OMB No. 3145-0279 (Expiration Date 10/31/2026)

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Vazquez, Maribel

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0002-6184-3103

Position Title: Professor of Biomedical Engineering

Organization and Location: Rutgers, The State University of New Jersey, New Brunswick, New Jersey, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
Massachusetts Institute of Technology (MIT), Cambridge, Massachusetts, United States	Doctor of Science (SCD)	09/1998	09/2001	Mechanical Engineering
Massachusetts Institute of Technology (MIT), Cambridge, Massachusetts, United States	Master of Science (MS)	01/1996	08/1996	Mechanical Engineering
Cornell University, Ithaca, New York, United States	Bachelor of Science (BS)	08/1988	06/1992	Mechanical and Aerospace Engineering

Appointments and Positions

2019 - present	Professor of Biomedical Engineering, Rutgers, The State University of New Jersey, New Brunswick, New Jersey, United States
2025 - 2028	Executive Board of Directors, Biomedical Engineering Society (BMES), Arlington, Virginia, United States
2022 - 2025	Board of Directors, Vice President at Large, American Institute of Medical and Biological Engineers (AIMBE), Arlington, Virginia, United States
2007 - 2018	Associate Professor of Biomedical Engineering, City University of New York (City College) , New York, New York, United States
2001 - 2006	Assistant Professor of Biomedical Engineering, City University of New York (City College) , New York, New York, United States
1998 - 2001	Research Associate, MIT Whitehead Institute for Biomedical Research, Cambridge, Massachusetts, United States
1996 - 1998	Teaching Fellow: Department of Mechanical Engineering, Massachusetts Institute of Technology (MIT), Cambridge, Massachusetts, United States
1992 - 1996	Mechanical Engineer, Intel Corporation , Portland, Oregon, United States

Products

Products Closely Related to the Proposed Project

1. Koul S, Devecka LA, Pierce MC, Vazquez M. A Microscale-Optical Interface to Examine Electric Field-Induced Cell Motility Within Whole-Eye Facsimiles. Micro (Basel). 2025 Mar;5(1) PubMed Central PMCID: PMC12208019.
2. Oprysk LM, Vazquez M, Shinbrot T. Internal cohesion gradient as a novel mechanism of collective cell migration. PLoS Comput Biol. 2025 Mar;21(3):e1012769. PubMed Central PMCID: PMC12077783.
3. Markey MW, Pena CD, Venkatesh T, Cai L, Vazquez M. Retinal Progenitor Cells Exhibit Cadherin-Dependent Chemotaxis across Transplantable Extracellular Matrix of In Vitro Developmental and Adult Models. J Tissue Eng Regen Med. 2023;2023:1381620. PubMed Central PMCID: PMC11919238.
4. Vazquez M. Microfluidic and Microscale Assays to Examine Regenerative Strategies in the Neuro Retina. Micromachines (Basel). 2020 Dec 9;11(12) PubMed Central PMCID: PMC7763644.
5. Mishra S, Peña JS, Redenti S, Vazquez M. A novel electro-chemotactic approach to impact the directional migration of transplantable retinal progenitor cells. Exp Eye Res. 2019 Aug;185:107688. PubMed Central PMCID: PMC6698415.

Other Significant Products Highlighting Contributions to Science

1. Peña JS, Vazquez M. Harnessing the Neuroprotective Behaviors of Müller Glia for Retinal Repair. Front Biosci (Landmark Ed). 2022 May 30;27(6):169. PubMed Central PMCID: PMC9639582.

