

**Professor Qingbo Xu MBBS, MD, PhD**



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Professor Qingbo Xu studied Medicine at the Qingdao Medical School, China, where he obtained his MBBS in 1983. Between 1983 and 1988 he spent 5 years as a PhD student in Peking Union Medical School, Beijing, China. In the spring of 1988 he joined Professor Georg Wick's group at the Institute for Experimental Pathology, University of Innsbruck, Austria working as a postdoctoral fellow.

Meanwhile, he obtained his MD in 1992. As a Forgarty fellow he spent two years at the Laboratory of Biological Chemistry, National Institutes of Health, USA. In 1996 he returned to Austria employed by the Institute for Biomedical Aging Research, Austrian Academy of Sciences, where he created his own research group and promoted to Associate Professor. In 2000 he was appointed a Professor at St George's Hospital Medical School. Professor Xu was appointed as BHF John Parker Chair of Cardiovascular Science at King's College London in July 2006. He has published more than 150 original papers, and is often invited to present papers nationally and internationally.

**Research Areas:** Stem/progenitor cells in atherosclerosis, vascular proteomics, transgenic mouse models, signal transduction and gene expression.

**Research Interests**

1. Stem cells

2. The pathogenesis of atherosclerosis
  3. Vascular biology
  4. Animal models
  5. Proteomics
  6. Signal transduction

The Vascular Biology Unit is a basic science research unit. It undertakes a wide range of research in the field of cardiovascular diseases with the broad objective of improving the understanding of molecular mechanisms in the pathogenesis and the treatment of cardiovascular disease. The overarching aim of our study is to elucidate the molecular mechanisms underlying cardiovascular diseases, especially atherosclerosis. Our task is to bridge the gap between basic science research and its application through cardiology and cardiac surgery. In keeping with this, several research projects are carried out in the Unit as follows:

#### **Stem/progenitor cells in atherosclerosis**

Recent evidence indicates that stem/progenitor cells play a crucial part in the development of atherosclerosis and heart disease. Using mouse models, Professor Xu's group demonstrated that both endothelial cells and smooth muscle cells within atherosclerotic lesions of vein grafts and allografts were derived from stem/progenitor cells of the vessel wall and blood. Very recently his group found that abundant stem/progenitor cells exist in the arterial adventitia. These cells could have a therapeutic potential for cardiovascular diseases. His team is studying the contribution of stem/progenitor cells to the pathogenesis of atherosclerosis, and clarifying a potential use of these cells for therapy.

#### **Proteomics**

Since human genes have been sequenced, it is essential to understand functions of these genes in the pathophysiological conditions. Proteomics provides us an opportunity to study protein functions, modifications and interactions. We are using our in vitro and in vivo models to investigate the role of specific protein functions in the process of signalling for stem cell differentiation and for the pathogenesis of atherosclerosis. We have completed proteome mapping for stem/progenitor cells and vascular cells as well which were published in our specific website ([www.vascular-proteomics.com](http://www.vascular-proteomics.com)) as references. Eventually, these findings could lead to a new therapeutic strategy in the treatment of vascular diseases in humans.

### **Mechanical stress-initiated signalling**

His group has established an in vitro system studying mechanical stress-initiated signal transductions in SMCs and successfully cultivated vascular SMCs from transgenic and knockout mice. They discovered several signal pathways in SMCs that are initiated by mechanical stress, including PDGF receptor-ras-raf-ERK-AP-1, rac-PKC-p38MAPK, rac-HSF1-HSP70, MKP-1-regulated pathways. Recently, they also partially clarified the signal pathways for stem cell differentiation into endothelial and smooth muscle cells.

### **Mouse models of atherosclerosis**

Professor Xu and his co-workers have established the first mouse model of vein graft atherosclerosis. The features of this mouse vascular graft model resemble those of human venous bypass graft atheroma. His model has been proven to be powerful in the study of the pathogenesis and treatment of the vein graft disease. Many people from Europe and USA have visited his laboratory to learn the techniques for establishing the model, which is now used widely.

