血红蛋白α Hemoglobin A1c 抗体

产品货号： mlR9889

英文名称： Hemoglobin alpha

中文名称： 血红蛋白 α/Hemoglobin A1c 抗体

别　　名： Hemoglobin A1c; Alpha globin; Hba1; HBA2; HBAM; Hemoglobin alpha 1; Hemoglobin alpha adult chain 1; HBA_HUMAN.

研究领域： 心血管　细胞生物　糖尿病　内分泌病　糖蛋白

抗体来源： Rabbit

克隆类型： Polyclonal

交叉反应： Human,

产品应用： ELISA=1:500-1000 IHC-P=1:400-800 IHC-F=1:400-800 IF=1:50-200 （石蜡切片需做抗原修复）
not yet tested in other applications.
optimal dilutions/concentrations should be determined by the end user.

分 子 量： 15kDa

细胞定位： 细胞浆 细胞外基质 分泌型蛋白

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原： KLH conjugated synthetic peptide derived from human Hemoglobin alpha:1-100/142

亚　　型： IgG

纯化方法： affinity purified by Protein A

储 存 液： 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件： Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

PubMed： PubMed

产品介绍： 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].

Function:

Involved in oxygen transport from the lung to the various peripheral tissues.

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).

Subcellular Location:

hemoglobin complex.

Tissue Specificity:

Red blood cells.

Post-translational modifications:

The initiator Met is not cleaved in variant Thionville and is acetylated.

DISEASE:

Defects in HBA1 may be a cause of Heinz body anemias (HEIBAN) [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.

Defects in HBA1 are the cause of alpha-thalassemia (A-THAL) [MIM: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.

Note=Alpha(0)-thalassemia is associated with non-immune hydrops fetalis, a generalized edema of the fetus with fluid accumulation in the body cavities due to non-immune causes. Non-immune hydrops fetalis is not a diagnosis in itself but a symptom, a feature of many genetic disorders, and the end-stage of a wide variety of disorders.

Defects in HBA1 are the cause of hemoglobin H disease (HBH) [MIM:613978]. HBH is a form of alpha-thalassemia due to the loss of three alpha genes. This results in high levels of a tetramer of four beta chains (hemoglobin H), causing a severe and life-threatening anemia. Untreated, most patients die in childhood or early adolescence.

Similarity:

Belongs to the globin family.

SWISS:

P69905

Gene ID:

3039

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

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