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

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Gibson, Heather

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-2652-8252

Position Title: Associate Professor

Organization and Location: Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States	Postdoctoral Fellow	05/2011	04/2016	Cancer Immunotherapy
Wayne State University, Detroit, Michigan, United States	Doctor of Philosophy (PHD)	08/2007	05/2011	Immunology and Microbiology
Michigan State University, East Lansing, Michigan, United States	Bachelor of Science (BS)	08/1999	05/2004	Biochemistry and Molecular Biology

Appointments and Positions

2018 - present	Associate Professor, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2025 - present	Associate Professor (tenured), Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2018 - 2025	Assistant Professor, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2016 - 2018	Research Scientist, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2014 - 2016	Research Associate, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2011 - 2014	Postdoctoral Fellow, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2007 - 2011	Graduate Research Assistant, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2007 - 2008	Graduate Teaching Assistant, Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States
2004 - 2007	Senior Research Assistant, Henry Ford Health System, Detroit, Michigan, United States
2001 - 2003	Undergraduate Research Assistant, Michigan State University, East Lansing, Michigan, United States

Products

Products Closely Related to the Proposed Project

1. Hackett JB, Glassbrook JE, Muñiz MC, Bross M, Fielder A, Dyson G, Movahhedin N, McCasland J, McCarthy-Leo C, Gibson HM. A diversity outbred F1 mouse model identifies host-intrinsic genetic regulators of response to immune checkpoint inhibitors. Oncoimmunology. 2022;11(1):2064958. PubMed Central PMCID: PMC9037414.
2. Hackett JB, Ramos N, Barr S, Bross M, Viola NT, Gibson HM. Interferon gamma immunoPET imaging to evaluate response to immune checkpoint inhibitors. Front Oncol. 2023;13:1285117. PubMed Central PMCID: PMC10735274.
3. Gregory M, Hackett JB, Hughes S, Muñiz MC, Ayoub S, Boerner A, Alrabaa H, Taylor L, Rivera B, Holland H, Dyson G, Daveluy S, Gibson H. Pharmacologic targeting of the dopamine D2 receptor impacts the efficacy of immune checkpoint blockade in melanoma. J Immunother Cancer. 2026 Mar 27;14(3) PubMed Central PMCID: PMC13034299.
4. Wallace-Povirk A, Rubinsak L, Malysa A, Dzinic SH, Ravindra M, Schneider M, Glassbrook J, O'Connor C, Hou Z, Kim S, Back J, Polin L, Morris RT, Gangjee A, Gibson H, Matherly LH. Targeted therapy of pyrrolo[2,3-d]pyrimidine antifolates in a syngeneic mouse model of high grade serous ovarian cancer and the impact on the tumor microenvironment. Sci Rep. 2022 Jul 5;12(1):11346. PubMed Central PMCID: PMC9256750.
5. Viola NT, Glassbrook JE, Kalluri JR, Hackett JB, Wicker MN, Sternberg J, Gibson HM. Evaluation of an ImmunoPET Tracer for IL-12 in a Preclinical Model of Inflammatory Immune Responses. Front Immunol. 2022;13:870110. PubMed Central PMCID: PMC9130849.

Other Significant Products Highlighting Contributions to Science

1. Gibson HM, McKnight BN, Malysa A, Dyson G, Wiesend WN, McCarthy CE, Reyes J, Wei WZ, Viola-Villegas NT. IFNγ PET Imaging as a Predictive Tool for Monitoring Response to Tumor Immunotherapy. Cancer Res. 2018 Oct 1;78(19):5706-5717. PubMed Central PMCID: PMC6443251.
2. Gibson HM, Mishra A, Chan DV, Hake TS, Porcu P, Wong HK. Impaired proteasome function activates GATA3 in T cells and upregulates CTLA-4: relevance for Sézary syndrome. J Invest Dermatol. 2013 Jan;133(1):249-57. PubMed Central PMCID: PMC4284066.
3. Gibson HM, Veenstra JJ, Jones R, Vaishampayan U, Sauerbrey M, Bepler G, Lum L, Reyes J, Weise A, Wei WZ. Induction of HER2 Immunity in Outbred Domestic Cats by DNA Electrovaccination. Cancer Immunol Res. 2015 Jul;3(7):777-86. PubMed Central PMCID: PMC4491035.
4. Gibson H, Munns S, Freytag S, Barton K, Veenstra J, Bettahi I, Bissonette J, Wei WZ. Immunotherapeutic intervention with oncolytic adenovirus in mouse mammary tumors. Oncoimmunology. 2015 Jan;4(1):e984523. PubMed Central PMCID: PMC4368120.
5. Trendowski MR, Watza D, Lusk CM, Lonardo F, Ratliff V, Wenzlaff AS, Mamdani H, Neslund-Dudas C, Boerner JL, Schwartz AG, Gibson HM. Evaluation of the Immune Response within the Tumor Microenvironment in African American and Non-Hispanic White Patients with Non-Small Cell Lung Cancer. Cancer Epidemiol Biomarkers Prev. 2024 Sep 3;33(9):1220-1228. PubMed Central PMCID: PMC11371519.

