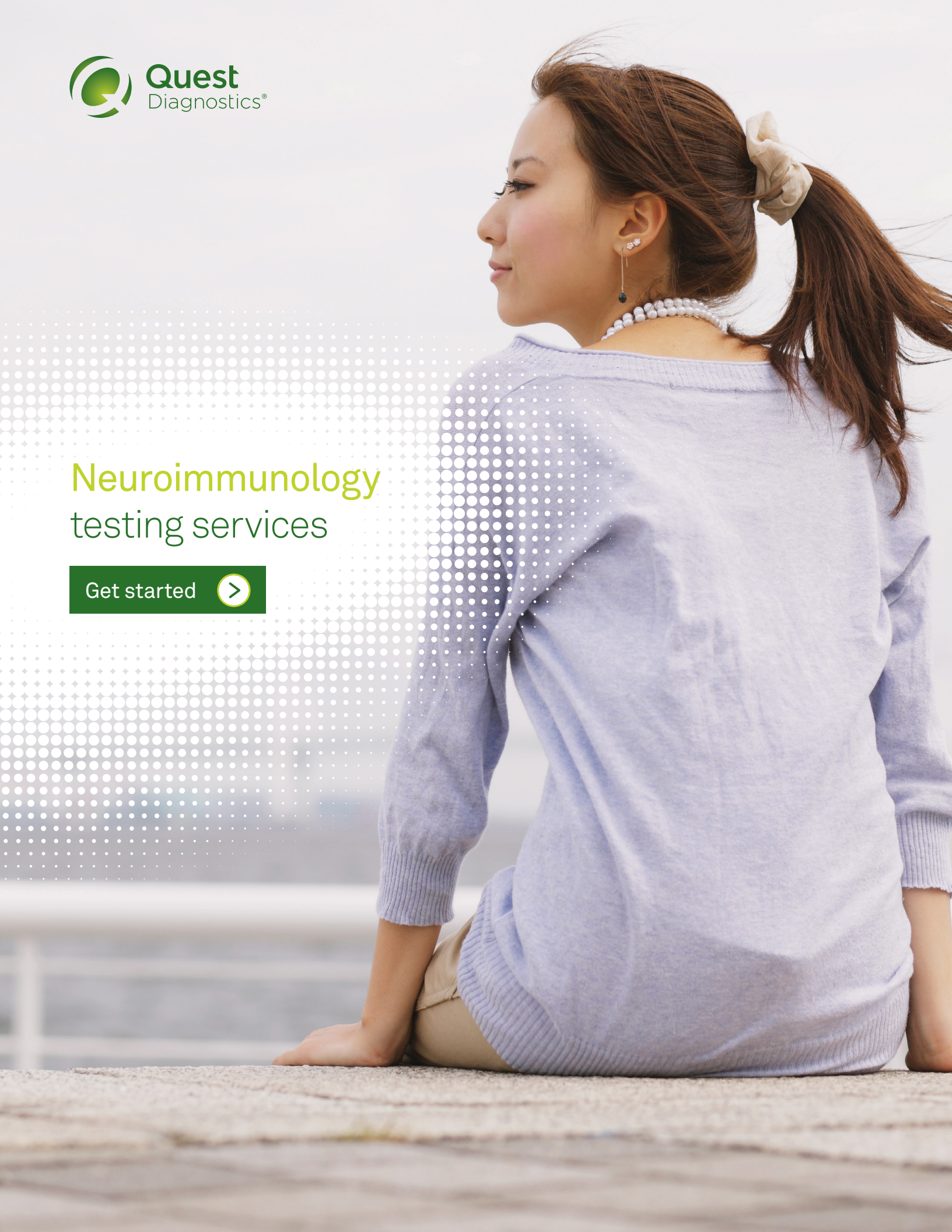
Quest Diagnostics®

Neuroimmunology testing services

Get started



Neuroimmunology testing through Quest Diagnostics

Neuroimmunological diseases are complex. Testing for them shouldn't be. We make advanced neuroimmunology testing more accessible and actionable. When you need to know more, you'll find more here.

At Quest Advanced™ Neurology, everything we do is about our **commitment to the patient as a person.**

Provider:

Expansive test menu
- Neuroimmunology testing is performed only at Quest facilities to ensure test quality and performance
- Over 40 neurologic antibodies available

Wide-ranging medical expertise
- Over 650 medical and scientific experts on staff
- Available for one-on-one consultations

Single antibody test offerings
- Over 40 antibodies that can be ordered individually

Gold standard methodologies
- Target tissues and/or cells are specifically selected for optimal diagnostic detection
- Methodologies include CBA, RIA, and line blot

Patient:

Extensive health plan coverage
- Participating provider with most major health plans, including traditional Medicare and Medicaid
- UnitedHealthcare® Preferred Lab Network provider

Access to more than 2,250 Patient Service Centers (PSCs)
- Convenient locations for specimen collection
- Walk-in and appointment scheduling to promote patient compliance

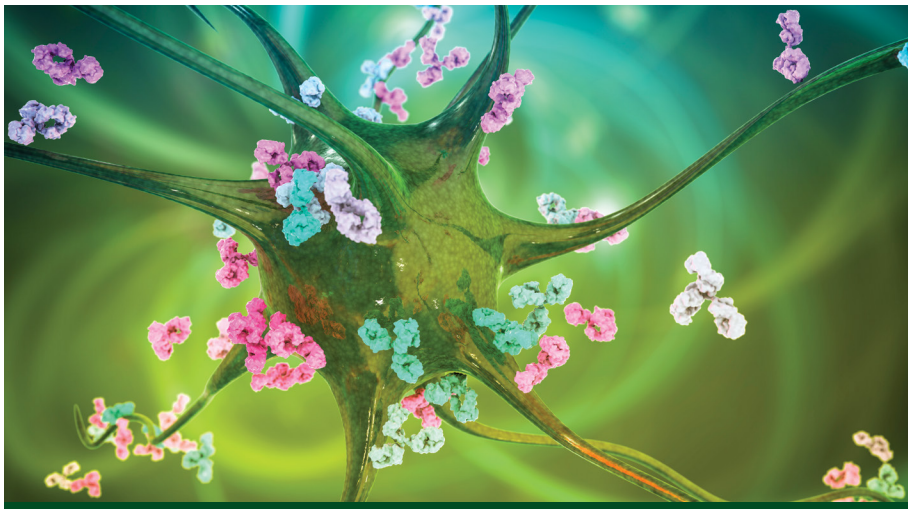
Practice:

EHR integration
- Interfaces with over 600 EHR systems, more than any other lab company

Autoantibody testing services for autoimmune neurology disorders

When patients present with a range of neurological symptoms, it can be difficult to identify what is causing them and select the best treatment. In most cases, early detection and prompt therapy can improve patient outcomes.[1]

Quest Diagnostics is commited to delivering neuroimmunological diagnostic solutions built around a data-driven selection of neuronal-specific antibodies known to be associated with specific conditions and diseases.



Comprehensive testing may:

- Identify idiopathic neurological disease in the absence of a tumor
- Identify a malignancy early to optimize treatment and help improve outcomes
- Inform targeted immunosuppressive therapy for immunological disease

Knowing where to look first

Patients often manifest a variety of clinical syndromes that can be caused by one or more autoantibodies. To complicate the evaluation, a given autoantibody can exhibit a variety of clinical appearances and a given neurological presentation can often be caused by several different autoantibodies. The complexity of the neurological system and the absence of a specific clinical presentation make it difficult to know where to look first.

In a clinical study of 16,700 samples tested for neurological autoantibodies, approximately 50% tested positive for autoantibodies other than those included in the initial testing order.[2]

By screening patients for multiple autoantibodies, the detection rate for diagnostically relevant autoantibodies **increased by 87%,** compared to single testing of requested analytes.[2]

46.6%
positive for unrequested parameters

53.4%
positive for requested parameters

We offer expanded neuroimmunological testing for many autoimmune neurological disorders

Early identification of all clinically relevant central nervous system (CNS) autoantibodies

It is important to test for newly discovered neural autoantibodies as many are associated with treatable neurological diseases.

Autoantibody-mediated neuroimmunological disorders can arise from tumors, genetic predisposition, or even infections such as polyneuropathy disorder and Guillain-Barré syndrome (GBS).

