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

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Johnson, Gary

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-2867-0551

Position Title: Distinguished Professor

Organization and Location: Department of Pharmacology, University of North Carolina School of Medicine, Chapel Hill, North Carolina, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
University of California, San Francisco, California, United States	Other (OTH)	03/1976	05/1979	Biochemistry
University of Colorado Medical School, Denver, Colorado, United States	Doctor of Philosophy (PHD)	08/1971	02/1976	Pharmacology
California State University, Northridge, California, United States	Bachelor of Arts (AB)	08/1968	06/1971	Biology

Appointments and Positions

2019 - present	Distinguished Professor, Department of Pharmacology, University of North Carolina School of Medicine, Chapel Hill, North Carolina, United States
2014 - present	Member, American Association for Cancer Research (AACR), Philadelphia, Pennsylvania, United States
2014 - 2017	Co-Founder, KinoDyn, Inc., Durham, North Carolina, United States
2011 - 2019	Kenan Distinguished Professor, Department of Pharmacology, University of North Carolina School of Medicine, Chapel Hill, North Carolina, United States
2003 - present	Member, American Society of Pharmacology and Experimental Therapeutics (ASPET), Rockville, Maryland, United States
2003 - 2025	Co-directory, Program in Molecular Therapeutics, Lineberger Comprehensive Cancer Center, Chapel Hill, North Carolina, United States
2003 - 2017	Professor, Chair, Department of Pharmacology, University of North Carolina School of Medicine, Chapel Hill, North Carolina, United States
2002 - 2003	Vice Chair, Dept. of Pharmacology, University of Colorado Medical Sch., Denver, Colorado, United States
2001 - 2005	Consultant, Atherogenics , Inc, Alpharetta, Georgia, United States
1999 - 2003	Member, Molecular Biology Program, University of Colorado Medical School, Denver, Colorado, United States
1999 - 2003	Associate Director of Basic Sciences, University of Colorado Cancer Center, Denver, Colorado, United States
1996 - 2000	Director, Program in Molecular Signal Transduction, National Jewish Health, Denver, Colorado, United States
1994 - 1999	Director of Cell Biology, Cadus Pharmaceuticals, Inc., Tarrytown, New York, United States
1989 - 2003	Member, Cancer Center, University of Colorado School of Medicine, Denver, Colorado, United States
1989 - 2003	Professor, Dept. of Pharmacology, University of Colorado School of Medicine, Denver, Colorado, United States
1988 - 2000	Senior Scientist, Div. Basic Sciences, National Jewish Health, Denver, Colorado, United States
1981 - 1988	Assoc. Professor, Dept. of Biochemistry, Univ. of Massachusetts Med. Ctr., Worcester, Massachusetts, United States
1979 - 1981	Assist. Professor, Div. of Biology/Med, Sec. of Physiological Chem, Brown Univ, Providence, Rhode Island, United States

Products

Products Closely Related to the Proposed Project

1. Prasath V, Boutrid H, Wesolowski R, Abdel-Rasoul M, Timmers C, Lustberg M, Layman RM, Macrae E, Mrozek E, Shapiro

C, Glover K, Vater M, Budd GT, Harris L, Isaacs C, Dees C, Perou CM, Johnson GL, Poklepovic A, Chen H, Villalona-Calero M, Carson W, Stover DG, Ramaswamy B. Phase II study of MEK inhibitor trametinib alone and in combination with AKT inhibitor GSK2141795/uprosertib in patients with metastatic triple negative breast cancer. Breast Cancer Res Treat. 2025 Feb;210(1):179-189. PubMed Central PMCID: PMC12796985.