2. Vazquez M. Electro-chemotactic stimuli for cell replacement therapy in neurosensory retina. Neural Regen Res. 2020 Mar;15(3):450-452. PubMed Central PMCID: PMC6921348.
3. McCutcheon S, Majeska R, Schaffler M, Vazquez M. A multiscale fluidic device for the study of dendrite-mediated cell to cell communication. Biomed Microdevices. 2017 Aug 8;19(3):71. PubMed Central PMCID: PMC6350513.
4. Unachukwu UJ, Warren A, Li Z, Mishra S, Zhou J, Sauane M, Lim H, Vazquez M, Redenti S. Predicted molecular signaling guiding photoreceptor cell migration following transplantation into damaged retina. Sci Rep. 2016 Mar 3;6:22392. PubMed Central PMCID: PMC4776098.
5. Dudu V, Rotari V, Vazquez M. Sendai virus-based liposomes enable targeted cytosolic delivery of nanoparticles in brain tumor-derived cells. J Nanobiotechnology. 2012 Feb 17;10:9. PubMed Central PMCID: PMC3352066.

Certification:

I certify that the information provided is current, accurate, and complete. This includes, but is not limited to, information related to current, pending, and other support (both foreign and domestic) as defined in 42 U.S.C. § 6605.

In accordance with Section 10632 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19232), each individual identified as a senior/key person must certify that they are not a party to a malign foreign talent recruitment program.

Research Security Training Requirement for Federal Award Personnel: In accordance with Section 10634 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19234), each individual identified as a senior/key person must certify that they have completed the requisite research security training that meets the requirements specified in Item 2 of Important Notice No. 149 within 12 months prior to proposal submission.

Misrepresentations and/or omissions may be subject to prosecution and liability pursuant to, but not limited to, 18 U.S.C. §§287, 1001, 1031 and 31 U.S.C. §§3729-3733 and 3802.

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NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: Vazquez, Maribel

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0002-6184-3103

Position Title: Professor of Biomedical Engineering

Organization and Location: Rutgers, The State University of New Jersey, New Brunswick, New Jersey, United States

Personal Statement

Integrated Bioengineering Platform.
My research laboratory develops bioengineering approaches for emerging neural therapies by combining engineering technology with mechanistic cellular responses. Extensive engineering training from industry and applied research in academia has guided my development of nano and microfluidic systems that correlate extracellular cues with activated signaling pathways in progenitor and mature cells of the nervous system (PMC7763644). We have produced several microfluidic models to recapitulate critical biophysical barriers and confined physiological geometries (PMC8009310) of multi-partite, neural tissues. These include microphysiological systems to examine cell migratory responses in a variety of external fields and molecular transport of critical compounds across extracellular matrix and cellular barriers. Our quantitative systems have helped enrich the understanding of cellular integration processes in degenerated and apoptotic tissue. Bioengineering projects from our group to develop microfluidic, biomaterial, and organotypic explant system models of the retina have been sponsored by awards from the NIH (EY031439; EY026752) and the NSF (2243644; 18044). My lab has collaborated with Dr. Stephen Redenti for over a decade in the development of regenerative approaches to advance transplantation outcomes in degenerated retina.

Treatments for Vision Loss.
Studies from my group advance the development of cell-based therapies in the visual system by examining the effects of electric fields, ligand concentration gradients, and hybrid stimuli of electro-chemotactic (EC) fields. Tests measure dynamic responses of retinal neurons, glia, and neurovascular tissue barriers (PMC6698415) to extracellular cues. We apply analytical and numerical models (MATLAB, COMSOL) to precisely describe the microenvironments created in our devices and employ computational, agent-based models to investigate emerging cell behaviors therein (PMC12077783). Our results have demonstrated that responses of neural progenitors are finely regulated by superior cell sensitivity to the chemical and electric field gradients in adult tissues. Moreover, we have examined the significance of transplantable matrix in cell adhesion, displacement, and migratory response to external cues (PMC5764132) significant to cell replacement therapy. We recently developed an omics approach to target mechanistic interactions between membrane signaling proteins in different retinal progenitor cell groups and biochemical environments of degenerated retina (PMC4776098).