When a patient presents with symptoms suggesting a CNS autoimmune disorder, early identification of antibodies can help direct therapy in patients likely to improve with treatments such as immunotherapy.

Table 1 Autoimmune neurological disorders

Movement disorders	Autoimmune ataxia, epilepsy, chorea
Neuromuscular disorders	Myasthenia gravis, Lambert-Eaton
Neuro-oncology	Paraneoplastic neurological syndrome (PNS)
Brain function disorders	Autoimmune dementia, encephalopathy
Demyelinating disorders	Multiple sclerosis (MS), neuromyelitis optica (NMO), myelin oligodendrocyte glycoprotein (MOG)
Peripheral nervous system (PNS) disorders	Guillain-Barré syndrome (GBS), peripheral and sensory neuropathies

Table 2 ICD-10 codes[a]

ICD-10 code	Symptom description
CNS disorders	
G62.9	Polyneuropathy, unspecified
G60.9	Hereditary and idiopathic neuropathy, unspecified
R20.2	Paresthesia of skin
M62.81	Muscle weakness (generalized)
R20.9	Unspecified disturbances of skin sensation
R41.3	Other amnesia
R27.0	Ataxia, unspecified
G60.3	Idiopathic progressive neuropathy
R20.0	Anesthesia of skin
R53.83	Other fatigue
R26.9	Unspecified abnormalities of gait and mobility
G13.0	Paraneoplastic neuromyopathy and neuropathy
D49.9	Neoplasm of unspecified behavior of unspecified site

ICD-10 code	Symptom description
Neuromyelitis optica spectrum disorders	
G36.0	Neuromyelitis optica [Devic]
G35	Multiple sclerosis
R42	Dizziness and giddiness
G95.9	Disease of spinal cord, unspecified
Z79.899	Other long term (current) drug therapy
E78.5	Hyperlipidemia, unspecified
G37.9	Demyelinating disease of central nervous system, unspecified
M12.9	Arthropathy, unspecified
G37.3	Acute transverse myelitis in demyelinating disease of central nervous system
H46.9	Unspecified optic neuritis
E53.8	Deficiency of other specified B group vitamins
G04.90	Encephalitis and encephalomyelitis, unspecified
E77.9	Disorder of glycoprotein metabolism, unspecified
E77.8	Other disorders of glycoprotein metabolism
E77.1	Defects in glycoprotein degradation
E74.00	Glycogen storage disease, unspecified

ICD-10 code	Symptom description
Myasthenia gravis	
H53.2	Diplopia
G70.00	Myasthenia gravis without (acute) exacerbation
G70.01	Myasthenia gravis with (acute) exacerbation
M62.81	Muscle weakness (generalized)
H02.409	Unspecified ptosis of unspecified eyelid
H02.403	Unspecified ptosis of bilateral eyelids
H02.402	Unspecified ptosis of left eyelid
H02.401	Unspecified ptosis of right eyelid
R53.1	Weakness
E03.9	Hypothyroidism, unspecified

[a] This list of commonly submitted diagnoses is intended to assist ordering physicians in providing ICD-10-CM codes. This is not a comprehensive list and an ICD-10-CM book should be used as the official reference.

Note: Diagnoses must always be documented in the patient's medical record. The ultimate responsibility belongs to the ordering physician to correctly assign the patient's diagnosis based on the patient's history, symptoms, and medical condition.

Paraneoplastic and other CNS disorders

Paraneoplastic neurological syndromes (PNS) are a set of degenerative autoimmune disorders due to the remote effects of cancer. Identification of a specific paraneoplastic antibody can guide the search for an underlying malignancy.

Early detection and quick treatment can make a difference in patient outcomes

The positive identification of specific antibodies can help direct therapy to improve patient outcomes, avoid treatment that may harm the patient, and/or aid in early detection and treatment of cancer.

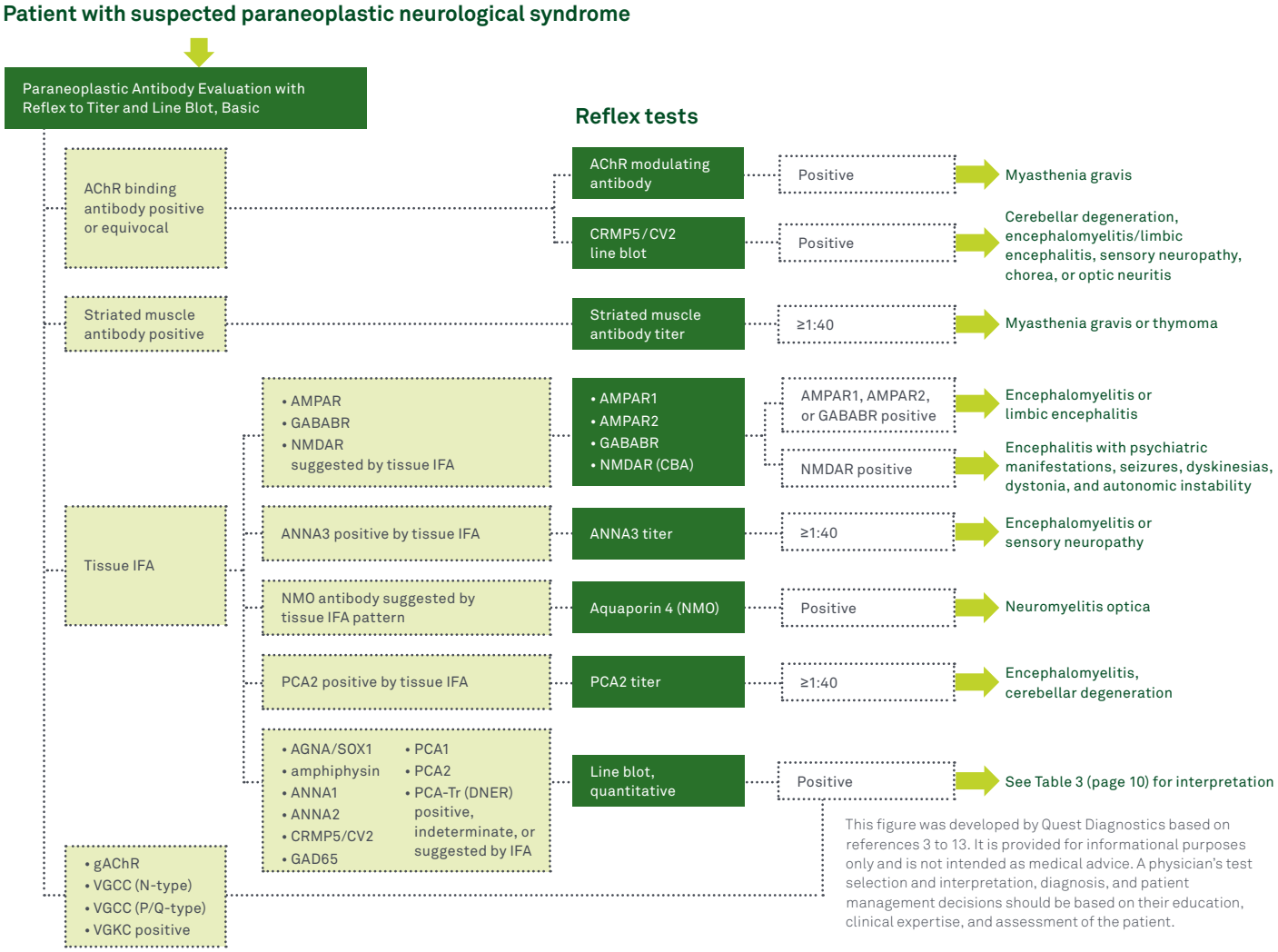
Paraneoplastic antibodies

In a majority of PNS, the neurological symptoms appear before the cancer has been identified. Identification of paraneoplastic antibodies can direct the search for an underlying cancer, increasing the likelihood of making an early diagnosis of the tumor and treating the neurological symptoms.

