SELF-ASSESSMENT

Cole J. Ferguson, MD, PhD

Assistant Professor in Residence, Step V (50%)

University of California at San Diego School of Medicine

Director of Ophthalmic Pathology, Neuropathology Division, Department of Pathology

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At my fourth-year appraisal in 2024, I received a *Favorable with Recommendations* rating from the Department of Pathology and the campus-wide Committee on Academic Personnel (CAP). Of note, Senior Assistant Vice Chancellor of Academic Affairs Robert S. Ross put forward a *Favorable* fourth year appraisal "based on the level of engagement and the caliber of activities in all areas." CAP brought up research independence and productivity as the areas to strengthen, with reviewers asking for two things in particular: additional senior-authored publications without my former mentor as co-author, and major extramural funding as principal investigator. The record I present here addresses both issues.

One year ago I deferred my 6th year appraisal to bring several maturing studies to completion. My laboratory has since published four studies on which I am senior or co-senior author, none with a former mentor. Another senior-author study was recently submitted. I now hold R01 and R21 awards from the NINDS as principal investigator, the R21 having scored in the 8th percentile, with another R01 in preparation.

Since establishing my laboratory at the UC San Diego School of Medicine in October 2020, my major research focus has been epigenetic control of neurodevelopment. My lab's work has opened a new field of study into the unexpected role of ubiquitin signaling in shaping chromatin. UC San Diego has provided an especially conducive home for this work, as our Neuroscience, Computational Biology, and Genetics each rank among the strongest in the country, and my lab relies heavily on the tools of each field as well as the gifted undergraduates from the adjacent main campus who develop their bioinformatic theses based on our projects.

In addition to conducting extramurally funded research, I co-direct UCSD's only graduate survey course in Genetics; direct the Ophthalmic Pathology service within the Neuropathology Division under a 50% clinical appointment; mentor a steady succession of undergraduate and graduate students as well as medical students, residents, and fellows in Pathology; and contribute to the University through the Academic Senate, multiple admissions committees, and service to the Department of Pathology. I have also delivered three invited lectures at outside institutions.

In short, I have met all criteria for achieving rank of Associate Professor with tenure: i) minimum time in rank, ii) independent, peer-reviewed publications, iii) consistent grant funding, iv) national recognition, v) service to the department, university, and field, vi) demonstration of effective teaching and mentorship, vii) strong Chair support, and viii) ongoing upward career trajectory.

In what follows below, I describe my contributions to the research, clinical, teaching, and service missions of the University.

RESEARCH AND SCHOLARLY ACTIVITY

Ubiquitin operates in two fundamentally different modes: it can act as a degradative signal, in which polyubiquitin chains target proteins for destruction by the proteasome organelle, or as a regulatory modification,

in which a single ubiquitin alters a substrate's activity or interactions without destroying it. Over the past several years my lab has shown that both modes shape the chromatin landscape during neuronal maturation, placing our work at the nexus of ubiquitin biology and neuronal epigenetics. Below, I first describe our work on ubiquitin in its degradative capacity, before turning to our discovery that ubiquitin reshapes chromatin identity as the brain matures and how this led to an interesting side project on interorgan communication. I close with a project on neuroinflammation and neurodegeneration that has its roots in my doctoral thesis work, to which we have now been able to return with far more sophisticated technology, and how this relates to a clinical collaboration on the pathogenesis of an eye disease I see in my pathology practice.

I. Ubiquitin as a degradative signal: clearing chromatin regulators to allow neuronal maturation

I came to this problem from the clinical side of human genetics. As a postdoctoral fellow with Azad Bonni at Washington University, I set out to understand a new recessive intellectual-disability syndrome we found to be caused by loss of *ANAPC7* (Ferguson–Bonni neurodevelopmental syndrome; OMIM 619699). *ANAPC7* encodes APC7, a core subunit of the Anaphase-Promoting Complex (APC)—a ubiquitin E3 ligase long studied for clearing mitotic proteins so that dividing cells can exit mitosis. None of the other 15 core APC subunits had ever been associated with an inherited human disease. Indeed, why should non-dividing neurons be susceptible to a defect in the clearance of mitotic proteins? In Ferguson et al. (*Molecular Cell*, 2022), we used state-of-the-art structural biochemistry, human genetics, and functional analyses in several mouse models to show that APC7 has a crucial, previously unsuspected role in neuronal differentiation. Specifically, it is required for efficient ubiquitination by the APC and, through proteomics analysis of the Apc7-deficient mouse brain, we identified Ki-67 as a key neuronal substrate. When insufficiently cleared from post-mitotic neurons, Ki-67 shifts from the chromosome periphery to concentrate in constitutive heterochromatin (densely compacted genomic regions that that must be silenced for neurons to function). We speculated that, because Ki-67 is rich in intrinsically disordered repeat domains, its accumulation could alter the condensation of constitutive chromatin that occurs through phase separation.

