Instructions: This sample document includes a sample invention and prior art examples. Read through the listed claims and features of the sample invention and prior art, analyze the inventions, and decide which claims and features are the key components. Key components can be claims or sub-claims that differentiate the claims between invention and prior art. In a table (the sample deliverable), compare the claims and features of the invention and prior art. Based on your expertise, decide whether claims or features are directly overlapping, somewhat overlapping, or not overlapping at all.

Notes: These inventions are pulled from a publicly accessible patent list available through Google Patent. These examples have been adapted for the purposes of this exercise. We have abbreviated the claims section for the purpose of this exercise. This preliminary prior art search was performed to gather information to get you started. If you wish to conduct additional prior art research on your own, use USPTO PAIR system or google patent.

Summary of Invention:

Methods to identify markers for brain damage using fresh brain tissue and methods and compositions for detecting these markers are disclosed.

Invention: US7985555B2 (https://patents.google.com/patent/US7985555B2/en)

Claims and Features of Invention:

Claim 1. A method to detect brain damage in a test subject which method comprises analyzing a sample comprising biological fluid, or fraction thereof, of said subject for the presence of a marker which is visinin-like protein 1 (VLP-1), wherein any enhanced presence of said marker in the fluid, or fraction thereof, of the test subject as compared to normal subjects indicates an increased probability of brain damage in the test subject.

Claim 2. The method of claim1, wherein the subject is human.
Claim 3. The method of claim 1, wherein the biological fluid is CSF, serum or plasma.
Claim 4: the method of claim 3, wherein the biological fluid is serum or plasma.

Claim 5. The method of claim 1, which further includes analyzing said sample for at least one additional marker selected from the group summarized below.

Claim 6. The method of claim 5, where in the additional marker is selected from the group summarized below.

Claim 7. The method of claim 5, wherein the additional marker is selected from the second group summarized below.

Claim 8. The method of claim 5, wherein the additional marker is selected from the third group summarized below.

Group 1. synaptosomal-associated protein	Group 2.	Group 3.

(25 kDa, SNAP25), glutamate decarboxylase 1 (brain, 67 kDa, GAD67), myelin-associated oligodendrocyte basic protein, synaptotagmin 1, tubulin beta 4, fasciculation and elongation protein zeta 1 (zygin I), glycine receptor beta, vesicular membrane protein p24, olfactomedin 1, Zic family member 1 (odd-paired homolog, *Drosophila*), protein kinase C and casein kinase substrate in neurons 1, proteolipid protein 1 (Pelizaeus-Merzbacher disease, spastic paraplegia 2), internexin neuronal intermediate filament protein alpha, solute carrier family 32 (GABA vesicular transporter, member 1), serine (or cysteine) proteinase inhibitor (clade I, member 1 (neuroserpin)), Neuronatin, gamma-aminobutyric acid (GABA) A receptor (gamma 2), vesicle-associated membrane protein 2 (synaptobrevin 2), and neurogranin (protein kinase C substrate, RC3).	Zic family member 1 (odd-paired homolog, *Drosophila*), protein kinase C and casein kinase substrate in neurons 1, proteolipid protein 1 (Pelizaeus-Merzbacher disease, spastic paraplegia 2), internexin neuronal intermediate filament protein alpha, solute carrier family 32 (GABA vesicular transporter, member 1).	serine (or cysteine) proteinase inhibitor (clade I, member 1 (neuroserpin)), Neuronatin, gamma-aminobutyric acid (GABA) A receptor (gamma 2), vesicle-associated membrane protein 2 (synaptobrevin 2), and neurogranin (protein kinase C substrate, RC3).

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Prior Art A, Summary of Invention

The present invention identifies a number of gene markers whose expression is altered in multiple sclerosis (MS). These markers can be used to diagnose or predict MS in subjects, and can be used in the monitoring of therapies. In addition, these genes identify therapeutic targets, the modification of which may prevent MS development or progression.

Invention: US20040018522A1
(https://patents.google.com/patent/US20040018522A1/en?oq=US2004%2f0018522)

Claims and Features of Prior Art A:

Claim 1. A method or predicting whether a subject is or will be afflicted with multiple sclerosis (MS) comprising

a. Obtaining an mRNA-containing sample from said subject;
b. Determining expression information for one or more genes from the group summarized below.
c. Comparing expression information for said selected genes with the expression information of the same gene in a subject not afflicted with MS; and
d. Predicting whether said subject is or will be afflicted with MS.

