

Technical Data Sheet

FlowMAb PE anti-mouse PD-1 (CD279)



Attention: Use of this product constitutes an agreement to Bio X Cell's Terms and Conditions which are included with this product in print and can also be found at <https://bioxcell.com/terms-and-conditions>.

Lot Specific Information

Lot Number: Lot Specific*
Volume: Lot Specific*
Concentration: Lot Specific* (generally 4 to 11 mg/ml) *
Total Protein: Lot Specific*

*This information will be noted on the certificate of analysis that ships with this product.

Product Information

Catalog Number: FM0273-PE
Clone: 29F.1A12™
Isotype: Rat IgG2a
Conjugation: PE
Excitation Source: Yellow-Green 488 nm, 532 nm, 561 nm
Excitation Max: 496 nm, 566 nm
Emission Max: 576 nm
Recommended Dilution Buffer: InVivoPure pH 7.0 Dilution Buffer
Immunogen: Recombinant PD-1-Ig fusion protein
Reported Applications: *in vivo* blocking of PD-1/PD-L signaling
in vitro PD-1 neutralization
Immunohistochemistry (frozen)
Immunofluorescence
Western blot
Flow cytometry
Formulation: PBS, pH 7.0
Contains 0.09% Sodium Azide
Endotoxin: <2EU/mg (<0.002EU/μg)
Determined by LAL gel clotting assay
Purity: >95%
Determined by SDS-PAGE
Sterility: 0.2 μm filtration
Production: Purified from cell culture supernatant in an animal-free facility
Purification: Protein G
RRID: [AB_2687796](https://abnova.com/AB_2687796)
Molecular Weight: 150 kDa

Description

The 29F.1A12™ monoclonal antibody reacts with mouse PD-1 (programmed death-1) also known as CD279. PD-1 is a 50-55 kDa cell surface receptor encoded by the *Pdcd1* gene that belongs to the CD28 family of the Ig superfamily. PD-1 is transiently expressed on CD4 and CD8 thymocytes as well as activated T and B lymphocytes and myeloid cells. PD-1 expression declines after successful elimination of antigen. Additionally, *Pdcd1* mRNA is expressed in developing B lymphocytes during the pro-B-cell stage. PD-1's structure includes a ITIM (immunoreceptor tyrosine-based inhibitory motif) suggesting that PD-1 negatively regulates TCR signals. PD-1 signals via binding its two ligands, PD-L1 and PD-L2 both members of the B7 family. Upon ligand binding, PD-1 signaling inhibits T-cell activation, leading to reduced proliferation,

cytokine production, and T-cell death. Additionally, PD-1 is known to play key roles in peripheral tolerance and prevention of autoimmune disease in mice as PD-1 knockout animals show dilated cardiomyopathy, splenomegaly, and loss of peripheral tolerance. Induced PD-L1 expression is common in many tumors including squamous cell carcinoma, colon adenocarcinoma, and breast adenocarcinoma. PD-L1 overexpression results in increased resistance of tumor cells to CD8 T cell mediated lysis. In mouse models of melanoma, tumor growth can be transiently arrested via treatment with antibodies which block the interaction between PD-L1 and its receptor PD-1. For these reasons anti-PD-1 mediated immunotherapies are currently being explored as cancer treatments. Like the RMP1-14 and J43 antibodies the 29F.1A12™ antibody has been shown to block the binding of PD-1 to its ligands in vivo.

Storage

Store at the stock concentration at 4°C . **Do not freeze.**

It is not uncommon for a floccule or precipitate to appear during storage. The floccule is typically buffer salts precipitating out of solution or a small bit of protein aggregation. For information on how to remove floccules or precipitates see our FAQ's at <https://bioxcell.com/faqs>.

Protocol Information

Since applications vary, each investigator should use the application references as a guide to help estimate the appropriate dose or concentration. The dose or concentration can be further optimized experimentally in a dose response or titration experiment.

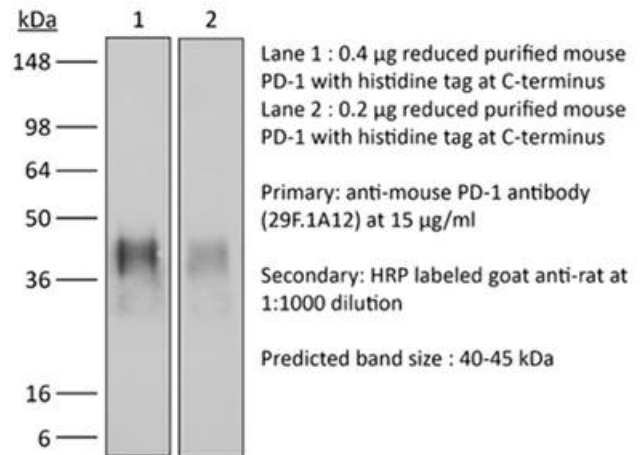
Application References

For a complete list of references, visit https://bioxcell.com/fm0273-pe?bxcs=9k1b3a#tab_references or scan the QR code below.



Binding Validation

Validation data shown below confirms that this clone binds to its target antigen. For lot specific binding validation data, e-mail technicalservice@bioxcell.com.



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