

## Overview

### Useful For

Diagnosing congenital alpha-2 plasmin inhibitor deficiencies (rare)

Providing a complete assessment of disseminated intravascular coagulation, intravascular coagulation and fibrinolysis, or hyperfibrinolysis (primary fibrinolysis), when measured in conjunction with fibrinogen, fibrin D-dimer, fibrin degradation products, soluble fibrin monomer complex, and plasminogen

Evaluating liver disease

Evaluating the effects of fibrinolytic or antifibrinolytic therapy

### Special Instructions

- [Coagulation Guidelines for Specimen Handling and Processing](#)

### Method Name

Chromogenic

### NY State Available

Yes

## Specimen

### Specimen Type

Plasma Na Cit

### Specimen Required

**Specimen Type:** Platelet-poor plasma

**Collection Container/Tube:** Light-blue top (3.2% sodium citrate)

**Submission Container/Tube:** Plastic vial

**Specimen Volume:** 1 mL

#### Collection Instructions:

1. For complete instructions, see [Coagulation Guidelines for Specimen Handling and Processing](#).
2. Centrifuge, transfer all plasma into a plastic vial, and centrifuge plasma again.
3. Aliquot plasma into a plastic vial leaving 0.25 mL in the bottom of centrifuged vial.
4. Freeze plasma immediately (no longer than 4 hours after collection) at -20 degrees C or, ideally, -40 degrees C or below.

#### Additional Information:

1. Double-centrifuged specimen is critical for accurate results as platelet contamination may cause spurious results.
2. Each coagulation assay requested should have its own vial.

Forms

If not ordering electronically, complete, print, and send a [Coagulation Test Request](#) (T753) with the specimen.

Specimen Minimum Volume

0.5 mL

Reject Due To

Gross hemolysis	Reject
Gross lipemia	Reject
Gross icterus	Reject

Specimen Stability Information

Specimen Type	Temperature	Time	Special Container
Plasma Na Cit	Frozen	14 days	

Clinical & Interpretive

Clinical Information

Alpha-2 plasmin inhibitor (antiplasmin) is synthesized in the liver with a biological half-life of approximately 3 days. It inactivates plasmin, the primary fibrinolytic enzyme responsible for remodeling the fibrin thrombus, and binds fibrin together with factor XIIIa making the clot more difficult to lyse. Absence of alpha-2 plasmin inhibitor results in uncontrolled plasmin-mediated breakdown of the fibrin clot and is associated with increased risk of bleeding.

Reference Values

Adults: 80-140%

Normal, full-term, and premature infants may have mildly decreased levels (> or =50%) that reach adult levels within 90 days postnatal.\*

\*See Pediatric Hemostasis References section in [Coagulation Guidelines for Specimen Handling and Processing](#).

Interpretation

Patients with congenital homozygous deficiency (with levels of <10%) are clinically affected (bleeding). Heterozygous individuals having levels of 30% to 60% of mean normal activity are usually asymptomatic.

Lower than normal levels may be suggestive of consumption due to activation of plasminogen and its inhibition by alpha-2 plasmin inhibitor.

The clinical significance of high levels of alpha-2 plasmin inhibitor is unknown.

Cautions

Alpha-2 plasmin inhibitor results are potentially affected by the following:

-Heparin, unfractionated or low-molecular-weight >4 U/mL

- Alpha-2-macroglobulin >7 mg/mL; potentially leading to a falsely increased result
- Hemoglobin >200 mg/dL
- Bilirubin >20 mg/dL
- Triglycerides >1000 mg/dL

**Clinical Reference**

1. Lijnen HR, Collen D. Congenital and acquired deficiencies of components of the fibrinolytic system and their relation to bleeding or thrombosis. Blood Coagul Fibrinolysis. 1989;3(2):67-77. doi:10.1016/0268-9499(89)90034-9
2. Francis RB Jr. Clinical disorders of fibrinolysis: A critical review. Blut. 1989;59(1):1-14
3. Aoki N. Hemostasis associated with abnormalities of fibrinolysis. Blood Rev. 1989;3(1):11-17
4. Singh S, Saleem S, Reed GL. Alpha2-antiplasmin: The devil you don't know in cerebrovascular and cardiovascular disease. Front Cardiovasc Med. 2020;7:608899

**Performance****Method Description**

This assay is performed using the HemosIL Plasmin Inhibitor Kit on the Instrumentation Laboratory ACL TOP Family. Patient plasma, containing alpha-2 plasmin inhibitor, is mixed with reagent containing excess plasmin. Plasmin activity in the reagent is rapidly inhibited by alpha-2 plasmin inhibitor. Residual plasmin activity is then measured using an amidolytic activity assay, in which residual plasmin lyses a synthetic chromogenic substrate and subsequently releases para-nitroaniline (detected at 405 nm) to a level that is inversely proportional to the amount of alpha-2 plasmin inhibitor in the sample.(Teger-Nilsson AC, Friberger P, Gyzander E. Determination of a new rapid plasmin inhibitor in human blood by means of a plasmin specific tripeptide substrate. Scand J Clin Lab Invest. 1977;37(5):403-409; package insert: HemosIL Plasmin Inhibitor. Instrumentation Laboratory; 11/2019)

**PDF Report**

No

**Day(s) Performed**

Monday through Friday

**Report Available**

3 to 7 days

**Specimen Retention Time**

7 days

**Performing Laboratory Location**

Mayo Clinic Laboratories - Rochester Main Campus

**Fees & Codes**

Fees

- Authorized users can sign in to [Test Prices](#) for detailed fee information.
- Clients without access to Test Prices can contact [Customer Service](#) 24 hours a day, seven days a week.
- Prospective clients should contact their account representative. For assistance, contact [Customer Service](#).

Test Classification

This test has been modified from the manufacturer's instructions. Its performance characteristics were determined by Mayo Clinic in a manner consistent with CLIA requirements. This test has not been cleared or approved by the US Food and Drug Administration.

CPT Code Information

85410

LOINC® Information

Test ID	Test Order Name	Order LOINC® Value
A2PI	Alpha-2 Plasmin Inhibitor, P	27810-1

Result ID	Test Result Name	Result LOINC® Value
A2PI	Alpha-2 Plasmin Inhibitor, P	27810-1