

# Rosenfeld Science Scholars Symposium

Yale

Becton Engineering and Applied Science Center  
Davies Auditorium  
15 Prospect Street

September 7, 2018  
4:30-7:00 PM

## Schedule of Presentations

5:30	Dr. Sandy Chang <i>Associate Dean of Science and QR Education</i>	Welcoming remarks
5:30-5:45	Emma Garcia <i>Dept. of Chemistry</i>	Characterization of para-cyano-phenylalanine tRNA synthetase and variants as a tool for incorporating $\beta$ -amino acids <i>in vivo</i>
5:45-6:00	Nicole Eskow <i>Dept. Molecular, Cellular and Developmental Biology</i>	Role of MKL1 in megakaryocyte ontogeny
6:00-6:15	Kazemi Adachi <i>Dept. of Applied Physics</i>	Refractive scattering without angular restriction for tandem photovoltaic design
6:15-6:30	Ellen Kan <i>Dept. of Biomedical Engineering</i>	Investigating the biological effects of oxygen tension on lung epithelial cells
6:30-6:45	Kevin Biju <i>Dept. of Molecular Biophysics and Biochemistry</i>	Characterization of stalled replication fork-induced telomeric recombination
6:45-7:00	Stephanie Smelyansky <i>Dept. of Chemistry</i>	Regulation of mRNA decay by phosphorylation of NoBody, a human microprotein

## Poster Presentations (4:30-5:30)

Razvan Azamfirei <i>Dept. of Molecular, Cellular and Developmental Biology</i>	Examining nuclear envelope repair in the <i>C. elegans</i> early embryo
Kevin Chang <i>Dept. of Molecular Biophysics and Biochemistry</i>	Engineering a synthetic bacterial co-culture to degrade and metabolize PET plastics
Meibin Chen <i>Dept. of Biomedical Engineering</i>	Exploring heterogeneity of IL-12 secretion in murine bone marrow-derived macrophages
Chunyang Ding <i>Dept. of Applied Physics</i>	Design of broadband multipolar microwave waveguide attenuators for superconducting Qubits
Adam Fine <i>Dept. of Physics</i>	Unsupervised behavioral classification of <i>Drosophila melanogaster</i> from positional data in olfactory assay

Benjamin Koleske  
*Dept. of Molecular Biophysics and Biochemistry*

Characterization of an artificial signal peptide with  
secondary activity

Hudson Lee  
*Dept. of Biomedical Engineering*

Genetic modification of diabetic fibroblasts and  
extracellular matrix for improved wound healing

Jacob Zavatone-Veth  
*Dept. of Physics*

Dissecting asymmetric locomotor coordination  
during turning in *Drosophila*

## Characterization of para-cyano-phenylalanine tRNA Synthetase and Variants as a Tool for Incorporating $\beta$ -amino acids *in vivo*

Emma Garcia<sup>1,2</sup>, Omer Ad<sup>2</sup>, and Alanna Schepartz<sup>2,3</sup>

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It has been known for decades that protein synthesis involves the incorporation of the ~20 canonical  $\alpha$ -amino acids into polypeptides encoded by mRNA. However, if this system can be modified to incorporate monomers other than  $\alpha$ -amino acids into a polymer, then novel sequence-controlled macromolecules can be synthesized with extensive potential applications in materials science, industry, and medicine. Our lab's recent discovery of a mutant ribosome has shown promise as a tool for  $\beta$ -amino acid incorporation *in vivo*, but due to the lack of an orthogonal tRNA – synthetase pair, it is currently impossible to exploit the full potential of this ribosome to incorporate  $\beta$ -amino acids in a site-specific manner. Therefore, an enzyme known to be orthogonal towards natural amino acids in preference of  $\alpha$ -unnatural para substituted phenylalanine derivatives, *p*-CNF-RS, has been selected for evolution experiments. This enzyme has been purified for use *in vitro* and shown by mass spectrometry to be able to charge small amounts of  $\beta$ -amino acid to a cognate amber suppressor tRNA *in vitro*. Additionally, two small saturation mutagenesis libraries of residues L32 and W108 of *p*-CNF-RS were generated for analysis *in vivo* through a GFP screen and a flow cytometry and mass spectrometry based assay. Together, these experiments work toward the goal of developing an active tRNA synthetase that would allow for site specific incorporation of  $\beta$ -amino acids into full length proteins *in vivo*.

