDH1 mRNA expression levels. Our data suggest that rutin suppresses TNBC cell proliferation and stemness, potentially via down-regulation of stemness markers such as ITGA6 and EMT markers such as VIM. RNA-sequencing analyses are underway to identify additional pathways affected by rutin and may inform future therapeutic strategies for TNBC to improve survival, particularly among patients of West African ancestry.

CARLOS SILVEYRA
ABC PARTICIPANT 2026

As an incoming senior at Patriot High School in Jurupa Valley, I have observed firsthand the implications of health disparities and the withheld healthcare aid in underrepresented populations. In my community, I have the privilege of serving as Secretary for the Youth Advisory Council for Riverside County, where I advocate for youth involvement to unify communities through service. I view education as a foundational right that enables individuals to maximize their intellectual potential and drive positive societal change. For Hispanic communities, higher education serves as a powerful engine for equity, connecting passionate advocates determined to provide resources to those who need them most.

These experiences, alongside a myriad of mentors, have propelled my future aspiration to become a family medicine physician. This summer, I have had the honor and opportunity to work in Dr. Sean Wilson's laboratory alongside Melanie Morales and PhD student Aditi Bhatnagar. Our project encompasses evaluating stem cell therapies for infant bronchopulmonary dysplasia and examining hyperoxia/normoxia-induced lung damage in mice. We utilized brightfield microscopy to image tissues stained with Hematoxylin and applied the FIJI software to analyze and quantify the resulting fibrosis.

This opportunity has shown me that the role of a scientist is not to achieve continuous success, but to confront the failures of the scientific world to achieve meaningful implications. I am incredibly grateful for the mentorship Dr. Wilson and Claudia Rodriguez have given me, not only to explore the world of science but also to understand who I am as a researcher/scientist.

SEMI-AUTOMATED DIGITAL MORPHOMETRY FOR UNBIASED ASSESSMENT OF HYPEROXIA-INDUCED BROCHOPULMONARY DYSPLASIA MOUSE MODEL

Carlos Silveyra, Melanie Morales, Ian Forncrook, Aditi Bhatnagar, Claudia Rodriguez Torres, Ciprian P Gheorghe, Sean M Wilson

Lawrence D. Longo Center for Perinatal Biology, Advanced Imaging and Microscopy Core (AIM), School of Medicine, Loma Linda University, Loma Linda, CA

Bronchopulmonary dysplasia remains a critical complication among preterm infants, characterized by disrupted pulmonary alveologenesis. In clinical settings, this morphological simplification is primarily driven by prolonged supplemental oxygen therapy, a necessary life-saving intervention that is recapitulated in our murine model via sustained hyperoxic exposure (95% O-2) relative to ambient room air controls (21% O2). To evaluate emerging therapeutic interventions using mesenchymal stem cell derivative, our preclinical research involves reproducible baseline injury models and objective quantification pipelines that eliminate detection bias.

Our study modeled hyperoxia-induced parenchymal disruption in three-week-old mouse lung tissue and validated an ImageJ-based computer-assisted digital framework for classifying structural degradation. Murine cohorts were assigned to normoxic (21% O2) and hyperoxic (95% O2) groups with five mice per group. Following exposure, lung tissues were harvested, sectioned, mounted, and stained with hematoxylin and eosin. Brightfield images were captured using an Evident APEXVIEW APX100 microscope. Randomly-selected lung tissue fields were formatted for downstream morphometric analysis using a semi-automated FIJI macro to examine septal point counts, mean linear intercept, and alveolar septal volume density. Ongoing morphometric analysis and preliminary comparisons show anticipated increase in mean linear intercept and decrease in alveolar septal volume density in hyperoxic groups compared to normoxic controls. These findings demonstrate that hyperoxic environments exacerbate alveolar simplification and arrest of secondary septae development, essential for gas exchange. By validating this automated framework, our study establishes a clinically-translational and highly reproducible pathological baseline to quantitatively characterize hyperoxia-induced structural degradation in future studies with mesenchymal stem cell therapies.

CAROLINE CORONADO
ABC PARTICIPANT 2026

I am a rising homeschooled senior on track to earn an associate degree in psychology next fall. Homeschooling has given me the flexibility to explore many different fields, but science has always been my favorite subject to study. I have been fortunate to serve in leadership roles as Vice President of my local homeschool co-op and as an intern at a nonprofit organization in Riverside, but the experiences I value most are the ones that allow me to teach and serve my community. These include teaching STEM classes to homeschool students and founding a nonprofit initiative that raised more than $1,500 for local health organizations. Outside of academics and service, I enjoy dancing, music, traveling, and playing cornhole.

This summer, I returned to Dr. Alfonso Durán's lab, where our research focuses on ferroptosis from a health disparities perspective. In addition to developing new laboratory skills, I have had the opportunity to contribute to the preparation of a research manuscript, an experience that has further strengthened my interest in translational research and the physician-scientist pathway.

In the future, I hope to pursue an MD/PhD and become a surgeon-scientist, combining patient care with research to improve health outcomes. I also hope to open my own medical practice, continue conducting impactful research, and establish a scholarship and educational program that expands opportunities for students without monetary restrictions.

I am incredibly grateful to Dr. Alfonso Durán, Dr. Francis Zamora, and Dr. Viet Hoang Dinh for their mentorship, encouragement, and investment in my growth as both a student and researcher.

FERROPTOTIC VULNERABILITY INDEX (FVI) AND ALLOSTATIC LOAD (AL) AS JOINT PREDICTORS OF 15-YEAR MORTALITY AMONG U.S. ADULTS: A POPULATION-BASED STUDY HIGHLIGHTING DISPARITIES BY POVERTY AND RACE

Caroline Coronado [1], Viet Hoang Dinh [1], Gary TA-Wei Yu [1], Alfonso Durán [1,2]

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Ferroptosis is a regulated type of cell death that depends on iron, dysregulated antioxidant pathways, resulting in lipid peroxidation of organelle and cellular membranes. Scientists hypothesize that ferroptotic stress may play a role in chronic disease, but this theory has not been tested to see whether markers of ferroptosis-prone biology predict death in a large population, or how they relate to the body's physiological cumulative "wear and tear" from chronic stress, known as allostatic load (AL).

To study this, we built a new six-part Ferroptotic Vulnerability Index (FVI) from blood biomarkers linked to ferroptotic stress and an established eight-part AL score. We applied both to 15,819 U.S. adults from a national health survey (NHANES, 1999–2004) linked to the National Death Index (NDI). In the 13,580 adults with complete data, 3,433 died during the 15-year follow-up window.

Using statistical models that account for age, sex, race/ethnicity, and income, we found that higher scores on both indices predicted a greater risk of mortality. Each one-point rise in AL was linked to about a 14% higher risk of death, and each one-point rise in FVI to about a 28% higher risk. The two scores measured largely different things, and each stayed predictive even after accounting for the other, without significant statistical correlation. Adults who scored high on both FVI and AL had a much higher death rate of 53.6%, compared to 11.3% among those low on both panels.

The FVI was an especially strong predictor of cancer death, a pattern the AL score did not capture, suggesting FVI, suggesting a potential ferroptosis-specific cancer-mortality signal. Death risk also varied by race/ethnicity and income, with the highest-risk patterns concentrated in disadvantaged groups. Together, these findings suggest that combining the two scores could help identify high-risk populations who might benefit from targeted screening.

CATHERINE LI

ABC PARTICIPANT 2026

I am a rising senior at Loma Linda Academy, planning to pursue a career in biomedical research. I have the opportunity to work in William H. R. Langridge's lab, where our research focuses on developing a plant-based vaccine. What fascinates me most is the idea that a living plant can be engineered one day to help combat one of today's significant health challenges: respiratory viruses.

Outside of research, I participate in a variety of volunteer activities. The most prominent and meaningful one has been serving at REGEN sabbath school, supporting high school students. In my free time, I enjoy playing basketball, reading, and hanging out with friends.

The ABC program has taught me many things, one of most important being progress is not linear. Experiments don't always turn out as expected, but every result provides valuable information that guides the next step. As Dr. Casiano said, most of your scientific research career is made up of failures and setbacks. I learned to see "failed" experiments as opportunities to improve and better understand the science behind it. This experience strengthened my understanding of molecular biology, critical thinking skills, and hands-on laboratory techniques, while teaching me that meaningful discovery requires curiosity, patience, meticulousness, and tenacity. This principle extends beyond scientific research and applies to many aspects of life.

I would like to thank Dr. Langridge for welcoming me into his lab and for patiently mentoring me throughout this summer program. His support and encouragement made this experience even more memorable and valuable.

EVALUATION OF CTB-SARS-CoV-2- RECEPTOR-BINDING DOMAIN (RBD) GENE AND PROTEIN EXPRESSION IN TRANSFORMED TOBACCO LEAVES

Catherine Li and William Langridge

Center for Health Disparities and Molecular Medicine, Department of Biochemistry, School of Medicine, Loma Linda University, Loma Linda, CA 92354

Plant genetic engineering has emerged as a promising strategy for developing next-generation mucosal vaccines. Plant-based vaccine advantages, include lower production costs, a reduced risk of contamination with human pathogens, and improved stability at ambient temperature. In addition, oral mucosal delivery has the potential to induce immune responses at the respiratory mucosa, the primary site of SARS-CoV-2 infection. This study tests the hypothesis that the cholera toxin B subunit (CTB)-fused SARS-CoV-2 receptor binding domain (RBD) gene can be successfully integrated into the tobacco plant genome, and that stable expression of the recombinant CTB-RBD vaccine protein can be detected in tobacco leaf tissues. Tobacco leaves from previously generated transgenic plants putatively containing the CTB-SARS-CoV-2 RBD gene were harvested, frozen in liquid nitrogen, and homogenized for total protein extraction. The plant proteins were separated by acrylamide gel electrophoresis. The recombinant CTB-RBD protein was further isolated by electroelution from gel strips corresponding to the known molecular weight of the vaccine protein. This was then followed by protein staining to estimate protein yield. A distinct protein band at the expected molecular weight (between 37–50 kDa) was detected, consistent with the predicted size of the recombinant CTB-RBD fusion protein. The vaccine protein was further identified by immunoblotting using antibodies specific for the CTB SARS-CoV-2-ACE-RBD protein. The vaccine protein was identified, followed by X-ray film detection of the corresponding immunoreactive bands. Immunoblotting was performed to confirm the identity of the vaccine protein. We predict these findings will demonstrate successful plant transformation and stable transgene expression of the CTB-RBD vaccine protein, supporting the feasibility of plant genetic engineering as an effective alternative platform for producing the recombinant mucosal vaccine. Future studies will focus on increasing vaccine protein production, evaluating vaccine immunogenicity, and vaccine protective efficacy in cell culture and in animal models prior to the initiation of clinical trials.

LANGYUE (DIANA) HU
ABC PARTICIPANT 2026

As an incoming senior at Eleanor Roosevelt High School in Eastvale, my passion for healthcare is driven by service and scientific inquiry. On campus, I serve on the Pink Dot Club board to raise awareness for organ donation and perform with the Colorguard program. Beyond school, I volunteer at San Antonio Regional Hospital, represent youth interests on the Eastvale City Youth Council, and help manage my mother's home baking business. My background in the culinary arts translates directly into the lab, where precise procedures mirror the careful measuring, stirring, and heating of a recipe.

This past summer at Loma Linda University, I researched gold nanoparticles (GNPs) as radiosensitizers in cancer radiation therapy. Furthermore, attending insightful seminars from diverse researchers provided valuable real-world perspectives. Hearing their expertise solidified my desire to pursue this future path in medicine and research. Through the program, I gained proficiency in culturing glioblastoma cells and utilizing advanced equipment like UV-Vis and Dynamic Light Scattering. In particular, I successfully increased GNP uptake in glioblastoma cells by attaching the Angiopep-2 peptide, ultimately enhancing radiation efficacy. These foundational skills will be crucial as I pursue a healthcare career, continue research in university, and work to care and solve community health disparities.

I am grateful to Dr. Patil and the Patil Lab for providing this exceptional laboratory experience, and to my mentors, Sanjay Yadav and Cedric Lansangan, for their meticulous guidance and support. Finally, I extend my sincere gratitude to the CDHMM team for making this an unforgettable summer.

BLOOD BRAIN BARRIER PENETRATING GOLD NANOPARTICLES FOR RADIOSENSITIZATION OF MALIGNANT GLIOMA

Langyue Hu[1], Sanjay Yadav[1], Cedric Lansangan[1], Kevin Nick[2], Rameshwar Patil[1*]

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[2]Department of Earth and Biological Sciences, School of Medicine, Loma Linda University, Loma Linda, CA

Glioblastoma (GBM) remains the most common and lethal malignant brain cancer originating from astrocytes. It is characterized by rapid cellular invasion and a poor prognosis, making the development of advanced therapeutic strategies critical. Radiation therapy plays a critical role in management of GBM and has been part of the standard of care. This study aimed to improve the therapeutic efficacy of radiation therapy, by utilizing gold nanoparticles (GNPs) as radiosensitizers to amplify targeted radiation-induced damage within tumor cells. Our novel, targeted GNPs were synthesized using a co-reduction method of tannic acid and sodium citrate, followed by characterization via UV-Vis spectroscopy and Dynamic Light Scattering (DLS) to confirm size uniformity. The GNPs were subsequently surface coated with polyethylene glycol for stabilization and functionalized with a blood-brain barrier (BBB) penetrating peptide Angiopep-2 (Ap2). Ap2 peptide acts as a substrate for low-density lipoprotein receptor-related protein 1 (LRP-1), a receptor highly expressed in tumor capillary cells, allowing the particles to successfully cross the restrictive BBB. Successful surface modification and peptide conjugation were verified via agarose gel electrophoresis, which demonstrated distinct sample migration shifts indicative of altered molecular weight and charge. Colloidal stability of GNP was studied using 2 MBT method. Future studies will focus on nanoparticle uptake in GBM cell lines. Amount of gold will be measured using MP-AES (Microwave Plasma - Atomic Emission Spectroscopy) to quantitatively determine the GNP uptake in GBM cell lines. We successfully achieved a synthesis and functionalization of GNPs. The particles were characterized by state-of-the-art analytical methods and wet chemical assays developed in our lab. This biocompatible, targeted platform offers a highly promising strategy to bypass the BBB, maximize localized delivery for enhanced radiation intensity, and ultimately improve therapeutic outcomes for glioblastoma patients.

LINDA MARTINEZ
ABC PARTICIPANT 2026

As a 2026 ABC participant, my understanding that healing is both a scientific and spiritual calling has only deepened through this transformative experience. Under the mentorship of Dr. Seth A. Wiafe, I have been working on the project titled Mapping the Gap: Pediatric Healthcare Access Across the Inland Empire. This work has illuminated the powerful intersection of data, geography, and compassion, revealing the pressing needs of vulnerable pediatric populations across our region.

As this summer progresses, I continue to develop specialized skills in geospatial research using ArcGIS Pro and ArcGIS Online. Through the integration of spatial and non-spatial data at the census tract level, I am learning how to assemble and manipulate complex datasets that define and highlight disparities in healthcare access. This technical training has been accompanied by a profound realization: protecting children does not begin in an operating room or research lab, but in the preventive efforts that address the social and environmental conditions placing them at risk.

One of the biggest blessings of this program has been the mentorship of Dr. Wiafe. His wisdom, integrity, and faith have guided my growth not only as a student and researcher, but as a person seeking to serve with purpose. I am deeply grateful for his investment in my development and for the example he sets of leadership grounded in both excellence and compassion.

I truly believe God places certain people with intention to fulfill His plans. I sincerely hope that I may be used as His instrument to continue serving children and families with love, humility, and unwavering faith.

MAPPING PEDIATRIC HEALTHCARE ACCESS AND GEOGRAPHIC DISPARITIES ACROSS CALIFORNIA'S INLAND EMPIRE

Linda Martinez, Seth A. Wiafe

Center for Health Disparities and Molecular Medicine, Health Geoinformatics Lab, School of Public Health, Loma Linda University, Loma Linda, CA

Geographic access to pediatric healthcare is a critical determinant of child health outcomes, yet substantial disparities in pediatric specialty care persist across California's Inland Empire. San Bernardino and Riverside counties span more than 27,000 square miles and serve a rapidly growing pediatric population. Reliance on a single dedicated children's hospital and a limited network of specialty facilities may create barriers to timely care. This study used Geographic Information Systems (GIS) to identify communities with the greatest unmet pediatric healthcare needs and examine the relationship between healthcare accessibility and population vulnerability.

Using ArcGIS Pro 3.7, we integrated census tract-level American Community Survey data—including child population, poverty, insurance coverage, and racial/ethnic composition—with geocoded locations of children's hospitals and hospitals offering pediatric specialty services. Analyses included choropleth mapping, buffer analysis, Getis-Ord Gi* hotspot analysis, spatial overlays, and a weighted composite index to identify priority areas with limited access.

Estimated travel distances to the nearest pediatric specialty facility ranged from 0.2 to 171 miles (mean = 16; SD = 22), demonstrating marked geographic variation. Statistically significant clusters of underserved census tracts were identified in southwestern San Bernardino County and northwestern Riverside County, including Rancho Cucamonga, Moreno Valley, Perris, and Indio. These findings suggest that geographic proximity alone does not ensure adequate access; disparities also reflect travel burden, socioeconomic disadvantage, insurance coverage, and population concentration.

Priority maps identified pediatric care deserts where expansion of specialty services, workforce placement, and transportation planning may improve access. Integrating geospatial accessibility with social vulnerability measures provides a decision-support framework for healthcare planning, resource allocation, and policy development to advance equitable pediatric specialty care throughout the Inland Empire.

MILEY THERESE MIRANDA
ABC PARTICIPANT 2026

Throughout my life I have been afraid to step out of my comfort zone and try new things. The thought of leaving my group of friends to attend a STEM academy 45 minutes away from my home was terrifying to 12-year-old me. However, as I moved forward with my journey, I realized that a greater fear of mine was never knowing what could have been.

It wasn't until the second semester of 7th grade where I began to realize the priceless opportunities that a highly ranked STEM school had to offer. Now that I'm an incoming junior at Western Center Academy, I learned that everything I do should push me one step closer to my ultimate goal of becoming a pediatrician. As I continue my years in high school, I have made it a priority to take the high-level science classes that will expand my knowledge on complex scientific topics. Next year I will be taking on the leadership role of being a president of our school's Future Doctors Club. This summer, being an intern for the ABC program has allowed me to step out of my comfort zone even further to explore the complexities of Ovarian Cancer. Taking that step has not only allowed me to gain an abundance of new knowledge but has also taught me the valuable benefits of not being afraid to pursue a new environment. I now know that each time I step away from comfort, I am taking one step closer to my dreams.

CULTURING PATIENT-DERIVED OVARIAN CANCER CELLS IN VITRO CHANGES THE GENE EXPRESSION OF STEMNESS-RELATED MARKERS

Miley Miranda, Linh Nyguen, Mia Pierce, Brigette Vasquez, Juli Unternaehrer

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

Ovarian Cancer (OC) is the 5th deadliest gynecological malignancy targeting women worldwide. Previous studies suggest that cancer stem cells (CSCs) are one of the prominent factors that contribute to the foundation of tumor initiation, recurrence, and chemoresistance in OC. CSCs are recognized for their ability to differentiate and self-renew. These characteristics are identified as stemness characteristics. Identification of CSC relies on the presence of certain CSC markers. Our study focuses on testing the hypothesis that culturing cells in vitro results in a decrease in stemness characteristics of OC cells in later passages. Patient-derived OC (PDOC) cells were cultured in vitro under general cell culture conditions (37°C and 5% CO2). The cells were monitored daily through light microscopy. Once they reached the appropriate confluency (80%), they were passaged and harvested for reverse transcriptase quantitative PCR (RT-qPCR). RT-qPCR was performed after creating complementary DNA (cDNA) from RNA isolated from PDOC cells, measuring the mRNA expression of stem cell markers, POU5F1, NANOG, HGMA2, and LIN28 in comparison to the actin control. A segment of this experiment was conducted using PD8 cells at passage 0 and 10. After RT-qPCR was successfully completed, data results suggested a decrease in the mRNA expression of stemness markers POU5F1, NANOG, HGMA2, and LIN28 in the later passages of PDOC in vitro. For instance, there was a significant decrease in NANOG for later passaged PD8 OC cells in comparison to PD8 OC cells at passage 0. Future studies include a wider variety of PDOC samples and utilize different stemness markers in RT-qPCR testing, providing a better understanding of how CSCs behave, while allowing for an expansion on effective and more targeted therapies.

Undergraduate Training Program (UTP)

ADETAYO ADEOGUN
UTP PARTICIPANT 2026

Taking part in the UTP Program this summer changed how I view medicine. After years of shadowing physicians, I knew I wanted to care for patients, but I was also curious about how new treatments are discovered. Working in a research laboratory for the first time helped answer some of those questions while inspiring many more. The program challenged me to think like a scientist, strengthened my problem-solving skills, and gave me greater confidence in research.

I'm an upcoming junior at Texas Tech University, where I am majoring in Biochemistry. Outside the classroom, I play on the men's volleyball team and volunteer in hospice care. These experiences have taught me the importance of teamwork, compassion, and serving others during difficult moments. This summer, I conducted research at the Center for Health Disparities and Molecular Medicine under the mentorship of Dr. Danilo Boskovic and Ph.D. student Lidia Michelle Malina Wells. We studied how inflammation and bacterial stimulation affect platelet function to better understand bleeding complications in premature newborns.

