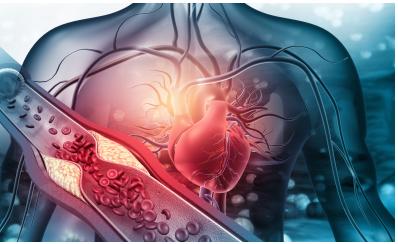
Novel peptide therapy for vascular inflammation

Atherosclerosis is driven by chronic vascular inflammation, lipid accumulation and immune cell recruitment, leading to plaque formation and vessel narrowing. Even in optimally treated patients, substantial residual inflammatory risk remains. CXCR4 is widely expressed but has a key role on leukocytes and aortic endothelial cells: activation by the pro-inflammatory cytokine MIF drives vascular inflammation, foam cell formation and plaque progression, whereas CXCR4 engagement by CXCL12 can exert both cell-dependent atheroprotective and disease-promoting effects.



IP RIGHTS

PCT filed in AU, CA, EP, US

2021

Chemokine-selective peptide therapy in Atherosclerosis

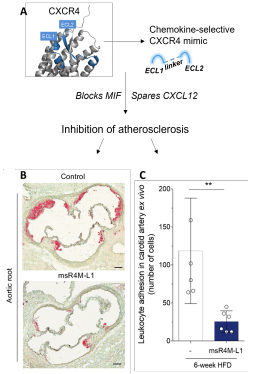
The invention provides soluble CXCR4-mimicking peptides built from engineered ECL1/ECL2 loop sequences linked by a short spacer. These small, stable molecules selectively bind MIF but not CXCL12, localize to atherosclerotic plaques and block MIF-driven leukocyte recruitment and foam cell formation, thereby directly targeting residual inflammatory risk.

01 Chemokine-selective CXCR4 mimetic peptides block MIF-driven atherosclerosis while sparing CXCL12/CXCR4

02 Short synthetic peptides (<30 aa) enable cost-efficient, robust and scalable manufacturing

03 High affinity and stability with targeted accumulation in atherosclerotic plaques

04 Platform concept expandable to other chemokine receptors and inflammatory diseases



A) CXCR4-mimicking peptides are engineered from the ECL1/ECL2 loops and linked by a short spacer to form a soluble ectodomain that selectively binds MIF but not CXCL12. B) Aortic root sections from atherosclerotic mice show reduced inflammatory plaque burden after msR4M-L1 treatment versus control. C) Ex vivo carotid artery model demonstrating that msR4M-L1 markedly decreases leukocyte adhesion to atherosclerotic vessels.

REFERENCES:

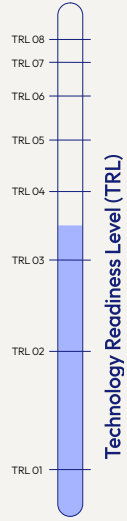
Kontos et al., Nature Communications vol. 11, 5981 (2020)

CHALLENGE

Atherosclerosis is driven by chronic vascular inflammation, lipid deposition and chemokine-mediated immune cell recruitment, leading to plaque growth and unstable lesions. In many patients, a high residual inflammatory risk persists despite optimal lipid-lowering therapy. Existing anti-inflammatory and chemokine-targeted drugs often lack selectivity and disrupt protective pathways, underscoring the need for pathway-specific interventions.

INNOVATION

The approach uses soluble CXCR4- mimicking peptides that bind selectively to the pro-inflammatory cytokine MIF while sparing the atheroprotective CXCL12/CXCR4 axis. These small, stable peptides localize to atherosclerotic plaques, inhibit MIF-driven leukocyte recruitment, foam cell formation and vascular inflammation, and reduce plaque burden in relevant preclinical models. This chemokine-selective, plaque-targeted mode of action directly addresses residual inflammatory risk and provides a modular template for extending the concept to other chemokine receptors and indications.



01 Basic principles observed · **02** Technology concept formulated · **03** Experimental proof of concept · **04** Technology validated in lab · **05** Technology validated in relevant environment · **06** Technology demonstrated in relevant environment · **07** System prototype demonstrated in operational environment · **08** System complete and qualified

BayPAT

Innovation from Research & Science – BayPAT markets inventions on behalf of the Bavarian universities and universities of applied sciences.

An invention of **Technical University of Munich**

Technische Universität München

As one of the largest technology transfer offices in Germany, BayPAT evaluates and commercializes innovations from 28 research institutions. With our extensive experience and expertise in patenting and licensing technologies and our comprehensive range of services, we are your partner of choice. For more information visit www.baypat.de

Bayerische Patentallianz GmbH
Dr. Linda Keil
lkeil@baypat.de
+49 (0)89 5480177-30
Prinzregentenstr. 52
80538 München