Petra Rocic, PhD

Work Address Department of Physiology and Pharmacology
Sam Houston State University
College of Osteopathic Medicine
[redacted]

PROFESSIONAL EXPERIENCE

5/2020-present	Associate Professor, Department of Physiology and Pharmacology, Sam Houston State University, College of Medicine, Conroe, TX, USA
11/2013-5/2020	Associate Professor, Department of Pharmacology, New York Medical College (NYMC), Valhalla, NY, USA
06/2019-present	Grant Writer, ScienceDocs.com
05/2008-11/2013	Assistant Professor, Department of Biochemistry and Molecular Biology, University of South Alabama (USA), Mobile, AL, USA
05/2007-05/2008	Assistant Professor, Department of Integrative Medical Sciences, Northeast Ohio Medical University (NEOMED), Rootstown, OH, USA
11/2005-05/2007	Instructor, Department of Physiology, Louisiana State University Health Sciences Center (LSUHSC), New Orleans, LA, USA
05/2003-11/2005	Postdoctoral Research Fellow, Department of Physiology, Louisiana State University Health Sciences Center, New Orleans, LA, USA; Advisor: William M. Chilian
09/2001-05/2003	Postdoctoral Research Fellow, Department of Medicine, Division of Cardiology, Emory University, Atlanta, GA, USA; Advisor: Kathy K. Griendling

EDUCATION

2001	Doctor of Philosophy, Physiology and Biophysics, University of Alabama at Birmingham, Birmingham, AL, USA; Advisor: Pamela A. Lucchesi, Ph.D. *Dissertation Title*: "Regulation of angiotensin II-induced vascular smooth muscle cell protein synthesis by multiple signaling cascades", August 2001
1997	Bachelor of Arts, Biology, Oberlin College, Oberlin, OH, USA, May 1997

TEACHING EXPERIENCE

- 10 years experience with medical curriculum development and delivery via different methods (including didactic lecture, flipped classroom, case-based learning, interactive large and small group discussion) and using different methods (including Blackboard, Canvas, Zoom)
- 10 years experience writing USMLE style exam questions (and assisting faculty in this area)
- 10 years experience mentoring MD and PhD students in scholarly and laboratory activities
- Experience with M1 and M2 systems-based curriculum design

COURSE DIRECTOR/SECTION HEAD

University of South Alabama

2011-2013 Fundamentals of Basic Medical Science II (graduate) – Course director for cardiovascular and renal blocks (organization of lectures, case studies, review sessions, quizzes and exams)

LECTURER (DIDACTIC)

Sam Houston State University

2020-present Scientific Foundations 1 - Muscle Physiology

New York Medical College

2020-present Cardiovascular Pharmacology (graduate) – Therapies for migraine
2017-present Molecular pharmacology (graduate) - microRNAs in cardiovascular disease
2014-present Medical Pharmacology (medical, class size ~200 students) – Antihypertensive drugs (9 hours), Therapies for Heart Failure (9 hours), using the Blackboard teaching platform
2015-present Cardiovascular Pharmacology (graduate) – New therapies for the treatment of heart failure
2014-present Endocrine Pharmacology (graduate) – Metabolic syndrome: Mechanisms and pharmacological therapies

University of South Alabama

2013-2013 Medical curriculum year I (class size ~70 students): Cardiovascular Block – Metabolic syndrome, atherosclerosis, myocardial blood flow, angina and collateral circulation
2009-2013 Fundamentals of Basic Medical Science II (graduate) – Complete cardiovascular and renal physiology based on medical physiology course content
2008-2012 Medical Biochemistry – Signal transduction: Angiotensin II, insulin and aldosterone signaling in heart failure and metabolic syndrome
2008-2010 Research Design and Methods (graduate) – Experimental design and formulation of hypothesis
2009-2011 DREAM (Diversity Recruitment and Enrichment for Admission into Medicine Program) – Biochemistry: Endocrine block

Louisiana State University Health Sciences Center

2006-2007 Biological Systems (graduate) - Cardiovascular and endocrine physiology
2006-2007 Advanced pathophysiology (School of Nursing) – Cardiovascular pathophysiology,

ACTIVE LEARNING

Sam Houston State University

2020-present Scientific Foundations 1 – Muscular dystrophy, Urinary tract infections

New York Medical College

2013-present Medical Pharmacology, Clinical case studies (case-based, flipped classroom/TBL learning; 8 cases per year, 2h each, ~20 students per faculty facilitator, split in working groups of 5-6 students)

University of South Alabama

2011-2013 Clinical Case Studies – Hypertension and heart failure (medical student presentations followed by faculty questions)

2011-2013 Fundamentals of Basic Medical Science II (graduate) - Cardiovascular and renal physiology block quizzes and associated review sessions, TBL modules

2011-2013 Medical Biochemistry – Case study (on-line self-study module): Myocardial infarction

2010-2013 Fundamentals of Basic Medical Science II (graduate) – Restenosis, heart failure and hypertension case studies (interactive discussion sessions)

2008-2013 Literature Reports (interactive journal club for 1st year grauate students)

Louisiana State University Health Sciences Center

2004-2007 Medical Physiology and Anatomy: cardiovascular anatomy and function laboratory for 1st year medical students

INDIVIDUAL MENTORING

Tracy Dodd	2009-2012, University of South Alabama, Ph.D. awarded June, 2012
Rebecca Hutcheson	2010-2013, University of South Alabama, Ph.D. awarded October, 2013
Patricia Villalta	2009-2013, University of South Alabama, Ph.D. awarded November, 2013
Luke Wiggins	2010-2012, University of South Alabama, M.D. with Research Honors awarded May, 2012
Russell Terry	2011-2013, University of South Alabama, M.D. with Research Honors awarded May, 2013
Ian Hunter	2014-2016, New York Medical College, M.S. (with Dissertation Research) in Integrative Biomedical Sciences, awarded June 2016
Amanda Soler	2014-2018, New York Medical College, Ph.D. awarded October, 2018
Gregory Joseph	2015-2018, New York Medical College, Ph.D. awarded June 2018

