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

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: SARKAR, DEVANAND

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-0973-2323

Position Title: Professor with tenure

Organization and Location: Department of Cellular, Molecular and Genetic Medicine, Virginia Commonwealth University, Richmond, VA, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
Nagoya University, Nagoya, Not Applicable, N/A, Japan	DOCTOR OF PHILOSOPHY	04/1996	03/2001	Endocrinology and Metabolism
Dhaka Medical College, Dhaka, Not Applicable, N/A, Bangladesh	FOREIGN - BACHELOR OF MEDICINE AND SURGERY	07/1987	09/1994	Medicine

Appointments and Positions

2018 - present	Professor with tenure, Department of Cellular, Molecular and Genetic Medicine, Virginia Commonwealth University, Richmond, VA, United States
2016 - present	Associate Director for Cancer Research Training and Education Coordination (CRTEC), Massey Comprehensive Cancer Center, Virginia Commonwealth University, Richmond, VA, USA
2013 - 2018	Associate Professor with Tenure, Department of Human and Molecular Genetics, Virginia Commonwealth University, Richmond, VA, USA
2008 - 2013	Assistant Professor, Department of Human and Molecular Genetics, Virginia Commonwealth University, Richmond, VA, USA
2006 - 2007	Adjunct Assistant Professor, New York Institute of Technology, New York, NY, USA
2003 - 2007	Associate Research Scientist, Departments of Pathology and Urology, Columbia University, New York, NY, USA

Products

Products Closely Related to the Proposed Project

1. Reghupaty SC, Raha S, Mendoza R, Manna D, Gawi Ermi A, Davis E, Aqeel Y, Subler MA, Koblinski J, Idowu M, Mukhopadhyay N, Lai Z, Fisher PB, Windle JJ, Dozmorov MG, Sarkar D. TATA-box binding protein-associated factor 2 (TAF2) in hepatocyte survival and tumorigenesis. Hepatology. 2026 Mar 1;83(3):497-512. PubMed Central PMCID: PMC12310156.
2. Manna D, Reghupaty SC, Camarena MDC, Mendoza RG, Subler MA, Koblinski JE, Martin R, Dozmorov MG, Mukhopadhyay ND, Liu J, Qu X, Das SK, Lai Z, Windle JJ, Fisher PB, Sarkar D. Melanoma differentiation associated gene-9/syndecan binding protein promotes hepatocellular carcinoma. Hepatology. 2023 Dec 1;78(6):1727-1741. PubMed Central PMCID: PMC11261751.
3. Robertson CL, Mendoza RG, Jariwala N, Dozmorov M, Mukhopadhyay ND, Subler MA, Windle JJ, Lai Z, Fisher PB, Ghosh S, Sarkar D. Astrocyte Elevated Gene-1 Regulates Macrophage Activation in Hepatocellular Carcinogenesis. Cancer Res. 2018 Nov 15;78(22):6436-6446. PubMed Central PMCID: PMC6239947.
4. Srivastava J, Robertson CL, Ebeid K, Dozmorov M, Rajasekaran D, Mendoza R, Siddiq A, Akiel MA, Jariwala N, Shen XN, Windle JJ, Subler MA, Mukhopadhyay ND, Giashuddin S, Ghosh S, Lai Z, Chen Y, Fisher PB, Salem AK, Sanyal AJ, Sarkar D. A novel role of astrocyte elevated gene-1 (AEG-1) in regulating nonalcoholic steatohepatitis (NASH). Hepatology. 2017 Aug;66(2):466-480. PubMed Central PMCID: PMC5519412.
5. Srivastava J, Siddiq A, Gredler R, Shen XN, Rajasekaran D, Robertson CL, Subler MA, Windle JJ, Dumur CI, Mukhopadhyay ND, Garcia D, Lai Z, Chen Y, Balaji U, Fisher PB, Sarkar D. Astrocyte elevated gene-1 and c-Myc cooperate to promote hepatocarcinogenesis in mice. Hepatology. 2015 Mar;61(3):915-29. PubMed Central PMCID: PMC4309751.

