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

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Shiozawa, Yusuke

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0001-9814-9230

Position Title: Associate Professor

Organization and Location: Wake Forest University Health Sciences, Winston-Salem, NC, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
University of Michigan, Ann Arbor, MI, USA	Postdoctoral Fellow	07/2006	09/2010	Molecular Biology
Juntendo University School of Medicine, Tokyo, Not Applicable, N/A, Japan	DOCTOR OF PHILOSOPHY	04/2002	03/2006	Pediatrics
Juntendo University School of Medicine, Tokyo, Not Applicable, N/A, Japan	DOCTOR OF MEDICINE	04/1994	05/2000	Medicine
Wake Forest University School of Medicine, Winston-Salem, NC, USA	Other training		08/2017	Faculty Early Career Development

Appointments and Positions

2021 - present	Associate Professor, Wake Forest University Health Sciences, Winston-Salem, NC, United States
2026 - present	Director of the Cancer Biology PhD Program, Wake Forest University School of Medicine, Winston-Salem, NC, United States
2015 - 2021	Assistant Professor, Wake Forest University School of Medicine, Winston-Salem, NC, USA
2014 - 2015	Research Assistant Scientist, University of Michigan Dental School, Ann Arbor, MI, USA
2010 - 2014	Research Investigator, University of Michigan Dental School, Ann Arbor, MI, USA
2006 - 2010	Postdoctoral Research Fellow , University of Michigan Dental School, Ann Arbor, MI, USA
2004 - 2006	Research Fellow, National Research Institute for Child Health and Development, Tokyo, Not Applicable, N/A, Japan
2002 - 2006	Staff Doctor, Junstendo University School of Medicine, Tokyo, Not Applicable, N/A, Japan
2000 - 2002	Resident, Juntendo University School of Medicine, Tokyo, Not Applicable, N/A, Japan

Products

Products Closely Related to the Proposed Project

1. Contino KF, Ollodart J, Yu Y, Park SH, Tsuzuki S, Rollins K, Heethouse TM, Chu J, Steele LR, Kimura T, Lee J, Furdui CM, Miller LD, Hsu FC, Shiozawa Y. The contribution of stem cell factor and its receptor c-Kit to cancer-induced bone pain. JCI Insight. 2026 Jun 22;11(12) PubMed Central PMCID: PMC13313531.
2. Park SH, Tsuzuki S, Contino KF, Ollodart J, Eber MR, Yu Y, Steele LR, Inaba H, Kamata Y, Kimura T, Coleman I, Nelson PS, Muñoz-Islas E, Jiménez-Andrade JM, Martin TJ, Mackenzie KD, Stratton JR, Hsu FC, Peters CM, Shiozawa Y. Crosstalk between bone metastatic cancer cells and sensory nerves in bone metastatic progression. Life Sci Alliance. 2024 Dec;7(12) PubMed Central PMCID: PMC11393574.
3. Jimenez-Andrade JM, Ramírez-Rosas MB, Hee Park S, Parker R, Eber MR, Cain R, Newland M, Hsu FC, Kittel CA, Martin TJ, Muñoz-Islas E, Shiozawa Y, Peters CM. Evaluation of pain related behaviors and disease related outcomes in an immunocompetent mouse model of prostate cancer induced bone pain. J Bone Oncol. 2023 Dec;43:100510. PubMed Central PMCID: PMC10701434.
4. Park SH, Eber MR, Fonseca MM, Patel CM, Cunnane KA, Ding H, Hsu FC, Peters CM, Ko MC, Strowd RE, Wilson JA, Hsu W, Romero-Sandoval EA, Shiozawa Y. Usefulness of the measurement of neurite outgrowth of primary sensory neurons to study cancer-related painful complications. Biochem Pharmacol. 2021 Jun;188:114520. PubMed Central PMCID:

PMC8154668.