Progress from Early Career.
My academic career began as a co-founder of the first public Biomedical Engineering department in the state of New York (Harlem). Publication during this early career (2001-2007) was heavily impacted by foundational development of an undergraduate curriculum and its engineering accreditation. This endeavor demanded extraordinarily high administrative and instructional duties that reduced my research productivity. In addition, maternity leave was not supported by New York state until 2016, requiring that I use time accrued for medical and sabbatical leaves to financially support each 12-week period protected by the FMLA. I relocated my laboratory in 2019 to a more research-focused environment at Rutgers University, where I remain committed to public engineering education via undergraduate research programs sponsored by the university and NSF (EEC-23-49225; EEC-19-50509).

Honors

2024	Cheryl Wall Fellowship for Faculty Excellence, Rutgers, The State University of New Jersey
2024	Plenary Lecture: Inspiring Health Disparities Research and Education within Biomedical Engineering, Biomedical Engineering Society (BMES), Council of Chairs
2023	Professional Impact Award, American Institute of Medical and Biological Engineers (AIMBE)
2023	International Keynote: Microfluidic Modeling of Neurovascular Barriers and its Impacts in Health Disparities, Gordon Research Conference in Microscale Fluidic Phenomena
2022	Elected Fellow, Biomedical Engineering Society, BMES
2020	Elected Fellow, American Institute of Medical and Biological Engineers, AIMBE

2018	President's Award for Excellence (Inaugural), City University of New York, City College
2017	Invited Expert Lecture: Microfluidics for the Visual System, Gordon Research Conference in Physics and Chemistry of Microfluidics
2002 - 2008	MIT Review Committee, Department of Mechanical Engineering, Invited Alumna Member

Contributions to Science

1. **Microfluidic Modeling of Adult Neural Environments**

   Background.
   The retina is a seminal developmental model where local cell–cell communication directs cell fate and behavior. Regenerative medicine, by contrast, relies upon cell responses to environmental cues from adult retina, which are less understood. Models of mature retina are challenging because its anatomical landmarks and physiological constraints are defined by microscale parameters, while its multi-partite cell responses occur at the mesoscale. We have addressed this unmet need by developing microfluidic models of adult retinal environments to anatomical scale.

   Central Findings.
   Our studies have demonstrated the surprising chemosensitivity of retinal progenitors to ligands produced by damaged adult retina, which complement its known responses to well-examined developmental gradients (PMC11919238). We have further illustrated a dosage response, where varied gradient profiles of the same ligand induce different modalities of migration, average velocities, and cell directionality upon a variety of biomaterials comprised of retinal substrate proteins.

   Impact.
   Our group has become a pioneer in microfluidic modeling of the neuroretina, where our technologies recapitulate ultra-low cytokine gradients that are maintained by interstitial flows for homeostasis and repair (PMC7763644). These controllable microenvironments facilitate quantitative study of intra- and inter-cellular responses to external stimuli (PMC3352066), which advance development of bioengineering approaches to regenerative medicine.

   My Role.
   As the principal investigator, I led the design and development of microfluidic assays, validation of their transport phenomena using analytical and computational models, and measurement of biological impacts using immunocytochemical staining and imaging analysis. I also ensured proper microfabrication protocols, statistical analysis, and edited the manuscripts and data figures for publication quality.

2. **Bioengineering Approaches to Cell Replacement Therapy**

   Background.
   Cell replacement therapies show great promise to restore vision by transplanting stem-cell derived, donor cells to replace dystrophic neurons. However, synaptic integration requires donor cells to traverse a network of retinal neurons whose confined spacing requires enormous configurational changes that raise the energy barrier for migration. Successful transplantation outcomes require new approaches to guide donor cells toward desired synaptic targets.

   Central Findings.
   We have shown that chemotactic gradients alone are insufficient to overcome mechanical and adhesion-mediated constraints of the confined microenvironment (PMC6350513). We further demonstrated dramatic increases in donor cell migration when ligand gradients were combined with electrical fields used in modern ophthalmic treatments (PMC6698415).

   Impact.
   Our group has produced an original and novel external stimulus of electro-chemotactic (EC) fields to guide donor cell migration (PMC6921348). Studies illustrate mechanistic changes in EC-induced migration but no effect on receptor expression. Moreover, we recently validated the superior migration of donor cells across EC fields using 3D eye facsimiles of biomimetic retinal hydrogels (PMC12208019).

