

Recombinant CPS1 Monoclonal Antibody

catalog number: **AN301906L**

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

Description	
Reactivity	Human;Mouse;Rat
Immunogen	Recombinant human CPS1 fragment
Host	Rabbit
Isotype	Rabbit IgG
Clone	C121527
Purification	Protein A purified
Buffer	PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protein protectant.

Applications	Recommended Dilution
WB	1:1000-1:5000
IHC	1:50-1:100

Data	
 Western Blot with CPS1 Monoclonal Antibody at dilution of 1:1000. Lane 1: HeLa Observed-MW:165 kDa Calculated-MW:165 kDa	 Western Blot with CPS1 Monoclonal Antibody at dilution of 1:5000. Lane 1: Human liver, Lane 2: Mouse liver, Lane 3: Rat liver Observed-MW:165 kDa Calculated-MW:165 kDa
 Immunohistochemistry of paraffin-embedded Human hepatocellular carcinoma using CPS1 Monoclonal Antibody at dilution of 1:100.	 Immunohistochemistry of paraffin-embedded Human liver using CPS1 Monoclonal Antibody at dilution of 1:100.
 Immunohistochemistry of paraffin-embedded Mouse small intestine using CPS1 Monoclonal Antibody at dilution of 1:100.	 Immunohistochemistry of paraffin-embedded Rat liver using CPS1 Monoclonal Antibody at dilution of 1:100.

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

Background
Carbamoyl phosphate synthetase 1 (CPS1), a rate-limiting enzyme in the urea cycle, catalyzes the conversion of ammonia and bicarbonate to carbamoyl phosphate in mitochondria. Non-small cell lung carcinoma (NSCLC) cells with oncogenic KRAS and loss of the tumor suppressor LKB1 express CPS1 and depend on this enzyme for growth. CPS1 maintains pyrimidine/purine balance in these cancer cells. Silencing CPS1 reduces the pyrimidine to purine ratio and stalls DNA synthesis, leading to DNA damage and cancer cell death. In addition, the tumor suppressor p53 represses the expression of urea cycle enzymes CPS1, OTC, and ARG1 and, therefore, causes the accumulation of ammonia, suppressing cancer growth. Furthermore, research studies suggest that hypermethylation-mediated downregulation of CPS1 expression may contribute to the progression of normal hepatocytes to hepatocellular carcinoma (HCC).

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