


## Product datasheet

# HRP Anti-Hemoglobin antibody ab19362

[4 References](#) [1 Image](#)

### Overview

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<b>Product name</b>	HRP Anti-Hemoglobin antibody
<b>Description</b>	HRP Goat polyclonal to Hemoglobin
<b>Host species</b>	Goat
<b>Conjugation</b>	HRP
<b>Specificity</b>	The antibody has been tested in ELISA and IEP with a Human Hemaglobin Calibrator/Standard but has yet to be tested against endogenous protein.
<b>Tested applications</b>	<b>Suitable for:</b> WB
<b>Species reactivity</b>	<b>Reacts with:</b> Human <b>Predicted to work with:</b> Rabbit, Dog, Chimpanzee, Rhesus monkey 
<b>Immunogen</b>	Full length protein corresponding to Human Hemoglobin conjugated to bovine serum albumin. Goats were immunized with purified human hemoglobin A1 of adult red blood cell origin.
<b>Positive control</b>	recombinant human hemoglobin
<b>General notes</b>	Molar enzyme/antibody protein ratio is 4:1.  The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.  If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As

### Properties

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<b>Form</b>	Liquid
<b>Storage instructions</b>	Shipped at 4°C. Store at +4°C.
<b>Storage buffer</b>	pH: 6.8 Preservative: 0.01% Thimerosal (merthiolate) Constituents: 1.19% HEPES, 0.58% Sodium chloride
<b>Purity</b>	Immunogen affinity purified
<b>Purification notes</b>	Antibody concentration was determined by extinction coefficient prior to conjugation: absorbance

at 280 nm of 1.4 equals 1.0 mg of IgG.

**Clonality**

Polyclonal

**Isotype**

IgG

**Applications**

**The Abpromise guarantee**

Our **Abpromise guarantee** covers the use of ab19362 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
WB		1/1000 - 1/30000. Predicted molecular weight: 16 kDa.

**Target**

**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**

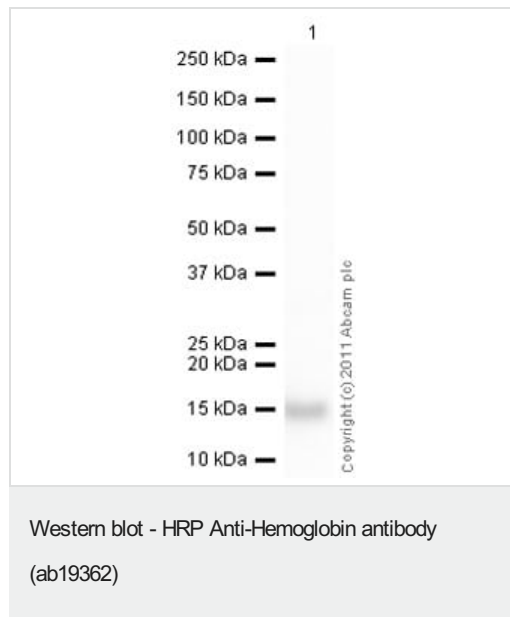
The initiator Met is not cleaved in variant Thionville and is acetylated.

## modifications

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## Images

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