2. Xu Y, Peng XL, East MP, McCabe IC, Stroman GC, Jenner MR, Chan PS, Morrison AB, Shen EC, Hererra SG, Joisa CU, Rashid NU, Iuga AC, Gomez SM, Miller-Phillips L, Boeck S, Heinemann V, Haas M, Ormanns S, Johnson GL, Yeh JJ. Tumor-Intrinsic Kinome Landscape of Pancreatic Cancer Reveals New Therapeutic Approaches. Cancer Discov. 2025 Feb 7;15(2):346-362. PubMed Central PMCID: PMC11805639.
3. Lin B, Shelton AK, Smithberger E, Ziebro J, Skinner KR, Bash RE, Kirkman R, Stamper A, Butler M, Flores A, Angus SP, East MP, Cloughesy TF, Nathanson DA, Berens ME, Sarkaria JN, Binder ZA, O'Rourke DM, Howton TC, Lasseigne BN, Willey CD, Johnson GL, Hjelmeland AB, Furnari FB, Miller CR. Identifying and exploiting combinatorial synthetic lethality by characterizing adaptive kinome rewiring of EGFRvIII-driven glioblastoma. Acta Neuropathol Commun. 2025 Jun 28;13(1):143. PubMed Central PMCID: PMC12205505.
4. Angus SP, Stuhlmiller TJ, Mehta G, Bevill SM, Goulet DR, Olivares-Quintero JF, East MP, Tanioka M, Zawistowski JS, Singh D, Sciaky N, Chen X, He X, Rashid NU, Chollet-Hinton L, Fan C, Soloway MG, Spears PA, Jefferys S, Parker JS, Gallagher KK, Forero-Torres A, Krop IE, Thompson AM, Murthy R, Gatza ML, Perou CM, Earp HS, Carey LA, Johnson GL. FOXA1 and adaptive response determinants to HER2 targeted therapy in TBCRC 036. NPJ Breast Cancer. 2021 May 12;7(1):51. PubMed Central PMCID: PMC8115531.
5. Fisher MJ, Shih CS, Rhodes SD, Armstrong AE, Wolters PL, Dombi E, Zhang C, Angus SP, Johnson GL, Packer RJ, Allen JC, Ullrich NJ, Goldman S, Gutmann DH, Plotkin SR, Rosser T, Robertson KA, Widemann BC, Smith AE, Bessler WK, He Y, Park SJ, Mund JA, Jiang L, Bijangi-Vishehsaraei K, Robinson CT, Cutter GR, Korf BR, Blakeley JO, Clapp DW. Cabozantinib for neurofibromatosis type 1-related plexiform neurofibromas: a phase 2 trial. Nat Med. 2021 Jan;27(1):165-173. PubMed Central PMCID: PMC8275010.

Other Significant Products Highlighting Contributions to Science

1. Matkar S, East MP, Stuhlmiller TJ, Witek GM, Farrel A, Pastor S, Okumu DO, Kennedy A, Kalna JR, Berko ER, Casey CE, Krytska K, Patel K, Rokita JL, Gerelus M, Maris JM, Johnson GL, Mossé YP. Kinome Reprogramming of G2/M Kinases and Repression of MYCN Contribute to Superior Efficacy of Lorlatinib in ALK-Driven Neuroblastoma. Mol Cancer Ther. 2025 Sep 2;24(9):1389-1401. PubMed Central PMCID: PMC12322148.
2. Joisa CU, Chen KA, Beville S, Stuhlmiller T, Berginski ME, Okumu D, Golitz BT, East MP, Johnson GL, Gomez SM. Combined kinome inhibition states are predictive of cancer cell line sensitivity to kinase inhibitor combination therapies. Pac Symp Biocomput. 2024;29:276-290. PubMed Central PMCID: PMC11413988.
3. Flint AC, Mitchell DK, Angus SP, Smith AE, Bessler W, Jiang L, Mang H, Li X, Lu Q, Rodriguez B, Sandusky GE, Masters AR, Zhang C, Dang P, Koenig J, Johnson GL, Shen W, Liu J, Aggarwal A, Donoho GP, Willard MD, Bhagwat SV, Clapp DW, Rhodes SD. Combined CDK4/6 and ERK1/2 Inhibition Enhances Antitumor Activity in NF1-Associated Plexiform Neurofibroma. Clin Cancer Res. 2023 Sep 1;29(17):3438-3456. PubMed Central PMCID: PMC11060649.
4. Zawistowski JS, Bevill SM, Goulet DR, Stuhlmiller TJ, Beltran AS, Olivares-Quintero JF, Singh D, Sciaky N, Parker JS, Rashid NU, Chen X, Duncan JS, Whittle MC, Angus SP, Velarde SH, Golitz BT, He X, Santos C, Darr DB, Gallagher K, Graves LM, Perou CM, Carey LA, Earp HS, Johnson GL. Enhancer Remodeling during Adaptive Bypass to MEK Inhibition Is Attenuated by Pharmacologic Targeting of the P-TEFb Complex. Cancer Discov. 2017 Mar;7(3):302-321. PubMed Central PMCID: PMC5340640.
5. Duncan JS, Whittle MC, Nakamura K, Abell AN, Midland AA, Zawistowski JS, Johnson NL, Granger DA, Jordan NV, Darr DB, Usary J, Kuan PF, Smalley DM, Major B, He X, Hoadley KA, Zhou B, Sharpless NE, Perou CM, Kim WY, Gomez SM, Chen X, Jin J, Frye SV, Earp HS, Graves LM, Johnson GL. Dynamic reprogramming of the kinome in response to targeted MEK inhibition in triple-negative breast cancer. Cell. 2012 Apr 13;149(2):307-21. PubMed Central PMCID: PMC3328787.

