

## Molecular hereditary qualities of coronary artery disease.

Kaoru Tada\*

Department of Cardiology, Kanazawa University, Kanazawa, Japan

Positional cloning based on genome-wide linkage analysis of a large family identified MEF2A (encoding a transcription factor), the causative gene for coronary artery disease and the first non-lipid-related disease in myocardial infarction. MEF2A mutations can make up to 1.93% of the diseased population. Therefore, genetic testing based on MEF2A mutation analysis may soon be available to many patients with coronary artery disease / myocardial infarction. Genome-wide association studies have identified a significant association between myocardial infarction and the LTA gene (encoding lymph toxin  $\alpha$ ), and follow-up studies have also shown that the LTA interaction gene LGALS2 (encoding galectin 2) is also myocardial infarction susceptibility. It turned out to be a gene. Studies using genome-wide linkage scans with hundreds of small nuclear families revealed novels, including ALOX5AP (encoding 5-lipoxygenase-activating protein) and PDE4D (encoding phosphodiesterase 4D) associated with myocardial infarction and stroke. Coronary artery disease and myocardial infarction susceptibility genes have been identified. In the case of ischemic stroke [1].

Genetic studies provide new insights into the etiology of coronary artery disease and myocardial infarction. Future studies will identify genes for the causes and susceptibility of new diseases, understand the molecular mechanisms by which mutations cause coronary artery disease / myocardial infarction, and focus on gene-specific treatment of patients. Genes associated with complex human diseases such as CHD and MI can be divided into three main categories: disease-causing genes, susceptibility genes, and disease-related genes. Disease-causing genes are genes that are directly involved in the cause of the disease when mutated. In this case, mutations are clearly defined or established as the leading cause of the disease. For example, mutations in the potassium channel genes KCNQ1 and KCNH2 cause LQTS and mutations in the cardiac sodium channel gene SCN5A cause Brugada syndrome. Disease-causing genes are highly predictive and can be used directly for genetic testing. Susceptible genes are genes that increase or decrease the risk of developing a disease and may or may not cause the disease in connection with other genetic and environmental factors [2].

Mutations in these genes, or SNPs, are present in both the normal and diseased populations, but the frequency varies between the two populations. For individuals, susceptibility genes have a lower predictive value for the onset and prognosis of the disease. Disease-related genes are genes that

are involved in the disease by molecular biology, microarray, or proteomics analysis, but their relationship to the disease as a cause or result has not been established. Some disease-related genes function as biomarkers for the disease. This section describes the causative genes for CAD and MI, and the next two sections describe susceptibility genes and CAD / MI-related genes. The causative genes for familial hypercholesterolemia and tangier disease have been identified, both of which increase the risk of early atherosclerosis and CAD [3]. Hypercholesterolemia is characterized by increased LDL-binding plasma cholesterol levels, tendonitis, and atheroma, and mutations in the LDL receptor (LDLR) gene, apoB100 gene and the proprotein convertase subtilisin. Can be caused by Kexin type 9 gene (PCSK9), cholesterol 7 $\alpha$ -hydroxylase gene (CYP7A1) and ARH gene. Tanzier's disease is a rare disease characterized by lack of plasma HDL cholesterol and cholesterol ester deposition in the reticular endothelial system, with splenomegaly and enlarged tonsils and lymph nodes, hepatic splenoma and peripheral neuropathy. The Tangier disease gene has been identified as ABCA1 encoding a member of the ATP-binding cassette transporter family involved in the outflow of lipids from peripheral cells to ApoAI, thereby forming nascent HDL particles.

MEF2A, the only disease-causing gene encoding a member of the MEF2 family of transcription factors, has been identified in primary CAD and MI without other associated clinical features such as hypercholesterolemia and Tanzler's disease [4]. This discovery was named one of the top 10 advances of the American Heart Association in 2004. We assume that the disease behaves like a single gene disorder in a single large family of CAD and MI, and that the gene responsible for the disease can be mapped and identified by typical location cloning using model-based linkage analysis. To test this hypothesis, a genome-wide chain scan was performed on a large family of 13 patients showing autosomal dominant inheritance patterns of CAD and MI. This study led to the localization of the first loci of autosomal dominant CAD and MI on chromosome 15q26 (adCAD1). Further molecular genetic studies have established MEF2A as the gene that causes CAD and MI in families, and a 21 base pair deletion was confirmed in the last exons (Exxon 11) of MEF2A in all affected families. However, it was not confirmed in families with normal phenotype. Or in 119 people with normal angiography. Functional studies have shown that deletion of 21 base pairs disrupts MEF2A transcriptional activation activity via a dominant negative mechanism.

\*Correspondence to: Kaoru Tada, Department of Cardiology, Kanazawa University, Kanazawa, Japan, E-mail: [tdkr@med.kanazawa-u.ac.jp](mailto:tdkr@med.kanazawa-u.ac.jp)

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