Summer M.D. Student Research

Robert E. Levy (Summer 2012), Anna Crutchfield (Summer 2013), Martha O'Hea (Summer 2013)

High School and Undergraduate Student Research

Nihaar Parikh (2015-2016, High School Honors Research), Melanie Anaya (2015-2016, High School Honors Research), Jessica Correra (Summer 2016, High School Summer Research), Tyana Brown (Undergradate Research, Summer 2016-2019), Corinna Lozano (2017-2020, High School Honors Research)

Dissertation committees

University of South Alabama: Samar Swedan (Ph.D. May, 2010), Rebecca Torres (Ph.D. October, 2010), Abdallah AlZoubi (Ph.D. March, 2013), Kendra Reed (Ph.D. awarded September, 2014), Pierre Kadeba (Ph.D. awarded May, 2015), Jared McLendon (Ph.D. awarded June, 2015)

New York Medical College: Vidi Daghia (Ph.D. awarded June, 2017), Elizabeth Newman (Ph.D. awarded December, 2018), Ankit Gilani (Ph.D. awarded March, 2020)

CURRICULUM DELIVERY TECHNIQUES: Blackboard, Canvas, Zoom, PowerPoint, didactic lectures, flipped classroom, team-based learning (TBL), case-based learning, interactive small and large group presentation and/or discussion, audience response systems (clickers)

RESEARCH EXPERIENCE

Current Research Interests

Patophysiological consequences of metabolic syndrome in the cardiovascular system:

- 20-HETE as a biomarker for Covid-19 disease severity, development of post-Covid-19 syndrome and inhibition of lasting immunity against SARS-Cov2
- Beneficial effect of 20-HETE antagonists on myocardial infarct size, post-infarct cardiac remodeling and failure and patient survival, with emphasis on obesity and metabolic syndrome
- Mechanistic effects of bariatric surgery and lipectomy on cardiac and vascular function in obesity and the metabolic syndrome
- Effect of environmental pollution (emphasis on air pollution) on cardiovascular function in obesity and metabolic syndrome
- Beneficial effects of 20-HETE antagonists on isolated systolic hyprtension in metabolic syndrome and obesity

PENDING GRANT SUPPORT

20-HETE in the regulation of myocardial remodeling and function after myocardial infarction
PI: Petra Rocic
Agency: NIH NHLBI
Type: RO1 $375,000 Annual Direct Cost
The goal of this project is to identify 20-HETE-dependent mechanisms responsible for infarct enlargement and eccentric left ventricular remodeling after myocardial infarction in rat models and human patients. Moreover, we will determine the ability of 20-HETE antagonists, administered at onset of reperfusion, to minimize infarct size and prevent detrimental eccentric remodeling and left ventricular failure, as well as decrease mortality. Findings in animal models will be correlated with observations in patients enrolled in our ongoing clinical study.

Vevo 3100 Ultrasound and LAZR-X Photoacoustic Imaging System
PI: Petra Rocic
Agency: NIH NHLBI
Type: HEI (High-end Instrument Grant)
Period: 4/1/2020 $928,345 Annual Direct Cost
The goal of this application is to obtain funding for the purchase of the VisualSonics Vevo 3100/LAZR-X echocardiography system.

COMPLETED GRANT SUPPORT

Mechanistic Basis of miR-145-mediated Restoration of Coronary Collateral Growth
PI: Petra Rocic
Agency: NIH NHLBI
Type: RO1
Period: 4/1/2015-3/31/2020 $250,000 Annual Direct Cost
The goal of this project is to understand why normalization of miR-145 levels in vascular smooth muscle cells (VSMC) resulted in complete recovery of coronary collateral growth (CCG) in metabolic syndrome rats. We hypothesize that restoration of normal VSMC miR-145 levels in the late stage of CCG in the metabolic syndrome restores CCG by normalizing ECM composition, reducing inflammation and restoring endothelial function. Therefore, we will determine whether: 1) VSMC-specific miR-145 delivery normalizes ECM remodeling during CCG in the metabolic syndrome, 2) VSMC-specific miR-145 delivery and normalized ECM remodeling ameliorate excessive inflammation (inflammatory cell infiltration, cytokine, ROS and MMP production from these cells) during CCG in the metabolic syndrome, and 3) VSMC-specific miR-145 delivery and normalized ECM remodeling restore endothelial function during CCG in the metabolic syndrome.

Mechanistic Basis of miR-145-mediated Restoration of Coronary Collateral Growth
PI: Petra Rocic
Agency: NIH NHLBI
Type: Diversity Supplement (for Gregory Joseph)
Period: 4/1/2017-05/31/2018 $45,800 Annual Direct Cost
The goal of this supplement is to provide support for Gregory Joseph, who is completing his Ph.D. dissertation work in the PI's laboratory.

20-HETE Increases Large Artery Stiffness and Systolic Blood Pressure in Metabolic Syndrome
PI: Amanda Soler
Spnsor/Mentor: Petra Rocic
Agency: NIH NHLBI
Type: Ruth L. Kirschstein National Research Service Award Individual Predoctoral Fellowship to Promote Diversity in Health-Related Research
Period: 8/1/2017-7/31/2018 $30,537 Annual Direct Cost
The goal of this fellowship is to support dissertaion research for Amanda Soler. The hypothesis of the proposal matches the main hypothesis of her dissertation, that elevated 20-HETE in metabolic syndrome increases MMP12 activation, large artery stiffness and systolic blood pressure.

