human PD-1, His-Tag

Programmed cell death protein 1, immune checkpoint receptor

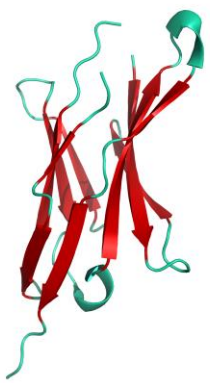
Cat. no. P2020-163

Product Information

Protein:	human PD-1, His-Tag (~ 18.7 kDa)
Uniprot#:	Q15116
Sequence:	MLDSPDRPWNPPTFSPALLVVTEGDNATFTCSFSNTSESFVLNWYRMSPSNQTDKLAAFPE DRSQPGQDCRFRVTQLPNGRDFHMSVVRARRNDSGTYLCGAISLAPKAQIKESLRAELRV TERRAEVPTAHPSPSPRPAGQFQT Methionine at pos. 1 might be present due to cloning constraints, C-terminal His-tag not shown in sequence.
Source:	Recombinantly expressed in HEK293.
Tag(s):	His-tag, C-terminal
Purification:	Purified by affinity chromatography and subsequent buffer exchange.
Formulation:	PBS; pH 7.4. Liquid, stored and shipped at -80 °C.
Purity:	> 95 % (will be determined by densitometry of Coomassie stained gel, example next page)
Concentration:	Will be determined by BCA-Assay.
Long-term storage:	No recommendations.
Comment:	Protein migrates at higher molecular weight during SDS-PAGE due to posttranslational modifications.

Background Information:

Activated T cells express the programmed cell death protein 1 (PD-1), which belongs to the CD28 family of the immunoglobulin superfamily. As immune checkpoint receptor, PD-1 represents a key player in modulating adaptive immune responses to prevent both excessive stimulation of the immune system and tissue damage. It interacts with two ligands, PD-L1 and PD-L2. PD-L1 is expressed on antigen-presenting cells (APCs) and many other tissue cells, whereas PD-L2 is mainly expressed on APCs. Structurally, PD-1 consists of an extracellular immunoglobulin domain, a transmembrane domain, and a cytoplasmic tail. The cytoplasmic tail contains two tyrosine-based signaling motifs, referred to as immunoreceptor tyrosine-based inhibitory motif (ITIM) and immunoreceptor tyrosine-based switch motif (ITSM). Engagement of PD-1 with its ligands results in the recruitment of phosphatases to these signaling motifs und induces inhibitory signals

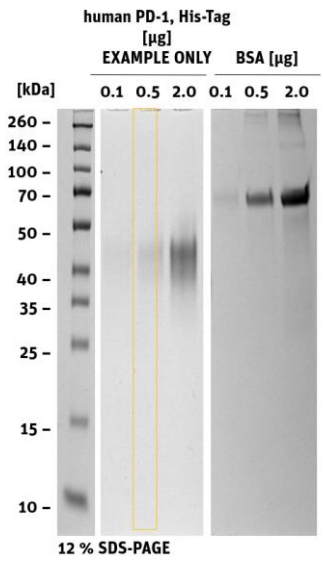


Structural model of human PD-1

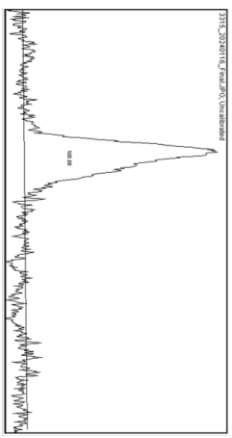
Product Information

that suppress T cell activation. In contrast to CTLA-4, PD-1 is not a competitive inhibitor, but inhibits kinase-dependent signals from CD28 and T cell receptor (TCR) upon ligand engagement. Moreover, PD-1 rather limits the responses of effector T cells, predominantly CD8+ T cells, in peripheral tissues. This regulatory function is essential for preventing autoimmunity and maintaining immune tolerance. Cancer cells often upregulate the expression of PD-L1, enabling interaction with PD-1 on infiltrating T cells and thus, inducing immune tolerance within the tumor microenvironment. This immune evasion strategy allows cancer cells to escape destruction by the immune system, promoting tumor growth and progression. Immune checkpoint inhibitors, such as monoclonal antibodies targeting PD-1, have demonstrated remarkable clinical efficacy in various cancer types by unleashing the anti-tumor immune response.

Quality Information (provided for each lot):



SDS-PAGE/Coll.Coomassie



	Area	Percent
1	17781.995	100

Histogram (of marked lane in gel picture)