BIOGRAPHICAL SKETCH

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NAME: GARRETT, MICHAEL, R.

eRA COMMONS USER NAME (agency login): MGARRETT

POSITION TITLE: Associate Professor and Director

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

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
University of California-Riverside, Riverside, CA	BS	05/1993	Biochemistry
University of California-Riverside, Riverside, CA	MS	05/1994	Biochemistry
Bowling Green State University, Bowling Green, OH	MBA	05/1999	Finance
University of Toledo-College of Medicine, Toledo, OH	PHD	12/2006	Molecular Basis of Disease

A. PERSONAL STATEMENT

I have 20 years' experience in molecular biology, animal models of disease, and genetic and genomics techniques, including DNA based methods (cloning, sequencing, and NGS, etc.) and RNA based methods (quantitative real-time PCR, microarray, and RNAseq, etc.). I have an active research program involving studying the genetics of complex disease including hypertension, kidney disease, and diabetes. My laboratory takes a multidisciplinary approach to achieve this goal by utilizing animal models, cell-culture based systems, genetic and genomics methods, proteomics, and bioinformatics. My demonstrated record of accomplishment, including an independent research program with extramural funding (NIH, AHA, etc.), a strong publication history in high-quality journals (JASN, Hypertension, JCI, and Genome Research), a record of conducting collaborative research, along with my leadership abilities and diverse skill set provides the necessary tools to successfully achieve the proposed aims. Aside from my research activities, I currently serve as the Director of the Institutional Molecular and Genomics Core. I am responsible for meeting with research investigators, aiding in the design of experiments, providing oversight of technical staff and training, evaluating equipment needs, and conducting seminars to educate and promote core capabilities. My business training and education also provides a solid foundation to administer the proposed projects through frequent communication among project members and constructing realistic research plans, timeline, and managing budgets.

B. POSITIONS AND HONORS

Positions and Employment

1995 - 1999 **Laboratory Supervisor**, Medical College of Ohio, Department of Physiology and Molecular Medicine, Toledo, OH

1999 - 2001 **Research Instructor**, Medical College of Ohio, Department of Physiology and Molecular Medicine, Toledo, OH

2001 - 2003 **Research Assistant Professor**, University of Toledo-College of Medicine, Department of Physiology and Pharmacology, Toledo, OH

2003 - 2007 **Assistant Professor**, University of Toledo-College of Medicine, Department of Physiology and Pharmacology, Toledo, OH

2007 - 2010 **Assistant Professor**, Medical College of Wisconsin, Department of Medicine (Nephrology), Milwaukee, WI

2010 - **Associate Professor**, University of Mississippi Medical Center, Department of Pharmacology, Department of Medicine (Nephrology), Jackson, MS

2010 - **Director**, Molecular and Genomics Core Facility, University of Mississippi Medical Center, Jackson, MS

2014 - **Associate Director**, Center of Genetic Medicine, University of Mississippi Medical Center, Jackson, MS

Other Experience and Professional Memberships

2001 -	Member, American Association for Advancement of Science (AAAS)
2006 -	Member, American Society of Nephrology (ASN)
2007 -	Member, American Physiological Society (APS)
2009 -	Member, American Heart Association (AHA)
2012 -	Member, American Society for Pharmacology and Experimental Therapeutics (ASPET)
2012 -	Member, Association of Biomolecular Resource Facilities
2005	Ad hoc reviewer, NIH Peer Review Committee: Genomics of Transplantation
2010	Ad hoc reviewer, NIH Peer Review Committee: Mechanisms of Arterial Stiffening
2010	Ad hoc reviewer , NIH Peer Review Committee: Conference Grant Applications (2 times)
2012	Ad hoc reviewer, NIH Peer Review Committee: Comparative Medicine
2012 - current	Member, AHA Peer Review Committee: Basic Cell, Genetics and Epigenetics
2013	Ad hoc reviewer, NIH Peer Review Committee: SEP- EDIC/GoKind DP3
2014	Ad hoc reviewer, NIH Peer Review Committee: Biomarkers of Diabetes, Digestive, and Kidney
2003 - 2006	Editorial Board, Journal of Hypertension
2008 - 2012	Editorial Board, Physiological Genomics
2008 - 2012	Editorial Board, Frontiers in Genomics Physiology
2015 - current	Associate Editor, Physiological Genomics