Other CNS autoantibodies

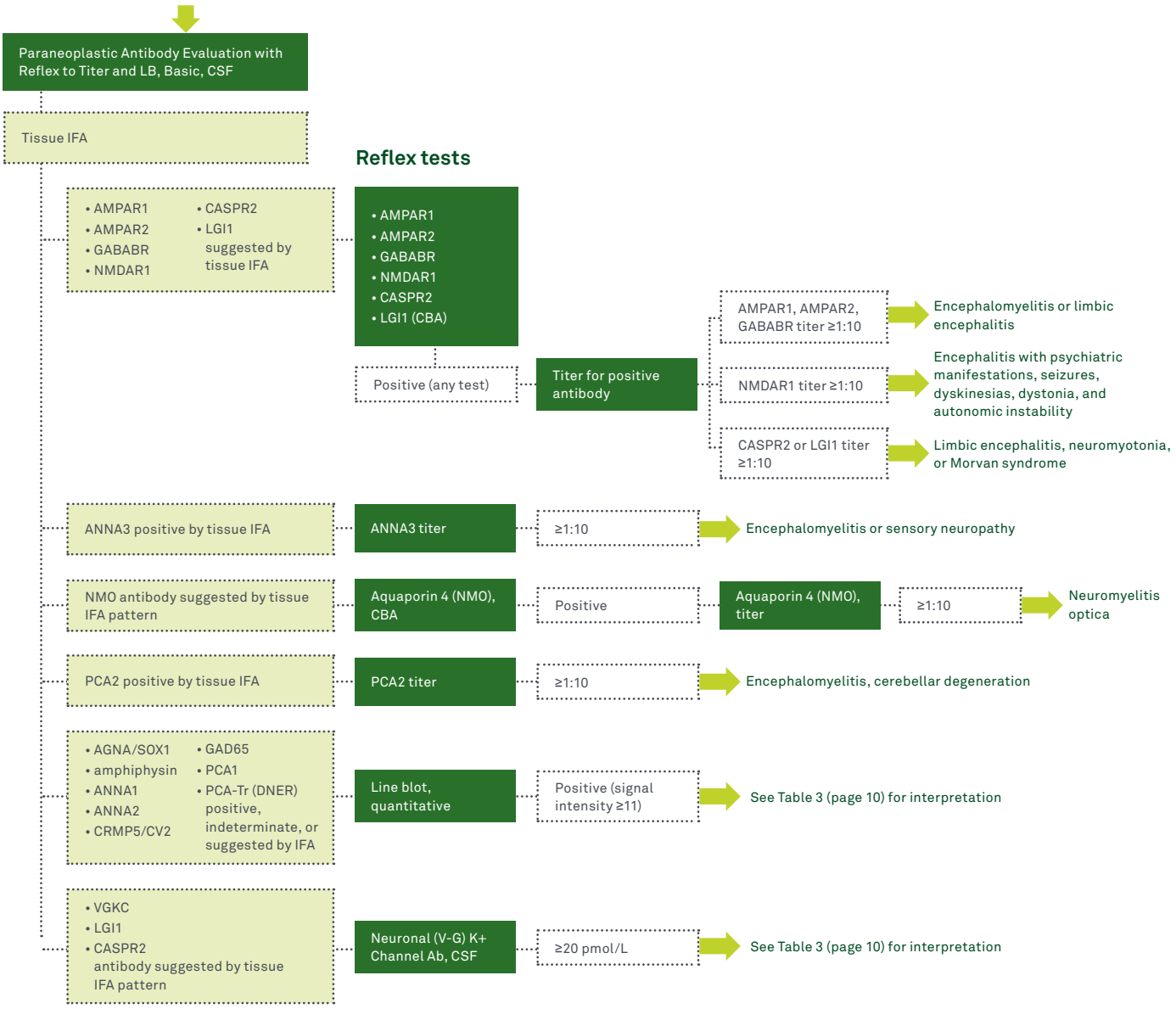
Immunotherapy and other treatments have been successful in patients with antibodies against LGI1, CASPR2, VGKC, NMDA (NR1), and GAD65. Early detection may enable better outcomes.[1]

Figure 1 Paraneoplastic Antibody Evaluation with Reflex to Titer and Line Blot, Basic (test code 93876) panel
See Table 3 (page 10) for interpretations



This figure was developed by Quest Diagnostics based on references 3 to 13. It is provided for informational purposes only and is not intended as medical advice. A physician's test selection and interpretation, diagnosis, and patient management decisions should be based on their education, clinical expertise, and assessment of the patient.

Figure 2 Paraneoplastic Antibody Evaluation with Reflex to Titer and LB, Basic, CSF (test code 94536) panel
See Table 3 (page 10) for interpretations



This figure was developed by Quest Diagnostics based on references 8, 12, 14, and 15. It is provided for informational purposes only and is not intended as medical advice.
A physician's test selection and interpretation, diagnosis, and patient management decisions should be based on his/her education, clinical expertise, and assessment of the patient.

Paraneoplastic Antibody Expanded Evaluation

In 60% of patients with paraneoplastic neurological syndromes, the symptoms occur before the diagnosis of cancer is made[16]

In most cases, neurological symptoms include memory disturbance, cognitive impairment, seizures, psychosis, loss of consciousness, or even coma.

The Paraneoplastic Antibody Expanded Evaluation from Quest always uses a cell-based assay (CBA) as part of the initial panel assessment and has the ability to identify 25 prevalent antibodies, including Ma2/Ta and Zic4

Detection of central nervous system autoantibodies is generally better achieved with CBAs[17]

65%
missed by IFA alone

Utilizing CBA increases the likelihood of **identifying membrane-embedded protein targets, LGI1, CASPR2, NMDA (NR1), AMPAR, and GABABR**

Reasons to test with Quest's expanded panel:

1. **Identify PNS antibodies** that can increase the likelihood of early diagnosis and treatment
2. **Understand disease progression and prognosis** so you and your patients know what to expect
3. **Discover comorbidities or underlying conditions** such as encephalitis, ataxia, or myasthenia gravis so you can plan the right care pathway

Figure 3 Paraneoplastic Antibody Expanded Evaluation with Reflex to Titer and LB, Serum (test code 94957) consists of 3 distinct panels, with the appropriate titer reflex if an antibody is positively identified. See Table 3 (page 10) for interpretations.