Regulation of Coronary Collateral Growth in the Metabolic Syndrome
PI: Petra Rocic
Agency: NIH NHLBI
Type: RO1
Period: 8/1/2008-7/31/2015 $250,000 Annual Direct Cost
The goals of this project are: 1) to establish myocardial oxidative stress and redox-sensitive signaling profiles for normal vs. metabolic syndrome during repetitive ischemia-induced coronary collateral growth, 2) to determine the effect of Ang II on coronary collateral growth, myocardial oxidative stress and redox-dependent signaling in vivo in normal vs. metabolic syndrome animals, and 3) to establish treatment paradigms (AT1R blockers, NAD(P)H oxidase inhibition, dominant negative and/or constitutively active signaling molecule expression) to achieve maximal restoration of coronary collateral growth in the metabolic syndrome.

Regulation of Coronary Collateral Growth in the Metabolic Syndrome
PI: Petra Rocic

Agency: NIH NHLBI
Type: ARRA Supplement
Period: 8/1/2009-7/31/2011 $48,918 Annual Direct Cost
The goal of this project was to obtain repeated measurments of oxidative stress in anesthethised rats in vivo in order to reduce the number of animals used and be able to use each animal as its own internal control. This project also allowed for hiring of new personel and purchase of US-made equipment (rodent anesthesia station).

Regulation of Coronary Collateral Growth in the Metabolic Syndrome
PI: Petra Rocic
Agency: NIH NHLBI
Type: Diversity Supplement
Period: 8/1/2009-8/15/2012 $36,500 Annual Direct Cost
The goal of this supplement was to provide support for Dr. Tracy Dodd, who graduated with a Ph.D. in June, 2012.

microRNA-mediated Regulation of Coronary Collateral Growth in the Metabolic Syndrome
PI: Rebecca Hutcheson
Sponsor: Petra Rocic
Agency: AHA
Type: Predoctoral Fellowship
Period: 07/01/2011-06/30/2013 $28,460 Annual Direct Cost
The goals of this project were to determine if miR-143/145 were involved in the regulation of vascular smooth muscle phenotype and coronary collateral growth in normal and metabolic syndrome rats.

Reactive Oxygen Species and Redox- Sensitive Signaling in Coronary Collateral Growth
PI: Petra Rocic
Agency: AHA (National)
Type: SDG
Period: 01/01/2006-12/31/2009 $65,000 Annual Direct Cost
The goals of this project were to determine the effect of mimicking the human metabolic syndrome on ROS concentrations, coronary collateral development and the activation of critical redox-dependent signaling pathways in a rat model of repetitive ischemia *in vivo* and to determine the effect of Ang II on ROS-concentration, coronary collateral development and critical redox-dependent signaling pathway activation *in vivo*, under both normal and pathological conditions.

Cell-Specific ROS Regulation of Coronary Collateral Growth
PI: Rocic
Agency: NIH
Type: COBRE
Period: 07/01/2006-06/30/2007 $180,000 Annual Direct Cost
The goal of this project was to determine the cell type(s) that is (are) the most sensitive to the effects of alterations in ROS concentrations in the rat model of coronary collateral growth, and to determine whether normalization of ROS levels in this cell type(s) could rescue coronary collateral growth in the rat model.

"Redox Regulation of Angiogenesis"
PI: Petra Rocic
Sponsor: William M. Chilian
Agency: AHA
Type: Postdoctoral fellowship
Period: 07/01/2004-06/30/2005 $31,500 Annual Direct Cost

The goal of this project was to determine the boundaries of the redox window that allows for angiogenesis in response to VEGF *in vitro* and for coronary collateral development *in vivo*, as well as to determine redox-dependent signaling pathways involved in angiogenesis.

"Redox-Sensitivity of Translation Initiation"
PI: Petra Rocic
Sponsor: Kathy K. Griendling
Agency: AHA
Type: Postdoctoral fellowship
Period: 07/01/2002-5/1/2003 $31,500 Annual Direct Cost
The goal of this project was to determine the redox-sensitivity of the intracellular signaling pathways that regulate translation initiation, including PHAS-1, eIF4E, p38 MAPK and Akt, both in cultured vascular smooth muscle cells *in vitro* and in a transgenic mouse model overexpressing components of the vascular NAD(P)H oxidase.

PUBLICATIONS

Abstracts

1. **Rocic P**, Griffin T, McRae C, Lucchesi PA. Increased expression and activation of PYK2 in hypertensive vascular smooth muscle. *FASEB J*. 13:A1008, 1999.

2. **Rocic P**, Griffin T, McRae C, Lucchesi PA. Increased expression and activation of PYK2 in hypertensive vascular smooth muscle. Presented at the UAB Hypertension Retreat, Sandestin, Florida, October 2000.

3. **Rocic P**, Lucchesi PA. PYK2 Antisense inhibits angiotensin II-induced protein synthesis through ERK1/2 MAP kinases and Akt in vascular smooth muscle. *FASEB J*. 15:A488, 2001.

4. Perez J, **Rocic P**, Luchesi PA. A role for PYK2 in PDGF-induced vascular smooth muscle cell proliferation. *Experimental Biology*, 2002.

