Abstract

Introduction: This article attempts to summarise the genetic research that has taken place during the past decade to determine the identity of genes causing high blood pressure. Methods: Candidate gene studies and genome-wide scanning have been the methods primarily employed, and studies have been performed in both experimental models (rats and mice) and human volunteers (sibling-pairs and case-control). Key studies from the past 10 years are discussed, in addition to the congenic strains. Results: Genome-wide scans and candidate gene studies in both rat and man have generated many chromosomal regions and loci involved in blood pressure regulation. However, much work is still required to fine map the large chromosomal regions found in the genome-wide scans and to isolate variants in candidate genes and prove that they are disease-causing. Conclusions: It is anticipated that within the next 5 to 10 years at least one blood pressure susceptibility gene will be identified in rat and possibly some in man. It is hoped that the identification of genes controlling blood pressure will enable investigators to determine physiological/biochemical pathways defective in hypertensive patients. This information may then be utilised to identify specific hypertensive phenotypes, to tailor therapy appropriately for patients and hopefully to develop novel therapeutic agents for hypertension.

Key words: Candidate genes, Comparative mapping, Essential hypertension, Experimental models, Genome-wide screens, Quantitative trait loci