God willing, I hope to earn an M.D./Ph.D. and build a career that combines patient care with biomedical research. I want to care for people during their most vulnerable moments while contributing to discoveries that improve many lives. I am deeply grateful to Dr. Boskovic and Lidia Michelle Malina Wells for their mentorship, patience, and encouragement. Their guidance has helped me grow as both a scientist and a person, and this summer is an experience I will always remember.

EFFECTS OF PORPHYROMONAS GINGIVALIS LIPOPOLYSACCHARIDE ON HUMAN PLATELET FUNCTIONS

Adetayo Adeogun, Lidia Michelle Malina Wells, Danilo S. Boskovic

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

Platelets are critical for hemostasis and contribute to immune responses during infection and inflammation. In premature infants, platelet functions may be impaired despite normal circulatory platelet counts, increasing the risks of prolonged bleeding and intracranial hemorrhage. This study investigates whether lipopolysaccharide (LPS) from Porphyromonas gingivalis modifies platelet hemostatic function or immune-related activity. Whole blood from 22 donors was incubated with 0, 1, or 20 μg/mL P. gingivalis LPS for progressively increasing time intervals. Platelet plug formation was assessed using the Siemens platelet function analyzer, PFA-100, which measures capillary closure time. Platelet activation markers and platelet–neutrophil aggregate formation was evaluated using flow cytometry. Exposure to 1 μg/ml LPS yielded closure times similar to baseline, while exposure to 20 μg/mL LPS led to a moderate prolongation in closure time, indicating delayed platelet plug formation. This effect was further pronounced with extended incubation. However, considerable blood-donor variability is observed. Flow cytometry reveals an increase in CD62P expression at 20 μg/mL LPS, minimal changes in CD63, and no consistent dose-dependent trend in PAC-1 binding. The most evident preliminary immune-associated response was an increase in platelet–neutrophil aggregate formation at higher LPS concentration, particularly in the presence of ADP. These results demonstrate that P. gingivalis LPS can induce measurable alterations in platelet behavior, especially at elevated concentrations and longer incubation periods. However, these effects are less substantial than those previously reported with the whole bacterium, suggesting that other bacterial components may also contribute to platelet dysfunction. Elucidating how inflammatory bacterial signals influence platelet function may clarify the mechanisms underlying bleeding complications during infection, particularly in vulnerable premature infants, and inform future research on biomarkers or therapeutic targets for inflammation-related platelet dysfunction.

ANDREA ISABEL LATORRE MARRERO

UTP PARTICIPANT 2026

I was born in Bayamón, Puerto Rico, a small island in size, but powerful in everything it represents to me. Being from Puerto Rico has allowed me to see how illness affects not only one person, but entire families who are searching for answers. Through my own family experience with cancer, especially with my mom, I have seen how destructive a disease can be when there is not enough information behind it. Because of that, I began to understand the importance of research and hope to do my part by adding my small "granito de arena" into the unknown world that it is. I am currently completing my undergraduate degree in Nursing, along with my pre-medical requirements and a minor in Chemistry. Because of this, I strive to pursue an MD/PhD, a dream many may find crazy coming from a nursing student. But my parents always told me there is no limit to the sky, reminding me to dream beyond what others may expect from me.

Participating in the UTP program has given me the opportunity to grow in a way I did not expect in such a short time. In Dr. Carlos Casiano's lab, I have been able to research and learn about prostate cancer and the LEDGF/p75 IBD interactome, including proteins such as JPO2, menin, MLL, IWS1, ASK1, PogZ, Med-1, HRP2, and c-MYC, and how they may contribute to cancer cell survival. I am grateful to my mentors, Dr. Carlos Casiano and Nathaly Manrique, for believing in me, guiding me, and patiently teaching me throughout this experience. I do this for Puerto Rico, for families like mine, and for every family that deserves answers and hope.

EXPRESSION OF THE LEDGF/P75 INTEGRASE BINDING DOMAIN INTERACTOME IN ENZALUTAMIDE-RESISTANT PROSTATE CANCER CELLS

Andrea Latorre, Nathaly Manrique, Pedro Ochoa, Yasmine Baca, Carlos A. Casiano

Center for Health Disparities and Molecular Medicine, School of Medicine, Loma Linda University, Loma Linda, CA; and Caris Life Sciences, Phoenix, AZ

Prostate cancer (PCa) is the most diagnosed cancer and the second leading cause of male cancer deaths in the U.S. Although androgen receptor signaling inhibitors (ARSIs) such as enzalutamide, have improved the treatment of advanced PCa, therapeutic resistance inevitably develops leading to cross-resistance to docetaxel-based chemotherapy. A key mechanism underlying this cross-resistance is glucocorticoid receptor (GR) bypass signaling, in which GR compensates for AR inhibition by activating transcriptional programs that promote tumor cell survival. GR signaling in PCa cells promotes the expression of LEDGF/p75, a transcriptional co-activator encoded by the PSIP1 gene that promotes the expression of genes involved in cellular stress survival, DNA repair, and therapeutic resistance. The C-terminal integrase-binding domain (IBD) of LEDGF/p75 functions as a protein interaction hub that recruits multiple transcriptional regulators to active chromatin. This LEDGF/p75 interactome is upregulated in docetaxel-resistant PCa cells and contributes to PCa aggressiveness. PCa patient data from the Caris Life Sciences' clinico-genomic database showed that the expression of genes encoding members of the GR-LEDGF/p75 transcriptional network was positively associated with PSIP1 expression in prostate tumors. We hypothesized that GR also activates this network in enzalutamide-resistant cells, leading to coordinated expression of multiple LEDGF/p75-associated transcriptional regulators that promote therapeutic cross-resistance. We performed Western blotting analyses of protein lysates from enzalutamide-sensitive and -resistant LNCaP cell lines to confirm concurrent GR and LEDGF/p75 upregulation. Lysates were subsequently evaluated for the expression of additional LEDGF/p75 IBD-interacting proteins to determine if they are likewise upregulated in enzalutamide-resistant cells. We anticipate these studies will determine whether the upregulation of the LEDGF/p75 IBD-interactome represents a molecular feature of both ARSI and taxane resistance, further defining its contribution to PCa drug cross-resistance.

BRIGITTE FONSECA MARROQUIN

UTP PARTICIPANT 2026

My journey into medicine began at the Inland Empire Women's Clinic, watching nurse practitioners guide patients through uterine tumor diagnoses. Having lost relatives to cancer, I had always carried a quiet fear of oncology, but seeing these clinicians inspired me to confront my anxiety through the Undergraduate Training Program. As a University of Redlands undergraduate double majoring in Health, Medicine, and Society and Women, Gender, and Sexuality Studies, this summer immersion transformed my fear into deep academic curiosity. Instead of shying away, I wanted to understand cancer's inner workings, sparking an interest in becoming a surgeon-scientist bridging patient care and research.

This passion came alive in Dr. Ying Nie's laboratory, where PhD candidate Kristian Holgersson guided my investigation into how lung cancer cells metabolically impair the heart during radiation. Stepping into the lab each day went beyond merely executing routine protocols; instead, the experience resembled solving a complex puzzle. Witnessing the rapid growth of animal tumor models in just a week pushed me past basic laboratory boundaries to uncover the fundamental mechanisms driving therapy resistance.

Outside the lab, the program's seminars and workshops deeply refueled my medical school motivation. I felt a clear sign from God confirming this path is exactly where he wants me to be. I am incredibly grateful to Dr. Nie, Kristian, and the team for helping me overcome my fears and foster my scientific curiosity. Moving forward, I will carry this foundational discipline to evaluate health disparities and serve underserved populations with a faith-centered heart.

SYSTEMIC METABOLIC ALTERATIONS OF LLC1 TUMORS DRIVES CARDIOTOXICITY

Brigitte Fonseca Marroquin, Kristian Holgersson, Ying Nie, Guangyu Zhang, Reinhard Schulte,

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

This ongoing study investigates the hypothesis that LLC1 murine tumors induce systemic metabolic alterations, with tumor-derived metabolites driving cardiotoxicity. Three groups were included: healthy mice with no tumor, tumor-bearing mice injected subcutaneously with LLC1 cells, and tumor-bearing mice treated with the anti-glycolytic agent TEPP-46. Five mice were included per group. Tumors were allowed to reach 2 cm3 before ending the experiment. Plasma metabolomics with mass spectrometry evaluated the top 25 most significantly altered metabolites with heatmap analysis, and plasma cardiac troponin T was quantified with ELISA between the groups. Additionally, hearts were resected, and immunohistochemistry and H&E staining were applied to assess microvascular architecture and overall tissue morphology. The metabolomics data demonstrated distinct clustering between the experimental groups, with principal component analysis (PCA) indicating systemic metabolic alterations. Moreover, heatmap analysis revealed metabolic alterations between the experimental groups, mapping the involvement of specific metabolites. ELISA revealed elevated plasma cardiac troponin T in the untreated tumor-bearing group, while the TEPP-46 treatment group had significantly lower cardiac troponin T levels. Immunohistochemistry showed distinct heart capillary fragmentation and significant reduction of microvascular network length, junction counts, and network loop density in the non-treated tumor-bearing group. TEPP-46-treated mice showed a significant delay in tumor growth. Overall, our results support that the presence of LLC1 lung tumors induces systemic metabolic alterations and cardiac microvascular deformation. Further investigations are required to determine whether TEPP-46 can normalize these alterations and reduce early cardiotoxicity in tumor-bearing mice.

CAMERON SCOTT
UTP PARTICIPANT 2026

I attend Oakwood University in Alabama as a rising junior for a bachelor's degree in biology. Currently, I am working under Doctor Rameshwar Patil on using Boron Carbide nanoparticles for BNCT treatment for head and neck cancers. I have already completed two presentations, one on Zinc Finger Homeobox 3 dysfunction in relation to prostate cancer, and another on Venous Scaffolding for Varicose Veins. Since coming here, I have been exposed to technologies and developments completely novel to me, such as iron and gold nanoparticle synthesis, a rotary vapor device, and more. I am grateful that I had the opportunity to witness and participate in cutting-edge technology such as Dr. Patil's research.

I have a litany of creative hobbies, including writing, drawing, and music. He hopes to get accepted into medical school and become an Orthotist when he graduates in two years.

TARGETED BORON CARBIDE NANOPARTICLES FOR BNCT IN HEAD AND NECK CANCER

Cameron Scott1, Sanjay Yadav1, Kevin Nick2, Rameshwar Patil1*

1Department of Basic Sciences, Division of Cancer Sciences, School of Medicine, Loma Linda University, Loma Linda, CA

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Annually, over half a million individuals are diagnosed with Head and neck squamous cell carcinoma (HNSCC), with about 300,000 of those dying from HNSCC worldwide. Epidermal Growth Factor Receptor (EGFR) is a cell surface receptor that is commonly overexpressed in HNSCC and plays a crucial role in the growth and development of HNSCC tumors. Increased EGFR activity aids HNSCC resistance to radiotherapy. Boron Neutron Capture Therapy (BNCT) is emerging as a promising treatment approach for many malignancies, including HNSCC. The effectiveness of BNCT relies on the selective delivery and sufficient accumulation of a high amount of boron-10 within tumor cells, which is a major issue for BNCT. Boron Carbide nanoparticles (B4C) contain high amounts of boron as they are made of a 4:1 ratio of boron to carbon. In this study, boron carbide nanoparticles were functionalized with polyethylene glycol and a tumor-targeting EGFR peptide for improved tumor targeting and cell uptake. An EGFR-specific targeting peptide was attached to the B4C nanoparticle surface via a stable covalent bond for effective tumor targeting and accumulation in HNSCC. The nanoparticle surface was coated with polyethylene glycol to increase the colloidal stability. Functionalized nanoparticles were characterized by various analytical methods such as UV-vis spectrometry, Dynamic Light Scattering, and wet chemical assays, established in Dr. Patil's lab. In vitro evaluation is ongoing to quantify the cellular uptake of targeted v.s. nontargeted nanoparticles in multiple EGFR-expressing cell lines. Cellular uptake of targeted nanoparticles will be studied by quantifying the internalized gold using Microwave Plasma Atomic Emission Spectroscopy (MP-AES). The development of this multifunctional EGFR-targeted nanoparticle platform has the potential to improve targeted boron delivery, enhancing BNCT for effective treatment of HNSCC.

DARINE ABU HILAL
UTP PARTICIPANT 2026

I started conducting research in the Fig Neuro-Lab as a senior in high school with hopes of learning lab techniques and gaining research experience. At the time, I simply wanted to be a physician. Under the mentorship of Dr. Johnny Figueroa, I had the opportunity to lead my own project, analyzing how adolescent stress can lead to binge eating behaviors. From that experience, I learned more than just how to use software for data analysis; I learned how to critically think, effectively work with others, and clearly communicate difficult concepts. More importantly, I developed a passion for studying neuroscience and health disparities which has led me to where I am today. This past spring, I wrapped up my second year at UCLA where I am pursuing a bachelor's degree in neuroscience and a minor in public health. My future goal is to become a clinician working to reduce health disparities and improve global health.

I am grateful to Dr. Figueroa for allowing me to volunteer in the lab for the past three years and welcoming me back for the Undergraduate Training Program (UTP). This summer, I am leading a project to develop a non-invasive method for fecal microbiota transplantation, which reduces stress exposure, making results from stress and microbiome studies more accurate. This opportunity has allowed me to grow as a researcher and as an individual. I will forever be grateful for the people I met and the learning experiences I gained through this program and the Fig Neuro-Lab.

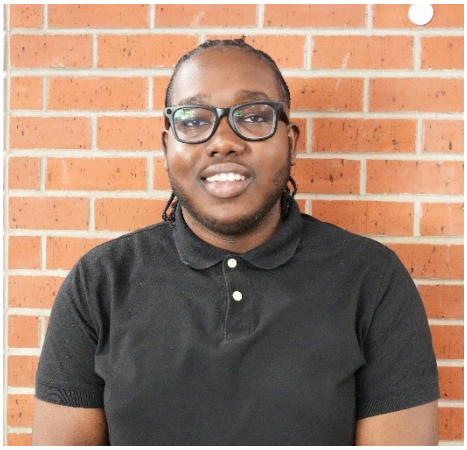
DEVELOPMENT OF A NON-INVASIVE FECAL MICROBIOTA TRANSPLANTATION METHOD FOR RATS USING NUTRIENT-ENRICHED FROZEN PELLETS

Darine Abu Hilal, Katherine Granados, Allison Rhee, Leanne Mercado, Julio Sierra, Johnny D. Figueroa

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

The gut microbiome plays a critical role in regulating metabolism, immune function, and brain health. Fecal microbiota transplantation (FMT) is widely used in preclinical research to investigate causal relationships between gut microbial communities and disease. Conventional FMT in rodents is typically administered via oral gavage, an invasive procedure that requires repeated restraint and may introduce stress that can influence behavioral and physiological outcomes. To address this limitation, we evaluated the feasibility of a non-invasive FMT approach in rats using nutrient-enriched frozen pellets containing donor fecal material. Recipient rats were provided access to the pellets for two hours, during which voluntary consumption was quantified to assess acceptance of the delivery method. Rats readily consumed an average of 0.8 g, corresponding to 58% of each pellet. Ongoing studies will determine microbial engraftment using 16S rRNA sequencing and compare the effectiveness of pellet-based FMT with conventional oral gavage. This approach has the potential to reduce procedural stress while providing.

JAMES MITCHELL
UTP PARTICIPANT 2026



Participating in the UTP has been a wonderful opportunity for my personal and academic growth. For as long as I can remember, I have always been fascinated by the intertwined fields of medicine and science. One reason science intrigues me so much is its ability to provide deep explanations while simultaneously leaving endless opportunities for further discovery.

I am a rising junior at Oakwood University, majoring in Biology on the pre-medicine track. My long term goal is to become a practicing physician and an active medical researcher. I aspire to be the type of physician that can relate to my patients on multiple levels, approach situations with expertise, and ultimately display the love of Christ to everyone I encounter. This program has truly been a blessing to me, and I am certain that the skills I have developed during the UTP will translate directly into my academic journey. In addition, the connections I have built and the knowledge I have acquired will definitely support me in my future professional endeavors.

I am especially thankful to my mentor, Dr. Jiang Zhong for welcoming me into his lab, providing me with the information necessary for meaningful research, and fostering an environment of collaboration.

ANALYSIS OF MDM2 mRNA VARIANTS IN ACUTE MYELOID LEUKEMIA

James Mitchell, Yingjie Fu, Emelie Zhu, Henry Trinh, Jiang F. Zhong

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

Acute myeloid leukemia (AML) is a highly aggressive form of cancer of the blood and bone marrow, characterized by the overproduction of immature myeloid cells. Unlike other types of leukemia that mainly affect children, AML is typically identified in older adults, with a median age of diagnosis between 65 and 72 years. AML is clinically important because it is one of the most common adult leukemias and continues to present significant treatment challenges despite recent advances. Dysregulation of the MDM2 gene, the primary negative regulator of the p53 tumor suppressor gene, has been implicated in a notable amount of AML cases. Overexpression or variation of MDM2 can lead to the inappropriate degradation of p53, decreasing proper cell apoptosis and enabling leukemic cell survival.

During this study, MDM2 mRNA variants were investigated in a specimen collected from an AML patient (10C1D8). Sanger sequencing was utilized to evaluate variations among the MDM2 mRNA transcript. The resulting sequences were analyzed using the NCBI BLAST tool to confirm alignment with MDM2. These were compared with a reference MDM2 sequence, allowing for further identification of differences among MDM2 mRNA transcripts.

Sequence analysis allowed us to discover some heterogeneity among MDM2 transcripts. In some transcripts, exon 5 was missing, while in others it was retained. The detection of exon 5 skipping may be biologically significant, as transcript variation within MDM2 could potentially have an effect on its interaction with p53, influencing cell survival pathways.

These results suggest the potential significance of MDM2 mRNA variation in AML patients and support the value of patient gene sequence analysis. Continued investigation would be necessary to determine the frequency of exon 5 skipping and whether this occurrence has clinical and functional significance in AML. These findings may contribute to further research involving targeted therapies by improving understanding of MDM2 variation in AML.

JASMYN VANTERPOOL
UTP PARTICIPANT 2026

I am a rising junior attending Oakwood University located in Huntsville Alabama, majoring in Applied Mathematics with a concentration in Chemistry and Biology. During the school years, I tutored high school students and college students in mathematics, ranging from Algebra 1 to calculus III with hopes of bridging the gaps between minority and the lack of understanding and attraction to this field.

Once I graduate, I plan on obtaining a PhD in Geochemistry and working in that respective field. I have received honors and recognition for my academic prowess, and intense research oriented undergraduate career in preparation for graduate school, attending and presenting at many research conferences. This summer I am working with Dr. Christopher C. Perry with assistance from Cedric Lansangan on Plasmon enhanced Au-Ag Decorated Fe doped TiO_2 for Photocatalysis and Antibiofilm Applications. Chemistry and Geology are my passions, so the most interesting part of this project was understanding the chemistry behind the mechanics, as well as the optimization and enhancement of the structure for our own synthesized materials. Contrary to my scientific and academic passions and interests, my hobbies include puzzles, arts and crafts of any kind, reading, dancing, playing volleyball, and most of all, learning.

I would like to thank Dr. Christopher C. Perry for opening his lab to me, and creating an environment where learning is boundless.

LASMON-ENHANCED AU–AG DECORATED FE DOPED TiO2 NANOFIBERS FOR PHOTOCATALYSIS AND ANTIBIOFILM APPLICATION

Jasmyn Vanterpool[1], Dr. Christopher C. Perry[2], Cedric Lansangan[3]

Mathematics and Computer Science, School of Basic Sciences,
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In recent years, environmental pollutants such as organic dyes and biofilm associated infections have increased, posing significant threats to both environment sustainability and public health. Titanium dioxide (TiO_2) is a well-known, cost-effective photocatalyst; however, its responsivity under visible light is limited due to its large bandgap. In this study, TiO2 nanofibers (NFs) were synthesized and combined with various combinations of plasmonic metals such as gold (Au) and silver (Ag), as well as iron (Fe) nanoparticles (NPs). Au and Ag nanoparticles were first synthesized and subsequently used to produce bimetallic Au-Ag seeds and nanostars, which were characterized using UV-vis spectroscopy and Dynamic Light Scattering (DLS). These NPs were then used to decorate the NFs using a one-pot photodeposition process. The prepared samples included TiO_2 NFs, Fe:TiO_2 NFs, Fe:TiO_2/Au-Ag NFs, and TiO2/Au-Ag NFs. These samples were characterized using Scanning Electron Microscopy (SEM) to evaluate their morphology and compositions. Photocatalytic properties were assessed using a spectrophotometric system. We anticipate that the Fe-doped TiO_2 NFs will exhibit enhanced photocatalytic activity in comparison to the undecorated NFs. However, the bimetallic Au-Ag sample will have stronger photocatalytic properties in comparison to the Fe-doped sample due to plasmon-enhanced visible-light absorption. Finally, the trimetallic Fe:TiO_2/Au-Ag system will exhibit the highest photocatalytic activity compared with the other samples under visible-light irradiation due to plasmonic behavior, charge separation, and increased receptivity to visible light caused by the Fe-doping. Future directions include the antimicrobial and biofilm applications of these NFs.

