HBB Rabbit pAb

CatalogNo: YN2466

Key Features

Host Species
- Rabbit

Reactivity
- Human,Rat

Applications
- WB,ELISA

MW
- 16kD (Observed)

Isotype
- IgG

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

WB 1:500-2000
ELISA 1:5000-20000

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	Synthesized peptide derived from part region of human protein. AA range:1-22
Specificity	HBB Polyclonal Antibody detects endogenous levels of protein.

Target Information

Gene name	HBB

Protein Name Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7]

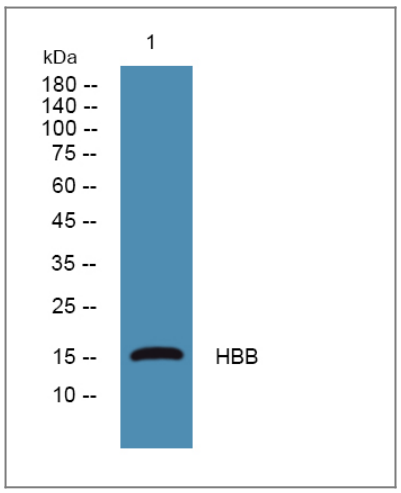
Organism	Gene ID	UniProt ID
Human	3043;	P68871;

Cellular Localization extracellular region,cytosol,hemoglobin complex,integral component of membrane,haptoglobin-hemoglobin complex,extracellular exosome,endocytic vesicle lumen,blood microparticle,

Tissue specificity Red blood cells.

Function Disease:Defects in HBB are the cause of beta-thalassemia [MIM:141900, 604131]. The thalassemias are the most common monogenic diseases and occur mostly in Mediterranean and Southeast Asian populations. The hallmark of beta-thalassemia is an imbalance in globin-chain production in the adult HbA molecule. Absence of beta chain causes beta(0)-thalassemia, while reduced amounts of detectable beta globin causes beta(+)-thalassemia. In the severe forms of beta-thalassemia, the excess alpha globin chains accumulate in the developing erythroid precursors in the marrow. Their deposition leads to a vast increase in erythroid apoptosis that in turn causes ineffective erythropoiesis and severe microcytic hypochromic anemia. Clinically, beta-thalassemia is divided into thalassemia major (transfusion dependent), thalassemia intermedia (of intermediate severity), and thalassemia minor (asymptomatic).,Disease:Defects in HBB are the cause of dominant beta-thalassemia inclusion body type [MIM:603902]. This form of beta-thalassemia is transmitted in an autosomal dominant fashion and is characterized by anemia, enlargement of the spleen, and gross abnormalities of the erythrocytes and their precursors.,Disease:Defects in HBB are the cause of sickle cell anemia [MIM:603903]; also known as sickle cell disease. Sickle cell anemia is characterized by abnormally shaped red cells resulting in chronic anemia and periodic episodes of pain, serious infections and damage to vital organs. Normal red blood cells are round and flexible and flow easily through blood vessels, but in sickle cell anemia, the abnormal hemoglobin (called Hb S) causes red blood cells to become stiff. They are C-shaped and resembles a sickle. These stiffer red blood cells can led to microvascular occlusion thus cutting off the blood supply to nearby tissues.,Disease:Defects in HBB may be a cause of Heinz body anemias [MIM:140700]. This is a form of non-spherocytic hemolytic anemia of Dacie type 1. After splenectomy, which has little benefit, basophilic inclusions called Heinz bodies are demonstrable in the erythrocytes. Before splenectomy, diffuse or punctate basophilia may be evident. Most of these cases are probably instances of hemoglobinopathy. The hemoglobin demonstrates heat lability. Heinz bodies are observed also with the Ivemark syndrome (asplenia with cardiovascular anomalies) and with glutathione peroxidase deficiency.,Function:Involved in oxygen transport from the lung to the various peripheral tissues.,Function:LVV-hemorphin-7 potentiates the activity of bradykinin, causing a decrease in blood pressure.,mass spectrometry: PubMed:1575724,miscellaneous:One molecule of 2,3-bisphospoglycerate can bind to two beta chains per hemoglobin tetramer.,online information:Hemoglobin entry,online information:Human hemoglobin variants and thalassemias,online information:The Singapore human mutation and polymorphism database,PTM:Acetylated on Lys-60, Lys-83 and Lys-145 upon aspirin exposure. PubMed:16916647 reports the identification of HBB acetylated on Lys-145 in the cytosolic fraction of HeLa cells. This may results from a contamination of the sample.,PTM:Glucose reacts non-enzymatically with the N-terminus of the beta chain to form a stable ketoamine linkage. This takes place slowly and continuously throughout the 120-day life span of the red blood cell. The rate of glycation is increased in patients with diabetes mellitus.,PTM:S-nitrosylated; a nitric oxide group is first bound to Fe(2+) and then transferred to Cys-94 to allow capture of O(2).,similarity:Belongs to the globin family.,subunit:Heterotetramer of two alpha chains and two beta chains in adult hemoglobin A (HbA).,tissue specificity:Red blood cells.,

Validation Data



Western blot analysis of lysates from DU145 cells, primary antibody was diluted at 1:1000, 4°over night

Contact information

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