

BIOGRAPHICAL SKETCH

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| | | | |
|-----------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------|---------|----------------|
| NAME Rong Ma, PhD | POSITION TITLE | | |
| eRA COMMONS USER NAME rongma | Associate Professor | | |
| EDUCATION/TRAINING (<i>Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.</i>) | | | |
| INSTITUTION AND LOCATION | DEGREE (if applicable) | YEAR(s) | FIELD OF STUDY |
| Anhui Medical University, P.R. China | B.S. | 1983 | Medicine |
| Anhui Medical University, P.R. China | M.S. | 1989 | Physiology |
| University of Nebraska Medical Center | PhD | 1999 | Physiology |
| University of Nebraska Medical Center | Post-Doc | 2002 | Physiology |

A. Personal Statement

Dr. Ma studies the physiology and pathology of kidneys. Specifically, electrophysiology, calcium imaging, biochemical and molecular approaches are used to investigate the function and biology of canonical transient receptor potential (TRPC) channels and store-operated calcium channels (SOC) in glomerular mesangial cells and podocytes to determine their role in the development of diabetic nephropathy and other kidney diseases. In a separate series of studies, Dr. Ma's lab is utilizing these same approaches to investigate TRPC mechanisms that mediate diabetic vascular complications.

B. Positions and HonorsPositions and Employment

| | | | |
|-----------|---------------------|---------------------|------------------------------|
| 1983-1989 | Res & Teach Assist, | Physiology, | Anhui Med. Univ., China |
| 1990-1995 | Assist Prof, | Physiology, | Anhui Med. Univ., China |
| 1999-2002 | Res. Assoc., | Physiology, | Univ. of Nebraska Med. Cent. |
| 2002-2004 | Res Assist. Prof | Cell Biology, | Univ. of Oklahoma HSC |
| 2004-2009 | Assist. Prof. | Physiology, | Univ. of North Texas HSC |
| 2007- | Member | O'Brien Kidney Cent | UT Southwestern |
| 2010- | Associate Prof. | Physiology | Univ. of North Texas HSC |

Professional Memberships

| | |
|-----------|--------------------------------|
| 1997- | American Physiological Society |
| 2002-2014 | American Hear Association |
| 2004-2014 | American Society of Nephrology |
| 2007-2011 | American Diabetes Association |

Honors

| | |
|------|---------------------------------------------------------------------------------------------------------------|
| 1998 | Losartan Travel Award, Merck & Company, Inc |
| 1998 | Caroline Tum Suden / Frances A. Hellegrandt Professional Opportunity Award, American Physiological Society |
| 2000 | The Second Place Award for Oral Presentation, Midwest Student Biomed Research Forum |
| 2002 | The First Place in Excellence in Renal Research American Physiological Society |
| 2005 | Lazaro J. Mandel Young Investigator Award, American Physiological Society |
| 2005 | Research Recognition Award, American Physiological Society Renal Section |
| 2007 | Travel Award Advances in Research Conference, American Society of Nephrology |
| 2008 | Investigator Award, Santa Cruz Biotechnology Inc. |

- 2009 Research Rising Star Award, University of North Texas HSC
2011 President's Faculty Award in Research Excellence, University of North Texas HSC
2012 President's Faculty Award in Research Excellence, University of North Texas HSC

Member of Study Section and Editorial Board

- 2005-2008: AHA Western Review Consortium
2009-2010: *Ad hoc* reviewer, National Scientific Foundation
2010- Editorial Board Member, Experimental Biology and Medicine
2011- Editorial Board Member, World Journal of Diabetes (WJD)
2011-2013: AHA National Center, Vascular Biology and Blood Pressure Regulation Study Section
2012- Editorial Board Member, Frontiers in Renal and Epithelial Physiology
2013- Member, Scientific Review Board Panel for Nephrology, Department of VA
2015- Editorial Board Member, Journal of Nephrology Research
2015- AHA Cell Transporter I Study Section

