BIOGRAPHICAL SKETCH

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NAME: Anthony J. Baucum II

eRA COMMONS USER NAME (credential, e.g., agency login): baucuma

POSITION TITLE: Associate Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Loyola Marymount University, Los Angeles, CA	B.S.	05/1999	Biology
University of Utah, Salt Lake City, UT	Ph.D	06/2004	Pharmacology
University of Utah, Salt Lake City, UT	Postdoctoral	06/2006	Drugs of abuse, dopamine transporter
Vanderbilt University, Nashville, TN	Postdoctoral	12/2011	Proteomics, phosphatases, kinases, protein interactions, and Parkinson disease

A. Personal Statement

I started my independent career at Indiana University-Purdue University Indianapolis (IUPUI) in the Biology Department in 2013. I was recently promoted to Associate Professor (August 1st, 2019) with Tenure (Starting July 1st, 2020). My independent research program focuses on signaling changes in the striatum under different pathological conditions. Specifically, I want to understand the function of the protein phosphatase 1 (PP1) interacting proteins, spinophilin and neurabin (collectively termed the neurabins), in the direct and indirect pathway medium spiny neurons of the striatum and other specific cell types in different brain regions such as the cerebellum. Protein phosphatases, such as PP1, obtain substrate specificity by targeting proteins. The neurabins, spinophilin and its homolog, neurabin, are the most abundant PP1 interacting protein in the PSD and are thought to be **critical hub proteins** that coordinate the phosphorylation and function of myriad synaptic proteins involved in neurodegenerative and neuropsychiatric diseases. Therefore, pathological changes in the association of the neurabins with PP1 and PP1 substrates will greatly influence the function of this critical phosphatase. I am well-versed in biochemical, molecular biological, imaging, proteomic, and electrophysiological methods to identify and characterize the function of the neurabins. Our previous studies have identified multiple spinophilin interacting proteins that are regulated by dopamine depletion (animal model of Parkinson disease (PD; Hiday et al., 2017)) or hyperdopaminergic signaling, such as is observed following psychostimulants (Watkins et al., 2018). In addition to changes in spinophilin protein interactions following altered dopamine signaling, we have found that whole-body spinophilin KO animals do not undergo amphetamine locomotor sensitization (Morris et al., 2018), a behavioral change associated with addiction. We also have found that cell-type specific loss of spinophilin modulates other striatal-based motor behaviors, including grooming induced by agonism of the metabotropic glutamate receptor 5. Together, our studies suggest a role for spinophilin in modulating striatal behaviors and the goal of the laboratory is to delineate mechanisms by which spinophilin modulates these behaviors.

I am part of the training programs in the Department of Biology, the Medical Neurosciences Program, and Pharmacology and Toxicology. I currently have Ph.D. 2 students from Biology and 2 students from Medical Neurosciences program.

Relevant publications

1) Morris CW, Watkins DS, Salek AB, Michael C. Edler, **Baucum II, AJ.** (2018). The association of spinophilin with disks large-associated protein 3 (SAPAP3) is regulated by metabotropic glutamate receptor (mGluR) 5. Mol Cell Neuro. Jun 14;90:60-69. PMC6294707

2) Hiday AC, Edler MC, Salek AB, Morris CW, Thang M, Rentz TJ, Rose KL, Jones LM, and **Baucum 2nd AJ**. (2017) Mechanisms and consequences of dopamine depletion-induced attenuation of the spinophilin/neurofilament medium interaction. Neural Plasticity 2017, 4153076. PMCID:PMC5467389

3) Salek, A.B.; Edler, M.C.; McBride, J.P.; **Baucum, A.J., 2nd**. Spinophilin regulates phosphorylation and interactions of the GluN2B subunit of the N-Methyl-D-Aspartate Receptor. *J Neurochem* **2019**. PMID 31325175

4) Watkins DS, True JD, Mosley AL, **Baucum AJ 2nd**. (2018). Proteomic analysis of the spinophilin interactome in rodent striatum following psychostimulant sensitization. Proteomes. Dec 17;6(4). Pii: E53. PMCID: PMC6313900

B.Positions and Honors

Positions

2011-2013: Research Instructor, Vanderbilt University
8/2013-07/2019: Assistant Professor, Biology, IUPUI
08/2019 – Present: Associate Professor, Biology, IUPUI
8/2013 – Present: Primary Faculty Stark Neurosciences Research Institute, Indiana University School of Medicine
04/2018 – Present: Adjunct Appointment, Pharmacology and Toxicology, Indiana University School of Medicine

