# PYRUVAT KINASE M2 ISOFORM ENZYME AND ATHEROSECLEROSIS

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| **ABSTRACT** Atherosclerosis is a chronic inflammatory vascular condition that causes coronary heart disease, and peripheral vascular disease. Which caused plaques in the intima (1). The metabolic risk factors activate endothelial cells (ECs), lead to endothelial dysfunction causes local blood mononuclear cell infiltration (2–4). The recruited monocytes differentiate into macrophages subsequently, which engulf large amounts of oxidized -low density lipoprotein (Ox-LDL), leading to lipid agregation and foam cell formation. Foamy macrophages infiltrate the lipid accumulation into the pathological intima-thickening lesions, inducing their transformation into necrotic cores. In the next step, smooth muscle cells (SMCs) migrate from the medium to the intima, proliferation & migration, plaques ocure. ECs, immune cells, and smooth muscle cells (SMCs) participate in atherosclerotic plaque formation (5) when arterial walls get thickened by fat and fibrous tissue. As the arterial lumen narrows, the plaque hardens and eventually ruptures (6,7). According to recent studies, PKM2-dependent glycolysis promotes the proliferation and migration of vascular smooth muscle cells (VSMCs), gamma interferon (IFN-γ) induces reversible metabolic reprogramming to sustain proinflammatory activity, and Homocysteinemia activates the glycolysis-lipogenic pathway in CD4+ T cells via PKM2 (8–10). Herein that pyruvate kinase M2 isoform enzyme depended glycolysis play critical role in atherosclerosis progression and treatment. **References:** 1. Kobiyama K, Ley K. Atherosclerosis. Circ Res [Internet]. 2018 Oct 26 [cited 2023 Dec 16];123(10):1118–20. Available from: https://www.ahajournals.org/doi/abs/10.1161/CIRCRESAHA.118.3138162. Libby P. Inflammation in atherosclerosis. Arterioscler Thromb Vasc Biol [Internet]. 2012 Sep [cited 2023 Dec 16];32(9):2045–51. Available from: https://pubmed.ncbi.nlm.nih.gov/22895665/3. Lu H, Daugherty A. Atherosclerosis. Arterioscler Thromb Vasc Biol. 2015 Mar 1;35(3):485–91. 4. Libby P, Buring JE, Badimon L, Hansson GK, Deanfield J, Bittencourt MS, et al. Atherosclerosis. Nature Reviews Disease Primers 2019 5:1 [Internet]. 2019 Aug 16 [cited 2023 Dec 16];5(1):1–18. Available from: https://www.nature.com/articles/s41572-019-0106-z5. Johnson JL. Emerging regulators of vascular smooth muscle cell function in the development and progression of atherosclerosis. Cardiovasc Res [Internet]. 2014 Sep 1 [cited 2023 Dec 16];103(4):452–60. Available from: https://dx.doi.org/10.1093/cvr/cvu1716. Ahmadi A, Leipsic J, Blankstein R, Taylor C, Hecht H, Stone GW, et al. Do Plaques Rapidly Progress Prior to Myocardial Infarction? Circ Res [Internet]. 2015 Jun 19 [cited 2023 Dec 16];117(1):99–104. Available from: https://www.ahajournals.org/doi/abs/10.1161/circresaha.117.3056377. Qiao JH, Walts AE, Fishbein MC. The severity of atherosclerosis at sites of plaque rupture with occlusive thrombosis in saphenous vein coronary artery bypass grafts. Am Heart J. 1991 Oct 1;122(4):955–8. 8. Zhao X, Tan F, Cao X, Cao Z, Li B, Shen Z, et al. PKM2-dependent glycolysis promotes the proliferation and migration of vascular smooth muscle cells during atherosclerosis. Acta Biochim Biophys Sin (Shanghai) [Internet]. 2019 Jan 3 [cited 2023 Dec 16];52(1):9–17. Available from: https://www.sciengine.com/10.1093/abbs/gmz1359. Yang Q, Xu J, Ma Q, Liu Z, Sudhahar V, Cao Y, et al. PRKAA1/AMPKα1-driven glycolysis in endothelial cells exposed to disturbed flow protects against atherosclerosis. Nat Commun [Internet]. 2018 Nov 7 [cited 2023 Dec 16];9(1):4667–4667. Available from: https://europepmc.org/articles/PMC622020710. Lü S, Deng J, Liu H, Liu B, Yang J, Miao Y, et al. PKM2-dependent metabolic reprogramming in CD4+ T cells is crucial for hyperhomocysteinemia-accelerated atherosclerosis. J Mol Med [Internet]. 2018 Jun 1 [cited 2023 Dec 16];96(6):585–600. Available from: https://link.springer.com/article/10.1007/s00109-018-1645-6  |

# Keywords: Atheroseclerosis, Cornary Heart Disease, PKM2.

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