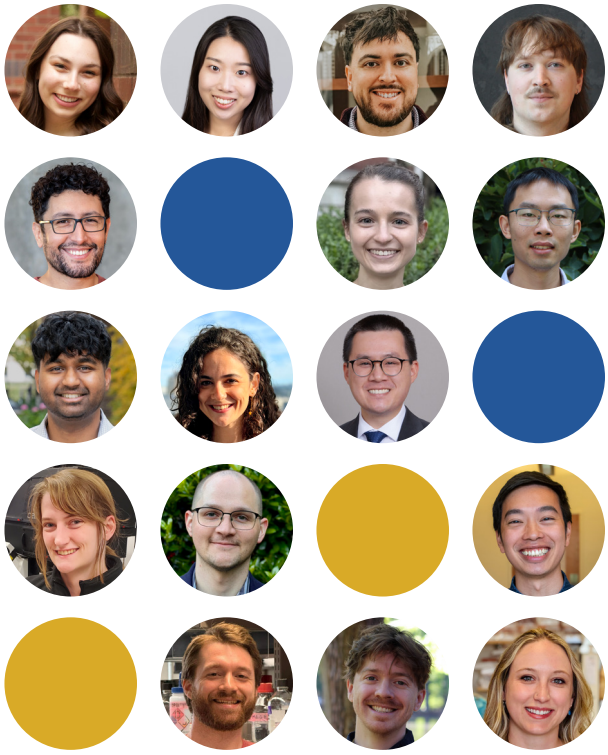
JANE COFFIN CHILDS

Fund for Medical Research



2026 JANE COFFIN CHILDS FELLOWS

KEVIN ARIS, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF AARON STREETS, Ph.D.
DEPARTMENT OF BIOENGINEERING
UNIVERSITY OF CALIFORNIA, BERKELEY

DNA usually forms a right-handed double-helix, known as the canonical B-form. But DNA can also be wound more tightly or more loosely, called positive and negative supercoiling. DNA supercoiling can impact how genes are regulated yet is difficult to study in its native cellular context.

During his thesis research in Zev Bryant, Ph.D.'s lab at Stanford University Kevin Aris examined how DNA supercoiling affects the energetics of DNA-protein interactions. He showed that DNA shape, or topology, affects how genes can be edited by different mechanisms. Specifically, Aris demonstrated that DNA supercoiling imparts physical reasons for why enzymes, such as Cas9 and Cas12a, naturally differ in how they bind to their DNA target.

As a Robertson Foundation - Jane Coffin Childs Fellow in Aaron Streets' lab at UC Berkeley, Aris will use his expertise in the CRISPR-Cas gene editing system not just to edit genes, but as a tool to measure DNA supercoiling across the genome in living cells. His goal is to build a genome-wide map of DNA shape to understand how it affects binding by gene regulators (like transcription factors). In addition to providing fundamental new knowledge about DNA shape in cells and gene regulation, Aris will investigate if and how this parameter is impacted in diseases such as cancer.

FELLOW

MINWOO BAE, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF JOSEPH MOUGOUS, Ph.D.
DEPARTMENT OF MICROBIAL PATHOGENESIS
YALE UNIVERSITY

FELLOW

Minwoo Bae, Ph.D., believes microbes contain a huge, largely unexplored supply of proteins that can be discovered and engineered for new medical and biotech uses. His past work found microbial proteins with unusual functions. Now, as a Robertson Foundation - Jane Coffin Childs Fellow, he plans to build "Trojan-horse" delivery systems inspired by microbial toxins that can smuggle next-generation antibiotics into bacteria.

As a graduate student in Emily Balskus's lab at Harvard, Bae identified gut microbial enzymes that break down dietary polyphenols. He first found an enzyme that metabolizes hydrocaffeic acid abundant in coffee, and later discovered multiple enzymes that convert ellagic acid into urolithin A, a compound linked to anti-aging and anti-inflammatory effects. This work helps explain how the gut microbiome changes the chemistry of what we eat and shows that microbes have evolved proteins that can carry out rare, complex chemical reactions.

Because many clever microbial tools come from bacteria competing with each other, Bae joined Joseph Mougous's lab at Yale to study and repurpose these systems. Antibiotic resistance is a major global health challenge, including for Gram-negative pathogens whose outer membranes are an impenetrable fortress keeping antibiotics out. His goal is to engineer microbial protein toxins that can pass through outer-membrane transporters, enabling antibiotics to be brought inside "by deception." He's especially interested in a pathogen associated with colorectal cancer, and his work could support both new antibiotic development and potential new strategies related to colorectal cancer.

JENNA BEYER, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF RYAN MEHL, Ph.D.
DEPARTMENT OF BIOCHEMISTRY AND BIOPHYSICS
OREGON STATE UNIVERSITY

FELLOW

Jane Coffin Childs Fellow Jenna Beyer, Ph.D. believes that we are in a golden age of chemical biology, which is a field that uses chemical approaches to manipulate and understand complex biological systems. The illuminating tools that Beyer developed during her Ph.D. research and is developing during her Robertson Foundation – Jane Coffin Childs Fellowship are unquestionably helping this field shine bright.

During her graduate work in George Burslem's lab at the University of Pennsylvania she generated a new way to edit protein sequences directly inside living mammalian cells. This method quickly adds chemical labels, like biotin or fluorescent dyes, to proteins, which helps researchers study them using tools like pull-down assays or microscopy. Compared with older approaches, her method works faster, lets scientists control timing more precisely, and avoids using large tags or antibodies that can interfere with a protein's normal function.

For the fellowship in Ryan Mehl's lab at Oregon State University, Dr. Beyer will develop new tools to study phosphorylation at specific spots on proteins. Phosphorylation is when a phosphate group is added to an amino acid, and it can happen at many places on many proteins. Most current methods are "all-or-nothing": they change a kinase, which is an enzyme that adds a phosphate group to a protein, and end up affecting lots of proteins at once. Beyer is creating a way to add a stable phosphate mark at one chosen site on one protein inside mammalian cells—more like a precise scalpel than a blunt tool. To show it works, she will first study how Protein Kinase A (PKA) may help protect against cardiovascular disease. Beyer's approach should also help researchers understand the role of phosphorylation in many other diseases.

ANNALISE BOND, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF JANELLE AYRES, Ph.D.
NOMIS CENTER FOR IMMUNOBIOLOGY AND MICROBIAL PATHOGENESIS
SALK INSTITUTE FOR BIOLOGICAL STUDIES

FELLOW

There are two ways the body survives an infection, the immune system can kill the germ, or the body can reduce harm from the infection even if the germ isn't eliminated. Annalise Bond, Ph.D., created a new research tool during her Ph.D. that improved our understanding of how immune cells identify and destroy targets. As a Jane Coffin Childs Fellow, she will now focus on the second strategy—helping the body tolerate infection and limit damage, known as "cooperative defense".

During Bond's graduate work in Meghan Morrissey's lab at UC Santa Barbara, she studied how macrophages (immune cells that act as the first responders) pick out pathogens among many healthy cells. She realized that the field lacked a tool to precisely control the duration and intensity of macrophage signaling, so she designed a synthetic, light-activated switch to turn on the signal. Using it, she showed that earlier activation can "prime" macrophages to engulf more of their target (in her experiments, cancer cells). She also found this priming works through a fast mechanism and a longer-lasting one, making the effect both quick and durable. These insights could help researchers design better ways to regulate immune responses, including against cancer.

Much less is known about the mechanisms of cooperative defense, which also means that this strategy remains essentially untapped in terms of therapeutic interventions. Dr. Bond will shift her studies to cooperative defense in Janelle Ayres, Ph.D.'s lab at the Salk Institute using a mouse model of sepsis. By analyzing neural-system signaling that correlates with survival, Bond is uncovering how the nervous and immune systems communicate to help the host survive an infection. In addition to discovering fundamental principles about cooperative defense, her work may lead to new ideas for improving outcomes for people with sepsis.

MATTHEW CAPEK, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF NICHOLAS BELLONO, Ph.D.
DEPARTMENT OF MOLECULAR AND CELLULAR BIOLOGY
HARVARD UNIVERSITY

FELLOW

Matthew Capek, Ph.D. believes that by examining how organisms sense and respond to their closest neighbors we can deepen our understanding of basic biology while also gaining insight into human health, disease, and ecosystem stability. During his graduate research he uncovered the distinct ways through which flies sense and adapt to environmental temperatures. Now, as a Jane Coffin Childs Fellow, Capek will investigate the adaptation and cooperation between some of the first plant and animal pioneers to transition from living in the waters to living on land.

As a graduate student in Marco Gallio, Ph.D.'s lab at Northwestern University, Capek showed how responses to temperature evolved in fly species hailing from different environments, from temperate forests to hot deserts. Flies from mild climates avoid heat and have molecular differences in their receptors that directly sense temperature. Mojave Desert flies are instead attracted to heat, and this shift in behavior arises from a change in how the brain processes and interprets the signal. He also studied the cold-adapted fly Chionea alexandriana, showing that they generate heat in response to rapid cold challenges, carry molecular changes in pathways to cope with stress, and produce antifreeze proteins that prevent freezing in sub-zero temperatures.

For his fellowship in Nicholas Bellono's lab at Harvard University, Capek will focus on understanding how interactions between mosses and springtails established the first terrestrial ecosystems. In water, moss sperm can swim to eggs to achieve fertilization, but life on land makes that journey far more difficult. Springtails help mosses reproduce on land by carrying sperm between moss sex organs. Capek will examine how mosses compel springtails to facilitate their reproduction, and determine what benefit motivates the springtails' efforts. He predicts that understanding this ancient plant-animal cooperation will yield a new framework for understanding how molecular communication drives the evolution of complex life.

LAURA BLASCO-CHAMARRO, Ph.D.

HOPE FUNDS FOR CANCER RESEARCH – JANE COFFIN CHILDS FELLOW

LABORATORY OF MARIELLA FILBIN, M.D., Ph.D.
DEPARTMENT OF PEDIATRIC ONCOLOGY
DANA-FARBER CANCER INSTITUTE

FELLOW

Pediatric brain tumors arise in the developing brain, yet how they interact and communicate with their neighboring cells to promote tumor growth is not well understood. In Laura Blasco-Chamarro's previous research she discovered how neural stem cells pause cell division to maintain a quiescent state. Now, as a Hope Funds for Cancer Research – Jane Coffin Childs Fellow, Blasco-Chamarro will study how pediatric brain tumors, called gliomas, misuse normal developmental programs to trigger abnormal cell division.

As a graduate student in Isabel Fariñas' lab at the University of Valencia, Blasco-Chamarro explored how localized cues support neural stem cell (NSC) quiescence. She found that in response to a specific signal, NSCs secrete a supportive material, called the extracellular matrix (ECM) that induces quiescence. This matrix then activates specific proteins called YAP and TAZ, which move into the nucleus and turn on genes that reinforce the resting state. Her work showed how a cell's environment can push neural stem cells toward staying inactive.

In Dr. Mariella Filbin's lab at Dana-Farber Cancer Institute, Blasco-Chamarro will study the opposite process: how pediatric high-grade gliomas activate developmental signaling to keep dividing. She will map how tumor cells interact with surrounding cells in the tumor microenvironment and identify the signals that promote tumor growth. She expects that blocking these support signals could slow or stop tumor growth. This research could lead to new treatments for pediatric high-grade gliomas and offer a broader strategy for targeting similar, lineage-specific signaling pathways in other cancers.

RUOYU (ROY) CHEN, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF CATHERINE DULAC, Ph.D.
DEPARTMENT OF MOLECULAR AND CELLULAR BIOLOGY
HARVARD UNIVERSITY

FELLOW

Biology often reuses the same basic "building blocks" for similar functions. Dr. Ruoyu Chen studied RNA–protein clusters (called RNP granules) in fruit fly reproductive cells and made a surprising discovery that changed how scientists think these granules work. Because similar granules also play important roles in neuronal functions, Chen became interested in neuroscience and now wants to study the cellular and molecular underpinnings of the drive for social interaction.

As a graduate student in Ruth Lehmann's lab at the Whitehead Institute for Biomedical Research, Chen studied RNP granules, which are small cell compartments made of RNA and RNA-binding proteins. Scientists had long thought these granules turn off protein production from the RNAs inside them. Chen found the opposite in fly germ cells: the RNAs in these granules are actively being used to make proteins. This was a paradigm-shifting finding for the field and led Chen to consider other areas of biology where this kind of translational regulation is important.

Since similar RNA granules help move RNAs around long nerve cells and make proteins in specific places, Chen began focusing on how these processes affect brain function and behavior. As a Jane Coffin Childs Fellow in Catherine Dulac's lab at Harvard, he will study the brain circuits and molecular signals that control the desire for social interaction. He will use social isolation to probe these systems, with the hope of understanding why loneliness is linked to poor health and some mental health disorders.

RYAN CHOW, M.D., Ph.D.

JJJ CHARITABLE FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORIES OF PRANAM CHATTERJEE, Ph.D.
DEPARTMENT OF BIOENGINEERING AND
KATALIN SUSZTAK, M.D., Ph.D.
DEPARTMENT OF MEDICINE
UNIVERSITY OF PENNSYLVANIA

FELLOW

As a hematology and oncology fellow, Ryan Chow, M.D., Ph.D., is often faced with the limitations of our current cancer therapies. His goal as a JJJ Charitable Foundation-Jane Coffin Childs Fellow is to build on his graduate work of identifying genetic insults to develop a new class of anti-cancer therapies that function by disarming oncogenic transcription factors.

