

Product datasheet for **KN307584RB**

Havcr2 Mouse Gene Knockout Kit (CRISPR)

Product data:

Product Type:	Knockout Kits (CRISPR)
Format:	2 gRNA vectors, 1 RFP-BSD donor, 1 scramble control
Donor DNA:	RFP-BSD
Symbol:	Havcr2
Locus ID:	171285
Components:	<p>KN307584G1, Havcr2 gRNA vector 1 in pCas-Guide CRISPR vector (GE100002)</p> <p>KN307584G2, Havcr2 gRNA vector 2 in pCas-Guide CRISPR vector (GE100002)</p> <p>KN307584RBD, donor DNA containing left and right homologous arms and RFP-BSD functional cassette.</p> <p>GE100003, scramble sequence in pCas-Guide vector</p>
Disclaimer:	<p>These products are manufactured and supplied by OriGene under license from ERS. The kit is designed based on the best knowledge of CRISPR technology. The system has been functionally validated for knocking-in the cassette downstream the native promoter. The efficiency of the knock-out varies due to the nature of the biology and the complexity of the experimental process.</p>
RefSeq:	<u>NM_134250</u>
UniProt ID:	<u>Q8VIM0</u>
Synonyms:	TIM-3; Tim3; Timd3



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Summary:

Cell surface receptor implicated in modulating innate and adaptive immune responses. Generally accepted to have an inhibiting function. Reports on stimulating functions suggest that the activity may be influenced by the cellular context and/or the respective ligand (PubMed:18006747). Regulates macrophage activation (PubMed:11823861). Inhibits T-helper type 1 lymphocyte (Th1)-mediated auto- and alloimmune responses and promotes immunological tolerance (PubMed:14556006, PubMed:18006747). In CD8+ cells attenuates TCR-induced signaling, specifically by blocking NF-kappaB and NFAT promoter activities resulting in the loss of IL-2 secretion. The function may implicate its association with LCK proposed to impair phosphorylation of TCR subunits (By similarity). In contrast, shown to activate TCR-induced signaling in T-cells probably implicating ZAP70, LCP2, LCK and FYN (PubMed:21807895). Expressed on Treg cells can inhibit Th17 cell responses (By similarity). Receptor for LGALS9. Binding to LGALS9 is believed to result in suppression of T-cell responses; the resulting apoptosis of antigen-specific cells may implicate HAVCR2 phosphorylation and disruption of its association with BAG6 (PubMed:22863785). Binding to LGALS9 is proposed to be involved in innate immune response to intracellular pathogens. Expressed on Th1 cells interacts with LGALS9 expressed on Mycobacterium tuberculosis-infected macrophages to stimulate antibactericidal activity including IL-1 beta secretion and to restrict intracellular bacterial growth (PubMed:20937702). However, the function as receptor for LGALS9 has been challenged (By similarity). Also reported to enhance CD8+ T-cell responses to an acute infection such as by Listeria monocytogenes (PubMed:24567532). Receptor for phosphatidylserine (PtSer); PtSer-binding is calcium-dependent (PubMed:20083673). May recognize PtSer on apoptotic cells leading to their phagocytosis. Mediates the engulfment of apoptotic cells by dendritic cells (PubMed:19224762). Expressed on T-cells, promotes conjugation but not engulfment of apoptotic cells (PubMed:20083673). Expressed on dendritic cells (DCs) positively regulates innate immune response and in synergy with Toll-like receptors promotes secretion of TNF-alpha (PubMed:18006747). In tumor-infiltrating DCs suppresses nucleic acid-mediated innate immune response by interaction with HMGB1 and interfering with nucleic acid-sensing and trafficking of nucleic acids to endosomes (PubMed:22842346). Can enhance mast cell production of Th2 cytokines IL-4, IL-6 and IL-13 (PubMed:17620455). Expressed on natural killer (NK) cells acts as a coreceptor to enhance IFN-gamma production in response to LGALS9. In contrast, shown to suppress NK cell-mediated cytotoxicity (By similarity). Negatively regulates NK cell function in LPS-induced endotoxic shock (PubMed:25337993).[UniProtKB/Swiss-Prot Function]

Product images:

