

**Supplementary Table S1.** Epigenetic biomarkers for atherosclerosis, carotid stenosis and vulnerable plaque.

<b>Epigenetic modification</b>	<b>Biomarker</b>	<b>Regulation in atherosclerosis</b>	<b>Sample source and type</b>	<b>Reference</b>
<b>DNA methylation</b>	5-mC	Decreased in atherosclerotic vs. non-atherosclerotic aortic tissue and in SCAS vs. ACAS	Human and animal aortic tissue and human carotid plaques	[1,2]
	ESR1/2 promoter region	Hypermethylated in CAS vs. controls	Human blood samples and HUVEC	[1,3]
	ABCA1 promoter region	Hypermethylated is related to atherosusceptibility	HAEC and pig aortic Tissue	[4]
	KLF4 promoter region	Hypermethylated in CAS vs. controls,	Human carotid plaques	[4]
	AIRE1 and ALOX12 promoter regions	Hypomethylated in high-calcified vs. low-calcified carotid plaques	Human carotid plaques	[5]
	RAMP1 promoter region	Hypomethylated in CAS vs. controls	Human carotid plaques	[6]
	Inflammation genes [PLA2G7]	Hypomethylated in vulnerable plaques	Human carotid plaques and blood	[7]
	DNMT1	Increased in atherosclerosis	Human blood and mice aortic tissue	[8]
		It negatively regulates arterial stiffening via maintaining the contractile phenotype of VSMCs	VSMCs Culture	[9]
	TET1	Increased in CAS vs. controls	Humans carotid plaques	[10]
	TET2	Its loss correlates with the degree of injury in murine models of vascular injury and	Human blood and murine bone marrow macrophages and VSMCs culture	[11–13]

<b>Histone PTMs</b>		human atherosclerotic disease		
	HDAC3	Its deficiency results in atherosclerosis	Mice aortic tissue and aortic SMC culture	[14]
	H3K9 and H3K27 methylation	Decreased in carotid plaques and significantly associated with disease severity	Human and animal carotid plaques, cardiac and aortic tissue and plaque-derived VSMCs culture	[1,10,14,15]
	EZH2	Its overexpression exaggerates atherosclerotic lesion	Mice aortic Tissue	[10]
	SIRT3	Its deficiency impairs endothelium-dependent relaxation without affecting plaque instability	Mice aortic Tissue	[10,16]
	JMJD3	Its depletion attenuates expression of pro-inflammatory genes	Mice bone marrow-derived and peritoneal macrophages	[10]
<b>miRNAs</b>	miR-let7c	Increased in hypertensive patients with atherosclerotic plaque	Human Serum	[17]
	miR-9-5p	Decreased in ACAS vs. controls	Human Serum	[18]
		Decreased as a CIE predictor	Human Serum	[18]
	miR-21	Decreased in vulnerable plaque	Micro-dissected fibrous caps in mice plaques	[19,20]
	miR-23a-5p	Increased in vulnerable plaque	Human plasma	[21]

miR-24-3p	Increased in vulnerable plaque	Human plasma	[22]
miR-27b-3p	Increased in vulnerable plaque	Human plasma	[22]
miR-28-5p	Decreased in ACAS vs. controls	Human Serum	[23]
miR-30a-5p	Increased in PN plaques vs. CC plaques	Human carotid plaques	[24]
miR-30d	Increased in PN plaques vs. CC plaques	Human carotid plaques	[24]
miR-92a	Increased in ACAS vs. controls	Human Serum	[25]
	Increased in SCAS vs. ACAS*	Human plasma	[26]
	Increased as a CIE predictor	Human Serum	[25]
miR-100	Increased in SCAS vs. ACAS	Human carotid plaques	[27–29]
miR-106-5p	Increased in ACAS vs. controls	Human Serum	[27]
	Increased as a CIE predictor	Human Serum	[27]
miR-124-3p	Increased in SCAS vs. ACAS	Human Serum	[30]
miR-125a	Increased in SCAS vs. ACAS	Human carotid plaques	[27,28]
miR-126	Increased in vulnerable plaque	Human plasma	[31]
miR-127	Increased in SCAS vs. ACAS	Human carotid plaques	[27–29]
miR-130a-3p	Increased in vulnerable plaque	Human plasma	[22]

miR-133a	Increased in SCAS vs. ACAS	Human carotid plaque	[28,29]
miR-133a-3p	Decreased in SCAS vs. ACAS	Human Serum	[30]
miR-133b	Increased in SCAS vs. ACAS	Human carotid plaques	[28,29]
	Increased in vulnerable plaque	Human carotid plaques	[32]
miR-134-5p	Increased in SCAS vs. ACAS	Human Serum	[30]
miR-143	Increased in SCAS vs. ACAS*	Human plasma	[26]
miR-145	Increased in SCAS vs. ACAS	Human carotid plaques	[28,29]
	Increased in vulnerable plaque	Human plasma	[31]
	Increased in SCAS vs. ACAS*	Human plasma	[26]
miR-145-5p	Increased in hypertensive patients with atherosclerotic plaque	Human Serum	[17]
miR-146a	Increased in CAS vs. controls	Human Serum	[33]
	Increased in vulnerable plaque	Human Serum	[33]
miR-186-5p	Increased in ACAS vs. controls	Human Serum	[34]
	Increased as a CIE predictor	Human Serum	[34]

