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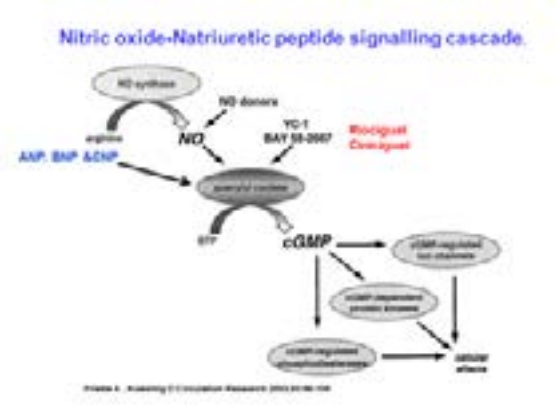
Advances in the genomics of blood pressure: Time for translation

Statement of the problem: Hypertension is the commonest cardiovascular worldwide with an anticipated 1.5 billion people with high blood pressure by 2025. It arises from a complex interplay between genes and lifestyle. From family studies between 30-50% of the heritability of blood pressure is due genetic influences or gene plus lifestyle. Approximately 8-12% of hypertensives cannot tolerate or are resistant to current therapies. Understanding the genes underpinning blood pressure could identify new biological pathways for innovative therapeutics. In genome wide studies of blood pressure now expanded to more than 1000 gene loci for blood pressure discovered and validated in over 1 million people. Many of these loci identify new biological pathways and some repurposing opportunities for existing therapies used for other disorders. A genetic risk score of all aggregate variants at 1000 loci suggested that in the over 50 year olds these loci cause a potential 10 mm Hg rise in blood pressure. This prompts the question is it tie to translate these findings into the clinic. First a targeted gene chip could identify those at risk in early life and enable lifestyle measures such as exercise a diet rich in fruit and vegetables, maintenance of an ideal body weight and reduced alcohol intake. In addition in mechanistic studies we and others have identified potential therapies acting on the nitric oxide-natriuretic peptide pathway including beetroot juice and c-natriuretic peptide mimetics. We can also now deploy next generation sequencing techniques to diagnose the cause of rare syndromic forms of hypertension and the impact of that will be explored.



Recent Publications

1. Warren HR, Evangelou E, Cabrera CP, Gao H, Ren M, Mifsud B, and multiple co-authors then, Caulfield M., Elliott P. Genome-wide association analysis identifies novel blood pressure loci and offers biological insights into cardiovascular risk. Nat Genet. 2017 Mar; 49(3):403-415. doi: 10.1038/ng.3768.

2. Georg B. Ehret then multiple co-authors then Mark J. Caulfield, Toby Johnson. Genetic variants from novel pathways influence blood pressure and cardiovascular disease risk. Nature 2011; 478(7367):103-9.

3. Louise V Wain, then multiple authors then Mark J Caulfield, Dabeeru C Rao, Martin D Tobin, Paul Elliott, Cornelia M van Duijn. Genome-wide association study identifies six new loci influencing pulse pressure and mean arterial pressure. Nature Genetics 2011 Sep 11; 43(10):1005-11.

4. Newton-Cheh C, then 152 co-authors then Elliott P, Abecasis GR, Caulfield M, Munroe PB. Genome-wide association study identifies eight loci associated with blood pressure. Nature Genetics 2009 May 10. [Epub ahead of print] PubMed PMID: 19430483.

Biography

Professor Mark Caulfield graduated in medicine in 1984 and trained in Clinical Pharmacology at St Bartholomew's Hospital where he developed a major programme in genetics of blood pressure regulation. In 2002 he became Co-Director of the William Harvey Research Institute at Queen Mary University of London which he grew from 140 to 530 clinicians and scientists and a major worldwide pharmacological centre focused on cardiovascular, inflammation and endocrine research. He was President of the British Hypertension Society and served on the council of the European Society of Hypertension. In 2013 he became an NIHR Senior Investigator and Chief Scientist for the 100,000 Genomes Project.

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