In Sidi Chen's lab at Yale University, Chow created new CRISPR screening methods to study which genes drive cancer. It should be noted that Chow's overwhelming research production as a graduate student cannot be completely covered in this space, but we'll look at a few examples. For example, he used AAV viruses to deliver CRISPR tools into animals and find tumor-suppressor genes in glioblastoma and liver cancer. Then, Chow adapted his CRISPR screens to enable stepwise mutation of multiple genes which can capture the sequential nature of mutations, for instance in non-small cell lung cancer. While Chow's findings emphasize the power of CRISPR screens in revealing novel cancer genetics, he quickly realized the impracticality of this modality for cancer therapies.

Now in Pranam Chatterjee's and Katalin Susztak's labs at the University of Pennsylvania, Chow is taking a different approach: designing brand-new peptides to target cancer-driving transcription factors. These proteins are important targets but have been hard to drug with traditional medicines because they are flexible and located in the cell nucleus. Chow plans to use deep learning to design peptides that bind these transcription factors and test whether they can rewrite cancer gene programs and eliminate tumors.

ADAM FENTON, Ph.D.

ACHELIS AND BODMAN FOUNDATION– JANE COFFIN CHILDS FELLOW

LABORATORY OF SAMARA RECK-PETERSON, Ph.D.
DEPARTMENT OF BIOCHEMSITRY AND BIOPHYSICS
WEILL CORNELL MEDICINE

FELLOW

Kinesins are motor proteins that "walk" along microtubules to carry cargo inside cells. Adam Fenton, Ph.D., studied how kinesins move mitochondria (the cells' power source) to where cells need energy. As an Achelis and Bodman-Jane Coffin Childs Fellow, he will now investigate a surprising possibility: that a neuron-specific kinesin also helps cancer cells invade other tissues.

In Erika Holzbaur's and Thomas Jongens's labs at the University of Pennsylvania, Fenton found several key things about kinesin motors and mitochondrial transport in neurons. In addition to his work on mitochondrial fission in neurons, he learned that a kinesin is turned on by a protein that connects it to mitochondria. He found that some ALS-linked kinesin mutations make this kinesin too active by removing a normal "off" mechanism.

Next, in Samara Reck-Peterson's lab at Weill Cornell Medicine, Fenton will study cancer progression. Early evidence suggests cancer cells may increase organelle-transport systems that are usually only active in neurons. He will test how a neuron-specific kinesin and its partner proteins contribute to cancer cell invasion and test ways to block this process in tumor organoid models. Fenton anticipates that his research will uncover novel roles for organelle transport in cancer cell biology and may illuminate the path toward new cancer therapies.

CHRIS GIULIANO, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF KIVANÇ BIRSOY, Ph.D., (JCC CLASS OF 2010)
LABORATORY OF METABOLIC REGUALTION AND GENETICS
THE ROCKEFELLER UNIVERSITY

FELLOW

What does microbial metabolism have to do with finding cancer therapies? Dr. Chris Giuliano thinks studying how microbes share and trade nutrients can help reveal novel ways to treat cancer. His goal is to find important metabolic "give-and-take" interactions between tumors and healthy tissues that could be targeted with drugs.

As a graduate student in Sebastian Lourido, Ph.D.'s lab at the Whitehead Institute for Biomedical Research, Giuliano studied the parasite *Toxoplasma gondii*. Because very little of its genome had been tested for genes that help it cause disease, he used a genome-wide CRISPR screen during infection and identified 300+ previously unknown virulence factors. He then studied three of these genes in depth and suggested a possible way to block infection.

Now as a Jane Coffin Childs Fellow in Kivanç Birsoy's lab at The Rockefeller University, Giuliano will shift his focus to metabolic exchange across human organs. He wants to create an unbiased method to detect metabolic exchange between organs and tissues, using an idea inspired by microbial metabolism. He will apply this novel approach to cancer—studying how tumors interact metabolically with nearby cells, with immune cells, and even with distant organs—to uncover metabolic vulnerabilities that could be disrupted for therapy.

JAKE KLEMM, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF DON FOX, Ph.D., (JCC CLASS OF 2007)
DEPARTMENT OF PHARMACOLOGY AND CANCER BIOLOGY
DUKE UNIVERSITY

FELLOW

Fly and worm researchers have long debated the unique advantages of their favorite model organism. Dr. Jake Klemm finds that flies provide a powerful genetic toolkit to study animal physiology. He previously used flies to find unexpected roles for proteins involved in cell death processes in tissue repair and regeneration. As a Robertson Foundation - Jane Coffin Childs Fellow, he will use fruit flies and mammalian cells to study how tRNAs, small RNA molecules that act as a physical adaptor during protein synthesis, help control which proteins are made in specific tissues.

Klemm developed his appreciation for flies during his thesis research in Rob Harris, Ph.D.'s lab at Arizona State University. Klemm built a fly model, using the fly wing, to study the tissue response to necrotic injury (a type of cell death different from apoptosis). He unexpectedly found that necrosis can trigger apoptosis in cells far away from the injury, something he called "necrosis-induced apoptosis." He also showed this process is required for tissue regeneration, and that enzymes called caspases are important for regeneration. This suggested that molecules best known for killing cells can also help tissues regrow.

Now in Don Fox's lab at Duke University, Klemm will study "tissue-adapted" tRNAs, which may help regulate gene expression in a tissue-specific way. Instead of being just routine parts of the protein-making machinery, these tRNAs may influence what gets translated in different cell types. He will test this idea in both reproductive (germ) cells and specialized (differentiated) cells, aiming to build a strong model for understanding how tRNAs function in animal development and physiology.

FRANCESCO (FRANK) LANFRANCHI, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF TIRIN MOORE, Ph.D.
DEPARTMENT OF NEUROBIOLOGY
STANFORD UNIVERSITY

FELLOW

Francesco (Frank) Lanfranchi, Ph.D. is baffled by how little we truly understand about the neural mechanisms underlying behavior. As a Robertson Foundation - Jane Coffin Childs Fellow, Lanfranchi wants to help fill this knowledge gap by understanding the neural circuits that facilitate how internal goals are maintained and translated into action – one of the fundamental functions that neurological diseases disrupt.

During his graduate studies in Doris Tsao's lab at UC Berkeley, Lanfranchi investigated how visual information is transformed into meaningful object representations across mammalian species. Lanfranchi studied how brains turn visual input into recognizable objects. Comparing macaques with tree shrews, he found that tree shrews can show primate-like abilities such as recognizing objects and faces. This suggests that "hierarchical" visual processing is conserved, but more compact, in the smaller tree shrew brain.

