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

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Singh, Soudamani

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0001-6689-9942

Position Title: Associate Professor

Organization and Location: Marshall University, Huntington, WV, USA

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
Marshall University, Huntington, West Virginia, United States	Postdoctoral Fellow	07/2013	07/2017	Gastroenterology
West Virginia University, Morgantown, West Virginia, United States	Postdoctoral Fellow	07/2010	06/2013	Gastroenterology
University of Madras, Chennai, Tamil Nadu, India	Doctor of Philosophy (PHD)	10/2002	07/2008	Endocrinology
University of Madras, Chennai, Tamil Nadu, India	Master of Philosophy (MPHIL)	06/1999	05/2000	Zoology
University of Madras, Chennai, Chennai, India	Master of Science (MS)	06/1997	05/1999	Zoology

Appointments and Positions

2024 - present Associate Professor, Marshall University, Huntington, WV, USA
2026 - present Member, International Society for Extracellular Vesicles, Milwaukee, WI, USA
2025 - present Member, American Society for Intercellular Communication, Manassas, VA, USA
2018 - 2024 Assistant Professor, Marshall University, Huntington, WV, USA
2017 - present Member, American Gastroenterological Association , Bethesda, MD, USA
2017 - 2018 Research Assictant Professor, Marshall University, Huntington , WV, USA
2006 - present Member, Society for Reproductive Biology and Comparative Endocrinology , Chennai, Tamil Nadu, India

Products

Products Closely Related to the Proposed Project

1. Wellington VNA, Singh S. The Adipose Tissue-Derived Secretome (ADS) in Obesity Uniquely Regulates the Na-Glucose Transporter SGLT1 in Intestinal Epithelial Cells. Cells. 2025 Aug 12;14(16) PubMed Central PMCID: PMC12384572.
2. Thompson C, Rahman MM, Singh S, Arthur S, Sierra-Bakhshi C, Russell R, Denning K, Sundaram U, Salisbury T. The Adipose Tissue-Derived Secretome (ADS) in Obesity Uniquely Induces L-Type Amino Acid Transporter 1 (LAT1) and mTOR Signaling in Estrogen-Receptor-Positive Breast Cancer Cells. Int J Mol Sci. 2021 Jun 23;22(13) PubMed Central PMCID: PMC8268498.
3. Palaniappan B, Arthur S, Sundaram VL, Butts M, Sundaram S, Mani K, Singh S, Nepal N, Sundaram U. Inhibition of intestinal villus cell Na/K-ATPase mediates altered glucose and NaCl absorption in obesity-associated diabetes and hypertension. FASEB J. 2019 Aug;33(8):9323-9333. PubMed Central PMCID: PMC6662973.

Other Significant Products Highlighting Contributions to Science

1. Selvaraj R, Kanakarajan S, Singh S. Role of MicroRNAs in Intestinal Epithelial Transport and Its Mechanism Leading to Colorectal Cancer. Advances in Biochemistry in Health and Disease [Internet] Cham: Springer Nature Switzerland; 2026. Chapter Chapter 19417-449p. Available from: https://link.springer.com/10.1007/978-3-032-08824-6_19 DOI: 10.1007/978-3-032-08824-6_19

2. Butts M, Singh S, Haynes J, Arthur S, Sundaram U. Moderate Alcohol Consumption Uniquely Regulates Sodium-Dependent Glucose Co-Transport in Rat Intestinal Epithelial Cells In Vitro and In Vivo. J Nutr. 2020 Apr 1;150(4):747-755. PubMed Central PMCID: PMC7138678.
3. Manoharan P, Sundaram S, Singh S, Sundaram U. Inducible Nitric Oxide Regulates Brush Border Membrane Na-Glucose Co-transport, but Not Na:H Exchange via p38 MAP Kinase in Intestinal Epithelial Cells. Cells. 2018 Aug 19;7(8) PubMed Central PMCID: PMC6115905.
4. Butts M, Sundaram VL, Murughiyan U, Borthakur A, Singh S. The Influence of Alcohol Consumption on Intestinal Nutrient Absorption: A Comprehensive Review. Nutrients. 2023 Mar 24;15(7) PubMed Central PMCID: PMC10096942.
5. Manoharan P, Gayam S, Arthur S, Palaniappan B, Singh S, Dick GM, Sundaram U. Chronic and selective inhibition of basolateral membrane Na-K-ATPase uniquely regulates brush border membrane Na absorption in intestinal epithelial cells. Am J Physiol Cell Physiol. 2015 Apr 15;308(8):C650-6. PubMed Central PMCID: PMC4398852.

