

# Breakthrough in understanding the genetics of high blood pressure

November 9 2011

---

A researcher from the University of Leicester's Department of Cardiovascular Sciences has been involved in a ground-breaking study into the causes of high blood pressure.

The study, published in the academic journal *Hypertension*, analysed [genetic material](#) in human kidneys in a search for genes that might contribute to high blood pressure. The findings open up new avenues for future investigation into the causes of high blood pressure in humans.

The study identified key genes, messenger RNAs and micro RNAs present in the kidneys that may contribute to human hypertension. It also uncovered two microRNAs that contribute to the regulation of renin – a hormone long thought to play a part in controlling blood pressure.

Although scientists have long known that the kidneys play a role in regulating blood pressure, this is the first time that key genes involved in the process have been identified through a large, comprehensive gene expression analysis of the human kidneys. It is also the first time that researchers have identified miRNAs that control the expression of the hormone renin.

The scientists studied tissue samples from the kidneys of 15 male hypertensive patients (patients with high blood pressure) and 7 male patients with normal blood pressure, and compared their messenger RNA (mRNA) and micro RNA (miRNA).

[Messenger RNA](#) (mRNA) is a single-stranded molecule that helps in the production of protein from DNA. Genetic information is copied from DNA to mRNA strands, which provide a template from which the cell can make new proteins. MicroRNA (miRNA) is a very small molecule that helps regulate the process of converting mRNA into proteins.

The study was co-authored by the University of Leicester's Dr Maciej Tomaszewski, Senior Clinical Lecturer in Cardiovascular Medicine in the Department of Cardiovascular Sciences, and a Consultant Physician in Leicester Blood Pressure Clinic - European Centre of Excellence.

Dr Tomaszewski commented: "I am very excited about this publication. Renin is one of the most important contributors to blood pressure regulation. The novel insights into its expression within the human kidney from this study open up new avenues for the development of new antihypertensive medications. The collection of hypertensive and normotensive kidneys is available for our studies in Leicester thanks to a long-term international collaboration. We will continue using this unique research resource in our further studies to decipher the genetic background of human hypertension."

Researchers described the discovery of these miRNAs as "the first real evidence to implicate renin" as a cause of high blood pressure. The findings also indicate which genes and miRNAs are involved in renin production. This increased understanding of the mechanisms underlying [hypertension](#) could lead to innovative new treatments for high blood pressure.

Researchers used samples of human kidneys stored in the Silesian Renal Tissue Bank (SRTB), all of which came from Polish males individuals of white European ancestry. The SRTB stores human kidney samples for use in genetic research into cardiovascular diseases. Samples were selected from 15 patients known to have [high blood pressure](#), along with

7 patients with normal [blood pressure](#) who were used as a control group for the study. The scientists used a range of techniques to study the [genes](#), mRNAs and miRNAs present in the medulla (the inner part of the kidney) and the cortex (the outer part).

**More information:** The research paper is available online at:  
[hyper.ahajournals.org/content/.../111.180729.abstract](http://hyper.ahajournals.org/content/.../111.180729.abstract)

Provided by University of Leicester

Citation: Breakthrough in understanding the genetics of high blood pressure (2011, November 9) retrieved 16 April 2024 from  
<https://medicalxpress.com/news/2011-11-breakthrough-genetics-high-blood-pressure.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.