

The Detection of OLR1 RNA in Human Coronary Plaque Tissue

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Abstract: This investigation looked at whether the OLR1 gene plays a role in coronary artery disease. This was done by examining whether OLR1 RNA would be detectable in human coronary plaque tissue using NCBI Blast against RNA sequencing data. Coronary artery disease develops when cholesterol, lipids, and inflammatory cells build up in the coronary arteries, and OLR1 is a key gene involved with the disease since it plays a role in oxidized LDL uptake and foam-cell formation. The human OLR1 mRNA sequence was confirmed through BLAST and then compared to data from experiment SRX20907944, an RNA-seq dataset from human coronary-plaque tissue of people affected by the disease. The BLAST search produced 150-base alignments with 98.64%–100% identity and extremely small E-values (as low as 1×10^{-69}), demonstrating that OLR1 transcripts were present in the plaque sample. A separate BLAST analysis also detected OLR1 RNA in diseased aortic-valve tissue affected by calcific aortic valve disease, with 100% identity matches and E-values down to 2×10^{-69} . These findings support the hypothesis that OLR1 is found in atherosclerotic tissue and show that the gene plays a role in not only coronary artery disease, but also calcific aortic valve disease.

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I. INTRODUCTION

The circulatory system carries oxygen, nutrients, hormones, white blood cells, and antibodies throughout the body and helps remove metabolic waste. The coronary arteries supply oxygen-rich blood directly to the heart muscle. When these arteries become narrowed or blocked, the heart receives less oxygen and cannot work normally.

Coronary artery disease (CAD) develops when atherosclerotic plaque accumulates in the walls of the coronary arteries. This plaque includes cholesterol, lipids, and inflammatory cells. As the plaque grows, it reduces blood flow and can cause chest pain, also called angina. If a plaque breaks and a blood clot completely blocks the artery, a heart attack can occur. Major risk factors include high cholesterol, high blood pressure, smoking, diabetes, obesity, physical inactivity, age, and inherited genetic risk.

Genetic research has identified 60 genetic loci linked to coronary artery disease. These discoveries have helped researchers identify biological pathways involved in cholesterol transport, inflammation, vascular-cell behavior, and plaque development. Genetic studies have also supported treatments that target pathways such as PCSK9 and this has helped with precision-medicine approaches for identifying people with increased inherited risk.

OLR1 encodes oxidized low-density lipoprotein receptor 1, commonly called LOX-1. LOX-1 is a cell-surface receptor that binds and absorbs oxidized LDL. The intake of oxidized LDL can fill vascular cells and macrophages with cholesterol, contribute to foam-cell formation, and lead to atherosclerotic plaque.

The purpose was to determine whether OLR1 RNA could be detected in RNA-sequencing data obtained directly from a human coronary plaque. The hypothesis was that the OLR1 mRNA sequence would produce significant matches against the coronary-plaque RNA-seq sample because OLR1 is involved in oxidized-LDL uptake and therefore leads to atherosclerotic plaque.

The KEGG lipids and atherosclerosis pathway (hsa05417) was used, and five genes linked to fat handling, immune cells sticking to the valve, and plaque growth were also originally looked at.

Table 1 Five Genes Considered for the Coronary Artery Disease Investigation.

Gene	Relation to Coronary Artery Disease
APOB	This gene is related to the metabolism of cholesterol. One of the factors that build up to cause coronary artery disease.
CD36	The rs1761667 AG genotype is a small DNA change in the CD36 gene that raises CD36 activity and increases coronary artery disease risk.
OLR1	This gene can cause coronary artery disease because it pulls oxidized LDL into artery walls, creating foam cells and plaque that block blood flow.
VCAM1	This gene relates to coronary artery disease because it helps monocytes stick to artery walls. The VCAM1 mutation makes this process stronger, leading to bigger plaques.
SELP	Can relate to coronary artery disease because it helps blood cells stick to artery walls.

OLR1 RNA will be detectable in RNA-sequencing data from human coronary plaque because OLR1 encodes a receptor involved in oxidized-LDL uptake and foam-cell formation during coronary atherosclerosis.

II. METHODS

The human OLR1 mRNA code was found in the NCBI Nucleotide database. The OLR1 gene sequence was first confirmed using NCBI BLAST to ensure it matched the known human OLR1 gene.

Description	Scientific Name	Max Score	Total Score	Query Cover	E value	Per. Ident	Acc. Len	Accession
Homo sapiens chromosome 12 GRCh38.p14 Primary Assembly	Homo sapiens	3179	4297	100%	0.0	100.00%	133275309	NC_000012.12
Homo sapiens isolate CHM13 chromosome 12 alternate assembly T2T-CHM13v2.0	Homo sapiens	3149	4267	100%	0.0	99.65%	133324548	NC_060936.1

Fig 1 The Human OLR1 mRNA Sequence Maps to Chromosome 12 in the Human Genome.

Then, Standard Nucleotide BLAST was used and the database was changed to the Sequence Read Archive experiment SRX20907944. This experiment contains RNA-sequencing data from human coronary plaque tissue so we could see if OLR1 is found and therefore related.

