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EDUCATION

1998-2001 Doctor of Science (Sc.D.), Mechanical Engineering
Massachusetts Institute of Technology (MIT) Cambridge, MA, USA

1995-1996 Master of Science (M.Sc.), Mechanical Engineering
Massachusetts Institute of Technology (MIT) Cambridge, MA, USA

1988-1992 Bachelor of Science (B.Sc.), Mechanical and Aerospace Engineering
Cornell University, Ithaca, NY, USA

CURRENT POSITIONS

2026- 2027 Visiting Sabbatical Scholar, Biosensors for Bioengineering Group, Institute for Biomedical Engineering of Catalonia (IBEC), Barcelona, Spain, EU

2026- 2027 Collaborating Scholar, Department of Physiological Sciences, Faculty of Medicine and Health Sciences, University of Barcelona (UB), Spain, EU

2025- 2028 Board of Directors, Biomedical Engineering Society (BMES), Landover, MD, USA

2019- Now Professor of Biomedical Engineering (BME, with Tenure),
Rutgers, The State University of New Jersey, New Brunswick, NJ, USA

PREVIOUS POSITIONS

2022- 2025 Board of Directors, American Institute of Medical and Biological Engineering (AIMBE),VA, USA

2007- 2018 Associate Professor of Biomedical Engineering (BME, with Tenure),
City University of New York (CUNY-City College), New York, NY, USA

2001- 2006 Department Co-founder and Assistant Professor of Biomedical Engineering
City University of New York (CUNY-City College), New York, NY, USA

1998- 2001 Research Assistant, MIT Whitehead Institute for Biomedical Research, Cambridge, MA

1996- 1998 Teaching Fellow, MIT Department of Mechanical Engineering, Cambridge, MA, USA

1992- 1996 Mechanical Engineer (Cleanroom/Process contamination), Intel Corporation, CA, OR, USA

RESEARCH ACHIEVEMENTS (SUMMARY AND 10 SIGNIFICANT OUTPUTS)

Summary. *As a Principal Investigator (PI) at the interface of microfluidics, biophysics, and tissue engineering, I have developed my career in pursuit of a fundamental question:* ***How does the tissue environment govern the behavior of living cells?*** *Early training in the engineering industry paired with decades of applied academic research has guided my creation of in vitro models that reconstruct critical biophysical barriers and geometries of adult neural tissues. My group has developed quantitative guidance environments to represent damaged tissues using biochemical ligands, electrical signals, and biomatrix modalities. Our established high precision platforms have challenged presumptions of cell passivity in mature tissues to reveal that: a.) Exogenous cells retain latent capacities to sense biochemical guidance; b.) Adhesion constraints of restrictive tissue architecture control cell navigation despite concurrent cues; and c.) Overlapping biochemical and bioelectric signals produce non-linearly coordinated cell behaviors. Our recent attempts to control these cell responses through a single molecular regulator exposed the critical limitation of reductionist strategies and shifted our research program toward coordinated system level analyses. We moved beyond descriptive observation of cell behavior toward predictive modeling by developing agent-based computational models that convert our prior measurements of cell interactions into rules of navigation. These mounting discoveries expose the core bioengineering question driving our research:* ***What coordinated rules of intracellular signaling transform tissue guidance cues into predictable cell behavior?*** *This progression demonstrates my capacity to integrate engineering, cell biology, and computation around scientific questions that cannot be resolved within any single discipline. Key outputs are arranged in three themes of discovery, mechanism, and modeling below. To pursue scientific expansion, I established collaborations to extend my research program into lab-chip platforms, phosphoproteomics, and biological validation, while retaining the core scientific question and integrative framework within my research program.*

Theme-1 (Discovery): Donor Cells Retain Latent Guidance Capacity in Dense Environments

1. Ligand signaling activates baseline motility in donor cells.

Unachukwu, U., et al., PLoS One. 2013 Dec 23;8(12):e83906 (doi: 10.1371/journal.pone.0083906)

This initial study challenged presumptions that donor cells were non-motile in adult biochemical microenvironments. We demonstrated that microfluidic EGF gradients induced chemokinetic motility in donor cells via EGFR signaling. The findings provide experimental microfluidic baselines for ligand-

driven motility and target molecular pathways (PI3K) that achieve directed migration. As co-corresponding author, I led this project to demonstrate precise microfluidic control of donor motility.