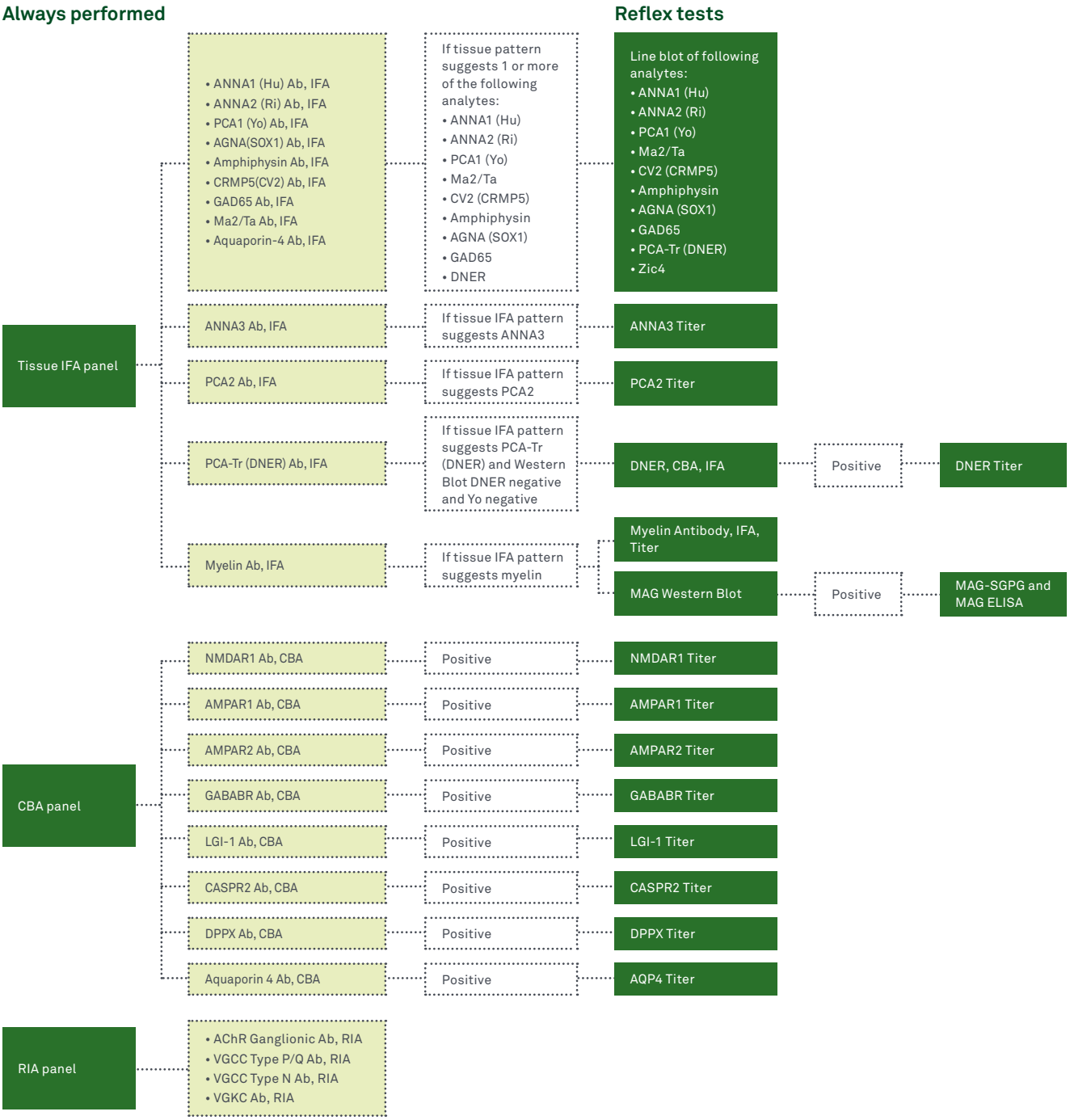


Figure 4 Autoimmune Neurology Antibody Comprehensive Panel with Reflexes, Serum (test code 93888)
See Table 3 (page 10) for interpretations

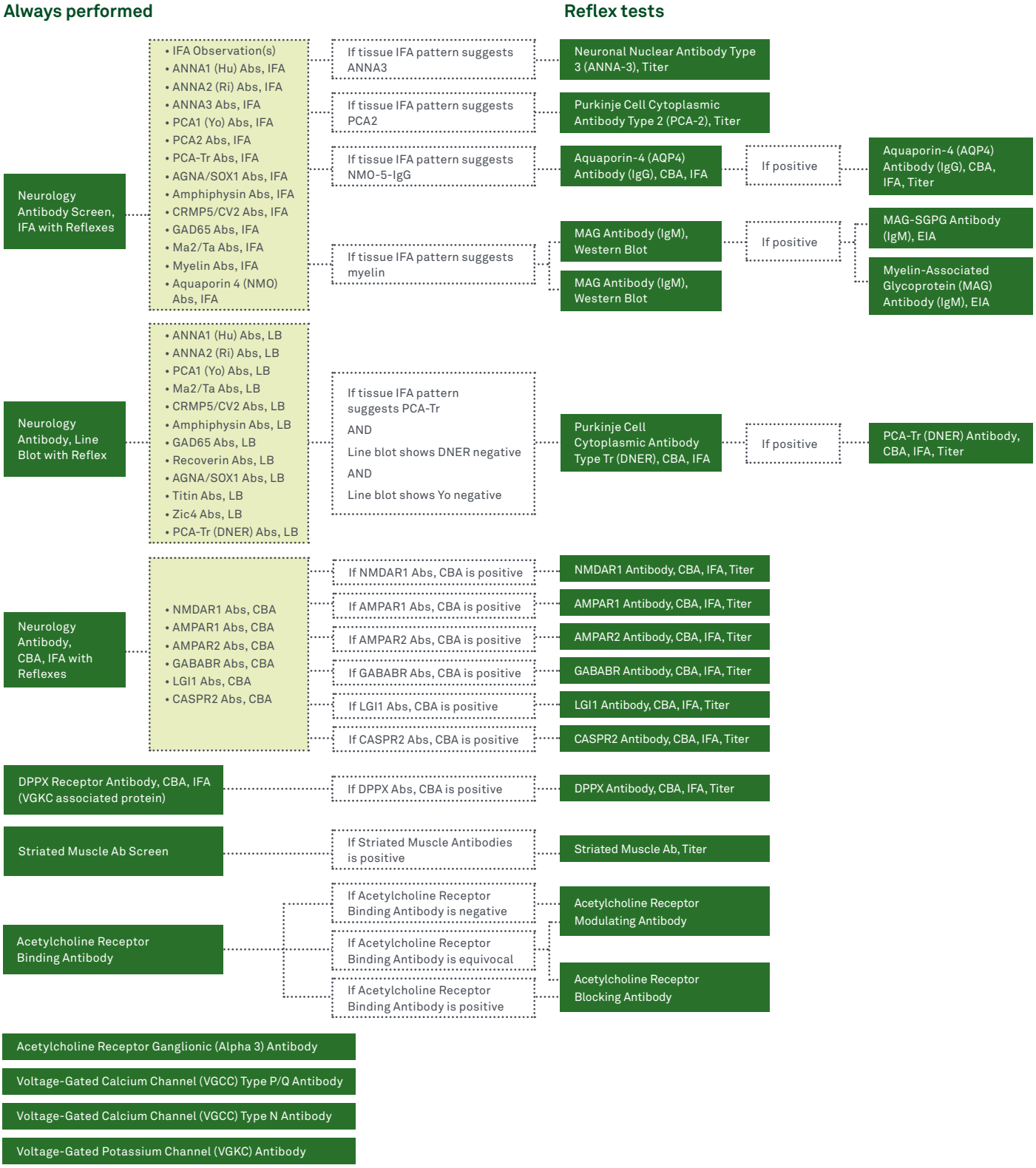


Table 3 Paraneoplastic antibodies and associated cancers and clinical significance

	Associated neurologic syndrome[b]												Associated tumor or cancer type									
Autoantibody target[b]	Autonomic neuropathy	Brainstem encephalitis/ opsoclonus-myoclonus	Cerebellar degeneration	LE/encephalomyelitis	LEMS	Myasthenia gravis	Neuromyelitis optica	Neuromyotonia	Sensory neuropathy	Stiff person	Other	Breast cancer	Hodgkin lymphoma	Lung cancer	Ovarian cancer	Prostate cancer	Renal cell cancer	SCLC	Testicular tumor	Thymoma	Other
AChR						●														○	
AGNA/SOX1			●	●	●				●					●				○			
AMPAR				●								●		●	●			●		●	
Amphiphysin				●					●	●		○			●			○			
ANNA1 (Hu)	●	●	●	●					●			●		●	●	●		○			bladder, gastrointestinal, pancreas
ANNA2 (Ri)		●	●									○		○				●			gynecological
ANNA3[c]				●					●									●			
Aquaporin 4 (NMO)							●					●		●							
CASPR2				●				●			●[d]									○	melanoma
CRMP5/CV2	●		●	●					●		●[d]							○		○	
DPPX				●																	
GABABR				●										●[e]				○			
gAChR	●								●		●[d]	●		●		●	●	●		●	gastrointestinal, lymphoid, melanoma, bladder
GAD65			●	●						●		●		●				●		●	pancreatic, thymic
LGI1				●								●				●					nonmelanoma skin, colon
Ma2/Ta		●	●	●															○		
MAG									●												MGUS[e]
NMDAR1											●[d]				○[e]				●[e]		
PCA1 (Yo)			●									●			○						
PCA2[c]			●	●					●									●			
PCA-Tr (DNER)			●										○								
Recoverin											●[d]							○			
RyR						●														○	
Striated muscle						●														○	
Titin						●														○	
VGCC, N-type					●													○			
VGCC, P/Q-type			●		●													○			
VGKC				●				●			●[d]							●		●	
Zic4			●															○			

○ Indicates tumor type(s) most frequently associated with the antibody.
GI, gastrointestinal; LE, limbic encephalitis; LEMS, Lambert-Eaton myasthenic syndrome; MGUS, monoclonal gammopathy of uncertain significance; SCLC, small-cell lung cancer.

[b] Autoantibodies and syndromes can occur in the absence of cancer or a tumor.

[c] Case report.

[d] Other reported syndromes: CASPR2, Morvan syndrome; CRMP5/CV2, chorea and optic neuritis; gAChR, cortical and neuropsychiatric presentation; NMADR1, encephalitis with psychiatric manifestations, seizures, dyskinesias, dystonia, and autonomic instability; recoverin, cancer-associated retinopathy; VGKC, Morvan syndrome.

[e] Notes on cancers: GABABR, lung tumor; MAG, MGUS is not cancer but can progress to cancer; NMADR1, teratoma.

