

BIOGRAPHICAL SKETCH

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NAME Winston Campbell (Cam) Patterson		POSITION TITLE Earnest Craige Distinguished Professor of Cardiovascular Medicine	
eRA COMMONS USER NAME WINSTON PATTERSON			
EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)			
INSTITUTION AND LOCATION	DEGREE (if applicable)	YEAR(s)	FIELD OF STUDY
Vanderbilt University, Nashville, TN	B.A.	1985	Psychology
Emory University School of Medicine, Atlanta, GA	M.D.	1989	Medicine

A. Positions and Honors.

- 1989-1993 Resident and Chief Resident, Internal Medicine, Emory University Affiliated Hospitals
 1993-1996 Research Fellow (Mentor: Edgar Haber), Cardiovascular Biology Laboratory, Harvard University
 1996-2000 Assistant and Associate Professor of Medicine (Cardiology) and Pharmacology & Toxicology, University of Texas Medical Branch
 2000-present Professor of Medicine (Cardiology), Pharmacology, and Cell and Developmental Biology (with Tenure), University of North Carolina at Chapel Hill
 2000-present Director, Carolina Cardiovascular Biology Center, University of North Carolina at Chapel Hill
 2005-present Division Chief, Cardiology, Department of Medicine, University of North Carolina at Chapel Hill

1985 Summa Cum Laude Graduate, Vanderbilt University; 1981-85 Harold Sterling Vanderbilt Scholar, Vanderbilt University; 1992 Alpha Omega Alpha Medical Honor Society, Emory University School of Medicine; 1994-96 National Research Service Award, National Institutes of Health; 1997 First Prize, Ignacio Chavez Young Investigator Award, Interamerican Society of Cardiology; 1997-02 Clinical Investigator Award, National Institutes of Health; 1998 Finalist, Young Investigator Award, American College of Cardiology; 2000-pres, Editorial Board, *Circulation* and *Circulation Research*; 2002, Established Investigator Award, American Heart Association; 2003, American Society of Clinical Investigation; 2003, Ruth and Philip Hettleman Prize for Artistic and Scholarly Achievement, University of North Carolina; 2003-pres, Editorial Board, *ATVB* and *JMCC*; 2004, Association of University Cardiologists

B. Selected peer-reviewed publications (in chronological order; selected from 117).

1. Wu Y, **Patterson C**. The human KDR/FLK-1 gene contains a functional initiator element that is bound and transactivated by TFII-I. *Journal of Biological Chemistry*, **274**: 3207-3214 (1999).
2. Ballinger C, Connell P, Yin LY, Wu, Hu Z, Thompson LJ, **Patterson C**. Identification of CHIP, a novel Tetratricopeptide repeat-containing protein that interacts with heat shock proteins and negatively regulates chaperone function. *Molecular and Cellular Biology*, **19**: 4535-4545 (1999).
3. Ruef J, Meshel A, Hu Z, Horaist C, Runge MS, Ballinger CA, Thompson LJ, Dumont JA, Runge MS, **Patterson C**. Flavopiridol inhibits vascular smooth muscle cell proliferation in vitro and neointimal formation in vivo after carotid injury in the rat. *Circulation*, **100**: 659-665 (1999).
4. Connell P, Ballinger CA, Jiang J, Wu Y, Höhfeld J, **Patterson C**. Regulation of heat shock protein-mediated protein triage decisions by the co-chaperone CHIP. *Nature Cell Biology*, **3**: 93-96 (2001).
5. Meacham GC, **Patterson C**, Zhang W, Younger JM, Cyr DM. The Hsc70 co-chaperone CHIP targets immature CFTR for proteasomal degradation. *Nature Cell Biology*, **3**: 100-105 (2001).
6. Moon S-K, Thompson LJ, Madamanchi N, Ballinger SW, Runge MS, **Patterson C**. Aging, oxidative responses and proliferative capacity in cultured mouse aortic smooth muscle cells. *American Journal of Physiology*, **280**: H2779-H2789 (2001).

