

Overview

Useful For

Detection of individuals with an ongoing immune process

First-tier screening test for congenital complement deficiencies

Method Name

Automated Liposome Lysis Assay

NY State Available

Yes

Specimen

Specimen Type

Serum Red

Specimen Required

Patient Preparation: Fasting preferred.

Supplies: Sarstedt 5 mL Aliquot Tube (T914)

Collection Container/Tube: Red top (serum gel/SST are **not** acceptable)

Submission Container/Tube: Plastic vial

Specimen Volume: 1 mL

Collection Instructions:

1. Immediately after specimen collection, place the tube on wet ice.
2. Centrifuge and aliquot serum into plastic vial.
3. Immediately freeze specimen.

Specimen Minimum Volume

0.5 mL

Reject Due To

Gross hemolysis	OK
Gross lipemia	OK
Gross icterus	OK

Specimen Stability Information

Specimen Type	Temperature	Time	Special Container
Serum Red	Frozen	28 days	

Clinical & Interpretive

Clinical Information

Complement (C) proteins are components of the innate immune system. There are 3 pathways to complement activation: 1) the classical pathway, 2) the alternative (or properdin) pathway, and 3) the lectin (or mannan-binding lectin) pathway. The classical pathway of the complement system is composed of a series of proteins that are activated in response to the presence of immune complexes. A single IgM molecule or two IgG molecules are sufficient to trigger activation of the recognition complex initiated by C1q. The activation process triggers a cascade that includes an amplification loop. The amplification loop is mediated by C3, with cleavage of a series of proteins, and results in three main end products: 1) anaphylatoxins which promote inflammation (C3a, C5a), 2) opsonization peptides that are chemotactic for neutrophils (C3b) and facilitate phagocytosis, and 3) the membrane attack complex (MAC), which promotes cell lysis.

The absence of early components (C1, C2, C3, C4) of the complement cascade results in the inability of immune complexes to activate the cascade. Patients with deficiencies of the early complement proteins are unable to generate the peptides that are necessary to clear immune complexes and to attract neutrophils or to generate lytic activity. These patients have increased susceptibility to infections with encapsulated microorganisms. They may also have symptoms that suggest autoimmune disease; complement deficiency may be an etiologic factor in the development of autoimmune disease.

Patients with deficiencies of the late complement proteins (C5, C6, C7, C8, and C9) are unable to form the MAC and may have increased susceptibility to neisserial infections.

Undetectable complement levels are found in patients with specific component deficiencies. Decreased complement levels are found in infectious and autoimmune diseases due to fixation and consumption of complement.

Reference Values

30-75 U/mL

Interpretation

Low levels of total complement (total hemolytic complement) may occur during infections, disease exacerbation in patients with systemic lupus erythematosus, and in patients with immune complex diseases such as glomerulonephritis.

Undetectable levels suggest the possibility of a complement component deficiency. Individual complement component assays are useful to identify the specific deficiency.

Cautions

Because this is a functional assay, the results are dependent on appropriate specimen transport and storage.

Clinical Reference

1. Daha MR: Role of complement in innate immunity and infections. Crit Rev Immunol. 2010;30(1):47-52. doi: 10.1615/critrevimmunol.v30.i1.30

2. Prohaszka Z, Varga L, Fust G: The use of 'real-time' complement analysis to differentiate atypical haemolytic uraemic syndrome from other forms of thrombotic microangiopathies. Br J Haematol. 2012 Aug;158(3):424-425. doi: 10.1111/j.1365-2141.2012.09168.x

3. Cataland SR, Holers VM, Geyer S, Yang S, Wu HM: Biomarkers of terminal complement activation confirm the diagnosis of aHUS and differentiate aHUS from TTP. Blood. 2014 Jun;123(24):3733-3738. doi: 10.1182/blood-2013-12-547067

4. Frazer-Abel A, Sepiashvili L, Mbughuni MM, Willrich MA: Overview of laboratory testing and clinical presentations of complement deficiencies and dysregulation. Adv Clin Chem. 2016;77:1-75. doi: 10.1016/bs.acc.2016.06.001

Performance

Method Description

An automated method is performed using liposomes as the target for the serum complement system. The dinitrophenyl (DNP)-labeled liposomes are sensitized with antibody to DNP. Serum complement causes lysis and release of entrapped glucose-6-phosphate dehydrogenase. Glucose-6-phosphate dehydrogenase reacts with glucose-6-phosphate and nicotinamide adenine dinucleotide (NAD⁺). NAD⁺ is then reduced to NADH and the conversion is measured at 340 nm. The assay correlates with the total complement assay based on sheep red blood cell lysis, has lower variability, and is simpler to perform. (Package insert: Fujifilm Autokit CH50. Fujifilm Wako Pure Chemical Corporation; 04/2018; Yamamoto S, Kubotsu K, Kida M, et al: Automated homogeneous liposome-based assay system for total complement activity. Clin Chem. 1995 Apr;41(4):586-590)

PDF Report

No

Day(s) Performed

Monday through Friday

Report Available

1 to 2 days

Specimen Retention Time

14 days

Performing Laboratory Location

Rochester

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 cleared, approved, or is exempt by the US Food and Drug Administration and is used per manufacturer's instructions. Performance characteristics were verified by Mayo Clinic in a manner consistent with CLIA requirements.

CPT Code Information

86162

LOINC® Information

Test ID	Test Order Name	Order LOINC® Value
COM	Complement, Total, S	4532-8

Result ID	Test Result Name	Result LOINC® Value
COM	Complement, Total, S	4532-8