C. Peer-reviewed publications (selected)

1. **Ma, R.**, Zucker, I.H., and Wang, W. Central gain of the cardiac sympathetic afferent reflex in dogs with heart failure. *Am. J. Physiol Heart Circ Physiol* 273: H2664-H2671, 1997 (PMID: 9435602)
2. **Ma, R.**, Zucker, I.H., and Wang, W. Reduced NO enhances the central gain of the cardiac sympathetic afferent reflex in dogs with heart failure. *Am J Physiol Heart Circ Physiol* 276: H19-H26, 1999 (PMID: 9887012)
3. **Ma, R.**, Schultz, H.D., and Wang, W. Chronic central infusion of angiotensin II potentiates cardiac sympathetic afferent reflex. *Am J Physiol Heart Circ Physiol* 277: H15-H22, 1999 (PMID: 10409176)
4. Wang, W., Schultz, H.D., and **Ma, R.** Cardiac sympathetic afferent sensitivity is enhanced in heart failure. *Am J Physiol Heart Circ Physiol* 277: H812-H817, 1999 (PMID: 10444509)
5. Wang, W. and **Ma, R.** Cardiac sympathetic afferent reflexes in heart failure. *Heart Failure Rev* 5: 57-71, 2000 (PMID: 16228916)
6. **Ma, R.**, Smith, S., Child, A., Carmines, P.K., and Sansom, S.C. Store-operated calcium channels in human mesangial cells. *Am J Physiol Renal Physiol* 278: F954-F961, 2000 (PMID: 10836983)
7. Sansom, S. C., **Ma, R.**, Carmines, P.K., and Hall, D.A. Regulation of Ca²⁺-activated K⁺ channels by multifunctional Ca²⁺/calmodulin-dependent protein kinase. *Am J Physiol Renal Physiol* 279: F283-F288, 2000 (PMID: 10919847)
8. **Ma, R.** and Sansom, S.C. Epidermal growth factor activates store-operated calcium channel in human glomerular mesangial cells. *J Am Soc Nephrol* 12:47-53, 2001 (PMID: 11134249)
9. Wang, W., Schultz, H.D., and **Ma, R.** Volume expansion potentiates cardiac sympathetic afferent reflex in dogs. *Am J Physiol Heart Circ Physiol* 280: H576-H581, 2001 (PMID: 11158954)
10. **Ma, R.**, Pluznick, J.L., Kudlacek, P.E., and Sansom, S.C. Protein kinase C activates store-operated Ca²⁺ channels in human glomerular mesangial cells. *J Biol Chem* 276: 25759-25765, 2001 (PMID: 11352899)
11. **Ma, R.**, Kudlacek, P.E., and Sansom, S.C. Protein kinase C α participates in activation of store-operated Ca²⁺ channels in human mesangial cells. *Am J Physiol Cell Physiol* 283:C1390-1398, 2002 (PMID: 12372800)
12. Kudlacek, P. E., Pluznick, J.L., **Ma, R.**, Padalinam, B., and Sansom, S.C. The role of H β 1 in activation of human mesangial cell BK channels by cGMP-kinase. *Am J Physiol Renal Physiol* 285:F289-F294, 2003 (PMID: 12670831)
13. **Ma, R.**, Rundle, D., Jacks, J., Koch, M., Downs, T., and Tsiokas, L. Inhibitor of myogenic family: a novel suppressor of store-operated Ca²⁺ currents through an interaction with TRPC1. *J Biol Chem* 278:52763-52772, 2003 (PMID: 14530267)
14. Li, W.P., Tsiokas, L., Sansom, S.C., and **Ma, R.** Epidermal growth factor activates store-operated Ca²⁺ channels through an IP₃ independent pathway in human glomerular mesangial cells. *J Biol Chem* 279:4570-4577, 2004 (PMID: 14612458)

15. **Ma, R.**, Pluznick, J.L., and Sansom, S.C. Ion channels in mesangial cells: Function, Malfunction or Fiction. *Physiology* 20:102-111, 2005 (PMID: 15772299)
16. **Ma, R.**, Zhu, G.Q., and Wang, W. Interaction of central Ang II and NO on the cardiac sympathetic afferent reflex in dogs. *Auton Neurosci Basic Clin* 118:51-60, 2005 (PMID: 15795177)
17. **Ma, R.**, Li, W.P., Rundle, D., Kong, J., Akbarali, H., and Tsiokas, L. PKD2 functions as an EGF-activated plasma membrane channel. *Mol Cell Biol* 25:8285-8298, 2005 (PMID: 16135816)
18. **Ma, R.**, Du, J., Sours, S., Ding, M. Store-operated Ca²⁺ channel in renal microcirculation and glomeruli. *Exp Biol Med* 231:145-153, 2006 (PMID: 16446490)
19. Sours, S., DU, J., Chu, S., Zhou, X.J., Ding, M., and **Ma, R.** Expression of canonical transient receptor potential (TRPC) proteins in human glomerular mesangial cells. *Am J Physiol Renal Physiol* 290:F1507-F1515, 2006 (PMID: 16418302)
20. Du, J., Sours-rothers, S., Coleman, R., Ding, M., Graham, S., Kong, D., and **Ma, R.** Canonical transient receptor potential 1 channel is involved in contractile function of glomerular mesangial cells. *J Am Soc Nephrol* 18:1437-1445, 2007 (PMID: 1738936)
21. Graham, S., Ding, M., Sours-Brothers, S., Yorio, T., and **Ma, R.** Downregulation of TRPC6 protein expression by high glucose, a possible mechanism for the impaired Ca²⁺ signaling in glomerular mesangial cells in diabetes. *Am J Physiol Renal Physiol* 293: F1381-F1390, 2007 (PMID: 17699555)
22. Du, J., Sours-Brothers, S., Ding, M., Graham, S., and **Ma, R.** Mediation of angiotensin II-induced Ca²⁺ signaling by polycystin 2 in glomerular mesangial cells. *Am J Physiol Renal Physiol* 294:F909-F918, 2008 (PMID: 18256307)
23. Sours-Brothers, S., **Ma, R.**, and Koulen, P. Ca²⁺-sensitive transcriptional regulation: from kinase/phosphatase-mediated activity to direct DNA interaction. *Front Biosci (Landmark Ed)* 14: 1851-1856, 2009 (PMID: 19273168)
24. Sours-Brothers, S., Ding, M., Graham, S., and **Ma, R.** Interaction between TRPC1/TRPC4 assembly and STIM1 contributes to store-operated Ca²⁺ entry in mesangial cells. *Exp Biol Med* 234:673-682, 2009 (PMID: 19307462)
25. Sours-Brothers, S. and **Ma, R.** Canonical transient receptor potential (TRPC) in renal microcirculation. *Microcirculation: Function, Malfunction, and Measurement*. Frank Columbus, *Nova Publishers* pp 25-38, 2009
26. Wu, Z., Xu, Q., Zhang, L., Kong, D., **Ma, R.**, and Wang, L. Protective effect of resveratrol against kainic acid-induced temporal lobe epilepsy in rats. *Neurochem Res* 34:1393-1399, 2009
27. Graham, S., Ding, M., Ding, Y., Sherry-Brothers, S., Luchowski, R., Gryczynski, Z., Yorio, T., Ma, H., and **Ma, R.** Canonical transient receptor potential 6 (TRPC6), a redox-regulated cation channel. *J Biol Chem* 285:23466-23476, 2010 (PMCID: PMC2906337)
28. Graham, S., Gorin, Y., Abboud, H.E., Ding, M., Lee, D.Y., Shi, H., Ding, Y., and **Ma, R.** Abundance of TRPC6 protein in glomerular mesangial cells is decreased by ROS and PKC in diabetes. *Am J Physiol Cell Physiol* 301:C304-C315, 2011 (PMCID: PMC3154551)
29. Ding, Y., Winters, A., Ding, M. Graham, S., Akopova, I., Muallem, S., Hong, J.H., Gryczynski, Z., Yang, S.H., Birnbaumer, L., and **Ma, R.** Reactive oxygen species-mediated TRPC6 activation in vascular myocytes, a mechanism for vasoconstrictor-regulated vascular tone. *J Biol Chem* 286:31799-31809, 2011 (PMCID: PMC3173128)
30. Graham, S., Yuan, J., and **Ma, R.** Canonical transient receptor potential (TRPC) channels in diabetes. *Exp Biol Med* 237:111-118, 2012 (PMCID: PMC3307128)
31. Winters, A., Taylor, J.C., Ren, M., **Ma, R.**, Liu, R., and Yang, S.H. Translational focal cerebral ischemia induces long-term cerebral vasculature dysfunction in a rodent experimental stroke model. *Transl Stroke Res* 3:279-285, 2012 (PMCID: PMC3418819)
32. Luan, J., Li, W., Han, J., Zhang, W., Gong, H. and **Ma, R.** Renal protection of in vivo administration of tempol in streptozotocin-induced diabetic rats. *J Pharmacol Sci* 119:167-176, 2012 (PMCID: PMC3539787)
33. Ding, Y., Stidham, R., Bumeister, R., Winters, A., Sprouse, M., Ding, M., Ferguson, D.A., Meyer, C.J., Wigley, W.C., and **Ma, R.** The synthetic triterpenoid, RTA405, increases glomerular filtration rate and reduces angiotensin II-induced contraction of glomerular mesangial cells. *Kidney Intl* 83:845-854, 2013 (PMCID: PMC3600401)

