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Genetic Variants in Artery Diseases

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Description

Despite a plethora of knowledge on predisposing risk factors and patho mechanisms, the precise molecular pathways that lead to Coronary Artery Disease (CAD) and Myocardial Infarction (MI) are unknown. CAD and MI are complicated genetic disorders that are caused by a combination of environmental and genetic variables. Atherosclerosis of the coronary arteries and subsequent clinical disease are caused by a combination of environmental and hereditary factors. The biological complexity of atherosclerotic disease stems from the unknown or unanticipated combinations of numerous genetic and environmental variables that have only been partially recognized on their own. Genetic differences in causal or susceptibility genes, according to current knowledge, provide the basis of biological mechanisms that, in combination with environmental factors, cause disease. In the hunt for chromosomal loci and candidate genes involved in these complicated disorders, researchers use linkage analysis, which follows 'disease' alleles in families, or genetic connection in a group of unrelated individuals. Progress in human genome sequencing and mapping, as well as efforts to discover all of the estimated one million Single Nucleotide Polymorphisms (SNPs) found in humans, will enable novel methodologies such as genome-wide association analyses. The current state of knowledge on human genetic diversity makes a sobering contribution to the deconstruction of CAD/MI as complex characteristics.

Raised expectations about the power of molecular genetic studies versus traditional pathophysiological experimental approaches, a lack of precise clinical phenotyping, a lack of functional characterization of gene variants, and the vast number of genes that have yet to be discovered may all play a role. Except for polymorphisms in lipid genes (e.g., apo E) and uncommon genetic variants (e.g., LDL receptor), which have a causal effect on both the intermediate (LDL-cholesterol level in plasma) and clinical phenotypes (CAD/MI), the role of most gene polymorphisms is debated or unknown. Despite huge advances in human genome sequencing and molecular genetics and bioinformatic approaches over the last decade, progress in mapping and identifying genes responsible for complex features like CAD/MI has been slow, posing a tremendous challenge to medical research in the twenty-first century [1,2].

Coronary Artery Disease (CAD) is a chronic inflammatory illness characterized by remodeling and constriction of the coronary arteries, which deliver oxygen to the heart. Stable angina, acute coronary syndrome, and sudden cardiac death are some of the clinical symptoms. It has a complicated etiopathogenesis linked to environmental factors like food, smoking, and physical exercise, as well as genetic factors 2 that modulate disease risk both individually and by interaction. The principal etiopathogenic process that causes CAD is atherosclerosis, and its progression is linked to a complex combination of environmental and genetic variables, with the latter having impacts either directly or through cardiovascular risk factors. Atherosclerosis is a silent, chronic condition marked by the accumulation of lipids, fibrous components, and inflammatory chemicals in the walls of major arteries. The outflow of Low-density Lipoprotein (LDL) cholesterol into the subendothelial region starts the process, which can then be changed and oxidised by a variety of factors. Oxidized/modified LDL particles are powerful chemotactic agents that increase monocyte adherence and migration to the subendothelial region by inducing production of vascular cell adhesion molecule and intercellular adhesion molecule at the endothelial surface.

In the intima medium, monocytes transform into macrophages. Different monocyte subsets have recently been identified, and their functions appear to differ depending on the stage of atherosclerosis in which they are involved. Macrophages have proinflammatory functions, such as the release of cytokines including interleukins and tumor necrosis factor and bind oxidized LDL *via* scavenger receptors to produce foam cells. The creation of the first classic atherosclerotic lesion, the fatty streak, in which foam cells are present in the subendothelial region, is the end outcome of this process. In the subendothelial region, other types of leukocytes, such as lymphocytes and mast cells, also accumulate. The interaction of monocytes, macrophages, foam cells, and T cells causes cellular and humoral immune responses, as well as a persistent inflammatory state with the production of many proinflammatory chemicals. The migration of smooth muscle cells from the artery's medial layer into the intima continues this process, culminating in the transformation from a fatty stripe to a more complex lesion. Smooth muscle cells create extracellular matrix molecules in the intima media, forming a fibrous cap that covers the original fatty streak. Foam cells inside the fibrous cap die and release lipids into the extracellular space, generating the necrotic core, a lipid-rich pool. As a result of this process, the second atherosclerotic lesion, the atherosclerotic plaque, forms. Traditional cardiovascular risk factors like hypertension, diabetes, dyslipidemia, and obesity, as well as CAD, are considered complex features generated by the interaction of hereditary and environmental variables. Genetic research can reveal new metabolic pathways linked to the onset and progression of atherosclerosis, as well as lead to the discovery of new pharmaceutical targets [3-5].

Conflict of Interest

None.

References

1. Sayols-Baixeras, Sergi, Carla Lluís-Ganella, Gavin Lucas, and Roberto Elosua. "Pathogenesis of coronary artery disease: Focus on genetic risk factors and identification of genetic variants." *Appl Clin Genet* 7 (2014): 15.
2. Waterworth, Dawn M. and Sally L. Ricketts. "Genetic variants influencing circulating lipid levels and risk of coronary artery disease." *Arterioscler Thromb Vasc Biol* 30 (2010): 2264-2276.
3. Takeuchi, Fumihiko, Masato Isono and Tomohiro Katsuya. "Association of genetic variants influencing lipid levels with coronary artery disease in Japanese individuals." (2012): e46385.
4. LeBlanc, Marissa, Verena Zuber and Bettina Kulle Andreassen. "Identifying novel gene variants in coronary artery disease and shared genes with several cardiovascular risk factors ." *Circulation Res* 118 (2016): 83-94.
5. Christiansen, Morten Krogh and Steen Dalby Kristensen. "Coronary artery disease-associated genetic variants and biomarkers of inflammation." *PloS one* 12 (2017): e0180365.

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