ZBTB7B Protein, Human (Sf9, His, Strep)

Cat. No.:	HY-P701600
Synonyms:	ZBTB7B; Zinc finger and BTB domain-containing protein 7B; Krueppel-related zinc finger protein cKrox; hcKrox; T-helper-inducing POZ/Krueppel-like factor; Zinc finger and BTB domain-containing protein 15; Zinc finger protein 67 homolog; Zfp-67; Zinc finger protein 857B; Zinc finger protein Th-POK
Species:	Human
Source:	Sf9 insect cells
Accession:	O15156 (G2-S539)
Gene ID:	51043
Molecular Weight:	

PROPERTIES

Appearance	Solution.
Formulation	Supplied as a 0.22 μm filtered solution of 50 mM Tris-HCl, pH7.5, 200 mM NaCl, 20% glycerol.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	Please use rapid thawing with running water to thaw the protein.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice.

DESCRIPTION

Background

ZBTB7B, a transcription regulator, plays a pivotal role in determining the lineage commitment of immature T-cell precursors, acting as a key determinant of CD4 and CD8 cell fates. Its tissue-specific functions are evident in mammary epithelial cells and T cells. ZBTB7B is indispensable and sufficient for CD4 commitment, counteracted by its absence leading to CD8 commitment. The development of immature T-cell precursors correlates precisely with their T-cell receptor specificity for major histocompatibility complex class II or class I molecules. The interplay between ZBTB7B and CBF complexes is decisive in the CD4 versus CD8 cell fate decision. ZBTB7B suppresses RUNX3 expression and imposes CD4+ lineage fate by inducing SOCS suppressors of cytokine signaling. It functions as a transcriptional activator for SOCS genes, repressing RUNX3 expression and promoting CD4+ lineage fate. During CD4 lineage commitment, it associates with multiple sites at the CD8 locus, acting as a negative regulator through epigenetic silencing by recruiting class II histone deacetylases, such as HDAC4 and HDAC5. Additionally, ZBTB7B acts as a metabolic regulator in lactating mammary glands, serving as a critical feed-forward regulator of insulin signaling by directly regulating the expression of insulin receptor substrate-1 (IRS-1) and insulin-induced Akt-mTOR-SREBP signaling. It functions as a transcriptional repressor of collagen genes and may also repress fibronectin and other extracellular matrix genes. Moreover, ZBTB7B is a potent driver of brown fat development, thermogenesis, and cold-induced beige fat formation, recruiting the brown fat lncRNA 1 (Blnc1):HNRNPU ribonucleoprotein

complex to activate thermogenic gene expression in brown and beige adipocytes. ZBTB7B homodimerizes and interacts with various partners such as NCL, NEDD4, YBX1, HNRNPU, HDAC4, and HDAC5, each interaction contributing to its multifaceted regulatory functions.

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