



THE ONLY PROVEN OBJECTIVE PATHWAY FOR DIAGNOSIS AND MANAGEMENT OF TRAUMATIC BRAIN INJURY (TBI)

A Fully-Integrated Network for TBI Diagnostics, Education, & Care



BioConnetiX TBI Biomarker Diagnostic Pathway - for Personal Injury Case Management

Replacing Guesswork with Science: Precision Biomarkers for Every Phase of TBI

By measuring brain-derived protein markers and genetic risk indicators, biomarker testing delivers objective, definitive confirmation of TBI and highly specific, individualized risk stratification tied to precise clinical interventions. Tests available through our network are validated to detect and track TBI across all phases – acute (<72 hours), post-acute (up to 6 months), and chronic/long-term (up to 5 years).



p-tau216 (*phosphorylated tau protein*)

Marker of disrupted tau processing after TBI; elevations reflect neuronal stress, emerging tangle pathology, and risk for ongoing or delayed neurocognitive impairment.



GFAP (*glial fibrillary acidic protein*)

Released during brain's active response to injury; distinguishes recent damage & helps confirm whether persistent elevations in other markers stem from the same event.



UCH-L1 (*ubiquitin carboxyl-terminal hydrolase L1*)

Protein released from brain neurons soon after injury; detects even mild TBI that imaging may miss; levels correlate with injury severity and functional outcome.



NF-H, NF-L (*neurofilament heavy or light chain*)

"Slow-burn" neuronal protein that peaks in post-acute phase; strong indicator of recovery trajectory and treatment efficacy when tracked over time



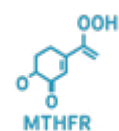
S100B (*S100 calcium-binding protein beta*)

Astrocyte-derived protein that rises as the brain initiates repair; degree of elevation indicates severity; persistence over time may signal chronic neurological damage.



ApoE (*apolipoprotein E*)

Lipid transport protein influencing neural repair. E4 genotype is associated with delayed recovery after TBI & elevated risk of neurodegeneration, including Alzheimer's.



MTHFR (*methylenetetrahydrofolate reductase*)

Regulates folate metabolism and homocysteine conversion. C677T/A1298C variants can disrupt neuronal repair & increase risk for vascular or cognitive complications.

TBI Biomarkers PROVEN ACCURACY

>90% CLINICAL SENSITIVITY >95% SPECIFICITY

Ideal First Test Timing: 30 days - 12 weeks post-injury

Defensible Biomarker Evidence Efficient Case Management

BioConnetiX brings deep expertise in molecular diagnostics and multidisciplinary care coordination for complex, analytic-heavy medicine – transforming a fragmented medical landscape into a streamlined, attorney-friendly pathway that removes friction & accelerates case progress.

We connect your clients with neurologists specialized in TBI care, coordinate biomarker testing through a highly qualified laboratory network, and pair every test result with an independent Molecular Pathologist or Neurointensivist Interpretive Report. Subspecialists are available for eConsults with treating providers, attorney consultations, and expert-witness support.

Through BioConnetiX, legal teams, patients, and treating providers gain one-touch access to the largest and fastest-growing network of biomarker-pathway-trained TBI specialists and resources – supported by comprehensive patient and provider education and continuously updated clinical pathways aligned with the latest science.

We deliver HIPAA-compliant care-team connections, medical-records management, clinical decision-support, and lab solutions designed to lower costs and expand access – powered by growing software capabilities to streamline case workflow, automate document exchange, and drive meaningful analytics and optimization.

Objective, Quantifiable, and Defensible: Modernizing TBI Diagnosis Across the Injury Timeline

Acute
(0 - 72 hours)

S100B, UCH-L1, GFAP

Is there acute brain injury?
Is a CT needed?

Early Sub-Acute
(4 days - 6 weeks)

**GFAP, UCH-L1, NF
(± S100B) (+ApoE, MTHFR)**

Recent damage or prior injury?
How severe?
Genetic risk factors?

Late Sub-Acute
(6 - 12 weeks)

**GFAP, NF, TAU
(± S100B, UCH-L1)**

Resolving or evolving?
How severe? QEEG/DTI?
Return-to-duty/play?

Chronic
(3 - 12 months)

**NF, TAU
(± GFAP)**

Persistent deficits?
Risk of neurocognitive decline?
Therapy effective?

Long-Term
(12 months - 5+ years)

**NF, TAU
(± GFAP)**

Therapeutic impact?
Recovery trajectory?
New injury? Secondary or delayed
neurodegeneration?