## Role of MKL1 in Megakaryocyte Ontogeny

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Hematopoiesis is the process by which hematopoietic stem cells develop into every blood cell type. In mature organisms, hematopoiesis is primarily carried out in the bone marrow, whereas in a developing fetus, hematopoiesis occurs in the liver. Many questions remain regarding the differences that underlie the development of megakaryocytes, the cells that produce platelets, in the fetal liver and adult bone marrow. As megakaryocytes mature, surface expression of CD41 is induced, and the cells undergo progressive rounds of endomitosis to become polyploid. By studying how this maturation differs between fetal liver and adult bone marrow, a better understanding will be gained regarding the possible *in utero* development of several megakaryocyte-associated pathologies, such as acute megakaryoblastic leukemia (AMKL) associated with the RBM15-MKL1 fusion oncogene. In adult bone marrow, MKL1 (megakaryoblastic leukemia 1) enhances megakaryocytic maturation via SRF (serum response factor) transcriptional coactivation. This study aims to identify the role that MKL1 plays in fetal megakaryopoiesis. Fetal liver-derived hematopoietic stem cells were harvested from E12.5-14.5 embryos and cultured for 96 h with thrombopoietin to promote megakaryopoiesis. Megakaryocyte maturation was assessed by flow cytometric analysis of polyploidization of CD41+ cells. Our results demonstrate that megakaryocyte maturation is enhanced in cells from MKL1 knockout fetal liver compared to cells from wild-type fetal liver. This enhanced maturation is inhibited when MKL1 knockout fetal liver cells are transduced to express MKL1. However, maturation is not inhibited when these cells are transduced to overexpress a variant of MKL1 in which the SRF-binding domain is removed. Therefore, MKL1 may inhibit the maturation of fetal liver-derived megakaryocytes in an SRF-dependent manner. Future studies will involve the characterization of megakaryocyte differentiation in fetal livers from SRF knockout mice and mice expressing the RBM15-MKL1 fusion protein to further define the role of MKL1 in megakaryopoiesis and potentially leukemogenesis *in utero*.

## Refractive Scattering Without Angular Restriction for Tandem Photovoltaic Design

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In a seminal 1961 paper, William Shockley and Hans-Joachim Queisser conducted a “detailed balance” calculation for the efficiency limit of p-n junction semiconductor solar cells. For a single junction photovoltaic (PV) cell, the SQ limit is ~33.2%. Tandem PVs can exceed this limit – a theoretical infinite junction system has an ideal efficiency of ~86.8%. These multi-junction PVs increase efficiency by allocating light of only certain wavelengths to carefully tuned solar cells.

Vertically stacked PV systems exploit the bandgaps of the semiconductors themselves to allocate light but encounter the difficulties of material-sensitive lattice- and current-matching. Laterally organized PVs have much simpler electronics yet require mechanical stabilizers and tracking to supplement optical splitters.

Using computer simulations, we model various optical devices for light splitting. Using MATLAB, we developed a regime using total internal reflection, thin film filter lenses, and rear mirrors to direct light. This design had an ideal efficiency of ~38% for multi-junction cells. Our second design simulated a spectral splitter using finite-difference time-domain solver *Meep*. This regime has a much more promising ideal efficiency of ~43% for two-junction PVs and ~48% for three-junction PVs, though our optimizations have not realized splitters functional across a broad spectrum of angles.

## Investigating the Biological Effects of Oxygen Tension on Lung Epithelial Cells

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For patients suffering from end-stage lung diseases, a promising alternative to lung transplantation involves the creation of patient-specific, tissue-engineered lungs for *in vivo* implantation. One of the biggest obstacles to this goal is the maintenance of the alveolar-capillary barrier needed to preserve separation between fluid and air. In particular, the level of dissolved oxygen ( $pO_2$ ) requires careful control, as hypoxic or hyperoxic environments can induce apoptosis or dedifferentiation in alveolar epithelial cells (AECs), respectively, leading to loss of overall tissue function. Thus, bioreactor culture conditions must be optimized to maximize AEC survival and differentiation. In this study, we designed a tunable bioreactor system that allows for predictive control of  $pO_2$  and provides a non-invasive method to transform dissolved oxygen measurements directly into real-time oxygen consumption rates, regardless of the bioreactor setup or sweep gas applied to the cultured lung. We then performed native rat lung cultures to experimentally validate our model, subjecting the lungs to sweep gas mixtures simulating hypoxic, physiologic, and hyperoxic conditions in order to identify  $pO_2$  environments that promote both survival and differentiation of AECs. Our data suggest that hypoxic conditions lead to upregulation of apoptotic markers, while hyperoxic conditions lead to upregulation of proliferation markers and downregulation of epithelial markers. Mesenchymal markers were found to be upregulated in hyperoxic environments as well, suggesting that epithelial-mesenchymal transition (EMT) may occur and contribute to loss of barrier function under these conditions. These native experiments lay the groundwork for future studies that will investigate the optimal culture conditions needed to build tissue-engineered lungs with full barrier function.

