

<b>Product name:</b>	THP Rabbit Polyclonal Antibody
<b>Cat number:</b>	ABN18887
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100µL
<b>Clone:</b>	Polyclonal
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rabbit
<b>Isotype:</b>	IgG
<b>Immunogen:</b>	The antiserum was produced against synthesized peptide derived from human THP. AA range:329-378
<b>Reactivity:</b>	Human,Rat,Mouse
<b>Applications:</b>	WB 1:500-1:2000,IHC 1:50-1:300,ELISA 1:2000-1:20000
<b>Molecular Weight:</b>	70kDa
<b>Purification:</b>	Affinity purification
<b>Form:</b>	Liquid
<b>Buffer:</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
<b>Storage:</b>	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

**Background:**

The protein encoded by this gene is the most abundant protein in mammalian urine under physiological conditions. Its excretion in urine follows proteolytic cleavage of the ectodomain of its glycosyl phosphatidylinositol-anchored counterpart that is situated on the luminal cell surface of the loop of Henle. This protein may act as a constitutive inhibitor of calcium crystallization in renal fluids. Excretion of this protein in urine may provide defense against urinary tract infections caused by uropathogenic bacteria. Defects in this gene are associated with the renal disorders medullary cystic kidney disease-2 (MCKD2), glomerulocystic kidney disease with hyperuricemia and isosthenuria (GCKDHI), and familial juvenile hyperuricemic nephropathy (FJHN). Alternative splicing of this gene results in multiple transcript variants. [provided by RefSeq, Jul 2013],disease:Defects in UMOD are a cause of glomerulocystic kidney disease with hyperuricemia and isosthenuria [MIM:609886]. Glomerulocystic kidney disease (GCKD) and medullary cystic disease/familial juvenile hyperuricemic nephropathy (MCKD/HNFJ) are two distinct renal disorders that share some common clinical features. The former is characterized by a cystic dilatation of Bowman's space and a collapse of glomerular tuft. Familial GCKD can be associated with either hypoplastic or normal sized kidneys. A GCKD clinical variant presents the association with hyperuricemia due to low fractional excretion of uric acid and severe impairment of urine concentrating ability that are reminiscent of MCKD/HNFJ.,disease:Defects in UMOD are the cause of familial juvenile hyperuricemic nephropathy (HNFJ) [MIM:162000]. HNFJ is a heritable autosomal dominant renal disease characterized by juvenil onset of hyperuricaemia, polyuria, progressive renal failure, and gout. The disease is associated with interstitial pathological changes resulting in fibrosis.,disease:Defects in UMOD are the cause of medullary cystic kidney disease 2 (MCKD2) [MIM:603860]. MCKD2 and HNFJ constitute a group of heritable renal diseases with a common mode of transmission (autosomal dominant) and shared features including polyuria, hyperuricaemia, progressive renal failure, and gout. Both diseases are associated with interstitial pathological changes resulting in fibrosis. While corticomedullary cysts are well documented in MCKD2, their presence in HNFJ is not well documented. The primary clinical features of MCKD2 and HNFJ vary in presence and severity, complicating the diagnosis of these conditions, particularly in milder cases. Both diseases are considered to be allelic diseases.,function:Not known. May play a role in regulating the circulating activity of cytokines as it binds to IL-1, IL-2 and TNF with high affinity.,similarity:Contains 1 ZP domain.,similarity:Contains 3 EGF-like domains.,subcellular location:Secreted after cleavage in the urine.,tissue specificity:Synthesized by the kidneys and is the most abundant protein in normal human urine.,