5. Park SH, Eber MR, Tsuzuki S, Booker ME, Sunil AG, Widner DB, Parker RA, Peters CM, Shiozawa Y. Adeno-associated virus serotype rh10 is a useful gene transfer vector for sensory nerves that innervate bone in immunodeficient mice. Sci Rep. 2017 Dec 12;7(1):17428. PubMed Central PMCID: PMC5727257.

Other Significant Products Highlighting Contributions to Science

1. Fonseca MM, Gelblung O, Pennypacker SD, Brooks T, Limia M, Morgan JW, Zhu X, Tovias-Sanchez LC, Pluma-Pluma A, Martinez RE, Eber MR, Park SH, Furdui CM, Awasthi D, Emmanuelli A, Cho BA, Tan C, Shalowitz DI, Lentz SS, Kelly M, O'Brien Cox A, Macnamara L, Hsu FC, Shiozawa Y, Hsu W, Iwawaki T, Miller LD, Lesser GJ, Strowd R, Cubillos-Ruiz JR, Romero-Sandoval EA. Leukocyte-intrinsic ER stress responses contribute to chemotherapy-induced peripheral neuropathy. Sci Transl Med. 2025 Oct 29;17(822):eady5288. PubMed Central PMCID: PMC13215384.
2. Eber MR, Park SH, Contino KF, Patel CM, Hsu FC, Shiozawa Y. Osteoblasts derived from mouse mandible enhance tumor growth of prostate cancer more than osteoblasts derived from long bone. J Bone Oncol. 2021 Feb;26:100346. PubMed Central PMCID: PMC7779864.
3. Shiozawa Y, Pedersen EA, Havens AM, Jung Y, Mishra A, Joseph J, Kim JK, Patel LR, Ying C, Ziegler AM, Pienta MJ, Song J, Wang J, Loberg RD, Krebsbach PH, Pienta KJ, Taichman RS. Human prostate cancer metastases target the hematopoietic stem cell niche to establish footholds in mouse bone marrow. J Clin Invest. 2011 Apr;121(4):1298-312. PubMed Central PMCID: PMC3069764.
4. Ollodart J, Contino KF, Steele LR, Yu Y, Shiozawa Y. Radiographic Assessment of Osteosclerotic Lesions in Mice with Bone Metastasis. Methods Mol Biol. 2025;2885:423-432. PubMed PMID: 40448774.
5. Eber MR, Jiménez-Andrade JM, Peters CM, Shiozawa Y. A Method of Bone-Metastatic Tumor Progression Assessment in Mice Using Longitudinal Radiography. Methods Mol Biol. 2022;2413:1-6. PubMed Central PMCID: PMC9082521.

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: Shiozawa, Yusuke

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0001-9814-9230

Position Title: Associate Professor

Organization and Location: Wake Forest University Health Sciences, Winston-Salem, NC, United States

Personal Statement

My ultimate career goal is to become an independent translational researcher and leader in cancer bone metastasis, bone pain, and treatment-related complications. I aim to define how cancer, including bone-metastatic cancer, interacts with sensory nerves in bone, with the long-term goal of developing new treatments that reduce cancer-related mortality and improve quality of life for patients with advanced cancer. With my clinical and research background, I believe I am well positioned to achieve this goal with the support of this award.

I have substantial experience studying interactions between the bone marrow microenvironment and metastatic disease. My medical career began in Pediatric Hematology and Oncology, where I cared for patients with recurrent leukemia and solid tumors involving bone. These experiences led me to focus on why tumors preferentially colonize the marrow. I pursued a Ph.D. and then joined the University of Michigan as a postdoctoral fellow, where I continued to study the hematopoietic niche. One of my key contributions was establishing a direct link between hematopoietic stem cell homing to the marrow and cancer bone metastasis. We found that bone-metastatic cancer targets the hematopoietic stem cell niche during dissemination to the bone marrow and directly competes with hematopoietic stem cells for niche occupancy. In 2015, I was recruited to the Department of Cancer Biology at Wake Forest School of Medicine as an Assistant Professor. As PI and Co-Investigator on several NIH- and DOD-funded grants, I have developed preliminary data, established assays and animal models to measure pain behavior, and built collaborations with basic scientists and clinicians that support the proposed work. My current research focuses on identifying factors that regulate bone metastasis and its painful complications, developing targeted therapies for bone metastasis, and defining the molecular mechanisms of cancer treatment-associated complications, including chemotherapy-induced peripheral neuropathy. To date, I have published 56 peer-reviewed papers and 28 invited review articles.