In Tirin Moore's lab at Stanford University, Lanfranchi will study how the brain uses sensory information to guide actions—especially when sensory cues aren't available. For example, you can still write your name with your eyes closed because the goal is internally maintained. He will map the circuits that support this kind of goal-directed behavior in macaques, with the hope that understanding these normal circuits will help explain why internally driven actions are especially affected in Parkinson's disease.

JING LI, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF SHIYU XIA, Ph.D., (JCC CLASS OF 2022)

DEPARTMENT OF CHEMICAL AND BIOMOLECULAR ENGINEERING, COLLEGE OF CHEMISTRY

UNIVERSITY OF CALIFORNIA, BERKELEY

FELLOW

Cells are constantly threatened by pathogens, so they need ways to sense danger and turn on defenses at the right time. In her graduate work, Jing Li, Ph.D., elucidated elegant and mechanistic details of how bacteria know when to fight back against invading bacteriophages. As a Robertson Foundation - Jane Coffin Childs Fellow, Li will leverage insight from her previous studies to reengineer human cells to explore the programmable activation of immune defense.

As a graduate student in Longfei Wang's lab at Wuhan University, Li revealed how bacterial defense systems sense when to activate. She solved a number of novel and insightful structures of the GajA/GajB proteins with different cofactors and substrates. Her structures showed that when ATP is abundant, GajA remains in a closed, inactive state. However, when ATP is depleted during phage infection, GajA converts into an open state which binds to and cleaves DNA, thereby activating GajB and leading to prokaryotic cell death. This research helped Li appreciate the role of small molecules, such as ATP, to act as information-rich signals that gate biological decisions.

Now in Shiyu Xia's lab at UC Berkeley, Dr. Li plans to bring similar "small-molecule control" ideas into human cells. She will build synthetic protein circuits to reprogram the cGAS–STING immune pathway so she can choose when it turns on and how strongly it responds. Because cGAS–STING is involved in cancer, aging, autoimmune disease, and infections, this controllable system could help researchers understand these conditions and eventually support new therapies.

MENGYAO LI, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF XIAO WANG, Ph.D.
BROAD INSTITUTE OF MIT AND HARVARD
MASSACHUSETTS INSTITUTE OF TECHNOLOGY

How does a single cell develop into a complete and complex organism? Dr. Mengyao Li is fascinated by the question of how cells, despite sharing an identical genome, achieve such distinct identities and tissue types through epigenetic regulation. During her graduate studies in Fuchou Tang's lab at Peking University and Kehkooi Kee's lab at Tsinghua University, she traced the epigenetic dynamics and lineage differentiation that guide cell fate decisions during early mammalian embryogenesis, utilizing an ultra-sensitive long-read sequencing-based chromatin accessibility profiling method she developed for scarce, single-cell-input samples. This method enabled her to dissect the epigenetic regulation of repetitive elements and the X chromosome, systematically delineating the cell-type-specific transcription factor regulatory networks that drive early development.

As a Robertson Foundation – Jane Coffin Childs Fellow in Xiao Wang's lab at the Broad Institute and MIT, she seeks to decipher the hidden spatial code governing how cells translate RNA into functional proteins. By exploring how the subcellular organization of transcripts dictates their translation kinetics, she aims to uncover how dynamic shifts in RNA translational efficiency ultimately orchestrate cellular states, tissue architecture, and disease.

FELLOW

KE LIANG, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF TOM RAPOPORT, Ph.D.
DEPARTMENT OF CELL BIOLOGY
HARVARD MEDICAL SCHOOL

FELLOW

Ke Liang, Ph.D. studies how proteins are transported in cells and why these processes matter for human disease. In her earlier work, Liang used structural biology to map major protein-transport machines. As a Jane Coffin Childs Fellow, she will focus on how proteins are transported into peroxisomes, small organelles found in the cytoplasm that act as the cell's "cleanup crew," breaking down and detoxifying substances generated by metabolic processes.

As a graduate student in Yigong Shi's and Zhen Yan's labs at Westlake University, Liang's research illuminated different mechanisms for protein transport across organelle boundaries. She made key contributions to determining the structures of the frog cytoplasmic, inner, and nuclear rings of the nuclear pore complex (NPC), providing unprecedented insights of the organization of this complex. Additionally, Liang's structures of chloroplast protein import complexes in land plants and green algae demonstrated how related transport systems are conserved and specialized across species.

Now as a JCC Fellow in Tom Rapoport's lab at Harvard Medical School, Liang will investigate protein import into peroxisomes. Peroxisomes are unusual because they can import fully folded proteins using receptors that shuttle in and out and must be extracted and recycled. Liang aims to clarify how these steps work. Because failures in peroxisomal import cause serious diseases with few or no treatments, her work could also point toward new therapeutic ideas.

JIALIN LIU, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF TZUMIN LEE, M.D., Ph.D.
LIFE SCIENCES INSTITUTE
UNIVERSITY OF MICHIGAN

FELLOW

Jialin Liu, Ph.D., has been building "snapshots" of brain development using single-cell and spatial gene-expression data. But because those datasets were incomplete, he often had to infer how development unfolded rather than directly observe it. Now he aims to create a new way to track neuronal development in space over time inside an intact brain.

In Joshua Welch, Ph.D.'s lab at The University of Michigan Liu developed computational tools to analyze cell states based on gene expression. Examples include a pipeline that jointly analyzes single-cell sequencing data from a variety of experiments and can be used by all scientists, as well as a model for inferring spatial and temporal dynamics of cell states from spatial transcriptomic data. Liu's research has provided incredibly useful and broadly accessible tools for analyzing cell state based on gene expression, which can be used to infer cell state transitions among other purposes.

In Tzumin Lee's lab at The University of Michigan, Liu will generate the kind of data his models need: 3D spatial transcriptomics across multiple time points during fruit fly brain development, both in normal flies and in flies with targeted genetic changes. With these richer datasets and new analysis tools, he hopes to produce a "ground-truth" map of how cell lineages and brain wiring develop over time—more like watching the whole movie rather than predicting from a few frames. Ultimately, he hopes to reveal how the brain wires itself and how genetic changes alter that process.

ADAM LOWET, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF MICHAEL YARTSEV, Ph.D.
DEPARTMENT OF BIOENGINEERING AND NEUROSCIENCE
UNIVERSITY OF CALIFORNIA, BERKELEY

FELLOW

Adam Lowet, Ph.D. is fascinated by the neural and computational basis of social behavior in both health and disease. During his graduate work, Lowet used insights from AI to uncover a novel way in which the brain learns from rewards and punishments. As a Jane Coffin Childs Fellow, Lowet will now investigate the computational and biological mechanisms underlying social decision-making using an unorthodox model system: the Egyptian fruit bat, a highly social animal.

