

**DESCRIPTION**

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human PD-1 in direct ELISAs.
<b>Source</b>	Recombinant Monoclonal Rabbit IgG Clone # 2705J
<b>Purification</b>	Protein A or G purified from cell culture supernatant
<b>Immunogen</b>	Human PD-1 synthetic peptide
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

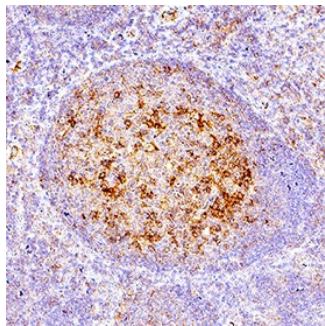
**APPLICATIONS**

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Immunohistochemistry</b>	3-25 µg/mL	Immersion fixed paraffin-embedded sections of human tonsil

**DATA**

**Immunohistochemistry**



**PD-1 in Human Tonsil.** PD-1 was detected in immersion fixed paraffin-embedded sections of human tonsil using Rabbit Anti-Human PD-1 Monoclonal Antibody (Catalog # MAB10858) at 3 µg/mL for 1 hour at room temperature followed by incubation with the Anti-Rabbit IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC003). Before incubation with the primary antibody, tissue was subjected to heat-induced epitope retrieval using Antigen Retrieval Reagent-Basic (Catalog # CTS013). Tissue was stained using DAB (brown) and counterstained with hematoxylin (blue). Specific staining was localized to lymphocytes in germinal centers. Staining was performed using our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 0.5 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

Programmed Death-1 receptor (PD-1), also known as CD279, is type I transmembrane protein belonging to the CD28 family of immune regulatory receptors (1). Other members of this family include CD28, CTLA-4, ICOS, and BTLA (2-5). Mature human PD-1 consists of a 148 amino acid (aa) extracellular region (ECD) with one immunoglobulin-like V-type domain, a 24 aa transmembrane domain, and a 95 aa cytoplasmic region. The human PD-1 ECD shares 65% aa sequence identity with the mouse PD-1 ECD. The cytoplasmic tail contains two tyrosine residues that form the immunoreceptor tyrosine-based inhibitory motif (ITIM) and immunoreceptor tyrosine-based switch motif (ITSM) that are important for mediating PD-1 signaling. PD-1 acts as a monomeric receptor and interacts in a 1:1 stoichiometric ratio with its ligands PD-L1 (B7-H1) and PD-L2 (B7-DC) (6, 7). PD-1 is expressed on activated T cells, B cells, monocytes, and dendritic cells while PD-L1 expression is constitutive on the same cells and also on nonhematopoietic cells such as lung endothelial cells and hepatocytes (8, 9). Ligand of PD-L1 with PD-1 induces co-inhibitory signals on T cells promoting their apoptosis, anergy, and functional exhaustion (10). Thus, the PD-1: PD-L1 interaction is a key regulator of the threshold of immune response and peripheral immune tolerance (11). Finally, blockade of the PD-1: PD-L1 interaction by either antibodies or genetic manipulation accelerates tumor eradication and shows potential for improving cancer immunotherapy (12, 13, 14).

**References:**

1. Ishida, Y. *et al.* (1992) *EMBO J.* **11**:3887.
2. Sharpe, A.H. and G. J. Freeman (2002) *Nat. Rev. Immunol.* **2**:116.
3. Coyle, A. and J. Gutierrez-Ramos (2001) *Nat. Immunol.* **2**:203.
4. Nishimura, H. and T. Honjo (2001) *Trends Immunol.* **22**:265.
5. Watanabe, N *et al.* (2003) *Nat. Immunol.* **4**:670.
6. Zhang, X. *et al.* (2004) *Immunity* **20**:337.
7. Lázár-Molnár, E. *et al.* (2008) *Proc. Natl. Acad. Sci. USA* **105**:10483.
8. Nishimura, H *et al.* (1996) *Int. Immunol.* **8**:773.
9. Keir, M.E. *et al.* (2008) *Annu. Rev. Immunol.* **26**:677.
10. Butte, M.J. *et al.* (2007) *Immunity* **27**:111.
11. Okazaki, T. *et al.* (2013) *Nat. Immunol.* **14**:1212.
12. Iwai, Y. *et al.* (2002) *Proc. Natl. Acad. Sci. USA* **99**: 12293.
13. Nogrady, B. (2014) *Nature* **513**:S10.
14. Swaika, A. *et al.* (2015) *Mol. Immunol.* **67**: 4