
BIOGRAPHICAL SKETCH

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NAME Victoria L. King		POSITION TITLE Assistant Professor	
eRA COMMONS USER NAME (credential, e.g., agency login) Victoria.king			
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)	MM/YY	FIELD OF STUDY
University of Kentucky, Lexington, KY	B.S.	1994	Biology
University of Kentucky, Lexington, KY	Ph.D.	1999	Pharmaceutical Sciences
University of Kentucky, Lexington, Kentucky	Post- doctoral	2003	Cardiovascular Medicine

A. Personal Statement

The goal of the research in my laboratory is to investigate the role of inflammatory mediators, with a particular focus on the role of prostanoids in the development and progression of diet-induced obesity, diabetes and cardiovascular disease. In these studies we plan to investigate the role of microsomal prostaglandin E synthase 1 (mPGES-1) deficiency in mice on the development and progression of these pathophysiological states. mPGES-1 is the primary source of prostaglandin E₂ (PGE₂) during inflammation. A common major component of these pathophysiological states is inflammation. As a postdoctoral fellow and junior faculty member I have focused on the role of inflammation in the development and progression of atherosclerosis and abdominal aortic aneurysms. During the past three years, I have expanded my research program to include studies investigating the link between obesity induced inflammation and its role in the atherosclerotic lesion formation. As the PI of mentored grants from both the American Heart Association and the NIH, I have established the appropriate animal models and techniques in my laboratory to investigate the role of inflammatory mediators, such as mPGES-1 generated PGE₂ in the pathophysiology of these diseases. I have published peer reviewed publications as well as invited publications in both of these areas. Moreover, I have set up successful collaborations with other investigators in these areas, which has enhanced my ability to address unexpected findings including alterations in weight gain in response to feeding a high fat diet in our mouse model. These collaborations will facilitate my ability to determine mechanisms that mediate these alterations at the cellular level. In summary I have established a successful and productive research program in my laboratory in highly relevant areas given there is no sustained treatment for obesity. Moreover, my studies focus on a relevant but novel therapeutic target, mPGES-1, for treatment of this disease.

B. Positions and Honors

Positions and Employment

2003 – 2008 Research Assistant Professor of Internal Medicine
2008 – present Assistant Professor of Internal Medicine

Honors and Awards

2005 – 2007 Atorvastatin Research Award
2003 Young Investigators Award – Atherosclerosis, Thrombosis and Vascular Biology Meeting
2001 - 2003 American Heart Association Post Doctoral Fellowship
1999 - 2001 National Institutes of Health - RO1 Supplemental Post-Doctoral Fellowship
1998 - 1999 National Research Service Award

C. Publications

Selected Peer-Reviewed Publication (from 21 publications)

Most relevant to the current application

1. **King VL**, Szilvassy SJ, Daugherty A. Interleukin-4 deficiency decreases atherosclerotic lesion formation in a site-specific manner in female LDL receptor-/-mice. *Arterioscler Thromb Vasc Biol.* 2002; Mar 1;22(3):456-61.
2. **King VL**, Trivedi D, Gitlin J.R., Loftin CD. Selective cyclooxygenase-2 inhibition with celecoxib decreases angiotensin II-induced abdominal aortic aneurysm formation in mice. *Arterioscler Thromb Vasc Biol* 2006 May;26(5):1137-43.
3. **King VL**, Cassis LA, Daugherty A. Interleukin-4 does not influence development of hypercholesterolemia or angiotensin II-induced atherosclerotic lesions. *Am. J. Pathol.* 2007 171: 2040-2047.
4. Arsenescu V, Arsenescu RI, **King VL**, Swanson H, Cassis LA. Polychlorinated Biphenyl 77 Induces Adipocyte Differentiation and Proinflammatory Adipokines and Promotes Obesity and Atherosclerosis. *Environ Health Perspect* 2008; 116(6); 761-768.
5. Wilson P, Thompson JC, Webb NR, **King VL**, Tannock LR. Serum Amyloid A, but not C-Reactive Protein Stimulates Vascular Proteoglycan Synthesis in a Pro-Atherogenic Manner. *Am. J. Pathol.* 2008; 173(6): 1902-1910.
6. **King VL**, Hatch N, deBeer FC, Tannock LR. A Murine Model of Obesity with Accelerated Atherosclerosis. *Obesity* 2010; 18(1): 35-41.
7. Jahangiri A., Wilson P. Hou T, Brown A, **King VL**, Tannock LR. SAA is Found on ApoB-Containing Lipoproteins in Obese Diabetic Humans. Accepted for publication. *Obesity* 9/2012.
8. Hatch NW, Srodulski SJ, Chan HW, Zhang X, Tannock LR, **King VL**. Endogenous androgen enhances hypercholesterolemia and atherosclerosis in low-density lipoprotein receptor deficient mice. *Genet Med.* 2012; 9(5):319-28.

