Sara Margaret McMillin, PhD
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Education

PhD, Biochemistry and Molecular Genetics
George Washington University, Washington, DC
Graduate Partnership Program with the National Institutes of Health
Graduation Date: January 2011

BA, Biology
Appalachian State University, Boone, NC
Graduation Date: December 2003

Current Position

August 2016 – Present
Fred Wilson School of Pharmacy, High Point University, High Point, NC
Assistant Professor of Basic Pharmaceutical Sciences
Establishing an independent research program focusing on diabetes and metabolism. Teaching 1st and 2nd year pharmacy students in physiology, pharmacology, and medicinal chemistry.

Prior Scientific Research and Training

July 2015 – July 2016
Robert Wood Johnson Medical School, Rutgers University, New Brunswick, NJ
Instructor
Worked closely with Dr. Fredric Wondisford, setting up his new laboratory. Investigated the mechanisms of hepatic gluconeogenesis under various hormonal and nutritional stresses using mouse models and primary cell lines. Also studied the dose dependent effects of the drug metformin on cell metabolism in hepatocytes.

August 2013 – July 2015
Johns Hopkins University Medical School, Baltimore, MD
Post-Doctoral Research Fellow, Laboratory of Fredric Wondisford, MD
Investigated the genetic regulation of hepatic gluconeogenesis *in vivo*. Established multiple mouse models that exhibit elevated gluconeogenesis. Utilized adenoviral/shRNA technology to target and knock down transcriptional co-activators *in vivo.* Performed a comprehensive panel of *in vivo* metabolic analyses. Utilized metabolic cages (CLAMS) to monitor food intake, energy expenditure, and respiratory exchange ratio.

January 2011 – May 2013
National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD
Post-Doctoral Research Fellow, Laboratory of Jurgen Wess, PhD
Worked on a number of projects analyzing the effects of activating (*in vivo*) specific G protein signaling pathways. Generated transgenic mouse lines selectively expressing designer GPCRs known as DREADDs (Designer Receptors Exclusively Activated by Designer Drugs) in different metabolic tissues. Used a synthetic ligand (CNO) to selectively activate these receptors *in vivo*. Investigated the effects on whole body glucose homeostasis, insulin sensitivity, and energy expenditure.

January 2005 – December 2010

National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD
Pre-Doctoral Research Fellow, Laboratory of Jurgen Wess, PhD
Completed a thesis project analyzing the molecular and functional properties of G protein-coupled receptor dimerization. Introduced a wide array of mutations into the M3 subtype of the muscarinic acetylcholine receptor. Analyzed receptor dimerization through bioluminescence resonance energy transfer (BRET), cysteine cross-linking, and co-immunoprecipitation methods. Investigated functional properties through ligand binding, calcium mobilization, and inositol phosphate assays. Monitored receptor expression and cell surface localization via Western Blots, ELISA, and confocal microscopy.

Prior Teaching Experience

November 2013 & 2014
Johns Hopkins University Medical School, Baltimore, MD
Small group leader for the Metabolism block of "Scientific Foundations in Medicine", the first year curriculum for Johns Hopkins medical students. Covered carbohydrate, fat, and amino acid metabolism, as well as the TCA cycle and oxidative phosphorylation.

January 2013 – May 2013; January 2012 – May 2012
Foundation for the Advancement of Education in the Sciences (FAES)
National Institutes of Health, Bethesda, MD
Co-instructor and course organizer for a new course entitled Biochemistry in Health and Disease for the Layman. Course was designed for a general audience and provided a basic yet broad overview of biochemistry and molecular biology. Taught carbohydrate, fat, and protein metabolism, as well as the TCA cycle and the electron transport chain/ATP synthesis. Also taught two lectures covering DNA/RNA, gene transcription and translation, and the consequences of genetic mutations.

