

About the MORL

MORL focuses on two ultra-rare complement-mediated renal diseases, C3G and aHUS. With a mission to enhance patient care, we employ two key initiatives -

1) Offer advanced diagnostic services to precisely measure complement deregulation, identify disease-driving auto-antibodies, and assess genetic variants in complement genes. These tests provide you with a comprehensive view of complement deregulation in patients.

2) Conduct complement research to better understand the underlying disease pathophysiology, using cutting-edge technology and novel complement assays.

These testing panels are backed by over 25 years of experience offered by a multidisciplinary team that includes scientists and clinicians who review all data to offer expert contextual interpretation of the findings.

The MORL also plays a pivotal role in the development of complement-targeted therapeutics, partnering with stakeholders to advance treatment options.

Understanding complement-mediated kidney diseases through genetic and functional testing.

Why the MORL

Cutting-edge technology: Our state-of-the-art lab is equipped with advanced technology for accurate and precise biomarker and genetic analysis.

Expert team: Our team of experienced scientists and medical professionals provides expert insight into complement disorders.

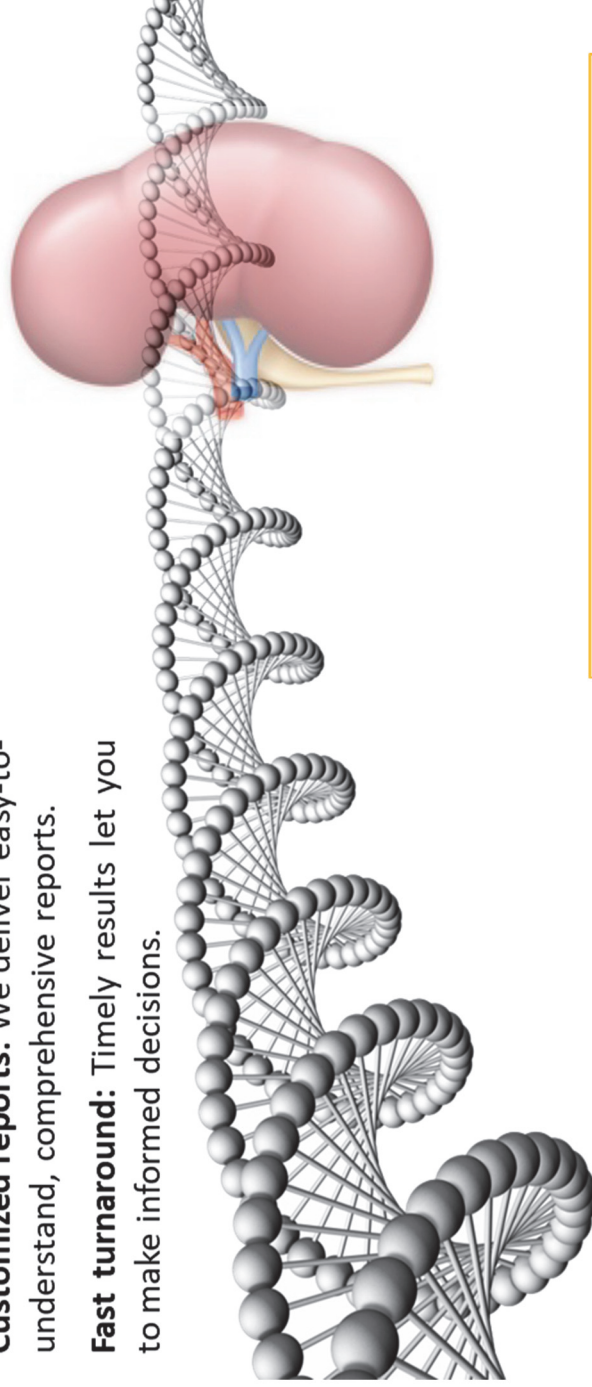
Customized reports: We deliver easy-to-understand, comprehensive reports.

Fast turnaround: Timely results let you to make informed decisions.

Advancing Nephrology Care
Through Complement Testing

C3 Glomerulopathy

Atypical Hemolytic Uremic Syndrome



Complement Serological Panels
Complement Genetic Panels

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For a thorough evaluation of complement-mediated disorders, four vital data types are essential.

1. **Complement Pathway Activity:** Monitoring complement activity aids in predicting disease status and therapeutic response.
2. **Biomarker Profile:** Specific biomarkers offer insight into complement-related pathology, aiding in disease definition and activity assessment.
3. **Acquired Drivers of Disease:** Detecting antibodies to components like C3 and C5 convertases, factor H, and factor B can quantify a patient's clinical progression.
4. **Genetic Drivers of Disease:** Recognizing genetic factors sheds light on disease etiology, heritability and recurrence chance.

These data, when combined, provide a comprehensive understanding of the variety of complement disorders and facilitate personalized tailored patient care.

Complement Pathway Activity

- CH50
- AP activity
- C3b deposition assay
- Fluid-phase activity assay

Biomarker Profiling

- C3, C3c, C4, FB, Ba, Bb, FD
Biomarkers reflect C3 convertase activity
- C5, Properdin, Soluble C5b-9
Biomarkers reflect C5 convertase activity
- Factor H and Factor I
Fluid-phase complement regulators

TMA Differential Testing

- ADAMTS-13 testing
Distinguish TTP from other TMAs

Acquired Disease Drivers

- C3 nephritic factors
- C4 nephritic factors
- C5 nephritic factors
- FH autoantibodies
- FB autoantibodies

Genetic Disease Drivers

- Next-Gen Targeted Panel
C3, CFB, CFH, CFI, CD46, DGKE, G6PD, MMACHC, PLG, THBD, ADAMTS13
- CNV analysis
Structural variation in the *CFH-CFHR5* region by multiplex ligation-dependent probe amplification