After starting my independent lab at UCSD, we soon realized that we had found not a single, especially important substrate but a general principle: the canonical mitotic regulators—Ki-67, the chromosome-passenger complex (CPC) and its kinase Aurora B, and topoisomerase 2α—are all normally ubiquitinated to be cleared from neurons as they exit mitosis and differentiate (Ledvin et al., *Developmental Cell*, 2023; senior author). When the APC loses activity, these proteins persist in phosphorylated, mitotic-like forms, with Aurora B accumulating in heterochromatin and driving aberrant phosphorylation of its substrate histone H3. The CPC had been shown to affect chromatin state if present during interphase, but its roles outside mitosis were still unknown. Proteins long thought to be purely mitotic are thus repurposed to regulate heterochromatin in post-mitotic neurons. This study, my first as an independent senior author (though Azad Bonni was listed as a middle author based upon his support during the early stages of the project), established that ubiquitination in the context of neurodevelopmental disorders merited scientific interest.

Ledvin et al. also produced an unexpectedly promising translational result: we administered two brain-penetrant Aurora B inhibitors subcutaneously and found that they fully corrected abnormal histone H3 phosphorylation in the mutant brain. To my knowledge, this is the first time a neuronal chromatin phenotype has been rescued by a peripherally delivered small molecule. Lowering Ki-67 genetically likewise restored heterochromatin function. With Nick Brown (UNC–Chapel Hill), a biochemist and co-author on three of our papers, we are now defining how this machinery recognizes its neuronal substrates—work supported by an R21 from the NINDS that scored in the 8th percentile.

Although this is a nascent field, the *Molecular Cell* paper (2022) has been cited 17 times, and the *Developmental Cell* (2023) paper has already been cited 14 times by other groups working on APC or its substrates in various contexts, including cancer. A parallel, or rather anti-parallel, project that I began in the Bonni lab on the function of the Hao-Fountain neurodevelopmental syndrome protein USP7 (a *de*ubiquitinating enzyme) in the brain was published in *Cell Reports* in 2025, with me second author after the PhD student Hao Chen. This paper has been cited 13 times already, which signals the growth of the field.

Ki-67 in phase separation of neuronal heterochromatin

The APC work drew our attention to Ki-67 itself—a protein universally used as a marker of cell proliferation, but whose function in post-mitotic neurons was utterly unknown. We generated the first brain-specific Ki-67 knockout mouse and, comparing these with *Anapc7* mutant mice that have too much Ki-67, discovered that Ki-67 determines the size and number of the silenced heterochromatic particles within the nucleus. It does so without changing chromatin marks or which genes neurons express, suggesting Ki-67 operates through a physical rather than a chemical mechanism. Live-cell imaging revealed that Ki-67-rich structures behave like liquid droplets that merge and split, one hallmark of phase separation. Collaborating with our colleague Galia Debelouchina (UCSD Dept. of Chemistry and Biochemistry), we used purified proteins and biophysical methods, as well as molecular dynamics simulations, to show how Ki-67 drives this behavior. These findings, just accepted in *Nature Communications*, recast a familiar proliferation marker as a physical organizer of the neuronal nucleus, and they provide one of the clearest examples of phase separation setting heterochromatin architecture in neurons, with direct consequences for genome integrity in the developing brain. The work is supported by my NINDS R01 ("Regulation of phase separation in neuronal heterochromatin," now beginning year 3 of 5). Moving forward, we will focus on the mechanism of Ki-67-driven heterochromatin phase separation via mutagenesis experiments, molecular dynamics simulations, and nuclear magnetic resonance (NMR) and hydrogen-deuterium exchange studies of phosphorylation—while performing the Hi-C analysis of Ki-67 mutant neurons and mapping the neuronal Ki-67 interactome using APEX2 proximity labeling of endogenous Ki-67 locus.