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 Johnson, Gary in SciENcv on 2026-04-28 13:16:42

NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: Johnson, Gary

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-2867-0551

Position Title: Distinguished Professor

Organization and Location: Department of Pharmacology, University of North Carolina School of Medicine, Chapel Hill, North Carolina, United States

Personal Statement

My academic position at UNC is Distinguished Professor in the Department of Pharmacology where I served as chair of the department for 14 years from 2003-2017. I was also the Kenan Distinguished Professor from 2011-2019. I served from 2004-2025 as co-director of the Program in Molecular Therapeutics for the Lineberger Comprehensive Cancer Center. I have had a research laboratory continuously funded by NIH since 1979. I have served on many NIH committees including the Board of Counselors for the NIDDK, NIGMS Council (ad hoc), chaired the NIGMS Pharmacogenetics Review Committee and served on the NIGMS Glue Grant Review Panel. I have served on the scientific advisory boards of two publicly traded biotechnology companies. I have trained 56 postdoctoral fellows and 28 PhD students. Representative past students and fellows currently have positions at Vanderbilt SOM, Oklahoma HSC, Minnesota HSC, Emory SOM, Colorado HSC, North Carolina SOM (in Cell Biology independent of my lab), Fox Chase Cancer Center, Indiana SOM and Arizona SOP. Two trainees have successfully started their own companies (one a student, one a fellow). Several have had significant leadership roles including Vice President at Bayer Pharmaceutical, Associate Director for Research at the Oklahoma Cancer Center, chair of Craniofacial Biology, University of Colorado HSC, chair of Pediatric Oncology at Emory, and scientific director of the Manitoba Institute for Cell Biology. Thus, I have extensive experience successfully mentoring post-doctoral fellows and students, directing a large basic science department and evaluating innovative scientific initiatives. As a translational basic scientist my research interests focus on understanding the behavior of the kinome en masse in cancer. My laboratory has developed chemical proteomics methods that allow measurement of the functional state of ~90% of the kinome that can be applied to cell lines, preclinical animal models, patient-derived xenografts, organoids and patient biopsies including archival FFPE tumors. Our studies integrate kinome proteomics with next generation sequencing and chromatin epigenetics to define the dynamic behavior of the kinome at both baseline and with perturbation in preclinical cancer models and patient clinical trials. Our quantitative human kinase PRM stable isotope peptide library uniquely positions us to systematically elucidate the baseline kinome specific to the 20% of PDAC patients that are basal subtype and most chemotherapy resistant. This can be done with archival FFPE or flash frozen primary and metastatic PDAC needle biopsies requiring less than 10 mcg of protein. Measurement of kinases at attomole-femtomole proteomic expression levels allows for determining dynamics of adaptive kinome responses to kinase inhibitors over time of patient treatment. The proteomic analysis integrated with RNAseq analysis will be used to develop predictive-experimental platforms for targeting basal subtype PDAC kinase vulnerabilities.

Projects that I would like to highlight:
R01 CA288145 2/1/2024-12/31/2029 Yeh (MPI-Contact), Johnson, co-MPI, Targeting EGFR in basal subtype cancer

P50-CA257911 Yeh (PI) 09/16/2022-08/31/2027 Johnson, co-I Project 3, Selective Targeting of Pancreatic Cancer SPORE

U01CA274298 Yeh (PI) 9/1/2022 – 8/31/2027 Johnson, co-I, Integrating tumor and stroma to understand and predict treatment response

