Dr Melanie Madhani

Lecturer in Cardiovascular Medicine

Cardiovascular and Respiratory Sciences

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Biography

Dr Melanie Madhani graduated from the University of Wales College Of Medicine in 1998 with a BSc (Hons) in Pharmacology. She obtained a PhD in Cardiovascular Sciences from the Ivy League School, Dartmouth College, New Hampshire, USA and University of Wales in 2002. She then joined University College London (UCL) as a post-doctoral research fellow, to work under Dr. Adrian Hobbs. Here, she investigated the role of cGMP in cardiovascular diseases. During her time at UCL, she was awarded a UCL Bogue Fellowship to work with Nobel Laureate Dr Louis Ignarro, University of California, Los Angeles, USA. In 2005, she joined Dr. Philip Eaton's research group at The Rayne Institute, King's College London, where she broadened her experience in cardiac physiology in order to complement her vascular knowledge and skills into cGMP regulation during myocardial ischaemia reperfusion injury. In 2009, Dr. Madhani was appointed as a Lecturer in Cardiovascular Medicine at the University of Birmingham. Currently, Melanie collaborates with Prof Frenneaux (University of Aberdeen), Prof Horowitz (Queen Elizabeth Hospital, University of Adelaide, Australia), and Prof Bonser (Queen Elizabeth Hospital, University of Birmingham). Melanie is currently an associate editor for Pharmacology and Therapeutics and Bulletin Editor for the British Society for Cardiovascular Research.

Teaching

- 2nd Year MBChB: Cardiovascular System
- 2nd Year BSc/B Med Sci Cardiovascular
- 3rd BSc/B Med Sci Cardiovascular

Research

Following myocardial infarction, a healing process results in replacement of ischaemic myocardium by a collagen rich scar. Even after scar formation there is continual activity in the border zone that regulates expansion of the infarct. The non-infarcted myocardium undergoes chronic remodelling over the following period such that the heart may become hypertrophic or dilated and function compromised. The extent of this later remodelling and degree of dysfunction is largely determined by infarct size and expansion. We are particularly interested to understand the mechanisms of myocardial injury during ischaemia and reperfusion. Of particular interest is to understand the physiological and pathological actions of nitric oxide-cGMP pathway on the cardiovascular system. These processes are interrogated with a range of in-vitro and in-vivo myocardial infarction models.

Other activities

- Associate Editor for International Review: Pharmacology & Therapeutics Journal
- Bulletin Editor for the British Society for Cardiovascular Research

Publications

Madhani M, Hall AR, Cuello F, Burgoyne JR, Fuller W, Hobbs AJ, Shattock MJ & Eaton P. Ser-69 phosphorylation of phospholemman contributes to Sildenafil-induced cardioprotection against reperfusion injury. **Am J Physio Heart** 2010; June 11

Baliga RS, Zhao L, Madhani M, Lopez-Torondel B, Visintin C, Selwood D, Wilkins MR, MacAllister RJ, Hobbs AJ. (2008) **Synergy between natriuretic peptides and phosphodiesterase 5 inhibitors ameliorates pulmonary arterial hypertension (http://www.ncbi.nlm.nih.gov/pubmed/18689467?ordinalpos=1&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_DefaultReportPanel.Pubmed_RVDocSum)**. **Am J Respir Crit Care Med.**15;178:861-9.

Burgoyne J, Madhani M, Cuello F, Brennan JP, Charles R L, Browning D and Eaton P. (2007) Interprotein disulfide bond formation activates PKGI independently of cGMP. **Science,** 317:1393-7.

Villar IC, Panayiotou CM, Sheraz A, Madhani M, Scotland RS, Nobles M, Kemp-Harper B, Ahluwalia A and Hobbs AJ. (2007) Definitive role for natriuretic peptide receptor-C in mediating the vasorelaxant activity of C-type natriuretic peptide and endothelium-derived hyperpolarising factor **Cardiovasc Res** 74:515-25.

Leiper J, Nandi M, Torondel B, Murray-Rust J, Malaki M, O'hara B, Rossiter S, Anthony S, Madhani M, Selwood D, Smith C, Wojciak-Stohard B, Rudiger A, Stidwell R, McDonald N Q, Vallance P. (2007) **Disruption of methylarginine metabolism impairs vascular homeostasis. (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=17273169&query_hl=1&itool=pubmed_docsum)** **Nat Med** ;13:198-203.

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