II. Ubiquitin as a regulatory mark: reshaping chromatin identity as the brain matures

The second major focus of my laboratory concerns ubiquitin's other modus operandi. Rather than marking a protein for destruction, a single ubiquitin attached to a protein can regulate its activity, for instance by serving as a bunding platform for recruiting interactors. As the brain develops, each neuron must switch precise subsets of genes on and off, and we have found that ubiquitination of histone proteins is a master regulator of that process. I will describe major discoveries from my laboratory relevant to this area of investigation.

Polycomb complexes at active loci in the developing brain

The specific modification we study, H2AK119ub (hereafter H2Aub), is placed onto histone H2A by Polycomb Repressive Complex 1 (PRC1) and removed by the deubiquitinase BAP1. Loss of PRC1 function de-represses genes and has been studied primarily in the context of various cancers; little was known about what it might do in neurons, except that mutations in PRC1 components cause inherited neurodevelopmental disease. We therefore mapped H2Aub in the mouse brain at an early postnatal and adult timepoints, while developing new bioinformatic methods to deepen our analyses of our CUT&RUN data. To our surprise, we found that as neurons mature, this "silencing" mark is depleted from genes that are silenced and redirected toward *active* loci (specifically, active enhancers and promoters) (Parmar et al., *PLoS Genetics*, 2025). Mapping other PRC-dependent histone modifications showed that Polycomb Repressive Complex 2 likewise controls actively expressed genes in maturing neurons through the modification H3K27me1, which we found to be much more dynamic than the widely known PRC2-dependent, heterochromatin-associated modification H3K27me3.

These findings overturn the textbook view that Polycomb complexes only act at repressive loci. Reviewers recognized the significance of the work:

> *"... compelling chromatin data ... reveal new ways of thinking about polycomb complex functions in neuronal differentiation… The data are presented and evaluated in rigorous fashion – this was one of the best descriptions of bioinformatics choices for pipelines and normalization strategies that I have seen."*
>
> *"The findings are significant, not only advancing our understanding of chromatin regulation in neural development but also introducing computational and statistical methodological innovations that will likely be valuable for future studies—especially those involving dynamic, multifactorial chromatin landscapes."*
>
> *"The authors' findings are highly novel and interesting for the field of Polycomb and chromatin research...."*

Chromatin control of inter-organ small-molecule handling

One offshoot of the PRC chromatin mapping project led my lab in a new direction with our colleague Sanjay Nigam (UCSD Dept. of Pediatrics), who studies small molecule transporters. We decided together to explore how the roughly 1,000 genes that absorb, distribute, metabolize, and clear small molecules—and so mediate communication between organs—are regulated as the body matures. This question had never been examined at the chromatin level, but the approaches we had developed for the *PLoS Genetics* paper made this feasible. Mapping several chromatin marks in mouse kidney, liver, and brain at embryonic and adult stages, we found that the liver remodels chromatin state at these loci to the greatest degree during development, with coordinated activation in kidney and liver that tracked with changes in gene expression (Parmar et al., *Epigenetics & Chromatin*, 2025). The findings bear on how the body achieves metabolic autonomy after birth, on handling of exogenous drugs, and on diseases such as chronic kidney disease and liver failure. We are now focusing on transporters for specific disease-relevant molecules, including bile acids in the liver and urate, uremic toxins, and creatinine in the kidney.

BAP1 and the formation of chromatin identity in neurons

If H2Aub is redistributed during neurodevelopment, what happens when its removal goes wrong? Children born with mutations in *BAP1*, the deubiquitinase that erases H2Aub, develop Küry–Isidor syndrome, which causes severe global delay, behavioral problems, and seizures. Like PRC1/2, BAP1 had been studied almost exclusively in the context of cancer; its role in the brain had never been examined. Because complete loss of BAP1 is embryonic-lethal, we generated conditional mouse models removing BAP1 from excitatory neurons of the forebrain or cerebellum, and paired behavioral analyses with CUT&RUN, ATAC-seq, and transcriptomics. We found that BAP1 orchestrates a multifaceted genetic program essential for neuronal gene expression—a program distinct from its roles in other cell types. Remarkably, when Bap1 is lost, the distinction between active (euchromatin) and silenced (heterochromatin) chromatin breaks down and these two functional compartments begin to resemble one another. The genes most affected by Bap1 loss are involved in the formation and function of synapses; in collaboration with Kim Dore's lab (UCSD Dept. of Neurosciences), we performed brain-wave recordings and single-cell electrophysiology to show that the mutant neurons are abnormally hyperexcitable. This work, which we recently submitted with me a senior author, recasts BAP1 as a central regulator of the neuronal epigenome and gene regulation.