Lowet's graduate research in Naoshige Uchida's lab at Harvard University was motivated by the observation that many AI algorithms are significantly improved when considering the entire probability distribution of outcomes rather than just their mean value. Lowet thought this principle might apply to how our brains work and investigated this hypothesis in the context of the mesolimbic dopamine system. Lowet demonstrated that the brain indeed encodes more than just the mean and uses this distributional information to speed up learning. Surprisingly, Lowet discovered that the upper and lower tails of reward distributions are encoded in different types of neurons, suggesting brain information processing is organized in a more detailed way than previously understood.

Now in Michael Yartsev's lab at UC Berkeley, Lowet will use the Egyptian fruit bat, an ultrasocial mammal, as a model for studying social decision behavior. Lowet will record behavior and neural activity while groups of bats forage collectively and compare these to normative models of social decision-making from machine learning and behavioral ecology. Ultimately, Lowet hopes that this foundational research into how healthy brains coordinate with others will eventually inform approaches to disorders where this ability is compromised.

HEANKEL LYONS, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF AARON GITLER, Ph.D.
DEPARTMENT OF GENETICS
STANFORD UNIVERSITY

FELLOW

Heankel Lyons, Ph.D. became motivated to do biomedical research while growing up due to a family member developing a rare neurodegenerative disease. As a JCC Fellow, she will take her expertise from her graduate work on how biomolecular condensates regulate gene activity to ask how condensates regulate neuronal gene expression, how these processes shape normal cell biology, and how their dysregulation leads to brain disease.

As a graduate student in Ben Sabari's lab at UT Southwestern, Lyons uncovered fundamental principles into how biomolecular condensates, membrane-less compartments that gather specific molecules, help regulate transcription. She found that condensates formed by a protein called MED1 recruit RNA polymerase II and helpful regulators while keeping out inhibitors. Impressively, Lyons also identified amino-acid "patterns" that determine which proteins get recruited, and showed how cancer fusion proteins exploit similar patterns to drive cancer-related gene programs.

Now as a JCC Fellow in Aaron Gitler's lab at Stanford University, Lyons returns to the subject that originally spurred her interest in science: neurodegeneration. She'll focus on a central protein in amyotrophic lateral sclerosis (ALS), named TDP-43. TDP-43 forms condensates, and most research in the neurodegeneration field has focused on TDP-43's role as an RNA-binding protein, yet this protein was originally discovered as a DNA-binding protein. Lyons will use her expertise in transcription and condensates to define TDP-43's role in neurons and investigate how transcriptional dysregulation involving TDP-43 contributes to ALS.

KIRA MARSHALL, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF MATT GIBSON, Ph.D., (JCC CLASS OF 2002)
STOWERS INSTITUTE FOR MEDICAL RESEARCH

Dr. Kira Marshall has combined her desire to do rigorous molecular research with her passion to preserve threatened species. As a graduate student, Marshall provided keen insights into the molecular mechanisms of spermatogenesis in marsupial and placental mammals. Now, she'll return to the sea to gain a better understanding of coral reproduction, to uncover ways to mitigate the rapidly declining coral populations around the world.

Marshall's graduate studies in Bluma Lesch's lab at Yale University provided fine detail into gene expression during sperm development – a deeply conserved process with obvious implications for fitness. By characterizing this process in the marsupial opossum and in the mouse, a placental mammal, Marshall was able to compare spermatogenesis across the placental-marsupial split. She uncovered a gene program that's conserved in both species, as well as genes that appear to contribute to the placental mammalian lineage. Marshall's findings furthered our understanding of germ cell biology as well as infertility.

As a JCC Fellow at the Stowers Institute in Matt Gibson's lab, Marshall will extend her research on reproduction to a different branch of the evolutionary tree by studying Acropora millepora, a hermaphroditic free spawning coral species. These organisms release bundles of eggs and sperm into the water, yet self-fertilization is exceedingly rare, suggesting that there are mechanisms that control gamete attraction and compatibility. Marshall will investigate the fertilization of this coral species and decipher the means by which they ensure proper mating. Ultimately, Marshall aims for her research to have a direct and positive effect on marine ecosystems by helping to preserve threatened coral species.

FELLOW

SASHA McDOWELL, Ph.D.

MERCK – JANE COFFIN CHILDS FELLOW

LABORATORY OF JOSEFINA DEL MÁRMOL, Ph.D.
DEPARTMENT OF BIOLOGICAL CHEMISTRY AND MOLECULAR PHARMACOLOGY
HARVARD MEDICAL SCHOOL

FELLOW

Taste can be instructive: for example, the taste of a calorie-rich pastry is highly pleasurable while spoiled food is off-putting. Sasha McDowell, Ph.D.'s graduate research revealed the molecular details of how fruit flies are repelled by too much salt yet attracted to just the right amount. As a Merck-Jane Coffin Childs Fellow McDowell will now explore a related mechanism for how mosquitoes are attracted to the odor of their human prey.

During her thesis research in Michael Gordon's lab at the University of British Columbia, McDowell investigated how fruit flies taste and respond to salt. She identified the first salt-specific receptor in *Drosophila melanogaster* and demonstrated how this ionotropic receptor (IR) functions to avoid high salt concentrations. McDowell then investigated a related receptor which is involved in salt attraction. She found that the receptor activity is tuned in response to prior salt consumption. Together, McDowell's studies reveal how IRs are involved in both salt attraction and repulsion to balance overall dietary intake.

Near the end of her graduate research McDowell contracted dengue fever through a mosquito bite. Naturally, she was curious about what attracts mosquitoes to their human prey, which will be her focus in Josefina del Mármol's lab at Harvard. Previous research had shown that a mosquito IR is involved in their attraction, though it remains unclear what component of human odor this receptor detects. Dr. McDowell aims to discover this missing attractant, understand how the receptor binds to the attractant, and find inhibitors that prevent mosquitoes' attraction to humans. In addition to providing fundamental information about mosquito biology, McDowell's research may reveal new strategies for bio-control efforts.

SANDRA NAKANDAKARI-HIGA, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF MINSOO KIM, Ph.D.
DEPARTMENT OF MICROBIOLOGY AND IMMUNOLOGY
UNIVERSITY OF ROCHESTER MEDICAL CENTER

FELLOW

Dr. Sandra Nakandakari-Higa wants to understand how a cell's fate and function are determined. This process is not shaped in isolation; rather, it is shaped through continuous interactions with neighboring cells, forming dynamic networks of communication that orchestrate development, homeostasis, and immune responses. As a Jane Coffin Childs Fellow, she'll use Labeling Immune Partnerships by SorTagging Intercellular Contacts (LIPSTIC), an approach she improved in her graduate work, to evaluate the persistence of memory T cells within the lung.

