

Native Human Hemoglobin protein ab77858

画像数 1

製品の詳細

製品名	Native Human Hemoglobin protein
精製度	> 95 % SDS-PAGE. Multi-step procedure including ion exchange chromatography. 0.96mg hemoglobin/mg powder (Coomassie) (prior to lyophilization).
発現系	Native
アクセッション番号	<u>P69905</u> <u>P68871</u>
タンパク質長	Full length protein
Animal free	No
由来	Native
アミノ酸配列 1	
生物種	Human
アミノ酸配列 2	
生物種	Human
予測される分子量	15 kDa

特性

Our **Abpromise guarantee** covers the use of **ab77858** in the following tested applications.

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

アプリケーション	Western blot SDS-PAGE
製品の状態	Lyophilized
備考	All human source materials have tested negative for HIV1, HIV2, HCV antibodies, HBsAg and HIV Antigen by approved FDA methods. No test guarantees a product to be non-infectious. Therefore, all material derived from human fluids or tissues should be considered as potentially infectious.

前処理および保存

保存方法および安定性	Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.
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Constituent: Sodium chloride

再構成

Protein can be reconstituted using deionized water.

関連情報

機能

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

組織特異性

Red blood cells.

関連疾患

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.

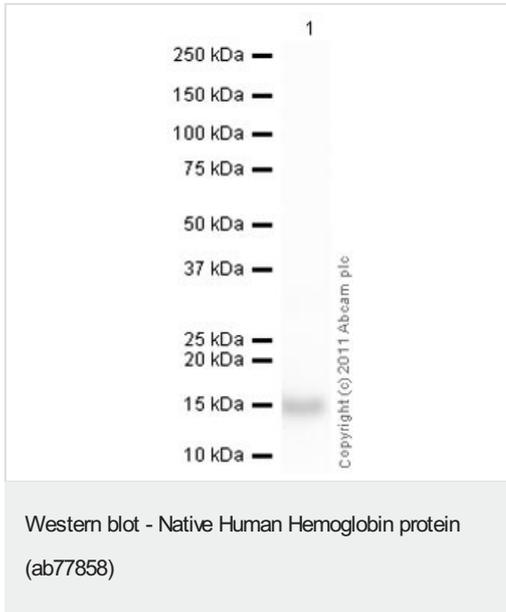
配列類似性

Belongs to the globin family.

翻訳後修飾

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

画像



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