## **Characterization of Stalled Replication Fork-Induced Telomeric Recombination**

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The shelterin complex is a crucial protector of telomeres, preventing recognition of telomere ends as double strand breaks by the DNA damage response machinery. When components of this complex are disrupted, tumorigenic DNA damage response pathways, such as homologous recombination (HR), can occur. We have recently shown that, when certain shelterin components are disrupted, cells exhibit a high degree of HR and the formation of large telomeric foci. These foci are visualized as spots of high fluorescence intensity upon staining of telomeric G-rich sequence by PNA-FISH. However, the characterization of these foci has not yet been determined. In this study, we characterized the mechanisms required for foci formation and examined the role of HR in this process. Our results provide an enhanced understanding of the relationship between shelterin and HR, revealing new insights into telomeric repair and DNA damage response pathways.



## **Regulation of mRNA Decay by Phosphorylation of NoBody, a Human Microprotein**

Stephanie Smelyansky<sup>1,2</sup>, Zhenkun Na<sup>2</sup>, Sarah Slavoff<sup>2</sup>

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Small proteins and polypeptides, defined here as any polypeptide shorter than 100 amino acids long, have recently been discovered to comprise a larger part of the eukaryotic proteome than previously thought. NoBody, short for non-annotated P-body dissociating polypeptide, is one such small protein. The NoBody protein regulates 5'-to-3' mRNA decay via interaction with EDC4, a scaffolding protein that holds together the mRNA decapping complex, as well as DCP1a, an activator of the decapping complex. These two proteins are critical parts of P-bodies, which are cellular granules associated with mRNA decay. However, little is known about how NoBody itself is regulated. The Slavoff lab recently identified a NoBody phosphorylation site located at serine residue 61. We hypothesize that phosphorylation at this site alters NoBody's interaction with DCP1a, subsequently affecting mRNA decay rates. To determine the effect that phosphorylation has on NoBody's activity, we have developed five stable mammalian cell lines from an existing NoBody knockout HEK293T cell line that express wild type, alanine mutant, and phosphomimic aspartate mutant at serine residue 61 of NoBody. Using these cell lines, we are currently using RT-qPCR to determine the rate of cellular mRNA decay as a result of the charge state of residue 61 as well as cell imaging to quantify cellular P-body numbers as a result of the charge state. Furthermore, we plan to conduct a series of co-immunoprecipitation experiments to determine how the S61 charge state affects the cellular association of DCP1a with NoBody. Collectively, these experiments are some of the first to document the regulatory effect of a post-translational modification on a small protein and the subsequent effect on cellular processes.

## Examining Nuclear Envelope Repair in the *C. elegans* Early Embryo

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The eukaryotic nuclear envelope (NE) is composed of two lipid bilayers and establishes a permeability barrier between cytoplasmic and nucleoplasmic contents to protect and regulate the genome. In the event of a loss of the permeability barrier, the NE can reseal in a process similar to nuclear envelope reformation after mitosis or meiosis. In addition to membrane resealing machinery, this process requires membrane incorporation to reduce the size of the holes. Recent work showed that ESCRTs, components of the membrane sealing machinery, are responsible for the sealing of the nuclear envelope. However, the process through which the ESCRT machinery senses rupture and is coordinated to reseal the NE is unknown. We study this process by examining the nuclear envelope sealing of the oocyte-derived pronucleus following meiosis II in the one-cell stage *C. elegans* embryo. Following pronuclear migration, the oocyte-derived pronucleus maintains its permeability barrier until cell cycle-regulated nuclear envelope breakdown. By monitoring oocyte-derived pronuclear sealing, our lab identified that CNEP-1, a phosphatase regulating membrane biogenesis that limits membrane incorporation at the NE, coordinates with LEM-2, an inner nuclear membrane protein coordinating ESCRT recruitment, during nuclear envelope sealing. However, the exact signaling pathway has not been established.

