



3415A 3 Ave NW, Calgary, Alberta, T2N 0M4, Canada

**Patient Name:** PATIENT, NAME**Specimen ID (SID):** 26001-0000-00**DOB:** 01-Jan-2000**PHN:** AB 00000000**Reason for Testing:** -**Relevant Medications:** -**External SID:****Doctor:** Dr. Doctor**Report Date:** 27-Mar-2026**Specimen Type:** Plasma**Date/Time Collected:** 01-Jan-2026 / 00:00**Complement Profile Panel****Laboratory Developed Test (LDT)****Report Summary:****Sample Comments:**

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Results Summary:**High Analytes:** C1q, C4, C5a, Factor B, Factor H**High Normal Analytes:** C5, C9**Low Analytes:** C3b/iC3b**Results Interpretation:**

- Recognition-dependent complement initiation via the classical pathway is suggested by high C1q, which may reflect increased antigen-antibody or pattern recognition-driven activation and potential for downstream C4/C2 convertase assembly.
- Increased generation of terminal complement effectors is suggested by high C5a and high normal C5 and C9, which could indicate enhanced production of anaphylatoxic fragments and membrane attack complex components.
- Reduced central C3 effector fragment abundance is supported by low C3b/iC3b, which may reflect consumption or altered convertase activity with potential impact on opsonization.
- Enhanced fluid-phase regulatory control is suggested by high Factor H, which could limit C3 convertase-mediated amplification and modulate opsonic and inflammatory signaling.
- Potential for alternative pathway C3 convertase formation is supported by high Factor B, consistent with increased substrate availability for amplification in the absence of dominant regulation.
- Greater availability for classical/lectin pathway C3 convertase assembly is suggested by high C4, which may act with recognition signals to promote C3 activation.

Disclaimer:

The interpretation of these test results should be correlated with clinical findings and other diagnostic tests. Biomarker levels can vary due to many biological, physiological, and diurnal factors; their clinical significance must be assessed by a qualified healthcare professional. This information is not intended to be used as the sole basis for diagnosis or treatment decisions.

***In vitro* complement activation is likely to occur if samples are not stored and transported appropriately.**

Reviewed by: DP**Eve Technologies Corporation is a CLIA certified High Complexity International Laboratory**


PATIENT, NAME

PHN: AB 00000000; DOB: 01-Jan-2000; SID: 26001-0000-00; External SID:

Complement Profile Panel

Laboratory Developed Test (LDT)

Analyte	Results	Reference Interval†	Units
CLASSICAL PATHWAY INITIATION			
C1q	83.7 HIGH	16.7 - 59.7	µg/ml
LECTIN PATHWAY INITIATION			
Mannose-Binding Lectin	426	65 - 3259	ng/ml
CLASSICAL & LECTIN PATHWAYS C3 CONVERTASE			
C2	2621	0 - 4284	ng/ml
C4	434 HIGH	55 - 187	µg/ml
C4b	22.4	13.6 - 39.3	µg/ml
ALTERNATIVE PATHWAY C3 CONVERTASE			
Factor B	242 HIGH	65 - 212	µg/ml
Factor D	873	569 - 1774	ng/ml
Properdin	5895	4748 - 8591	ng/ml
MAJOR COMPLEMENT EFFECTOR			
C3	62.9	10 - 161	µg/ml
C3b/iC3b	< 14.4 LOW	15 - 122	µg/ml
TERMINAL PHASE			
C5	34.0	9.5 - 36.4	µg/ml
C5a	6274 HIGH	667 - 4073	pg/ml
C9	14.6	4.2 - 15.0	µg/ml
REGULATORY FACTORS			
Factor H	312 HIGH	105 - 275	µg/ml
Factor I	36.9	18.0 - 52.0	µg/ml

† Reference intervals estimated by data-mining ≥390 PLASMA samples drawn from both healthy and pathological subjects.