

Recombinant Prealbumin/Transthyretin Monoclonal Antibody

catalog number: **AN302048L**

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

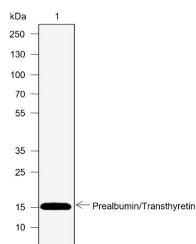
Description

Reactivity	Human;
Immunogen	Peptide. This information is proprietary to PTMab.
Host	Rabbit
Isotype	IgG, κ
Clone	A768
Purification	Protein A purified
Buffer	PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protein protectant.

Applications Recommended Dilution

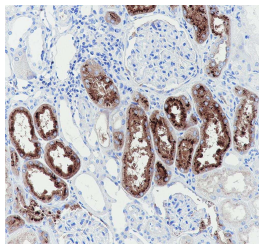
WB	1:1000
IHC	1:1000

Data

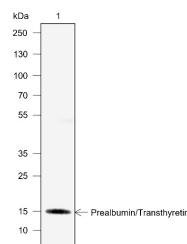


Western Blot with Prealbumin/Transthyretin Monoclonal Antibody at dilution of 1:1000. Lane 1: Human plasma

Observed-MW:35 kDa
Calculated-MW:16 kDa



Immunohistochemistry of paraffin-embedded Human kidney using Prealbumin/Transthyretin Monoclonal Antibody at dilution of 1:1000.



Western Blot with Prealbumin/Transthyretin Monoclonal Antibody at dilution of 1:5000. Lane 1: Human plasma

Observed-MW:35 kDa
Calculated-MW:16 kDa

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	Ice bag

Background

For Research Use Only

Toll-free: 1-888-852-8623
Web: www.elabscience.com

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Rev. V1.1

Transthyretin (TTR) is a highly conserved homotetrameric protein that is synthesized in the liver and choroid plexus of the brain. TTR was originally discovered as a protein found in human plasma and cerebrospinal fluid (CSF). TTR transports thyroid hormones (TH) and retinol by binding to retinol-binding protein. Although TTR is synthesized in the liver and choroid plexus, TTR is detected in blood plasma and cerebrospinal fluid migrating as monomers, dimers, and tetramers. Beyond its function as a carrier protein of TH and retinol in plasma and CSF, several additional TTR functions have been described, including proteolytic cleavage of specific substrates like apolipoprotein, neuropeptide Y (NPY), and APP. These neuronal substrates suggest a functional role for TTR in the central nervous system. Consistent with a CNS function, TTR null mice exhibit memory impairments and altered sensorimotor behavior. TTR may also be linked to neurodegenerative disease: TTR levels in Alzheimer's disease (AD) patients are negatively correlated with disease progression, and a protective role for TTR, at least in AD mouse models, has been described. TTR itself may play a more direct role in disease as gain-of-function mutations in TTR cause the protein to misfold and aggregate into amyloid fibrils, contributing to autosomal dominant hereditary amyloidosis in diseases such as familial amyloid polyneuropathy, familial amyloid cardiomyopathy, and familial leptomeningeal amyloidosis.