

Volume 1, May 2023

The following case studies demonstrate the clinical utility of the Cunningham Panel™ as an aid in identifying an underlying autoimmune etiology in patients with certain neuropsychiatric symptoms. Test results can support a clinician's diagnosis with laboratory evidence.

Patient Symptomatic

Patient symptoms

Initial treatment consistent with elevated autoantibodies to ganglioside GM1 (tics, dysgraphia), elevated anti-albumin autoantibodies (OCD, poor concentration), and dopamine D1 autoantibodies (anxiety, sleep problems, emotional lability, depression, regression).



Case Study #1 - Autoimmune-induced neuropsychiatric symptoms in an 8-year-old female

Antibiotic treatment leads to resolution of multiple neurologic and psychiatric symptoms

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Case Study #2 - Gradual onset of tics and severe cognitive regression in 20-year-old male

Initially diagnosed with bipolar disorder, testing reveals immune-mediated condition

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Case Study #3 - Severe OCD and tics improve with immune-modulatory treatment

Clinical and laboratory results support autoimmune-based illness

[READ](#)

Case Study #4 - 20-year-old female with OCD improves with rituximab treatment

Testing revealed elevated autoantibodies against Dopamine D2L receptor

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Case Study #5 - Multiple neuropsychiatric symptoms improve with immune-modulatory treatment

Reduction in symptoms following treatment with anti-inflammatory drugs and IVIg therapy

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The Cunningham Panel™ - An Overview

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CASE STUDY #1

Autoimmune-induced neuropsychiatric symptoms in an 8-year-old female

Antibiotic treatment leads to resolution of multiple neurologic and psychiatric symptoms

Presentation: 8-year-old girl with abrupt onset of obsessive-compulsive behaviors, vocal tics, poor concentration, sensory/motor abnormalities, emotional lability, regression, aggression, and dysgraphia. Parents report urinary and sleep problems.

Course of Illness: Relapsing and remitting symptoms with improvement after 2-3 days of azithromycin.

Previous Diagnoses: Anxiety, OCD, unspecified disorder of immune mechanism

Previous Lab Results: ASO <50, Anti-DNase B <50, normal IgG titers

Medical History: Significant for two recent streptococcal infections with high fever, sore throat, sinus pain and cough. History of frequent ear infections, sinus infections and idiopathic fevers.

Family History: Positive for mycoplasma infections, autoimmune disorders, allergies, frequent bacterial and viral infections.

Pre-Treatment Cunningham Panel™ Results: Elevated anti-Lysoganglioside GM1 autoantibodies 640 (normal range 80-320), elevated anti-Tubulin autoantibodies 2,000 (normal range 250-1,000), and borderline anti-Dopamine Receptor D1 autoantibodies 2,000 (normal range 500-2,000)

Treatment: Azithromycin with rapid symptom improvement.

Post-Treatment Cunningham Panel™ Results: Anti-Lysoganglioside GM1 returned to normal (147); anti-Tubulin returned to normal (250) and anti-Dopamine Receptor D1 autoantibodies returned to normal (500)

Status: All symptoms resolved. Returned to baseline after 2-3 days of treatment.



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Cunningham Panel™

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Borderline 2000
Anti-Dopamine D2L Receptor Autoantibodies	Normal 2000
Anti-Lysoganglioside GM1 Autoantibodies	Elevated 640
Anti-Tubulin Autoantibodies	Elevated 2000
CaMKinase II	Normal 95

POST-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Normal 500
Anti-Dopamine D2L Receptor Autoantibodies	Normal 2000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 40
Anti-Tubulin Autoantibodies	Normal 250
CaMKinase II	Normal 112

SUMMARY

An 8-year-old girl with a history of strep, sinus and ear infections presented with numerous neuropsychiatric symptoms exhibited clinical and laboratory response to antibiotics.