7. Thompson LJ, Jiang J, Madamanchi, N, Runge MS and **Patterson C**. PTP ϵ , a tyrosine phosphatase expressed in the endothelium, negatively regulates endothelial cell proliferation. *American Journal of Physiology*, **281**: 396-403 (2001).
8. Aitsebaomo J, Kingsley-Kallesen M, Wu Y, Quertermous T, **Patterson C**. Vezf1/DB1 is an endothelial cell-specific transcription factor that regulates expression of the endothelin-1 promoter. *Journal of Biological Chemistry*. **276**: 39197-205 (2001).
9. Jiang J, Ballinger CA, Wu Y, Dai Q, Cyr DM, Höfeld J, **Patterson C**. CHIP is a U-box-dependent E3 ubiquitin ligase: identification of Hsc70 as a target for ubiquitylation. *Journal of Biological Chemistry*, **276**: 42938-44 (2001).
10. Citri A, Alroy I, Lavi S, Rubin C, Xu Y, Grammatikakis N, **Patterson C**, Neckers L, Fry DW, Yarden Y. Drug-induced ubiquitylation and degradation of ErbB receptor tyrosine kinases: Implications for cancer therapy. *EMBO Journal* **21**: 2407-2417 (2002).
11. Asimakis GK, Lick S, **Patterson C**. Post-ischemic recovery of contractile function is impaired in SOD2 (+/-) but not SOD1 (+/-) mouse hearts. *Circulation*, **105**: 981-986 (2002).
12. **Patterson C**. A new gun in town: the U-box is a ubiquitin ligase domain. *Science*, **116**: 1-4 (2002).
13. Smyth S and **Patterson C**. Tiny dancers: the integrin-growth factor nexus in angiogenic signaling. *Journal of Cell Biology*, **158**: 17-21 (2002).
14. Xu W, Marcu M, Yuan X, Mimnaugh E, **Patterson C**, Neckers L. The chaperone-dependent E3 ubiquitin ligase CHIP mediates a novel degradative pathway for c-ErbB2/Neu. *Proceedings of the National Academy of Sciences USA*, **99**: 12847-12852 (2002).
15. Lenihan D, Osman A, Sriram V, Aitsebaomo J, **Patterson C**. Evidence for association of coronary sinus levels in hepatocyte growth factor and collateralization in human coronary disease. *American Journal of Physiology*, **284**: 1507-1512 (2003).
16. Jackson LF, Qiu TH, Sunnarborg SW, Chang A, Zhang C, **Patterson C**, Lee DC. Defective valvulogenesis in HG-EGF and TACE-null mice is associated with aberrant BMP signaling. *EMBO Journal*, **22**: 2704-2716 (2003).
17. Kampinga HH, Kanon B, Salomons FA, Kabakov AE, **Patterson C**. Overexpression of the co-chaperone CHIP enhances in vivo Hsp70 folding activity. *Molecular and Cellular Biology*, **23**: 4948-4958 (2003).
18. Wu Y, Moser M, Bautch VL, **Patterson C**. HoxB5 is an upstream transcriptional switch for differentiation of vascular endothelium from precursor cells. *Molecular and Cellular Biology*, **23**: 5680-5691 (2003).
19. Moser M, Binder O, Wu Y, Aitsebaomo J, Ren R, Bode C, Bautch VL, Conlon FL, **Patterson C**. BMPER, a novel endothelial cell precursor-derived protein, antagonizes BMP signaling and endothelial differentiation. *Molecular and Cellular Biology*, **23**: 5664-5679 (2003).
20. Dai Q, Zhang C, Wu Y, McDonough H, Whaley RA, Godfrey V, Li H-H, Madamanchi N, Xu W, Neckers L, Cyr D, **Patterson C**. CHIP activates HSF1 and confers protection against apoptosis and cellular stress. *EMBO Journal*, **22**(20): 5446-58 (2003).
21. Aitsebaomo J, Wennerberg K, Der CJ, Zhang C, Kedar V, Kingsley-Kallesen ML, Zeng G.-Q, **Patterson C**. p68RacGAP is a novel GTPase-activating protein that interacts with Vezf1 and modulates endothelial cell capillary formation. *Journal of Biological Chemistry*, **279**: 17963-17972 (2004).
22. Moser M, Knoth R, Bode C, **Patterson C**. LE-PAS, a novel Arnt-dependent HLH-PAS protein is expressed in limbic tissues and transactivates the CNS midline enhancer element. *Molecular Brain Research*, **128**: 141-149 (2004).
23. Kedar V, McDonough H, Arya R, Li H-H, Rockman HA, **Patterson C**. Muscle-specific Ring Finger 1 is a bona fide ubiquitin ligase that degrades cardiac troponin I. *Proceedings of the National Academy of Sciences USA*, **101**: 18135-18140 (2004).
24. Arya R, Kedar V, Hwang JR, McDonough H, Li H-H, Taylor J, **Patterson C**. Muscle ring finger protein-1 inhibits PKC ϵ activation and prevents cardiomyocyte hypertrophy. *Journal of Cell Biology*, **167**: 1147-1159 (2004).
25. Li H-H, Kedar V, Zhang C, McDonough H, Arya R, Wang D-Z, **Patterson C**. Atrogin-1/muscle atrophy F-box inhibits calcineurin-dependent cardiac hypertrophy by participating in an SCF ubiquitin ligase complex. *Journal of Clinical Investigation*, **114**: 1058-1071 (2004).
26. Roberts D, Stanford W, **Patterson C**, Bautch VL. A vascular gene trap defines RasGRP3 as an angiogenesis-regulated gene required for the endothelial response to phorbol esters. *Molecular and Cellular Biology*, **24**: 10515-10528 (2004).
27. Younger, JM, Ren H-Y, Chen L, Fan, C-Y, Fields A, **Patterson C**, Cyr DM. A foldable CFTR Δ F508 biogenic intermediate accumulates upon inhibition of the hsc70/CHIP E3 ubiquitin ligase. *Journal of Cell Biology*, **164**: 1075-1085 (2004).
28. Zhang C, Xu Z, He X-R, Michael LH, **Patterson C**. CHIP, a co-chaperone/ubiquitin ligase that regulates protein quality control, is required for maximal cardioprotection after myocardial infarction in mice. *American Journal of Physiology*, **288**: 2836-2842 (2005).
29. Hwang JR, Zhang C, **Patterson C**. CHIP facilitates degradation of apoptosis signal-regulating kinase 1 and inhibits ASK1-dependent apoptosis. *Cell Stress and Chaperones*, in press (2005).