Other Significant Products Highlighting Contributions to Science

1. Saverino A, Qu X, Mendoza RG, Raha S, Manna D, Ermi AG, Subler MA, Windle JJ, Liu J, Sarkar D. Spatial transcriptomics unravels palmitoylation and zonation-dependent gene regulation by AEG-1 in mouse liver. J Biol Chem. 2024 Jun;300(6):107322. PubMed Central PMCID: PMC11134871.
2. Rajesh Y, Reghupaty SC, Mendoza RG, Manna D, Banerjee I, Subler MA, Weldon K, Lai Z, Giashuddin S, Fisher PB, Sanyal AJ, Martin RK, Dozmorov MG, Windle JJ, Sarkar D. Dissecting the Balance Between Metabolic and Oncogenic Functions of Astrocyte-Elevated Gene-1/Metadherin. Hepatol Commun. 2022 Mar;6(3):561-575. PubMed Central PMCID: PMC8870024.
3. Jariwala N, Rajasekaran D, Mendoza RG, Shen XN, Siddiq A, Akiel MA, Robertson CL, Subler MA, Windle JJ, Fisher PB, Sanyal AJ, Sarkar D. Oncogenic Role of SND1 in Development and Progression of Hepatocellular Carcinoma. Cancer Res. 2017 Jun 15;77(12):3306-3316. PubMed Central PMCID: PMC5488274.
4. Jariwala N, Mendoza RG, Garcia D, Lai Z, Subler MA, Windle JJ, Mukhopadhyay ND, Fisher PB, Chen Y, Sarkar D. Posttranscriptional Inhibition of Protein Tyrosine Phosphatase Nonreceptor Type 23 by Staphylococcal Nuclease and Tudor Domain Containing 1: Implications for Hepatocellular Carcinoma. Hepatol Commun. 2019 Sep;3(9):1258-1270. PubMed Central PMCID: PMC6719750.
5. Raha S, Reghupaty SC, Qu X, Mendoza RG, Farrar D, Ermi AG, Davis E, Martin RK, Subler MA, Fisher PB, Windle JJ, Liu J, Sarkar D. Myeloid AEG-1/MTDH drives inflammation and hepatocellular dysfunction in diet-induced steatohepatitis. J Biol Chem. 2026 Feb;302(2):111101. PubMed Central PMCID: PMC12818226.

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.

Certified by SARKAR, DEVANAND in SciENcv on 2026-05-01 10:30:52

NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: SARKAR, DEVANAND

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-0973-2323

Position Title: Professor with tenure

Organization and Location: Department of Cellular, Molecular and Genetic Medicine, Virginia Commonwealth University, Richmond, VA, United States

Personal Statement

My research aims at understanding the molecular mechanism of metabolic dysfunction-associated steatohepatitis (MASH) and hepatocellular carcinoma (HCC) and developing novel, targeted and effective therapeutic strategies, based on the derived knowledge. Amplification of chromosome 8q is a key event in hepatocarcinogenesis and the laboratory studies genes at this location, namely AEG-1/MTDH, MDA-9/SDCBP, TAF2 and MYC, analyzing their oncogenic properties either alone or in cooperation using complex mouse models. We also study the oncogene SND1, which interact and cooperate with AEG-1, and the tumor suppressor IGFBP7, which promotes tumor antigen presentation. These mechanistic studies pave the way for evaluating diverse therapeutic strategies, such as targeted nanoparticle-delivered siRNAs for oncogenes, small molecule inhibitors (such as MDA-9 inhibitor PDZ1i or MYC inhibitor MYCi975) and immunomodulators using endogenous mouse models of HCC. I have been studying HCC for 18 years and have the necessary expertise and experience to serve as a co-investigator in this grant.

Ongoing projects that I would like to highlight include:

NIH NCI: P01CA275740 Sarkar 08/08/2024–07/31/2029
ExpLoiting thErapeutic VulnerAbilities in hepaTocEllular carcinoma (ELEVATE)
Goal: This program project grant aims to evaluate multiple combinatorial treatment strategies for HCC associated with metabolic dysfunction-associated steatohepatitis (MASH)

NIH NCATS: T32TR004362 Sarkar, Mosavel, Neigh 12/01/2023–11/30/2028
CTSA Predoctoral T32 at Virginia Commonwealth University
Goal: This is a training grant for predoctoral students in clinical and translational science

NIH NCI: U54CA283762 Trevino, Harrell 07/19/2023–06/30/2028
United for Health Equity – Living PDX Program (U4HELPP)
Goal: To generate Patient-derived xenografts from diverse cancers
Role: Co-investigator

Honors

2025	Scientist of the Year Award, Massey Comprehensive Cancer Center, VCU
2024	National/International Recognition Award (NIRA), Virginia Commonwealth University
2019	Inducted to Member, National Academy of Inventors, Virginia Commonwealth University Chapter
2015	Harrison Foundation Distinguished Professor in Cancer Research, Virginia Commonwealth University Massey Comprehensive Cancer Center
2011	Rosemary Holden Kelso Fellowship to Participate in the Harris-Manchester College Summer Research Institute, Oxford University, UK
2010 - 2013	Blick Scholar Award, Virginia Commonwealth University School of Medicine
2008 - 2015	Harrison Endowed Scholar in Cancer Research, Virginia Commonwealth University Massey Comprehensive Cancer Center
2005	Milstein Young Investigator Award, The International Society for Interferon and Cytokine Research
2003 - 2006	Postdoctoral Fellowship Award, DAMD17-03-1-0290, Department of Defense
2001 - 2002	Postdoctoral Fellowship Award, DAMD17-98-1-8053 , Department of Defense
2000	Quest Diagnostics Young Investigator Award, The Endocrine Society