2. Injury-associated cytokines activate persistent environmental control of donor cell migration.

Unachukwu, U., et al., Sci. Rep. 2016 March 3:2, 69 (doi: 10.1038/srep22392)

We demonstrate that donor cells retain intrinsic motility programming and sustain migration along microfluidic gradients of injury cytokines. The discovery challenged the view that donor cells are intrinsically passive in adult tissue and established environmental signaling as a mechanism to control cell guidance. As the senior researcher and co-corresponding author, I led this bioengineering project.

3. Gradient microstructure determines the spatial resolution of chemotactic sensing in donor cell groups.

Mccutcheon, S., et al., Biomed Microdevices. 2015 Dec;17(6):107 (doi: 10.1007/s10544-015-0002-6)

Studies illustrate that donor cell groups resolve precisely structured biochemical gradients in our customized microfluidic platform to indicate that spatial patterning of local cues constrains chemotactic sensing. These findings establish the spatial constraints and resolution needed to translate donor cell guidance to 3D microenvironments. As principal investigator (PI) and corresponding author, I conceived the research direction and led technology development with biological validation.

4. Nanoscale geometry, not cell identity, gates cell-cell communication in our multiscale platform.

Mishra, S., et al., Biomed. Microdevices, 2017 Aug 8;19(3): 71 (doi: 10.1007/s10544-017-0212-1)

The work reveals that synaptic capacity is constrained by nanoscale geometry rather than cell identity. We developed a technology platform that recreates tissue scale geometry for analysis of cell morphology, migration, and cell-cell communication in physiological constraints. Donor cells showed 10-fold elongation to establish gap junctional contacts with distal neighbors, revealing physical architecture as a determinant of cell communication. As PI and corresponding author, I guided design, prototyping, and validation of the platform to hold pending patent rights (Ser. No. 63/521,975).

Theme-2 (Mechanism): Collective Environmental Cues Reshape Donor Cell Guidance

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5. Electro-chemotactic (EC) stimulation reveals integrated control of directional migration.

Mishra, S., et al., Biosensors (Basel) 2017 Nov 21;7(4) (doi: 10.3390/bios7040054),

Our group introduced EC stimulation to address how overlapping physical and biochemical cues jointly regulate sustained migration, a significant gap in donor cell guidance. We balanced chemotactic gradients with electric fields to illustrate 3-fold increases in donor cell migration compared with either stimulus alone. As PI and corresponding author, I conceived the EC paradigm and directed its validation.

6. Transcriptional mechanisms link electro-chemotactic (EC) cues to donor cell migration.

Pena, J., et al., Exp. Eye Res. 2019, Aug;185:107688 (doi: 10.1016/j.exer.2019.06.002)

Studies showed that electro-chemotactic migration is associated with transcriptional changes in adhesion and cytoskeletal remodeling, rather than surface-receptor expression. These findings elevate electro-chemotaxis from descriptive to mechanistic by providing evidence that extrinsic cues reprogram cellular motility through downstream pathways. As PI and corresponding author, I conceived the study, led the experimental design, and directed the mechanistic testing.

7. Single gene control fails to predict donor cell motility across microenvironments

Dabrowski, A., et al., Explor Biomat-X 2025;2:101335 (doi: 10.37349/ebmx.2025.101335),

We demonstrated that Top2b, as a single genetic switch, regulates migration in an extracellular matrix-dependent manner but cannot universally control donor motility across microenvironments. These findings revealed a key limitation of single gene strategies and point to context dependent signaling networks as the next mechanistic challenge. As PI and corresponding author, I directed this genetics collaboration to integrate the mechanistic basis of donor motility into my research program.

Theme-3 (Model): Multimodal Guidance Cues Predict Cell Navigation

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8. Chemotactic signaling and adhesion jointly drive donor cell migration.