5. **Rocic P**, Griendling K. Redox-sensitivity of Ang II-dependent translation initiation. *American Heart Association Scientific Sessions, 2002.*

6. **Rocic P**, Chilian WM, Zhang C**.** Src and PI3-kinase play a role in flow-induced dilation of coronary arterioles. *American Heart Association Scientific Sessions, 2004.*

7. **Rocic P**, Kolz C, Toyota E, WM Chilian. Coronary angiogenesis and collateral development require optimal concentrations of superoxide and are associated with Akt and p38 MAP kinase activation. *Keystone Cardiovascular Symposia, 2005.*

8. **Rocic P**, Kolz C, Toyota E, WM Chilian. Coronary angiogenesis and collateral development require optimal concentrations of superoxide. *Experimental Biology, 2005.*

9. **Rocic P**, Kolz C, WM Chilian. Requirement for p38 MAP kinase and optimal superoxide concentrations in coronary collateral growth. *Experimental Biology, 2006.*

10. Reed R, Kolz C, **P Rocic.** Mechanistic basis for disparate effects of Ang II on coronary collateral growth. *Experimental Biology, 2007.*

11. Smith E, Jadhav R, **Rocic P**. Coronary Artery Vascular Smooth Muscle-Specific Contractile Protein Expression in the Metabolic Syndrome. *Experimental Biology, 2009.*

12. Bradley K, **Rocic P**, Weber DS. Determining the Effects of p38 MAPK Inhibition on Matrix Metalloproteinase-9 Expression. *Experimental Biology, 2009.*

13. Dodd T, Wiggins L, Jadhav R, Smith E, **Rocic P**. p38 MAPK-dependent regulation of MMPs during coronary collateral growth. *Experimental Biology, 2010.*

14. Dodd T, Wiggins L, Jadhav R, Smith E, **Rocic P**. MMP regulation during coronary collateral growth in the metabolic syndrome. *Keystone Symposia, 2011.*

15. Dodd T, Jadhav R, Wiggins L, Smith E, Russell JC, **Rocic P**. p38 MAPK-dependent regulation of MMPs during coronary collateral growth. *Experimental Biology, 2011*

16. Hutcheson R, Terry R, Musiyenko A, Smith E, **Rocic P**. microRNA regulation of vascular smooth muscle phenotype and coronary collateral growth. *Experimental Biology, 2011*

17. Dodd T, Wiggins L, Smith E, Musiyenko A, **Rocic P**. Increased MMP8 and 12 activation correlates with elevated endostatin and angiostatin and impaired coronary collateral growth in the metabolic syndrome. *Experimental Biology, 2012*

18. Hutcheson R, Terry R, Musiyenko A, Smith E, **Rocic P**. miR-mediated regulation of coronary collateral growth in the metabolic syndrome. *Experimental Biology, 2012*

19. Weber DS, Rashmi J, Dodd T, Smith E, Bennet J, **Rocic P**. Sustained activation of p38 MAPK and MMP2 and 9 exacerbate neointima formation following vascular injury in metabolic syndrome rat. *Experimental Biology, 2012*

20. Hutcheson R, Terry R, Smith E, Chaplin J, Musiyenko A, Russell JC, Lincoln T, **Rocic P**. Delivery of microRNA-145 restores coronary collateral growth in metabolic syndrome rats. *American Heart Scientific Sessions, 2012*

21. Hutcheson R, Chaplin J, Smith E, Hysell B, Jadhav R, **Rocic P**. Excessive proliferation underlies impaired coronary collateral growth in the metabolic syndrome. *Experimental Biology, 2012*

22. Soler A, Hutcheson B, Hunter I, Falck JR, Schwartzman M, **Rocic P**. 20-HETE antagonist, 20-SOLA, restores coronary colalteral growth in metabolic syndrome. *American Heart Association Council on Hypertension, 2015*

23. Soler A, Hutcheson B, Hunter I, Zhang F, Gotlinger K, Schwartzman ML, Falck JR, **Rocic P**. 20-HETE increases large artery stiffness and systolic blood pressure in metabolic syndrome. *Winter Eicosanoid Conference, 2016.*

24. Joseph G, Hutcheson B, Hunter I, Soler A, Zhang F, Gotlinger K, Falck JR, Schwartzman ML, **Rocic P**. Neutrophil-derived 20-HETE is a major determinant of coronary collateral growth. *Winter Eicosanoid Conference, 2016.*

25. Joseph G, Hutcheson B, Hunter I, Soler A, Zhang F, Gotlinger K, Falck JR, Schwartzman ML, **Rocic P**. Neutrophil-derived 20-HETE is a major determinant of coronary collateral growth. *American*

Heart Scientific Sessions, 2016.

26. Soler A, Hunter I, Joseph G, Hutcheson R, Hutcheson B, Yang J, Zhang F, Joshi SR, Bradford C, Gotlinger KH, Maniyar R, Falck JR, Proctor S, Schwartzman ML, Gupte SA, **Rocic P**. 20-HETE antagonist normalize large artery stiffness and systolic blood pressure in metabolic syndrome. *American Heart Association Council for Hypertension, 2017.*

27. Soler A, Hutcheson B, Yang J, Bradford C, Zhang F, Gotlinger K, Schwartzman ML, **Rocic P**. Intra-Abdominal Lipectomy and 20-HETE Antagonism Reduce Large Arterial Stiffness and Systolic Blood Pressure in Metabolic Syndrome. *American Heart Association Council for Hypertension, 2017.*

Manuscripts

Peer-reviewed manuscripts

1. **Rocic P**, Govindarajan G, Sabri A, Lucchesi PA. A role for PYK2 in regulation of ERK1/2 MAP kinases and PI3-kinase by Ang II in vascular smooth muscle. *Am. J. Physiol*. 2001;280:C90-C99.

2. **Rocic P**, Lucchesi PA. Downregulation by antisense oligonucleotides establishes a role for the proline-rich tyrosine kinase PYK2 in Ang II-induced signaling in vascular smooth muscle. *J.Biol. Chem*., 2001 Jun 15;276(24):21902-6.