JAVAN CHARLES
UTP PARTICIPANT 2026

Growing up in Queens, New York, I have been drawn to medicine from a young age and have always known I wanted to become a physician. Pursuing that dream led me to Oakwood University, where I am currently an incoming junior majoring in Pre-Med Biology. As I continue my studies, I have developed a growing interest in scientific research through the UTP program, where I have gained valuable experience and deepened my appreciation for the role research plays in advancing medicine. I plan to be a medical doctor, with an interest in cardiology, while remaining open to the many opportunities within medicine. These experiences have strengthened my desire to pursue a career with compassionate patient care and scientific discovery.

Outside of academics, music has been one of the greatest influences in my life. As a drummer and pianist, I perform in church services, ensembles, and other live settings while continuing to explore music production. Music has taught me the value of discipline, teamwork, and serving others. Whether through music, ministry, or community service, serving others has become one of the most meaningful parts of my college experience. My faith in God inspires me to pursue excellence in all that I do.

I am sincerely grateful to Dr. Langridge for welcoming me into the research environment, teaching me to work meticulously and with patience, and providing me with the opportunity to expand my scientific knowledge and further develop as a student, aspiring scientist, and future healthcare professional.

ISOLATION, PURIFICATION, AND CHARACTERIZATION OF A RECOMBINANT CTB-SARS-CoV-2-ACE2-RBD FUSION PROTEIN FOR MUCOSAL VACCINE DEVELOPMENT

Javan Charles, William H. Langridge

Center for Health Disparities and Molecular Medicine, Basic Sciences, Biochemistry Division,
School of Medicine, Undergraduate Training Program,
Loma Linda University, Loma Linda, CA

The respiratory mucosa serves as the first line of defense against viral pathogens and is the primary site where many respiratory infections are established. The COVID-19 pandemic demonstrated the importance of developing vaccine strategies that extend beyond systemic immunity by promoting targeted protective responses at mucosal surfaces, where respiratory pathogens initially establish infection. This approach addresses a major gap in vaccine design by focusing immune protection at the site where respiratory infections begin. Therefore, to enhance protection against emerging respiratory viral pathogens, it is important to develop a more effective mucosal vaccine platform. This work aims to isolate, purify, and characterize a recombinant CTB-SARS-CoV-2 ACE2 receptor-binding domain (RBD) fusion protein expressed in *Escherichia coli* as a step towards development of a more effective mucosal respiratory virus vaccine. The CTB-RBD fusion protein combines the cholera toxin B subunit (CTB), a mucosal adjuvant that enhances immune activation at mucosal surfaces, with the SARS-CoV-2-ACE2-RBD, a viral protein recognized by the immune system to generate protective responses. The vaccine fusion protein was expressed in E. coli BL-21 cells containing expression plasmid pS-30, which encodes the CTB-SARS-RBD gene. The vaccine protein was isolated from bacterial cultures by lysis of the bacteria and removal of cell fragments by centrifugation followed by purification of the vaccine by acrylamide gel electrophoresis, electroelution, and Western blot immunodetection of the vaccine protein. Our experimental results support production of the vaccine protein in bacteria establishing a basis for evaluating vaccine potential to promote an effective mucosal immune response. Future studies will assess vaccine protein interaction with dendritic cells and T cells to stimulate virus antigen specific T cell activation and proliferation, key components of cellular immunity. Ultimately, this work will establish a basis for development of CTB-based mucosal vaccine strategies that strengthen the level of immune protection against novel respiratory virus pathogens.

MIA PIERCE
UTP PARTICIPANT 2026

I have never been satisfied with simply knowing *what* happens. I have found that I must understand *why*. I began college at Azusa Pacific University as a kinesiology major, but quickly realized I wasn't fulfilled by my studies. I found that I was drawn more to intricate molecular mechanisms instead of the broad outcomes of biological processes. The more I learned, the more I fell in love with science and discovered my passion for scientific research.

While juggling playing Division II basketball and taking on leadership roles in my school's TriBeta Biological Honors Society and Student Athlete Advisory Committee, I have pursued research opportunities in cancer biology and immunology, both reinforcing my fascination with understanding disease at its most fundamental level. As I head into my senior year, my interests have gradually narrowed in on women's reproductive health, an area that remains largely underrepresented despite its importance. I hope to help close that gap by investigating the molecular and immunological mechanisms that drive gynecologic diseases and translating those discoveries into improved diagnostics and therapies.

Joining Dr. Unternaehrer's laboratory this summer has been an exciting opportunity to continue pursuing that goal. Working with ovarian cancer has been truly eye-opening. Every experiment reminds me that answering one question almost always uncovers ten more, which is exactly what I love most about research. My goal is to pursue an MD/PhD and combine patient care with translational research dedicated to improving women's health. I am incredibly grateful to Dr. Unternaehrer, Jacqueline Coats, and Brigitte Vazquez, as well as the other members of the laboratory for their mentorship and for the opportunity to continue growing as a scientist and future physician.

SERIAL PASSAGING ALTERS CANCER STEM CELL BIOMARKER EXPRESSION IN PATIENT-DERIVED OVARIAN CANCER CULTURES

Mia Pierce, Miley Miranda, Linh Nyguen, Jacqueline Coats, Brigitte Vazquez, Juli Unternaehrer

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

Ovarian cancer (OC) is the most fatal gynecological disease due largely to late diagnosis, high rates of recurrence, and the acquisition of chemoresistance following treatment. Cancer stem cells (CSC) are a rare population of self-renewing cells found within tumors that are linked to recurrence, metastasis, and chemoresistance. The purpose of this study was to determine how CSC-associated biomarker abundance in patient-derived cultures is altered in a 2D *in vitro* model after serial passaging. Following tumor removal, serial passaging in a nutrient-rich 2D culture environment may gradually alter the expression of stemness markers as the cells adapt to an environment different from their native microenvironment. It was hypothesized that the abundance of stemness markers CD133, CD44, CD117, and ALDH would decrease with serial passaging, thereby affecting relevance of the tumor model. To answer this question, six patient-derived cell lines were grown in 2D culture and analyzed at the initial passage (P0) and a later passage (P7-P12). At these time points, flow cytometry was utilized to quantify the expression levels of the above-mentioned CSC markers. Flow cytometry data were analyzed using FlowJo 10.8.1 and the abundance of each marker was determined, along with ALDH+ CD133+ cells, which are reportedly enriched for ovarian CSCs. In parallel, RT-qPCR was used to evaluate the expression of the CD133, CD44, and CD117 biomarkers. Results confirm that the expression of these biomarkers is altered with serial passaging, with varying results among the six patient-derived cells. These findings suggest that the use of patient-derived 2D cell cultures may present itself as a reliable model for studying ovarian cancer CSCs only for a limited period of serial passaging.

SHANIA MCKELVEY
UTP PARTICIPANT 2026

As I pursue a career in medicine, I carry with me the belief that every setback can become a foundation for future success. To me, medicine is an opportunity to understand one human being through the experiences of another. This summer challenged me to broaden my perspective on healthcare through scientific research. Beyond gaining valuable laboratory experience, I developed a deeper understanding of myself, strengthened my passion for knowledge, and further inspired my desire to integrate my Christian values into my future practice as a physician.

As a rising junior at Oakwood University in Huntsville, Alabama, I am pursuing a Bachelor of Science degree with a concentration in Pre-Medicine. I am also a full-time student-athlete and will soon begin my third year as a member of Oakwood University's women's volleyball team. I have been privileged to complete numerous hours of shadowing, clinical, and volunteer experience; however, I am eager to continue expanding my understanding of healthcare while selflessly serving those around me.

As a participant in CHDMM's Undergraduate Training Program, I had the distinct opportunity to analyze the expression of various immune cells in ovarian cancer tissues and examine their correlation with the cancer's clinical classification. I would like to sincerely acknowledge and thank Dr. Salma Khan, Joseph Cruz, Romi Yamauchi, Alena McQuarter, and every member of Dr. Khan's laboratory for their guidance and mentorship, which will undoubtedly have a lasting impact on my future career as a physician.

DETERMINING THE DIFFERENTIAL EXPRESSION OF CANCER-ASSOCIATED FIBROBLASTS AND TUMOR-ASSOCIATED MACROPHAGES IN THE TUMOR MICROENVIRONMENT OF HIGH GRADE SEROUS OVARIAN CANCER

Shania McKelvey, Alena McQuarter, Joseph Cruz, Cody S. Carter, Salma Khan

Center for Health Disparities and Molecular Medicine, Pathology & Human Anatomy, School of Medicine, Loma Linda University, Loma Linda, CA

Epithelial ovarian cancer, particularly high-grade serous ovarian carcinoma (HGSOC), is the deadliest gynecologic malignancy due to its late diagnosis and poor response to therapy. The tumor microenvironment (TME) plays a critical role in tumor progression and treatment resistance. Two key components of the TME are cancer-associated fibroblasts (CAFs) and M2-like tumor-associated macrophages (TAMs), both of which promote tumor growth and immune evasion. CAFs remodel the extracellular matrix, stimulate angiogenesis, and secrete factors that support tumor survival, whereas M2 TAMs enhance metastasis and suppress anti-tumor immune responses. Together, these stromal cells create an immunosuppressive microenvironment that contributes to therapeutic resistance. This study evaluated the abundance of CAFs and TAMs in the TME of HGSOC and examined their association with patient outcomes. Multiplex fluorescent immunohistochemistry was performed on formalin-fixed, paraffin-embedded HGSOC tissues using alpha-smooth muscle actin (α-SMA) as a CAF marker and CD163 as an M2 TAM marker. Stained tissues were imaged with a Keyence fluorescence microscope, and biomarker expression was correlated with clinical data from the Loma Linda University HGSOC cohort. Our findings demonstrated that increased expression of α-SMA and CD163 was associated with more aggressive HGSOC and poorer overall survival. These results suggest that enrichment of CAFs and M2 TAMs contributes to an immunosuppressive, therapy-resistant tumor microenvironment and supports the development of combination strategies targeting both stromal and immune components to improve outcomes in HGSOC.

ZACHARY CHANG
UTP PARTICIPANT 2026

I attend Pacific Union College in Napa Valley, CA as a Biology major going into my sophomore year. At PUC, I serve as a Student Government Senator and propose legislation to improve the campus. I was recently awarded the "Senator of the Year" award. I also play guitar for Sabbath School, VESPERS, and PUC Church. I am also a part of Outreach Ministries at PUC where we serve the homeless in Clearlake and Berkeley.

My goals for the future are to do research for Loma Linda University while being a dental or medical student. I am currently working with Dr. Sean Wilson on a metabolomics project. Specifically, I am analyzing adult carotid tissue in sheep grown in high and low altitudes. The most interesting part about this research is that this project has significant implications in relation to hypertension and changes in metabolic pathways in humans. I also plan on going to mission trips next summer to Sri Lanka or India.

In my free time I like to play piano/guitar, golf, and go hiking. One thing I learned in this program is that conducting research requires a lot of internal motivation. This is because research does not always have a set deadline or "exam" unlike college classes.

Thank you, Dr. Wilson, for your guidance, patience, and passion for what you do. I can see why the medical students speak so highly of you.

LONG-TERM HYPOXIA INDUCES METABOLIC REPROGRAMMING IN SHEEP ADULT CAROTID ARTERIES

Zachary Chang2, Sean Wilson1

Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA1; Pacific Union College2

Long-term hypoxia (LTH) causes structural and functional changes to the adult carotid artery, leading to disruptions in cerebral blood flow and vascular homeostasis. While metabolic reprogramming and oxidative stress are well-studied in other vascular tissues, their roles in the adult carotid artery under long-term hypoxia remain poorly understood. We hypothesized that high-altitude LTH would induce significant metabolic reprogramming in the adult sheep carotid artery, disrupting energy metabolism, increasing inflammation, promoting oxidative stress, and altering lipid signaling. To investigate this, carotid artery samples were obtained from adult normoxic sheep raised at 355 meters in Loma Linda, CA, and hypoxic sheep raised at 3,801 meters above sea level in Bishop, CA. Metabolite levels were quantified using ultra-performance liquid chromatography tandem mass spectrometry (UPLC-MS/MS). Chemical similarity enrichment analyses and complex pathway visualization were performed on the datasets. Enrichment analysis revealed changes in key metabolic pathways including glycolysis, purine metabolism, glutathione metabolism, cysteine metabolism, sphingolipid metabolism, and the glycerol phosphate shuttle. Ten metabolites were found to be significantly different between hypoxic and normoxic groups. Increased glycerol-alpha-phosphate suggests compensatory upregulation of the glycerol phosphate shuttle to regenerate NAD+ and sustain glycolytic flux when the electron transport chain is oxygen-constrained under hypoxia. Accumulation of inosine and guanosine, purine catabolism products of ATP and GTP, indicates accelerated nucleotide breakdown consistent with oxidative phosphorylation falling behind cellular energy demand. Increased 3-hydroxybutyric acid suggests a metabolic shift toward fat-based energy production when glucose oxidation is limited by reduced oxygen availability. Decreased 12(13)-EpOME, an anti-inflammatory oxylipin, indicates disrupted lipid signaling and is consistent with a pro-inflammatory shift in the adult carotid artery. Together, our findings support that long-term hypoxia induces broad metabolic reprogramming in the adult carotid artery, promoting energetic strain, vascular inflammation, oxidative stress, and disrupted lipid metabolism.

MTP – Medical Training Program

ALLISON RHEE
MTP PARTICIPANT 2026

Growing up, I watched several members of my family develop Type 2 diabetes. Learning about my grandfather's experience surviving starvation during the Korean War sparked my curiosity about how early-life experiences and environmental exposures may influence health across generations. That interest led me to seek research exploring health disparities, ultimately drawing me to the Fig NeuroLab, where Dr. Johnny Figueroa investigates how obesity and environmental factors affect vulnerable populations in Southern California.

The Fig NeuroLab has been my scientific home since 2023. As a sophomore undergraduate volunteer, I learned cryosectioning for the first time and discovered how exciting it was to contribute to meaningful research. Three years later, I am grateful to return to the lab as an MD/PhD student in the Loma Linda University School of Medicine.

My research explores how interactions between the gut microbiome, brain, and behavior may contribute to neurodevelopment and health. Working in the Fig NeuroLab has sparked a deeper curiosity about the gastrointestinal system and the remarkable ways it communicates with the brain, broadening both my scientific interests and my appreciation for translational medicine.

I am deeply grateful for the mentorship of Dr. Johnny Figueroa, Dr. Timothy Simon, Julio Sierra, and my lab members whose guidance has shaped my development as a scientist. I look forward to a career where I can care for patients, ask meaningful scientific questions inspired by the clinic, and translate research discoveries into therapies that improve human health.

EVALUATING P-CRESOL AS A CANDIDATE MICROBIAL MEDIATOR OF GUT DYSBIOSIS–INDUCED FEEDING AND SOCIAL BEHAVIOR

Allison Rhee, Julio Sierra, Katherine Granados, Darine Abu Hilal, Leanne Mercado, Johnny D. Figueroa

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

The gut microbiome communicates bidirectionally with the brain through bioactive metabolites that regulate neurodevelopment, metabolism, and behavior. However, the specific microbial metabolites responsible for mediating gut–brain dysfunction remain poorly understood. Previous studies from our laboratory demonstrated that adolescent exposure to psychosocial stress and a Western diet induces gut dysbiosis and persistent behavioral abnormalities. Untargeted metabolomic profiling identified elevated p-cresol (4-methylphenol), a tyrosine-derived bacterial metabolite, as a candidate mediator of gut–brain dysfunction. Increased p-cresol abundance was associated with altered hippocampal microglial morphology and maladaptive behaviors following adolescent psychosocial stress and Western diet exposure. These findings led us to investigate whether chronic p-cresol exposure is sufficient to induce alterations in brain and behavior associated with gut dysbiosis.

Female Lewis rats were pair-housed, matched by weight and acoustic startle reactivity, and randomly assigned to receive either p-cresol (0.25 g/L) or vehicle in drinking water throughout adolescence. Body weight and water intake were monitored longitudinally, and home-cage behavior was assessed biweekly using the PhenoTyper system to quantify feeding behavior and locomotor activity. Behavioral phenotyping also includes assessments of anxiety-like and social behaviors to determine the impact of chronic p-cresol exposure on gut–brain function. By administering p-cresol in the absence of psychosocial stress, this study isolates the contribution of a single microbiome-derived metabolite to behavioral outcomes.

This study will determine whether p-cresol is sufficient to reproduce behavioral abnormalities associated with gut dysbiosis, providing mechanistic insight into how individual microbial metabolites influence adolescent brain development and gut–brain communication. Establishing causal roles for microbiome-derived metabolites will advance our understanding of microbiome–brain interactions and may identify novel therapeutic targets for disorders characterized by maladaptive eating and social dysfunction.

JOCELYN CHOW
MTP PARTICIPANT 2026

Growing up in an immigrant household, I witnessed how language barriers can limit patients' understanding of their health and access to care. Wanting to better support my family and others facing similar healthcare barriers, I was inspired to pursue medicine. Currently, I am a second year medical student at Western University of Health Sciences. Before medical school, I worked as a research assistant in a translational neuro-oncology lab and served as the after-school coordinator for a Christian organization working with the local refugee community. Through the relationships I built with my students, I developed a strong desire to work with underserved communities. After moving back home to Southern California, I continued to explore this interest as a medical assistant at a federally qualified health center that served the immigrant community in Los Angeles. These experiences shaped my career aspirations to integrate research with health advocacy to develop evidence-based interventions that address health disparities in marginalized communities. This summer, I am working in Dr. Carlos Casiano's lab to characterize ENO1 and ENO2 as potential biomarkers of glioblastoma. I am grateful for the privilege of being at Loma Linda University, where faith is integrated into both scientific discovery and compassionate patient care. It has been such an exciting time being immersed in a community of faith at the Center for Health Disparities and Molecular Medicine with like-minded peers that are also passionate about addressing healthcare inequity, and I would love to have the opportunity to continue my training at this wonderful institution!

CHARACTERIZING THE EXPRESSION AND LOCALIZATION OF ENO1 AND ENO2 IN GLIOBLASTOMA CELLS

Jocelyn Chow, Kathleen Escobar-Acuña, Lemti Nyirendah, Krystal Santiago, Nathan R. Wall, Carlos A. Casiano

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

Glioblastoma (GBM) is a highly aggressive glioma that presents with poor patient outcomes due to its rapid recurrence and treatment resistant nature. Despite therapeutic interventions, average survival rates for patients range from 12–15 months, with five-year survival rate of 6.8%. Recent studies explored glycolytic metabolic reprogramming in GBM, implicating alpha-enolase (ENO1) overexpression in glioma progression. ENO1 and its neuron-specific isoform ENO2 have been studied as promising biomarkers in other malignancies such as prostate cancer (PCa), underscoring their value as pan-cancer therapeutic targets. We hypothesized that, like in PCa, the intracellular expression and surface abundance of ENO1 are higher than those of ENO2 in GBM. Neuroendocrine prostate cancer (NEPC) and GBM cell lines were obtained commercially through ATCC. NEPC cells served as positive controls due to their aggressive nature and elevated ENO1 and ENO2 expression. For Western blotting analysis, cell lysates were incubated with specific antibodies to ENO1 and ENO2. Flow cytometry was performed to characterize the intracellular and surface abundance of ENO1 and ENO2. The Cancer Genome Atlas (TCGA) GBM dataset was mined to construct Kaplan-Meier survival curves. The protein expression levels of ENO1 and ENO2 were similar in GBM and NEPC cells. The TCGA GBM dataset revealed that patients with high ENO1 (n=18) and ENO2 (n=17) expression had decreased survival rates within 12 months (log-rank p=0.0691 and p=0.0019, respectively). Collectively, our preliminary data suggest that ENO1 and ENO2 are highly expressed in GBM cells and associated with worse patient outcomes. We anticipate that ENO1 and ENO2 are present on the GBM cell surface, where they could serve as potential therapeutic targets.

LEANNE E. MERCADO THEN
MTP PARTICIPANT 2026

As a rising second-year medical student at Universidad Central del Caribe in Puerto Rico, my goal is to pursue a career in internal medicine in order to serve my community. I aspire to establish a clinical practice with a strong emphasis on health advice and preventive care. By encouraging healthy lifestyle changes, I hope to empower patients to regain control of their health. As I continue my medical training, I also plan to actively participate in research and community service to better understand my community's needs and become a more compassionate physician.

Participating in Loma Linda's summer Medical Training Program (MTP) reaffirmed my passion for science and healthcare. In Dr. Johnny Figueroa's laboratory, I was fascinated by the complexity of the gut microbiome and its impact on the body beyond the gut, including its roles in hormonal regulation and neurodevelopment. Despite years of research, many mechanisms remain a mystery, underscoring the complexity of the human body. What fuels my passion for medicine is that every discovery raises new questions, each with the potential to contribute to patient care. I hope to utilize the knowledge I gain as an MTP participant to empower others to take an active role in their health.

The problem-solving skills and confidence I acquired in this program mark the first milestone towards this goal. I am grateful for this amazing opportunity and for the mentors, colleagues, and friends who supported me along the way.