34. Shen, B., Zhu, J.H., Zhang, J., Jiang, F.F., Zhang, Y., Ke, D.P., **Ma, R.** and Du, J. Attenuated mesangial cell proliferation related to store-operated Ca^{2+} entry in aged rat: the role of STIM1 and Orai1. *Age (Dordr)* 35: 2093-2202, 2013 (PMID: 23334602)
35. Wang, Y., Ding, M., Chaudhari, S., Ding, Y., Yuan, J., Stankowska, D., He, S., Krishnamoorthy, R., Cunningham, J.T., and **Ma, R.** NF- κ B mediates suppression of canonical transient receptor potential 6 (TRPC6) expression by ROS and PKC in kidney cells. *J Biol Chem* 288: 12852-12865, 2013 (PMC3642329)
36. Chaudhari, S., Wu, P., Wang, Y., Ding, Y., Yuan, J., and **Ma, R.** High glucose and diabetes enhanced store-operated Ca^{2+} entry and increased expression of its signaling proteins in mesangial cells. *Am J Physiol Renal Physiol* 306: F1069-F1080, 2014 (PMID: 24623143)
37. Lee, K.P., Choi, S., Ahuja, M., Graham, S., **Ma, R.**, Insuk, S., Muallem, S. and Yuan, J. Molecular determinants mediating regulation of TRPC channels by STIM1. *J Biol Chem* 289: 6372-6382, 2014 (PMID: 24464579)
38. Ilatovskaya, D.V., Palygin, O., Chubinskiy-Nadezhdin, V., Negulyaev, Y.A., **Ma, R.**, Birnbaumer, L. and Staruschenko, A. Acute effect of angiotensin II on TRPC6 channels in the podocytes of freshly isolated glomeruli. *Kidney Int* 86:506-514, 2014 (PMID: 24646854)
39. Sun, L., Li, W., Li, W., Xiong, L., Li, G., and **Ma, R.** Astragaloside IV prevents damage to human mesangial cells through the inhibition of the NADPH oxidase/ROS/Akt/NF- κ B pathway under high glucose conditions. *Int J Mol Med* 34: 167-176, 2014 (PMID: 24718766)
40. Zuckerman, J.E., Gale, A., Wu, P., **Ma, R.**, and Davis, M.E. siRNA delivery to the glomerular mesangium using polycationic cyclodextrin nanoparticles containing siRNA. *Nucleic Acid Ther* 25: 53-64, 2015 (PMID: 25734248)
41. Wu, P., Wang, Y., Davis, M.E., Zuckerman, J.E., Chaudhari, S., Begg, M., and **Ma, R.** Store-operated Ca^{2+} channel in mesangial cells inhibits matrix protein expression. *J Am Soc Nephrol* 26: 2691-2702, 2015 (PMID: 25788524)
42. Wang, Y., Chaudhari, S., Ren, Y.Z., and **Ma, R.** Impairment of hepatic nuclear factor 4 α (HNF4 α) binding to *stim1* promoter contributes to high glucose-induced upregulation of STIM1 expression in glomerular mesangial cells. *Am J Physiol Renal Physiol* 308: F1135-F1145, 2015 (PMID: 25786776)
43. Meng, Y., Li, W.Z., Shi, Y.W., Zhou, B.F., **Ma, R.**, and Li, W.P. Danshensu protects against ischemia/reperfusion injury and inhibits the apoptosis of H9c2 cells by reducing the calcium overload through the p-JNK-NF- κ B-TRPC6 pathway. *Int J Mol Med* 37: 258-266, 2016 (PMID: 26718129)
44. Chaudhari, S. and **Ma, R.** Store-operated calcium entry and diabetic complications. *Exp Biol Med* 241: 343-352, 2016 (PMID: 26468167)
45. **Ma, R.**, Chaudhari, S., and Li, W. Canonical transient receptor potential 6 (TRPC6) channel, a new target of reactive oxygen species in renal physiology and pathology. *Antioxid Redox Signal* 25:732-748 (selected as a High-Impact Article) (PMID: 26937558)
46. Jiang, H., Qin, X.J., Li, W.P., **Ma, R.**, Wang, T. and Li, Z.Q. LncRNAs expression in adjuvant-induced arthritis rats reveals the potential role of LncRNAs contributing to rheumatoid arthritis pathogenesis. *Gene* 593: 131-142, 2016