Activities, Memberships, Awards

New Investigator Award, American Association of Colleges of Pharmacy, January 2018

Member, Endocrine Society, January 2015 – present

Member, American Association for the Advancement of Science (AAAS), January 2011 - present

Helmsley Charitable Trust Travel Award, Endocrine Society Meeting (ENDO), San Diego, CA, 2015

Reviewer, PLOS One (2 manuscripts; 2014 - 2015) and Molecular Pharmacology (1 manuscript; 2014)

Member, NIH Diabetes Interest Group, 2009 – 2013

Co-Chair, NIH Graduate Student Council "Pathways" Career Development Committee, 2008 – 2010

Publications

Wang Y***, McMillin SM***, Kalemba K, Kwon H, Lu W, Xu H, Chauhan AV, White NE, Anacker KR, Rabinowitz JD, Su X, and Wondisford FE. 2019. Glycerol activates and is the major substrate for fasting gluconeogenesis. Nature; *Under Review*.

Kaiser E, Tian Q, Wagner M, Barth M, Xian W, Schroeder L, Ruppenthal S, Kaestner L, Boehm U, Wartenberg P, Lu H, **McMillin SM**, Bone DB, Wess J, Lipp P. 2018. DREADD technology reveals major impact of Gq signaling on cardiac electrophysiology. Cardiovasc Res; cvy251, doi: 10.1093/cvr/cvy251

Rossi M, Zhu L, **McMillin SM**, Pydi SP, Jain S, Wang L, Cui Y, Lee RJ, Cohen AH, Kaneto H, Birnbaum MJ, Ma Y, Rotman Y, Liu J, Cyphert TJ, Finkel T, McGuinness OP, Wess J. 2018. Hepatic Gi signaling regulates whole body glucose homeostasis. J Clin Invest; 128: 746-759.

Lu Z, Almaca J, Dadi PK, Hong H, Sakamoto W, Rossi M, Lee RJ, Lu H, Cui Y, **McMillin SM**, Kuo B, Leapman RD, Matchinsky FM, Doliba NM, Urs NM, Caron MG, Jacobson DA, Caicedo A, and Wess J. 2017. Beta-Arrestin-2 is an essential regulator of pancreatic beta-cell function under physiological and pathophysiological conditions. Nat Commun; 8: 14295.

He L, Chang E, Peng J, An H, **McMillin SM**, Radovick S, Stratakis CA, and Wondisford FE. 2016. Activation of the cAMP-PKA pathway antagonizes metformin suppression of hepatic glucose production. J Biol Chem; 291: 10562-10570.

Nakajima K, Jain S, Ruiz de Azua I, **McMillin SM**, Rossi M, and Wess J. 2013. Minireview: Novel Aspects of M3 Muscarinic Receptor Signaling in Pancreatic Beta Cells. Mol Endocrinol; 27: 1208-1216.

Li J, Jain S, **McMillin SM,** Cui Y, Gautum D, Lu H, Jou W, McGuinness OP, Gavrilova O, and Wess J. 2013. A Novel Experimental Strategy to Assess the Metabolic Effects of Selective Activation of a Gq-Coupled Receptor in Hepatocytes in vivo. Endocrinology; 154: 3539-3551.

Hu J, Thor D, Zhou Y, Wang Y, **McMillin SM**, Liu T, Mistry R, Challiss RA, Costanzi S, and Wess J. 2012. Structural aspects of M3 muscarinic acetylcholine receptor dimer formation and activation. FASEB J; 26: 604-616.

McMillin SM, Heusel M, Costanzi S, and Wess J. 2011. Structural Basis of M3 Muscarinic Receptor Dimer/Oligomer Formation. J Biol Chem; 286: 28584-28598.

Rosemond E, Rossi M, **McMillin SM**, Scarselli M, Donaldson JG, and Wess J. 2011. Regulation of M3 Muscarinic Receptor Expression and Function by Transmembrane Protein 147. Mol Pharmacol; 79: 251-261.

Li B, Scarselli M, Knudsen CD, Jacobson KA, **McMillin SM**, and Wess J. 2007. Rapid identification of functionally critical amino acids in a G protein-coupled receptor. Nat Methods; 4: 169-174.

Perrino FW, Harvey S, **McMillin S**, and Hollis T. 2005. The human TREX2 3'-> 5'-exonuclease structure suggests a mechanism for efficient nonprocessive DNA catalysis. J Biol Chem; 280: 15212-15218.

Grant Support

American Association of Colleges of Pharmacy. New Investigator Award. Co-PI. Project Title: Weight Loss with SGLT2 Inhibitor Use: An Uphill Battle. January 2018 – July 2019. $10,000