ANTIBIOTIC-INDUCED GUT DYSBIOSIS ALTERS HIPPOCAMPAL ACTIVITY DURING ADOLESCENT BRAIN DEVELOPMENT

Leanne Mercado, Katherine Granados, Terence Killebrew, Darine Abu Hilal, Allison Rhee, Julio Sierra, Jo-wen Liu, Johnny D. Figueroa

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

The gut microbiome plays an essential role in immune, metabolic, and neural development during adolescence. Emerging evidence suggests that microbial signals regulate microglial maturation and synaptic plasticity; however, the mechanisms through which gut dysbiosis influences adolescent brain development remain poorly understood. This study examined whether antibiotic-induced microbiome depletion alters hippocampal function during adolescence. Female Lewis rats received a broad-spectrum antibiotic cocktail in drinking water for three weeks to induce gut microbiota depletion. Following a 72-hour washout period, animals underwent behavioral testing and functional magnetic resonance imaging (fMRI). Antibiotic-treated rats exhibited reduced dorsal hippocampal activity compared with untreated controls, supporting a role for the gut microbiome in normal hippocampal maturation. Ongoing studies are evaluating neuroplasticity-associated proteins, including estrogen receptor β (ERβ), brain-derived neurotrophic factor (BDNF), and tropomyosin receptor kinase B (TrkB), together with microglial phenotyping and metabolomic profiling to identify mechanisms linking gut dysbiosis to altered brain development. These findings support the gut microbiome as a critical regulator of adolescent hippocampal maturation and provide a foundation for understanding how microbiome disruption influences long-term brain function.

MELISSA LEE

MTP PARTICIPANT 2026

I took my first step into scientific research when I volunteered in Dr. Erik Behringer's lab during the summer of 2024. This introduction to laboratory work led me to pursue my own research project in organic chemistry the following school year at my university. These experiences showed me the importance of careful and meticulous work at the basic science level in order to make discoveries. I chose to further explore my scientific interests and develop my research skills this summer in the Medical Training Program at LLU's CHDMM.

I graduated with a Bachelor's degree in Biology from Southern Adventist University in Collegedale, Tennessee, in May 2026. The classes and labs at SAU gave me a strong foundation in scientific knowledge and laboratory skills. This July, I will be starting my first year as a medical student at Loma Linda University School of Medicine. Pursuing a career in medicine has always been my aspiration, and I hope to be able to serve my community well as a healthcare provider and a scientist. It is my goal to be a physician who is knowledgeable in my field and excels in patient care. In the future, I aim to be involved in both the clinical aspect of medicine as well as in the research field, contributing to the discovery and development of treatments in the basic sciences.

This summer, I am working in Dr. Alfonso Duran's lab, with the added guidance of Dr. Viet Hoang Dinh. Our research focuses on better understanding the novel process of ferroptosis, a form of non-apoptotic regulated cell death. Ferroptosis-related stress may be involved in the development of neuropathic pain and age-related diseases. These are prevalent illnesses in the population, so the research of preventative treatments is crucial. I have enjoyed learning about this new topic and the impacts that our research could have on the community. I am also thankful for the opportunity to better develop my scientific thinking and laboratory skills under the CHDMM mentors. I would like to offer my deepest gratitude to Dr. Duran for his support, guidance, and mentorship, as well as to Dr. Dinh for his patient and thorough teaching during my time in this program.

EFFECTS OF PALMITIC ACID AND RSL3 ON FERROPTOSIS IN IMMORTALIZED SCHWANN CELLS AND THE PROTECTIVE POTENTIAL OF J147

Melissa Lee[1], Viet Hoang Dinh[1], Francis Zamora[1], Caroline Coronado[1], Salvador Soriano[1,2], Alfonso Duran[1,2]

[1]Center for Health Disparities and Molecular Medicine, School of Medicine, Loma Linda University, Loma Linda, CA, [2]Department of Pathology and Human Anatomy, School of Medicine, Loma Linda University, Loma Linda, CA

Long-term exposure to palmitic acid (PA), a saturated fatty acid, increases the susceptibility of Schwann cells to ferroptosis, a form of cell death driven by iron-dependent lipid peroxidation and failure of GPX4-dependent lipid-peroxide defense. In patients with metabolic diseases such as Type 2 diabetes, ferroptosis is thought to contribute to painful diabetic neuropathy (pDN) through damage to peripheral nerve cells. The aim of this study was to assess the effects of PA (to model chronic lipid overload) and RSL3 (a GPX4 inhibitor used to induce ferroptosis) in immortalized Schwann cells (iSC) and test the ability of the experimental drug J147 to protect against ferroptosis-associated injury.

iSC were treated with sublethal doses of PA and RSL3 to induce ferroptotic stress. Cell viability was measured using the WST1 assay, lipid peroxidation was measured using C11-BODIPY, and levels of GPX4, the enzyme that detoxifies oxidized membrane lipids, were assessed by western blot techniques. Cells were then treated with J147 to assess the drug's ability to rescue cells from ferroptosis-associated stress in comparison to Ferrostatin-1, a known ferroptosis inhibitor (positive control).

Our results show that sublethal doses of PA reduces GPX4 levels and increases lipid peroxidation in iSC, consistent with an early, sublethal ferroptotic-stress state; RSL3 produced comparable increases in lipid peroxidation as a positive ferroptosis control. Results on the potential of J147 to reverse cellular damage were inconclusive, and additional trials will be needed to optimize dosages and method of treatment.

Our work indicates that long-term exposure to PA lowers GPX4-dependent defenses and promotes lipid peroxidation in Schwann cells. By capturing this injury at an early, sublethal stage, this cell model offers a system for studying whether ferroptotic stress can be detected and reversed before irreversible nerve damage occurs in metabolic disease.

CHDMM Graduate Students

ADELAIDE MAKAMURE

CHDMM GRADUATE STUDENT PARTICIPANT 2026

Adelaide Makamure is a second-year PhD student at Loma Linda University and a young researcher focusing on prostate cancer. Her research aims to deepen the understanding of prostate cancer biology and potentially inform future therapeutic strategies. Born and raised in Zimbabwe, she completed high school there before earning a BSc in Biology: Biomedical option from the University of Eastern Africa, Baraton (Kenya) in 2021. She was on the dean's list in 2018 and 2019 for academic excellence. Adelaide is driven by excellence, showing motivation, discipline, and commitment to advancing cancer biology. Outside of research, she enjoys music and is often found humming a tune.

LOW TUMOR EXPRESSION OF PSIP1/LEDGF/P75 IS MORE PREVALENT IN PROSTATE CANCER PATIENTS OF AFRICAN AND EAST ASIAN DESCENT AND ASSOCIATED WITH IMPROVED IMMUNOTHERAPY OUTCOMES POSSIBLY VIA INCREASED PYROPTOTIC VULNERABILITY

Adelaide Makamure1,2, Yasmine Baca3, Pedro T. Ochoa1,2, Alfonso Duran1,2, Andrew Elliott3, Rana R. McKay4, Neeraj Agarwal5, Janani Nagasubramanya1,2, Sharan Bir1,2, Zhong Chen2, Issac Kremsky2, Charles Wang2, Carlos A. Casiano1,2

1Center for Health Disparities and Molecular Medicine, 2Department of Basic Sciences, Loma Linda University School of Medicine, Loma Linda, CA; 3Caris Life Sciences, Phoenix, AZ; 4University of California, San Diego, CA; 5University of Utah, Salt Lake City, UT

Lens epithelium derived growth factor/p75 (LEDGF/p75), encoded by the PSIP1 gene, drives prostate cancer (PCa) drug cross-resistance by promoting DNA damage repair and stress survival transcription. Its depletion induces genomic instability, likely leading to inflammatory responses. We hypothesized that PSIP1 expression levels in prostate tumors may differ among racial groups and influence immune pathways associated with immunotherapy resistance. 9,685 prostate tumors that underwent DNA- and RNA-sequencing at Caris Life Sciences were stratified by PSIP1 expression quartiles (Q1-low vs Q4-high). Infiltrating immune cell fractions of the tumor microenvironment (TME) were inferred by deconvolution of tumor RNA expression using QuantiSEQ. Overall survival (OS) from time of treatment start to last contact was estimated using the Cox proportional hazards model to calculate hazard ratio (HR) and log-rank tests to calculate p-values. Analysis of PSIP1 tumor expression by genomic ancestry revealed lower median expression in PCa patients of African and East Asian descent compared to other groups. Low PSIP1 expression was associated with lower tumor infiltration of M2 macrophages and regulatory T cells. Single cell RNA-seq of prostate tumors revealed that low PSIP1 expression was associated with infiltration of T and NK cells. Low PSIP1 expression was also associated with better OS in PCa patients, including those receiving immune checkpoint inhibition (ICI) immunotherapy, androgen receptor signaling inhibitors or taxanes. In taxane-resistant PCa cells that underwent PSIP1 silencing, we identified via RNA-seq 970 differentially expressed genes and significantly enriched pathways related to inflammation, pyroptosis, and lymphocyte function. Western blotting revealed upregulation of pyroptosis-associated proteins IL18 and gasdermins D and E upon PSIP1 silencing. Cleavage of these gasdermins, which leads to pyroptotic IL18 release, was also detected. Our results suggest that low PSIP1 expression is associated with favorable immune TME linked to increased pyroptotic vulnerability and improved OS in patients receiving ICI and other therapies. The relatively low PSIP1 tumor expression in PCa patients of African ancestry might be consistent with reports that this patient group shows better early responses to standard therapies compared to European Americans.

JULIO SIERRA

CHDMM GRADUATE STUDENT PARTICIPANT 2026

My parents immigrated from Mexico in search of better opportunities for themselves and, in turn, for me. Watching them navigate the challenges of acculturation taught me the value of education and hard work and instilled in me a determination to overcome the barriers they once faced. Driven by a love of learning and problem-solving, I pursued undergraduate studies in biomedical engineering, where I gained hands-on experience developing systems for use in research. That early experience sparked my interest in pursuing a career in basic science.

I am now a fourth-year Ph.D. candidate in the Neuroscience, Systems Biology, and Bioengineering program at Loma Linda University. As a first-generation college student, I understand firsthand the difficulty of navigating higher education without a support system. The IMSD program in the Center for Health Disparities and Molecular Medicine has provided a sense of community and allowed me to serve as a peer mentor, encouraging students to pursue higher education and strengthen their interest in biomedical research.

I am grateful for the mentorship and support of Dr. Johnny Figueroa, Dr. Timothy Simon, and many others for helping me develop as a neuroscientist, as well as my wonderful mentees for the hard work and determination they demonstrate in the lab. I expect to continue contributing to the field by investigating how early environmental factors affect neurodevelopment and behavior. I am excited to continue driving this research forward and help address this critical health disparities issue.

GUT MICROBIOME DEPLETION PERTURBS ENDOCRINE–GUT–BRAIN HOMEOSTASIS DURING ADOLESCENCE

Katherine Granados, Julio Sierra, Darine Abu Hilal, Leanne Mercado Allison Rhee, Jo-Wen Liu, Johnny, D. Figueroa

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

The gut microbiome plays a central role in regulating endocrine, metabolic, and neurobehavioral signaling. During adolescence, maturation of the gut microbiota coincides with major hormonal changes that influence brain development, yet the effects of microbiome disruption on endocrine signaling remain poorly understood. This study investigated how antibiotic-induced depletion of the microbiome alters fecal hormone profiles in female Lewis rats. Animals received a broad spectrum antibiotic cocktail in drinking water during adolescence and were maintained on either a Western diet or standard chow. Fecal samples were collected during adolescence (PND63) and adulthood (PND81). Enzyme immunoassays were used to quantify corticosterone, estrogen, progesterone, and serotonin as biomarkers of stress, endocrine function, and gut-brain communication. Antibiotic-treated animals exhibited significantly lower concentrations of corticosterone, estrogen, progesterone, and serotonin compared with controls, indicating that microbiome depletion disrupts multiple endocrine signaling pathways. Ongoing analyses are determining how these hormonal alterations relate to microbial composition and behavioral outcomes. Together, these findings suggest that perturbation of the adolescent gut microbiome produces persistent changes in endocrine signaling that may influence long-term gut-brain communication.

KATHLEEN ESCOBAR ACUÑA
CHDMM GRADUATE STUDENT PARTICIPANT 2026

My journey in science began in a rural area of Chile, where growing up in a high school teachers family taught me the value of hard work and education. After graduating with honors on Bioengineering and a Master of Science in Neurobiology from the University of Concepción, I explored metabolic regulation, nutrient transport mechanisms, and Vitamin C changes associated with oxidative stress in neurodegeneration.

In September 2023, I moved forward in my academic career by joining the PhD program in Neuroscience at Loma Linda University. Through research rotations—such as evaluating hippocampal phagocytic microglial markers in stress-induced models in Dr. Johnny Figueroa's lab and implementing human cell culture models to study insulin resistance in Dr. Konrad Talbot's lab—I built a strong foundation before joining Dr. Carlos Casiano's laboratory to study cellular metabolic pathways and molecular targets in cancer.

Though I faced a personal tragedy that required a leave of absence from October 2024 to December 2025, I chose to return to Loma Linda University because I found a community where I can pursue rigorous science while integrating my faith in God. Alongside my research, I am deeply committed to service, participating in humanitarian missions to Mexico with ALAS and volunteering at the LLU Open House. I am excited for what lies ahead as I continue my path in research and academic inquiry.

CHARACTERIZING THE EXPRESSION AND LOCALIZATION OF ENO1 AND ENO2 IN GLIOBLASTOMA CELLS

Jocelyn Chow, Kathleen Escobar-Acuña, Lemti Nyirendah, Krystal Santiago, Nathan R. Wall, Carlos A. Casiano

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

Glioblastoma (GBM) is a highly aggressive glioma that presents with poor patient outcomes due to its rapid recurrence and treatment resistant nature. Despite therapeutic interventions, average survival rates for patients range from 12–15 months, with five-year survival rate of 6.8%. Recent studies explored glycolytic metabolic reprogramming in GBM, implicating alpha-enolase (ENO1) overexpression in glioma progression. ENO1 and its neuron-specific isoform ENO2 have been studied as promising biomarkers in other malignancies such as prostate cancer (PCa), underscoring their value as pan-cancer therapeutic targets. We hypothesized that, like in PCa, the intracellular expression and surface abundance of ENO1 are higher than those of ENO2 in GBM. Neuroendocrine prostate cancer (NEPC) and GBM cell lines were obtained commercially through ATCC. NEPC cells served as positive controls due to their aggressive nature and elevated ENO1 and ENO2 expression. For Western blotting analysis, cell lysates were incubated with specific antibodies to ENO1 and ENO2. Flow cytometry was performed to characterize the intracellular and surface abundance of ENO1 and ENO2. The Cancer Genome Atlas (TCGA) GBM dataset was mined to construct Kaplan-Meier survival curves. The protein expression levels of ENO1 and ENO2 were similar in GBM and NEPC cells. The TCGA GBM dataset revealed that patients with high ENO1 (n=18) and ENO2 (n=17) expression had decreased survival rates within 12 months (log-rank p=0.0691 and p=0.0019, respectively). Collectively, our preliminary data suggest that ENO1 and ENO2 are highly expressed in GBM cells and associated with worse patient outcomes. We anticipate that ENO1 and ENO2 are present on the GBM cell surface, where they could serve as potential therapeutic targets.

KRYSTAL SANTIAGO

CHDMM GRADUATE STUDENT PARTICIPANT 2026

I was born in Mayagüez, Puerto Rico, where I learned early on the value of hard work and education. These experiences shaped my commitment to academic excellence and led me to pursue a career in scientific research. I earned my B.S. in Industrial Microbiology from the University of Puerto Rico and am currently a senior PhD student working toward my doctorate in cancer biology. My training has been driven by an interest in contributing to research that addresses health disparities. To support this goal, I chose Dr. Casiano and Dr. Almaguel as co-advisors, whose work focuses on diseases that disproportionately affect underrepresented populations. My research examines the role of Enolase-1 and Enolase-2 in prostate cancer progression, particularly their effects on cell proliferation, migration, invasion, and metastasis. I also investigate ENO1 antibody-based targeting to determine how it influences tumor cell behavior.

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.

NATHALY P. MANRIQUE
CHDMM GRADUATE STUDENT PARTICIPANT 2026

I developed an early fascination with biology and the molecular mechanisms underlying human disease, which inspired my pursuit of a career in cancer research. That passion led me to earn a Bachelor of Science in Biology with an emphasis in Biomedical Sciences from Andrews University and a Master of Science in Biomedical Sciences from Charles R. Drew University of Medicine and Science. I am currently a second-year Ph.D. student in the Cancer, Developmental, and Regenerative Biology program at Loma Linda University, where I conduct research in the Casiano Laboratory.

My current research focuses on the molecular mechanisms underlying therapeutic resistance in advanced prostate cancer. Specifically, I investigate how glucocorticoid receptor (GR)-mediated transcriptional signaling influences the LEDGF/p75 interactome in enzalutamide-resistant prostate cancer cells. Although androgen receptor signaling inhibitors have significantly improved outcomes for patients with advanced prostate cancer, the emergence of resistance remains a major clinical obstacle. My work aims to elucidate how stress-adaptive transcriptional networks promote tumor cell survival and therapeutic resistance, with the broader goal of identifying molecular vulnerabilities that could be leveraged to develop more effective treatment strategies.

Outside of the laboratory, I enjoy traveling and spending time at the beach. I also enjoy connecting with my community through volunteer work at Loma Linda University Church, where I serve on Sabbath mornings. As I continue my scientific training, I hope to contribute to research that deepens our understanding of cancer biology while accelerating the translation of laboratory discoveries into improved therapies for patients.

EXPRESSION OF THE LEDGF/P75 INTEGRASE BINDING DOMAIN INTERACTOME IN ENZALUTAMIDE-RESISTANT PROSTATE CANCER CELLS

Andrea Latorre, Nathaly Manrique, Pedro Ochoa, Yasmine Baca, Carlos A. Casiano

Center for Health Disparities and Molecular Medicine, School of Medicine, Loma Linda University, Loma Linda, CA; and Caris Life Sciences, Phoenix, AZ

Prostate cancer (PCa) is the most diagnosed cancer and the second leading cause of male cancer deaths in the U.S. Although androgen receptor signaling inhibitors (ARSIs) such as enzalutamide, have improved the treatment of advanced PCa, therapeutic resistance inevitably develops leading to cross-resistance to docetaxel-based chemotherapy. A key mechanism underlying this cross-resistance is glucocorticoid receptor (GR) bypass signaling, in which GR compensates for AR inhibition by activating transcriptional programs that promote tumor cell survival. GR signaling in PCa cells promotes the expression of LEDGF/p75, a transcriptional co-activator encoded by the PSIP1 gene that promotes the expression of genes involved in cellular stress survival, DNA repair, and therapeutic resistance. The C-terminal integrase-binding domain (IBD) of LEDGF/p75 functions as a protein interaction hub that recruits multiple transcriptional regulators to active chromatin. This LEDGF/p75 interactome is upregulated in docetaxel-resistant PCa cells and contributes to PCa aggressiveness. PCa patient data from the Caris Life Sciences' clinico-genomic database showed that the expression of genes encoding members of the GR-LEDGF/p75 transcriptional network was positively associated with PSIP1 expression in prostate tumors. We hypothesized that GR also activates this network in enzalutamide-resistant cells, leading to coordinated expression of multiple LEDGF/p75-associated transcriptional regulators that promote therapeutic cross-resistance. We performed Western blotting analyses of protein lysates from enzalutamide-sensitive and -resistant LNCaP cell lines to confirm concurrent GR and LEDGF/p75 upregulation. Lysates were subsequently evaluated for the expression of additional LEDGF/p75 IBD-interacting proteins to determine if they are likewise upregulated in enzalutamide-resistant cells. We anticipate these studies will determine whether the upregulation of the LEDGF/p75 IBD-interactome represents a molecular feature of both ARSI and taxane resistance, further defining its contribution to PCa drug cross-resistance.

SURGE – Student Undergraduate Research Generating Evidence

ABEL MASSEY
SURGE PARTICIPANT 2026

As a senior nursing student at Loma Linda University School of Nursing, I have had the opportunity to connect with many patients and interact with a variety of healthcare professionals. If my time as a nursing student has taught me anything, it is that everyone has a deep desire for compassionate care. To play a small part in the translation of clinical research into actionable, compassionate care is more than a job or an experience; it is a calling. Under Dr. Lisa Roberts and her team, I seek to address healthcare disparities relating to maternal health and Sickle Cell Disease care. What excites me most about this project is getting to make the historically vulnerable and marginalized population of those who have Sickle Cell Disease feel seen and better understood.

Parallel to my academic pursuits, I am a professional musician. I spend many of my weekends serving various church communities as a drummer. Interfacing with music has given me the opportunity to connect with people from all walks of life; something that I will forever cherish. Connection at the emotional and spiritual levels has sparked my desire to deepen my understanding of science and biology. I hope to one day combine my gift of connection with my desire for understanding to heal patients holistically.