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Cunningham Panel™ test results

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

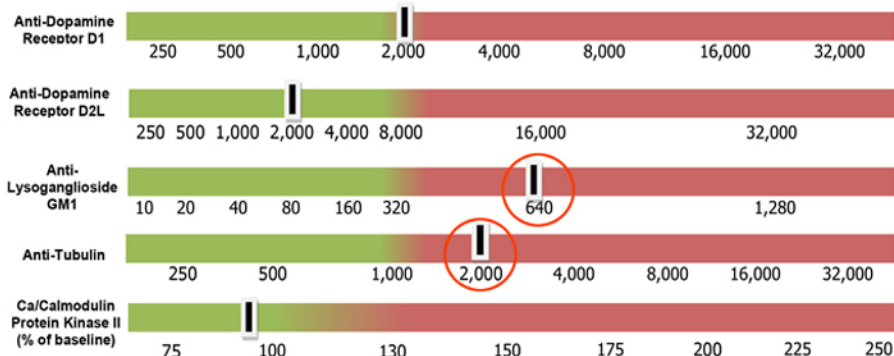
PRE-TREATMENT: Patient Symptomatic

Patient symptoms pre-treatment consistent with elevated autoantibodies to Lysoganglioside GM1 (tics, dysgraphia), elevated anti-Tubulin autoantibodies 2000 (OCD, poor concentration), and borderline Dopamine D1 autoantibodies (anxiety, sleep problems, emotional lability, aggression, regression).

LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:2,000	1:2,000	1:640	1:2,000	95
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	BORDERLINE	NORMAL	ELEVATED	ELEVATED	NORMAL

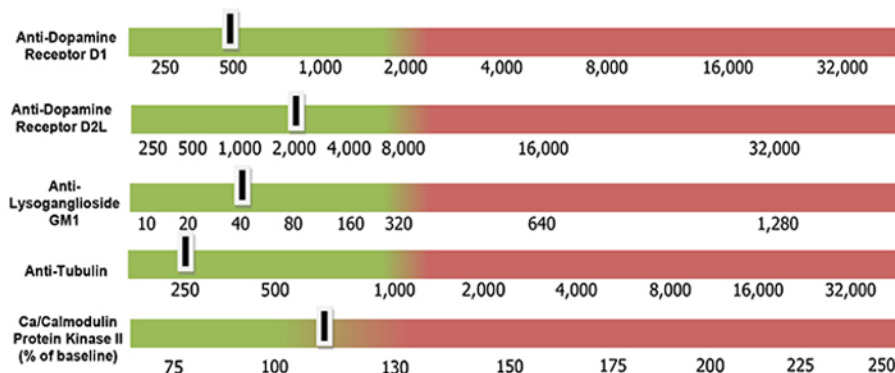
LABORATORY TEST RESULTS



LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:500	1:2,000	1:40	1:250	112
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	NORMAL	NORMAL	NORMAL	NORMAL	ELEVATED

LABORATORY TEST RESULTS



POST-TREATMENT: Symptom Resolution

Symptom resolution complete.
All Cunningham Panel™ markers within normal limits.



CASE STUDY #2

Gradual onset of tics and severe cognitive regression in 20-year-old male

Initially diagnosed with bipolar disorder, testing reveals immune-mediated condition

Presentation: 20-year-old male with gradual onset of tics, inability to concentrate, sensory abnormalities, emotional lability, severe cognitive regression, separation anxiety, developmental regression, sleep disturbances, handwriting disturbance and aggressiveness.

Previous Diagnoses: Unspecified bipolar disorders

Previous Lab Results: Positive for Lyme disease, Babesia, and Rickettsia

Medical History: Rocky Mountain Spotted Fever, multiple ear and strep infections throughout childhood, known immunodeficiency.

Family History: Positive for rheumatoid arthritis, asthma and eczema.