Our map of the gene pathways disrupted by BAP1 loss also relates to its other life as a tumor suppressor gene in cancer. *BAP1* mutations occur in many malignancies but are most famously associated with uveal melanoma, a diagnosis I encounter frequently as an ophthalmic pathologist. Loss of BAP1 in uveal melanoma is strongly associated with metastasis and poor prognosis. We found that the gene expression changes that occur in Bap1

deficient neurons resemble those that occur in tumor cells that have lost Bap1, for instance, dysregulated expression of cell adhesion pathways and lineage-defining transcription factors. These shared signatures hint at a single unifying function for BAP1 that underlies both cancer's invasive properties and the neurite outgrowth required for neuron-to-neuron connectivity in the brain.

This foundational study has already yielded three additional projects that we are working on and believe will be competitive to receive additional extramural grant support in the next year or so:

1. Using Hi-C to study how DNA is folded in three dimensions, we found that BAP1 loss disrupts genome organization at every structural scale (topologically-associated domains, loops and stripes), with different effects in euchromatin versus heterochromatin.

2. We have found that BAP1 loss profoundly disturbs DNA methylation and hydroxymethylation, in a pattern that reinforces inappropriate gene silencing or activation.

3. We have mapped the specialized histone variant, H2A.z, during normal brain development and have found that BAP1 also controls its ubiquitin tagging—a role not previously suspected.

We now have established embryonic stem cells in the lab and are using gene editing to introduce patient *BAP1* mutations into them, so that we can grow human neurons carrying these mutations and study them directly. A complementary new direction examines mice lacking the PRC1 ubiquitin-ligase subunits Ring1A and Ring1B: where BAP1 erases H2Aub, these enzymes deposit it, and comparing the two will let us test how writing versus erasing the mark shapes gene expression, DNA methylation, chromatin folding, behavior, and neurophysiology.

Together, this body of work makes progress on one of the central questions in biology—how a single genome gives rise to cell types as different as a neuron and a hepatocyte—and I expect these projects to be of broad interest across the fields of epigenetics and neuroscience.

III. Neuroinflammation and neurodegeneration

A third area of interest in my lab is bringing unbiased and targeted methods to bear on disease pathogenesis.

Understanding the basis of Fig4-related neurodegeneration. My interest in inherited neurological disease began in graduate school with Miriam Meisler at the University of Michigan, where I studied FIG4, a phosphatase that, with PIKfyve and VAC14, controls the endolysosomal signaling lipid $PI(3,5)P_2$. *FIG4* mutations cause a fatal childhood neurodegeneration and the fatal form of Charcot–Marie–Tooth disease CMT4J and contribute to several forms of ALS. We had shown that insufficient $PI(3,5)P_2$ impairs autophagy in neurons and astrocytes and produces severe neurodegeneration (Ferguson et al., *Human Molecular Genetics*, 2009), and that neuronal Fig4 expression is both necessary and sufficient to prevent it (Ferguson et al., *Human Molecular Genetics*, 2012). We also found that Fig4 loss causes congenital CNS hypomyelination through a non-cell-autonomous effect on oligodendrocytes (Winters & Ferguson et al., *Journal of Neuroscience*, 2011; co-first author), defined the destabilizing mechanism of the common CMT4J allele (Lenk et al., *PLoS Genetics*, 2011), and helped delineate the defining features of the human disease (Nicholson et al., *Brain*, 2011).

Yet despite decades of study, it has remained unclear how $PI(3,5)P_2$ deficiency actually harms the brain, because the lysosome is involved in so many cellular processes. Borrowing the proteomic strategy that had worked for the APC, I applied unbiased proteomics to the lysosome with Steve Gygi (Harvard) and Brandon Gassaway (BYU), quantifying more than 10,000 brain proteins to define the full set of $PI(3,5)P_2$-dependent lysosomal substrates—and found that lysosomal failure drives hyperactivation of innate immunity. With Andrew

Mendiola (UCSD, Department of Pharmacology) we showed that microglia contribute to oxidative damage, and that these abnormalities begin in the first days of life, supporting the idea that disturbances during development can set the stage for later neurodegeneration. This first comprehensive account of how disrupted lysosomal lipids injure the brain appeared, with only minor revision, as Wong et al. (*Neurobiology of Disease*, 2026; senior author). Reviewers wrote:

"The amount of data generated using complementary approaches is substantial, and the analyses are carefully performed."