Autoimmune encephalitis

Autoimmune encephalitis is a relatively new category of immune-mediated disease involving the central nervous system.[18] It can impair function, and present via a subacute onset of memory disturbance, cognitive impairment, seizures, psychosis, and a loss of consciousness or even coma.

The direct causes of autoimmune encephalitis are unknown; it is often accompanied by a paraneoplastic disorder or exposure to common bacteria (streptococcus or mycoplasma pneumonia, with or without active infection).

The importance of an early diagnosis

Autoimmune encephalitis can be a difficult clinical diagnosis for physicians due to:

- Overlapping clinical, imaging, and laboratory features that **mimic other disorders**
- Symptoms that can appear at **various times and intensities**

Screening tests that can identify the correct pathophysiology of autoimmune encephalitis can help physicians select an appropriate first-line therapy, which often consists of corticosteroids, IV immunoglobulin (IVIg), plasma exchange, or tumor removal.

Timely initiation of the appropriate therapy gives patients the best chance at a successful recovery.[18] Research shows that 50% of patients with anti-NMDA receptor encephalitis show improvement within 4 weeks of receiving treatment, and 80% of patients have partial or complete recovery following treatment.[19]

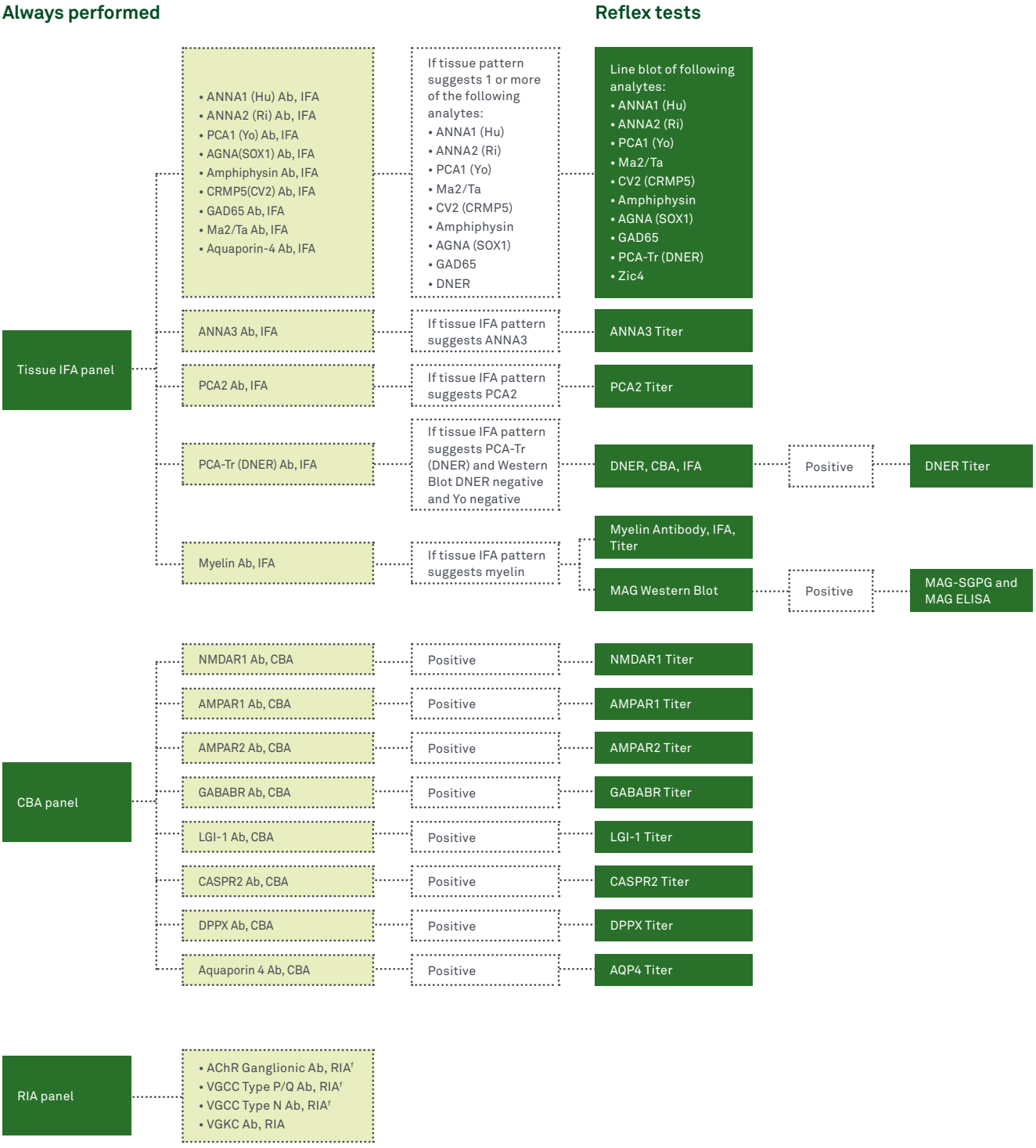
A comprehensive testing solution that streamlines the path to diagnosis

The Autoimmune Encephalitis Evaluation panel (test codes 94955 and 94958) is built on 25 antibodies commonly found in autoimmune encephalitis. A 3- to 14-day turnaround can be significant, allowing physicians to establish an effective treatment protocol and halt the progression of devastating symptoms.

3 reasons to order autoimmune encephalitis testing from Quest

- **Turnaround time** of 3 to 14 days can help physicians make a differential diagnosis and initiate appropriate treatment.
- **CBA panel** is always performed, and includes NMDA antibodies that are consistent with limbic encephalitis.
- This panel includes the **most prevalent encephalitis antibodies,** including Ma2/Ta.

Figure 5 The Encephalitis Antibody Evaluation with Reflex to Titer and Line Blot, Serum (test code 94955) consists of 3 distinct panels, with the appropriate titer reflex if an antibody is positively identified.



[f] gAChR, VGCC (N-type), VGCC (P/Q type) are not included in the CSF panel: Encephalitis Antibody Evaluation with Reflex to Titer and Line Blot, CSF (test code 94958).

Neuromyelitis optica spectrum disorder (NMOSD)

What is NMOSD?

Neuromyelitis optica spectrum disorder (NMOSD) is an immune-mediated, chronic, and often relapsing inflammatory disease that predominantly affects the optic nerve and spinal cord.[20] It can sometimes be mistaken for multiple sclerosis (MS) because many of the symptoms overlap between the 2 diseases.

NMOSD attacks generally have a sudden, severe onset, causing immediate disability, including blindness and paralysis, while MS episodes tend to be more variable or milder, especially in the early stages of the disease.

NMOSD patients often simultaneously present with **optic neuritis and transverse myelitis.**[20]

NMOSD is generally **mediated by anti-aquaporin (AQP4) antibodies**. An AQP4 test is generally negative in MS patients.[21]

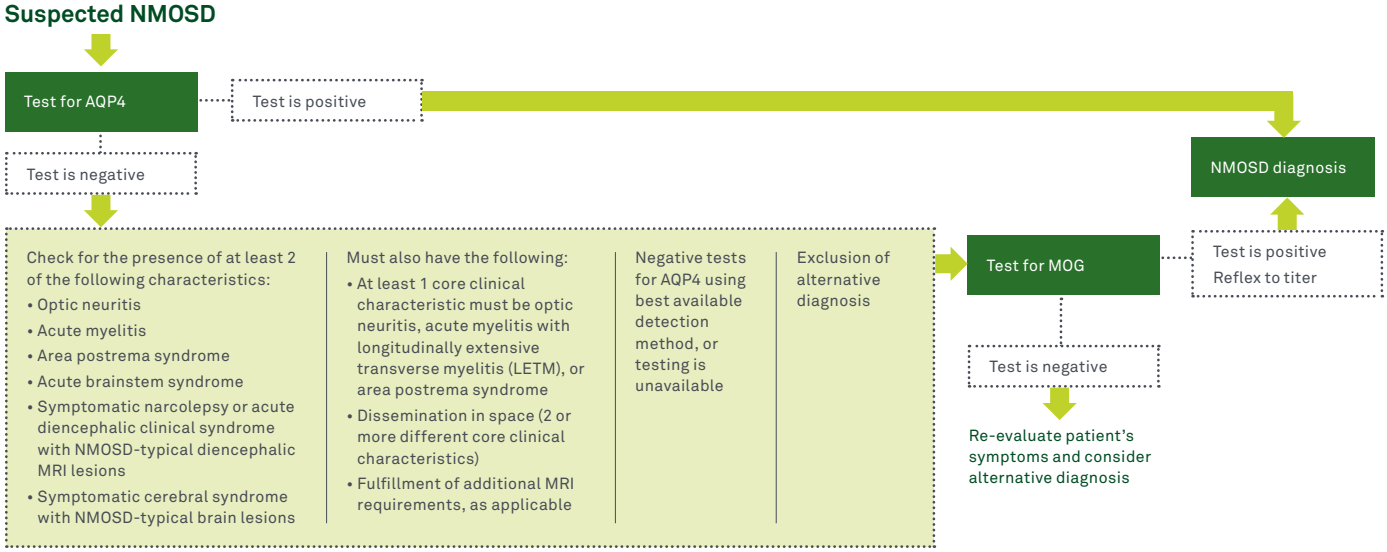
Early diagnosis is critical because treatments that are effective for MS or other demyelinating disorders might be ineffective, or even harmful, for patients with NMOSD.[22] When used in conjunction with other clinical testing, the NMO Spectrum Evaluation (AQP4 with reflex to MOG) can help physicians make an informed diagnosis, and aid in clinical decision management.