In addition to maintaining an active laboratory program, I am deeply committed to research training at the undergraduate, graduate, postdoctoral, professional student, and junior faculty levels. At the University of Michigan, I mentored more than 25 basic science and clinical research trainees, including eight postdoctoral fellows. At Wake Forest School of Medicine, I have mentored three postdoctoral fellows and 34 research students, including four medical students and two Ph.D. students, and I currently mentor two Ph.D. students. By inspiring and training the next generation of investigators, I hope to extend my impact on patient care beyond my own laboratory.

Honors

2025	The winner of the 2024-2025 Graduate Student Association Graduate Faculty Excellence Award, Wake Forest University School of Medicine
2023	The Research Excellence Award, Wake Forest University School of Medicine
2022	The Research Excellence Award, Wake Forest University School of Medicine
2021	The Research Excellence Award, Wake Forest University School of Medicine
2011	Prostate Cancer Foundation Young Investigator Award , Prostate Cancer Foundation
2007	Best Paper Award , Japan Cytometry Society

Contributions to Science

1. Clinical experience in bone marrow disease as a pediatric hematologist/oncologist.
   My medical career began as a pediatric hematologist/oncologist. While caring for cancer patients with bone metastases, I recognized the need to better understand how tumors co-opt the bone marrow. Since then, my research has focused on bone metastasis. Patients with bone metastases and their families face a tremendous physical and emotional burden. In 2006, I was

awarded a Pediatric Oncology Research Fellowship from the Children's Cancer Association of Japan, which allowed me to come to the United States to expand my research as a postdoctoral fellow.

2. Bone marrow osteoblastic niche supports hematopoiesis.
   Osteoblasts are thought to be a major component of the hematopoietic stem cell (HSC) niche in the bone marrow. We discovered that the bone marrow osteoblastic niche supports early hematopoiesis both in vitro and in vivo. These findings support the hypothesis that bone-metastatic prostate cancer (PCa) targets the osteoblastic HSC niche during dissemination.

3. Prostate cancer targets the hematopoietic stem cell niche to establish a foothold in bone marrow.
   One of our major contributions to the field of prostate cancer (PCa) bone metastasis is the discovery that disseminated PCa targets the hematopoietic stem cell (HSC) niche, which regulates HSC function. Our work showed that the osteoblastic HSC niche is critical for the homing and binding of both HSCs and PCa cells. Based on these findings, we provided direct evidence that PCa targets the HSC niche and competes with HSCs for niche occupancy.

4. The bone marrow niche regulates the phenotype of disseminated tumor cells.
   Our research supports the hypothesis that the hematopoietic stem cell niche contributes to the accumulation of tumor-initiating, or dormant, prostate cancer (PCa) cells in the marrow. This suggests that disseminated tumor cells (DTCs) may act as metastatic seeds. However, little is known about how disseminated PCa cells remain viable in bone and later progress to metastasis. Cancer stem cells and dormant tumor cells are thought to play a key role in this process.

5. Molecular crosstalk between bone-metastatic cancer and nociceptive neurons.
   We also investigate the mechanisms underlying cancer-induced bone pain and tumor outgrowth. Recent studies have shown that interactions between nerves and cancer cells promote cancer progression and metastasis, but how these interactions contribute to cancer-induced bone pain remains unclear. Our work defines how interactions between bone-metastatic cancer cells and bone marrow nerve cells contribute to both bone pain and the outgrowth of bone-metastatic cancer.

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