Honors

2007	APS Star Reviewer, American Physiological Society-Physiological Genomics
2010	Robert M. Hearin Foundation Research Scholar, Robert M. Hearin Foundation
2011	Silver Medal for Excellence in Research, University of Mississippi Medical Center
2012	Leadership Development Program, University of Mississippi Medical Center
2012	Fellow of American Heart Association (FAHA), American Heart Association-Council for High Blood Pressure Research (HBPR)
2013	Gold Medal for Excellence in Research, University of Mississippi Medical Center

C. Contribution to Science

1. **Research Area 1:** The genetic basis of chronic kidney disease (CKD) in the Dahl salt-sensitive (S) rat. My early work (2003) in this area reported some of the first genetic analyses of kidney injury and decline in renal function in an animal model. These genetic studies were especially unique as they utilized two models of hypertension (S and SHR) that differ with respect to kidney injury. The advantage of these analyses has been the ability to identify loci that influence kidney injury on genetic backgrounds (either S or SHR) permissive for hypertension. Subsequently, I have moved from identifying genomic regions (linkage analysis) to identifying specific genes/genetic variants and molecular mechanisms that explain susceptibility of the S rat to develop CKD (2007- current). There have been few research investigator that have achieved this breath of success in positional cloning novel genes. The significance of this body of work is that the identification of gene/genetic variants related to kidney injury in the context of hypertension could have a significant impact on human health by serving as novel therapeutic targets for CKD.
    a. **Garrett MR**, Dene H, Rapp JP. Time-course genetic analysis of albuminuria in Dahl salt-sensitive rats on low-salt diet. J Am Soc Nephrol. 2003 May;14(5):1175-87. PubMed PMID: 12707388.
    b. **Garrett MR**, Gunning WT, Radecki T, Richard A. Dissection of a genetic locus influencing renal function in the rat and its concordance with kidney disease loci on human chromosome 1q21. Physiol Genomics. 2007 Aug 20;30(3):322-34. PubMed PMID: 17504948; PubMed Central PMCID: PMC3153419.
    c. Williams JM, Johnson AC, Stelloh C, Dreisbach AW, Franceschini N, Regner KR, Townsend RR, Roman RJ, **Garrett MR**. Genetic variants in Arhgef11 are associated with kidney injury in the Dahl

salt-sensitive rat. Hypertension. 2012 Nov;60(5):1157-68. PubMed PMID: 22987919; PubMed Central PMCID: PMC3505884.

d. Jia Z, Johnson AC, Wang X, Guo Z, Dreisbach AW, Lewin JR, Kyle PB, and **Garrett MR**. Allelic Variants in Arhgef11 via the Rho-Rock Pathway are linked to Epithelial–Mesenchymal Transition and Contributes to Kidney Injury in the Dahl S Rat. PLOS One (accepted), 2015

2. **Research Area 2**: The development and analysis of novel genetic models of disease including calcium oxalate stone disease, diabetic nephropathy, and renal agenesis/congenital abnormalities of kidney and urinary tract (CAKUT). I have been involved in the development of novel animal models to study human complex disease, both as the primary investigator and co-investigator. In particular, my laboratory was first to perform an analysis of renal phenotypes in the Heterogeneous stock (HS) rat model to explore its potential utility in identifying the genes involved in kidney disease. The study identified a family of rats born with a single kidney and other urogenital abnormalities. Subsequently these animals were used to develop a new inbred model (HSRA rat) that exhibit a high incidence of congenital solitary kidney/CAKUT (50-75%). The model has allowed for the first time to address the hypothesis that early development (*in utero*) with a solitary kidney increases susceptibility to renal injury and hypertension more than what is observed in either two-kidney animals or two-kidney animals subjected to uninephrectomy with age. The significance of this body of work is that provides a firm foundation to better understand the genetic basis of nephrogenesis/kidney development as well as understand confounding factors (hypertension and diabetes) that may impact and/or modulate susceptibility toward kidney injury in single kidney individuals.

   a. Wiessner JH, **Garrett MR**, Roman RJ, Mandel NS. Dissecting the genetic basis of kidney tubule response to hyperoxaluria using chromosome substitution strains. Am J Physiol Renal Physiol. 2009 Aug;297(2):F301-6. PubMed PMID: 19493966; PubMed Central PMCID: PMC2724241.