### **Selected Publications**

1. Foteinos G, Hu Y, Xiao Q, Metzler B and Xu Q. Rapid endothelial turnover in atherosclerosis-prone areas coincides with stem cell repair in apolipoprotein E-deficient mice. [Circulation](#). 2008; 117:1856-1863.
2. Xu Q. Stem cells and transplant arteriosclerosis. [Circ. Res](#). 2008; 102:1011-1024.
3. Mayr M, Zampetaki A, Sidibe A, Mayr U, Yin X, De Souza AI, Chung YL, Madhu B, Quax PH, Hu Y, Griffiths JR, and Xu Q. Proteomic and metabolomic analysis of smooth muscle cells derived from the arterial media and adventitial progenitors of apolipoprotein E-deficient mice. [Circ. Res](#). 2008;102:1046-1056.
4. Kiechl S, Schett G, Schwaiger J, Seppi K, Eder P, Egger G, Santer P, Mayr A, Xu Q, and Willeit J. Soluble receptor activator of nuclear factor-kappaB ligand and risk for cardiovascular disease. [Circulation](#) 2007;116:385-391.
5. Sidibe A, Yin X, Tarelli E, Xiao Q, Zampetaki A, Xu Q, Mayr M. Integrated membrane protein analysis of mature and embryonic stem cell-derived smooth muscle cells using a novel combination of Cy-dye/biotin labelling. [Mol. Cell Proteomics](#) 2007;6:1788-1797.
6. Zeng L, Xiao Q, Margariti A, Zhang Z, Zampetaki A, Patel S, Capogrossi MC, Hu Y, Xu Q. HDAC3 is crucial in shear- and VEGF-induced stem cell differentiation toward endothelial cells. [J. Cell Biol.](#) 2006;174:1059-1069.

7. Mayr U, Zou Y, Zhang Z, Dietrich H, Hu Y, Xu Q. Accelerated arteriosclerosis of vein grafts in inducible NO synthase(-/-) mice is related to decreased endothelial progenitor cell repair. [Circ Res](#). 2006;98:412-420.
8. Foteinos G, Afzal AR, Mandal K, Jahangiri M, Xu Q. Anti-heat shock protein 60 autoantibodies induce atherosclerosis in apolipoprotein E-deficient mice via endothelial damage. [Circulation](#). 2005;112:1206-1213.
9. Schett G, Kiechl S, Redlich K, Oberhollenzer F, Weger S, Egger G, Mayr A, Jocher J, Xu Q, Pietschmann P, Teitelbaum S, Smolen J, Willeit J. Soluble RANKL and risk of nontraumatic fracture. [JAMA](#). 2004;291:1108-1113.
10. Mayr M, Siow R, Chung YL, Mayr U, Griffiths JR, Xu Q. Proteomic and metabolomic analysis of vascular smooth muscle cells: role of PKCdelta. [Circ Res](#). 2004;94:e87-96.
11. Wick G, Knoflach M, Xu Q. Autoimmune and inflammatory mechanisms in atherosclerosis. [Annu Rev Immunol](#). 2004;22:361-403.
12. Hu Y, Zhang Z, Torsney E, Afzal AR, Davison F, Metzler B, Xu Q. Abundant progenitor cells in the adventitia contribute to atherosclerosis of vein grafts in ApoE-deficient mice. [J Clin Invest](#). 2004;113:1258-1265.
13. Torsney E, Mayr U, Zou Y, Thompson WD, Hu Y, Xu Q. Thrombosis and neointima formation in vein grafts are inhibited by locally applied aspirin through endothelial protection. [Circ Res](#). 2004;94:1466-1473.
14. Mandal K, Jahangiri M, Mukhin M, Poloniecki J, Camm AJ, Xu Q. Association of anti-heat shock protein 65 antibodies with development of postoperative atrial fibrillation. [Circulation](#). 2004;110:2588-2590.
15. Hu Y, Davison F, Zhang Z, Xu Q. Endothelial replacement and angiogenesis in arteriosclerotic lesions of allografts are contributed by circulating progenitor cells. [Circulation](#). 2003;108:3122-3127.
16. Mayr U, Mayr M, Li C, Wernig F, Dietrich H, Hu Y, Xu Q. Loss of p53 accelerates neointimal lesions of vein bypass grafts in mice. [Circ Res](#). 2002;90:197-204.
17. Hu Y, Mayr M, Metzler B, Erdel M, Davison F, Xu Q. Both donor and recipient origins of smooth muscle cells in vein graft atherosclerotic lesions. [Circ Res](#). 2002;91:e13-20.
18. Hu Y, Davison F, Ludewig B, Erdel M, Mayr M, Url M, Dietrich H, Xu Q. Smooth muscle cells in transplant atherosclerotic lesions are originated from recipients, but not bone marrow progenitor cells. [Circulation](#). 2002;106:1834-1839.

19. Leitges M, Mayr M, Braun U, Mayr U, Li C, Pfister G, Ghaffari-Tabrizi N, Baier G, Hu Y, Xu Q. Exacerbated vein graft arteriosclerosis in PKCdelta-null mice. [J. Clin. Invest.](#) 2001;108:1505-1512.
20. Kiechl S, Egger G, Mayr M, Wiedermann CJ, Bonora E, Oberhollenzer F, Muggeo M, Xu Q, Wick G, Poewe W, Willeit J. Chronic infections and the risk of carotid atherosclerosis: prospective results from a large population study. [Circulation.](#) 2001;103:1064-1070.