abcam

Product datasheet

Rat Hemoglobin ELISA Kit ab157733

1 Image

Overview

Product name Rat Hemoglobin ELISA Kit

Detection method Colorimetric

Precision

Intra-assay

Sample	n	Mean	SD	CV%
Overall				< 10%

Inter-assay

Sample	n	Mean	SD	CV%
Overall				< 10%

Sample type Serum, Plasma, Other biological fluids

Assay type Sandwich (quantitative)

Sensitivity 1.4284 ng/ml

Range 0.006 µg/ml - 16.92 µg/ml

Recovery

Sample specific recovery

Sample type	Average %	Range
Serum	> 85	% - %

Assay duration Multiple steps standard assay

Species reactivity **Reacts with:** Rat

Product overview Abcam's Hemoglobin Rat ELISA Kit is a highly sensitive two-site enzyme linked immunoassay (ELISA) for measuring Hemoglobin in biological samples of rats.

In this assay the Hemoglobin present in samples reacts with the anti-Hemoglobin antibodies which have been adsorbed to the surface of polystyrene microtitre wells. After the removal of unbound proteins by washing, anti-Hemoglobin antibodies conjugated with horseradish peroxidase (HRP) are added. These enzyme-labeled antibodies form complexes with the previously bound Hemoglobin. Following another washing step, the enzyme bound to the immunosorbent is assayed by the addition of a chromogenic substrate, 3,3',5,5'-tetramethylbenzidine (TMB). The quantity of bound enzyme varies directly with the concentration

of Hemoglobin in the sample tested; thus, the absorbance, at 450 nm, is a measure of the concentration of Hemoglobin in the test sample. The quantity ofHemoglobinin the test sample can be interpolated from the standard curve constructed from the standards, and corrected for sample dilution.

Platform Microplate

Properties

Storage instructions Please refer to protocols.

Components	1 x 96 tests
100X HRP-conjugated anti-rat Hemoglobin antibody	1 x 150µl
20X Wash Buffer Concentrate	1 x 50ml
5X Diluent Concentrate	1 x 50ml
Chromogen Substrate Solution	1 x 12ml
Rat Hemoglobin Calibrator (Lyophilized)	1 vial
Rat Hemoglobin ELISA Microplate	1 unit
Stop Solution	1 x 12ml

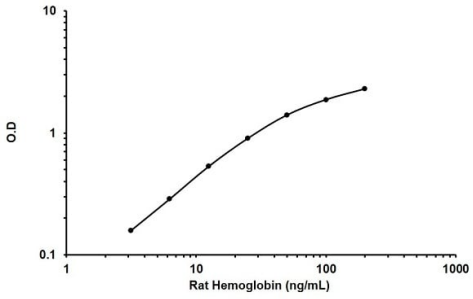
Function Involved in oxygen transport from the lung to the various peripheral tissues.

Tissue specificity Red blood cells.

Involvement in disease 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.
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. 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.
Sequence similarities	Belongs to the globin family.
Post-translational modifications	The initiator Met is not cleaved in variant Thionville and is acetylated.

Images



Standard Curve

Representative standard curve using ab157733 Hemoglobin Rat ELISA Kit.

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