   b. Solberg Woods LC, Stelloh C, Regner KR, Schwabe T, Eisenhauer J, **Garrett MR**. Heterogeneous stock rats: a new model to study the genetics of renal phenotypes. Am J Physiol Renal Physiol. 2010 Jun;298(6):F1484-91.PMID: 20219828; Central PMCID: PMC2886820.

   c. Slaughter TN, Paige A, Spires D, Kojima N, Kyle PB, **Garrett MR**, Roman RJ, Williams JM. Characterization of the development of renal injury in Type-1 diabetic Dahl salt-sensitive rats. Am J Physiol Regul Integr Comp Physiol. 2013 Oct 1;305(7):R727-34. PubMed PMID: 23926133; PubMed Central PMCID: PMC3798803.

   d. Wang X, Johnson AC, Williams JM, White T, Chade AR, Zhang J, Liu R, Roman RJ, Lee JW, Kyle PB, Solberg-Woods L, **Garrett MR**. Nephron Deficiency and Predisposition to Renal Injury in a Novel One-Kidney Genetic Model. J Am Soc Nephrol. 2014 Oct 27;PubMed PMID: 25349207.

3. **Research Area 3**: The investigation, identification, and characterization of genes involved in monogenic and polygenic diseases using of genetic analyses, cutting-edge genomic technologies, and knockout animal models. I have been involved in the several project that have utilized animal models to identify single gene defects and the functional consequence of loss a gene (knockout animals) in complex disease, both as the primary investigator and co-investigator. For example, my laboratory was first to report that loss of nuclear hormone receptor NR4A1 can contribute to progressive kidney injury, specifically through an immune-mediated mechanism. This study is an example of my laboratory's thoroughness as the work spanned from physiological characterization (blood pressure, renal hemodynamics, histological analysis) to whole transcriptome analysis to bone marrow cross transplantation studies and *in vitro* assays.

   a. Joe B, Saad Y, Dhindaw S, Lee NH, Frank BC, Achinike OH, Luu TV, Gopalakrishnan K, Toland EJ, Farms P, Yerga-Woolwine S, Manickavasagam E, Rapp JP, Garrett MR, Coe D, Apte SS, Rankinen T, Pérusse L, Ehret GB, Ganesh SK, Cooper RS, O'Connor A, Rice T, Weder AB, Chakravarti A, Rao DC, Bouchard C. Positional identification of variants of Adamts16 linked to inherited hypertension. Hum Mol Genet. 2009 Aug 1;18(15):2825-38. PMID: 19423552; Central PMCID: PMC2706685.

   b. Johnson AC, Lee JW, Harmon AC, Morris Z, Wang X, Fratkin J, Rapp JP, Gomez-Sanchez E, **Garrett MR**. A mutation in the start codon of γ-crystallin D leads to nuclear cataracts in the Dahl SS/Jr-Ctr strain. Mamm Genome. 2013 Apr;24(3-4):95-104. PMID: 23404175; PubMed Central PMCID: PMC3628938.

   c. Westbrook L, Johnson AC, Regner KR, Williams JM, Mattson DL, Kyle PB, Henegar JR, **Garrett MR**. Genetic susceptibility and loss of Nr4a1 enhances macrophage-mediated renal injury in

CKD. J Am Soc Nephrol. 2014 Nov;25(11):2499-510. PMID: 24722447; PubMed Central PMCID: PMC4214519.

d. Muroya Y, Fan F, Regner KR, Falck JR, **Garrett MR**, Juncos LA, Roman RJ. Deficiency in the Formation of 20-Hydroxyeicosatetraenoic Acid Enhances Renal Ischemia-Reperfusion Injury. J Am Soc Nephrol. 2015 Feb 2; PMID: 25644108.