Contributions to Science

1. Analysis of a novel role of AEG-1 in regulating retinoid x receptor (RXR) function: RXR is a transcription factor that heterodimerizes with a wide variety of nuclear receptors mediating effects of vitamins, hormones, and lipids. We documented that AEG-1 interacts with RXR and prevents RXR-mediated functions of other nuclear receptors. In AEG-1 KO mice there is hyperactivation of RXR partners LXR and PPARα in the intestines, resulting in inhibition of intestinal lipid absorption. AEG-1 KO mice are, thus, lean and live significantly longer. These findings identify a novel role of AEG-1 in lipid metabolism and pave the way for studies on AEG-1 regulating obesity-associated cancers. We have shown that in Alb/AEG-1 mice overexpressed AEG-1 blocks thyroid hormone function thereby inducing peripheral hypothyroidism, a condition known as non-thyroidal illness syndrome (NTIS), often associated with cancers including HCC. AEG-1 overexpression blocks RXR-RAR function and, thus, retinoic acid-mediated killing. We have generated hepatocyte-specific nanoparticles to deliver AEG-1 siRNA and demonstrated that this approach in combination with all-trans-retinoic acid (ATRA) synergistically inhibits orthotopic human HCC xenografts in nude mice. ATRA is an approved anticancer drug, and the liver is an ideal organ for RNAi therapy since IV administration of any agent will go to the liver first. Thus, identification of AEG-1/RXR interaction resulted in unraveling of novel roles of AEG-1, not only as an oncogene but as an important regulator of lipid metabolism and non-alcoholic steatohepatitis (NASH), and resulted in the development of a targeted therapeutic approach that might have high translational potential for HCC patients.

2. Identification and analysis of non-8q genes regulating HCC: While studying AEG-1 function we identified several genes, which do not reside in chromosome 8q, to play a seminal role in regulating HCC. The transcription factor Late SV40 Factor (LSF)/TFCP2 is induced by AEG-1. Prior to our studies, a role of LSF in cancer had not been documented. We demonstrated that LSF is overexpressed in a high percentage of HCC patients and plays an important role in proliferation, chemoresistance, and metastasis by transcriptionally regulating osteopontin and MMP-9 and activating oncogenic c-Met signaling. We have evaluated the therapeutic efficacy of small molecule inhibitors of LSF using a transgenic mouse model of HCC, thus paving the way for potential clinical trials using these inhibitors. On the other hand, the secreted protein Insulin-like growth factor binding protein-7 (IGFBP7) was found to be downregulated by AEG-1. We generated an Igfbp7 knockout mouse model and demonstrated its tumor suppressor function by inhibiting antigen presentation. This study identified for the first time an immunomodulatory function of IGFBP7, paving the way for its potential therapeutic use in combination with immune checkpoint inhibitors.

3. Identification of Staphylococcal nuclease and tudor domain containing-1 (SND1) as an AEG1-interacting protein and as an oncogene for HCC: My laboratory identified SND1 as an AEG-1 interacting protein and documented their role in RNA-induced silencing complex (RISC) contributing to HCC. We have shown that SND1 activates NF-κB, TGFβ and Akt signaling to promote angiogenesis, epithelial-mesenchymal transition (EMT), and cell cycle progression. Using a hepatocyte-specific transgenic mouse expressing SND1 we documented that SND1 functions as a bona fide oncogene for HCC and established that inhibition of SND1 by a small-molecule inhibitor might be an effective anti-HCC strategy.

4. Analysis of oncogenic function of genes co-amplified with MYC: Amplification of chromosome 8q is observed in many cancers including HCC. The driver oncogene MYC is located at 8q24.21 and molecular classification of HCC has identified a subclass of MYC-driven HCC with poor prognosis. We have been studying several genes in 8q which are co-amplified with MYC, such as Astrocyte elevated gene-1/metadherin (AEG-1/MTDH), melanoma differentiation associated gene-9/syndecan binding protein (SDCBP) and TATA box binding protein associated factor 2 (TAF2). I have been working with AEG-1 and MTDH since their initial cloning and my laboratory is the first to demonstrate an oncogenic role of TAF2 in HCC. We have demonstrated the oncogenic function of these genes either alone or in combination with MYC, using a variety of mouse models, and elucidated the underlying molecular mechanism.

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