Using LIPSTIC, Nakandakari-Higa studied how brief interactions between immune cells and their cellular partners shape lasting immune responses during her graduate work in Gabriel Victora's lab at The Rockefeller University. Nakandakari-Higa redesigned LIPSTIC so it no longer depends on one specific receptor–ligand pair. This made it broadly applicable for many types of cell interactions. She used this "universal" LIPSTIC to follow how dendritic cells activate T cells and how virus-specific T cell interactions change over time, and the tool can now help other researchers track immune contacts in detail.

As a JCC Fellow in Minsoo Kim's lab at the University of Rochester, Nakandakari-Higa will focus on a key aspect of protective immunity: the generation and persistence of memory T cells in the lung. Infection with respiratory viruses generates these T cells and provides protection against reinfection. However, over time the numbers of these T cells wane which limits their effectiveness. Nakandakari-Higa will use her universal LIPSTIC technology to map the cellular interactions of memory T cells in the lung, and to analyze how those interactions change. She'll also invert LIPSTIC to determine how signals delivered by the local microenvironment contribute to T cell survival. Her research may provide new clues that could inform ways to make vaccine protection last longer.

KANGBO NG, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF RUTH LEHMANN, Ph.D.
DEPARTMENT OF BIOLOGY
WHITEHEAD INSTITUTE FOR BIOMEDICAL RESEARCH
MASSACHUSETTS INSTITUTE OF TECHNOLOGY

FELLOW

KangBo Ng, Ph.D., has long been fascinated by how somatic cells, the non-reproductive cells of the body, and germ cells, the reproductive cells, work together to ensure the proper development of an organism. During his graduate research, Ng studied how these cells organize themselves in space to build the embryo. Now, as a Robertson Foundation - Jane Coffin Childs Fellow, Ng will investigate how somatic and germ cells exchange metabolic resources to help kick-start embryonic development.

Ng's thesis research in Nathan Goehring's lab at the Francis Crick Institute addressed how cell polarity shapes animal development. Because polarity systems are used across many different cellular contexts, they must be able to respond sensitively to spatial cues while still producing stable outcomes. Ng demonstrated that oscillatory polarity feedback, coupled to the cell cycle, allows cells to resolve these seemingly contradictory requirements. He also found that mechanical flows generated during cell division can directly transport polarity proteins to organize the embryo. Altering these flows changed division patterns, suggesting a simple mechanism by which embryos could generate different body plans.

In Ruth Lehmann's lab at the Whitehead Institute, Ng will focus on metabolic communication between somatic and germ cells. Germ cells switch between phases of rest, division, and quality control, and somatic cells appear to help control these transitions, but the underlying mechanism remains unclear. Ng hypothesizes that somatic cells may orchestrate these processes by transferring metabolic resources to germ cells. His work could reveal new principles of embryo development and inform future research into reproductive health.

JORDAN NGO, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF REBECCA VOORHEES, Ph.D.
DEPARTMENT OF BIOLOGY AND BIOLOGICAL ENGINEERING
CALIFORNIA INSTITUTE OF TECHNOLOGY

FELLOW

Jordan Ngo, Ph.D. is interested in uncovering the mechanistic principles that govern organelle biogenesis and membrane assembly in both normal physiology and human disease. As a graduate student, Ngo provided important insight into how extracellular vesicles are formed and how the plasma membrane is repaired. As a Robertson Foundation – Jane Coffin Childs Fellow, Ngo will continue to study organelle biogenesis, investigating how peroxisomes, membrane-bound organelles that play essential roles in human physiology, form.

During Ngo's thesis research in Randy Schekman's lab at UC Berkeley, he made important discoveries around extracellular vesicles and the plasma membrane. First, Ngo discovered that exosomes, a specific subtype of extracellular vesicles, form in response to plasma membrane damage and that the protein Annexin A6 is crucial for this process. Then, he demonstrated that the selective autophagy receptor p62 is important for sorting protein and RNA cargo into exosomes. Finally, he identified sorcin as a scaffold that couples Annexin A11 recruitment to ESCRT-III assembly for plasma membrane repair.

For his Robertson Foundation – Jane Coffin Childs Fellowship in Rebecca Voorhees's lab at Caltech, he will search for genes that control peroxisome assembly and build a new test to study how early peroxisome-related vesicles form. Because defects in peroxisome formation cause serious disorders, such as Zellweger spectrum disorders, and have been implicated in cancer progression, this work could clarify how peroxisome problems contribute to disease.

ANDREAS OBERS, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF SHRUTI NAIK, Ph.D.
DEPARTMENT OF IMMUNOLOGY AND IMMUNOTHERAPY
ICAHN SCHOOL OF MEDICINE AT MOUNT SINAI

FELLOW

Andreas Obers, Ph.D. investigates the biological mechanisms that determine whether tissues recover after injury and inflammation or become trapped in persistent, maladaptive states that contribute to chronic disease and aging. Inspired by his graduate research showing that biological responses are shaped by local tissue environments and prior experiences, Obers now explores how a key regulator of cellular stress responses influences the balance between tissue repair and persistent dysfunction.

Obers conducted his doctoral research in the laboratories of Laura Mackay and Christoph Wilhelm through a joint program between the University of Melbourne and the University of Bonn. In his first-author work, he revealed that retinoic acid, a metabolite derived from vitamin A, shapes the durability and distribution of immune surveillance across tissues. In related work, he contributed to the discovery that immune cells occupying the same tissue can adopt distinct functional identities, allowing them to either promote tissue protection or contribute to disease.

Now in the laboratory of Shruti Naik at Mount Sinai, Obers studies a key regulator of cellular stress responses whose expression is consistently elevated in aged tissues. Although it is widely associated with aging, scientists are only beginning to explore whether it has functions beyond its classical role. By investigating how inflammation reshapes its localization and activity within cells, Obers aims to uncover how tissues transition from successful repair to persistent dysfunction and chronic disease.

BELÉN PACHECO-FIALLOS, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF SEYCHELLE VOS, Ph.D.
DEPARTMENT OF BIOLOGY
MASSACHUSETTS INSTITUTE OF TECHNOLOGY

FELLOW

Belén Pacheco-Fiallos, Ph.D. studies how gene activity is regulated. Although scientists know the structures of many individual parts, a major next step is learning how these molecular "machines" work together. In graduate school, she studied how cells export the right messenger RNAs (mRNAs) from the nucleus out of roughly 20,000 different transcripts. As a Jane Coffin Childs Fellow, she will now study how cells prevent unhelpful ("non-productive") transcription from happening everywhere in the genome.