3. **Rocic P**, Griffin TM, McRae CN, Lucchesi PA. Altered PYK2 phosphorylation by ANG II in hypertensive vascular smooth muscle. Am J Physiol Heart Circ Physiol. 2002 Feb;282(2):H457-65.

4. Hanna I, Taniyama Y, **Rocic P**, Szocs K, Griendling KK. NAD(P)H oxidase-derived reactive oxygen species as mediators of Angiotensin II signaling. *Antiox Redox Signal.* (Review) 2002; 4(6):899-914.

5. Seshiah PN, Weber D, **Rocic P**, Valppu L, Taniyama Y, Griendling KK. Angiotensin II stimulation of NAD(P)H oxidase activity: Upstream mediators. *Circ Res.* 2002; 91(5):406-13.

6. **Rocic P**, Seshiah P, Griendling KK. Reactive oxygen species sensitivity of angiotensin II-dependent translation initiation in vascular smooth muscle cells. *J Biol Chem.* 2003 Sep 19;278(38):36973-9.

7. **Rocic P**, Jo H, Lucchesi PA. A role for PYK2 in ANG II-dependent regulation of the PHAS-1-eIF4E complex by multiple signaling cascades in vascular smooth muscle. *Am J Physiol Cell Physiol.* 2003 Dec;285(6):C1437-44.

8. Taniyama Y, Weber DS, **Rocic P**, Hilenski L, Akers ML, Park J, Hemmings BA, Alexander RW, Griendling KK. Pyk2- and Src-dependent tyrosine phosphorylation of PDK1 regulates focal adhesions. *Mol Cell Biol.* 2003 Nov;23(22):8019-29.

9. Weber DS, Taniyama Y, **Rocic P**, Seshiah PN, Dechert MA, Gerthoffer WT, Griendling KK. Phosphoinositide-dependent kinase 1 and p21-activated protein kinase mediate reactive oxygen species-dependent regulation of platelet-derived growth factor-induced smooth muscle cell migration. *Circ Res.* 2004 May 14;94(9):1219-26.

10. Taniyama Y, Ushio-Fukai M, Hitomi H, **Rocic P**, Kingsley MJ, Pfahnl C, Weber DS, Alexander RW, Griendling KK. Role of p38 MAPK and MAPKAPK-2 in angiotensin II-induced Akt activation in vascular smooth muscle cells. *Am J Physiol Cell Physiol.* 2004 Aug;287(2):C494-9.

11. Weber DS, **Rocic P**, Mellis AM, Laude K, Lyle AN, Harrison DG, Griendling KK. Angiotensin II-induced hypertrophy is potentiated in mice overexpressing p22phox in vascular smooth muscle. *Am J Physiol Heart Circ Physiol*. 2005 Jan;288(1):H37-42.

12. Toyota E, Warltier DC, Brock T, Ritman E, Kolz C, O'Malley P, **Rocic P**, Focardi M, Chilian WM. Vascular endothelial growth factor is required for coronary collateral growth in the rat. *Circulation.* 2005 Oct 4;112(14):2108-13.

13. Koshida R, **Rocic P**, Saito S, Kiyooka T, Zhang C, Chilian WM. Role of focal adhesion kinase in flow-induced dilation of coronary arterioles. *Arterioscler Thromb Vasc Biol.* 2005 Dec;25(12):2548-53.

14. **Rocic P**, Rezk B, Lucchesi PA. PPAR-γ Agonists Decrease Hyperhomcysteinemia and Cardiac Dysfunction: New Hope for the Ailing Diabetic Heart? *Am J Physiol Heart Circ Physiol.* 2006 Jul;291(1):H26-8.

15. **Rocic P**, Kolz C, Reed R, Potter B, Chilian WM. Optimal superoxide concentration and p38MAP kinase are required for coronary collateral growth. *Am J Physiol Heart Circ Physiol.* 2007 Jun;292(6):H2729-36.

16. Hattan N, Chilian WM, Park F, **Rocic P**. Restoration of Coronary Collateral Growth In The Zucker Obese Rat: Impact of VEGF and ecSOD. *Basic Res Cardiol.* 2007 May;102(3):217-23.

17. Saitoh S, Kiyooka T, **Rocic P**, Rogers PA, Zhang C, Swafford A, Dick GM, Viswanathan C, Park Y, Chilian WM. Redox-dependent coronary metabolic dilation. *Am J Physiol Heart Circ Physiol.* 2007 Dec;293(6):H3720-5.

18. Reed R, Kolz C, Potter B, **Rocic P**. The mechanistic basis for disparate effects of Ang II on coronary collateral growth. *Arterioscler Thromb Vasc Biol.* 2008 Jan;28(1):61-7.

19. Reed RE, Potter B, Smith E, Jadhav R, Villalta P, Jo H, **Rocic P**. Redox-sensitive Akt and Src Regulate Coronary Collateral Growth in the Metabolic Syndrome. *Am J Physiol Heart Circ Physiol.* 2009 June: 296(6):H1811-21.

20. Carrão AC, Chilian WM, Yun J, Kolz C, **Rocic P**, Lehmann K, van den Wijngaard JP, van Horssen P, Spaan JA, Ohanyan V, Pung YF, Buschmann I. Stimulation of coronary collateral growth by granulocyte stimulating factor: role of reactive oxygen species. *Arterioscler Thromb Vasc Biol.* 2009 Nov;29(11):1817-22.

21. Redox-Dependent Mechanisms in Coronary Collateral Growth: The Redox Window Hypothesis. Yun J, **Rocic P**, Pung YF, Belmadani S, Carrao AC, Ohanyan V, Chilian W. *Antioxid Redox Signal.* 2009 Aug;11(8):1961-74.

22. Rocic B, Bajuk NB, **Rocic P**, Weber DS, Boras J, Lovrencic MV. Comparison of antihyperglycemic effects of creatine and metformin in type II diabetic patients. *Clin Invest Med.* 2009;32(6):E322.

