DCAF1 抗原（重组蛋白）

中文名称： DCAF1 抗原（重组蛋白）

英文名称： DCAF1 Antigen (Recombinant Protein)

别　　名： DDB1 and CUL4 associated factor ; RIP; VPRBP

相关类别： 抗原

储　　存： 冷冻（-20℃）

概述

Fusion protein corresponding to a region derived from 1308-1507 amino acids of human DCAF1

技术规格

Full name:	DDB1 and CUL4 associated factor
Synonyms:	RIP; VPRBP
Swissprot:	Q9Y4B6
Gene Accession:	BC022792
Purity:	>85%, as determined by Coomassie blue stained SDS-PAGE
Expression system:	Escherichia coli
Tags:	His tag C-Terminus, GST tag N-Terminus
Background:	Acts both as a substrate recognition component of E3 ubiquitin-protein ligase complexes and as an atypical serine/threonine-protein kinase, playing key roles in various processes such as cell cycle, telomerase regulation and histone modification. Probable substrate-specific adapter of a DCX (DDB1-CUL4-X-box) E3 ubiquitin-protein ligase complex, named CUL4A-RBX1-DDB1-DCAF1/VPRBP complex, which mediates ubiquitination and proteasome-dependent degradation of proteins such as NF2. Involved in the turnover of methylated proteins: recognizes and binds methylated proteins via its chromo domain, leading t

	o ubiquitination of target proteins by the RBX1-DDB1-DCAF1/VPRBP complex (PubMed:23063525). The CUL4A-RBX1-DDB1-DCAF1/VPRBP complex is also involved in B-cell development: DCAF1 is recruited by RAG1 to ubiquitinate proteins, leading to limit error-prone repair during V(D)J recombination. Also part of the EDVP complex, an E3 ligase complex that mediates ubiquitination of proteins such as TERT, leading to TERT degradation and telomerase inhibition (PubMed:23362280). Also acts as an atypical serine/threonine-protein kinase that specifically mediates phosphorylation of 'Thr-120' of histone H2A (H2AT120ph) in a nucleosomal context, thereby repressing transcription. H2AT120ph is present in the regulatory region of many tumor suppressor genes, down-regulates their transcription and is present at high level in a number of tumors (PubMed:24140421). Involved in JNK-mediated apoptosis during cell competition process via its interaction with LLGL1 and LLGL2 (PubMed:20644714).