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: Singh, Soudamani

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0001-6689-9942

Position Title: Associate Professor

Organization and Location: Marshall University, Huntington, WV, USA

Personal Statement

I am a tenured Associate Professor in the Department of Biomedical Sciences at the Joan C. Edwards School of Medicine, Marshall University, and I have the scientific expertise, leadership, and mentorship experience required to successfully execute this SuRE-First project. My laboratory investigates the cellular and molecular mechanisms of regulation of intestinal epithelial nutrient transport in both normal physiology and pathophysiology, with a specific focus on obesity and environmental disruptors like microplastics. My long-term goal is to advance exosome bioengineering to address transporter changes in obesity, potentially leading to innovative therapeutic strategies for obesity-related diabetes. This grant focuses on exploring molecular mechanisms underlying altered SGLT1 phosphorylation in both in vitro and in vivo systems. This award will provide undergraduate and master's students at Marshall University with high-quality research and hands-on training to uncover the molecular mechanisms behind altered glucose absorption. My lab has shown that adipose-derived exosomes can regulate intestinal epithelial transporters. Adipose-derived exosomes from obese Zucker rats, but not lean Zucker rats, increased intestinal glucose absorption by altering SGLT1 phosphorylation. My extensive studies on the role of intestinal epithelial transporters in the pathogenesis of IBD and obesity have resulted in 20 peer-reviewed publications in high-impact journals. I have experience in mentoring undergraduates, graduate students, and medical students. My students have earned the best poster award, best oral presentation, and poster of distinction. For this SuRE-First grant, I have the skills and experience to successfully mentor undergraduate and graduate students at Marshall University in the exosome-mediated mechanism of intestinal nutrient absorption in obesity.

Honors

2022	Poster of Distinction, Digestive Disease Week , American Gastroenterological Association (AGA)
2021	Poster of Distinction, Digestive Disease Week , American Gastroenterological Association (AGA)
2020	Excellence in Diversity & Inclusion Award , Marshall University
2018	Poster of Distinction, Digestive Disease Week, American Gastroenterological Association (AGA)
2016	Young Investigator Award , American Gastroenterological Association (AGA)
2015	Poster of Distinction, Digestive Disease Week, American Gastroenterological Association (AGA)
2007	Senior Research Fellow , Indian Council of Medical Research
2007	Women Scientists Award , Department of Science and Techonology

Contributions to Science

1. Deregulation of glucose homeostasis during obesity: Recently, our study investigated how adipose-derived secretome (ADS) from obese Zucker rats (OZR) regulate SGLT1 in intestinal epithelial cells. We found that OZR-ADS stimulates SGLT1, increasing its glucose affinity without altering protein levels. This effect is due to increased SGLT1 phosphorylation. In contrast, ADS from lean Zucker rats (LZR) showed no such effect. Our results suggest that during obesity, ADS enhances SGLT1, contributing to glucose homeostasis dysregulation and potentially Type 2 diabetes. In FASEB, we have shown that during obesity, SGLT1 and DRA are stimulated, whereas NHE3 is unaffected. The mechanism of stimulation of SGLT1 was due to increased affinity (Km) while the mechanism of stimulation of DRA was increased transporter number (Vmax). These observations provide the pathophysiologic basis for obesity-mediated dysregulation of glucose and NaCl homeostasis, an underlying cause of diabetes and hypertension.

2. A moderate dose of alcohol differentially regulates intestinal nutrient transporters: In another collaborative study, we demonstrated that moderate alcohol consumption inhibited both SGLT1 and B0AT1 in intestinal epithelial cells, both in vitro and in vivo in Sprague-Dawley rats. However, the mechanisms of inhibition of these Na-dependent co-transport processes differed: B0AT1 was reduced because of a decrease in the number of co-transporters (Vmax), with no change in affinity

(1/Km). SGLT1 activity decreased because of reduced affinity (1/Km) for its substrate, with no change in the number of co-transporters (Vmax). We have now extended these studies to adipose tissue-derived exosomes in alcohol-treated SD rats.

3. Inimitable regulation of SGLT1 during inflammation in intestinal epithelial cells. In the journal Cells, we showed that during chronic enteritis, peroxynitrite (OONO), a potent oxidant, inhibits SGLT1 while NHE3 activity remains unaltered. OONO inhibition of SGLT1 was secondary to a reduction in the number of co-transporters (Vmax), without an alteration in affinity. We further demonstrated that the p38 mitogen-activated protein (MAP) kinase pathway mediates OONO inhibition of SGLT1. In another study, we showed that chronic, specific inhibition of basolateral NKA in IEC-18 cells increases intracellular Na+. In this study, we showed that NKA stimulates glucose transport via brush border membrane SGLT1, but not Na+ influx via NHE3. The mechanism of SGLT1 stimulation is secondary to an increase in the co-transporter's affinity for glucose. NKA regulates brush border membrane SGLT1, possibly as a compensatory response to the loss of trans-membrane Na+, leading to its inhibition.

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