23. Jadhav R, Dodd T, Smith E, Bailey E, Delucia AL, Russell JC, Madison R, Potter BJ, Walsh K, Jo H, **Rocic P**. Angiotensin Type I Receptor Blockade in Conjunction with Enhanced Akt Activation Restores Coronary Collateral Growth in the Metabolic Syndrome. *Am J Physiol Heart Circ Physiol.* 2011:300(5):H1938-49.

24. Pérez J, Torres RA, **Rocic P**, Cismowski MJ, Weber DS, Darley-Usmar VM, Lucchesi PA. PYK2 Signaling is Required for PDGF-dependent Vascular Smooth Muscle Cell Proliferation. *Am J Physiol Cell Physiol.* 2011 Jul;301(1):C242-51.

25. Rocic B, Znaor A, **Rocic P**, Weber D, Vucic-Lovrencic M. Comparison of antihyperglycemic effects of creatine and glibenclamide in type II diabetic patients. *Wien Med Wochenschr.* 2011 Nov;161(21-22):519-23.

26. Dodd T, Jadhav J, Wiggins L, Stewart J, Smith E, Russell JC, **Rocic P**. MMPs 2 and 9 are essential for coronary collateral growth and are prominently regulated by p38 MAPK. *J Mol Cell Cardiol.* 2011:51(6):1015-25.

27. Torres RA, Drake DA, Solodushko V, Jadhav R, Smith E, **Rocic P**, Weber DS. Slingshot isoform-specific regulation of cofilin-mediated vascular smooth muscle cell migration and neointima formation. *Arterioscler Thromb Vasc Biol.* 2011 Nov;31(11):2424-31.

28. Pung YF, **Rocic P**, Murphy MP, Smith RA, Hafemeister J, Ohanyan V, Guarini G, Yin L, Chilian WM. Resolution of Mitochondrial Oxidative Stress Rescues Coronary Collateral Growth in Zucker Obese Fatty Rats. *Arterioscler Thromb Vasc Biol.* 2012:32(2):325-34.

29. **Rocic P**. "Why is coronary collateral growth impaired in type II diabetes and the metabolic syndrome?" *Vascular Pharmacology.* 2012 Nov-Dec;57(5-6):179-86.

30. Hutcheson R, **Rocic P**. The metabolic syndrome, oxidative stress, environment and cardiovascular disease: the great exploration. *Exper. Diabet. Res.* 2012:doi:10.1155/2012/271028.

31. Hutcheson R, Terry R, Chaplin J, Smith E, Musiyenko A, Russell JC, Lincoln T, **Rocic P**. MicroRNA-145 restores contractile vascular smooth muscle phenotype and coronary collateral growth in the metabolic syndrome. *Arterioscler Thromb Vasc Biol*. 2013 Apr;33(4):727-36.

32. Alzoubi A, Toba M, Abe K, O'Neill KD, **Rocic P**, Fagan KA, McMurtry IF, Oka M. Dehydroepiandrosterone Restores Right Ventricular Structure and Function in Rats with Severe Pulmonary Arterial Hypertension. *Am J Physiol Heart Circ Physiol.* 2013 Jun;304(12):H1708-18.

33. Dodd T, Wiggins L, Hutcheson R, Smith E, Alla M, Hysell B, Russell JC, **Rocic P**. Impaired Coronary Collateral Growth in the Metabolic Syndrome Is in Part Mediated by Matrix Metalloproteinase 12-Dependent Production of Endostatin and Angiostatin. *Arterioscler Thromb Vasc Biol.* 2013 Apr;33(6):1339-49.

34. Leavesley SJ, Ledkins W, **Rocic P**. A device for performing automated balloon catheter inflation ischemia studies. *PLoS One.* 2014 Apr 25;9(4):e95823.

35. Hutcheson R, Chaplin J, Hutcheson B, Borthwick F, Proctor S, Gebb S, Jadhav R, Smith E, Russell JC, **Rocic P**. miR-21 normalizes vascular smooth muscle proliferation and improves coronary collateral growth in metabolic syndrome. *FASEB J*. 2014 Sep;28(9):4088-99.

36. Villalta PC, **Rocic P**, Townsley MI. Role of MMP2 and MMP9 in TRPV4-induced lung injury. Am J *Physiol Lung Cell Mol Physiol.* 2014 Oct 15;307(8):L652-9.

37. Hutcheson R, Terry R, Hutcheson B, Jadhav R, Chaplin J, Smith E, Barrington R, Proctor SD, **Rocic P**. miR-21-mediated decreased neutrophil apoptosis is a determinant of impaired coronary collateral growth in metabolic syndrome. *Am J Physiol Heart Circ Physiol.* 2015 Jun 1;308(11):H1323-35.

38. Diane A, Pierce WD, Kelly SE, Sokolik S, Borthwick F, Jacome-Sosa M, Mangat R, Pradillo JM, Allan SM, Ruth MR, Field CJ, Hutcheson R, **Rocic P**, Russell JC, Vine DF, Proctor SD. Mechanisms of Comorbidities Associated With the Metabolic Syndrome: Insights from the *JCR:LA-cp* Corpulent Rat Strain. *Front Nutr*. 2016 Oct 10;3:44.

39. Joseph G, Soler A, Hutcheson R, Hunter I, Bradford C, Hutcheson B, Gotlinger KH, Jiang H, Falck JR, Proctor S, Laniado Schwartzman M, **Rocic P**. Elevated 20-HETE Impairs Coronary Collateral Growth in Metabolic Syndrome Via Endothelial Dysfunction. *Am J Physiol Heart Circ Physiol.* 2017 Mar 1; 312(3): H528–H540.

