abcam

Product datasheet

Mouse Hemoglobin ELISA Kit ab157715

2 References 1 Image

Overview

Product name Mouse 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

Assay type Sandwich (quantitative)

Sensitivity 2.62 ng/ml

Range 12.5 ng/ml - 400 ng/ml

Recovery

Sample specific recovery

Sample type	Average %	Range
Serum	> 85	% - %

Assay duration Multiple steps standard assay

Species reactivity **Reacts with:** Mouse

Product overview Abcam's Hemoglobin Mouse ELISA kit is an *in vitro* enzyme-linked immunosorbent assay (ELISA) for the quantitative measurement of Hemoglobin in biological samples of mice.

In this assay the Hemoglobin present in samples reacts with the anti-Hemoglobin antibodies which have been adsorbed to the surface of polystyrene microtiter wells. After the removal of unbound proteins by washing, anti-HM antibodies conjugated with horseradish peroxidase (HRP), are added. These enzyme-labeled antibodies form complexes with the previously bound HM. 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 HM in the sample tested; thus, the absorbance, at 450 nm, is a measure of the concentration of HM in the test sample. The quantity of HM in the test sample can be interpolated from the standard curve constructed from the standards, and corrected for sample dilution.

Platform Microplate

Properties

Storage instructions Store at +4°C. Please refer to protocols.

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

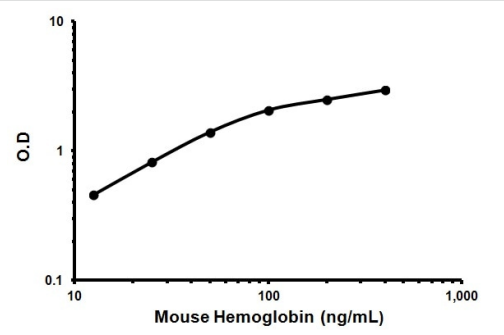
Function Involved in oxygen transport from the lung to the various peripheral tissues.

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



Typical standard curve

Typical standard curve using ab157715 Hemoglobin Mouse ELISA Kit

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

Our Abpromise to you: Quality guaranteed and expert technical support

- Replacement or refund for products not performing as stated on the datasheet
- Valid for 12 months from date of delivery
- Response to your inquiry within 24 hours
- We provide support in Chinese, English, French, German, Japanese and Spanish
- Extensive multi-media technical resources to help you
- We investigate all quality concerns to ensure our products perform to the highest standards

If the product does not perform as described on this datasheet, we will offer a refund or replacement. For full details of the Abpromise, please visit https://www.abcam.com/abpromise or contact our technical team.

Terms and conditions

- Guarantee only valid for products bought direct from Abcam or one of our authorized distributors