Markey, M., et al., Tissue Eng Regen Med 2023, Aug 24;2023:1381620 (doi: 10.1155/2023/1381620)

This study illustrates divergent motility between individual and collective donor cell cohorts from both invertebrate and mouse cells. We demonstrated that chemotactic fields increase cell-to-cell contacts to drive group migration and that these micro-clusters enhance overall motility via coordinated cell-matrix adhesion mechanisms. These findings target adhesion as a key determinant of donor cell migration and

provide critical data to develop predictive modeling of motile behaviors. As PI and corresponding author, I conceived the study and directed its experimental design and analysis.

9. An agent-based computational model (ABM) predicts donor cell migration.

Oprysk, L., et al., PLoS Comput. Biol. 2025 Mar 10;21(3):e1012769 (doi: 10.1371/journal.pcbi.1012769)

We developed a computational framework that predicts donor migration into restrictive architectures via cell-cell interactions and mechanical constraints. Independent datasets using donor cells from mouse and drosophila validated model predictions, demonstrating conserved principles across cell systems. This work shifted our research program from measurement of donor cell behavior toward prediction of emergent responses using this quantitative ABM framework to test the effects of cell guidance cues. As co-PI and corresponding author, I led experimental validation and oversaw code development to add these computational tools into my research platform.

10. Closed loop optical tracking enables rapid 3D analysis of donor cell guidance.

Koul, S., et al., Micro 2025, 5(1), 10 (doi: 10.3390/micro5010010)

Our group engineered a modular 3D biomaterial platform that combines cues from transient electric field guidance (EF) with non-destructive optical tracking of donor cell behavior. This system permits longitudinal cell measurement in tissue-like samples without sectioning, to accelerate iterative testing fivefold. EF is applied across a hydrogel resistor to prevent Joule heating and preserve integrity of samples for cell tracking. This platform extends our research toward analysis of cue-induced navigation with predictive modeling. As PI and corresponding author, I conceived and validated this platform.

PEER RECOGNITION OF INTERNATIONAL SCIENTIFIC LEADERSHIP

These selected distinctions highlight my recognized scientific contributions and leadership within biomedical engineering, including 150 invited presentations and 100 mentored students. (Full listing on website.)

A. Fellow Status and Academy Memberships (elected by peer committee)

1. **Elected Fellow**, American Institute of Medical and Biological Engineers (2020). Conferred upon elite bioengineers who drive global discoveries and innovation. Awarded for my contributions of microfluidic bioassays to tissue engineering.
2. **Study Section Member**, Special Emphasis Panels for National Institutes of Health (2018-2024). Invited to review competitive research proposals twice annually in scientific review panels of bioengineering.
3. **Elected Fellow**, Biomedical Engineering Society (2022). Awarded for contributions of translational microfluidic bioassays into BME research and educational programs.

B. International Keynotes and Plenary Addresses (invited by scientific committee)

1. **Gordon Lecture,** The Physics and Chemistry of Microfluidics (2017, Italy). Selected by an international committee of experts for my contributions at the intersection of microfluidics and guided cellular integration. I was the first-ever invited speaker to this renown conference from a USA Minority Serving Institution. The lecture helped establish an EU-based network for collaboration.
2. **Gordon Keynote,** Research Seminar in Microscale Fluid Phenomena (2023, Italy). Invited by over 100 early-career researchers in microfluidics for contributions of my multidisciplinary cell-based biotechnologies.
3. **Keynote,** TERMIS-AM (2025, USA). Invited to present my platform of electro-chemotactic guidance in tissue microenvironments: Tissue Engineering and Regenerative Medicine International Society (Americas).

C. Recent Professional Distinctions, Honors, and Awards

1. **Institutional Impact Award,** Biomedical Engineering Society (2015). The award reflects my capacity to develop lasting institutional infrastructure as a co-founder of the BME department at CUNY.
2. **Inaugural President's Award for Excellence**, CUNY (2017). The university's highest honor recognizes my development of a competitive research program to advance science, technology, and student mentoring.
3. **Professional Impact Award**, AIMBE (2023). Awarded for spearheading development of accessible cell-based technologies into modern BME curricula and under/graduate mentoring.

