
Overview**Useful For**

Detecting increased or decreased fibrinogen (factor 1) concentration of acquired or congenital origin

Monitoring severity and treatment of disseminated intravascular coagulation and fibrinolysis

Method Name

Only orderable as part of a profile or reflex. For more information, see:

ALBLD / Bleeding Diathesis Profile, Limited, Plasma

APROL / Prolonged Clot Time Profile, Plasma

AATHR / Thrombophilia Profile, Plasma

ADIC / Disseminated Intravascular Coagulation/Intravascular Coagulation and Fibrinolysis (DIC/ICF) Profile, Plasma

ALUPP / Lupus Anticoagulant Profile, Plasma

Claus

NY State Available

Yes

Specimen**Specimen Type**

Plasma Na Cit

Specimen Required

Only orderable as part of a profile or reflex. For more information, see:

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APROL / Prolonged Clot Time Profile, Plasma

AATHR / Thrombophilia Profile, Plasma

ADIC / Disseminated Intravascular Coagulation/Intravascular Coagulation and Fibrinolysis (DIC/ICF) Profile, Plasma

ALUPP / Lupus Anticoagulant Profile, Plasma

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 (preferred)	14 days	

Clinical & Interpretive**Clinical Information**

Fibrinogen, also known as factor 1, is a plasma protein that can be transformed by thrombin into a fibrin gel ("the clot"). Fibrinogen is synthesized in the liver and circulates in the plasma as a disulfide-bonded dimer of 3 subunit chains. The biological half-life of plasma fibrinogen is 3 to 5 days.

An isolated deficiency of fibrinogen may be inherited as an autosomal recessive trait (afibrinogenemia or hypofibrinogenemia) and is one of the rarest of the inherited coagulation factor deficiencies.

Acquired causes of decreased fibrinogen levels include acute or decompensated intravascular coagulation and fibrinolysis (disseminated intravascular coagulation), advanced liver disease, L-asparaginase therapy, and therapy with fibrinolytic agents (eg, streptokinase, urokinase, tissue plasminogen activator).

Fibrinogen function abnormalities, dysfibrinogenemias, may be inherited (congenital) or acquired. Patients with dysfibrinogenemia are generally asymptomatic. However, the congenital dysfibrinogenemias are more likely than the acquired to be associated with bleeding or thrombotic disorders. While the dysfibrinogenemias are generally not associated with clinically significant hemostasis problems, they characteristically produce a prolonged thrombin time clotting test. Congenital dysfibrinogenemias usually are inherited as autosomal codominant traits.

Acquired dysfibrinogenemias mainly occur in association with liver disease (eg, chronic hepatitis, hepatoma) or renal diseases associated with elevated fibrinogen levels.

Fibrinogen is an acute-phase reactant, so a number of acquired conditions can result in an increase in its plasma level:

- Acute or chronic inflammatory illnesses
- Nephrotic syndrome
- Liver disease and cirrhosis
- Pregnancy or estrogen therapy
- Compensated intravascular coagulation

The finding of an increased level of fibrinogen in a patient with obscure symptoms suggests an organic rather than a functional condition. Chronically increased fibrinogen has been recognized as a risk factor for development of arterial and venous thromboembolism.

Reference Values

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ALUPP / Lupus Anticoagulant Profile, Plasma

Males: 200-500 mg/dL

Females: 200-500 mg/dL

In normal full-term newborns and in healthy premature infants (30-36 weeks gestation) fibrinogen is near adult levels (>150) and reaches adult levels by <21 days postnatal.

Interpretation

This test assesses levels of functional (clottable) fibrinogen (see Cautions). Fibrinogen may be decreased in acquired conditions such as liver disease and acute intravascular coagulation and fibrinolysis and disseminated intravascular coagulation (ICF/DIC). Fibrinogen may be decreased in rare conditions including congenital afibrinogenemia or hypofibrinogenemia. Fibrinogen may be elevated with acute or chronic inflammatory conditions.

Cautions

In patients with dysfibrinogenemias, this assay may give spuriously low results.

In patients with markedly elevated plasma levels of fibrin degradation products (eg, thrombolytic therapy or disseminated intravascular coagulation and fibrinolysis), clottable fibrinogen determined by this method may be lower than when measured by an end point method (eg, nephelometric) assay.

Patients with antibodies to bovine thrombin (which can arise in association with surgical application of topical bovine thrombin) may have spuriously decreased fibrinogen when assayed by this assay.

The presence of heparin above 1.0 U/mL may cause erroneously low kinetic estimates of fibrinogen, or make it impossible to measure fibrinogen by the nephelometric end point technique. In these cases, end point determinations of clottable fibrinogen by a gravimetric/spectrophotometric (biuret) technique or fibrinogen immunoassay may be helpful.

Clinical Reference

1. Dang CV, Bell WR, Shuman M: The normal and morbid biology of fibrinogen. *Am J Med* 1989;87:567-576
2. Bowie EJW, Owen CA Jr: Clinical and laboratory diagnosis of hemorrhagic disorders. In *Disorders of Hemostasis*. Edited by OD Ratnoff, CD Forbes. Philadelphia, WB Saunders Company, 1991, pp 1342-1354
3. Martinez J: Quantitative and qualitative disorders of fibrinogen. In *Hematology: Basic Principles and Practice*. Edited by R Hoffman, EJ Benz Jr. SH Shattil, et al. New York, Churchill Livingstone, 1991, pp 1342-1354
4. Mackie IJ, Kitchen S, Machin SJ, Lowe GD: Hemostasis and Thrombosis Task Force of the British committee for standards in haematology. Guidelines for fibrinogen assays. *Br H Haematol* 2003;121:396-304

Performance**Method Description**

The Clauss fibrinogen assay is performed using the HemosIL Fibrinogen-C kit on the Instrumentation Laboratory ACL TOP. Patient plasma, containing fibrinogen, is mixed with reagent containing excess thrombin. The excess thrombin

converts the fibrinogen in the patient plasma to fibrin. The amount of time it takes to form a clot is inversely proportional to the amount of fibrinogen present in the patient plasma. (Clauss A: Rapid physiological coagulation method in determination of fibrinogen. Acta Haematol 1957;17:237-246; Rossi E, Mondonico P, Lombardi A, Preda L: Method for the determination of functional [clottable] fibrinogen by the new family of ACL coagulometers. Thromb Res 1988;52:453-468; Hollensead SC, Triplett DA: Review of fibrinogen methods: clinical considerations. ASCP Check Specimen: 10[4] 1988 [TH 88-4]; Palareti G, Maccaferri M, Manotti C, et al: Fibrinogen assays: a collaborative study of six different methods. Clin Chem 1991;37:714-719)

PDF Report

No

Specimen Retention Time

7 days

Performing Laboratory Location

Rochester

Fees & Codes**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

85384

LOINC® Information

Test ID	Test Order Name	Order LOINC Value
CLFIB	Fibrinogen, Clauss, P	48664-7

Result ID	Reporting Name	LOINC®
CLFIB	Fibrinogen, Clauss, P	48664-7