

Designation	246-D
Product	human monoclonal antibody to HIV-1 gp41 epitope QQLLGIWG
Product no.	AB013
Lot no.	xxx
Shelf life	unopened at least until: mm yyyy
Volume	xxx µl
Concentration	x.xx mg/ml
Method of analysis	OD 280 nm
Isotype	IgG1 (κ)
Host cell	hetero-hybridoma
Purification	protein A affinity chromatography
Product buffer	PBS – sterile, no preservatives <i>Handling under non-sterile conditions can cause contamination leading to protein degradation!</i>
Formulation	liquid
Storage conditions	+ 2-8°C

Note: For use as research reagent only

The permission to use the cell line for the manufacture of this product was obtained from New York University School of Medicine, 650 First Avenue, 6 Floor, New York, NY 10016, USA. Special acknowledgement is given to Dr. Susan Zolla-Pazner and Dr. Mirosław K. Gorny, both New York University School of Medicine, who established and characterized this antibody.

See page 2 for more detailed information on antibody 246-D

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Polymun Scientific Immunbiologische Forschung GmbH, March 30th 2010

Human mAb 246-D was generated using a cellular method (1) from an asymptomatic HIV-1-positive individual living in the New York area. It reacts in ELISA with a gp41 peptide spanning amino acids 579-613 of gp160. mAb 246-D binds to overlapping hexapeptides which identified its core epitope as qqLLGIwg (amino acids 591-598) which is located in the immunodominant region (cluster I) of gp41, just upstream from the disulfide loop (amino acids 598-604) (1); the capital letters represent the core epitope and the lowercase letters, the flanking amino acids which probably contribute to the binding of the epitope. 246-D does not bind to either the peptide complex N51/C43 that approximates the core of the fusogenic form of gp41 or to the individual peptides N51 or C43 that form this structure (2). MAb 246-D recognizes a conserved epitope and binds strongly to all tested intact primary isolates of clades A, B, C, D, F and CRF01 (clade E) (3, 4).

MAb 246-D has no neutralizing activity but mediates ADCC activity (5). MAb 246-D was shown to inhibit HIV-1 BaL replication in macrophages but not in PHA-stimulated PBMCs. It is suggested that this occurs by an IgG-Fc γ R-dependent interaction leading to endocytosis and degradation of HIV particles. It is also suggested that mAb 246-D will not impair virus entry into PBMCs, but that it could participate in the protection of mucosal HIV transmission by preventing the infection of macrophages and dendritic cells (6).

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