The “somewhat similar sequences (blastn)” option was selected. After the search completed, the descriptions table was reviewed, and the number of displayed hits, alignment length, percent identity, query coverage, E-value, and read accessions were recorded. A significant match was defined as a high-identity alignment with an E-value below 0.001.

III. RESULTS

The SRA-BLAST search found significant alignments between the human OLR1 mRNA sequence and reads from coronary-plaque experiment SRX20907944. The visible reads were 150 nucleotides long and aligned to portions of the OLR1 transcript with approximately 98.64% to 100.00% identity. The strongest visible alignments had E-values of 1×10^{-69} , and other displayed alignments had E-values ranging from 4×10^{-69} to 2×10^{-29} . These extremely small E-values show that the alignments were not random matches. Together, these results demonstrate that OLR1 RNA was present in the human coronary-plaque sample.

Description	Max Score	Total Score	Query Cover	E value	Per. Ident	Acc. Len	Accession
SRX20907...	271	271	0%	1e-69	100.00%	150	SRR25159844.13286579.1
SRX20907...	271	271	0%	1e-69	100.00%	150	SRR25159844.11096428.1
SRX20907...	271	271	0%	1e-69	100.00%	150	SRR25159844.4274862.1
SRX20907...	269	269	0%	4e-69	100.00%	150	SRR25159844.11096428.2
SRX20907...	269	269	0%	4e-69	100.00%	150	SRR25159844.4274862.2
SRX20907...	267	267	0%	5e-68	99.33%	150	SRR25159844.5198371.1
SRX20907...	267	267	0%	5e-68	99.33%	150	SRR25159844.5197649.1
SRX20907...	265	265	0%	2e-67	99.33%	150	SRR25159844.5197649.2

Fig 2 NCBI SRA-BLAST Results for OLR1 mRNA (NM_002543.4) Against Coronary-Plaque Experiment SRX20907944. The Displayed Reads were 150 Bases Long, had 98.64%–100.00% Identity, and Included E-values as Low as 1×10^{-69} .

IV. CONCLUSION

The BLAST results supported the hypothesis that OLR1 RNA would be detectable in human coronary-plaque tissue. The high sequence identity, 150-base alignments, and extremely small E-values show direct evidence that OLR1 transcripts were present in the sample. This finding is consistent with the biological role of OLR1 in atherosclerosis because its protein product, LOX-1, binds oxidized LDL and participates in cellular lipid uptake and foam-cell formation.

The tissue sample showing OLR1 being expressed in people with coronary artery disease supports its link to the disease. Experiment SRX20907944 came from RNA taken directly from a human coronary plaque, so finding OLR1 in this sample shows that the gene was active in tissue affected by atherosclerosis.

Furthermore, calcific aortic valve disease (CAVD) is a condition where the aortic valve slowly becomes thick and

hard. It happens because calcium builds up on the valve over time. This buildup is not just aging but an active process caused by inflammation and fat deposits, similar to atherosclerosis. As the calcium increases, the valve becomes stiff and cannot open well. This makes it harder for blood to flow out of the heart. Mild cases cause slight thickening, while severe cases lead to aortic stenosis. If the disease gets worse, it can cause chest pain, shortness of breath, and heart failure.

An additional finding was that OLR1 RNA was also detected in a human aortic-valve RNA-seq sample affected by calcific aortic valve disease. In that separate BLAST search, at least 100 displayed reads were 150 bases long, matched OLR1 at 100% identity, and had E-values as low as 2×10^{-69} . This demonstrates that OLR1 were present in both coronary-plaque tissue and diseased aortic-valve tissue. This overlap matters because it means damaged fats and inflammation play a role in both plaque buildup in CAD and calcific aortic valve disease.

Description	Max Score	Total Score	Query Cover	E value	Per. Ident	Acc. Len	Accession
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.22807170.1
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.22834414.2
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.20966636.2
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.20966636.1
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.16941126.1
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.10535821.2
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.9779854.1
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.8666758.2
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.8667720.2
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.6283638.1
SRX20795...	271	271	6%	2e-69	100.00%	150	SRA-SRR25040676.6281500.1
SRX20795...	269	269	6%	7e-69	100.00%	150	SRA-SRR25040676.21802632.1
SRX20795...	269	269	6%	7e-69	100.00%	150	SRA-SRR25040676.21802629.1
SRX20795...	269	269	6%	7e-69	100.00%	150	SRA-SRR25040676.8666758.1
SRX20795...	269	269	6%	7e-69	100.00%	150	SRA-SRR25040676.8667720.1

Fig 3 NCBI SRA-BLAST Results Showing OLR1 RNA Matches in a Diseased Aortic-Valve RNA-seq Sample from Experiment SRX20795471. The Figure Shows that the Gene and the Sample had 100% Identity, and Included E-values as Low as 2×10^{-69} Meaning it wasn't due to Chance.

All of these results provide direct evidence that OLR1 was present in the plaque sample and show that OLR1 plays a role in coronary artery disease. A separate BLAST analysis also detected OLR1 RNA in diseased aortic-valve tissue, demonstrating that OLR1 transcripts were present in tissue affected by calcific aortic valve disease.

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