LITERACY OF SICKLE CELL DISEASE/TRAIT AND ITS IMPACT ON YOUNG ADULT DATING

Abel Massey, Emma Karpov, Crystal Lee, Ashra Tugung, Samuel Habimana, Fatima Alramdhan, Carlene Fider, Susanne Montgomery, and Lisa Roberts

School of Nursing, Loma Linda University, Loma Linda, CA

Sickle Cell Disease and Sickle Cell Trait (SCD/T) are inherited hematological disorders characterized by abnormally shaped red blood cells that impair microcirculation and oxygen delivery. Beyond clinical management, future planning and navigating the dating landscape represent increasingly complex, yet understudied, psychosocial challenges for individuals living with SCD/T. This study aimed to evaluate public knowledge of SCD/T and determine how baseline awareness influences young adults' attitudes, perceptions, and willingness regarding dating individuals with SCD/T. A mixed-methods design was executed. First, a cross-sectional quantitative pilot survey was administered to African American/Black identifying young adults aged 18–30 without SCD/T. Second, qualitative depth was established via three semi-structured key informant interviews. The interview inclusion criteria were expanded to include all racial backgrounds to capture a comprehensive pool of prospective partners. Quantitative analysis revealed a mean knowledge score of 5.76 out of. Most respondents demonstrated deficits concerning SCT and the hereditary transmission patterns of SCD. Statistically significant associations ($p < .05$) emerged between total knowledge scores and demographic variables including gender, educational attainment, housing status, and food insecurity, as well as partner preferences like valuing intelligence and dependability. While housing status served as a significant predictor of baseline knowledge, relational and psychosocial factors did not predict total knowledge scores. Qualitative coding revealed four primary themes: Experiential/Clinical SCD/T Knowledge Gaps, Perceived Frequent Healthcare Interaction of SCD/T Individuals, Medical Labeling as a Chronic Illness Psychosocial Stressor, and The Desire to Understand SCD/T Complexity to Enable Informed Decision Making. Low public literacy surrounding SCD/T inheritance and traits highlights a need for targeted public health education. Addressing these knowledge deficits is critical to support informed reproductive and relational choices among young adults.

ASHRA TUGUNG
SURGE PARTICIPANT 2026

As a Filipino immigrant, I grew up surrounded by nurses whose compassion and dedication shaped my understanding of what it means to care for others, making a healthcare career feel inevitable. While I always knew I wanted to serve others, my path to healthcare unfolded in unexpected ways, revealing a passion for both research and nursing.

I earned a Bachelor of Science in Microbiology, Immunology, and Molecular Genetics with a minor in Global Health from UCLA. Returning to the Inland Empire led me to the Loma Linda University Perinatal Institute, where I work as a Certified Clinical Research Professional, contributing to maternal and neonatal research. While my undergraduate studies sparked a love for laboratory science, clinical research taught me that behind every research question is a person, a family, and a story.

Currently, I am pursuing my Entry-Level Master of Science in Nursing at Loma Linda University with a concentration in Population Health, reflecting my commitment to advancing health equity through nursing and research. This summer, I have the privilege of participating in the SURGE program under Dr. Lisa Roberts, whose research focuses on improving health outcomes for individuals with sickle cell disease and trait (SCD/T). My project explores how perceptions of SCD/T may influence romantic relationship decisions. Looking ahead, I hope to build a career that brings together nursing, research, advocacy, and education to advance evidence-based care, improve patient outcomes, and promote health equity. My sincere thanks to Dr. Lisa Roberts and the SURGE team for their mentorship.

BEHAVIORAL INTENTIONS FOLLOWING DISCLOSURE OF SICKLE CELL DISEASE: A MIXED-METHODS STUDY OF YOUNG ADULTS' DATING PERSPECTIVES

Ashra Tugung, Crystal Lee, Abel Massey, Emma Karpov, Samuel Habimana, Fatimah Alramdhan, Carlene Fider, Susanne Montgomery, Lisa Roberts

School of Nursing, Loma Linda University, Loma Linda, CA

Sickle cell disease (SCD) is a chronic inherited blood disorder that disproportionately affects individuals of African descent and presents unique challenges for romantic relationships because of its medical, genetic, and psychosocial implications. Disclosure of one's chronic illness risks may influence relationship formation, and little is known about how young adults respond when a potential dating partner discloses having SCD. The purpose of this mixed-methods study was to explore young adults' openness to dating individuals with SCD and examine factors associated with intentions following disclosure of SCD by a potential dating partner. Survey data were collected from 106 African Americans (18-30 years) without SCD or trait using a vignette assessing behavioral intentions following a dating partner's disclosure of SCD. Interviews with three young adults further explored perspectives on dating individuals with SCD. Quantitative data were analyzed using descriptive and nonparametric statistics, and qualitative data were analyzed using thematic analysis. Among the 90 vignette respondents, greater openness to dating someone with SCD was associated with a lower likelihood of leaving the date early ($\rho = -.394$, $p < .001$), not contacting the individual again ($\rho = -.515$, $p < .001$), or choosing to remain friends only ($\rho = -.330$, $p = .001$), and with a greater likelihood of going on another date ($\rho = .493$, $p < .001$). Perceived emotional support needs, social stigma, frequent medical care, and selected partner values also affected dating openness. Qualitative findings suggested that knowledge gaps, concerns about disclosure, anticipated caregiving responsibilities, and personal relationship values shaped participants' responses to potential partners with SCD. These findings highlight the importance of improving young people's understanding of SCD and reducing stigma surrounding disclosure to support informed and equitable relationship decisions.

CRYSTAL LEE
SURGE PARTICIPANT 2026

Pursuing nursing has allowed me to combine my love of science and serving others into one meaningful career. I am currently an Entry-Level Master of Science in Nursing student at Loma Linda University (LLU) with a concentration in Population Health, and I earned my BS in Biology from UCR. As a first-generation graduate student, every milestone has been especially meaningful and has strengthened my determination to make a difference in healthcare. Over the past eight years, I have worked as a certified pharmacy technician and now work as a nursing assistant here at LLU. These experiences have demonstrated the importance of compassion, teamwork, and treating every patient with dignity.

This summer, I had the privilege of participating in the SURGE program in the School of Nursing under the mentorship of Dr. Lisa Roberts, contributing to research focused on sickle cell disease and family planning. Watching the research process unfold has deepened my appreciation for the role nurses play in advancing research and improving the lives of patients and families. It has reinforced my desire to help bridge the gap between research, clinical practice, and community health.

Outside of healthcare, you'll most likely find me spending time with family and friends, getting creative through crafting, or spoiling my six cats. I hope to build a career that blends compassionate patient care, research, and community outreach while advocating for underserved communities. I am sincerely grateful to Dr. Lisa Roberts and the SURGE team for helping me take another step toward that goal.

PREFERRED ROMANTIC PARTNER CHARACTERISTICS AND OPENNESS TO DATING INDIVIDUALS WITH SICKLE CELL DISEASE/TRAIT

Crystal Lee, Ashra Tugung, Abel Massey, Emma Karpov, Fatimah Alramdhan, Samuel Habimana, Carlene Fider, Susanne Montgomery, Lisa Roberts

School of Nursing, Loma Linda University, Loma Linda, CA

Romantic relationships and family planning are important aspects of young adulthood, yet little is known about the partner characteristics that influence willingness to pursue a relationship with someone living with sickle cell disease (SCD). This mixed-methods study examined preferred romantic partner characteristics associated with openness to dating someone with SCD among young adults. A survey of 106 African American adults aged 18-30 years assessed demographic characteristics, preferred partner characteristics, knowledge of SCD, and willingness to date someone with SCD. Quantitative data were analyzed using descriptive statistics, Spearman correlations, and three semi-structured interviews were analyzed thematically to provide context for the survey findings. Spearman correlations showed that valuing a dependable character was associated with greater openness to dating someone with SCD ($\rho = .238$, $p = .023$), whereas greater emphasis on similar hobbies or interests ($\rho = -.330$, $p = .001$), similar dietary preferences ($\rho = -.228$, $p = .031$), and being a good cook and housekeeper ($\rho = -.270$, $p = .010$) were associated with lower openness. Interview findings reinforced the importance of trust, shared values, family planning, and knowledge of SCD when considering a potential romantic partner, highlighting how these factors shaped participants' attitudes toward dating someone living with the condition. Together, these findings suggest that interpersonal qualities may play a greater role in dating decisions than physical or socioeconomic characteristics. Furthermore, these findings suggest that relationship values and future family considerations influence willingness to pursue relationships with individuals living with SCD. Understanding these preferences may help inform culturally responsive educational and counseling interventions that address misconceptions, support informed reproductive decision-making, and promote more inclusive attitudes toward individuals living with SCD.

EMMA KARPOV

SURGE PARTICIPANT 2026

Growing up with parents who were both involved in research in their respective fields inspired a lifelong curiosity about the world around me and motivated me to pursue research within the nursing discipline. Throughout high school and college, I have found a passion for helping vulnerable populations. Now, as a senior at Loma Linda University School of Nursing, this passion has grown into a strong interest in neonatology and women's health.

Before beginning nursing school, I completed my prerequisite coursework at Walla Walla University. I have maintained a 4.00 GPA through both my prerequisite and nursing education and was honored to receive the Loma Linda University Alumni Merit Scholarship this year. To continue connecting with vulnerable populations through nursing school, I serve as the Co-Community Vice President for our Association of Student Nurses and have volunteered more than 100 hours in the Cardiovascular Intensive Care Unit.

This summer, I have had the opportunity to work under Dr. Lisa Roberts' HRSA research grant, which addresses maternal health disparities with a focus on sickle cell disease. My research studying the intersection of sickle cell disease, relationships, and family planning has reinforced my passion for women's health and demonstrated how nursing research can shape more equitable care for patients and families.

I would like to sincerely thank Dr. Lisa Roberts and the SURGE team for their mentorship and for inspiring me to continue integrating research into my future nursing career.

PERCEIVED RELATIONSHIP CHALLENGES WHEN CONSIDERING DATING PARTNERS WITH SICKLE CELL DISEASE/TRAIT

Emma Karpov, Crystal Lee, Abel Massey, Ashra Tugung, Fatimah Alramadahn, Samuel Habimana, Susanne Montgomery, Carlene Fider, Lisa Roberts

School of Nursing, Loma Linda University, Loma Linda, CA

Sickle cell disease (SCD) is an inherited genetic blood disorder, whereas individuals with sickle cell trait (SCT) are carriers. Because carrier status dictates reproductive risk, SCD/T presents unique health and genetic considerations. Therefore, romantic relationship decisions may involve unique personal and genetic family-planning considerations. Little is known about perceptions of young adults who may consider having a romantic relationship with persons living with SCD/T. This mixed-methods study explored relationship challenges these young adults perceive. Survey data (N=106 African Americans, aged 18-30) and semi-structured interviews (N=3) were analyzed. Quantitative analyses evaluated variables related to openness to dating someone with SCD/T and which challenges seemed most important when making such decisions. Thematic analysis identified key factors influencing relationship decisions. Descriptive statistics revealed that genetic risks to children, future child health impacts, and family planning were the three most impactful relationship challenges, unaffected by demographics. Notably, partner characteristics, such as desire for a home and children, significantly correlated with family planning concerns (r = .32, p = .002) and future child health impacts (r = .23, p = .028). Desire for a good financial prospect correlated with all three challenges: genetic risks to children (r = .29, p = .005), future child health impacts (r = .22, p = .037), and family planning (r = .34, p < .001). Qualitative interviews revealed concerns about reproductive risk, shaped by cultural and family expectations, preferences for early disclosure of SCD status, and anticipated emotional and lifestyle demands. Findings suggest that reproductive concerns, family planning, and financial stability are central considerations shaping perceptions of romantic relationships involving individuals with SCD/T. Findings will inform the development of interventions to improve public education and genetic counseling, and guide future studies.

Summer Undergraduate Research Fellowship (SURF)

AIDEN RILEY
SURF PARTICIPANT 2026

I am extremely grateful to have been accepted into the SURF program for the summer of 2026. I am currently a rising junior at Baylor University in Waco, Texas, where I am studying cell and molecular biology with the goal of attending medical school. During my time at Baylor, I have been involved in Greek life, volunteered as an EMT, and served in my church's children's ministry every Sunday. Outside of academics, I enjoy watching and playing a variety of sports, with volleyball being my favorite. Through sports, I have developed valuable skills such as teamwork, communication, and trust. This summer, I have had the privilege of conducting research in Dr. Christian Hurtz's lab. Our research focuses on understanding the pathogenesis of high-risk leukemia subtypes, primarily Ph-like ALL, and identifying new treatment strategies to improve clinical outcomes for those diagnosed. The aspect of this project that I have found most interesting is treating cell lines with varying concentrations of chemotherapies and other drugs, as it allows me to quantify experimental results and directly observe the effects of potential treatments. Participating in the SURF program has strengthened my understanding of cancer biology, teamwork, and what it means to conduct rigorous, testable research, while reinforcing my passion for medicine. I am especially thankful to Dr. Hurtz for his guidance, patience, and mentorship throughout this experience.

SELECTIVE BRD9 DEGRADATION REDUCES THE VIABILITY OF PH-LIKE B-CELL ACUTE LYMPHOBLASTIC LEUKEMIA CELLS

Aiden Riley, V. S. S. Abhinav Ayyadevara, Shreya Patil, Christian Hurtz

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

Ph-Like ALL is a high-risk leukemia subtype associated with frequent relapse and inferior clinical outcomes, highlighting the need to identify novel therapeutic vulnerabilities. BRG1, encoded by SMARCA4, is an ATPase subunit of the SWI/SNF chromatin-remodeling complexes and plays an important role in early B-cell development. We previously found that higher BRG1 expression was associated with poor clinical outcomes in Ph-like B-ALL patients and that Ph-like B-ALL cells were sensitive to BRG1/BRM inhibition. However, BRG1 and BRM provide ATPase activity to cBAF, pBAF, and ncBAF, and broad ATPase inhibition may disrupt multiple SWI/SNF assemblies. Clinical evaluation of broad BRG1/BRM inhibition has revealed tolerability challenges, emphasizing the importance of more complex-selective approaches. To identify a selective SWI/SNF dependency in Ph-like B-ALL, we compared the expression of genes associated with cBAF, PBAF, and ncBAF across B-ALL subtypes. PBAF-associated gene expression differed significantly among subtypes and was lowest in Ph-like B-ALL compared to other subtypes. cBAF-associated expression showed a nonsignificant trend toward lower expression in Ph-like B-ALL. In contrast, ncBAF-associated expression was maintained in Ph-like B-ALL at levels comparable to those observed in other B-ALL subtypes. Based on these findings, we hypothesized that Ph-like B-ALL cells may retain a dependency on ncBAF. BRD9 is a defining component of ncBAF that is not incorporated into cBAF or PBAF. We therefore evaluated the activity of FHD-609, a selective BRD9 degrader, in murine Ph-like ALL cells. Treatment with FHD-609 reduced leukemia-cell viability, indicating that selective disruption of BRD9-containing ncBAF complexes impairs the growth or survival of Ph-like ALL cells. These findings identify BRD9 as a potential therapeutic vulnerability in Ph-like B-ALL and support further investigation of ncBAF-selective degradation as an alternative to broad BRG1/BRM inhibition.

FATIMAH AHMED
SURF PARTICIPANT 2026

The more I learn about biomedical engineering, the more I am inspired by its potential to improve lives through innovation. What began as a curiosity about science has grown into a passion for developing technologies that can address some of medicine's most challenging problems. Research experience has strengthened my desire to contribute to discoveries that have a meaningful impact on patient care.

I am a second-year Bioengineering student at the University of California, Riverside. Before joining the SURF Program, I worked with molecular biology techniques and presented a research poster summarizing my work. I also had the opportunity to publish a peer-reviewed review article on what I had learned from my experience. This summer, I am conducting research in Dr. Rameshwar Patil's laboratory, where I investigate iron oxide nanoparticles as potential theranostic agents for glioblastoma treatment using boron neutron capture therapy. I have especially enjoyed learning how engineering, chemistry, and biology intersect to develop more targeted approaches to cancer treatment and seeing how collaborative research can transform ideas into meaningful scientific progress.

As I continue my undergraduate education, I hope to build upon the strong foundation this program has provided and attend graduate school to further my training as a researcher. I am sincerely grateful to Dr. Rameshwar Patil and everyone in the laboratory for their mentorship, encouragement, and the opportunity to be part of such an inspiring research experience.

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

ENGINEERING THERANOSTIC IRON OXIDE NANOPARTICLES FOR TARGETED BORON DELIVERY IN GLIOBLASTOMA

Fatimah Ahmed[1], Sanjay Yadav[1], Kevin Nick[2], Rameshwar Patil[1*]

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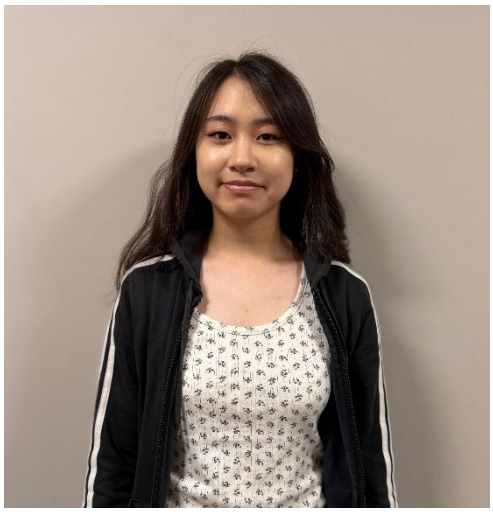
Glioblastoma (GBM) is the most aggressive primary brain tumor and remains difficult to treat due to its rapid progression, therapeutic resistance, and the restrictive nature of the blood-brain barrier (BBB). GBM accounts for approximately 52.2% of all malignant brain and central nervous system tumors. Current treatment strategies, including surgical resection, radiation therapy, and chemotherapy, provide limited long-term survival, highlighting the need for more effective targeted therapies. Boron Neutron Capture Therapy (BNCT) is a promising treatment approach that selectively destroys tumor cells through the accumulation of boron-10 followed by irradiation with thermal neutrons. This nuclear reaction produces high-linear energy transfer (LET) alpha particles and lithium-7 nuclei, resulting in localized tumor cell death while minimizing damage to surrounding healthy tissue. The effectiveness of BNCT relies on the selective delivery and sufficient accumulation of boron within tumor cells. In this study, iron oxide nanoparticles were engineered and synthesized as a multifunctional theranostic platform for targeted boron delivery. The iron oxide core provides magnetic resonance imaging (MRI) contrast capability, while a dopamine coating serves as a surface functionalization layer that enables conjugation of both a boron-containing compound and a targeting ligand. We are utilizing the Angiopep-2 (Ap2) peptide which promotes transport across the BBB, enhancing tumor cell targeting. The synthesized nanoparticles are characterized to evaluate their morphology, colloidal stability, magnetic properties, and surface functionalization. Future studies will investigate ways to improve boron loading capacity and target specificity in GBM tumor cells, followed by evaluation of BBB penetration, tumor accumulation, MRI imaging capability, and therapeutic efficacy after neutron irradiation. The development of this multifunctional nanoparticle platform has the potential to improve targeted boron delivery, enhance treatment precision, and advance BNCT as a safer and more effective therapeutic strategy for the treatment of GBM.

GABRIELLE KIM
SURF PARTICIPANT 2026



Before joining SURF, my understanding of biomedical research came primarily through reading and coursework, but working in Dr. Oberg's laboratory has allowed me to experience it firsthand and see what it truly involves. The experience has made me realize how finely tuned limb development is, and how there is so much to be understood. It's interesting to learn about the effects of specific manipulations on an embryo and how the results may be interpreted.

What I value about research is that it bridges the gap between theory and reality. In many cases the two are taught separately but scientific research stitches the two together, which is quite a fulfilling experience.

I am an incoming junior at Southern Adventist University and majoring in Biophysics. I'm passionate about math and physics and I enjoy being involved in my school's physics department research. I have also enjoyed volunteering for the LLU Hospital during the past few years, and I have been on a few medical mission trips and am always inspired by the way physicians can use their knowledge to remedy illnesses. I hope to pursue an MD/PhD so that I can contribute to research that uses mechanistic understanding of human biology to create tools that can be used in clinical practice.

I'm deeply grateful to everyone in Dr. Kerby Oberg's lab for giving me the opportunity to learn a lot of valuable new information, and for their patience and mentorship in teaching me to be an active participant in the research process.

AN *LHX9* ASSOCIATED *CIS*-REGULATORY MODULE IS ACTIVE IN ANTERIOR AND POSTERIOR REGIONS OF DEVELOPING CHICKEN LIMB BUDS

Gabrielle C. Kim, Jeanyoung Kim, Charmaine U. Pira, Kerby C. Oberg
Department of Pathology and Human Anatomy, School of Medicine, Loma Linda University, Loma Linda, CA

Lhx9 is a LIM homeodomain transcription factor that is responsible for regulating gene expression during development. A recent clinical report found *LHX9* disruption resulted in hypoplastic/absent thumbs and great toes in a child, suggesting a critical role in limb development. The expression of *Lhx9* is accentuated in the anterior (radial) margin of the limb. Interestingly, in two model systems of limb development, chickens and mice, the expression pattern differs with murine expression also extending into the distal-posterior (pinky) region. *Cis*-regulatory modules (CRMs) are DNA sequences which control spatiotemporal gene expression, and *Lhx9*-associated CRMs are likely responsible for the differential *Lhx9* expression pattern. We identified a potential CRM, pCRM (-7), by *in silico* analysis of conservation and chromatin accessibility and hypothesize that pCRM (-7) displays activity coincident with the *Lhx9* species-specific expression.

To determine the activity of pCRM (-7), we isolated it from mouse genomic DNA to generate a fluorescence reporter plasmid which was inserted/transfected into the anterior and posterior regions of chicken limb buds via electroporation during a stage where *LHX9* expression is robust. Embryos were harvested 24 hours after transfection, and the activity was visualized using a fluorescence microscope.