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 Gibson, Heather in SciENcv on 2026-08-25 11:09:21

NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: Gibson, Heather

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-2652-8252

Position Title: Associate Professor

Organization and Location: Wayne State University/Karmanos Cancer Institute, Detroit, Michigan, United States

Personal Statement

I completed my postdoctoral training and accepted promotion to Research Associate and Research Scientist in the lab of Dr. Wei-Zen Wei, a pioneer of HER2 DNA vaccination, which provided a solid foundation in cancer immunotherapy and animal model design. My studies included immune modulation with HER2 and TRAIL receptor DR5 vaccines, oncolytic virus therapy, and cyroablation, with subsequent intratumoral and peripheral anti-tumor immune evaluation for both humoral and cellular responses. I worked with genetically diverse feline and murine models for HER2 vaccine development, and have shown that genetic background plays a prominent role in maintenance of self-tolerance to cancer vaccination, which is supported by the variance in cancer immunotherapy response in the human population. It was through these studies that I realized my passion for both the discovery of host-intrinsic regulators of immunotherapy response and optimized methods to evaluate anti-tumor immunity in vivo to predict therapeutic outcomes. In July 2018 began my independent research program as an Assistant Professor with a focus on uncovering tumor immunotherapy resistance mechanisms. In collaboration with Dr. Nerissa Viola (WSU, Oncology Department) I developed novel antibody-based imaging tracers to the immune cytokines interferon gamma (IFN-γ) and interleukin 12 (IL12) to identify activated immune responses within the tumor microenvironment using positron emission tomography (PET). As a means of identifying host-intrinsic regulators of immunotherapy response, I have utilized tumor models in genetically heterogeneous Diversity Outbred (DO) and Collaborative Cross (CC) mice. We find host genetic background is a key regulator of immunotherapy outcomes for a variety of treatment modalities. Genetic linkage analysis in these models has identified putative resistance mechanisms for cancer vaccination, immune checkpoint inhibition, and targeted immunotherapy. Causal gene/pathway analysis has identified candidate biomarkers and secondary intervention targets to improve personalized medicine. My laboratory regularly conducts a myriad of in vitro/ex vivo, intratumoral, and peripheral immune assays combined with multi-omics approaches to determine the presence and functional status of anti-tumor immune cells. As such, we have multiple ongoing collaborations across the cancer center and university.

Honors

2025	Research Excellence Award, Wayne State University School of Medicine
2021	College Teaching Award, Wayne State University School of Medicine
2016	Young Investigator Award, Translational Research Cancer Centers Consortium
2015	Young Investigator Award, American Association of Immunologists
2014	Innovation Fellowship, Wayne State University
2014	Young Investigator Award, American Association of Immunologists
2014	Travel Award, Translational Research Cancer Centers Consortium
2013	Travel Award, Translational Research Cancer Centers Consortium
2012	Travel Award, Translational Research Cancer Centers Consortium
2010	2nd place poster award, Translational Research Cancer Centers Consortium
2005	Oral presentation winner, Henry Ford Health System Research Symposium