D. Contributions to Science

I. Identified and characterized store-operated Ca^{2+} channels (SOC) in mesangial cells (MC)

- First to describe electrophysiological and pharmacological properties of SOC in glomerular MC.
 - Revealed physiological role for SOC in MC signaling pathways, such as growth factor signaling.
 - Demonstrated SOC contributions to MC function.
1. **Ma, R.**, Smith, S., Child, A., Carmines, P.K., and Sansom, S.C. Store-operated calcium channels in human mesangial cells. *Am J Physiol Renal Physiol*. 278: F954-F961, 2000.
 2. **Ma, R.** and Sansom, S.C. Epidermal growth factor activates store-operated calcium channel in human glomerular mesangial cells. *J Am Soc Nephrol* 12:47-53, 2001.

3. Li, W.P., Tsiokas, L., Sansom, S.C. and **Ma, R.** Epidermal growth factor activates store-operated Ca^{2+} channels through an IP_3 independent pathway in human glomerular mesangial cells. *J Biol Chem* 279:4570-4577, 2004.
4. **Ma, R.**, Du, J., Sours, S., Ding, M. Store-operated Ca^{2+} channel in renal microcirculation and glomeruli. *Exp Biol Med* 231:145-153, 2006.

II. Advanced our understanding of molecular regulatory mechanisms of SOC

- Demonstrated that protein kinase C, particularly it's α isoform, is a physiological activator of SOC in MC.
 - Recently established physical interaction between SOC and specific isoforms of classic transient receptor potential channels (TRPC) in MC cells, highlighting a novel form of SOC activity regulation.
 - Showed that the α -isoform of the inhibitor of myogenic family (I-mfa) is a suppressor of SOC in cell lines.
1. **Ma, R.**, Pluznick, J.L., Kudlacek, P.E. and Sansom, S.C. Protein kinase C activates store-operated Ca^{2+} channels in human glomerular mesangial cells. *J Biol Chem* 276: 25759-25765, 2001.
 2. **Ma, R.**, Kudlacek, P.E., and Sansom, S.C. Protein kinase $\text{C}\alpha$ participates in activation of store-operated Ca^{2+} channels in human mesangial cells. *Am J Physiol Cell Physiol* 283:C1390-1398, 2002.
 3. **Ma, R.**, Dana, R., Jacks, J., Koch, M., Downs, T., and Tsiokas, L. Inhibitor of myogenic family: a novel suppressor of store-operated Ca^{2+} currents through an interaction with TRPC1. *J Biol Chem* 278:52763-52772, 2003.
 4. Sours-Brothers, S., Ding, M., Graham, S., and **Ma, R.** Interaction between TRPC1/TRPC4 assembly and STIM1 contributes to store-operated Ca^{2+} entry in mesangial cells. *Exp Biol Med* 234:673-682, 2009.