We found that CRM (-7) was active in the limb bud and present in both anterior and distal-posterior regions, which is consistent with the pattern of murine *Lhx9* expression.

Our data suggest that CRM (-7) is a good candidate for regulating the posterior expression of *Lhx9* evident in mice. Further tests in transgenic mice are required to confirm this notion. Further studies are ongoing to determine whether the activity of chicken CRM (-7) is also active in the posterior limb domain.

IAN FORNCROOK
SURF PARTICIPANT 2026

I attend Walla Walla University in College Place, Washington, and this fall I will return as a senior studying biophysics.

As a SURF participant, I have worked under the mentorship of Dr. Sean Wilson and his lab manager, Dr. Claudia Rodriguez. With their guidance, I have learned to capture images on the lab's LSM900 microscope and to use several imaging programs–ImageJ, Imaris, and ZEN Blue–to perform colocalization analysis on those samples. My project is to create standardized protocols for each program in order to advance consistent methodology for colocalization of both 2D and 3D images, and to draw conclusions about how useful each one is for that analysis. One of my favorite parts of this research has been chasing the rush that comes after something I have been troubleshooting for hours finally works. These are tools I expect to carry into a PhD in biophysics and, possibly, a career in industry research. Outside of my current project, I am also drawn to neuroscience–especially how sleep influences neurodegenerative disease.

Outside the lab, I am most often outdoors playing racket sports or throwing frisbees; when I am not, you might find me playing guitar or spending time with friends.

I am grateful to Dr. Wilson and Dr. Rodriguez for their mentorship and their willingness to teach me the things I am curious about. Their guidance has been deeply appreciated.

STANDARDIZED PROTOCOLS AND CROSS-PLATFORM REPRODUCIBILITY OF QUANTITATIVE COLOCALIZATION ANALYSIS IN FLUORESCENCE MICROSCOPY

Ian Forncrook, Claudia Rodrigues, Sean Wilson

Advanced Imaging and Microscopy Core (AIM), Loma Linda University, Loma Linda, CA

Colocalization analysis uses the intensity and spatial overlap of two fluorescently labeled channels to quantify the degree to which different fluorophores occupy the same space. Multiple software packages perform this analysis using different algorithms, thus, the same image can yield different findings depending on the workflow. The goal of this project was to develop standardized, step-by-step protocols for widely used platforms, Fiji , Imaris, along with Zen Blue, and to determine which metrics remain comparable across software. Pearson's correlation coefficient, Manders' colocalization coefficients (M1 and M2), and threshold Manders' coefficients (tM1 and tM2) were evaluated in Fiji, Imaris, and ZEN using fluorescent bead standards, brain tissue, plant tissue, and cerebral arteriole samples in 2D and 3D (z-stack) images. Bead standards served as a positive control in which near complete overlap was expected. The protocols specify background subtraction, region-of-interest or mask definition, intensity thresholding, and the point-spread function, which sets the block size used by the Costes randomization. Costes testing confirmed that overlap exceeded chance in all samples, validating the comparisons that followed. Among the magnitude measures, Pearson's coefficient was the most stable, generally agreeing within five percent for the same image, and M1 and M2 coefficients greed closely. tM1 and tM2 coefficients showed the largest disagreement, differing by up to 20% in the brain tissue sample, where colocalization was partial, while agreeing within 2% in the near-fully colocalized plant sample. Each program computes its own thresholds, while regions of interest were redrawn manually. Platforms differed in the Manders values. These differences reflect inherent properties of each platform and define how the data should be interpreted: Pearson's coefficient can be compared across software, whereas tM1 and tM2 are platform-specific and most meaningful within a single workflow. Accordingly, Fiji suits quick, automatable analysis and Imaris suits three-dimensional data.

JORGE HIROSHI BOYAS HOMMA

SURF PARTICIPANT 2026

I am currently a rising junior at the University of Redlands pursuing a B.S. in Biochemistry and Molecular Biology with a minor in Physics through the 3+2 Engineering program. Outside the classroom, I am actively involved in the Redlands International Student Association (RISA), the TECHO club, and as one of the co-captains of the University of Redlands Dance Company. I also serve as a tutor and certified paramedic, as well as volunteering in food drives and local hospitals.

Research has become one of the most rewarding parts of my academic journey. I have previously investigated the biomechanics of ballet dancers' footwork, participated in the UofR's SURF program under the mentorship of Dr. Kendra Nelson in the kinesiology department, and conducted an Environmental Impact Assessment (EIA) of Jenks Lake under the mentorship of Dr. Lyons in collaboration with the U.S. Forest Service. This summer, I am conducting research in Dr. Schulte's Lab at Loma Linda University, where I am investigating BSH-loaded exosomes as targeted delivery vehicles for Boron Neutron Capture Therapy (BNCT) against glioblastoma stem cells.

After completing Google's Professional Machine Learning Engineer certification, I hope to pursue a career in AI-driven biomedical engineering, and I am sincerely grateful to Dr. Schulte and everyone in his team for their mentorship, encouragement, and the opportunity to contribute to meaningful research this summer that can help me grow into this career path in the future.

DEVELOPMENT AND OPTIMIZATION OF AN ELECTROPORATION PROTOCOL FOR LOADING U87 MALIGNANT GLIOMA-DERIVED EXOSOMES WITH SODIUM BOROCAPTATE DEQUALINIUM COMPLEX

Jorge Hiroshi Boyas Homma, Dr. Rajesh K. Gaur, PhD, Dr. Kevin E. Nick, PhD, Dr. Nie Ying, PhD, Dr. Reinhard Schulte MD, MS, DABR

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

Glioblastoma (GBM) is a highly aggressive, grade 4 primary brain cancer with a five-year relative survival rate of 6.9%, one of the lowest in malignant human tumors. The current standard GBM treatment consists of maximal safe resection surgery, followed by six weeks of radiation therapy combined with temozolomide chemotherapy, and six months of adjuvant temozolomide. This treatment, however, fails due to a number of factors, including infiltrative tumor growth, heterogeneity, and high resistance to chemoradiation. Boron Neutron Capture Therapy (BNCT) presents a promising alternative to treat GBM tumors. The idea of BNCT is to selectively deliver ^{10}B to tumor cells, in particular to highly resistant tumor stem cells, while minimizing the ^{10}B uptake by healthy cells. When a ^{10}B nucleus captures a low-energy thermal neutron, it splits into a helium and lithium ion, respectively, which deposit a large, high linear energy transfer (LET) radiation dose within the cell's diameter, destroying its nuclear DNA. Successful BNCT requires (1) sufficiently high fluency of thermal neutrons in the tumor and (2) selective delivery of ^{10}B to tumor cells. Current ^{10}B compounds, including Sodium Borocaptate (BSH), only partially fulfill the latter condition. Our hypothesis is that exosomal delivery of BSH conjugated with dequalinium (DEQ), an FDA-approved drug known to target mitochondria of GBM stem cells with a large transmembrane potential, will create the selectivity required. The purpose of this summer research was to develop an efficient protocol for loading the BSH+DEQ complex into GBM derived exosomes.

MELANIE MORALES
SURF PARTICIPANT 2026

I am an incoming senior at the University of California, Berkeley, where I study nutrition and metabolic biology and minor in math and science education. I have always been drawn to physiology and the study of how the body works, because each system reveals the intricacy and purpose of God's creation. As a Gates Scholarship recipient, and through countless opportunities, I have learned that persistence, hard work, and faith can open doors I once only hoped to reach.

At Berkeley, I have sought opportunities that allow me to serve and learn. I have worked with students in local Bay Area public schools by helping design lessons, tutor, and support teachers in the classroom. I have also volunteered with organizations serving unhoused communities through meal preparation and service, as well as in hospital settings by assisting nurses, answering calls, and helping with medical equipment.

This summer, I am grateful to work alongside Carlos Silveyra and Aditi Bhatnagar. Our project studies stem cell therapy for premature infants who develop lung damage after high oxygen exposure like bronchopulmonary dysplasia. Using brightfield microscopy, I image mouse lungs stained with Masson's trichrome stain to analyze for fibrosis using FIJI due to hyperoxia-induced BPD.

This experience has taught me that research is not strictly a step-by-step process and that unexpected results can become meaningful opportunities to learn. Thank you to Claudia Rodriguez and Dr. Sean Wilson's lab for welcoming me, teaching me patiently, and giving me the opportunity to grow as a person and future scientist.

QUANTIFICATION OF HYPEROXIA-INDUCED PULMONARY FIBROSIS IN A NEONATAL MOUSE MODEL OF BRONCHOPULMONARY DYSPLASIA

Melanie Morales, Carlos Silveyra, Aditi Bhatnagar, Claudia Rodriguez Torres, Ciprian P Gheorghe, Sean M Wilson
Lawrence D. Longo Center for Perinatal Biology, Advanced Imaging and Microscopy Core, School of Medicine, Loma Linda University, Loma Linda, CA

Supplemental oxygen is a life-saving intervention for premature infants with respiratory insufficiency. However, prolonged hyperoxia can generate reactive oxygen species, leading to oxidative stress, inflammation, impaired alveolar development, and driving progressive pulmonary fibrosis, all of which are characteristic of bronchopulmonary dysplasia (BPD).
The objective of this study is to characterize hyperoxia-induced structural lung injury in a neonatal mouse model by quantifying fibrosis and airspace availability. We hypothesize that hyperoxia-exposed lungs will exhibit increased collagen deposition and reduced airspace availability, resulting in an elevated fibrosis-to-parenchyma ratio and a decreased airspace-to-sample ratio compared with normoxic controls.
Brightfield images of three-week-old mouse lung sections stained with Masson's trichrome were acquired using an Evident APEXVIEW APX100 microscope. Images were analyzed in FIJI using a semi-automated workflow to evaluate structural remodeling by measuring collagen deposition relative to total parenchyma and quantifying airspace available for gas exchange. Comparisons between normoxic and hyperoxic lungs generated measurements of parenchymal, fibrotic, and airspace areas, which were used to calculate fibrosis-to-parenchyma and airspace-to-sample ratios. We expect these airspace measurements to correspond with parallel morphometric assessments of mean linear intercept and alveolar septal volume density in H&E-stained lungs, while Masson's trichrome analysis extends this work by evaluating collagen matrix formation and pulmonary fibrosis.
This workflow establishes a reproducible method for assessing hyperoxia-induced lung damage in a neonatal mouse model of BPD. Ultimately, this model provides the framework for future studies evaluating the therapeutic efficacy of Wharton's jelly-derived mesenchymal stem cell exosomes (WJ-MEx). We hypothesize that WJ-MEx treatment will reduce pulmonary fibrosis and preserve alveolar wall structure, providing a foundation for developing regenerative therapies for premature infants with BPD.

SARAH LAWRENCE
SURF PARTICIPANT 2026

In my sophomore year at Biola University, I spoke to a professor about different careers in healthcare I was thinking about pursuing. As I talked about what I was interested in, I noticed that I was drawn to the research being done in each field rather than the direct patient care. This professor turned my attention towards biomedical research, and I was encouraged to apply to Loma Linda's SURF program. This summer I have had the chance to learn techniques like Western blotting and flow cytometry as well as the larger process of bringing a paper to publication. I've enjoyed learning about the process of research as much as completing assays and analyzing data. I am so thankful to Dr. Nathan Wall and Ann Morcos for mentoring me and patiently teaching me the skills I need to succeed in a future PhD program.

As I look forward to finishing my degree at Biola and a future contributing to medical research, I am more and more convinced that the Lord has made our world infinitely complex and beautiful. The more I learn about the molecular pathways in our cells, the more I am in awe of his workmanship. I can think of no better way to worship the Lord than a life spent learning about his creation and using that knowledge to love those around me.

CURCUMIN ACTIVATES PARALLEL APOPTOTIC, NECROPTOTIC, AND MITOCHONDRIAL CASPASE-INDEPENDENT DEATH PATHWAYS IN GEMCITABINE-RESISTANT PANCREATIC CANCER CELLS

Sarah Lawrence[2,4], Ann Morcos[1,2], Yeonkyu Jung[1,2], Victor Dominguez Porras[2,3], Hayden Dennis[2,4], David Caba Molina[3], Ryan N. Fuller[5], Nathan R. Wall[1,2*]

[1]Dept. of Pathology & Human Anatomy, Div. of Human Anatomy, [2]James M. Slater, MD Proton Treatment & Research Center, Dept. of Radiation Medicine, [3]Dept. of Surgery, [4]Loma Linda University School of Medicine, Loma Linda, CA 92350 USA, [5]Dept. of Biological Sciences, California Baptist University, Riverside, CA 92504, USA

Pancreatic ductal adenocarcinoma (PDAC) remains one of the most lethal malignancies, largely due to intrinsic and acquired resistance to gemcitabine-based chemotherapy. Curcumin, a bioactive polyphenol derived from turmeric, has been widely reported to modulate multiple oncogenic signaling pathways; however, the mechanisms by which it induces cell death in chemoresistant pancreatic cancer cells remain incompletely defined. In this study, we investigated the temporal activation of cell death pathways following curcumin treatment in gemcitabine-resistant PANC-1 cells. Cells were treated with 50 μM curcumin and harvested at three-hour intervals over a 24-hour period, with additional analysis at 36 hours. Protein expression and activation of cell death pathways were evaluated by immunoblotting while cell viability was evaluated using AnnexinV and 7AAD staining after treatment with Curcumin, necrostatin-1, and z-VAD-FMK. Curcumin treatment resulted in time-dependent activation of caspase-8, -9, and cleaved-3, indicating engagement of both extrinsic and intrinsic apoptotic pathways. Concurrently, increased expression and phosphorylation of MLKL, along with modulation of RIP1, suggested activation of necroptotic signaling. In addition, enhanced expression and cleavage of apoptosis-inducing factor (AIF) and Endonuclease G (EndoG) supported involvement of caspase-independent mitochondrial cell death. Treatment with necrostatin-1 and z-VAD-FMK resulted in partial rescue. Notably, these pathways were activated in a temporally coordinated but distinct manner, indicating that curcumin does not rely on a single cytotoxic mechanism but instead engages multiple, overlapping death programs in chemoresistant pancreatic cancer cells.

SYDNEY HANSON
SURF PARTICIPANT 2026

Neurodegenerative disorders and their impact on my family have played a primary role in my desire to pursue a Ph.D. in neuroscience. Seeing the effects of chronic diseases firsthand, I seek to research the mechanisms behind them and apply my findings translationally to improve patients' quality of life
In high school, I was accepted into several collegiate-level honors societies while in a dual-enrollment program, and in 2024, I was the first-ever recipient of Destroy Duchenne's Student Scholarship. Since then, I have been working with their nonprofit as an ambassador to raise awareness about Duchenne Muscular Dystrophy and to network with biotech companies to advance ongoing genetic and neuromuscular research.

Recently, I studied the physiological effects of acetaminophen and GABA on the optokinetic response of zebrafish, and I plan on shifting into pancreatic cancer research this next year. While in SURF, I hope to gain more specific neurobiological research experience and build upon my foundational understanding of neuroscience as a whole.

This fall, I will enter my senior year at California Baptist University, where I am pursuing a B.S. in biomedical sciences with a minor in psychology, ultimately working towards a graduate application in cognitive and clinical neuroscience. While at CBU, I am also blessed to mentor students by serving as a TA and club president, getting to pour into them as my professors have into me.

Thank you to Dr. Christopher G. Wilson for mentoring me at LLU and to Dr. Ryan Fuller at CBU.

Psalm 139:13-14

VAGUS NERVE STIMULATION ALTERS TNF-α EXPRESSION IN A RAT MODEL OF NECROTIZING ENTEROCOLITIS

Sydney Hanson

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

One in every twenty patients in the NICU develops necrotizing enterocolitis (NEC), a severe inflammatory disease of the gut in premature infants associated with high mortality rates and long-term neurological impairments for survivors. The cause of NEC is not well understood but typically shows infection and an increase in inflammatory markers, including cytokines and chemokines. On average, 25% of infants with NEC do not survive, and those who do face increased neurodevelopmental impairment. Current treatments include antibiotics, restricted feeds, or bowel resections, but these approaches are both limited and invasive. Most importantly, they do not cure NEC. In addition to uncertain treatment for intestinal injury, approaches to NEC-associated neuroinflammation remain underdeveloped. However, given the enteric nervous system's autonomic control of inflammation and the role of the vagus in homeostasis, pre-established pathways in the gut-brain axis, primarily the cholinergic anti-inflammatory pathway (CAP), serve as a foundational link. When inflammation occurs, CAP regulates the production of cytokines, mainly inhibiting the production of pro-inflammatory cytokines like TNF-α and IL-1β. The vagus nerve is the primary conduit for CAP action. Vagus nerve stimulation (VNS) has emerged as a promising, non-invasive approach to immunoregulation by activating CAP. We investigated the effects of VNS in NEC models by quantifying changes in cytokine expression within the nucleus tractus solitarius (NTS) of neonatal Sprague-Dawley rats in NEC-positive and NEC-negative, sham VNS, and VNS treatment groups. We used immunohistochemistry to selectively label TNF-α and IL-1β expression in the NTS regions associated with gut afferent input to the brainstem. We hypothesize that, relative to NEC-negative controls, pro-inflammatory cytokines will increase in rat pups with NEC, and VNS stimulation will reduce pro-inflammatory cytokines compared to non-VNS controls. By reducing NEC neuroinflammation, VNS treatment has potential for successful, non-invasive therapeutic intervention in preterm infants with NEC.

Macpherson Society Scholars

EMILY ROJAS
MACPHERSON PARTICIPANT 2026

Most of my undergraduate experience at Southern Adventist University was filled with ministry and service alongside my Biology degree studies. From serving as a Bible study leader on campus to taking a gap year to work in a high school girls' dormitory, I kept pursuing opportunities to create meaningful relationships. My love for people and desire for others to seek wholeness were major contributors to my decision to pursue a career in medicine.

As I applied to medical schools, I recognized that many of my peers had heavier research experiences. I wondered whether this gap would hurt my applications, but at the time, I was unsure if research would contribute to becoming a compassionate and patient-centered physician. When I started medical school at Loma Linda University, research became a larger focus. I was excited to learn about the McPherson Research Program because it seemed like an opportunity to explore that question for myself.

I matched into Dr. Sean Wilson's lab, which oversees projects ranging from youth athlete development to tissue hypoxic response. My summer project explores cycling coaching principles that can maximize athlete development and retention. I have enjoyed learning about these psychosocial principles because of the parallels in effective coaching and compassionate doctoring. Working in such a diverse lab has allowed me to learn from my peers and their projects, giving me a broader understanding of what research entails and how it can shape me into the physician I hope to become.

REIMAGINING YOUTH CYCLING: INTEGRATING LIFESTYLE MEDICINE, PSYCHOSOCIAL WELL-BEING, AND ECOLOGICAL DYNAMICS WITHIN THE LONG-TERM ATHLETE DEVELOPMENT FRAMEWORK

Matthew Rittenbach, Emily Rojas, Jacqueline Monteza, Micheal Norton, Freddie Rodriguez, Larry Nolan, Richard Cheetham, Sean M Wilson

Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA 92350, MSN Pro Coaching, Fast Freddie Foundation, Team Nolan, University of Winchester

Youth cycling participation in the United States remains concerningly low, with persistent retention challenges across all competitive levels. Coaches and individuals who value cycling's physical, mental, and sociological impacts have failed to close this gap through traditional coaching approaches, which emphasize an individual athlete's development. Demand for a more holistic perspective has led to this project, which draws on interdisciplinary research, empirical program evidence, and direct coaching experience. We advocate for a reappraisal of youth cycling development that considers athlete connectedness to themselves, their peers, and coaching communities. We propose an integrated model combining the Long-Term Athlete Development framework, Youth Physical Development, lifestyle pillars, ecological dynamics theory, and psychosocial health promotion as foundational principles for sustained sport engagement. By re-centering coaching practice on positive experiences, inclusivity, and evidence-based environmental design through constraints-led approaches, cycling can evolve from a marginalized activity into a lifelong pillar of American culture and public health.

HAREL SIMS

MCPHERSON PARTICIPANT 2026

As an incoming second-year medical student at Loma Linda University, I am conducting research this summer in Dr. Salma Khan's laboratory, where I am investigating the expression of CD163, TGF-β, PD-L1, VDR, and LAT1 in anaplastic thyroid cancer. Through this work, I hope to help identify potential therapeutic targets for radioligand therapy and immunotherapy in cancer treatment.

Prior to medical school, I earned a bachelor's degree in Biological Sciences from the University of California, Irvine. During my undergraduate studies, I worked in a urology research laboratory focused on improving urologic outcomes. I also served as Treasurer of IRIS, a federally recognized nonprofit organization dedicated to reducing barriers for medical students around the world by providing research opportunities, mentorship, and financial scholarships.

Outside of medicine and research, I enjoy going to the beach, beekeeping with my father, and serving in my Romanian church. My experience in Dr. Khan's laboratory has strengthened my appreciation for translational cancer research and its potential to improve patient care. I am sincerely grateful to Dr. Khan for her mentorship, wisdom, and unwavering support, as well as for the opportunity to contribute to her impactful and meaningful research.