40. Hunter I, Soler A, Joseph G, Hutcheson B, Bradford C, Zhang F, Potter BJ, Proctor SD, **Rocic P**. Cardiovascular function in male and female JCR:LA-cp rats: Effect of high fat/high sucrose diet. *Am J Physiol Heart Circ Physiol*. 2017 Apr 1;312(4):H742-H751.

41. Elevated 20-HETE in metabolic syndrome regulates arterial stiffness and systolic hypertension via MMP12 activation. Soler A, Hunter I, Joseph G, Hutcheson R, Hutcheson B, Yang J, Zhang FF, Joshi SR, Bradford C, Gotlinger KH, Maniyar R, Falck JR, Proctor S, Schwartzman ML, Gupte SA, **Rocic P**. *J Mol Cell Cardiol*. 2018 Apr;117:88-99.

Editorials and Reviews

1. Dell'Italia LJ, **Rocic P**, Lucchesi PA. Use of angiotensin-converting enzyme inhibitors in patients with diabetes and coronary artery disease. *Curr Probl Cardiol.* 2002 Jan;27(1):6-36.

2. **Rocic P**, Lucchesi P. NAD(P)H oxidases and TGF-beta-induced cardiac fibroblast differentiation: Nox-4 gets Smad. *Circ Res.* 2005 Oct 28;97(9):850-2.

3. **Rocic P.** Differential phosphoinositide 3-kinase signaling: implications for PTCA? *Am J Physiol Heart Circ Physiol.* 2009 Dec;297(6):H1970-1.

4. **Rocic P**. Can ErbB2 over-expression protect against doxorubicin cardiotoxicity? *Am J Physiol Heart Circ Physiol.* 2015 Oct;309(8):H1235-6.

5. **Rocic P.** Can microRNAs Be Biomarkers or Targets for Therapy of Ischemic Coronary Artery Disease in Metabolic Syndrome? *Curr Drug Targets*. 2017;18(15):1722-1732.

6. 20-HETE in the regulation of vascular and cardiac function. **Rocic P**, Schwartzman ML. *Pharmacol Ther*. 2018 Dec;192:74-87.

7. Comparison of Cardiovascular Benefits of Bariatric Surgery and Abdominal Lipectomy. **Rocic P**. *Curr Hypertens Rep.* 2019 Apr 5;21(5):37.

8. Patophysiology of chronic peripheral ischemia: new perspectives. Signorelli SS, Vanella L, Abraham NG, Scuto S, Marino E, **Rocic P**. *Ther Adv Chronic Dis.* 2020 Feb 5;11:2040622319894466.

Other Publications (book chapters)

1. Zang C, Rogers PA, Markus D. Muller-Delp JM, Teifenbacher CP, Potter B, Knudson JD, **Rocic P**, Chilian WM. (2007). "Regulation of Coronary Microvascular Resistance in Health and Disease" In: Handbook of Physiology: Microcirculation (RF. Tuma, WN. Duran, K. Ley eds.) Chpt 12, pp 521-549. Academic Press – Elsevier, San Diego, CA.

2. **Rocic P**, Gupte SA. (2015). "The Role of Vascualar Smooth Muscle Phenotype in Coronary Artery Disease" In: Translational Research in Coronary Artery Disease: Patophysiology to Treatment (WS. Aronow, JA. McClung eds.) Chpt 2, pp 15-21. Academic Press – Elsevier, San Diego, CA.

Selected Invited Scientific Presentations

2000	"Regulation of Ang II-induced vascular smooth muscle protein synthesis by multiple signaling pathways", Boston Biomedical Research Institute, Boston, MA
2000	Regulation of Ang II-induced vascular smooth muscle protein synthesis by multiple signaling pathways", Emory University, Atlanta, GA
2002	"Redox-sensitivity of Ang II-dependent translation initiation", Department of Physiology, LSHU Health Sciences Center, New Orleans, Louisiana
2006	"Therapeutic Angiogenesis, Current Problems and Future Solutions - Why Does VEGF Gene Therapy Stimulate Coronary Collateral Growth in Animals But Not In Patients?" American Heart Association Scientific Sessions 2006, Chicago, IL.
2007	"Why did therapeutic angiogenesis trials using VEGF fail?" North Eastern Universities College of Medicine, Rootstown, OH
2007	"Why did therapeutic Angiogenesis Trials Using VEGF Fail?", LSU Health Sciences Center, New Orleans, LA
2007	"The Importance of Mitochondrial Oxidative Stress and Integrity of Mitochondrial DNA in Coronary Arteriogenesis", American Heart Association Scientific Sessions 2007, Orlando, FL
2008	"Regulation of Coronary Collateral Growth by Oxidative Stress and AT1R-mediated Signaling", Cell Signaling Seminar Series, University of South Alabama, Mobile, AL
2008	"The Angiotensin II Paradox in Coronary Collateral Growth", Experimental Biology 2008, Sand Diego, CA
2009	"The Mechanistic Basis for the Disparate Effects of Angiotensin II on Coronary Collateral Growth", Experimental Biology 2008, San Diego, CA
2008	"Role of NAD(P)H oxidase- and Mitochondria-derived ROS in Coronary Collateral Growth", Experimental Biology 2008, San Diego, CA
2009	"Regulation of Coronary Collateral Growth by AT1R-mediated and Redox-sensitive Signaling", Medical College of Georgia, Augusta, GA
2009	"Redox dependency of Coronary Collateral Growth", American Heart Association Scientific Sessions 2008, New Orleans, LA
2009	"How to Measure Coronary Collateral Function in Rodents", American Heart Association Scientific Sessions 2008, New Orleans, LA
2010	"p38 MAPK-dependent regulation of MMPs during coronary collateral growth", Experimental Biology 2010, Anaheim, CA
2011	"Beneficial effects of physiological Angiotensin II and ROS levels on coronary collateral formation", Experimental Biology 2011, Washington, DC
2011	"Redox-sensitive pathways in coronary collateral growth", American Heart Association Scientific Sessions 2011, Orlando, FL
2012	"MMPs in coronary collateral growth", University of South Alabama, Mobile, AL

