NIH Biographical Sketch Common Form

Name: Jozic, Ivan

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0001-5114-9524

Position Title: Research Associate Professor, Dr. Phillip Frost Department of Dermatology and Cutaneous Surgery

Organization and Location:University of Miami Miller School of Medicine, Miami, Florida, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
University of Miami Miller School of Medicine, Miami, Florida, United States	Postdoctoral Fellow	04/2013	07/2018	Wound Healing
Florida International University, Miami, Florida, United States	Doctor of Philosophy (PHD)	08/2007	05/2013	Cell Biology
Nova Southeastern University, Davie, Florida, United States	Bachelor of Science (BS)	08/2004	05/2006	Biology

Appointments and Positions

2025 - present	Research Associate Professor, Dr. Phillip Frost Department of Dermatology and Cutaneous Surgery, University of Miami Miller School of Medicine, Miami, Florida, United States
2024 - present	Founding Director of Dermatopathology Spatial Omics Core Facility, University of Miami Miller School of Medicine, Miami, Florida, United States
2024 - present	Chief Operating Officer, AdaptBio Therapeutics, Fort Lauderdale, Florida, United States
2021 - present	Faculty Member Graduate Program in Biomedical Sciences, University of Miami Miller School of Medicine, Miami, Florida, United States
2019 - present	Reviewer, NIH SBIR/STTR ZRG1 study section, Bethesda, Maryland, United States
2014 - present	Member, Wound Healing Society, Beverly, Massachusetts, United States
2019 - 2025	Research Assistant Professor, Dr. Phillip Frost Department of Dermatology and Cutaneous Surgery, University of Miami Miller School of Me, Miami, Florida, United States
2018 - 2019	Instructor, Dr. Phillip Frost Department of Dermatology and Cutaneous Surgery, University of Miami Miller School of Medicine, Miami, Florida, United States
2014 - 2018	Adjunct Professor, Nova Southeastern University, Davie, Florida, United States

Products

Products Closely Related to the Proposed Project

1. Marjanovic J, Ramirez HA, Jozic I, Stone RC, Wikramanayake TC, Head CR, Abdo Abujamra B, Ojeh N, Kirsner RS, Lev-Tov H, Pastar I, Tomic-Canic M. Dichotomous role of miR193b-3p in diabetic foot ulcers maintains inhibition of healing and suppression of tumor formation. Sci Transl Med. 2022 May 11;14(644):eabg8397. PubMed Central PMCID: PMC9707408.
2. Marjanovic J, Jurczuk V, Tose LV, Cintron Diaz Y, Fernandez Lima F, Abdo Abujamra B, Danker S, Jabori S, Singh D, Burgess JL, Tam J, Samandari M, Stone RC, Davis SC, Kirsner RS, Tomic-Canic M, Andreopoulos FM, Jozic I. Scaffolds with spatiotemporally controlled growth factor delivery and cyclodextrin-enabled antagonism of growth factor receptor sequestration promote cutaneous wound healing. NPJ Regen Med. 2025 Sep 29;10(1):42. PubMed Central PMCID: PMC12480632.

Other Significant Products Highlighting Contributions to Science

1. Jozic I, Abujamra BA, Elliott MH, Wikramanayake TC, Marjanovic J, Stone RC, Head CR, Pastar I, Kirsner RS, Andreopoulos FM, Musi JP, Tomic-Canic M. Glucocorticoid-mediated induction of caveolin-1 disrupts cytoskeletal organization, inhibits cell migration and re-epithelialization of non-healing wounds. Commun Biol. 2021 Jun 18;4(1):757. PubMed Central PMCID: PMC8213848.
2. Kirsner RS, Pastar I, Krambrink A, Lev-Tov H, Burgess JL, Kolenic G, Jozic I, Catanuto P, Marjanovic J, Jones TLZ, Song

PX, Schmidt B, Pop-Busui R, Holmes CM, Spino C, Tomic-Canic M. Evaluation of c-Myc and Phosphorylated Glucocorticoid Receptor (p-GR) for Predicting Diabetic Foot Ulcer Healing-A Diabetic Foot Consortium Study. Wound Repair Regen. 2025 May-Jun;33(3):e70044. PubMed Central PMCID: PMC12138851.