Here, we present a screen aimed at identifying factors essential to the *C. elegans* NE repair pathway. We selected 18 candidate genes believed to be involved in the process. Using RNAi, we are depleting each candidate gene and screen for premature loss of the oocyte-derived pronucleus permeability barrier in a *cnep-1* deletion background. We can precisely identify a functional NE permeability barrier using a quantitative fluorescent assay developed in our lab. Our results suggest that LEM-4, a regulator of BAF phosphorylation, and other uncharacterized proteins play an important role in the establishment of the permeability barrier after meiosis. This could have far-reaching implications due to the functional similarity between post-meiotic NE formation and NE repair.

## Engineering a Synthetic Bacterial Co-culture to Degrade and Metabolize PET Plastics

Kevin Chang<sup>1,2,3</sup>, Cecily Gao<sup>3</sup>, Kevin Li<sup>3</sup>, Ricardo Moscoso<sup>3</sup>, Lauren Telesz<sup>3</sup>, Alice Tirad<sup>3</sup>, Jaymin Patel<sup>4</sup>, and Farren Isaacs<sup>4</sup>

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Polyethylene terephthalate (PET) is a polymer used to make plastic products ranging from synthetic fibers to water bottles. Although PET can be recycled, large amounts of PET end up accumulating in the environment as pollution. A recently discovered bacterium named *Ideonella sakaiensis* was found to degrade PET by using two enzymes, PETase and MHETase, to break PET into its two constitutive monomers: ethylene glycol (EG) and terephthalic acid (TPA). However, *I. sakaiensis*' inability to breakdown PET on a practical time scale undermines its usefulness in eliminating PET pollution. Our project aimed to tackle PET pollution by genetically engineering a synthetic *Escherichia coli* and *Aceintobacter baylyi* co-culture to degrade and metabolize PET. We used the following three-pronged approach: (1) engineer *E. coli* to express and secrete PETase and MHETase for extracellular degradation of PET, (2) engineer *E. coli* to uptake and metabolize EG, and (3) engineer *A. baylyi* to uptake and metabolize TPA by expressing foreign TPA utilization genes from *Comamonas* sp. strain E6. Once individually completed, these three components will then be combined to create a synthetic bacterial co-culture of cooperative *E. coli* and *A. baylyi* amino acid auxotrophs. Since both *E. coli* and *A. baylyi* are more characterized than *I. sakaiensis* and also capable of high-throughput mutagenesis, PET degradation and metabolism pathways in an engineered synthetic *E. coli* and *A. baylyi* co-culture potentially could be optimized to be even more efficient than those natively found in *I. sakaiensis*.

## **Exploring Heterogeneity of IL-12 Secretion in Murine Bone Marrow-Derived Macrophages**

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Macrophages coordinate immune responses through cytokine signaling pathways. Interleukin 12 (IL-12) is a pro-inflammatory cytokine secreted by macrophages that promotes an adaptive T cell response. While molecular mechanisms underlying IL-12 regulation have been studied in cell populations, few have focused on how IL-12 is regulated in single cells. Recent studies have shown that genetically identical macrophages stimulated with the same conditions in the same environment vary greatly in the amount of secreted cytokines that they produce. Given that abnormal concentrations on a population level have been associated with various disorders, understanding the regulations behind this heterogeneity has important implications for the design of therapeutic strategies involving IL-12.

To quantify cell-to-cell variability in IL-12 transcription and secretion, we stimulated murine bone marrow-derived macrophages (BMDMs) with the toll like receptor 4 (TLR4) ligand lipopolysaccharide (LPS) and measured IL-12p40 using different single-cell assays. We found that only a small fraction of cells secretes IL-12 and confirmed that a high concentration of IL-12p40 secreted in a population were primarily produced by this subset. Additional observations that IL-12 transcription and secretion follow similar patterns in our experiments suggest that cell-to-cell heterogeneity is regulated at the transcriptional level and is maintained by the cells through secretion. In future work, we will study how and why cells produce IL-12 heterogeneously. We will investigate how cell proximity affects transcription and secretion, and we will compare regulation of IL-12 to other pro-inflammatory cytokines produced in response to LPS stimulation.

# **Design of Broadband Multipolar Microwave Waveguide Attenuators for Superconducting Qubits**

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Quantum superconducting circuits coupled with cavity resonators are a leading candidate for digital quantum computation, with long energy relaxation lifetimes that are suitable for fault-tolerant quantum computing. However, dephasing due to thermal photons limits the coherence times of quantum bits. Previous work shows that microwave attenuator filters coupled to signal lines into the cavity reduce the number of thermal photons by an order of magnitude and improve qubit coherence times close to the  $2T_1$  limit. While past work has focused on narrow-band filters, which are specialized for a single frequency, a broadband filter with 1 GHz bandwidth is desirable for scalability and coupling to multiple qubits. Furthermore, a stronger understanding of the causes of attenuation would improve future design principles.