miR-199b-3p	Increased in vulnerable plaque	Human plasma	[22]
miR-200c	Increased in vulnerable plaque	Human carotid plaques	[35]
miR-206-3p	Decreased in CAS vs. controls	Mice left carotid artery tissue	[36]
miR-210	Decreased in vulnerable plaque	Human plasma sampled at the carotid lesion site	[37]
	Increased in SCAS vs. ACAS*	Human plasma	[26]
miR-216a	Increased in atherosclerosis progression	Human acute monocytic leukemia cell line	[38]
miR-216b	Decreased in unstable plaque	Human Serum	[39]
miR-221	Increased in SCAS vs. ACAS	Human carotid plaques	[28,29]
	Decreased in vulnerable plaque	Human carotid plaques shoulder	[40]
miR-221-3p	Increased in vulnerable plaque	Human plasma	[22]
miR-222	Decreased in vulnerable plaque	Human carotid plaques shoulder	[40]
miR-320b	Decreased in vulnerable plaque	Human Serum	[41]

**lncRNA**

miR-322-5p	Increased in CAS vs. controls	Mice left carotid artery tissue	[36]
miR-330-5p	Increased in vulnerable plaque	Human carotid plaques	[42]
miR-342-5p	Increased in ACAS vs. controls	Human Serum	[43]
	Increased as a CIE predictor	Human Serum	[43]
miR-466h-5p	Increased in vulnerable plaque	Mice left carotid artery tissue	[36]
miR-483-5p	Increased in ACAS vs. controls	Human Serum	[44]
	Increased as a CIE predictor	Human Serum	[44]
miR-503-5p	Decreased in ACAS vs. controls	Human Serum	[45]
miR-532-3p	Decreased in vulnerable plaque	HEK-293T cells	[46]
miR-638	Decreased in CAS vs. controls	Human Serum	[47]
miR-4530	Increased in vulnerable plaque	Human carotid plaques	[32]
ANRIL	Increased in atherosclerosis vs. non-atherosclerosis	Human atherosclerotic coronary Plaques	[48]
MIAT	Increased in atherosclerosis vs. non-atherosclerosis	Human atherosclerotic coronary plaque	[48]
	Increased in ACAS vs. controls and in SCAS vs. ACAS	Human serum	[49]

	Increased in atherosclerosis progression	Plaque and serum of an advanced atherosclerosis mouse model	[49]
	Increased in CAS vs. control and in vulnerable plaque	Human and murine carotid plaques	[50]
MALAT1	Decreased in atherosclerosis vs. non-atherosclerosis	Human atherosclerotic coronary plaque	[48]
lincRNA-p21	Decreased in atherosclerotic vs. non-atherosclerotic tissue	Mice aortic atherosclerotic plaques	[51]
LINC01123	Increased in CAS vs. controls	Human serum and carotid plaques	[52]
PEBP1P2	Decreased in atherosclerosis	Human serum of CAD patients and human advanced carotid plaques	[53]
NEAT1	Increased in CAS and CAD vs. controls	Human carotid plaques vs healthy arterial tissue and CAD PBMCs	[54]
CHROME	Increased in SCAS and ACAS vs. controls and in CAD vs controls	Human carotid plaques and plasma from CAD patients	[55]
UC.98	Increased in vulnerable plaques and involved in atherosclerotic progression	Human blood from ACS patients and MAEC culture	[56]
MSTRG.11455.17	Increased in unstable vs. stable plaques	Human carotid plaques	[57]
MSTRG.12845	Increased in unstable vs. stable plaques	Human carotid plaques	[57]

circRNA	CCAT2	Increased in unstable vs. stable plaques and vs. controls	Human serum of patients with carotid plaques	[39]
	PELATON	Increased in unstable vs. stable plaques	Human carotid plaques	[58]
	circANRIL	Increased in human vascular tissue	Human carotid plaques	[59]
		Its inhibition reduces vascular endothelial injury, oxidative stress and inflammation	Rat coronary artery tissue	[60]
	circ0003575	Increased in oxidized LDL treated cells	HUVEC culture	[61]
	circ0030042	Decreased in vulnerable plaques	Mice aortic root plaques	[62]
	circ0044073	Upregulated in CAS vs. controls	Human blood cells	[63]
	circR284	Increased in SCAS vs. ACAS	Human Serum	[64]
	circ0006896	Increased in unstable vs. stable plaques	Human serum exosomes of patients with carotid plaques	[65]
	circ000411	Decreased in unstable vs. stable plaques	Human carotid plaques	[57]
	circ0010729	Increased in dysfunctional vascular endothelial cells	HUVEC culture	[66]

ACAS: asymptomatic carotid artery stenosis. ACS: acute coronary syndrome. CAD: coronary artery disease. CAS: carotid artery stenosis. CIE: cerebrovascular ischaemic event. HAEC: human aortic endothelial cells. HUVEC: human umbilical vein endothelial cells. MAEC: murine aortic endothelial cells. SCAS: symptomatic carotid artery stenosis. SCM: smooth muscle cells. VSMCs: vascular smooth muscle cells. \*Not statistically significant, a tendency to be validated.

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