Other Experience and Professional Memberships

2001-2002: Student representative to the graduate training committee, Department of Pharmacology and Toxicology, University of Utah
2004: Student representative to the executive committee of the American Society of Pharmacology and Experimental Therapeutics – Neuropharmacology Division
2004-present: Member, Society for Neuroscience.
2006-2007: Junior co-chair Vanderbilt University postdoctoral association
2007-present: Member, American Society for Pharmacology and Experimental Therapeutics
2007-2008: Member board of directors Vanderbilt Medical Alumni Association
2008: American Society of Pharmacology and Experimental Therapeutics long-range planning committee member.
2008-2009: Senior advisor Vanderbilt University postdoctoral association
2008-2013 Faculty of 1000 Associate Member
2009: Search committee member for the dean of the Vanderbilt University Medical School
2009: Panel member on development of an individual development plan. National Postdoctoral Association annual meeting.
2010: Lecturer Vanderbilt University integrated graduate program course
2010: Internal mock grant reviewer for Tutorials in Physiology course
2010-2011: Board of directors, National Postdoctoral Association.
2013-2014: Search Committee Member, Department of Biology, IUPUI
2013-Present: Graduate Studies Committee Member, IUPUI Biology
2018-Present: Graduate Education Committee Member, School of Science
2019: Temporary member, SYN Study Section
2013-Present: Have served/am serving on over 30 graduate student thesis/dissertation committees.
2018-Present: Member Teaching Advisory Committee, Stark Neurosciences Research Institute
2019 -Present: Director of Thesis Graduate Studies, Department of Biology, IUPUI
2020-Present: Trainee Professional Development Awards Selection Committee – Society for Neuroscience

Honors and Awards (Intramural)

1999: Presidential Citation. Loyola Marymount University

1999: Kadner Biology Award for academic excellence, strong leadership, and dedicated service to the Biology Department. Loyola Marymount University
1999: Jerome Korth Award for highest core GPA in biology. Loyola Marymount University
1995-1999: Trustee Scholarship, Loyola Marymount University
2004: Wolf Prize in Teaching. An award that recognizes excellence in teaching
2009: Best poster award Vanderbilt Postdoc Poster Symposium – Neuroscience division
2009: Best poster award Vanderbilt Brain Institute Neuroscience Retreat
2009: Best poster award Kennedy Center Science Day, cellular and molecular neuroscience postdoc division. Vanderbilt, University
2010: Postdoc of the Year, Vanderbilt University Medical Center
2010: Best poster award Vanderbilt Postdoc Poster Symposium – Neuroscience division
2011: Best poster award Kennedy Center Science Day, cellular and molecular neuroscience postdoc division. Vanderbilt, University

Honors and Awards (Extramural)
2003: 2nd place American Society for Pharmacology and Experimental Therapeutics best paper competition, Neuropharmacology, graduate student division
2004: 1st place American Society for Pharmacology and Experimental Therapeutics best paper competition, Neuropharmacology, graduate student division
2004: Travel award to the Experimental Biology meeting in Washington D.C.
2005: 3rd Place American Society for Pharmacology and Experimental Therapeutics best paper competition, Neuropharmacology, postdoctoral division
2005: Young Scientist Travel Award, American Society for Pharmacology and Experimental Therapeutics, for Experimental Biology meeting
2007-2009: Neuroscience Scholar Award, Society for Neuroscience.
2009: 2nd Place Neuropharmacology Division of ASPET postdoctoral award competition, Experimental Biology
2014: Selected to attend a grant-writing workshop put on by NINDS for diverse researchers
2016-2017: Mentoring Institute for Neuroscience Diversity Scholars Fellow.

C. Contributions to Science

1. During graduate school, my research focused on effects of neurotoxic regimens of methamphetamine on pre-synaptic protein interactions. Methamphetamine is known to regulate levels of extracellular dopamine (DA) by causing reversal of the transporter. Methamphetamine is a highly abused psychostimulant that at high doses can lead to neurotoxicity. Moreover, individuals who abuse methamphetamine are at a higher risk for developing PD. This toxicity is dependent upon both hyperthermia and dopamine receptor agonism. Specifically, I was interested in how a neurotoxic regimen of methamphetamine altered DA transporter (DAT) oligormerization. We found that neurotoxic regimens of methamphetamine led to increased DAT oligomerization that was dependent upon agonism of the dopamine receptor and hyperthermia. Our data identified presynaptic, biochemical changes that occur early on following toxic administrations of methamphetamine that may contribute to methamphetamine toxicity. In addition to the dopamine transporter, I was involved on studies evaluating the effect of methamphetamine on the vesicular monoamine transporter-2. Specifically, this study was focused on effects of methamphetamine in the hippocampal serotonin transport of treated rats.