We designed and simulated a multipole filter that would achieve both the required attenuation and bandwidth. This filter is modeled with a 3-pole prototypical Chebyshev filter, using waveguide sections. Calculations based on desired center and corner frequencies guide the design. The filter is simulated both as a lumped model and as a full electromagnetic structure. Fabrication and experimental testing of the filter are to be conducted.

In addition, we examined how this filter attenuates using cavity, waveguide, and transmission line models. Under such models, we hypothesize that increasing surface roughness would further attenuate thermal photons, possibly leading to better results over a larger frequency range. We plan on testing this hypothesis by manufacturing a cavity similar to the current narrow-band model using a Flow NanoJet Abrasive water jet cutter, leaving a uniform roughness inside the cavity.

# Unsupervised Behavioral Classification of *Drosophila Melanogaster* from Positional Data in Olfactory Assay

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Navigation through odor plumes is a model paradigm for studying the interaction between sensory stimulus and behavioral response, with applications including pest control and artificial sensing. Studies of navigational strategies of model organisms typically use as their starting point a human-generated dictionary of navigational behaviors (e.g. ‘turning’, ‘accelerating’, etc.). This approach can fail to detect unique behaviors of a particular organism if the dictionary is too generic, or can mis-classify behaviors as a result of human error. In this work, I present a new method for behavioral classification based on data from an olfactory assay performed by the Emonet lab that records positional variables of flies walking to an odor source and the concentration of an odorant. My technique is preferable to manual classification of the resultant data since it is systematic and eliminates human biases in classifying behavior.

My technique transforms a data matrix consisting of the values of assay variables over time into the time-frequency domain using a wavelet transform, which is analogous to taking a Fourier transform at each time step. This equalizes the power between frequency components, to ensure repeated, high motions do not dominate the analysis. I then factor this matrix into a set of behavioral patterns and the extent to which they are present over time using an algorithm called seqNMF. I then reduce the dimensionality of the patterns by determining a set of smaller basis vectors that adequately explains the variance in the data, then clustering similar patterns together in two dimensions such that similar patterns appear in the same cluster.

Preliminarily, my method can effectively cluster patterns corresponding to fast turns, slow turns, and straight motion when the only data used is the orientation of the flies. In addition, the fact that this method extracts both behavioral patterns and the extent to which each pattern is active at each time means that one can analyze behavioral probabilities conditioned on variables such as odor concentration or position; first results of conditional analysis show that after entering an odor cloud, flies have a high probability of sharply turning. Manual inspection of the same dataset failed to produce a similar result. Further research areas include application to more complex datasets and to other organisms.

## Characterization of an Artificial Signal Peptide with Secondary Activity

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Protein targeting to the secretory pathway is achieved via recognition of signal peptides, short transmembrane-like regions located at the extreme N-terminus of nascent polypeptides. Signal peptides are typically cleaved from immature proteins in the endoplasmic reticulum (ER) by the action of signal peptidase, and it is currently believed that cleaved signal peptides are rapidly degraded following this step. However, a small number of known, structurally-complex signal peptides have been shown to escape degradation in the ER and enact secondary functions, often by modulating the activities of unrelated proteins. Our current work investigates the possibility of generating simpler artificial signal peptides with specific activity against transmembrane proteins.

The DiMaio Lab has extensively studied the E5 protein of bovine papillomavirus (BPV), the smallest known naturally-occurring oncoprotein. Although it is only 44 amino acids in length, the E5 protein binds specifically to the transmembrane domain of the platelet-derived growth factor  $\beta$  receptor (PDGF $\beta$ R), thereby activating the receptor in a ligand-independent manner and inducing oncogenic cell transformation. With the E5 protein as a scaffold, genetic libraries of artificial hydrophobic transmembrane protein aptamers ('traptamers') have been constructed and screened to characterize peptides with a range of specific activities against PDGF $\beta$ R or unrelated transmembrane proteins. One such traptamer previously found to activate PDGF $\beta$ R was successfully adapted into a signal peptide that, when expressed in place of the wild-type PDGF $\beta$ R signal peptide, results in a constitutively-active receptor capable of inducing cell growth and proliferation. The requirements for the activity of this construct were investigated to elucidate the mechanism of this active signal peptide. Traptamer library design schematics are being adapted to build a library of signal peptides expressed upstream of PDGF $\beta$ R, which will be screened for peptides with proper targeting function as well as secondary activity against the receptor.