QUANITIFICATION OF M2-LIKE TUMOR ASSOCIATED MACROPHAGE EXPRESSION IN ANAPLASTIC THYROID CANCER SUBTYPES AND STAGING

Harel Sims, Joseph Cruz, Alena McQuarter, Mia Perez, Andrea Shields, Alfred A. Semintal, Salma Khan
Center for Health Disparities and Molecular Medicine,
Otolaryngology, Loma Linda University School of Medicine

Anaplastic thyroid cancer (ATC) is a highly aggressive and undifferentiated thyroid malignancy. Although it accounts for 1–2% of thyroid cancer diagnoses, ATC is responsible for a disproportionate number of cancer-related deaths because of its rapid growth, metastasis, local invasion, and resistance to conventional therapies. Increasing evidence suggests that the tumor microenvironment plays a role in ATC progression and treatment resistance. A key component of the tumor microenvironment is tumor-associated macrophages (TAMs), which are immune cells that may adopt an M2-like, tumor-promoting phenotype. M2-like TAMs contribute to immune suppression, angiogenesis, tissue remodeling, tumor invasion, and metastatic progression. This study evaluated the expression of CD163, transforming growth factor-beta 1 (TGF-β1), and alpha-smooth muscle actin (α-SMA) in ATC specimens across tumor subtypes and staging. CD163 is a marker of M2-like macrophages and reflects an immunosuppressive macrophage population. TGF-β1 is a cytokine that can promote immune evasion, fibrosis, epithelial–mesenchymal transition, invasion, and metastasis in advanced cancers. α-SMA is a marker of activated fibroblasts and myofibroblasts, including cancer-associated fibroblasts, and indicates stromal activation and extracellular matrix remodeling. Multiplex immunohistochemistry was performed on formalin-fixed, paraffin-embedded ATC tissue samples to simultaneously visualize and analyze these markers while preserving their spatial relationships within the tissue. Malignant tumor regions and adjacent benign thyroid tissue were identified and compared using semi-qualitative image analysis. CD163, TGF-β, and α-SMA demonstrated elevated expression within malignant regions compared with benign tissue. Expression was particularly prominent within the tumor-associated microenvironment surrounding malignant cells, suggesting increased M2-like macrophage infiltration, immunosuppressive signaling, and stromal activation. These findings support the involvement of M2-like TAMs and associated stromal pathways in ATC progression and may provide a foundation for future tumor microenvironment-directed therapeutic strategies.

MATTHEW RITTENBACH

MACPHERSON SUMMER RESEARCH 2026

My interest in sports science and lifestyle medicine is shaped through my own experience as an athlete. I was involved in a wide variety of sports from a young age through college; particularly competing at a collegiate level in cross country before stepping away due to burnout. That experience sparked an interest in how coaching, training, and athlete development can better support long-term sports participation. I earned my Bachelor of Science in Biology from Walla Walla University and currently I am a second-year medical student at Loma Linda University School of Medicine.

This summer I had the privilege of working in Dr. Sean Wilson's laboratory on a research project that advocates for a comprehensive reappraisal of youth cycling development. The project proposes an integrated model that combines Long-Term Athlete Development, Youth Physical Development, lifestyle medicine principles, ecological dynamics, and psychosocial health promotion to encourage lifelong participation in sport and physical activity. This summer I have been able to learn how effective coaching and training do not have to be defined by the "suffer-fest" mentality that I experienced as an athlete. Instead, carefully designed environments can encourage skill progression, psychological well-being and long term engagement.

I am grateful to Dr. Sean Wilson for the guidance and encouragement this summer and for the opportunity to be a part of this project.

REIMAGINING YOUTH CYCLING: INTEGRATING LIFESTYLE MEDICINE, PSYCHOSOCIAL WELL-BEING, AND ECOLOGICAL DYNAMICS WITHIN THE LONG-TERM ATHLETE DEVELOPMENT FRAMEWORK

Matthew Rittenbach, Emily Rojas, Jacqueline Monteza, Micheal Norton, Freddie Rodriguez, Larry Nolan, Richard Cheetham, Sean M Wilson

Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA 92350, MSN Pro Coaching, Fast Freddie Foundation, Team Nolan, University of Winchester

Youth cycling participation in the United States remains concerningly low, with persistent retention challenges across all competitive levels. Coaches and individuals who value cycling's physical, mental, and sociological impacts have failed to close this gap through traditional coaching approaches, which emphasize an individual athlete's development. Demand for a more holistic perspective has led to this project, which draws on interdisciplinary research, empirical program evidence, and direct coaching experience. We advocate for a reappraisal of youth cycling development that considers athlete connectedness to themselves, their peers, and coaching communities. We propose an integrated model combining the Long-Term Athlete Development framework, Youth Physical Development, lifestyle pillars, ecological dynamics theory, and psychosocial health promotion as foundational principles for sustained sport engagement. By re-centering coaching practice on positive experiences, inclusivity, and evidence-based environmental design through constraints-led approaches, cycling can evolve from a marginalized activity into a lifelong pillar of American culture and public health.

Lab Assistant and Volunteers

ALYSSA SORRELL

LAB VOLUNTEER 2026

I graduated from the University of Redlands in the Spring of 2026 with a degree in Biochemistry and Molecular Biology and a minor in Public Policy. During my time at Redlands, I worked as a lifeguard and a peer tutor for biology and chemistry courses. I was also a member of the women's water polo team for my three years at Redlands. I served as captain of the team my senior season and was awarded All American Recognition. This upcoming fall, I will be attending the Loma Linda School of Pharmacy in hopes of pursuing a career as a pharmacist.

I joined Dr. Sean Wilsons lab in the Spring of 2024. My research involves investigating the effects of long-term hypoxia on sheep vasculature using metabolomic analysis. My main area of focus is fetal carotid artery tissues, but I also assist with pulmonary and adipose tissues. I find it interesting to see how alterations to something as small as a metabolite can add up and help produce changes to the human body.

Thank you to Dr. Sean Wilson's lab for welcoming me back again this summer and providing me with the opportunity to continue my research.

GESTATIONAL LONG TERM HIGH ALTITUDE HYPOXIA AND METABOLOMIC REPROGRAMMING IN ADIPOSE TISSUES OF NEWBORN SHEEP

Alyssa Sorrell, Delaney Werner, Hans Westenburg, Micheal La Frano, John Newman, Oliver Fein, Sean Wilson

Salk Institute for Biology Studies, La Jolla, CA; West Coast Metabolomics Center, University of California Davis, Davis, CA; MD Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA

During gestation, long term hypoxia (LTH) is known to upregulate brown adipose tissue (BAT) genes which express uncoupling protein 1 (UCP1) to support thermoregulation. In the weeks following birth, adipose tissue undergoes a dramatic phenotypic transition from BAT toward a more white adipose tissue (WAT) state characterized by increased lipid accumulation and reduced thermogenic capacity. This postnatal remodeling is especially dynamic during the first week of life, when UCP1 expression remains elevated before declining as the tissue shifts toward energy storage. The purpose of this study is to identify which pathways are reprogrammed under LTH in newborn adipose tissues. To investigate this, we obtained samples of adipose tissues from newborn normoxic and hypoxic sheep raised at 3,800 meters above sea level, starting 30 days within gestation, with tissues isolated 7-14 days post-natally. Metabolite levels were quantified oxylipin, endocannabinoid, and untargeted metabolomic analysis. Chemical similarity enrichment analyses and visualization by complex pathway analyses were performed on the datasets. Enrichment analysis revealed changes in key metabolic pathways such as glycolysis, amino acid metabolism, Citric Acid Cycle, and the Warburg effect. This is indicative that aerobic respiration was impacted due to the decrease oxygen concentration, observed in the metabolites involved in glycolysisglucose-6-phosphate, glucose-1-phosphate, dehydroascorbic acid all being increased under hypoxic stress conditions. Our findings suggest a shift in carbon flux towards glycolytic pathways supporting the BAT to WAT which results in decreased thermogenesis. Fully understanding how changes in cellular metabolism are tied to adaptations in adipose tissue following high altitude gestation is an active area of investigation.

DAN CELESTIN

LAB VOLUNTEER 2026

I am a 4th year medical student here at LLU School of Medicine and I will be applying for Anatomic and Clinical pathology at the end of this year. I am originally from Canada and graduated with biochemistry University of Ottawa and loved learning about cellular processes. I have always loved learning about the human body at microscopic scale and learning how those findings produce real and macroscopic effects. I have recently gotten the opportunity to complete a rotation in the pathology department and been able to witness how molecular staining and markers are used as critical supporting evidence in diagnosis and staging of cancers. I have learned how rapidly this field is evolving and that in a few years, the pathology landscape may look entirely different. In my future career as a pathologist, I envision being able to work in the clinical team as a crucial part of patient care. At the same time, I hope to continue devoting time to researching, developing better methods of detecting cancers for patient care. I have worked with Dr. Salma Khan previously on ovarian carcinoma and have been able to present posters. I am very appreciative of the opportunity to return this summer. As I am wrapping up my medical school journey, I am proud of the impacts I have made as a student and as a researcher.

INTERACTING PARTNERS OF TGF-β, PD-L1, AND ZEB2 IN IMMUNE MICROENVIRONMENT IN OVARIAN CANCER

Dan Celestin, Fariba Mamun, Romi Yamauchi, Cody S. Carter, Saeid Mirshahidi, Salma Khan

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High-grade serous ovarian carcinoma (HGSOC), the most common and lethal subtype, develops a highly immunosuppressive tumor microenvironment through dysregulated signaling pathways, including transforming growth factor-beta (TGF-β) and programmed death-ligand 1 (PD-L1), which facilitate immune evasion and tumor progression. The epithelial-mesenchymal transition (EMT) transcription factor ZEB2 is frequently upregulated in ovarian cancer and promotes tumor progression. This study aimed to identify protein-binding partners of TGF-β, PD-L1, and ZEB2 using immunoprecipitation to better understand the signaling networks that contribute to ovarian cancer progression. We hypothesized that TGF-β, PD-L1, and ZEB2 interact directly or indirectly within common signaling pathways that drive aggressive tumor behavior. We prepared protein lysates from frozen HGSOC tissues of the LLU patient cohort. Immunoprecipitation (IP) was performed using antibodies against TGF-β, PD-L1, and ZEB2 as baits. IP with TGF-β were probed for ZEB2, PD-L1, and integrin antibodies, PD-L1-IP were analyzed for AKT, α-SMA, and TGF-β and ZEB2-IP were examined for TGF-β and PD-L1 for Western Blotting. Protein interactions were detected by Licor and confirmed with molecular weights. This data demonstrated interaction of TGF-β with PD-L1 and integrin, whereas none in TGF-β with ZEB2. In addition, PD-L1 showed an interaction with AKT and MDM2, but not with TGF-β suggesting a potential link between immune checkpoint signaling and the PI3K/AKT pathway. Analysis of ZEB2-associated interactions is ongoing. These preliminary findings suggest that TGF-β and PD-L1 participate in interconnected signaling networks that may contribute to immune evasion and tumor progression in ovarian cancer. Defining these protein-protein interactions will improve our understanding of the molecular mechanisms underlying aggressive disease and may identify novel therapeutic targets and biomarkers for precision treatment of HGSOC.

DELANEY WERNER

LAB VOLUNTEER 2026

My interest in veterinary medicine and research has grown through every opportunity that I pursue that involves the intricate biological processes that occur through living systems. Particularly my fondness of development and health has begun to take shape as I have studied metabolomic processes this summer.

I currently attend the University of Redlands where I am about to return for my fourth year studying biology and human-animal studies. At Redlands, I have had the amazing opportunity to be a part of the Summer Science Research Program in partnership with Loma Linda University, working with Dr. Wilson. Working with his research has taught me to be excited about learning new processes within the human body and strengthened my love for scientific discovery.

During the school year I spend my time tutoring upper division chemistry and genetics courses, as I am very passionate about helping others find a love for science. After college, I hope to attend vet school to receive my DVM, becoming a doctor who serves the community both for animals and owners alike. Particularly, I have an interest in emergency medicine as it challenges my quick problem-solving skills in critical situations. Rationalizing the data I receive from the lab and turning it into evidence for the bigger picture is only just one way research helps guide me towards my path for the future.

Thank you to Dr. Sean Wilson for your guidance and for allowing me to get a start in my career in medicine.

IMPACT OF HIGH-ALTITUDE EXPOSURE ON METABOLIC PROFILES IN PERIRENAL ADIPOSE TISSUE OF FETAL SHEEP

Delaney Werner, Alyssa Sorrell, Hans Westenburg, Michael La Frano, John Newman, Oliver Fiehn, Charles Ducsay, Sean M Wilson

Salk Institute for Biology Studies, La Jolla, CA; West Coast Metabolomics Center, University of California Davis, Davis, CA; Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA

Chronic gestational hypoxia is associated with altered fetal development and metabolic dysfunction including an increase in thermoregulatory proteins, an overactive HPA axis, and increased endocrine signaling; however, the underlying metabolic pathways on perirenal adipose tissue (PAT) being reprogrammed remain unknown. In fetal sheep, hypoxia is known to cause "beiging" of PAT, increasing the phenotypic transformation of white-to-brown adipose tissue that are dense in mitochondria. The purpose of this study was to test the hypothesis that along with the upregulation of gene products critical to brown fat, PAT also undergoes metabolic reprogramming inclusive of increases in oxidative phosphorylation intermediates and oxidative stress metabolites. To test this theory, pregnant sheep were either held at a low elevation (~355m) or a high elevation (~3801m) for 110+ days during their gestational period, with PAT being extracted from the near-term fetal sheep, with metabolism assessed through untargeted along with targeted oxylipin and endocannabinoid analyses. Informatics on these metabolites from the fetal PAT were consequently run through univariant enrichment and pathway analysis using MetaboAnalyst to detect and interrogate changes in cellular metabolism. The data revealed significant changes in central carbon metabolism through an increase in the Citric Acid Cycle, acetyl-transfer pathways, and pathway intermediates suggestive of the Warburg effect. There was an increase in key oxidative metabolites indicating mitochondrial reprogramming and a transformation towards a brown adipose phenotype. There were also important alterations in fatty acid and amino acid derived metabolisms known to be associated with endocrine outputs and the sustainment of oxidative intermediates in the TCA cycle. Determining the physiological implications of the increases in BAT and enhanced oxidative stress on fetal PAT is essential to understanding how these metabolic consequences may affect the maturation of postnatal function and paves the way for future studies to further understand the impact of enhanced BAT function.

FARIBA MAMUN
LAB VOLUNTEER 2026

I graduated from the University of California, Riverside in 2025 and was given the opportunity to join Dr. Salma Khan's laboratory in January 2026. Currently, I am investigating differences in protein expression and biomarkers between low- and high-grade ovarian cancer. Using Western blot analysis, we apply specific antibodies to detect proteins that distinguish these two forms of the disease. This research is important because it provides evidence that low- and high-grade ovarian cancer are biologically distinct diseases that require different approaches to treatment.

Beyond research, I volunteered with A Friend in Me, a nonprofit organization dedicated to supporting pediatric cancer patients and their families. We collaborated with hospitals throughout Southern California to organize events that provided children and young adults with a sense of normalcy during treatment. Building relationships with these patients and witnessing their resilience strengthened my passion for pursuing a career in medicine, with the goal of specializing in pediatrics.

I am incredibly grateful to Dr. Salma Khan, Romi Yamauchi, and the entire laboratory team for their guidance and for fostering a welcoming and collaborative environment. Through this experience, I have gained a deeper appreciation for the vital role research plays in advancing medicine, driving innovation, and improving patient care. Outside of the laboratory, I enjoy baking, creating art, and building LEGO sets.

DIFFERENTIAL EXPRESSION OF SEVERAL ONCOGENIC PROTEINS IN OVARIAN CANCER

Fariba Mamun, Celena Romi Yamauchi, Alena McQuarter, Olabisi Bamidele, Salma Khan

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

Ovarian Cancer is one of the deadliest forms of female reproductive cancers due to the lack of screenings and early detections, often leading to late-stage diagnoses. High-grade ovarian cancer (serous carcinoma) is the most aggressive and common form, representing approximately 70 to 80% of malignant cases. This study focuses on the differential expressions of oncogenic and regulatory proteins in ovarian samples. The oncogene MDM2 regulates and degrades the tumor suppressor protein known as p53. Programmed death-ligand 1 (PD-L1), an oncogene, is an immune checkpoint protein that contributes to tumor evasion by inhibiting immune cell responses. Transforming Growth Factor-beta 1 (TGF-β1), a regulatory protein, controls cell growth and immune function, however, overexpression can lead to metastasis or tumor progression. Vitamin D Binding Protein, another regulatory protein, transports vitamin D in the bloodstream and regulates immune responses, and altered expressions can lead to tumor progression. Vitamin D Receptor, a regulatory gene, is a tumor suppressor and influences gene expression. Zinc Finger E-Box Binding Homeobox 2 (ZEB2) influences tumor growth and progresses diseases. Protein expression levels were analyzed using Western Blot of about 25 ovarian cancer patient samples. Specific antibodies targeting MDM2, PD-L1, TGF-β1, VDR, DBP, and ZEB2 were used to analyze protein expression, while GAPDH was used as a housekeeping gene and loading control. These proteins are differentially expressed and regulate the immune tumor micro-environment in high grade ovarian cancer. We conclude that differential expressions of these oncogenes can be targeted for the better survival of the patients based on their expression. These findings provide insight to the immune tumor micro-environment and support the development of targeted patient therapies, which lead to the overall improvement of patient outcomes.

JACQUELINE MONTEZA
LAB VOLUNTEER 2026

I grew up in Washington State, where I earned my undergraduate degree in biology from the University of Washington, and recently completed my first year of medical school at Loma Linda University School of Medicine. This summer, I had the privilege of conducting research in Dr. Sean Wilson's lab on youth cycling, an experience that allowed me to explore various aspects of lifestyle medicine, better understand the importance of promoting physical activity in the community, and deepen my interest in the field. Collaborating with my fellow researchers, Emily Rojas and Matthew Rittenbach, was one of the highlights of the summer, and I am grateful for their insights and support as we worked on this project. I am also grateful to Dr. Sean Wilson's mentorship, guidance, and encouragement throughout this summer, and appreciate the opportunity to contribute to the work of his research lab.

REIMAGINING YOUTH CYCLING: INTEGRATING LIFESTYLE MEDICINE, PSYCHOSOCIAL WELL-BEING, AND ECOLOGICAL DYNAMICS WITHIN THE LONG-TERM ATHLETE DEVELOPMENT FRAMEWORK

Matthew Rittenbach, Emily Rojas, Jacqueline Monteza, Micheal Norton, Freddie Rodriguez, Larry Nolan, Richard Cheetham, Sean M Wilson

Center for Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, CA 92350, MSN Pro Coaching, Fast Freddie Foundation, Team Nolan, University of Winchester

Youth cycling participation in the United States remains concerningly low, with persistent retention challenges across all competitive levels. Coaches and individuals who value cycling's physical, mental, and sociological impacts have failed to close this gap through traditional coaching approaches, which emphasize an individual athlete's development. Demand for a more holistic perspective has led to this project, which draws on interdisciplinary research, empirical program evidence, and direct coaching experience. We advocate for a reappraisal of youth cycling development that considers athlete connectedness to themselves, their peers, and coaching communities. We propose an integrated model combining the Long-Term Athlete Development framework, Youth Physical Development, lifestyle pillars, ecological dynamics theory, and psychosocial health promotion as foundational principles for sustained sport engagement. By re-centering coaching practice on positive experiences, inclusivity, and evidence-based environmental design through constraints-led approaches, cycling can evolve from a marginalized activity into a lifelong pillar of American culture and public health.

JAMES KUO
LAB VOLUNTEER 2026

I am a second-year medical student at Loma Linda University School of Medicine. I graduated from the University of Florida with a Bachelor of Science in Biology. During college, I participated in medical mission trips to Honduras and volunteered by performing music at assisted living facilities. I also conducted research on the development of a 12-plex respiratory virus diagnostic assay using PCR and Nanopore sequencing. During my gap year before medical school, I worked as an inpatient phlebotomist at UF Health Shands Hospital. In my free time, I enjoy playing piano, basketball, pickleball, and practicing yoyo tricks.

I am currently conducting research under the mentorship of Dr. Salma Khan and Romi Yamauchi, focusing on health disparities in papillary thyroid cancer by analyzing gene and protein expression patterns that may influence tumor behavior and clinical outcomes. Through this experience, I have enjoyed learning how research can connect molecular findings to broader questions about patient care and cancer disparities. I would like to acknowledge Dr. Salma Khan for providing me with this opportunity and thank her research team for their mentorship and support throughout this research experience.

