

## DPYD

**Reactivity:** Human Mouse Rat

**Tested applications:** WB IHC

**Recommended Dilution:** WB 1:500 - 1:2000 IHC 1:50 - 1:200

**Calculated MW:** 110kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human DPYD

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Synonym:**

DHP; DPD; DHPDHASE;

**Catalog #:** A1620

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 1806

**Isotype:** IgG

**Swiss Prot:** Q12882

**Purity:** Affinity purification

For research use only.

**Background:**

Dihydropyrimidine dehydrogenase (DPD, DPYD) catalyzes the initial and rate-limiting step in uracil and thymidine catabolism as well as catabolism of the chemotherapeutic drug 5-fluorouracil (5-FU) and its derivatives. DPYD deficiency, which results from mutations in the DPYD gene, causes errors in pyrimidine metabolism and potentially life-threatening side effects in cancer patients treated with 5-FU (reviewed in 1). As a result, ongoing work examines whether or how DPYD gene variation and protein expression can be used to predict 5-FU toxicity (1,2). Several genes that impart resistance to 5-FU were recently identified in human hepatocellular carcinoma (HCC). AEG-1, which is highly expressed in HCC, increases the expression of DPYD. DPYD is expressed more highly in HCC than in normal liver, and this is thought to be one mechanism of 5-FU resistance (3,4).

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