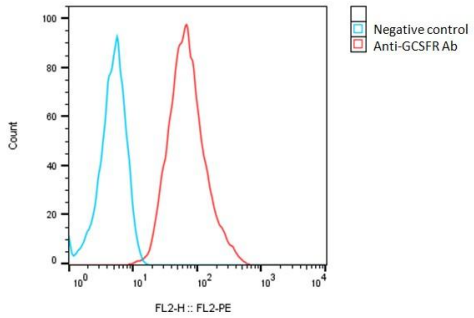
Human GCSFR-Ba/F3 Stable Cell Line
Catalog Number: C3077

SPECIFICATIONS

Catalog Number	C3077
Cell Line Name	Human GCSFR-Ba/F3 stable cell line
Accession Number	NM_000760.4
Host Cell	Suspension Ba/F3
Quantity	Two vials of frozen cells ($2x10^6$ per vial)
Culture Medium	DMEM/10% FBS, 2 ng/mL mIL-3 , 1 ug/ml Puromycin
Freezing Medium	90% FBS and 10% DMSO
Storage	Liquid nitrogen

DATA

Detection of human GCSFR expression on human GCSFR-Ba/F3 stable cells using a monoclonal antibody specific for human GCSFR (BD Pharmingen Cat#554538)



BACKGROUND

Granulocyte colony-stimulating factor receptor (GCSFR) is a crucial cell surface receptor of the cytokine receptor family that plays a pivotal role in regulating the production, differentiation, and function of neutrophils. GCSFR consists of an extracellular ligand-binding domain responsible for interacting with its ligand, granulocyte colony-stimulating factor (G-CSF). Upon binding to its ligand, GCSFR initiates a signaling cascade that promotes the proliferation, differentiation, and survival of myeloid progenitor cells, particularly granulocyte precursors, leading to the production and release of mature neutrophils into the bloodstream. This process, known as granulopoiesis, is crucial for maintaining a sufficient neutrophil count to combat infections and other inflammatory conditions. GCSFR is primarily expressed on the surface of cells within the myeloid lineage, including hematopoietic stem cells, myeloid progenitors, and mature neutrophils and reported in certain non-hematopoietic tissues under specific conditions, suggesting potential roles beyond hematopoiesis. Aberrant expression or dysregulation of GCSFR has been implicated in various hematological malignancies and solid tumors. In some cases, overexpression of GCSFR has been associated with disease progression, increased tumor aggressiveness, and poorer prognosis. Conversely, downregulation or loss of GCSFR expression may contribute to impaired immune surveillance and tumor escape mechanisms. Given its critical role in granulopoiesis and its involvement in cancer pathogenesis, GCSFR represents an attractive therapeutic target for various hematological malignancies and solid tumors. Strategies aimed at modulating GCSFR signaling, such as monoclonal antibodies targeting the receptor or its ligand, small molecule inhibitors, or immunomodulatory therapies, hold promise for the development of novel cancer treatments and immunotherapies.

References

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