Group 1. phosphatidylinositol transfer protein, inducible nitric oxide synthase, CIC-1 muscle chloride channel protein, placental bikunin, receptor kinase ligand LERK-3, GATA-4, thymopoietin, transcription factor E2f-2, S-adenosylmethionine synthetase, carcinoembryonic antigen, ret transforming gene, G protein-linked receptor, GTP-binding protein RALB, tyrosine kinase Syk, T cell leukemia LERK-2, tyrosine kinase (ELK1) oncogene, transcription factor SL1, phospholipase c, gastricsin, and D13S824E locus

See patent for claims 2-21 which are sub-claims of claim 1.

Claim 22. A chip or wafer comprising a nucleic acid microarray, wherein said nucleic acids hybridzie to target transcripts or cDNAs for group summarized in claim 1.

Claim 23. The chip of claim 22, wherein said chip is comprised of polymers, plastics, resins, polysaccharides, silica or silica-based material, fiberoptic materials, carbon, metals, inorganic glasses, or nitrocellulose.

Claim 24.The chip of claim 22, wherein said nucleic acids are cDNAs.

Claim 25. The chip of claim 22, wherein said nucleic acids are oligonucleotides.

Claim 26. The chip of claim 22, wherein said oligonucleotides are about 10 to about 50 base pairs or less in length.

Claim 27. A method for monitoring a therapy for multiple sclerosis comprising:

a. Obtaining an mRNA-containing sample from a subject receiving said therapy;
b. Determining expression information for one or more genes comprising material grouped material summarized above.
c. Comparing expresison information for said selected genes with the expression information of the same genes in an MS subject not receiving said therapy.

See patent for claims 28-41 which are sub-claims of claim 27.

Claim 42. A method for determining the efficacy of a therapy for multiple sclerosis comprising:

a. Obtaining an mRNA-containing sample from a subject receiving said therapy;
b. Determining expression information for one or more selected genes selected from the group summarized below;
c. Comparing expression information for said one or more selected genes with the expression information of the same gene or genes in an MS subject not receiving said therapy; and
d. Determining the efficacy of said therapy based on the ability of said therapy to alter the expression of said one more genes.

Group 2. skeletal muscle LIM-prot SLIM1, R kappa B, 815A9.1 myosin heavy chain, γ G2 psi from γ crystallin, thrombospondin 4, KIAA0178 (or Z97054)

See patent for claims 43-44 which are sub-claims of claim 42.

Claim 45. A method for treating multiple sclerosis (MS) comprising administering to a subject with MS a drug that causes an increase in the level of gene product selected from the group consisting of those genes indicated by a minus (-) sign in Tables 1-12.

Claim 46. A method for treating multiple sclerosis (MS) comprising administering to a subject with MS a drug that causes a decrease in the level of a gene product selected from the group consisting of those genes indicated by a plus (+) sign in Tables 1-12 and 16.

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Prior Art B, Summary of Invention

The present invention identifies biomarkers that are diagnostic of nerve cell injury and/or neuronal disorders. Detection of different biomarkers of the invention are also diagnostic of the degree of severity of nerve injury, the cell(s) involved in the injury, and the subcellular localization of the injury.

Invention: WO2005106038A2
(https://patents.google.com/patent/WO2005106038A2/en?oq=WO+2005%2f106038)

Claims and Features of Prior Art B:

Claim 1. A method of detecting and diagnosing neural injury and/or neuronal disorders comprising:

a. Detecting at least one or more protein biomarkers in a subject sample, and;
b. conelating the detection of one or more protein biomarkers with a diagnosis of neural injury and/or neuronal disorders, wherein the conelation takes into account the detection of one or more biomarker in each diagnosis, as compared to normal subjects wherein the one or more protein markers are selected from group summarized below and;
c. conelating the detection of one or more protein biomarkers with a diagnosis of neural injury and/or neuronal disorders, wherein the conelation takes into account the detection of one or more protein biomarkers in each diagnosis, as compared to normal subjects.