In Clemens Plaschka's lab at the Research Institute of Molecular Pathology in Vienna, Austria, Pacheco-Fiallos studied selective mRNA transport via the Transcription and Export (TREX) complex. She demonstrated that TREX lives up to its name by being an enormous oligomeric complex that's approximately 2 megadaltons in size. Using cryo-electron microscopy and tomography Pacheco-Fiallos revealed how TREX selectively recognizes mature mRNA-protein complexes for export to the cytosol and eventual translation of the transcript. Her research stresses the importance of studying molecular machines in relevant conditions and at the appropriate level of molecular complexity.

Pacheco-Fiallos will continue this approach to tackle a different selectivity problem in gene expression at Seychelle Vos's lab at MIT. There she will study a different selectivity problem: RNA polymerase II makes full mRNAs from genes, but it also makes very short, non-coding transcripts at enhancers and promoters. How the cell stops transcription in those regions isn't well understood. She will test whether a Restrictor complex helps recognize and shut down this inappropriate transcription, using integrative structural biology to figure out how the complex assembles and works.

IAN PRICE, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF BOB GOLDSTEIN, Ph.D.
DEPARTMENT OF BIOLOGY
UNIVERSITY OF NORTH CAROLINA AT CHAPEL HILL

FELLOW

Jane Coffin Childs Fellow Ian Price, Ph.D., studies how living things survive extreme stress. He focuses on tardigrades, microscopic animals that can survive the vacuum of space, extreme radiation, and being dried out completely for years before rehydrating and carrying on living.

For his thesis research in Wen Tang's lab at The Ohio State University Price examined protein-RNA assemblies called germ granules in *C. elegans*. He discovered novel proteins that regulate germ granule assembly, demonstrated that germ granules contribute to developmentally-appropriate gene silencing, and defined the molecular interactions that scaffold germ granule assembly. This work highlighted how powerful model organisms are for discovering new biology.

As a JCC Fellow in Bob Goldstein's lab at UNC, Chapel Hill, Price is investigating extremophile tardigrades, colloquially known as water bears. Tardigrades can tolerate drastic conditions including complete dehydration and levels of radiation that are one thousand times higher than what humans can survive. Previous work from the Goldstein lab demonstrated that a secreted protein is crucial for desiccation tolerance in tardigrades. Price suspects there are more protective secreted proteins and is searching for them to understand how they work. If some of these proteins protect other organisms too, they could be useful for preserving cells, medicines, and other medical materials.

AMY PRICHARD, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF LENA PERNAS, Ph.D.
DEPARTMENT OF MICROBIOLOGY IMMUNOLOGY AND MOLECULAR GENETICS
UNIVERSITY OF CALIFORNIA, LOS ANGELES

FELLOW

Amy Prichard, Ph.D., aims to understand how viruses reorganize their host cells to protect themselves from host defenses. During her Ph.D. research, Prichard examined how a family of bacteriophage, viruses that infect bacteria, build replication compartments in bacterial cells to shield viral genome replication from host defenses. Now, as a Jane Coffin Childs Fellow, Prichard will focus on how viruses that infect animals create a different type of replication compartment and how that may also allow them to evade host defenses during infection.

Prichard's graduate research in the labs of Joe Pogliano, Ph.D., and Elizabeth Villa, Ph.D., at UC San Diego investigated a family of bacteriophage that they named Chimalliviridae. This viral family is unique in that they form a nucleus-like replication compartment within bacteria. Prichard defined the core genes encoded by these bacteriophage, including chimallin, the namesake of this family, which is the major structural protein that forms the replication compartment. Additionally, Prichard and her colleagues revealed how chimallin self-assembles to form this subcellular compartment. Overall, her work clarified which viruses share this unique lifestyle and how these viruses protect their genome replication from the host.

During her JCC Fellowship in the lab of Lena Pernas, Ph.D., at UCLA, Prichard will investigate a different type of virus-induced subcellular compartment. Nodaviruses, such as Flock House Virus and Nodamura Virus, are unique in that they form replication compartments on the outer mitochondrial membrane. Prichard suggests that this location is an unusual "choice" for a viral replication site since mitochondria are home to an essential anti-viral signaling protein. Also, because mitochondria have their own genomes, they use cellular resources that other organelles do not, which could put them in direct competition with these viruses. Her project will examine how and why Nodaviruses replicate in this high-risk location, offering a clear example of how our cells' organelles, such as mitochondria, can help us fight off viral infection, and how viruses attempt to subvert these defenses by hiding their replication within subcellular compartments. By better understanding the ways viruses hijack our cells, scientists can build a biological toolkit to gain new ways to prevent disease.

ROHITH RAJASEKARAN, Ph.D.

ROBERTSON FOUNDATION – JANE COFFIN CHILDS FELLOW

LABORATORY OF KOLE ROYBAL, Ph.D., (JCC CLASS OF 2013)
DEPARTMENT OF MICROBIOLOGY AND IMMUNOLOGY
UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

FELLOW

Rohith Rajasekaran, Ph.D., wants to study how naturally evolved molecular systems can be redesigned to program new cellular behaviors. Rajasekaran's previous research repurposed a bacterial positioning system to turn mammalian cells into "two-way radios" that can send and receive biological information. Now, as a Robertson Foundation – Jane Coffin Childs Fellow, Rajasekaran will rewire T cell signaling in modular, tunable ways to improve their efficacy, durability, and safety in cancer immunotherapies.

Rajasekaran's thesis research in Scott Coyle's lab at the University of Wisconsin—Madison ported two bacterial proteins (MinD and MinE) into mammalian cells. MinD and MinE are normally involved in establishing bacterial polarity, but Rajasekaran repurposed them to control and pattern intracellular mammalian biology. He used them to track signaling (like kinase activity) and to organize processes such as condensate formation and actin filament growth. He also built genetic circuits that linked natural cellular processes to MinD/MinE signaling patterns, allowing those signaling "rhythms" (frequency and strength) to serve as a readout of cellular activity, revealing when genes turn on, how proteins are broken down, and how stem cells develop into different cell types.

In Kole Roybal's lab at UCSF, Rajasekaran will apply similar ideas to chimeric antigen receptor (CAR) T cell therapies. CAR T cancer therapies have shown great promise yet are only effective for a subset of patients and have limited duration for responding patients. Rajasekaran will build on the Roybal Lab's finding that oncogenic fusion genes enhance CAR T therapies, he will design synthetic fusion proteins that adjust the strength and timing of key signaling pathways in T cells. By linking signaling patterns to T cell behavior, he aims to create better, longer lasting, and safer CAR T therapies for more patients.