MOG antibody testing can provide insight and streamline the path to diagnosis and treatment

No one clinical characteristic is exclusive to NMOSD,[23] so it can be difficult to make a definitive diagnosis. The comprehensive test menu from Quest Diagnostics includes MOG, AQP4, and reflex options that can deliver clear, positive identification to help you diagnose NMOSD and treat patients sooner.

AQP4 is not the only antibody that can play a role in NMOSD. A growing body of research indicates that 10% to 50% of patients with NMOSD often test negative for AQP4,[24] and 15% to 35% of these patients test positive for MOG antibodies.[20, 22, 25-26]

Figure 5 Diagnostic criteria for NMOSD without AQP4 or NMOSD with unknown AQP4-IgG status[23]



This algorithm is intended as a guide for using Quest Diagnostics laboratory tests for diagnosing neuromyelitis optica spectrum disorder (NMOSD), based on Wingerchuck et al, 2015. The algorithm is provided for informational purposes only and is not intended as medical advice. A physician's test selection and interpretation, diagnosis, and patient management decisions should be based on his/her education, clinical expertise, and assessment of the patient.

Immunological testing for myasthenia gravis (MG)

Myasthenia Gravis Panel 2 with reflex— quantitative AChR binding, blocking, modulating antibodies with reflex to MuSK antibody testing

Myasthenia gravis (MG) is an autoimmune disorder characterized by muscle weakness that ranges from mild to severe for multiple muscle groups. MG affects approximately 20 per 100,000[27] patients and most commonly involves either acetylcholine receptors (AChR) or muscle-specific kinase receptors (MuSK) that ultimately inhibit muscle contraction. 90% have ptosis or diplopia with pupillary involvement. Onset of symptoms is generally gradual but can sometimes be acute following viral infection or pregnancy.

AChR

Quantifying AChR antibodies may be important as AChR antibody levels are directly proportional to disease severity in the population. AChR-positive patients may experience limb weakness, ptosis, diplopia, dysarthria, or dysphagia. **Binding antibodies were present in 82% of patients** with moderate/severe generalized disease; 69% of patients with mild, generalized disease; and 59% of patients with ocular myasthenia.[27]

MuSK

In seronegative AChR patients, 30% to 40% have antibodies to MuSK.[28] Overall, MuSK antibodies are seen in approximately 7% to 10% of all MG patients.[28] MuSK antibody-positive MG patients are also less likely to respond to acetylcholinesterase inhibitors (AChE), and symptoms may worsen with certain medications.[29]

MuSK antibody-positive patients may experience more pronounced bulbar weakness and may have tongue and facial atrophy. It is important that MuSK antibody–positive MG patients are identified, as one-third of patients experience a life-threatening respiratory crisis, and long-term immunosuppression is the sole treatment.[30] Patients who test positive for MuSK antibodies are much less likely to have thymomas.

LRP4

LRP4 antibodies are detected in a subset of MG patients lacking detectable AChR and MuSK antibodies. Particularly, **LRP4 antibodies were observed in double seronegative ocular MG.** LRP4-MG seems to present with more mild symptoms. Overall, the response of LRP4-MG patients to treatment was similar to published responses of AChR-MG rather than to MuSK-MG patients,[31] so understanding LRP4 presence can be helpful.

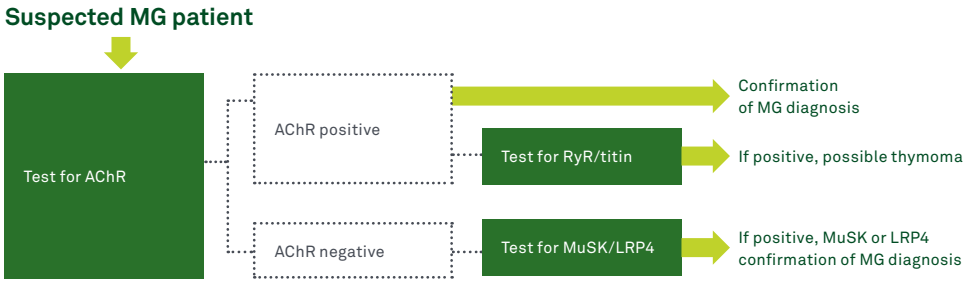
RyR

RyR antibodies are found in 13% to 38% of all AChR-positive patients. Their presence is associated with more severe and late-onset MG and can provide a strong indication of thymoma. RyR-postive patients may present with predominant bulbar and neck weakness, non-limb presentation, respiratory symptoms, and associated increased cardiac disease risk.

Titin

The presence of titin is associated with more severe and late-onset MG. It can provide **a strong indication of thymoma and ultimately involves the proximal compartment of both upper and lower limbs**. Titin-positive patients may experience pelvic girdle weakness, foot drop, and neck weakness and have variable degrees of Achilles tendon contractures, spinal rigidity, and muscle hypertrophy.[32]

Figure 6



Quest Diagnostics myasthenia gravis portfolio includes testing for LRP4 antibodies

Get the diagnosis you need so you can focus on treatment and patient care.

LRP4 antibodies are believed to be the **third-leading cause** of myasthenia gravis (MG)[33]

The mean age of patients testing positively for LRP4 is 57 years, **with approximately equal gender distribution**[35]

13%

LRP4 antibodies were found in **13% of patients testing negative for other MG antibodies** (AChR, MuSK), according to a recent study[34]

Most LRP4-positive patients **improve after standard MG therapy**[34]

Table 4 Test ordering information (see Table 5 on page 17 for additional tests)

