Recombinant Human CCL21/6Ckine

Catalog Number: 366-6C/CF

DESCRIPTION

Source	*E. coli*-derived human CCL21/6Ckine protein Ser24-Pro134 Accession # Q6ICR7
N-terminal Sequence Analysis	Ser24
Predicted Molecular Mass	12 kDa

SPECIFICATIONS

SDS-PAGE	16-17 kDa, reducing conditions
Activity	Measured by its ability to chemoattract BaF3 mouse pro-B cells transfected with human CCR7. The ED_{50} for this effect is 3-15 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

CCL21, also known as 6Ckine, TCA-4, SLC, Exodus-2, and A21, is a 12 kDa homeostatic chemokine that plays an important role in adaptive immune responses and inflammation (1). Unlike other CC chemokines, human CCL21 has a 37 amino acid (aa) C-terminal extension which mediates its attachment to carbohydrate structures and extracellular matrix components (2, 3). Mature human CCL21 shares 71% and 66% aa sequence identity with mouse and rat CCL21, respectively (4-6). Both human and mouse CCL21 signal through the chemokine receptor CCR7, while mouse CCL21 additionally can signal through CXCR3 (7). CCL21 is constitutively presented on initial lymphatic vessels, high endothelial venules (HEV), and lymph node dendritic cells (DC) (8-10). Immobilized CCL21 promotes the docking of DC to lymphatic vessels and the retention of T cells by lymph node DC, resulting in T cell priming for activation (8, 9). DC interaction with the anchored chemokine can induce CCL21 cleavage and release of an 8 kDa fragment that lacks the C-terminal extension (10). During chronic inflammation or tissue damage, CCL21 is up-regulated on local vascular endothelial cells, macrophages, T cells, and neurons (11-14). In these settings, it promotes fibrosis, inflammatory cytokine production, and neuropathic pain (12-14). The soluble chemokine is elevated in rheumatoid arthritis synovial fluid and in the serum of coronary artery disease patients (11, 13). CCL21 has been shown to exert either angiogenic or angiostatic effects (11, 15, 16). These effects, in combination with the ability of CCL21 to attract immune suppressor cells (Treg and MDSC) to a tumor site can have positive or negative effects on tumor progression (16, 17).

References:

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