2012 "Vascular smooth muscle phenotype is a critical determinant of coronary collateral growth", Louisiana State University Health Sciences Center, New Orleans, LA
2012 "Redox regulation of vascular growth", American Heart Association Scientific Sessions 2012, Los Angeles, CA
2012 "Mechanisms underlying impaired collateral growth in the metabolic syndrome: Role of MMP12", American Heart Association Scientific Sessions 2012, Los Angeles, CA
2013 "MicroRNA-145 restores coronary collateral growth in the metabolic syndrome", Gulf Coast Physiologic Society Meeting 2013, Mobile, AL
2013 "MicroRNA-145-dependent regulation of coronary collateral growth", New York Medical College, Valhalla, NY
2013 "Altered vascular smooth muscle phenotype impairs arteriogenesis in metabolic syndrome", American Heart Association Scientific Sessions 2013, Dallas, TX
2014 "Vascular smooth muscle phenotype is a critical determinant of coronary collateral growth", Tulane University, New Orleans, LA
2014 "MicroRNA-145-dependent regulation of coronary collateral growth", Department of Cardiology Grand Rounds, Westchester Hospital, Valhalla, NY
2014 "Cardiovascular effects of gastric bypass and intra-abdominal lipectomy in metabolic syndrome", Department of Gastroenterology Grand Rounds Westchester Hospital, Valhalla, NY
2015 "How to induce arteriogenesis in the heart in the metabolic syndrome", Cardiovascular Symposium, Valhalla, NY
2015 "The role of risk factors in attenuating collateral growth", American Heart Association Scientific Sessions 2015, Orlando, FL
2016 "20-HETE is a major regulator of coronary collateral growth in the metabolic syndrome", Winter Eicosanoid Conference, Baltimore, 2016
2016 "Deregulated apoptosis and proliferation are key components of impaired coronary collateral growth in the metabolic syndrome". Experimental Biology, San Diego, 2016
2016 "Myocardial adaptations and pathways to prevent cardiac failure". Westchester Cardiovascular Symposium, Rye Brook, NY, 2016
2017 "Effect of 20-HETE antagonists on myocarial infarct size and post-infarct ventricular remodeling", New York Methodist Hospital, Department of Cardiology, Brooklyn, New York, NY
2017 "Elevated 20-HETE impairs coronary collateral growth in metabolic syndrome via Neutrophil-mediated inflammation", Department of Physiology, INSERM, Paris, France
2017 "20-HETE antagonists normalize arterial stiffness and systolic hypertension in metabolic syndrome", Americhan Heart Association Council for Hypertension, San Francisco, CA
2018 "Neutrophil-dependent regulation of arteriogenesis", 11th World Congress for Microcirculation, Vancouver, Canada

LABORATORY TECHNIQUES: Rodent survival cardiac and vascular surgery (models of myocardial infarction, transient/repetitive ischemia, wire and balloon vascular injury, cardiac and vascular catheterization, echocardiography, cell isolation and cell culture (cardiac and vascular cells, immune cells, cell lines), DNA, RNA and protein isolation from cells and tissues, Western blot, immunohistochemistry, immunofluorescence, in-situ hybridization, PCR, RT-PCR, ELIZA, molecular biology techniques (restriction enzyme digestion, cloning, viral vector construction, propagation and amplification), plaque assay, in vitro and in vivo viral vector delivery, sterile technique

ACADEMIC SERVICE

University

University of South Alabama

2010-2013	Institutional Animal Care and Use Committee (IACUC) (Chair 2012-2013), University of South Alabama College of Medicine, Mobile, AL
2011-2013	College of Medicine Admissions Committee (Ad hoc), University of South Alabama College of Medicine, Mobile, AL
2008-2011	Medical Student Summer Research Committee (Chair 2010-2011), University of South Alabama College of Medicine, Mobile, AL
2008-2011	Medical Student Schoolarship Committee, University of South Alabama College of Medicine, Mobile, AL

New York Medical College

2015-2020	Intramural Research Grant/Fellowship Review
2015-2020	Course Evaluation Committee
2015-2020	Institutional Animal Care and Use Committee (IACUC)
2016-2020	Microbiology and Immunology Chair Search Committee
2017-2020	Faculty Evaluation Committee
2017-2020	Graduate Curriculum Committee

Sam Houston State University

2020-present	Faculty Evaluation Systems Committee

External

2012-2016	ATVB Programming Committee, American Heart Association

Honors, editorial boards and advisory committees

Honors

1995	Howard Hughes Undergraduate Summer Research Scholarship
2012	Red Sash Teaching Award
2013	Red Sash Teaching Award

Grant review

2006-2016	American Heart Association - Blood Pressure Regulation, Vascular Wall Biology
2013-2016	American Heart Association - Student Undergraduate & Health Science Fellowships
2010-2016	Italian Ministry of Health – Cardiovascular, Cardiology-Industry Collaboration Initiative
2016-present	National Institutes of Health – NHLBI, Myocardial Ischemia and Metabolism

Editorial boards

2012-present	**Associate Editor** - Journal of Biochemical and Pharmacological Research
2007-2020	American Journal of Physiology: Heart and Circulatory Physiology
2009-2020	Microcirculation
2012-2020	Circulation Research
2014-present	Annals of Vascular Medicine and Research