## Overview

**Product name**: Anti-Apolipoprotein A I antibody (HRP)  
**Description**: Goat polyclonal to Apolipoprotein A I (HRP)  
**Host species**: Goat  
**Conjugation**: HRP  
**Specificity**: Specific binding to Apo A-I. No cross-reaction with Apo AII, Apo B-100, Apo CI, Apo CII, Apo CIII, and Apo E.  
**Tested applications**: Suitable for: ELISA, WB  
**Species reactivity**: Reacts with: Human  
**Immunogen**: Full length native protein (purified) (Human).

## Properties

<table>
<thead>
<tr>
<th>Property</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Form</td>
<td>Liquid</td>
</tr>
<tr>
<td>Storage instructions</td>
<td>Shipped at 4°C. Store at +4°C.</td>
</tr>
</tbody>
</table>
| Storage buffer    | pH: 7.40  
Preservative: 0.01% Thimerosal (merthiolate)  
Constituents: 0.134% PBS, 0.58% Sodium chloride, 1% BSA |
| Purity            | Immunogen affinity purified                  |
| Clonality         | Polyclonal                                   |
| Isotype           | IgG                                          |

## Applications

Our [Abpromise guarantee](#) covers the use of **ab20784** in the following tested applications.

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

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>ELISA</td>
<td><strong>1/4000 - 1/32000.</strong></td>
<td></td>
</tr>
<tr>
<td>WB</td>
<td></td>
<td><strong>1/4000 - 1/32000.</strong> Predicted molecular weight: 31 kDa.</td>
</tr>
</tbody>
</table>
**Function**

Participates in the reverse transport of cholesterol from tissues to the liver for excretion by promoting cholesterol efflux from tissues and by acting as a cofactor for the lecithin cholesterol acyltransferase (LCAT). As part of the SPAP complex, activates spermatozoa motility.

**Tissue specificity**

Major protein of plasma HDL, also found in chylomicrons. Synthesized in the liver and small intestine.

**Involvement in disease**

Defects in APOA1 are a cause of high density lipoprotein deficiency type 2 (HDLD2) [MIM:604091]; also known as familial hypoalphalipoproteinemia (FHA). Inheritance is autosomal dominant.

Defects in APOA1 are a cause of the low HDL levels observed in high density lipoprotein deficiency type 1 (HDLD1) [MIM:205400]; also known as analphalipoproteinemia or Tangier disease (TGD). HDLD1 is a recessive disorder characterized by the absence of plasma HDL, accumulation of cholesteryl esters, premature coronary artery disease, hepatosplenomegaly, recurrent peripheral neuropathy and progressive muscle wasting and weakness. In HDLD1 patients, ApoA-I fails to associate with HDL probably because of the faulty conversion of pro-ApoA-I molecules into mature chains, either due to a defect in the converting enzyme activity or a specific structural defect in Tangier ApoA-I.

Defects in APOA1 are the cause of amyloid polyneuropathy-nephropathy Iowa type (AMYLIOWA) [MIM:107680]; also known as amyloidosis van Allen type or familial amyloid polyneuropathy type III. AMYLIOWA is a hereditary generalized amyloidosis due to deposition of amyloid mainly constituted by apolipoprotein A1. The clinical picture is dominated by neuropathy in the early stages of the disease and nephropathy late in the course. Death is due in most cases to renal amyloidosis. Severe peptic ulcer disease can occur in some and hearing loss is frequent. Cataracts is present in several, but vitreous opacities are not observed.

Defects in APOA1 are a cause of amyloidosis type 8 (AMYL8) [MIM:105200]; also known as systemic non-neuropathic amyloidosis or Ostertag-type amyloidosis. AMYL8 is a hereditary generalized amyloidosis due to deposition of apolipoprotein A1, fibrinogen and lysozyme amyloids. Viscera are particularly affected. There is no involvement of the nervous system. Clinical features include renal amyloidosis resulting in nephrotic syndrome, arterial hypertension, hepatosplenomegaly, cholestasis, petechial skin rash.

**Sequence similarities**

Belongs to the apolipoprotein A1/A4/E family.

**Post-translational modifications**

Palmitoylated.

Phosphorylation sites are present in the extracellular medium.

**Cellular localization**

Secreted.

---

**Target**

- Participates in the reverse transport of cholesterol from tissues to the liver for excretion by promoting cholesterol efflux from tissues and by acting as a cofactor for the lecithin cholesterol acyltransferase (LCAT). As part of the SPAP complex, activates spermatozoa motility.
- Major protein of plasma HDL, also found in chylomicrons. Synthesized in the liver and small intestine.
- Defects in APOA1 are a cause of high density lipoprotein deficiency type 2 (HDLD2) [MIM:604091]; also known as familial hypoalphalipoproteinemia (FHA). Inheritance is autosomal dominant.
- Defects in APOA1 are a cause of the low HDL levels observed in high density lipoprotein deficiency type 1 (HDLD1) [MIM:205400]; also known as analphalipoproteinemia or Tangier disease (TGD). HDLD1 is a recessive disorder characterized by the absence of plasma HDL, accumulation of cholesteryl esters, premature coronary artery disease, hepatosplenomegaly, recurrent peripheral neuropathy and progressive muscle wasting and weakness. In HDLD1 patients, ApoA-I fails to associate with HDL probably because of the faulty conversion of pro-ApoA-I molecules into mature chains, either due to a defect in the converting enzyme activity or a specific structural defect in Tangier ApoA-I.
- Defects in APOA1 are the cause of amyloid polyneuropathy-nephropathy Iowa type (AMYLIOWA) [MIM:107680]; also known as amyloidosis van Allen type or familial amyloid polyneuropathy type III. AMYLIOWA is a hereditary generalized amyloidosis due to deposition of amyloid mainly constituted by apolipoprotein A1. The clinical picture is dominated by neuropathy in the early stages of the disease and nephropathy late in the course. Death is due in most cases to renal amyloidosis. Severe peptic ulcer disease can occur in some and hearing loss is frequent. Cataracts is present in several, but vitreous opacities are not observed.
- Defects in APOA1 are a cause of amyloidosis type 8 (AMYL8) [MIM:105200]; also known as systemic non-neuropathic amyloidosis or Ostertag-type amyloidosis. AMYL8 is a hereditary generalized amyloidosis due to deposition of apolipoprotein A1, fibrinogen and lysozyme amyloids. Viscera are particularly affected. There is no involvement of the nervous system. Clinical features include renal amyloidosis resulting in nephrotic syndrome, arterial hypertension, hepatosplenomegaly, cholestasis, petechial skin rash.

**Sequence similarities**

Belongs to the apolipoprotein A1/A4/E family.

**Post-translational modifications**

Palmitoylated.

Phosphorylation sites are present in the extracellular medium.

**Cellular localization**

Secreted.

---

**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.