Test code	Test name	Turnaround time (TAT)	Specimen requirements	CPT codes
Neuro-oncology tests				
93876	Paraneoplastic Antibody Evaluation with Reflex to Titer and Line Blot, Basic (test components are listed in algorithm on page 5)	7 to 14 days	Serum; frozen; 6 mL preferred (3.5 mL minimum)	86255 (×10), 83519 (×5)
94536	Paraneoplastic Antibody Evaluation with Reflex to Titer and LB, Basic, CSF (test components listed in algorithm on page 6)	7 to 14 days	3.8 mL CSF collected in a sterile leak-proof container (2.4 mL minimum)	86255 (x10), 86341
94957	Paraneoplastic Antibody Expanded Evaluation w/Reflex to Titer and LB, Serum (test components listed in algorithm on page 8)	3 to 14 days	Serum; frozen; 6 mL preferred (3.5 mL minimum)	86255 (x20), 83519 (x4), 86341
93888	Autoimmune Neurology Antibody Comprehensive Panel with Reflexes, Serum (test components listed in algorithm on page 9)	7 to 16 days	7 mL (4.5 mL minimum) serum	86255 (x20), 86341 (x2), 84182 (x11), 83519 (x5)
Autoimmune encephalitis				
94955	Encephalitis Antibody Evaluation with Reflex to Titer and Line Blot, Serum (test components listed in algorithm on page 12)	3 to 14 days	6 mL serum preferred (3.5 mL serum minimum); collected in red-top tube	86255 (x20), 86341, 83519 (x4)
94958	Encephalitis Antibody Evaluation with Reflex to Titer and Line Blot, CSF	3 to 14 days	6 mL CSF preferred (3.5 mL CSF minimum); collected in sterile leak-proof container	86255 (x20), 83519, 86341
Neuromyelitis optica spectrum disorders				
90382	Aquaporin-4 (AQP4) Antibody (NMO-IgG), ELISA	6 to 8 days	2 mL serum (0.5 mL minimum)	83516
93893	Aquaporin-4 Antibody (IgG), CBA	7 days	0.5 mL (0.3 mL minimum) serum collected in a red-top tube (no gel) or CSF collected in a sterile, screw-cap container	86255
38321	Aquaporin-4 (AQP4) (NMO-IgG) Antibody with Reflex to Titer, Serum	3 to 7 days	2 mL (0.5 mL minimum) serum collected in a red-top tube (no gel)	86255
36952	Myelin Oligodendrocyte Glycoprotein (MOG) Antibody with Reflex to Titer, Serum	3 to 7 days	2 mL serum preferred (0.5 mL minimum); collected in red-top tube (no gel)	86255 reflex to 86256
36954	Myelin Oligodendrocyte Glycoprotein (MOG) Antibody with Reflex to Titer, CSF	3 to 7 days	2 mL serum preferred (0.5 mL minimum); collected in a screw-cap vial	86255 reflex to 86256
38312	NMO Spectrum Evaluation (AQP4 with Reflex to MOG), Serum	7 to 10 days	2 mL serum preferred (1 mL minimum); collected in a red-top tube (no gel)	86255
38313	NMO Spectrum Evaluation (AQP4 with Reflex to MOG), CSF	7 to 10 days	2 mL CSF preferred (1 mL minimum); collected in a sterile plastic screw-cap vial	86255
Myasthenia gravis				
93859	Myasthenia Gravis Panel 2 with Reflex to MuSK Antibody AChR Binding, Blocking, Modulating Antibody	7 to 14 days	3 mL serum (0.7 mL minimum)	83519 (×3) without reflex 83519 (×4) with MuSK reflex
206	Acetylcholine Receptor Binding Antibody	1 to 2 days	1 mL serum (0.5 mL minimum)	83519
34459	Acetylcholine Receptor Blocking Antibody	3 to 5 days	1 mL serum (0.5 mL minimum)	83519
26474	Acetylcholine Receptor Modulating Antibody	5 days	1 mL serum (0.5 mL minimum)	83519
94744	LRP4 Autoantibody Test	7-14 days	2 mL Serum (0.5 mL minimum)	86255
1490	MuSK and LRP4	7-14 days	2 mL Serum (0.5 mL minimum)	83519 and 86255
18842	MuSK Antibody Test	4 to 7 days	2 mL serum (0.5 mL minimum)	83519
266	Striated Muscle Antibody with Reflex to Titer	5 days	0.5 mL serum (0.1 mL minimum)	86255 (86256 with titer reflex)
7550	Myasthenia Gravis Panel 1 AChR Binding, Anti-Striated Muscle Antibody with Reflex	5 days	2 mL serum (0.4 mL minimum)	83519, 86255 (86256 with titer reflex)
10104	Myasthenia Gravis Panel 2 AChR Binding, Blocking, Modulating Antibody	5 days	2 mL serum (0.7 mL minimum)	83519 (×3)
10211	Myasthenia Gravis Panel 3 AChR Binding, Blocking, Modulating Antibody, Anti-Striated Muscle Antibody with Reflex	5 days	2 mL serum (0.8 mL minimum)	83519 (×3), 86255 (86256 with titer reflex)
Multiple sclerosis				
17728	Multiple Sclerosis Panel Myelin Basic Protein, Oligoclonal Bands IgG	4 to 6 days	2.2 mL CSF and 1 mL serum (1 mL CSF and 0.5 mL serum minimum)	83873, 83916
37581	Multiple Sclerosis Panel 1 Albumin IgG, Oligoclonal Bands, IgG Synthesis Rate/Index	3 to 5 days	3 mL CSF and 2 mL serum (1.5 mL CSF and 1 mL serum minimum)	82040, 82042, 82784 (×2), 83916
7085	Multiple Sclerosis Panel 2 Albumin IgG, IgG Synthesis Rate/Index Myelin Basic Protein,Oligoclonal Bands	4 to 6 days	4 mL CSF and 3 mL serum (2 mL CSF and 1.5 mL serum minimum)	82040, 82042, 82784 (×2), 83873, 83916

Reflex tests are performed at an additional charge.
Components of panels can be ordered separately.
Test codes valid through December 31, 2021 (subject to change).

Table 5 Test ordering information

Test code	Test name
AChR ganglionic	
93881	Acetylcholine Receptor Ganglionic (Alpha 3) Antibody
Amphiphysin	
4674	Recombx® Amphiphysin Antibody
ANNA1 (Hu)	
37053	Hu Antibody Screen with Reflex to Titer and Western Blot Includes ANNA1 (Hu) antibody (IFA) with reflex to WB with reflex to titer
37710	Hu Antibody Screen with Reflex to Titer and Western Blot, CSF Includes ANNA1 (Hu) antibody (IFA) with reflex to WB with reflex to titer
ANNA2 (Ri)	
10140	Ri Antibody Screen with Reflex to Titer and Western Blot Includes ANNA2 (Ri) antibody (IFA) with reflex to WB with reflex to titer
90121	Ri Antibody Screen with Reflex to Titer and Western Blot, CSF Includes ANNA2 (Ri) antibody (IFA) with reflex to WB with reflex to titer
CASPR2	
92413	CASPR2 Antibody
CRMP5/CV2	
4681	Recombx® CV2 Autoantibody
DPPX	
93891	DPPX Receptor Antibody, CBA IFA
gACHR	
93881	Acetylcholine Receptor Ganglionic (Alpha 3) Antibody
GAD65	
92414	GAD65 Neurological Syndrome Antibody
LGI1	
92416	LGI1 Antibody
Ma2/Ta	
4683	Recombx® MaTa Autoantibody
Myelin and MAG	
4639	Myelin Antibody (IgG), IFA
37438	Myelin Associated Glycoprotein (MAG) Antibody (IgM), EIA
37078	Myelin Associated Glycoprotein (MAG)-SGPG Antibody (IgM)
10063	Myelin Associated Glycoprotein (MAG) Antibody, with Reflex to MAG-SGPG and MAG, EIA Includes MAG antibody (WB) with reflex to MAG-SGPG antibody and MAG antibody (EIA)
NMDAR1	
92394	NMDA Receptor (NR1-subunit) Autoantibody
PCA1 (Yo)	
90119	Yo Antibody Screen with Reflex to Titer and Western Blot Includes PCA1 (Yo) antibody with reflex to WB with reflex to titer
90117	Yo Antibody Screen with Reflex to Titer and Western Blot, CSF Includes PCA1 (Yo) antibody with reflex to WB with reflex to titer
PCA-Tr	
93894	Purkinje Cell Cytoplasmic Antibody Type Tr (DNER), CBA IFA
Recoverin	
4684	Recombx® CAR (Anti-Recoverin) Autoantibody
Titin	
92792	Titin Autoantibody
VGCC (N-type)	
93882	Voltage-Gated Calcium Channel (VGCC) Type N Antibody
VGCC (P/Q-type)	
34057	Voltage-Gated Calcium Channel (VGCC) Type P/Q Antibody
VGKC	
93883	Voltage-Gated Potassium Channel (VGKC) Antibody
Zic4	
4689	Recombx® Zic4 Antibody

Test codes valid through December 31, 2021 (subject to change).