   My Role.
   As the principal investigator (PI), I led the development of electro-chemotactic fields and its analytical validation. I also supervised the creation and testing of 3D eye facsimiles to model in situ transplantation and statistical measurements of motile cells therein. I derived the primary manuscripts for publication and revised the data figures for accuracy and quality.

3. **Collective Behavior of Retinal Cells as Neuroprotection**

Background.
Modern advances in neuroscience have focused on the development, function, and plasticity of specialized neuronal cell groups. Surprisingly, a much smaller subset of projects has examined collective neuronal interactions in adult tissue or neuronal communication with cognate glial partners for collective responses to stimuli.

Central Findings.
Experiments from our group have demonstrated that collective behaviors of neural progenitors produce coordinated responses to external stimuli that are superior to that of individual cells (PMC5764132). This data was complemented by computational study, where agent-based modeling (ABM) illustrated that external gradient fields impact average cell velocity, directionality, and modality of migration (PMC12077783).

Impact.
We have become leaders in using ABM to predict responses of cell cohorts to external stimuli in adult retina. Computational results are likewise validated with experimental data from our microfluidic systems that facilitate study of natural cognate pairs, such as Muller glia and photoreceptor cells.

My Role.
As the principal investigator, I led the design and development of microfluidic testing and the validation of microenvironments using analytical and computational models. I supervised the coding elements of ABM and co-derived manuscripts for publication with precise data figures and statistical analysis.

4. **Muller Glia as Signaling Partners in Regeneration and Repair**

Background.
Despite the advancements of many contemporary therapies to restore vision, few approaches have considered the innate benefits of early gliosis, the endogenous processes of retinal repair that precede vision loss. Intrinsic behaviors of Muller glia hold promise to exploit glial reparative ability, while repressing neuro-disruptive MG responses.

Central Findings.
Our studies have demonstrated neuroprotective roles of Muller glia through chemotactic migration during early gliosis. Interestingly, the cells did not alter the cytokine environment sufficiently to impact protein expression of key receptors (e.g. VEGF), but rather demonstrated fine chemosensitivity to ligand gradients. Complementary studies illustrated collective roles of Muller glia to recover permeability of the hyperglycemic inner blood retinal barrier (iBRB), decrease oxidative stress, and upregulate receptor proteins to initiate retinal repair.

Impact.
Results demonstrate strong collective response of retinal barrier tissue specifically with cognate Muller glia, rather than astrocytes, to highlight the importance of these cells as partners in signaling and repair. Migration studies have also advanced the study of early stage gliotic processes in the visual system as a viable strategy to improve outcomes of regenerative therapies.

My Role. As the principal investigator, I supervised research design of microfluidic assays and testing, immunocytochemical staining, statistics, and edited the manuscripts for publication quality.

5. **A Bioengineering Approach to Reduce Health Disparities in Regenerative Medicine**

Background
Biomedical study of progressive neural disorders has underscored profound health disparities among adults in the United States and worldwide. Burden of disease will be lessened when regenerative treatments are developed cognizant of potential disparate impacts on medically underserved communities. My educational efforts are developed to increase awareness of health disparities among younger researchers and to motivate development of inclusive therapies by considering the medical differences between affected groups.

Central Findings.
We have developed undergraduate curricula and design projects for biomedical engineering courses that are openly shared online and via BMES. Interest in health disparities has also taken center stage at BMES and AIMBE to indicate rising awareness in the bioengineering community and the imperative to eliminate disparities in emerging therapies.

Impact.
I have become one of few bioengineering leaders to integrate development of regenerative therapies with needs to reduce health disparities. My plenary addresses and keynote lectures at inter/national societies demonstrate rising interest in development of new therapies that seek to eliminate health disparities in affected patients. My health disparities focus has also led to several education and funding awards.

My Role.
As the principal investigator and educator, I develop all materials and publications directly from my research laboratory and capstone design projects. I develop all of the manuscripts for publication.

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