III. Established complex alterations in MC SOC with diabetic nephropathy: potential therapeutic target

- First observation of SOC-mediated suppression of extracellular matrix protein expression in MC, suggesting a potentially important anti-fibrotic, beneficial impact on kidney in the setting of diabetes.
 - Observed distinct time-dependent effects of high glucose upon SOC function in cultured human MC.
Short term treatment with glucose tended to reduce SOC activity.
Long term treatment enhanced SOC activity, suggesting unique phases with development of diabetes.
 - Proposed early attenuation of SOC may advance DN, but later enhancement may serve to limit injury.
 - These studies highlight a novel potential therapeutic option for patients with DN.
1. Shen, B., Zhu, J.H., Zhang, J., Jiang, F.F., Zhang, Y., Ke, D.P., **Ma, R.** and J. Du. Attenuated mesangial cell proliferation related to store-operated Ca^{2+} entry in aged rat: the role of STIM1 and Orai1. *Age (Dordr)* 35: 2093-2202, 2013
 2. Chaudhari, S., Wu, P., Wang, Y., Ding, Y., Yuan, J., and **Ma, R.** High glucose and diabetes enhanced store-operated Ca^{2+} entry and increased expression of its signaling proteins in mesangial cells. *Am J Physiol Renal Physiol* 306: F1069-F1080, 2014.
 3. Wang, Y., Chaudhari, S., Ren, Y.Z., and **Ma, R.** Impairment of hepatic nuclear factor 4 α (HNF4 α) binding to *stim1* promoter contributes to high glucose-induced upregulation of STIM1 expression in glomerular mesangial cells. *Am J Physiol Renal Physiol* (Published online on March 18, 2015)
 4. Wu, P., Wang, Y., Davis, M.E., Zuckerman, J.E., Chaudhari, S., Begg, M., and **Ma, R.** Store-operated Ca^{2+} channel in mesangial cells inhibits matrix protein expression. *J Am Soc Nephrol* 26, 2015 (Published online on March 18, 2015; PMID: 25788524).

IV. Established physiological relevance of TRPC channels for MC function in health and with diabetes

- First to describe the distribution of TRPC isoforms in human MC.
(TRPC are G-protein-coupled and have 6 isoforms that are specific to cell types and tissues.)
 - First to demonstrate TRPC can regulate glomerular filtration rate (GFR) by altering MC contractile function
 - Showed abundance of TRPC6 protein is reduced with early diabetes: potential mechanism to raise GFR.
 - This line of work provides novel insights into physiological and pathological relevance of TRPC in MC.
1. Sours, S., Du, J., Chu, S., Zhou, X.J., Ding, M., and **Ma, R.** Expression of canonical transient receptor potential (TRPC) proteins in human glomerular mesangial cells. *Am J Physiol Renal Physiol* 290:F1507-F1515, 2006.
 2. Du, J., Sours-Brothers, S., Coleman, R., Ding, M., Graham, S., Kong, D., and **Ma, R.** TRPC1 channel is involved in contractile function of glomerular mesangial cells. *J Am Soc Nephrol* 18:1437-1445, 2007.
 3. Graham, S., Ding, M., Sours-Brothers, S., Yorio, T., and **Ma, R.** Downregulation of TRPC6 protein expression by high glucose, a possible mechanism for the impaired Ca²⁺ signaling in glomerular mesangial cells in diabetes. *Am J Physiol Renal Physiol* 293: F1381-F1390, 2007.
 4. Ding, Y., Stidham, R., Bumeister, R., Winters, A., Sprouse, M., Ding, M., Ferguson, D.A., Meyer, C.J., Wigley, W.C., and **R. Ma.** RTA405, a synthetic triterpenoid, increases glomerular filtration rate and reduces angiotensin II-induced contraction of glomerular mesangial cells. *Kidney Intl* 83:845-854, 2013.