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References

1. Vincent A, Bien CG, Irani SR, Waters P. Autoantibodies associated with diseases of the CNS: new developments and future challenges. *Lancet Neurol*. 2011;10(8):759-772. doi:10.1016/S1474-4422(11)70096-5
2. Stocker W, Probst C, Teegen B, et al. Stocker W, Probst C, Teegen B, et al. Multiparameter autoantibody screening in the diagnosis of neurological autoimmune diseases. Paper presented at: 2nd International Conference on Antibodies and Therapeutics; July 11-12, 2016; Philadelphia, PA. *Immunome Res*. 2016;12 (2)(Suppl). https://www.longdom.org/conference-abstracts-files/1745-7580.C1.005-009.pdf
3. Ariño H, Höftberger R, Gresa-Arribas N, et al. Paraneoplastic neurological syndromes and glutamic acid decarboxylase antibodies. *JAMA Neurol*. 2015;72(8):874-881. doi:10.1001/jamaneurol.2015.0749
4. Dalmau J, Gleichman AJ, Hughes EG, et al. Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies. *Lancet Neurol*. 2008;7(12):1091-1098. doi:10.1016/S1474-4422(08)70224-2
5. Graus F, Vincent A, Pozo-Rosich P, et al. Anti-glial nuclear antibody: marker of lung cancer-related paraneoplastic neurological syndromes. *J Neuroimmunol*. 2005;165(1-2):166-171. doi:10.1016/j.jneuroim.2005.03.020
6. Höftberger R, van Sonderen A, Leypoldt F, et al. Encephalitis and AMPA receptor antibodies. Novel findings in a case series of 22 patients. *Neurology*. 2015;84(24):2403-2412. doi:10.1212/WNL.0000000000001682
7. McKeon A, Lennon VA, Lachance DH, Fealey RD, Pittock SJ. Ganglionic acetylcholine receptor autoantibody. *Arch Neurol*. 2009;66(6):735-741. doi:10.1001/archneurol.2009.78
8. Pittock SJ, Lennon VA. Aquaporin-4 autoantibodies in a paraneoplastic context. *Arch Neurol*. 2008;65(5):629-632. doi:10.1001/archneur.65.5.629
9. Pittock SJ, Lucchinetti CF, Lennon VA. Anti-neuronal nuclear autoantibody type 2: paraneoplastic accompaniments. *Ann Neurol*. 2003;53(5):580-587. doi:10.1002/ana.10518
10. Pittock SJ, Lucchinetti CF, Parisi JE, et al. Amphiphysin autoimmunity: paraneoplastic accompaniments. *Ann Neurol*. 2005;58(1):96-107. doi:10.1002/ana.20529
11. Romi F, Skeie GO, Aarli JA, Gilhus NE. Muscle autoantibodies in subgroups of myasthenia gravis patients. *J Neurol*. 2000;247:369-375. doi: 0.1007/s004150050604
12. Titulaer MJ, Soffietti R, Dalmau J, et al. Screening for tumours in paraneoplastic syndromes: report of an EFNS Task Force. *Eur J Neurol*. 2011;18(1):19-e13. doi:10.1111/j.1468-1331.2010.03220.x
13. Yu Z, Kryzer TJ, Griesmann GE, Kim KK, Benarroch EE, Lennon VA. CRMP-5 neuronal autoantibody: marker of lung cancer and thymoma-related autoimmunity. *Ann Neurol*. 2001;49(2):146-154. doi:10.1002/1531-8249(20010201)49:2<146::AID-ANA34>3.0.CO;2-E
14. Chan KH, Vernino S, Lennon VA. ANNA-3 anti-neuronal nuclear antibody: marker of lung cancer-related autoimmunity. *Ann Neurol*. 2001;50(3):301-311. doi:10.1002/ana.1127
15. Irani SR, Alexander S, Waters P, et al. Antibodies to Kv1 potassium channel-complex proteins leucine-rich, glioma inactivated 1 protein and contactin-associated protein-2 in limbic encephalitis, Morvan's syndrome and acquired neuromyotonia. *Brain*. 2010;133(9):2734-2748. doi:10.1093/brain/awq213
16. National Organization for Rare Disorders. Paraneoplastic neurologic syndromes. Updated 2016. Accessed October 20, 2021. https://rarediseases.org/rare-diseases/paraneoplastic-neurologic-syndromes/
17. Abelhosn RW, Montana L, Rivera JG, et al. Tissue immunofluorescence confirmation of CNS autoantibodies identified by immunoblot or cell-based assay. Epub December 15, 2020. Preprint at *J Neurosci Neuropsych*. 2021; volume 4. https://www.researchsquare.com/article/rs-126755/v1. doi:10.21203/rs.3.rs-126755/v1
18. Kelley BP, Patel SC, Marin HL, Corrigan JJ, Mitsias PD, Griffith B. Autoimmune encephalitis: pathophysiology and imaging review of an overlooked diagnosis. *AJNR Am J Neuroradiol*. 2017:38(6):1070-1078. doi:10.3174/ajnr.A5086
19. Titulaer MJ, McCracken L, Gabilondo I, et al. Treatment and prognostic factors for long-term outcome in patients with anti-N-methyl-D-asparate (NMDA) receptor encephalitis: a cohort study. *Lancet Neurol*. 2013;12(2):157-165. doi:10.1016/s1474-4422(12)70310-1
20. Kitley J, Waters P, Woodhall M, et al. Neuromyelitis optica spectrum disorders with aquaporin-4 and myelin-oligodendrocyte glycoprotein antibodies: a comparative study. *JAMA Neurol*. 2014;71(3):276-283. doi:10.1001/jamaneurol.2013.5857
21. Kim SM, Kim SJ, Lee HJ, Kuroda H, Palace J, Fujihara K. Differential diagnosis of neuromyelitis optical spectrum disorders. *Ther Adv Neurol Disord*. 2017;10(7):265-289. doi:10.1177/1756285617709723
22. Sato DK, Callegaro D, Lana-Peixoto MA, et al. Distinction between MOG antibody-positive and AQP4 antibody-positive NMO spectrum disorders. *Neurology*. 2014;82(6):474-481. doi:10.1212/WNL.0000000000000101
23. Wingerchuck DM, Banwell B, Bennett JL, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurology*. 2015;85(2):177-189. doi:10.1212/WNL.0000000000001729
24. Pröbstel AK, Rudolf G, Dornmair K, et al. Anti-MOG antibodies are present in a subgroup of patients with a neuromyelitis optica phenotype. *J Neuroinflammation*. 2015;12:46. doi:10.1186/s12974-015-0256-1
25. Kezuka T, Usui Y, Yamakawa N, et al. Relationship between NMO-antibody and anti-MOG antibody in optic neuritis. *J Neuroophthalmol*. 2012;32(2):107-110. doi:10.1097/WNO.0b013e31823c9b6c
26. Waters P, Woodhall M, O'Connor KC, et al. MOG cell-based assay detects non-MS patients with inflammatory neurologic disease. *Neurol Neuroimmunol Neuroinflamm*. 2015;2(3):e89. doi:10.1212/NXI.0000000000000089
27. Haven TR, Astill ME, Pasi BM, et al. An algorithm for acetylcholine receptor antibody testing in patients with suspected myasthenia gravis. *Clin Chem*. 2010;56(6):1028-1029. doi:10.1373/clinchem.2009.140392
28. Rivner MH, Pasnoor M, Dimachkie MM, et al. Muscle-specific tyrosine kinase and myasthenia gravis owing to other antibodies. *Neurol Clin*. 2018;36(2):293-310. doi:10.1016/j.ncl.2018.01.004
29. Jowkar A, Goldenberg WD, Shah AK, et al. Myasthenia gravis workup. *Medscape*. Updated March 23, 2016. Accessed August 27, 2020. http://emedicine.medscape.com/article/1171206-workup
30. Huijbers MG, Zhang W, Klooster R, et al. MuSK IgG4 autoantibodies cause myasthenia gravis by inhibiting binding between MuSK and Lrp4. *Proc Natl Acad Sci US*. 2013;110(51):20783-20788. doi.org/10.1073/pnas.1313944110
31. Phillips WD, Vincent A. Pathogenesis of myasthenia gravis: update on disease types, models, and mechanisms. *F1000Res*. 2016;5(F1000 Faculty Rev):1513. doi:10.12688/f1000research.8206.1
32. Titin antibodies. Antibodies.com. Accessed August 27, 2020. https://www.antibodies.com/products/primary-antibodies/target=Titin
33. Zouvelou V, Zisimopoulou P, Rentzos M, et al. Double seronegative myasthenia gravis with anti-LRP4 antibodies. *Neuromuscul Disord*. 2013;23(7):568-570. doi:10.1016/j.nmd.2013.03.013
34. Rivner MH, Quarles BM, Pan JX, et al. Clinical features of LRP4/agrin-antibody-positive myasthenia gravis: a multicenter study. *Muscle Nerve*. 2020;62(3):333-343. doi:10.1002/mus.26985
35. Data on file. Athena Diagnostics, Inc; 2021.

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