HYOU1 AND ITS RELATED GENES AS POTENTIAL PROGNOSTIC MARKERS IN PAPILLARY THYROID CANCER

James Kuo, Samuel Chan, Luiza Barseghyan, Romi Yamauchi, Bamidele Olabisi, Alfred A. Simental, Salma Khan

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

Papillary thyroid cancer (PTC) exhibits differences in incidence, recurrence, and survival among racial and ethnic groups, suggesting underlying molecular mechanisms contributing to disease progression. In a previous bulk RNA-sequencing study of the Loma Linda University cohort, we identified HYOU1 as differentially expressed among four ethnic groups. HYOU1 is a gene involved in protein folding and adaptation during hypoxia and endoplasmic-reticulum stress. HYOU1 expression has been associated with tumor-cell survival, disease progression, and poor clinical outcomes in several malignancies. We hypothesized that HYOU1 and its related genes are associated with papillary thyroid cancer progression and survival. TCGA thyroid carcinoma datasets were analyzed using the UALCAN platform to evaluate HYOU1 and its positively correlated genes for tumor-versus-normal expression, race, sex, tumor stage, survival, and thyroid cancer cell-line dependency. Three positively correlated genes, SRPRB, DDOST, and TTC30A, were selected based on Pearson correlation strength, expression differences, survival associations, and DepMap gene-effect scores. HYOU1 was significantly downregulated in thyroid tumors and demonstrated reduced expression across racial, sex, and tumor-stage subgroups. Higher tumor expression was associated with poorer overall survival. DepMap CRISPR-knockout analysis showed that loss of HYOU1 reduced the viability of several thyroid cancer cell lines, suggesting these cells may depend on HYOU1 for survival. The three correlated genes were also downregulated in thyroid tumors, associated with poorer survival, and showed similar dependency in DepMap thyroid cancer cell lines. SRPRB and DDOST are involved in endoplasmic-reticulum protein targeting and processing, whereas TTC30A functions in ciliary transport. These findings suggest that HYOU1, SRPRB, DDOST, and TTC30A may serve as potential prognostic markers in papillary thyroid cancer. Additional studies are needed to determine their potential as prognostic biomarkers or therapeutic targets.

KATHERINE GRANADOS
LAB ASSISTANT 2026

My first two years at my local community college have been helpful in preparing me to pursue a doctoral degree. I dedicated both of my honors research projects to STEM-related studies and presented them to my peers. During my presentation on Neurological Effects of Repetitive Brain Injuries in Contact Sports, my 5-minute Q&A section turned into a class discussion, with everyone actively engaged in the topic. This experience made me comfortable with public speaking and interested me in teaching.

In 2023, I became a mentor at Loma Linda, teaching basic laboratory skills to new students. In this role, I encourage students to develop confidence in their techniques while practicing essential laboratory methods, helping prepare them for their own research journeys. Mentoring is important in supporting the next generation of scientists.

These experiences have solidified my decision to become a neurological researcher, studying molecular mechanisms and health disparities where gender or race affects development. I also hope to develop treatments for concussion-related injuries in sports for our youth. As a determined first-generation student, I hope to push forward the field of neurodegenerative diseases, increase awareness, and develop optimal treatments for underserved communities.

Now in 2026, I am fortunate to continue research under the guidance of Dr. Johnny Figueroa. In this lab, I am able to apply the skills and techniques that I will use in the future when finishing my Bachelor's degree in Neurobiology, Physiology, and Behavior at UC Davis and working toward a PhD in neuroscience.

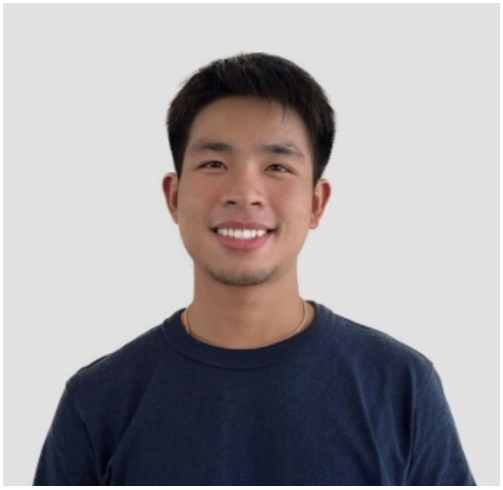
GUT MICROBIOME DEPLETION PERTURBS ENDOCRINE–GUT–BRAIN HOMEOSTASIS DURING ADOLESCENCE

Katherine Granados, Julio Sierra, Darine Abu Hilal, Leanne Mercado Allison Rhee, Jo-Wen Liu, Johnny, D. Figueroa

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

The gut microbiome plays a central role in regulating endocrine, metabolic, and neurobehavioral signaling. During adolescence, maturation of the gut microbiota coincides with major hormonal changes that influence brain development, yet the effects of microbiome disruption on endocrine signaling remain poorly understood. This study investigated how antibiotic-induced depletion of the microbiome alters fecal hormone profiles in female Lewis rats. Animals received a broad spectrum antibiotic cocktail in drinking water during adolescence and were maintained on either a Western diet or standard chow. Fecal samples were collected during adolescence (PND63) and adulthood (PND81). Enzyme immunoassays were used to quantify corticosterone, estrogen, progesterone, and serotonin as biomarkers of stress, endocrine function, and gut-brain communication. Antibiotic-treated animals exhibited significantly lower concentrations of corticosterone, estrogen, progesterone, and serotonin compared with controls, indicating that microbiome depletion disrupts multiple endocrine signaling pathways. Ongoing analyses are determining how these hormonal alterations relate to microbial composition and behavioral outcomes. Together, these findings suggest that perturbation of the adolescent gut microbiome produces persistent changes in endocrine signaling that may influence long-term gut-brain communication.

LINH NGUYEN
LAB VOLUNTEER 2026



As an international student from Vietnam and a lifelong competitive tennis player, I never expected tennis and medicine to cross paths. Traveling around the world for competitions exposed me to different cultures, communities, and healthcare systems, helping me realize that access to medicine is not the same everywhere. These experiences shaped my passion for becoming a physician who serves others with skill and compassion.

I am now a rising senior at the University of Redlands, pursuing a Bachelor of Science in Biology. This summer, I am representing my university through the Summer Undergraduate Research Fellowship while studying the role of BMI1 in ovarian cancer stemness and therapy resistance in Dr. Julie Unternaehrer's laboratory at Loma Linda University under the mentorship of Dr. Ethan Nyein. Being mentored by Dr. Nyein has helped me connect laboratory research to clinical questions in women's health.

My next major goal is to attend LLU's MD program, with the dream of becoming an ophthalmologist. I am fascinated by our eye's role in allowing us to perceive the world and translate light into images. I am also interested in longevity medicine and helping people live longer, healthier lives with those they love.

Outside of research, I volunteer at LLUH and Totally Kids Rehabilitation Hospital, and I enjoy piano, fitness, cooking, art, and time with friends. I am deeply grateful to Dr. Unternaehrer, Dr. Nyein, and the entire lab team for their guidance, patience, and for always meeting my many questions with a smile.

ONE DAMAGE, DIFFERENT REPAIRS: BMI1 AND DIVERGENT REPAIR PATHWAYS TO PLATINUM RESISTANCE IN HIGH-GRADE SEROUS OVARIAN CARCINOMA

Linh Nguyen, Juli Unternaehrer, Ethan Nyein, Ashlyn Conant, Brigitte Vasquez, Jacqueline Coats, Miley Miranda, Ann Morcos, Nathan Wall

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

Platinum resistance remains a major barrier in the treatment of high-grade serous ovarian carcinoma. We hypothesized that distinct platinum-resistant models exhibit different BMI1-associated DNA damage responses following irradiation. Prior work in the Unternaehrer laboratory identified elevated BMI1 in the PDX4 CR model and suggested that BMI1 redistributes to DNA damage sites, motivating its investigation across complementary resistance models. We compared the patient-matched PEO1/PEO4 system, in which PEO1 is platinum-sensitive and BRCA2-deficient whereas PEO4 is platinum-resistant with restored BRCA2 function, with the experimentally selected PDX4 CR model. Cisplatin sensitivity was confirmed by MTT assays, and cells were exposed to 4 Gy proton irradiation and collected at 0, 2, 12, and 24 hours. BMI1, BRCA1, and RAD51 mRNA expression was measured by reverse transcriptase quantitative PCR (RT-qPCR). PEO4 displayed greater cisplatin resistance than PEO1. Following irradiation, PEO1 and PEO4 showed increased BMI1 and BRCA1 mRNA expression, while RAD51 mRNA increased significantly only in PEO4 at 24 hours. This pattern is consistent with a BRCA2-restored, RAD51-associated transcriptional response in resistant PEO4 that was absent in BRCA2-deficient PEO1. In a separate irradiation experiment comparing PEO1 with PDX4 CR, PEO1 showed increased BMI1 mRNA at 2 hours, whereas PDX4 CR showed delayed BMI1 mRNA induction at 24 hours and decreased RAD51 mRNA over time, suggesting that platinum-resistant models may use divergent DNA damage response pathways rather than a single conserved mechanism. Differences in BMI1 induction across models support a context-dependent role for BMI1 in promoting DNA damage survival and platinum resistance. Planned protein and immunofluorescence analyses will assess BMI1 localization, γ-H2AX signaling, and BRCA2-RAD51 recruitment, followed by BMI1 gain- and loss-of-function studies to evaluate its therapeutic potential.

SAMANTHA NAJARRO
LAB VOLUNTEER 2026

Throughout my educational journey, I became captivated by the interdisciplinary nature of science. As I approach my third year at the University of California, Los Angeles, I have gained insight and value from hearing multiple perspectives. Majoring in Microbiology, Immunology, and Molecular Genetics with a minor in Biomedical Research has also broadened my view of what is capable in the field of science. I have seen numerous examples of the difference one can make in others' lives.

Service is a value that I have strived to orient towards. I began serving my community in high school, and as I pursue higher education, I continuously seek to bring about change for the betterment of others. With that, I am grateful to God for the opportunities I have been blessed with. Each day, I grow from these new learning experiences. Research is one of those experiences that inspires me because it brings many people together to design, collaborate, and innovate.

Thank you to Dr. Christian Hurtz for his leadership and for welcoming me to join the lab for the summer. I also want to thank Dr. V. S. S. Abhinav Ayyadevara for his mentorship and collaboration on his project, as well as all the wonderful members of the Hurtz Laboratory.

ENGINEERING CD22-TARGETED PEI NANOPARTICLES FOR NUCLEIC ACID DELIVERY TO LEUKEMIC B CELLS

Samantha Najarro[1, 2], V. S. S. Abhinav Ayyadevara[2], Christian Hurtz[2]

[1]University of California Los Angeles
[2]Division of Cancer Sciences, Loma Linda University

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. Many promising therapeutic targets in these subtypes are difficult to address with small-molecule inhibitors, which often carry disadvantages such as off-target effects, limited bioavailability, systemic toxicity, drug resistance, and short circulation half-life. To address this gap, we are developing a CD22-directed nanoparticle platform capable of delivering nucleic acids to B-ALL cells to induce or inhibit genes of interest in a subtype-specific manner. CD22 is a B-cell-specific receptor that undergoes clathrin-mediated endocytosis, with an observed vesicle size of approximately 200 nm, making it the physical size maximum of the nanoparticles to be delivered. Polyplexes were prepared using the gold-standard nucleic acid carrier 25 kDa polyethyleneimine (PEI), functionalized with GALA peptide for efficient endosomal escape and PEG for reduced immunogenicity, and surface-coated with anti-CD22 antibodies via streptavidin (SV) to enable selective uptake by B-ALL cells. PEI-based nanoparticles formulated with DNA or RNA exhibited hydrodynamic diameters ranging from 30–200 nm, with a peak size near 100 nm in serum-containing RPMI-based cell culture media. Although these sizes fall well within the endocytic limits of CD22, protein corona formation in the presence of serum proteins poses a significant challenge to nanoparticle delivery; ongoing efforts are therefore focused on preventing corona formation in culture media that mimic blood conditions. As proof of concept, PEI-based siRNA polyplexes targeting DYRK1A, a gene essential for survival in *KMT2A*-R ALL, were delivered via CD22-mediated endocytosis, silencing DYRK1A expression to 33% control levels and increasing apoptosis. These results demonstrate the platform's functional gene-silencing capability in a clinically relevant B-ALL model.

TERENCE KILLEBREW
LAB VOLUNTEER 2026

As a child, I was diagnosed with Guillain-Barré syndrome, which later progressed to Chronic Inflammatory Demyelinating Polyneuropathy. This motivated me to better understand diseases, to improve care for others facing similar challenges. Following my junior year at Oakwood University, I participated in the Undergraduate Training Program at Loma Linda University. During that time, I investigated how Aminoflavone, a synthetic anti-tumor drug, enhances the efficacy of endocrine therapy for breast cancer. These experiences were pivotal in my decision to pursue a career in research.

I am now a second-year student in the Master of Science in Biotechnology at Loma Linda University. This summer, I am conducting research In Dr. Figueroa's Lab, where I am investigating interactions between microglia and neuron using advanced microscopy techniques. Our research focuses on how poor gut health during adolescence disrupts brain development, increasing susceptibility to neurological disorders later in life. This research aligns closely with my academic and career goals pursuing research in nutrigenomics. My long term goal is to investigate how personalized nutrition can be used to prevent or reduce the risk of neurological disorders and improve patient outcomes. As a physician-scientist, I want to make evidence-based treatments to prevent and care for neurological disorders.

ANTIBIOTIC-INDUCED GUT DYSBIOSIS ALTERS HIPPOCAMPAL ACTIVITY DURING ADOLESCENT BRAIN DEVELOPMENT

Leanne Mercado, Katherine Granados, Terence Killebrew, Darine Abu Hilal, Allison Rhee, Julio Sierra, Jo-wen Liu, Johnny D. Figueroa

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

The gut microbiome plays an essential role in immune, metabolic, and neural development during adolescence. Emerging evidence suggests that microbial signals regulate microglial maturation and synaptic plasticity; however, the mechanisms through which gut dysbiosis influences adolescent brain development remain poorly understood. This study examined whether antibiotic-induced microbiome depletion alters hippocampal function during adolescence. Female Lewis rats received a broad-spectrum antibiotic cocktail in drinking water for three weeks to induce gut microbiota depletion. Following a 72-hour washout period, animals underwent behavioral testing and functional magnetic resonance imaging (fMRI). Antibiotic-treated rats exhibited reduced dorsal hippocampal activity compared with untreated controls, supporting a role for the gut microbiome in normal hippocampal maturation. Ongoing studies are evaluating neuroplasticity-associated proteins, including estrogen receptor β (ERβ), brain-derived neurotrophic factor (BDNF), and tropomyosin receptor kinase B (TrkB), together with microglial phenotyping and metabolomic profiling to identify mechanisms linking gut dysbiosis to altered brain development. These findings support the gut microbiome as a critical regulator of adolescent hippocampal maturation and provide a foundation for understanding how microbiome disruption influences long-term brain function.

TIMOTHY KIM

LAB VOLUNTEER SCHOOL OF PUBLIC HEALTH 2026

Living in four countries exposed me to diverse healthcare systems and cultural perspectives on health, illness, and access to care. These experiences taught me that health is shaped by more than biology, it reflects the interaction of environment, culture, opportunity, and lived experience. They also sparked my interest in understanding why health outcomes differ across populations and how social and structural factors contribute to health disparities.

Although I initially aspired to become a physician, my interests evolved toward understanding health beyond individual patient care. Through professional experiences in pharmaceuticals, medical devices, clinical research, and business development, I gained insight into how clinical evidence is generated, evaluated, and translated into practice. I also recognized how data quality and interpretation influence healthcare decisions and patient outcomes. These experiences ultimately led me to epidemiology, where I can investigate the patterns and determinants of health at the population level.

As a graduate student, I have developed strong interests in health disparities, epidemiologic data analysis, clinical data integrity, and evidence-based public health practice. My training has reinforced the importance of asking meaningful research questions, using reliable data, and critically evaluating evidence to address complex public health challenges.

I aspire to contribute to research that advances health equity, informs evidence-based policy, and supports interventions that improve the health and well-being of diverse populations.

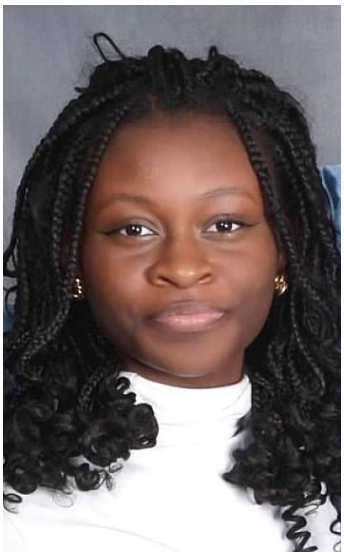
CYCLING AS AN EQUALIZER: ADOLESCENT MENTAL WELLBEING BENEFITS ARE ROBUST TO NATURE ACCESS, DEMOGRAPHICS, AND GEOGRAPHY IN THE RIDING FOR FOCUS PROGRAM

Timothy Hyunkyu Kim, Seth A. Wiafe, Sean M. Wilson, Esther Walker, Lauren Schuck, Cian L. Brown

Outride, California; University of Oklahoma; Health Geoinformatics Lab, School of Public Health, and School of Medicine, Loma Linda University, Loma Linda, CA

Adolescent mental health is a growing public health concern in the United States. Although physical activity is associated with better psychological wellbeing, whether these benefits differ by environmental or sociodemographic context remains unclear. This study examined whether the association between weekly cycling and mental wellbeing varied by nature access, social vulnerability, demographics, or geographic setting. Data comprised 39,938 surveys from 132 schools in the Riding for Focus (R4F) program across four academic years (2021–2025). Wellbeing was measured using the World Health Organization Well-Being Index (WHO-5), with weekly cycling participation as the primary exposure. Nature access was quantified using NatureScore, and neighborhood disadvantage using the CDC/ATSDR Social Vulnerability Index (SVI). Analyses in SAS 9.4 included multivariable regression, mixed-effects models, generalized estimating equations, and subgroup analyses; geographic analyses used ArcGIS Pro 3.7. Students cycling at least weekly reported significantly higher wellbeing than non-cyclists (mean WHO-5: 70.64 vs. 61.84; Cohen's $d = 0.383$; $p < 0.001$) and were less likely to fall below the low-wellbeing threshold (WHO-5 ≤ 50; 16.8% vs. 29.2%). The association remained robust after accounting for school-level clustering (ICC = 0.033), nature access, social vulnerability, gender, race/ethnicity, and geographic setting, with no significant effect modification. Although schools in more socially vulnerable communities had lower surrounding nature access ($r = −0.25$), the wellbeing association remained consistent across advantaged and disadvantaged settings. Weekly cycling was consistently associated with higher mental wellbeing regardless of context, suggesting that school-based cycling programs may be an equitable, scalable strategy for promoting youth mental health. Because the study is cross-sectional, findings represent associations rather than causal effects.

XARIA CLARK
LAB VOLUNTEER 2026



I' m an upcoming sophomore attending Stony Brook University in New York, currently studying biology and planetary sciences. By combining these two majors, I'm preparing myself for the field of astrobiology. Through this path, I hope to study extremophiles and the endless possibilities of God's creation. While I am still discovering exactly what my future path will look like, I plan to pursue a master's degree and a PhD after graduation, using this knowledge to eventually help and serve others. I've been inspired to look closer at the processes that govern both human health and survival in extreme conditions and how these resilient organisms could potentially have an impact. I view this opportunity as a crucial avenue that begins this journey.

My time in the lab this summer has given me a picture of how a professional research environment is paced, and has helped me develop the analytical, detail-oriented skills necessary for scientific discovery. I want to approach my future research as an exploration of the wonders of creation, recognizing that a posture of humble curiosity and service must accompany our search for answers. I am excited to see what God has in store.

I am incredibly grateful to Dr. Kerby Oberg for giving me the opportunity to support his lab's work, and for being such a welcoming, and inspiring PI. I also want to express my thanks to Charmaine Pira and Jeanyoung Kim for creating such a supportive learning environment and for being exceptionally patient and encouraging teachers.

LHX9 REGULATION DURING GONAD DEVELOPMENT IN THE CHICKEN

Xaria A. Clark, Jeanyoung Kim, Charmaine U. Pira
Kerby C. Oberg

Department of Pathology and Human Anatomy, School of Medicine, Loma Linda University, Loma Linda, CA.

Vertebrate reproductive organs begin as bipotential gonads that form in association with primitive kidneys during embryonic development. Lhx9, a LIM homeodomain transcription factor, is responsible for driving early gonad formation. Mutations in Lhx9 can result in complete failure of gonad formation or sex reversal, suggesting roles for Lhx9 in gonad formation and differentiation. Lhx9 functions via binding to *cis*-regulatory modules (CRMs). These sequences of DNA regulate the temporo-spatial transcription of a target gene. We have identified a potential CRM (CRM (-10)) in chickens that is near the *LHX9* gene and hypothesize that CRM (-10) is active in the gonad during early development.

To correlate early gonad development with *LHX9* expression, we performed *in situ* hybridization at various stages of development. To evaluate CRM(-10), the sequence was isolated by PCR and inserted upstream of a minimal promoter coupled to a fluorescent reporter. An *in ovo* electroporation was performed, transfecting the CRM-reporter into the developing gonad. We used the empty reporter construct as a negative control. We harvested and imaged the gonads using fluorescence microscopy 48 and 72 hours post-transfection.

Our results demonstrate *LHX9* expression in the surface epithelium of the forming bipotential gonads (Hamilton-Hamburger stage 16–32). Furthermore, CRM (-10) demonstrates weak activity in the developing gonads 48 hours post-transfection coincident with Lhx9 expression. The activity did not increase after 24 hours of further development. Changes in Lhx9 expression have also been implicated in gonad differentiation and we have not yet evaluated CRM(-10) at these later stages.

While our work demonstrates weak CRM (-10) activity in the bipotential gonad, its role in differentiation needs to be determined. Future work will investigate whether other CRMs will be required to help drive Lhx9 expression during gonad formation.

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