D. Competitive Grant Funding (most related completed proposals)

As an independent PI, I have continuously secured competitive funding to lead an interdisciplinary laboratory of 6-10 researchers across microfluidics, cell biology, and computational modeling. The selected awards illustrate scientific progression of my projects and represent a subset of my broader funding portfolio.

1. Photoreceptor integration via chemotactic honing, PI: Maribel Vazquez, Sc.D.
National Institutes of Health (USA), High Risk/High Reward Project (R21-EY026752), completed 2018.

Established a quantitative framework to examine donor cell chemotaxis. We revealed latent chemotactic capacity in contrast to prevailing concepts of donor cell passivity but discovered that biochemical gradients alone cannot sustain migration over extended distances. This unexpected limitation led us to develop electro-chemotactic guidance as a new strategy to manipulate donor cell migration.

2. Microfluidic models of emerging behaviors in tripartite systems, Multi-institutional Research Center, PI (10-year Sub-contract): Maribel Vazquez, Sc.D.
National Science Foundation (USA) Science and Technology Center (CBET-0939511): EBICS (Emerging Behaviors of Integrated Cellular Systems), completed 2020.

Developed microfluidics to reconstruct communication across multipartite systems of the neuro-muscular junction and neurosensory retina. Findings revealed interdependence between cell motility and synaptic communication, expanding my bioengineering focus from donor cell migration to coordinated cell behaviors that increasingly govern navigation across dense tissue systems.

3. A combined microfluidics and in vivo modeling approach to evaluate migration during retinogenesis, PI: Maribel Vazquez, Sc.D.
National Science Foundation (USA), Bioengineering Project (CBET-180441), completed 2022.

Evaluated conserved chemosensitivity of cell donors using microfluidic assays with primary and genetically modified retinal stem cells from invertebrates and mice. We discovered that cell adhesion had a greater influence on donor cell motility than cell identity or cohesion, revealing a significant determinant of migration. This finding expanded our research to include computational modeling of cell behaviors.

4. Modulating electro-chemotactic (EC) stimuli and Top2b-mediated pathways to promote integration of cone progenitor cells, PI: Maribel Vazquez, Sc.D.
National Institutes of Health (USA), High Risk/High Reward Project (R21-EY031439), completed 2024.

Examined how genetic manipulation (Top2b) influenced the electro and chemotactic migration of mouse donor retinal progenitor cells. We showed that altering Top2b failed to enhance migration and revealed constraints in downstream signaling. This unexpected result exposed limitations of single gene strategies and shifted our research toward study of intracellular signaling mechanisms that govern donor cell behavior.

ADDITIONAL NOTEWORTHY CONTRIBUTIONS TO THE RESEARCH COMMUNITY

1. **BME Department Co-founder,** CUNY. As one of four founding faculty, I worked to create a 128-credit (250-ECTS) undergraduate major, develop 11 of its 16 core technical courses, and lead its successful ABET accreditation (EUR-ACE). Via competitive funding, I established the first microfabrication facility in the school of engineering to modernize its educational and research programs.
2. **National Program Leadership and Cross-Institutional Infrastructure.** As PI, I now direct a national NSF Research Experiences for Undergraduates (REU) Site in Cellular Bioengineering, recruiting and mentoring undergraduate cohorts from over 50 institutions. Through competitive awards for NIH specialized centers (co-PI) and NSF Science and Technology Centers (Sub-contract PI), I established additional pipelines to expand BME research capacity and workforce across institutions. Through these programs my mentoring portfolio includes over 100 direct mentees across biomedical industry, academia, and start-up ventures.

CAREER BREAKS AND MAJOR LIFE EVENTS

1. **Instruction and Administrative Load (2001-2006).** To establish a new BME Department, I developed a distinctive, early-career leadership portfolio over research outputs. In tandem, I grew an independent microfluidics program by mentoring students from established local doctoral programs to secure funding.
2. **Maternity Leaves (2008, 2012).** I maintained the continuity of my research program despite a lack of USA institutional maternity leave and college policies of doubled instructional duties upon each return.
3. **Pandemic Closures (2020-2022).** Stringent restrictions suspended all laboratory operations and prohibited all interstate travel between my New York residence and New Jersey laboratory. I preserved research productivity by developing computational analyses to complement experiments until lab work resumed.