3. Jozic I, Vukelic S, Stojadinovic O, Liang L, Ramirez HA, Pastar I, Tomic Canic M. Stress Signals, Mediated by Membranous Glucocorticoid Receptor, Activate PLC/PKC/GSK-3β/β-catenin Pathway to Inhibit Wound Closure. J Invest Dermatol. 2017 May;137(5):1144-1154. PubMed Central PMCID: PMC7540219.
4. Seth N, Abujamra BA, Boulina M, Lev-Tov H, Jozic I. Upregulation of Caveolae-Associated Proteins in Lesional Samples of Hidradenitis Suppurativa: A Case Series Study. JID Innov. 2023 Nov;3(6):100223. PubMed Central PMCID: PMC10507649.

Certification:

I certify that the information provided is current, accurate, and complete. This includes but is not limited to information related to domestic and foreign appointments and positions.

I also certify that, at the time of submission, I am not a party to a malign foreign talent recruitment program.

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: Jozic, Ivan

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Position Title: Research Associate Professor, Dr. Phillip Frost Department of Dermatology and Cutaneous Surgery

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Personal Statement

I am a Research Assistant Professor in the Dr. Phillip Frost Department of Dermatology & Cutaneous Surgery at the University of Miami Miller School of Medicine with a research program focused on defining molecular and cellular mechanisms that regulate acute and chronic wound healing. My work integrates multidisciplinary “omics” approaches, including transcriptomics, lipidomics, proteomics, and spatial omics, to interrogate skin physiology and pathophysiology, with particular emphasis on the role of caveolae and membrane microdomains in wound repair, inflammation, autoimmunity, fibrosis, and scarring disorders. I am the founding Director of the Dermatopathology Spatial Omics Core at the University of Miami, where my group provides spatial transcriptomic analysis of human skin biopsies using the Visium platform, and I have extensive expertise in ex vivo human skin models for functional evaluation of cutaneous wound healing. I have an established and highly productive collaboration with Dr. Samandari, with whom I have generated robust preliminary data supporting the feasibility and translational relevance of the proposed studies in wound healing. I will lend my expertise to Dr. Samandari and I look forward to continuing our collaborative efforts.

Honors

2025	Basic Science Research Award, University of Miami
2023	Junior Faculty Development Award, American Diabetes Association
2022	H. Paul Ehrlich Rising Star Award, Wound Healing Foundation
2021	Research Scholar Award, American Skin Association
2020	Research Award, Stanley J. Glaser Foundation
2019	Junior Faculty Award, Wound Healing Society
2019	Medline Research Innovation Award, Wound Healing Foundation
2018	Young Investigator Award Winner, Wound Healing Society
2018	Basic Science Research Award, Brian V. Jegasothy Memorial Basic Research Endowment
2018	Poster Award Winner, Mancunian Skin Club
2017	Animal Model Award Winner, Cyagen, Inc.
2017	Best Oral Presentation Winner, Innovations in Wound Healing
2015	Best Poster Award, Innovations in Wound Healing
2010 - 2012	MAC Travel Award, American Society for Cell Biology