Pre-Treatment Cunningham Panel™ Results: Elevated anti-Dopamine Receptor D1 autoantibodies - 4,000 (normal range 500-2,000), borderline anti-Dopamine Receptor D2L - 8,000 (normal range 2,000-8,000), elevated anti-Tubulin autoantibodies - 2,000 (normal range 250-1,000), elevated Calcium/calmodulin-dependent protein kinase II (CaMKII) - 140 (normal range 53-130)

Treatment: Plasmapheresis followed by multiple IVIg treatments.

Post-Treatment Cunningham Panel™ Results: Anti-Dopamine Receptor D1 autoantibodies decreased to borderline (2,000), anti-Dopamine Receptor D2L autoantibodies remained at borderline (8,000), anti-Tubulin autoantibodies decreased to borderline (1,000), Calcium/calmodulin-dependent protein kinase II (CaMKII) decreased to borderline (124)

Status: Overall cognitive abilities improved, with mother reporting patient’s math skills returned after many years.



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Cunningham Panel™
An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Elevated 4000
Anti-Dopamine D2L Receptor Autoantibodies	Borderline 8000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 160
Anti-Tubulin Autoantibodies	Elevated 2000
CaMKinase II	Elevated 140

POST-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Borderline 2000
Anti-Dopamine D2L Receptor Autoantibodies	Borderline 8000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 160
Anti-Tubulin Autoantibodies	Borderline 1000
CaMKinase II	Borderline 124

SUMMARY

A 20-year-old male with gradual onset of tics, severe cognitive regression and multiple neuropsychiatric symptoms, who tested positive for multiple tick-borne illnesses, had elevated anti-Dopamine Receptor D1 and anti-Tubulin autoantibodies, along with an elevated cell stimulation assay (CaMKII) suggesting an infection-triggered autoimmune-based etiology.



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Cunningham Panel™ test results

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT:

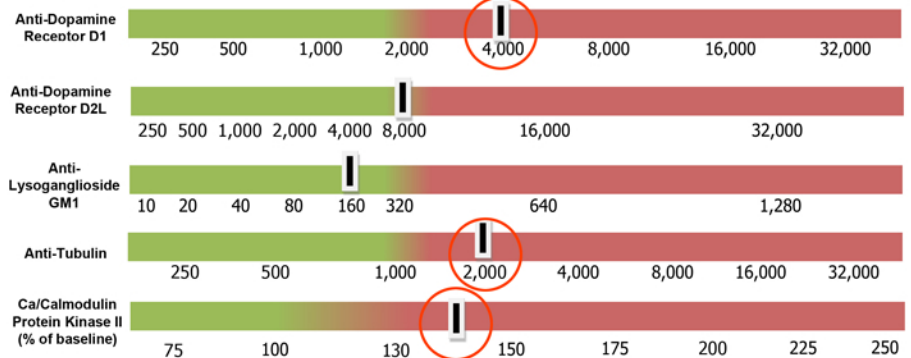
Patient Symptomatic

Elevated Dopamine Receptor D1 autoantibodies (4,000) consistent with emotional lability, anxiety, regression, sleep problems and aggressiveness; Tubulin autoantibodies (2,000) consistent with poor concentration and cognitive regression. Calcium/calmodulin-dependent protein kinase II (CaMKII) at 140 correlates with tics, sensory abnormalities, and dysgraphia.

LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:4,000	1:8,000	1:160	1:2,000	140
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	ELEVATED	BORDERLINE	NORMAL	ELEVATED	ELEVATED

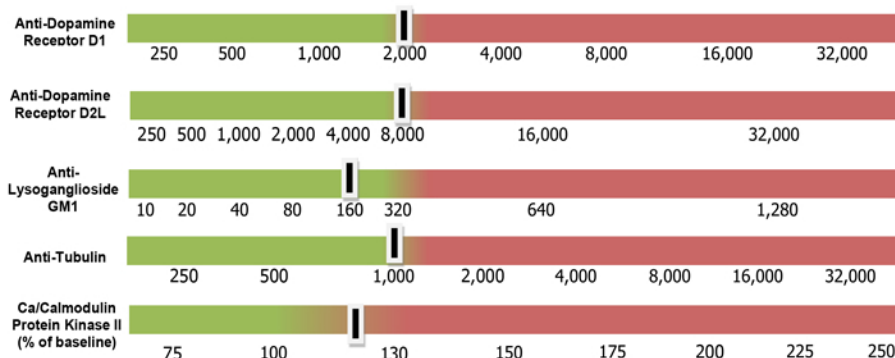
LABORATORY TEST RESULTS



LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:2,000	1:8,000	1:160	1:1,000	124
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	BORDERLINE	BORDERLINE	NORMAL	BORDERLINE	BORDERLINE