a. **Baucum AJ 2nd**, Rau KS, Riddle EL, Hanson GR, Fleckenstein AE. (2004). Methamphetamine increases dopamine transporter higher molecular weight complex formation via a dopamine- and hyperthermia-associated mechanism. Journal of Neuroscience. 24(13):3436-43. PMID: 15056723

b. Hadlock GC, **Baucum AJ 2nd**, King JL, Horner KA, Cook GA, Gibb JW, Wilkins DG, Hanson GR, Fleckenstein AE. (2009). Mechanisms underlying methamphetamine-induced dopamine transporter complex formation. Journal of Pharmacology and Experimental Therapeutics. 329(1):169-74. PMCID: PMC2670587

c. Hadlock GC, Nelson CC**, Baucum AJ 2nd**, Hanson GR, Fleckenstein AE. (2011). Ex vivo identification of protein-protein interactions involving the dopamine transporter. Journal of Neuroscience Methods. 196(2):303-7. PMCID: PMC3873427

d. Rau KS, Birdsall E, Volz TJ, Riordan JA, **Baucum AJ 2nd**, Adair BP, Bitter R, Gibb JW, Hanson GR, Fleckenstein AE. (2006). Methamphetamine administration reduces hippocampal vesicular monoamine transporter-2 uptake. Journal of Pharmacology and Experimental Therapeutics. 318(2):676-82. PMID: 16687477

2. During my postdoc, one of my areas of research focus was on the role of calcium/calmodulin-dependent protein kinase II (CaMKII) subcellular localization and protein-protein interactions. Specifically, I was interested in mechanisms that regulate CaMKII interactions and how those interactions modulate CaMKII subcellular localization. CaMKII is known to play a critical role in normal learning and memory. In addition, CaMKII activity is increased in animal models of PD and reduction of this activity ameliorates some of the disease pathologies, at least in rodents. Specifically, I found that the beta isoform of CaMKII is phosphorylated at multiple sites in its actin-binding domain and that phosphorylation of these sites is different in different subcellular fractions. In addition, I found that preventing phosphorylation of Thr286 on CaMKIIalpha has multiple effects on the phosphorylation of other sites on CaMKIIalpha as well as on sites on CaMKIIbeta. Moreover, in multiple collaborative studies, I helped to characterize the association of CaMKII with multiple known and novel interacting proteins. In addition, I helped to develop a novel, crude subcellular fractionation technique that is useful in isolating crude synaptic fractions. Together, these data have implications in multiple neurological disorders, as CaMKII activity is regulated in different disease states.

a. **Baucum 2nd AJ**, Shonesy BC, Rose KL, and Colbran RC. (2015). Quantitative proteomics analysis of CaMKII phosphorylation and the CaMKII interactome in the mouse forebrain. ACS Chemical Neuroscience. 6(4):615-31. PMID: 25650780.

b. Gustin RM, Shonesy BC, Robinson SL, Rentz TJ, Jalan-Sakrikar N, **Baucum 2nd AJ**, Winder DG, Stanwood GD, Colbran RJ. (2011). Loss of Thr286 phosphorylation disrupts synaptic CaMKII□ targeting, NMDAR activity and behavior in pre-adolescent mice. Molecular and Cellular Neuroscience. 47(4):286-92. PMCID: PMC3149813

c. Shonesy BC, Wang X, Rose KL, Ramikie TS, Cavener VS, Rentz T, **Baucum 2nd AJ**, Jalan-Sakrikar N, Mackie K, Winder DG, Patel S, Colbran RJ. (2013). CaMKII regulates diacylglycerol lipase-α and striatal endocannabinoid signaling. Nature Neuroscience. 16(4):456-63. PMCID: PMC3636998

d. Jalan-Sakrikar N, Bartlett RK, **Baucum 2nd AJ**, and Colbran RJ. (2012). Substrate-selective and calcium-independent activation of CaMKII by α-actinin. Journal of Biological Chemistry. 287(19):15275-83. PMCID: PMC3346149

3. I have a strong background and interest in using proteomics approaches to delineate synaptic protein interactions in different cell types and under different pathological conditions. To this end, I have collaborated on studies delineating pathological changes in the GluN2B NMDAR proteome. Moreover, I am currently using cutting-edge approaches such as Tandem-Mass-Tag and MudPIT analysis to characterize synaptic protein phosphorylation and interactions.

a. **Baucum, A. J.** Proteomic analysis of post-synaptic protein complexes underlying neuronal plasticity. ACS Chem Neurosci, 2017. 8, 689-701.