4. **Research Area 4**: Linkage analysis and positional cloning of genetic factors causative to hypertension using the Dahl salt-sensitive (S) rat. My early research reported some of the first systematic genetic analyses of blood pressure regulation using rodent models (1997-2000). These studies employed extensive linkage analyses using multiple F2 populations generated from the S rat and other strains to map genetic loci associated with high blood pressure. Subsequently, significant effort was aimed at generating a large number of congenic strains to narrow and focus the location of the causative genetic elements that underlie each locus. This work was instrumental in highlighting the extent of genetic complexity of a quantitative trait such as blood pressure, including the large number of genes/genomic regions that control blood pressure, the role of gene X gene interactions, and established many small genomic regions contained multiple genetic factors that control the trait (both positive and negative regulators). This body of work laid a strong foundation for many others that later came into the field.
    a. Rapp JP, **Garrett MR**, Deng AY. Construction of a double congenic strain to prove an epistatic interaction on blood pressure between rat chromosomes 2 and 10. J Clin Invest. 1998 Apr 15;101(8):1591-5. PMID: 9541488; PubMed Central PMCID: PMC508739.
    b. **Garrett MR**, Dene H, Walder R, Zhang QY, Cicila GT, Assadnia S, Deng AY, Rapp JP. Genome scan and congenic strains for blood pressure QTL using Dahl salt-sensitive rats. Genome Res. 1998 Jul;8(7):711-23. PubMed PMID: 9685318.
    c. **Garrett MR**, Zhang X, Dukhanina OI, Deng AY, Rapp JP. Two linked blood pressure quantitative trait loci on chromosome 10 defined by dahl rat congenic strains. Hypertension. 2001 Oct;38(4):779-85. PubMed PMID: 11641286.
    d. **Garrett MR**, Meng H, Rapp JP, Joe B. Locating a blood pressure quantitative trait locus within 117 kb on the rat genome: substitution mapping and renal expression analysis. Hypertension. 2005 Mar;45(3):451-9. PubMed PMID: 15655120.

D. RESEARCH SUPPORT

Ongoing Research Support

2013/08/01-2018/07/31
P30 GM103328, NIGMS
STOCKMEIER, CRAIG (PI)
Center for Psychiatric Neuroscience (CPN)
The overall goal of the project is to provide administrative and research core support to CPN investigators for the generation of knowledge of basic neurobiology and clinical psychiatry.
Role: Co-Investigator/Core Director

2014/08/01-2018/07/31
68105030618, Robert M Hearin Foundation
GARRETT, MICHAEL (PI)
Medical Student Research Program (MSRP)
The purpose of the MSRP is to foster the development of medical students into physician scientist. The program consists of 10-week “Summer of Research” portion M1-M2 (n=25 student per year X $3500 stipend), followed by “Full Option” for M2-M4 (n=15 per year X $5000 stipend)
Role: PI

2013/07/01-2018/06/30
P20GM103476 , NIGMS

ELASRI, MOHAMED (PI)
IDeA Networks of Biomedical Research Excellence (INBRE)
The overall goal of the project is to provide administrative and research core support to MS-INBRE investigators interested in molecular genetics and genomic technologies.
Role: Co-Investigator/Core Director (sub-contract)

2013/09/05-2018/04/30
1P20GM104357, NIGMS
HALL, JOHN (PI)
Cardiorenal and Metabolic Diseases Research Center
This application requests support for a Center of Biomedical Research Excellence focused on cardiovascular, renal and metabolic diseases.
Role: Co-Investigator/Core Director

2009/08/01-2015/07/31
R01-HL094446, NIH/NHLBI
GARRETT, MICHAEL (PI)
Genetics of Renal End Organ Damage in Hypertension
The major goal is to identify the gene(s) that promote glomerulosclerosis and renal interstitial fibrosis observed in the Dahl salt-sensitive rat (S), a model of hypertension induced renal disease.
Role: PI

Completed Research Support

2010/11/01-2011/10/31
UMMC-IRSP, UMMC-Intermural Research Support Program (IRSP)
GARRETT, MICHAEL (PI)
Renal Hemodynamics in New Genetic Model Born with a Single Kidney
The major goal is to characterize long-term cardiovascular and renal complications of being born with a single kidney versus undergoing a nephrectomy early in postnatal or adulthood.
Role: PI

2009/07/01-2011/06/30
P50 DK079306, NIH
AVNER, ELLIS (PI)
Characterizing a Novel Model of Proteinuria using Nr4a1 Nuclear Receptor Knockout Rats
The major goal of this projects is to characterize the onset and progression of proteinuria and renal injury in the Nr4a1-/-knockout (as compared to the FHH), as well as, to investigate the mechanism by which this nuclear receptor (Nr4a1) plays a role in renal injury.
Role: Pilot Grant PI

2007/07/01-2009/06/30
0755290B, AHA-GIA
GARRETT, MICHAEL (PI)
A Positional Cloning and Functional Approach to Identify a Renal Disease Gene Potentially Linked to Fibrosis
The major goal is to identify a gene(s) involved in susceptibility to develop renal damage using congenic strain analysis and to characterize differences in primary cultures of proximal tubular cells derived from the a renal protective congenic strain compared to the Dahl S parental.
Role: PI