"It provides a detailed and valuable resource that will likely serve as an important reference for future studies on the roles of lysosomal phosphoinositides in neurodegeneration and lysosome-associated neurological disease."

We are now deleting Fig4 one cell type at a time, with a surprising result: loss from astrocytes produces a more severe phenotype than loss from neurons, indicating that astrocytes drive most of the damage (likely by fueling neuroinflammation). This scenario is almost unheard of for a neurodegenerative phenotype. Our data implicate hyperactivation of the cGAS–STING pathway as the central hub in astrocytes that drives neuroinflammation. Because cGAS–STING inhibitors are already in trials for common neurodegenerative diseases, they may warrant evaluation for FIG4-related disease—a possibility I will explore in an upcoming R01 application.

Orbital Inflammatory Disease. As a small side-project, I am part of a multi-institutional study with our oculoplastics and ophthalmology colleagues on the pathology of orbital inflammatory disease. This study draws on my diagnostic work as a pathologist. We recently used periostin immunohistochemistry to show that this matrix protein is strongly and specifically expressed in zones of pathologic fibrosis—but not in the inflammatory infiltrate or epithelium—in both nonspecific orbital inflammation and IgG4-related orbital disease (Zuraski et al., *Orbit*, 2025), suggesting that periostin is a tissue biomarker and a possible contributor to fibrosis in these conditions.

In conclusion, I fully believe that in the past few years, my research laboratory has established an outstanding portfolio of independent and collaborative research projects, largely distinct from those I worked on as a trainee. We have demonstrated the ability to publish in extremely high impact journals and secure extramural grants at a time when all scientists find this a challenging goal.

TEACHING ACTIVITIES

Philosophy

My teaching is guided by the conviction that durable understanding comes from grasping the principles that govern how biological systems behave. Whether I am at the microscope with a clinical trainee, in the classroom with graduate students, or at the bench with a member of my own lab, my aim is the same: to help learners reason from first principles, so that they can approach problems they have never seen before. To truly understand the current state-of-the-art, we must understand how we came to know it—and whether, indeed, we are justified in claiming to know it—in order to build upon this knowledge and improve people's lives.

Classroom teaching

I have just completed my fourth year as co-director of the Genetics and Genomics course (BIOM252). It is the only survey genetics course for graduate students at UC San Diego, drawing 20–25 students each year from BMS, the Neurosciences Graduate Program, and Biological Sciences, among others; students in the Genetics Training Program (T32) take two years of it. My co-director Radha Ayyagari and I assemble ten weeks of lectures from across the San Diego genetics community, each pairing a leading scientist's lecture with student-

led discussion of related papers. The course alternates between two annual cycles—one on principles of inheritance and disease, the other on genetic technology. I attend every session to facilitate discussion, organize the Canvas materials, administer the exam, and assign grades.

Some of my contribution is behind the scenes. Because the course covers a different subject each week and students arrive with very different levels of preparation, I sequence the lectures so concepts build logically, acquaint each speaker with what came before to avoid overlap, and ask presenters to open broadly before taking a few well-chosen deep dives into key technical details or biology.

Students consistently rate the course's lectures, intellectual value, and overall effectiveness from "Very Good" to "Excellent," and they particularly value its breadth and its exposure to active research areas. My own lectures—on chromatin structure, histone modifications, and the methods used to study them—fall in alternating years; those in 2024 and 2026 were rated from "Very Good" to "Outstanding" across presentation, organization, and intellectual value. Students noted that the material was new to them yet came through clearly: one praised an "*in-depth explanation of very complicated concepts surrounding histone modification and chromatin structure*," another commented, *"I truly enjoyed the course… even as someone who only had basic knowledge in genetics, I was able to learn a lot and to appreciate the complexity and broadness of the field."* A student from outside the field praised the "*science storytelling*," noting that I "*care about students' general understanding, not just about delivering a talk*." A faculty evaluation described me as "*an incredible speaker who knows how to engage students and make them as excited about chromatin biology as he is*," crafting "*lectures like a story*" with a clear overall message. Feedback like this is very gratifying, as it reinforces how much students need and appreciate good teaching.

