

This product is for research use only (not for diagnostic or therapeutic use)

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## Product no AS16 3113

## Anti-Transthyretin 56-61, amyloid specific (mouse monoclonal)

## **Product information**

**Immunogen** Recombinant protein corresponding to the Human wild type Transthyretin.

GPTGTGESKCPLMVKVLDAVRGSPAINVAVHVFRKAADDTWEPFASGKTSESGELH GLTTEEFVEGIYKVEIDTKSYWKALGISPFHEHAEVVFTANDSGPRRYTIAALLSPYS

YSTTAVVTNPKE The epitope has been mapped to residue 56-61

**Host** Mouse

Clonality | Monoclonal

Subclass/isotype | IgG1

**Purity** Affinity purified in PBS pH 7.4.

Format Lyophilized

Quantity 100 μg

**Reconstitution** Add 100 μl sterile water to reconstitute to 1 mg/ml

Store lyophilized/reconstituted at 4°C, Please remember to spin the tubes briefly prior to opening them to avoid any

losses that might occur from material adhering to the cap or sides of the tube.

## **Application information**

Recommended dilution 1:1000 (ELISA), 1:500 (IHC), 1:1000 (WB)

Expected | apparent

MW

Confirmed reactivity | Human Transthyretin Amyloids

155

Not reactive in No confirmed exceptions from predicted reactivity are currently known

Additional information | Specifica

Specifically reactive to the amyloid form of human Transthyretin. Epitope mapped to residue 56-61 which remains buried within the native fold of transthyretin but becomes exposed within its amyloid form.

It has been suggested that that two distinct mechanisms of TTR-amyloidosis exists. The first, most common seen in wild type TTR Amyloidosis, consists of the full length TTR. Whereas the other type of amyloidosis mainly consists of the C-terminal region of the protein and is more common in mutant versions of TTR. Mouse IgG1 Anti-Transthyretin 56-61 (Amyloid Specific) epitope is located at the C-terminal strand of cleaved TTR and is suitable to detect amyloid formation derived from the C-terminal.

Selected references

Goldsteins et al. (1999). Exposure of cryptic epitopes on transthyretin only in amyloid and in amyloidogenic mutants.

Proc Natl Acad Sci U S A. 1999 Mar 16; 96(6): 3108-3113