Immunotag™ HBA Polyclonal Antibody

Antibody Specification

Catalog No.	ITN2315
Product Description	Immunotag™ HBA Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	HBA
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,ELISA
Recommended Dilution	WB 1:500-2000 ELISA 1:5000-20000
Concentration	1 mg/ml
Reactive Species	Human,Rat,Mouse
Host Species	Rabbit
Immunogen	Synthesized peptide derived from human protein, at AA range: 30-110
Specificity	HBA Polyclonal Antibody detects endogenous levels of protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, and 0.02% sodium azide.
Gene Name	HBA1; HBA2
Accession No.	P69905 P01942 P01946

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Description	hemoglobin subunit alpha 1(HBA1) Homo sapiens The human alpha globin gene cluster located on chromosome 16 spans about 30 kb and includes seven loci: 5'- zeta - pseudozeta - mu - pseudoalpha-1 - alpha-2 - alpha-1 - theta - 3'. The alpha-2 (HBA2) and alpha-1 (HBA1) coding sequences are identical. These genes differ slightly over the 5' untranslated regions and the introns, but they differ significantly over the 3' untranslated regions. Two alpha chains plus two beta chains constitute HbA, which in normal adult life comprises about 97% of the total hemoglobin; alpha chains combine with delta chains to constitute HbA-2, which with HbF (fetal hemoglobin) makes up the remaining 3% of adult hemoglobin. Alpha thalassemias result from deletions of each of the alpha genes as well as deletions of both HBA2 and HBA1; some nondeletion alpha thalassemias have also been reported. [provided by RefSeq, Jul 2008],
Protein Expression	Blood,Bone marrow,Brain,Lung,Platelet,Spleen,Thymus,Umbilical cord blood,Wh
Subcellular Localization	extracellular region,cytosol,hemoglobin complex,membrane,cytosolic small ribosomal subunit,haptoglobin-hemoglobin complex,extracellular exosome,endocytic vesicle lumen,blood microparticle,

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Protein Function	disease:Alpha(0)-thalassemia is associated with nonimmune hydrops fetalis [MIM:236750]. Hydrops fetalis is a generalized edema of the fetus with fluid accumulation in the body cavities.,disease:Defects in HBA1/HBA2 are the cause of alpha-thalassemia [MIM:141800, 604131]. The thalassemias are the most common monogenic diseases and occur mostly in Mediterranean and Southeast Asian populations. The hallmark of alpha-thalassemia is an imbalance in globin-chain production in the adult HbA molecule. The level of alpha chain production can range from none to very nearly normal levels. Deletion of both copies of each of the two alpha-globin genes causes alpha(0)-thalassemia, also known as homozygous alpha thalassemia. Due to the complete absence of alpha chains, the predominant fetal hemoglobin is a tetramer of gamma-chains (Bart hemoglobin) that has essentially no oxygen carrying capacity. This causes oxygen starvation in the fetal tissues leading to prenatal lethality or early neonatal death. The loss of three alpha genes results in high levels of a tetramer of four beta chains (hemoglobin H), causing a severe and life-threatening anemia known as hemoglobin H disease. Untreated, most patients die in childhood or early adolescence. The loss of two alpha genes results in mild alpha-thalassemia, also known as heterozygous alpha-thalassemia. Affected individuals have small red cells and a mild anemia (microcytosis). If three of the four alpha-globin genes are functional, individuals are completely asymptomatic. Some rare forms of alpha-thalassemia are due to point mutations (non-deletional alpha-thalassemia). The thalassemic phenotype is due to unstable globin alpha chains that are rapidly catabolized prior to formation of the alpha-beta heterotetramers.,disease:Defects in HBA1/HBA2 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.,miscellaneous:Gives blood its red color.,online information:Hemoglobin entry,online information:Human hemoglobin variants and thalassemias,online information:The Singapore human mutation and polymorphism database,PTM:The initiator Met is not cleaved in variant Thionville and is acetylated.,similarity:Belongs to the globin family.,subunit:Heterotetramer of two alpha chains and two beta chains in adult hemoglobin A (HbA); two alpha chains and two delta chains in adult hemoglobin A2 (HbA2); two alpha chains and two epsilon chains in early embryonic hemoglobin Gower-2; two alpha chains and two gamma chains in fetal hemoglobin F (HbF).,tissue specificity:Red blood cells.,
Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.