Contributions to Science

1. As a research assistant and graduate student, my studies focused on understanding the mechanisms of transcriptional dysregulation in cutaneous T cell lymphoma (CTCL) and the related leukemia Sezary syndrome. We investigated patient tumor cell transcriptomes and found distinct profiles of immune-related gene expression which could differentiate the progressive stages of the disease. Among these abnormally expressed genes is CTLA-4, which plays a role in immune suppression. I dissected the transcriptional regulation of this gene in both normal and CTCL/Sezary patient T cells and teased out various mechanisms controlling the inducible form of CTLA-4, including the role of transcription factors NFAT1 and GATA3 (PMIDs: 22951729, 17785820, 24173147). I also investigated the mechanisms underlying the transcriptional

regulation of the genes PLS3, GATA6 and TWIST1, which are aberrantly overexpressed in CTCL and Sezary Syndrome (PMID: 25806852). This work demonstrated a role for promoter hypomethylation in the elevated expression of these genes in the diseased T cell population.

2. Upon completion of my thesis work, I joined the Wei lab as a post-doctoral fellow and quickly gained experience in the development and testing of tumor antigen-specific DNA vaccines. I was fortunate to participate in the development of a new vaccine targeting TRAIL death receptor DR5 to find that agonist antibodies induced by human DR5 DNA trigger apoptosis in triple negative breast cancer (PMID: 22419388). I also implemented an innovative vaccine design platform to encode a structurally conservative single amino acid substitution within the vaccine cDNA, which we find promotes immunogenicity of the vaccine antigen resulting in circumvention of immune tolerance (PMID: 31177328). In an effort to develop a preclinical system to more closely mimic human immune tolerance to a tumor-associated antigen, I undertook the effort to test HER2 DNA vaccination in a novel feline mammary carcinoma model (PMID: 25711535). We find cats respond vigorously to DNA electroporation and their self-tolerance can be overcome by heterologous or mutant self HER2, however response is variable in these outbred animals. As a means of identifying host-intrinsic regulators of HER2 DNA vaccine response, I spearheaded the use of Diversity Outbred (DO) mice for genetic linkage analysis, where we identified the non-classical MHC Ib locus and regulatory T cells as playing prominent roles in vaccine outcomes (PMID: 32796024).

3. I have investigated modulation of macrophages the tumor microenvironment as a means of enhancing anti-tumor immunity. This includes testing of cryoablation (PMID: 25092895), cytokine producing adenovirus (PMID: 25949865), photodynamic therapy (PMID: 35235227), and de novo purine nucleotide biosynthesis inhibitors (PMID: 35790779) to determine their impact on local and systemic immunity. Each therapeutic modality initiates unique immune stimulating and suppressive responses, warranting mechanistic dissection in order to identify novel therapeutic approaches and logical co-therapeutic strategies.

4. As immunotherapy becomes a mainstay in cancer treatment, it is increasingly important to develop reliable methods to monitor therapeutic response to appropriately adjust the treatment regimen for improved patient outcomes. Toward this goal, I have co-developed positron emission tomography (PET) tracers to immune components including surface markers CD3 and CD8 (PMID: 32330387), and pro-inflammatory cytokines interferon gamma (IFNg) and interleukin 12 (IL-12). We have shown IFNg PET correlates with response to both HER2 cancer vaccines (PMID: 30115693) and immune checkpoint inhibitors (PMID: 38130991), and the FDA has approved an IND application to test a human IFNg PET tracer in clinical trials. For therapies that activate innate immunity upstream of T cell activation, such as oncolytic virus, we have found that IL-12 PET is a prognostic indicator of therapeutic response (PMID: 35634303).

5. Response to immunotherapy is variable in the human population, and it is unclear what factors differentiate patient outcomes. Beginning in my postdoctoral studies and extending through my current independent research projects, I have studied the role of the host genetic background in immunotherapy efficacy. My results show that genetic diversity within the host results in vastly different response to immunotherapy, including self antigen (HER2) vaccination (PMIDs: 32796024, 25711535), immune checkpoint inhibitors (PMID: 35481286), and targeted immunotherapy (in preparation). We also find differential intratumoral immune phenotypes by genetic ancestry in patients from the Karmanos Cancer Institute catchment area (PMID: 38953893). We have begun to identify key regulatory mechanisms and we are evaluating these genes/pathways for their utility as co-therapeutic targets and/or biomarkers to guide clinical strategy. An example is our recent discovery that dopamine receptor antagonism improves immune checkpoint inhibitor efficacy (PMID: 41895714), which stems from our genetic linkage analysis studies.

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