LABORATORY TEST RESULTS



POST-TREATMENT:

Symptom Resolution

Borderline Dopamine Receptor D1 autoantibodies (2,000), Dopamine Receptor D2L autoantibodies (8,000), Tubulin autoantibodies (1,000) and CaMKII (124) consistent with resolution of symptoms.

CASE STUDY #3

Severe OCD and tics improve with immune-modulatory treatment

Clinical and laboratory results support autoimmune-based illness

Presentation: 16-year-old female with abrupt onset at age 11 of severe OCD (food restriction), tics and choreiform movements, poor concentration, significant cognitive and academic decline, sensory abnormalities, severe mood dysregulation, urinary urgency and frequency, and dysgraphia. Aggressive behaviors prompted 6 calls to local police for assistance.

Course of Illness: Relapsing and remitting symptoms with three major episodes reported. Sudden increase in symptoms with strep exposure.

Previous Diagnoses: Autoimmune disease NOS, post-infectious encephalitis and encephalomyelitis, other diseases involving the immune system NOS

Previous Lab Results: Positive Lyme Western blot, elevated Mycoplasma and Coxsackie titers

Medical History: Non-contributory

Family History: Maternal and paternal grandmothers with autoimmune disorders, history of rheumatic fever. Sibling with Pediatric Autoimmune Neuropsychiatric Disorders Associated with Strep infection (PANDAS).

Pre-Treatment Cunningham Panel™ Results: Elevated Calcium/calmodulin-dependent protein kinase II (CaMKII) 167 (normal range 53-130)

Treatment: Plasmapheresis and IVIg with symptom improvement, low dose Zoloft, Abilify for maintenance.

Post-Treatment Cunningham Panel™ Results: Calcium / calmodulin-dependent protein kinase II (CaMKII) returned to borderline (123)

Status: Symptom improvement.



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Cunningham Panel™

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Borderline 2000
Anti-Dopamine D2L Receptor Autoantibodies	Normal 4000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 40
Anti-Tubulin Autoantibodies	Borderline 1000
CaMKinase II	Elevated 167

POST-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Normal 1000
Anti-Dopamine D2L Receptor Autoantibodies	Normal 4000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 80
Anti-Tubulin Autoantibodies	Borderline 1000
CaMKinase II	Borderline 123

SUMMARY

A 16-year-old female with severe food restriction, OCD, tics, choreiform movements, poor concentration, cognitive and academic decline, sensory abnormalities, mood dysregulation, urinary urgency and frequency, and dysgraphia exhibited clinical and laboratory response to plasmapheresis and IVIg.



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Cunningham Panel™ test results

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT:

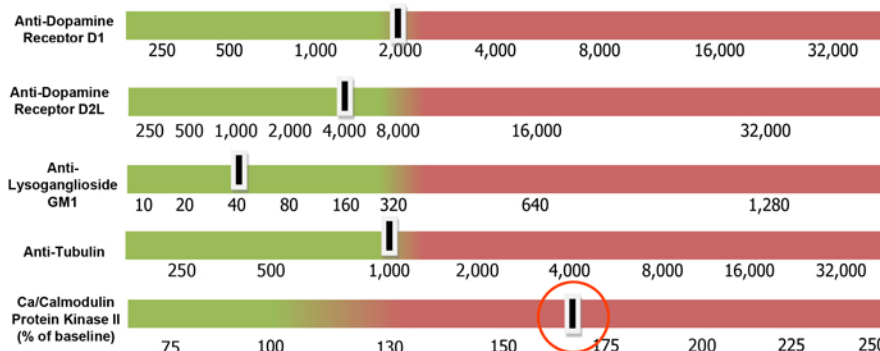
Patient Symptomatic

Patient symptoms likely due to CaMKII activation and subsequent increase in dopamine, epinephrine, and norepinephrine neurotransmitters resulting in adrenergic stimulation. Elevation in CaMKII associated with likely active infection.

LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:2,000	1:4,000	1:40	1:1,000	167
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	BORDERLINE	NORMAL	NORMAL	BORDERLINE	ELEVATED

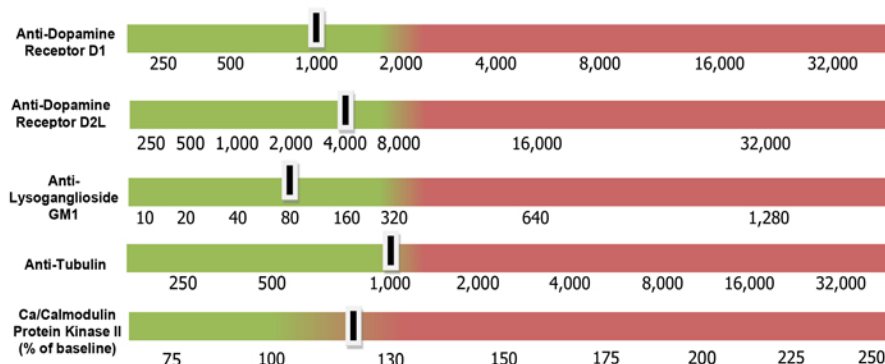
LABORATORY TEST RESULTS



LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:1,000	1:4,000	1:80	1:1,000	123
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	NORMAL	NORMAL	NORMAL	BORDERLINE	BORDERLINE

LABORATORY TEST RESULTS



POST-TREATMENT: Symptom Resolution

Symptom resolution after multiple plasma exchanges to mechanically remove antibodies followed by IVIg to replenish healthy antibodies.



CASE STUDY #4

20-year-old female with OCD improves with rituximab treatment

Testing revealed elevated autoantibodies against Dopamine D2L receptor

Presentation: 20-year-old female with gradual onset of OCD, poor concentration, sensory and motor abnormalities, emotional lability, separation anxiety, developmental regression, urinary frequency and urgency, sleep disturbances, and aggression.

Course of Illness: Symptoms relapsing and remitting with very frequent flares.

Previous Diagnoses: Idiopathic peripheral neuropathy, reflex sympathetic dystrophy, variants of migraine, Lyme disease, disorder of autonomic nervous system

Previous Lab Results: Positive for strep, Lyme disease, Babesia, Mycoplasma, Epstein-Barr virus, Herpes Zoster virus

Medical History: Frequent strep infections since childhood, low quantitative immunoglobulins, and dental infections.

Family History: Positive for autoimmune disease and allergies.

Pre-Treatment Cunningham Panel™ Results: Elevated anti-dopamine receptor D2L autoantibodies 16,000 (normal range 2,000-8,000), elevated Calcium/calmodulin-dependent protein kinase II (CaMKII) 144 (normal range 53-130)

Treatment: Rituximab (4 courses) and ozone treatments with significant symptom reduction.

Post-Treatment Cunningham Panel™ Results: Anti-dopamine receptor D2L autoantibodies returned to normal (500), Calcium / calmodulin-dependent protein kinase II (CaMKII) levels returned to normal (111)

Status: Significant reduction in symptoms.