C. Research Support

Ongoing Research Support

R01 HL61656 (Patterson) 7/1/99 - 6/30/09
NIH/NHLBI
Nuclear signaling in endothelial growth and angiogenesis

The major goal of this project is to examine the molecular mechanisms that regulate endothelial cell differentiation and cell type-specific gene expression. This project employs several models, including characterization of KDR/flk-1 gene regulation as a model for understanding early transcriptional events in endothelial cell differentiation and angiogenesis.

R01 GM61728 (Patterson) 6/1/01 - 5/31/05
NIH/NIGMS
CHIP: link between chaperone & proteasome system

The major goal of this project is to characterize the function of a ubiquitin ligase recently discovered in our laboratory, CHIP. Biochemical studies will determine how CHIP is regulated to affect the chaperone and proteasome systems; cellular assays will test the signaling pathways altered by CHIP and determine how CHIP modulates cell behavior in response to stress; and physiologic studies will characterize the role of CHIP in transgenic mice overexpressing CHIP, as well as knockout mice deficient in CHIP.

R01 HL 65619 (Patterson) 7/1/01 – 6/30/05
NIH/NHLBI
Myocyte signaling, and the response to stress

The major goal of this proposal is to characterize the functions of cardiac specific atrogenes belonging to the MuRF and atrogin family. Specifically, we intend to define the downstream targets and signaling pathways affected by these proteins, and to determine their roles in cardiac hypertrophy..

P01 AG024282 (Clemmons/Patterson) 8/01/04 – 7/30/09
NIH/NIA
Chaperones, ROS systems, & IGF1: Roles in vascular aging

The major goal of this proposal is explore interactions between oxidative events, IGF-1 signaling, and molecular chaperones as the 3 major pathways hypothesized to regulate vascular aging. Projects will take complementary cellular and physiologic approaches to address this problem. Dr. Patterson is co-principal investigator of the PPG and leader of Project #2.

R01 HL72347 (Patterson) 9/30/02 – 7/31/06
NIH/NHLBI
Carolina cardiopulmonary gene expression services

Although this grant uses the R01 mechanism, it funds a core facility dedicated to providing NHLBI-funded investigators at the University of North Carolina–CH with comprehensive microarray services that complement their existing research programs.

Established Investigator – 0340107N (Patterson) 1/1/03 – 12/31/07
American Heart Association
Transcriptional and signaling events mediated by VEZF1/DB1 in the vascular endothelium

The goal of this project is to define further the cellular and physiologic role of VEZF1/DB1 in the regulation of endothelium-dependent events in vitro and in vivo using molecular, genetic, and genomic approaches.

Clinical Scientist Award – 0340107N (Patterson) 1/1/04 – 12/31/08
Burroughs Wellcome Foundation
Oxidative Profiles In Cardiovascular Diseases

The goal of this project is to create a gene expression and proteomic profile that is characteristic of patients with or without coronary artery disease by performing microarray and mass spec profiling of blood samples from patients at risk of coronary artery disease.

H75/CCH424677-01 (Patterson)

6/6/05 – 6/5/06

CDC

Racial Disparities in Cardiovascular Disease

The major goal of this project is to develop programs and evaluations for understanding the causes and consequences of racial and ethnic disparities in cardiovascular care in the state of North Carolina, and to develop intervention strategies to reduce these disparities.

H75/CCH424675-01 (Patterson)

6/6/05 – 6/5/06

CDC

Genomic Data on Cardiovascular Disease

The major goal of this project is to develop programs and evaluations to understand the biological and ethical implications of gathering genomic data on cardiovascular disease to benefit the population of North Carolina.

P01HD031921 (Harris)

10/1/05 – 9/30/10

NIH/NICHD

National Study of Adolescent Health

The National Longitudinal Study of Adolescent Health (Add Health) is a nationally representative study that explores the causes of health-related behaviors of adolescents in grades 7 through 12 and their outcomes in young adulthood. Add Health seeks to examine how social contexts (families, friends, peers, schools, neighborhoods, and communities) influence adolescents' health and risk behaviors. Wave IV of Add Health focuses on 15,000 individuals within this cohort who are now young adults, and who will undergo evaluations for cardiovascular and metabolic risk, including genetic interrogations. Dr. Patterson is the director of the Scientific Core of this PPG.