

Product datasheet

Anti-Hemoglobin antibody ab19363

Overview

Product name	Anti-Hemoglobin antibody
Description	Goat polyclonal to Hemoglobin
Host species	Goat
Tested applications	Suitable for: Immunoelectrophoresis, Double Immunodiffusion
Species reactivity	Reacts with: Human
Immunogen	Human Hemoglobin F.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	pH: 7.20 Preservative: 0.1% Sodium azide Constituents: 1.21% Tris, 0.02% Sodium chloride
Purity	IgG fraction
Purification notes	Antiserum was solid phase adsorbed against hemoglobin A to ensure specificity. The antiserum was fractionated and passed over DEAE to yield an IgG fraction.
Clonality	Polyclonal
Isotype	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab19363** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
Immunoelectrophoresis		Use at an assay dependent dilution.
Double Immunodiffusion		Use at an assay dependent dilution.

Target

Function	Involved in oxygen transport from the lung to the various peripheral tissues.
Tissue specificity	Red blood cells.
Involvement in disease	<p>Defects in HBA1/HBA2 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.</p> <p>Defects in HBA1/HBA2 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.</p> <p>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.</p>
Sequence similarities	Belongs to the globin family.
Post-translational modifications	The initiator Met is not cleaved in variant Thionville and is acetylated.

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