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Cunningham Panel™

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Normal 1000
Anti-Dopamine D2L Receptor Autoantibodies	Elevated 16000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 80
Anti-Tubulin Autoantibodies	Normal 500
CaMKinase II	Elevated 144

POST-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Normal 1000
Anti-Dopamine D2L Receptor Autoantibodies	Normal 500
Anti-Lysoganglioside GM1 Autoantibodies	Normal 40
Anti-Tubulin Autoantibodies	Normal 250
CaMKinase II	Normal 111

SUMMARY

A 20-year-old female presented with gradual onset OCD and multiple neuropsychiatric symptoms. Testing revealed elevated autoantibodies directed against the Dopamine D2L receptor and elevated CaMKII, suggesting an autoimmune component. Elimination of memory B cells with rituximab resulted in a significant reduction in symptoms.



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Cunningham Panel™ test results

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT:

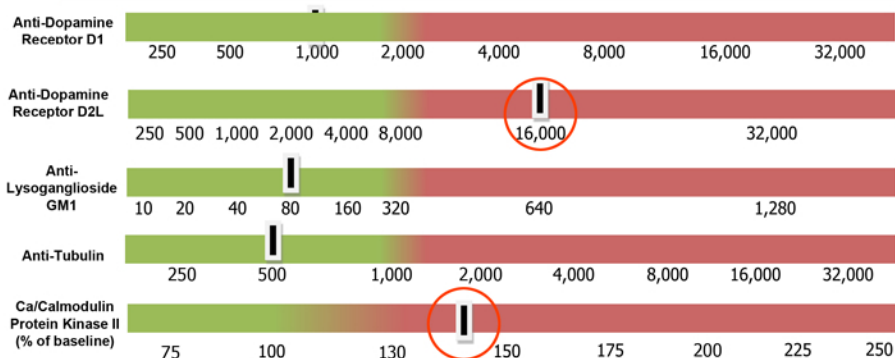
Patient Symptomatic

Patient symptomatic likely due to autoantibodies directed against D2L receptors resulting in motor abnormalities and increased CaMKII activation associated with OCD, poor concentration, emotional lability, separation anxiety, developmental regression, urinary issues, sleep disturbances, and aggression.

LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:1,000	1:16,000	1:80	1:500	144
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	NORMAL	ELEVATED	NORMAL	NORMAL	ELEVATED

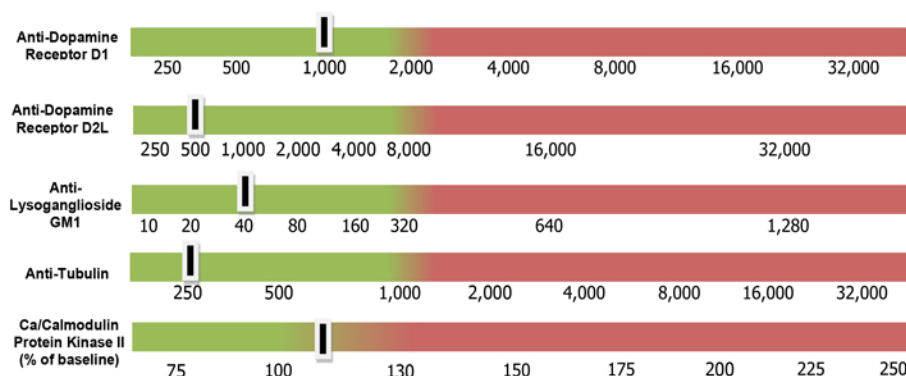
LABORATORY TEST RESULTS



LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:1,000	1:500	1:40	1:250	111
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL

LABORATORY TEST RESULTS



POST-TREATMENT:

Symptom Resolution

Symptom resolution after four courses of Rituxan (rituximab), a chemotherapeutic monoclonal antibody targeting and eliminating memory B cells.



CASE STUDY #5

Multiple neuropsychiatric symptoms improve with immune-modulatory treatment

Reduction in symptoms following treatment with anti-inflammatory drugs and IVIg therapy

Presentation: 17-year-old male with abrupt onset of OCD, inability to concentrate, sensory and motor abnormalities, emotional lability, separation anxiety, developmental regression, sleep disturbances, handwriting disturbance, aggressiveness, and choreiform movements.