# **Genetic Modification of Diabetic Fibroblasts and Extracellular Matrix for Improved Wound Healing**

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The extracellular matrix (ECM) is increasingly being recognized not just as a physical scaffold, but as a dynamic tissue component that both influences and is influenced by the cells residing within it. The hyperglycemic microenvironment of diabetic tissues induces phenotypic changes in both cells and ECM, leading to impaired wound healing. Fibroblasts, which deposit new ECM and secrete growth factors to coordinate the healing process, exhibit deficiencies in characteristics such as proliferation, mobility, and growth factor production. Diabetic ECM also demonstrates abnormalities such as higher levels of proteases and the presence of advanced glycation end products (AGEs).

Thrombospondin-2 (TSP-2), a homotrimeric matricellular protein, is known to regulate cell-ECM interactions, particularly ECM remodeling. While leptin receptor deficient (db/db) mice, a type II diabetic model, exhibit slowed healing of full-thickness skin wounds compared to wild-type (WT) mice, db/db mice that also possess a TSP-2 knock-out (KO) mutation demonstrate rescued wound healing ability comparable to that of WT mice. This suggests that pro-healing alterations in cells and ECM due to the TSP-2 KO modification may counteract the anti-healing alterations resulting from diabetes. In the present study, we aim to better understand the individual effects of a diabetic microenvironment and the TSP-2 KO mutation on fibroblasts, ECM, and their interactions. We used fibroblasts and cell-derived ECM from WT, diabetic, and diabetic TSP-2 KO models to assay fibroblast contraction and probe relevant signaling pathways; compare ECM collagen fiber properties using SEM image analysis; and analyze fibroblast morphology when different cells were mixed-and-matched onto different ECM.



# Dissecting Asymmetric Locomotor Coordination During Turning in *Drosophila*

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In order to navigate dynamic environments, animals must generate a wide variety of complex locomotor behaviors. Legged animals must be able to turn, coordinating limb movements to change their body orientation about a vertical axis. Though the neural basis of limb coordination during forward walking has been investigated in a variety of model systems, it is not well understood how the same circuitry executes a fundamentally different pattern of movement during turning, generating asymmetric coordination across the body. In particular, the suite of leg coordination patterns that generate turning remains unknown. Using machine vision methods, we have constructed a high-throughput assay for analyzing the posture and limb-tip positions of freely walking flies, allowing us to collect high-dimensional datasets of the limb configurations patterns used by *Drosophila* during turning behaviors. By applying an unbiased machine-learning approach to cluster these data, we show that turns which are similar at the level of centroid kinematics may be generated by distinct patterns of limb coordination. Yet, depending on the characteristics of turn being executed, different coordination patterns may be used preferentially. Our results demonstrate a principle of abundance in *Drosophila* motor control; as the fly has at its disposal many ways in which to generate a turn, the system is robust to neural or mechanical perturbations. We anticipate that this work will serve as a starting point for characterizations of how specific neural activity patterns generate ethological motor outputs in the fly.

### **Morton and Maggie Rosenfeld, J.D.**

Mort and Maggie Rosenfeld, born and raised in St. Louis and Chicago, respectively, have lived in Los Angeles for 45 years. Mort holds an undergraduate degree from Princeton and a JD from the University of Michigan Law School. He practiced law for more than 42 years in Los Angeles, the last 26 of which at a small business law firm which he founded. He retired three years ago and is presently writing novels. Maggie is a graduate of the University of Michigan and UCLA Law School. She practiced at a well-known Los Angeles law firm for 18 years before turning to part time practice for a period of time before assuming her current position as the head of business operations for a private elementary school where she previously served as the Chair of its Board of Trustees.



The Rosenfeld's are the parents of two sons who are Yale graduates. Andrew earned both a BA and MA in Economics as a member of the Class of 2004. Daniel received a BS and MS in Chemistry in 2007. Daniel spent each of his summers at Yale doing research, which, following discussion with Yale science administrators, inspired the funding for Rosenfeld Science Scholars originally known as Yale Science Scholars. The Yale College Dean's Office would like to thank the Rosenfelds for their generous support of Yale undergraduate STEM research.

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