Honors

2024	Keynote Speaker, Celebration of careers of Joan Heller Brown and Paul Insel, Department of Pharmacology, University of California, San Diego
2018	Hyman L. Battle Distinguished Cancer Research Award, University of North Carolina
2016	Keynote Speaker, Gordon Conference on G Protein Coupled Receptors & Protein Phosphorylation
2014	Keynote Speaker, FASEB Conference on Protein Phosphorylation, Cellular Plasticity & Signaling Rewiring
2012	Meet the Professor Lecture, AACR Annual Meeting
2011	Plenary Lecture, Seoul National University Symposium on Pharmacological Manipulation of Cancer Cell Proliferation & Transdifferentiation

2008	Directors Distinguished Lectureship, NIEHS
2008	Johnson-Sokatch Lecture, University of Oklahoma Health Sciences Center
2007	Keynote Speaker, Beijing Symposium on Cell Signaling: Cancer, Development and Stem Cells
2007	ISI Highly Cited Researcher in Biology and Biochemistry , Thomas Scientific, ISIHighlyCited.com
2002	Co-Chair, Protein Kinases Keystone Symposium
1998 - 2008	Merit Award, NIGMS
1996	Dean's Distinguished Seminar, University of Colorado Health Sciences Center
1995	Chair, Gordon Research Conference on Molecular Pharmacology
1993	Chair, Gordon Research Conference on Second Messengers & Protein Phosphorylation

Contributions to Science

1. We have used our methods to define the adaptive bypass mechanisms that result in the lack of durable responses to targeted kinase inhibitors in the clinic. We published a paper in Cell Reports that showed how BET-bromodomain inhibitors could block the transcriptional upregulation of receptor tyrosine kinases making the response to kinase inhibition durable. We are involved in multiple clinical trials using these methods. Blocking adaptive bypass resistance to kinase inhibitors at its epigenetic root has significant clinical implications for making therapeutic responses more durable. The relevance of such an approach is evident in our TNBC studies. The adaptive response signatures in BL versus CL cell lines and patient tumors are different but the two subtypes are treated similarly in the clinic. If the adaptive response to single kinase inhibitors such as trametinib could be blocked epigenetically, by targeting enhancer formation/remodeling by inhibiting P-TEFb constituents such as BRD4, p300, JMJD6, CDK7 or CDK9 adaptive resistance could be prevented and possibly reversed.

2. The Cell Stem Cell paper we published defined for the first time a mutation that captured a self-renewing tissue stem cell in a permanent state of EMT. This is one of several papers we published on function of the MAP3K, MEKK4, in controlling EMT. Our work was the first to show that MEKK4-JNK controlled the histone acetyltransferase, CBP, for histone acetylation regulating EMT. From these studies we used high-throughput microscopy screens to define the function of the SWI/SNF chromatin modifying complex in EMT. Similar screening methods defined specific microRNAs in synthetic lethality screens involving inhibition of the MEK-ERK1/2 pathway and the Tousled-like kinases in regulating herpes virus latency.

3. Narrow spectrum specificity chemical tools and probes are essential for use in functional studies of their target kinases. Mike East working in my laboratory developed methods for inhibitor bead enrichment of functional kinases from cell lysates coupled with mass spectrometry (MIB/MS) to profile the specificity of novel kinase inhibitors. This work has resulted in the informed design of novel kinase inhibitors and offers a physiologically relevant profile of compound specificity as it uses native, endogenous kinases. The method is being adopted by many chemical biology programs

4. Different agonists do not necessarily activate receptors through stabilization of the same active state. We discovered and I believe the first lab to publish that different agonists targeting the same GPCR have a “bias” and do not activate signaling pathways with the same intensity. We termed this “biased agonism” and “asymmetric signaling” in our studies with the bombesin receptor in small cell lung carcinoma. Biased agonism is now a major emphasis for guiding structure-activity relationships and the development of new drugs in the pharmaceutical industry.

5. My research laboratory was one of 2-3 laboratories in the early 90’s that demonstrated oncogenes including Ras and Src as well as specific GPCRs activated the MAPK, ERK1/2. We also cloned a series of MAP3Ks (MEKK1, 2, 3 & 4) and showed they differentially regulated ERK1/2, JNK and p38. This was groundbreaking because it defined the MAPK signaling network as a large network of MAP3Ks, MAP2Ks and MAPKs and not a series of linear pathways. We went on to define the role of MAPKs in proliferation, apoptosis, migration and invasion. Both gene knockouts and knockins were used to define MAP3K function in mice. My laboratory has published approximately 250 papers related to the function of MAPK networks in different aspects of human disease.

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