V. Demonstrated regulation of TRPC6 by reactive oxygen species in contractile cells

- TRPC6 is highly expressed in vascular smooth muscle cells and MC to regulate tone.
 - TRPC6 is believed to be activated by a Gq-coupled receptor/diacylglycerol pathway.
 - We are first to report that TRPC6 is redox sensitive and that reactive oxygen species have 2 opposing effects on TRPC6
 - Acute effect: Stimulate the channel by promoting membrane trafficking of the channel proteins
 - Chronic effect: Decrease abundance of channel protein by repression of *trpc6* gene transcription through protein kinase C/NF-κB pathway
 - We have provided novel mechanisms for controlling vascular and mesangial tone under normal and disease states associated with reactive oxygen species.
1. Graham, S., Ding, M., Ding, Y., Sours-Brothers, S., Luchowski, R., Gryczynski, Z., Yorio, T., Ma, H., and **Ma, R.** Canonical transient receptor potential 6 (TRPC6), a redox-regulated cation channel. *J Biol Chem* 285:23466-23476, 2010.
 2. Graham, S., Gorin, Y., Abboud, H.E., Ding, M., Lee, D.Y., Shi, H., Ding, Y., and **Ma, R.** Abundance of TRPC6 protein in glomerular mesangial cells is decreased by ROS and PKC in diabetes. *Am J Physiol Cell Physiol* 301:C304-C315, 2011.
 3. Ding, Y., Winters, A., Ding, M., Graham, S., Akopova, I., Muallem, S., Hong, J.H., Gryczynski, Z., Yang, S.H., Birnbaumer, L., and **Ma, R.** Reactive oxygen species-mediated TRPC6 activation in vascular myocytes, a mechanism for vasoconstrictor-regulated vascular tone. *J Biol Chem* 286:31799-31809, 2011.
 4. Wang, Y., Ding, M., Chaudhari, S., Ding, Y., Yuan, J., Stankowska, D., He, S., Krishnamoorthy, R., Cunningham, J.T., and **Ma, R.** NF-κB mediates suppression of canonical transient receptor potential 6 (TRPC6) expression by ROS and PKC in kidney cells. *J Biol Chem* 288:12852-12865, 2013.

Complete List of Published Work in MyBibliography:

<http://www.ncbi.nlm.nih.gov/sites/myncbi/1tsU8oCJVIFkE/bibliography/41689518/public/?sort=date&direction=ascending>

E. Research Support

Ongoing research supports

R25HL125447-01A1 Vishwanatha (PI) 4/1/16-3/30/21
NIH/NHLBI

Promoting diversity in research training for health professionals (PDRT)

The overall goal of PDRT is to promote diversity in health professional student populations by providing short-term research education support to stimulate career development in cardiovascular, pulmonary, hematologic, and sleep disorders research.

Role: Faculty mentor

R56DK108761 Ma (PI) 7/11/16-6/30/17
NIH/NIDDK

Inhibitor of myogenic family a, store-operated Ca²⁺ channel, and diabetic nephropathy

The major goal of this study is to determine specificity of in vivo nanoparticle delivery system and the contribution of I-mfa to glomerular injury in diabetes.

Role: PI

16GRNT27780043 Ma (PI) 1/1/16-12/31/17

American Heart Association South Western Affiliate

I-mfa, a potential therapeutic target of diabetic nephropathy

The major goal of this study is to investigate the contribution of increased I-mfa protein expression in glomerular mesangial cells to glomerular injury in diabetes and the underlying molecular mechanisms.

Role: PI

Completed research support (in the past 3 years)

5RO1DK079968-02 Ma (PI) 5/15/09-5/30/15 (no cost extension)
NIH/NIDDK

NADPH oxidases-derived ROS downregulate TRPC6 in mesangial cells in diabetes

The major goals of this study are to investigate the contribution of a decrease in TRPC6 channel expression to glomerular hyperfiltration at the early stages of diabetes and the underlying molecular mechanisms.

Role: PI

Research Grant Ma (PI) 11/1/13-12/30/14 (no cost extension)
SUNY Stony Brook

Impact of TBE-31 on glomerular filtration rate in rats

The major goal of this study was to determine the effect of TBE-31 on renal function.

Role: PI

Intramural seed grant Ma (PI) 5/1/15-8/31/16
UNTHSC

I-mfa, a potential target for the treatment of diabetic kidney disease

The major goal of this seed grant is to conduct pilot study to investigate if diabetes upregulates I-mfa protein expression in glomerular mesangial cells and if the increased I-mfa protein expression contributes to glomerular injury in diabetes.

Role: PI