Group 1. axonal proteins, amyloid precursor proteins, dendritic proteins, somal proteins, presynaptic proteins, post-synaptic proteins, demyelination biomarkers, glial proteins, neurotransmitter biomarkers, dopaminergic proteins, noradrenergic proteins, serotonergic proteins, glutamatergic proteins, GABAergic proteins, neurotransmitter receptors, neurotransmitter transporters, vimentin (P31000), CK-BB (P07335), 14-3-3-epsilon (P42655), MMP2, MMP9

See patent for claims 2-30 which are sub-claims of claim 1.

Claim 31. The method of claim 1 , wherein one or more protein biomarkers are detected using a biochip anay.

Claim 32. The method of Claim 31, wherein the biochip anay is a protein chip anay.
Claim 33. The method of Claim 31, wherein the biochip anay is a nucleic acid array.
Claim 34. The method of Claim 31, wherein the one or more markers are immobilized on a biochip anay.
Claim 35. The method of Claim 34, wherein immobilized one or more markers are subjected to laser ionization to detect the molecular weight of the markers.
Claim 36. The method of Claim 35, wherein the molecular weight of the one or more markers is analyzed against a threshold intensity that is normalized against total ion cunent.
Claim 37. The method of Claim 36, wherein logarithmic transformation is used for reducing peak intensity ranges to limit the number of markers detected.

Claim 38. The method of Claim 31, comprising: generating data on immobilized subject samples on a biochip anay, by subjecting anay to laser ionization and detecting intensity of signal for mass/charge ratio;

and, transforming the data into computer readable form; and executing an algorithm that classifies the data according to user input parameters for detecting signals that represent markers present in patients suffering from neural injury and/or neuronal disorders and are lacking in normal subject controls.

See patent for Claims 39-46 which are sub-claims of claim 38.

Claims 47-64. A method of detecting and diagnosing neural injury and/or neuronal disorders comprising: See patent for list of proteins and other information.

Claim 65. A kit for diagnosing neural injury and/or neuronal disorders in a subject, the kit comprising:
(a) a substrate for holding a biological sample isolated from a human subject suspected of having a damaged nerve cell,
(b) an agent that specifically binds at least one or more of the neural proteins; and,
(c) printed instructions for reacting the agent with the biological sample or a portion of the biological sample to detect the presence or amount of at least one marker in the biological sample.

See patent for claims 66-73 which are sub-claims of claim 65.

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Prior Art C, Scientific Article"Identification of synaptic vesicle, pre‐ and postsynaptic proteins in human cerebrospinal fluid using liquid‐phase isoelectric focusing."

Davidsson et al., Electrophoresis, Vol. 20, 1999, pages 431-437.

Link: https://doi.org/10.1002/(SICI)1522-2683(19990301)20:3%3C431::AID-ELPS431%3E3.0.CO;2-2

Abstract:

Synaptic pathology is central in the pathogenesis of several psychiatric disorders, for example in Alzheimer's disease (AD) and schizophrenia. Quantification of specific synaptic proteins has proved to be a useful method to estimate synaptic density in the brain. Using this approach, several synaptic proteins have been demonstrated to be altered in both AD and schizophrenia. Until recently, the analysis of synaptic pathology has been limited to postmortem tissue. In living subjects, these synaptic proteins may be studied through analysis of cerebrospinal fluid (CSF). In an earlier study performed by us, one synaptic vesicle specific protein, synaptotagmin, was detected in CSF for the first time using a procedure based on affinity chromatography, reversed‐phase chromatography, sodium dodecyl sulfate‐polyacrylamide gel electrophoresis (SDS‐PAGE) and chemiluminescence immunoblotting. However, other synaptic proteins were not detectable with this procedure. Therefore, we have developed a procedure including precipitation of CSF proteins with trichloroacetic acid, followed by liquid‐phase isoelectric focusing using the Rotofor Cell, and finally analysis of Rotofor fractions by Western blotting for identification of synaptic proteins in CSF. Five synaptic proteins, rab3a, synaptotagmin, growth‐associated protein (GAP‐43), synaptosomal‐associated protein (SNAP‐25) and neurogranin, have been demonstrated in CSF using this method. The major advantage of liquid‐phase isoelectric focusing (IEF) using the Rotofor cell is that it provides synaptic proteins from CSF in sufficient quantities for identification. This method may also be suitable for identification of other types of trace amounts of brain‐specific proteins in CSF. These results demonstrate that several synaptic proteins can be identified and measured in CSF to study synaptic function and pathology in degenerative disorders.

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