Additional recent publications of importance to the field (in chronological order)

1. Daugherty A, Rateri DL and **Victoria L. King**. IL-5 links adaptive and natural immunity in reducing atherosclerotic disease. *J. Clin. Invest.* 2004; 114: 317-19.
2. Daugherty A, Webb NR, Rateri DL, **King VL**. Cytokine regulation of macrophage functions in atherogenesis. *J Lipid Res.* Sep; 2005; 82(9):1812-22.
3. Cassis LA, Helton MJ, Howatt DA, **King VL**, Daugherty A. Aldosterone does not mediate angiotensin II-induced atherosclerosis and abdominal aortic aneurysms. *Brit J Pharm* 2005; 144(3):443-8.
4. Tannock LR, Kirk EA, **King VL**, LeBoeuf R, Wight TN, Chait A. Glucosamine supplementation accelerates early but not late atherosclerosis in LDL receptor deficient mice. *J. Nutr* 2006; Nov, 136(11): 2856-2861.
5. **King VL**, Lin AY, Ahluwalia N, Kristo F, Owens AP, Howatt DA, Shen D, Anderson, TJT, Tager AM, Luster AD, Daugherty A, Gerszten RE. Interferon- γ and the Interferon-Inducible Chemokine, CXCL10, Protect Against Aneurysm Formation and Rupture. *Circ* 2009; 119(3):426-35.
6. Kennedy AJ, Ellacott KLJ, **King VL**, Hasty AH. Mouse Models of the Metabolic Syndrome. *Dis Model Mech.* 2010; 3(3-4): 156-66.

D. Research Support:

Ongoing Research Support

1. R01 HL082835-05 King (PI) 2/01/08 – 1/31/14 (4.5 Calendar)
The role of Prostaglandin E2 in Angiotensin II-induced vascular disease
The proposed studies will determine the role of mPGES-1 generated PGE2 and the EP4 receptor in angiotensin II induced abdominal aortic aneurysm formation and atherosclerosis.

2. 8UL1TR000117-02 UK CCTS Pilot Project (PI) 7/1/12 – 12/31/13
The role of obesity, metabolic syndrome and diabetes on postprandial lipoprotein metabolism.
The proposed studies investigate Serum Amyloid A redistribution onto apolipoprotein B containing lipoproteins in diabetic and non-diabetic patients with metabolic syndrome in response to feeding a high fat diet.

Completed Research Support (within the past 3 years)

1. P20 RR021954 King (PI-Project 2) 9/08/08 – 6/30/12
Center for Research in Obesity and Cardiovascular Disease
The role mPGES-1 generated PGE2 in diet induced obesity.
The proposed studies investigate the role of mPGES-1 generated PGE2 in the development of atherosclerosis in model of diet induced obesity.
2. P01 HL080100 Webb (Co-I) 4/08/06 – 3/31/12
Mechanisms of Abdominal Aortic Aneurysm Formation
The proposed studies will determine the role of sPLA₂ on the development of AngII-induced abdominal aortic aneurysms.