b. Wills TA, **Baucum AJ** 2nd, Holleran KM, Chen Y, Pasek JG, Delpire E, Tabb DL, Colbran RJ, Winder DG. Chronic intermittent alcohol disrupts the GluN2B-associated proteome and specifically regulates group I mGlu receptor-dependent long-term depression. Addiction Biology. 2015 Nov 8. Epub ahead of print. PMCID: PMC4860359.

c. **Baucum 2nd AJ**, Jalan-Sakrikar N, Jiao Y, Gustin RM, Carmody LC, Tabb DL, Ham A-J L, Colbran RJ. (2010) Identification and validation of novel spinophilin-associated proteins in rodent striatum using an enhanced ex vivo shotgun proteomics approach. Molecular and Cellular Proteomics. 9(6):1243-59. PMCID:PMC2877984

4. The major focus of my postdoctoral studies and my independent research program is on the mechanisms that regulate the association of the synaptic protein spinophilin and the functional consequences of perturbations of spinophilin interactions. Spinophilin is an F-actin and protein phosphatase 1 interacting protein that is known to modulate the activity of multiple synaptic proteins and is important in normal striatal learning. I have a strong interest in how spinophilin interactions are altered in animal models of PD and other neurological disorders. Using biochemical and mass spectrometry-based techniques I have identified novel spinophilin interacting proteins and begun to characterize those interactions. Specifically, I have found that spinophilin interacts with multiple synaptic proteins, including CaMKII, NMDARs, synaptic scaffolding proteins, and cytoskeletal proteins. I have also identified mechanisms that regulate the association of spinophilin with multiple synaptic proteins as well as age-dependent and subcellular fraction-dependent modulation of these associations. We are currently characterizing these associations and determining the effect of DA depletion, an animal model of PD, on these interactions and have recently published a manuscript identifying pathological changes in the spinophilin interaction that occur in an animal model of PD. Additionally, we are beginning to determine which

specific cell types in the striata have altered spinophilin phosphorylation and protein interactions. Moreover, we are also currently evaluating the functional role of spinophilin in regulating substrate phosphorylation. Together these data suggest a critical role in spinophilin-dependent modulation of synaptic proteins in normal physiology and in pathological neurodegenerative diseases.

a. Salek, A.B.; Edler, M.C.; McBride, J.P.; Baucum, A.J., 2nd. Spinophilin regulates phosphorylation and interactions of the GluN2B subunit of the N-Methyl-D-Aspartate Receptor. *J Neurochem* **2019**. PMID 31325175

b. Watkins DS, True JD, Mosley AL, **Baucum AJ 2nd**. (2018). Proteomic analysis of the spinophilin interactome in rodent striatum following psychostimulant sensitization. Proteomes. Dec 17;6(4). Pii: E53. PMCID: PMC6313900

c. Morris CW, Watkins DS, Salek AB, Michael C. Edler, **Baucum II, AJ.** (2018). The association of spinophilin with disks large-associated protein 3 (SAPAP3) is regulated by metabotropic glutamate receptor (mGluR) 5. Mol Cell Neuro. Jun 14;90:60-69. PMC6294707

d. Edler MC, Salek AB, Watkins DS, Kaur H, Morris CW, Yamamoto BK, **Baucum II AJ**. (2018). Mechanisms regulating the association of protein phosphatase 1 with spinophilin and neurabin. ACS Chem Neuro. Nov 21;9(11):2701-2712. PMID 29786422

Complete List of Published Work in MyBibliography
https://www.ncbi.nlm.nih.gov/myncbi/anthony.baucum.1/bibliography/public/
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D. Research Support

Current:

1 R25 NS107173-01A1 Baucum mPI 07/01/2019 – 06/30/2024
Effort: 1 Summer Month.
Neuroscience Experience and Undergraduate Research Opportunities Program (NEUROP)
The goal of this training proposal is to increase neuroscience training in the School of Science for undergraduate and graduate URM students.

R33 4R33DA041876 Baucum (PI) 04/01/2018 - 03/31/2021
Effort: 2 Summer Months.
Spinophilin function in regulating pathological responses to psychostimulant drugs.
The goal of this study is to delineate cell-specific function of spinophilin in mediating psychostimulant pathologies.

R33 Minority Supplement 3R33DA041876-03S1 Baucum (PI) 04/01/2018 - 06/30/2020
Generation of cell-specific tools to determine the role of spinophilin in regulating pathological responses to psychostimulant drugs of abuse.
The goal of this study is to support the research of Mr. Darryl Watkins, a URM, on the R33 grant.