MOLLY SARGEN, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF BONNIE BASSLER, Ph.D.
DEPARTMENT OF MOLECULAR BIOLOGY
PRINCETON UNIVERSITY

FELLOW

Viruses have immense potential to influence the fates of individual cells, multicellular communities, and entire organisms. Nonetheless, vast gaps exist in our understanding of the interactions between viruses and their host cells. Molly Sargen, Ph.D., is specifically fascinated by how some viruses can co-exist with their hosts even though the interests of a virus and a cell are usually incompatible. Her goal is to use models of bacteria and their viruses (phages) to define the principles that drive host-virus interactions, including how each entity manipulates the interaction to its own advantage.

As part of her Ph.D. research in Sophie Helaine's lab at Harvard Medical School, she showed that phages that are embedded in *Salmonella* block other phages from infecting the same bacterium through defense mechanisms that they strategically avoid during their own replication. She found that phages also deploy these defense mechanisms to compete with other phages that inhabit the same host. Strikingly, these phage-phage interactions occur while *Salmonella* infects mammalian immune cells called macrophages and thereby can influence the outcome of *Salmonella* infections. Thus, Sargen showed how host-virus interactions have implications beyond one host cell and one virus.

Now as a Jane Coffin Childs Fellow in Bonnie Bassler's lab at Princeton University, Sargen will investigate how phages eavesdrop on bacterial communication called quorum sensing to inform their behavior: namely, whether they stably replicate with the host or undergo lytic replication that kills the host. In particular, she is interested in uncovering the mechanisms by which different cues influence these divergent virus lifestyles. Sargen notes that beyond advancing our basic understanding of viral behavior, her discoveries have the potential to inform the development of biomedical therapies that use or control viruses.

FRANCISCO TENJO CASTAÑO, Ph.D.

MERCK – JANE COFFIN CHILDS FELLOW

LABORATORY OF AKANKSHA THAWANI, Ph.D.
DEPARTMENT OF BIOCHEMISTRY AND MOLECULAR BIOPHYSICS
COLUMBIA UNIVERSITY

FELLOW

Francisco Tenjo Castaño, Ph.D. studies how DNA changes over time. In graduate school, he solved new protein structures showing how CRISPR-associated transposons (CAST) insert new DNA into the genomes of their bacterial hosts. As a Merck-Jane Coffin Childs Fellow, he will now study LINE-1, a major human DNA "jumping gene" that can reshape our genome and contribute to disease.

In Guillermo Montoya, Ph.D.'s lab at the University of Copenhagen, Tenjo Castaño used biochemistry and structural biology to reveal the molecular mechanism of CAST DNA insertions. First, he solved the structure of the CAST catalytic protein TnsB bound to the transposon ends and the target DNA, and found that the enzyme only becomes conformationally active when it is properly attached to the target DNA. Tenjo Castaño proposed that this coupling serves as a safety feature to ensure that CAST only starts integrating new DNA into the genome once the complex is in the proper location. Then, he reconstituted the complete ~1 MDa CAST system with target DNA and solved several structures of the entire complex and assembly intermediates at different stages. These results explained the fine details of DNA target detection and insertion site regulation. This work could help advance future gene-editing technologies.

During his thesis research, Tenjo Castaño increasingly appreciated the potential of transposons in gene therapy but also as drug targets. In Akanksha Thawani's lab at Columbia University, he will focus on LINE-1, the only active autonomous human retrotransposon, which has made up almost one-third of the human genome over evolutionary time. He will identify human proteins that help LINE-1 function, study how LINE-1 works using structural and biochemical methods and look for small-molecule drugs that inhibit it. Because LINE-1 activity is linked to cancer, neurodegeneration, and inflammation during aging, this research could point toward new treatments.

JAMES WHITLEY, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF IAN OLDENBURG, Ph.D.
DEPARTMENT OF NEUROSCIENCE AND CELL BIOLOGY
RUTGERS UNIVERSITY

FELLOW

James Whitley, Ph.D. has always been a fan of the underdog, and he notes that in neuroscience this tag applies broadly to any structure outside of the cortex. In his Ph.D., he showed that these regions do more than pass information along. As a Jane Coffin Childs Fellow, he will now study the brainstem, asking whether it plays a sophisticated role in filtering motor commands.

Historically the thalamus has been seen as a passive relay conveying sensory information to the cortex. Whitley's graduate research in Martha Bickford's lab at the University of Louisville challenged this passive view and established a more active, regulatory role for two visual thalamic nuclei. First, he demonstrated that the dorsal lateral geniculate nucleus enhances the flow of visual information following gaze shifts. Then, Whitley revealed an integrative role for the pulvinar nucleus whereby top-down and bottom-up signals are processed in the same neuron.

As Whitley has learned and discovered more about how information is transferred between different regions of the brain, he's come to the realization that traditional models fail to account for the diversity of behaviors and limit functional flexibility. In Ian Oldenburg's lab at Rutgers University, Dr. Whitley will examine information transfer between the cortex and the brain stem. He thinks motor commands may be represented as patterns of activity across groups of neurons, and he will test this idea using multiple methods. His goal is to have a better overall understanding of motor control and movement disorders.

AIRI YOSHIMOTO, Ph.D.

JANE COFFIN CHILDS FELLOW

LABORATORY OF LIQUN LUO, Ph.D.
DEPARTMENT OF BIOLOGY
STANFORD UNIVERSITY

FELLOW

Airi Yoshimoto, Ph.D. was influenced by her pharmacy training and a patient experience that showed her how early-life stress can affect brain development. Inspired by this experience, Yoshimoto focused her graduate work on how the brain controls body functions like heart rate. As a Jane Coffin Childs Fellow, she will study how hypothalamic circuits form during development and how they control basic needs such as thirst and temperature regulation.

During Yoshimoto's thesis research in Yuji Ikegaya's lab at The University of Tokyo she discovered the neural relay that allows voluntary control of one's heart rate. Heart rate and other physiological parameters are usually controlled by the autonomic nervous system. However, specialized training such as for free diving or meditation can teach individuals to voluntarily regulate these parameters. Yoshimoto developed a rat model of heart rate biofeedback, and used her model to uncover how the signal traveled through anterior cingulate cortical neurons through several relay stations all the way to parasympathetic neurons in the heart.

Now in Liqun Luo's lab at Stanford University, Yoshimoto will examine neurons in the hypothalamus that regulate homeostatic functions. Neurons that are thirst- and warmth-activated are anatomically intermingled in the hypothalamus, but are, by definition, triggered by different stimuli. Yoshimoto predicts that the presence of distinct surface adhesion proteins distinguishes these neural populations, and will build genetic mouse models to rewire hypothalamic circuits such that thirst will activate warmth-sensitive neurons. Yoshimoto's research promises to provide novel insight into the molecular mechanisms that dictate hypothalamic circuit specificity.