Course of Illness: Relapsing and remitting symptoms.

Previous Diagnoses: Panic disorder, emotional disorders with onset specific to childhood, selective deficiency of immunoglobulin A (IgA), personal history of traumatic brain injury, other encephalitis and encephalomyelitis

Previous Lab Results: High Coxsackie, Parvovirus and HHV6 titers, hypogammaglobinemia, MARCONS positive

Medical History: Chronic sinusitis and frequent viral infections.

Family History: Positive for rheumatic fever, tics, OCD, autoimmune disorders, hyper and hypothyroidism and allergies.

Pre-Treatment Cunningham Panel™ Results: Borderline anti-Tubulin autoantibodies - 1,000 (normal range 250-1,000), and elevated Calcium/calmodulin-dependent protein kinase II (CaMKII) - 184 (normal range 53-130)

Treatment: NSAIDs and multiple IVIg infusions.

Post-Treatment Cunningham Panel™ Results: Anti-Tubulin autoantibodies remained borderline (1,000) and Calcium / calmodulin-dependent protein kinase II (CaMKII) returned to normal (113)

Status: Symptom improvement with anti-inflammatory drugs and IVIG treatment.



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Cunningham Panel™

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

PRE-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Normal 500
Anti-Dopamine D2L Receptor Autoantibodies	Normal 4000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 80
Anti-Tubulin Autoantibodies	Borderline 1000
CaMKinase II	Elevated 184

POST-TREATMENT TEST RESULTS

Anti-Dopamine D1 Receptor Autoantibodies	Borderline 2000
Anti-Dopamine D2L Receptor Autoantibodies	Normal 2000
Anti-Lysoganglioside GM1 Autoantibodies	Normal 40
Anti-Tubulin Autoantibodies	Borderline 1000
CaMKinase II	Normal 113

SUMMARY

A 17-year-old male with abrupt onset OCD and other neuropsychiatric symptoms and a history of chronic sinusitis and viral infections experienced symptom improvement following treatment with anti-inflammatory drugs and IVIg therapy.



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Cunningham Panel™ test results

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

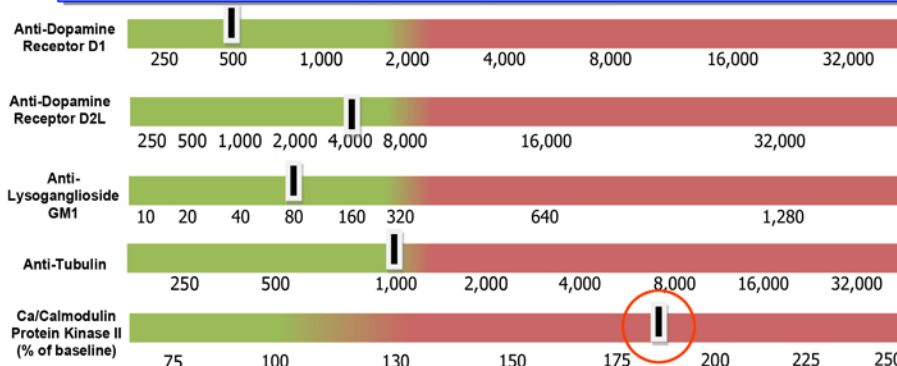
PRE-TREATMENT: Patient Symptomatic

Elevated Calcium/calmodulin-dependent protein kinase II (CaMKII) 184 consistent with sensory and motor abnormalities, emotional lability, separation anxiety, regression, sleep disturbances, dysgraphia, aggressiveness, and choreiform movements. Borderline anti-Tubulin autoantibodies 1,000 consistent with obsessive-compulsive symptoms and poor concentration.

LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:500	1:4,000	1:80	1:1,000	184
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	NORMAL	NORMAL	NORMAL	BORDERLINE	ELEVATED

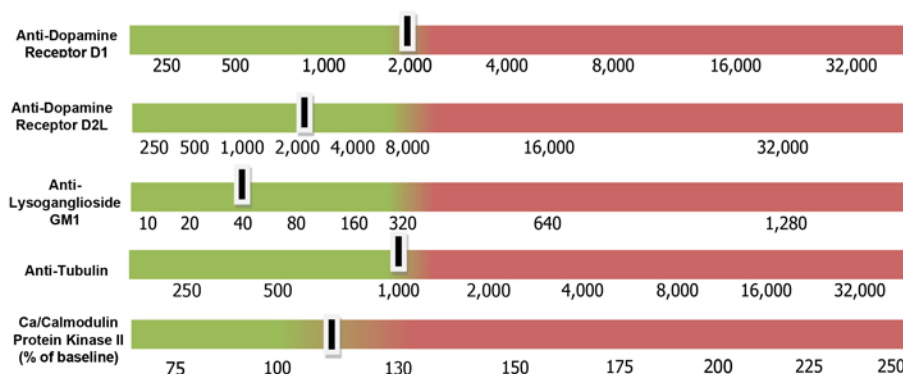
LABORATORY TEST RESULTS



LABORATORY TEST RESULTS COMPARED TO NORMAL RANGES

	Anti-Dopamine Receptor D1 (titer)	Anti-Dopamine Receptor D2L (titer)	Anti-Lysoganglioside GM1 (titer)	Anti-Tubulin (titer)	CaM Kinase II (% of baseline)
Patient Result	1:2,000	1:2,000	1:40	1:1,000	113
Normal Ranges	500 to 2,000	2,000 to 8,000	80 to 320	250 to 1,000	53-130
Normal Mean	1,056	6,000	147	609	95
INTERPRETATION*	BORDERLINE	NORMAL	NORMAL	BORDERLINE	NORMAL

LABORATORY TEST RESULTS



POST-TREATMENT: Symptom Resolution

Normal CaMKII (113) along with borderline Dopamine 1 autoantibodies (2,000) and anti-Tubulin autoantibodies (1,000) consistent with resolution of symptoms.



The Cunningham Panel™

An Autoimmune Encephalopathy & Basal Ganglia Encephalitis Panel

The Cunningham Panel™ comprises a series of high complexity blood tests that assists clinicians in identifying an underlying autoimmune etiology for various neurologic (i.e., involuntary movements, seizure-like episodes, tics) and/or psychiatric (i.e., OCD, severe anxiety, mood swings) manifestations.

The Panel measures elevations in autoantibodies directed against specific targets (Dopamine D1 & D2 receptors, Lysoganglioside, Tubulin) in the basal ganglia region of the brain, which may indicate an autoimmune basis for the patient's symptoms. A cell stimulation assay (CaMKinase II) measures the ability of these autoantibodies to stimulate activation of the CaMKII enzyme which can also indicate the likelihood of the presence of an active infection or re-occurring/reactivated infection.

Elevated levels on one or more of these tests indicate that a person's neuropsychiatric symptoms may be due to a treatable autoimmune disorder (potentially triggered by an infection(s), rather than a classic neurologic or psychiatric illness.

Test results can support a clinician's diagnosis with laboratory evidence of an underlying autoimmune etiology and assist clinicians in selecting an appropriate treatment regime. Studies have found that with proper treatment, a patient's symptoms can be greatly reduced or completely resolved.

Elevated levels on one or more of these tests indicate that neurologic and/or psychiatric symptoms may be due to a treatable autoimmune disorder.

Ordering the Cunningham Panel™

Moléculera Labs can accept authorized clinician test orders from all 50 states in the U.S., as well as other countries. (Note: New York patients must have their blood collected outside the State of New York.) ***The Panel can only be ordered by licensed clinicians.*** Click below to learn how to order the Cunningham Panel

Ordering The Panel

VISIT US AT [MOLECULERALABS.COM](https://moleculeralabs.com)

P. 405-239-5250

E. customerservice@moleculera.com

