

ATAGENIX LABORATORIES

Catalog Number:ATMP00013HU Recombinant Human CD47 protein ,C- His Tag

Product Details

Summary

English name Recombinant Human CD47 protein ,C- His Tag

Purity >90% as determined by SDS-PAGE

Endotoxin level <1.0 EU per μg of the protein as determined by the LAL method.

Construction A DNA sequence encoding the human CD47(Met1~Glu141) was fused with the C-

terminal His Tag

Accession # Q08722

Host Mammalian cells

Species Homo sapiens (Human)

Predicted Molecular Mass 15.32kDa

Formulation Supplied as solution form in 50 mM Tris-HCl, pH 7.5, 150 mM NaCl or lyophilized

from 50 mM Tris-HCl, pH 7.5, 150 mM NaCl.

Shipping In general, proteins are provided as lyophilized powder/frozen liquid. They are

shipped out with dry ice/blue ice unless customers require otherwise.

Stability &Storage Use a manual defrost freezer and avoid repeated freeze thaw cycles.

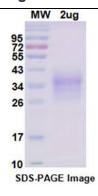
Store at 2 to 8 °C for one week .

Store at -20 to -80 °C for twelve months from the date of receipt.

Reconstitution Reconstitute in sterile water for a stock solution. A copy of datasheet will be

provided with the products, please refer to it for details.

SDS-PAGE image

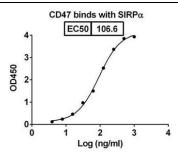




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Bioactivity



CD47(ATMP00013HU) bind with SIRPa(ATMP01936HU) measured by functional ELISA assay

Background

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Leukocyte surface antigen CD47 is also known as Antigenic surface determinant protein OA3, Integrin-associated protein (IAP) and Protein MER6. CD47 contains 1 Ig-like V-type (immunoglobulin-like) domain. CD47 is very broadly distributed on normal adult tissues. CD47 has a role in both cell adhesion by acting as an adhesion receptor for THBS1 on platelets, and in the modulation of integrins and plays an important role in memory formation and synaptic plasticity in the hippocampus by similarity. CD47 is the receptor for SIRPA, binding to which prevents maturation of immature dendritic cells and inhibits cytokine production by mature dendritic cells. CD47 Interaction with SIRPG mediates cell-cell adhesion, enhances superantigen-dependent T-cell-mediated proliferation and costimulates T-cell activation.

Alternative Names

CD47,MER6,IAP,OA3

References

Ghimire, Li, Chiba, Julovi, Li, Ross, Straub, O'Connell, Rüegg, Pagano, Isenberg, Rogers (2020) CD47 Promotes Age-Associated Deterioration in Angiogenesis,

Blood Flow and Glucose Homeostasis Cells 9(7)

Frontier progress

The aged population is currently at its highest level in human history and is expected to increase further in the coming years. In humans, aging is accompanied by impaired angiogenesis, diminished blood flow and altered metabolism, among others. A cellular mechanism that impinges upon these manifestations of aging can be a suitable target for therapeutic intervention. Here we identify cell surface receptor CD47 as a novel age-sensitive driver of vascular and metabolic dysfunction. With the natural aging process, CD47 and its ligand thrombospondin-1 were increased, concurrent with a reduction of self-renewal transcription factors OCT4, SOX2, KLF4 and cMYC (OSKM) in arteries from aged wild-type mice and older human subjects compared to younger controls. These perturbations were prevented in arteries from aged CD47-null mice. Arterial endothelial cells isolated



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from aged wild-type mice displayed cellular exhaustion with decreased proliferation, migration and tube formation compared to cells from aged CD47-null mice. CD47 suppressed ex vivo sprouting, in vivo angiogenesis and skeletal muscle blood flow in aged wild-type mice. Treatment of arteries from older humans with a CD47 blocking antibody mitigated the age-related deterioration in angiogenesis. Finally, aged CD47-null mice were resistant to age- and diet-associated weight gain, glucose intolerance and insulin desensitization. These results indicate that the CD47-mediated signaling maladapts during aging to broadly impair endothelial self-renewal, angiogenesis, perfusion and glucose homeostasis. Our findings provide a strong rationale for therapeutically targeting CD47 to minimize these dysfunctions during aging.