Contributions to Science

1.
    1. Role of glucocorticoid receptor signaling in wound healing. During my postdoctoral training, I defined a membrane-initiated glucocorticoid receptor (GR) signaling program that mechanistically links stress biology and glucocorticoid exposure to impaired keratinocyte migration and delayed re-epithelialization through activation of downstream pathways that increase nuclear c-Myc. Building on this mechanistic foundation, our group advanced these pathway readouts toward clinical translation by participating in a multicenter observational cohort study conducted by the NIDDK Diabetic Foot Consortium to evaluate whether two objective tissue biomarkers (c-Myc and phosphorylated GR [p-GR]), measured in baseline DFU wound tissue could predict complete healing at week 12. In this DFC study, wound tissue collected at the initial visit was analyzed by immunohistochemistry with quantification of nuclear c-Myc and p-GR in full-thickness epidermis, and biomarker measurements were correlated with week-12 clinical outcomes using primary analyses that included AUC-based assessment of predictive performance. Even where prediction performance required further refinement, this work operationalized a mechanistic GR/c-Myc hypothesis into a rigorous, multicenter biomarker evaluation framework and helped establish a well-characterized resource linking

wound biomaterials to longitudinal outcomes.

2. Role of lipid rafts/caveolae in wound epithelialization and therapeutic targeting. As I transitioned into my faculty role at UM, my research program centered around understanding the role of lipid rafts (specifically caveolae). My group demonstrated that lipid raft/caveolae signaling, which centered on caveolin-1 (Cav1), acts as a spatiotemporal regulator of keratinocyte migration and wound closure. Using human chronic wound tissue and complementary preclinical models, we demonstrated that Cav1-dependent membrane microdomains function as an inhibitory platform that suppresses EGFR signaling and epithelialization, and that physiological downregulation of caveolae is required for normal wound repair. Importantly, disruption of caveolae (either genetically or pharmacologically through cholesterol modulation) restores pro-migratory signaling and accelerates wound closure across multiple models. Building on this mechanistic foundation, our group translated caveolae-targeted biology into a topical, wound-localized therapeutic strategy. In a recent collaboration with Drs. Marjanovic and Tomic-Canic, we developed an extracellular matrix–mimetic hydrogel platform incorporating cyclodextrins to locally sequester cholesterol, thereby disrupting lipid raft–mediated receptor sequestration and restoring growth factor responsiveness. This approach significantly enhanced re-epithelialization and angiogenesis in both human ex vivo wound models and diabetic mouse wounds. Importantly, the intellectual property space surrounding this technology is protected. We have filed a patent application covering the sustained-release statin hydrogel platform for caveolae-targeted wound repair (PCT US2023/020173; U.S. Application 18/861,068), which has been published as US-2025-0367164-A1.

3. Proteomic and lipidomic profiling of wound healing. I advanced proteomic and lipidomic profiling strategies to capture molecular heterogeneity that drives variable healing trajectories. Using multimodal mass spectrometry imaging in human-relevant systems, we mapped dynamic lipid remodeling during epithelialization, including spatial depletion of cholesterol sulfate in neoepithelium, linking membrane composition to repair biology. Complementing spatial lipidomics, I helped drive scalable proteomic workflows for non-invasive biomarker discovery using routinely discarded wound dressings, enabling practical molecular stratification and longitudinal monitoring of healing vs. non-healing outcomes, and to this end I serve as the inaugural director of the spatial omics dermatopathology core at UM Dermatology department.

4. Lipid rafts/caveolae as unifying regulators of cutaneous biology. Building from the wound-healing membrane microdomain framework, I extended lipid raft/caveolae biology to broader cutaneous pathophysiology where epithelial behavior, inflammation, and tissue remodeling converge. In inflammatory skin disease, my group helped demonstrate caveolae-associated protein dysregulation in psoriasis, supporting caveolar components as disease-relevant biomarkers and potential therapeutic targets. In hidradenitis suppurativa, our work identified increased caveolae-associated proteins in lesional tissue and hair follicle compartments, highlighting membrane microdomains as candidate mechanistic hubs in chronic follicular inflammation. In hair disorders, we reported upregulation/localization of caveolae-associated structural proteins in scarring alopecias (lichen planopilaris and frontal fibrosing alopecia), supporting caveolar pathways as tractable targets linked to follicular stem cell niche biology and fibrotic remodeling.

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