

Novocastra™ Lyophilized Mouse Monoclonal Antibody Plasminogen Activator Inhibitor (Type 1)

Product Code: NCL-PAI-1

Intended Use	FOR RESEARCH USE ONLY.
Specificity	Human plasminogen activator inhibitor type 1 (PAI-1).
Clone	TJA6
Ig Class	IgG2b, kappa
Antigen Used for Immunizations	Prokaryotic recombinant protein corresponding to a 250 amino acid portion of the N-terminus of the human PAI-1 molecule.
Hybridoma Partner	Mouse myeloma (p3-NS1-Ag4-1).
Preparation	Lyophilized tissue culture supernatant containing 15 mM sodium azide. Reconstitute with 1 mL or 0.1 mL of sterile distilled water as indicated on vial label.
Effective on Frozen Tissue	Not fully evaluated.
Effective on Paraffin Wax Embedded Tissue	Yes
Recommendations on Use	Immunohistochemistry: Typical working dilution 1:20–1:40. 60 minutes primary antibody incubation at 25 °C. Standard ABC technique. Western Blotting: Not recommended.
Positive Controls	Immunohistochemistry: Tonsil.
Staining Pattern	Cytoplasmic.
Storage and Stability	Store unopened lyophilized antibody at 4 °C. Under these conditions, there is no significant loss in product performance up to the expiry date indicated on the vial label. The reconstituted antibody is stable for at least two months when stored at 4 °C. For long term storage, it is recommended that aliquots of the antibody are frozen at -20 °C (frost-free freezers are not recommended). Repeated freezing and thawing must be avoided. Prepare working dilutions on the day of use.
General Overview	Plasminogen activator inhibitor type 1 (PAI-1) is a 48 kD protein which inhibits the conversion of plasminogen to plasmin. It is the principal inhibitor of the plasminogen activators t-PA and u-PA. PAI-1 is structurally related to the serine protease inhibitor (serpin) superfamily. The serpins are known to undergo a conformational rearrangement upon cleavage of the reactive central peptide bond (P1-P1') and it is this conformational difference between the active and cleaved forms which determines their reactivity.
General References	Dublin E, Hanby A, Patel N K, et al. <i>American Journal of Pathology</i> . 157 (4): 1219–1227 (2000). de Witte J H, Sweep C G, Klijn J G, et al.. <i>British Journal of Cancer</i> . 80 (1–2): 286–294 (1999). Harbeck N, Thomssen C, Berger U, et al.. <i>Breast Cancer Research Treatment</i> . 54 (2): 147–157 (1999). Arai Y, Kubota T, Nakagawa T, et al.. <i>Acta Neurochir. (Wien)</i> . 140 (4): 377–385 (1998). Strojan P, Budihna M, Smid L, et al.. <i>European Journal of Cancer</i> . 34 (8): 1193–1197 (1998). Sweep C G, Geurts-Moespot J, Grebenschikov N, et al.. <i>British Journal of Cancer</i> . 78 (11): 1434–1441 (1998). Speiser P, Mayerhofer K, Kucera E, et al.. <i>Anticancer Research</i> . 17 (1B): 679–683 (1997). Ito H, Yonemura Y, Fujita H, et al.. <i>Virchows Arch</i> . 427 (5): 487–496 (1996). Zellinger R, Eder S, Schneeberger C, et al.. <i>Anticancer Research</i> . 16 (1): 449–453 (1996). Sancho E, Declerck P J, Price N C, et al.. <i>Biochemistry</i> . 34 (3): 1064–1069 (1995). Keijer J, Linders M, van Zonneveld A-J, et al.. <i>Blood</i> . 78 (2): 401–409 (1991). Munch M, Heegaard C, Jensen P H, et al.. <i>FEBS</i> . 295 (1,2,3): 102–106 (1991).