For three years I have lectured in the core cellular and molecular neuroscience course (NEUG200A, ~30 students) and, this past year, in NEUG221 (Epigenetics and Gene Regulation in the Brain, ~12 students). Each year I take part in the Mentoring Up sessions for incoming BMS students (BIOM203A; ~15 students); this year I was the only BMS faculty member to join all three. I am an active member of the Genetics Training Program (T32), whose journal clubs and annual retreat I attend with my students.

Mentoring

My lab benefits enormously from UC San Diego's strong culture of undergraduate research and from a feature that is easy to miss from outside: the immediate adjacency of the School of Medicine to a top-tier undergraduate campus gives a small lab like mine access to exceptional student volunteers. My lab is composed entirely of seven undergraduate students (who work part-time) and two graduate students, and much of our work depends on the computational analysis of large genomic datasets—a domain that rewards tenacity and fearlessness but requires a lot of instruction along the way.

Because the lab is small, the undergraduates take on their own projects and I mentor them directly in weekly one-on-one and lab meetings and frequent informal conversation. I emphasize rigorous, reproducible experimental design (in quantitative genomics and proteomics, the choice of controls and analytic parameters demands care from the outset). I also focus on written communication, asking students to draft their own figures and manuscripts, which we then revise together. It is worth noting how much this small team has accomplished, especially as competition for PhD students has stiffened while programs at UCSD and many other universities have contracted. My students routinely approach a problem in a way I would not have, write a more elegant pipeline, or identify a pattern in the data that reframes a project; I try to create the conditions for turning those observations into genuine insight by posing hard, open-ended questions and working beside them on the execution and analysis. I want them to leave not only as capable scientists but as people who have felt the exhilaration of discovery.

My trainees' outcomes are listed in my CV. What that list does not convey is the context: I founded the lab in an early wave of the pandemic (October 2020) and recruited and trained students through a period of remote work and limited in-person contact that did not ease until roughly 2023. Even so, several have gone on to competitive doctoral programs. Leya Ledvin, first author of our *Developmental Cell* paper, entered the Neurosciences Graduate Program here on a successfully funded NSF fellowship written from her work in my lab, and Aditya Parmar was sole first author on two papers as an undergraduate before entering a bioinformatics master's program. Others are enrolled in informatics PhD programs at UCSD and UCLA. I am proud that, given the circumstances, so many have continued in science.

I also serve on several PhD thesis committees across the Neurosciences, Biomedical Sciences, and Chemistry-Biochemistry programs (listed in my CV).

Current trainees

My current PhD students are Challana Tea (Chemistry and Biochemistry; 2023–present), who studies how BAP1 regulates chromatin and who built out our lab's embryonic stem cell facility and optimized our Hi-C experiments before advancing to candidacy with his committee's unanimous support, and Jai Ramchandra (BMS; 2025–present), who is beginning a project on how histone ubiquitination controls DNA methylation in the brain. They help me teach the undergraduates—including Zakir Alibhai, who has built several analysis pipelines and a web application for processing chromatin-mapping data—working across our genome-folding, DNA-tagging, H2A.z, Polycomb, and BAP1 mutant stem-cell projects.

CLINICAL SERVICE

I direct Ophthalmic Pathology within the Neuropathology Division. Eye pathology is a busy service—20–30 cases a week, which I have covered 40 weeks per year—and an unusually diverse one, demanding knowledge not only of the eye but of skin, head and neck, cytology, soft tissue, and retina/CNS, requiring frequent collaboration with ophthalmology, dermatology, and oncology. I take part in many multidisciplinary ophthalmology conferences, and I also provide the diagnostic pathology that has underpinned collaborative clinical-translational work in orbital disease (see Research section above).

In 2025 I became the neuropathologist for the Target ALS Foundation's multicenter postmortem tissue cores. Led at UC San Diego by John Ravits, ours is one of just six sites worldwide. I perform the microscopic evaluation and scoring of donated central nervous system tissue, rendering the neuropathological diagnosis and documenting the distribution and severity of pathology so that each case can be linked to its de-identified clinical and genetic data. This supports the foundation's mission of supplying researchers worldwide with high-quality postmortem tissue at minimal cost and without intellectual-property restrictions.

Clinical teaching

Neuropathology is a two-year subspecialty fellowship covering surgical neuropathology (e.g., brain tumors), autopsy neuropathology, muscle disease, eye pathology. Much of the teaching happens at daily microscopy sessions where we work through active cases together. My fellow from 2023 to 2025, Kari Hird, scored above the 90th percentile on the in-service examination and, after a surgical pathology fellowship at Utah, will return as faculty at UCSD. A former fellow, Connor Zuraski, completed an ophthalmic pathology fellowship at Stanford and has joined our division as faculty, as has another former fellow, Vanessa Goodwill. I also teach anatomic pathology residents on their neuropathology rotations, which I especially enjoy because the breadth of eye pathology means each resident takes away something useful whatever specialty they choose.

I oversee two medical-student experiences in ophthalmic pathology. Typically enrollees are interested in careers in Ophthalmology, but some are also pursuing Pathology or other medical specialties. I am preceptor for the third-year selective PATH412, a three-week rotation; last year all three enrolled students praised it warmly, noting that it gave them lasting insight into microscopic diagnosis. I also direct the four-week fourth-year elective PATH427; last year's three students were all headed for Ophthalmology and made remarkable progress in understanding the diseases they will encounter. I devote substantial time to these curricula, including curating collections of hundreds of teaching slides, developing lectures, and assembling self-study materials. Examples of student feedback are included below.

PATH 412: *"Incredibly kind and patient teacher. Always willing to teach even if busy. Down to earth and humble despite having a vast knowledge of pathology."*

PATH 427: *"Dr. Ferguson went above and beyond in ensuring a comprehensive and engaging learning experience. He took the time to review all incoming cases as well as a collection of both common and rare cases he had gathered over the years. His dedication to teaching was evident in the way he patiently explained fundamental pathology concepts and actively included me in case discussions. By the end of the course, I felt much more confident in identifying common ophthalmic pathology cases."*

I also give lectures and microscopy sessions for Ophthalmology residents on board-relevant topics, which they have consistently valued, and in 2022 I received a teaching award from the Department of Ophthalmology for excellence in resident education.

UNIVERSITY AND PUBLIC SERVICE

From 2023 to 2025 I served as a primary representative to the Academic Senate, a period that included several consequential votes—on the Chancellor's response to the student protest encampment near Library Walk, on the admission of clinical and adjunct faculty into the Senate, on student privacy, and on the University's response to visa revocations, free speech, and academic freedom.

I have served on the admissions committees for the Neurosciences Graduate Program (2023, 2024), Biomedical Sciences (2023), and the Medical Scientist Training Program (2025), each year reading and scoring roughly 75 applications, interviewing candidates, and helping decide admissions. For the past four years I have served on the planning committee for the Department of Pathology research retreat, including selecting keynote speakers.

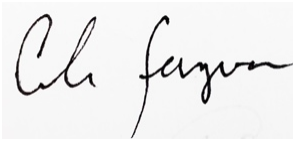
I am also a founding member of California Ophthalmic Pathologists, a monthly virtual gathering of 20 ophthalmologists and ophthalmic pathologists from UCSD, UC Irvine, UCLA, and USC that reviews challenging cases for second opinions and offers AMA Category 1 CME credit, and at which the neuropathology fellows and I frequently present.

Family challenges

I would like to also add that my family and I have faced many difficulties since relocating to San Diego in 2020. In 2023, a man drove his car into our home in Mission Hills, destroying the exterior wall and rendering our kitchen unusable for months. My wife underwent bilateral mastectomy surgery for high-grade ductal carcinoma in situ. A year prior she had been bedbound for nearly 6 months with a chronic illness from which she continues to suffer, though she has regained most of her previous level of function. These experiences have impacted my 13 and 11 year-old daughters, both of whom suffer from occasional bouts of severe anxiety. Finally, I underwent two orthopedic surgeries to my right and left ankles that required no weight bearing for a total of 3 months.

CONCLUSIONS

Across research, teaching, clinical service, and university citizenship, my aim has been to build something coherent, durable, and outstanding: an independent research program at the interface of ubiquitin biology and neuronal epigenetics, a clinical service and a group of trainees I am proud of, and a record of service to the institution. The questions that drive my science are ones that are well-supported by the stimulating intellectual environment at UCSD, as evidenced by my intra-institutional collaborations, and ones I expect to keep my laboratory productive for many years. I look forward to deepening these contributions and to taking on greater responsibility within the Department of Pathology and the broader campus community.



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