**Supplementary Material**

**CIDP antibodies target junction proteins and identify patient subgroups:**

**an autoantigenomic approach**

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**Supplementary Tables**

**Table e-1** Clinical and paraclinical data of CIDP patient subgroups obtained by PCA

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Parameters** | **CIDP**  **subgroup n°1 (clustered patients)** | **CIDP**  **subgroup n°2 of PCA** | **Median or frequency difference (95% CI)a** | ***P*-value**  **(MW or Chi² test)b** |
| **Age at onset – median year (25th-75th percentile)** | 49.2 (26.5-57.4) | 64.0 (57.2-71.6) | **15 (3 / 34)** | **0.01** |
| **Sex – F/M (% of F)** | 3/7 (30%) | 4/8 (33%) | 3% (-36 / 42) | 0.87 |
| **Type of symptoms – N (%)** |  |  |  |  |
| **Mixed (sensory and motor)** | 8/10 (80%) | 4/12 (33%) | **47% (10 / 83)** | **0.03** |
| **Purely sensory** | 0/10 (0%) | 4/12 (33%) | **33% (7 / 60)** | **0.05** |
| **Purely motor** | 2/10 (20%) | 4/12 (33%) | 13% (-23 / 50) | 0.49 |
| **Pain** | 4/10 (40%) | 3/12 (25%) | 15 (-24 / 54) | 0.46 |
| **Ataxia** | 3/8 (38%) | 7/11 (64%) | 26% (-18 / 70) | 0.27 |
| **Topography of symptoms – N (%)** |  |  |  |  |
| **Four limbs** | 5/10 (50%) | 10/12 (83%) | 33% (-4 / 71) | 0.10 |
| **Purely LL** | 5/10 (50%) | 2/12 (17%) | 33% (-4 / 71) | 0.10 |
| **Distal** | 0/10 (0%) | 2/12 (17%) | 17% (-4 / 38) | 0.19 |
| **Asymmetry** | 1/10 (10%) | 1/12 (8%) | 2% (-23 / 26) | 0.89 |
| **Cranial nerves** | 1/10 (10%) | 0/12 (0%) | 10% (-9 / 29) | 0.27 |
| **Type of progression – N (%)** |  |  |  |  |
| **Chronic evolution without relapse** | 6/10 (60%) | 7/12 (58%) | 2% (-40 / 43) | 0.94 |
| **Chronic evolution with relapses** | 3/10 (30%) | 3/12 (25%) | 5% (-33 / 43) | 0.80 |
| **Only relapses** | 1/10 (10%) | 2/12 (17%) | 7% (-21 / 35) | 0.66 |
| **Electroneuromyography – N (%)** |  |  |  |  |
| **Presence of conduction blocks** | 8/10 (80%) | 6/12 (50%) | 30% (-8 / 68) | 0.15 |
| **Axonal loss** | 5/10 (50%) | 2/12 (17%) | 33% (-4 / 71) | 0.10 |
| **Biological data – N (%)** |  |  |  |  |
| **Abnormal CSF total proteins** | 6/6 (100%) | 6/9 (67%) | **33% (3 / 64)** | 0.13 |
| **Monoclonal gammopathy** | 1/10 (10%) | 2/12 (17%) | 7% (-21 / 35) | 0.65 |
| **Treatment effects – N (%)** |  |  |  |  |
| **IVIg responders** | 7/9 (78%) | 8/10 (80%) | 2% (-35 / 39) | 0.91 |
| **Corticosteroid responders** | 3/6 (50%) | 1/3 (33%) | 17% (-50 / 83) | 0.65 |
| **Clinical score – median (25th-75th percentile)** |  |  | Cf. manuscript |  |
| **mRS - maximum value before treatment** | 3.0 (2.0-4.0) | 2.5 (2.0-4.0) | 0 (-2 / 2) | 0.82 |
| **mRS - minimum value after treatment** | 2.0 (1.0-2.0) | 1.0 (0.3-1.8) | 1 (0 / 2) | 0.19 |

Abbreviations: CI = confident interval; CIDP = chronic inflammatory demyelinating polyneuropathy; CSF = cerebrospinal fluid; F = female; IV Ig = intravenous immunoglobulins; LL = lower limbs; M = male; mRS = modified Rankin Score; MW = Mann-Whitney; N = number; PCA = principal component analysis.

a The bolded values highlight the difference for which the 95% CIs exclude the zero.

b The bold p-values are statistically significant (≤0.05).

**Table e-2** Potential confounder effects tested in IVIg responders vs. non-responders.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Parameters** | **IVIg responders** | **IVIg non-responders** | **Median or frequency difference (95% CI)a** | ***P*-value**  **(MW or Chi² test)b** |
| **Age at onset – median year (25th-75th percentile)** | 64.6  (50.3-71.4) | 54.8  (39.5-59.7) | 9 (-10 / 28) | 0.32 |
| **Sex – N of F (%)** | 5/15 (33%) | 1/4 (25%) | 8% (-40 / 57) | 0.76 |
| **Sampling delay (sample date - date of onset) – median year (25th-75th percentile)** | 2.4 (0.5-7.3) | 1.9 (0.9-3.2) | 0 (-10 / 3) | 1.00 |
| **IVIg treatment delay after sampling – median day (25th-75th percentile)** | 0.0 (0.0-11.3) | 1.0 (0.0-12.3) | 1 (-13 / 15) | 0.83 |
| **Proportion of patients naïve to any immunomodulatory treatment – N (%)** | 8/13 (62%) | 3/3 (100%) | 38% (12 / 65) | 0.21 |
| **Proportion of patients naïve to any immunomodulatory treatment – N (%)** | 9/14 (64%) | 4/4 (100%) | 36% (11 / 61) | 0.17 |
| **Sampling delay after last immunomodulatory treatment – median day (25th-75th percentile)** | 42 (37-79) | - |  | - |
| **Type of symptoms – N (%)** |  |  |  |  |
| **Mixed (sensory and motor)** | 7/15 (47%) | 3/4 (75%) | 28% (-21 / 78) | 0.33 |
| **Purely sensory** | 2/15 (13%) | 1/4 (25%) | 12% (-34 / 57) | 0.58 |
| **Purely motor** | 6/15 (40%) | 0/4 (0%) | 40% (15 / 65) | 0.14 |
| **Pain** | 6/15 (40%) | 0/4 (0%) | 40% (15 / 65) | 0.14 |
| **Ataxia** | 7/15 (47%) | 2/3 (67%) | 20% (-39 / 79) | 0.54 |
| **Topography of symptoms – N (%)** |  |  |  |  |
| **Four limbs** | 11/15 (73%) | 2/4 (50%) | 23% (-31 / 77) | 0.39 |
| **Purely LL** | 4/15 (27%) | 2/4 (50%) | 23% (-31 / 77) | 0.38 |
| **Distal** | 1/15 (7%) | 1/4 (25%) | 18% (-26 / 63) | 0.30 |
| **Asymmetry** | 1/15 (7%) | 1/4 (25%) | 18% (-26 / 63) | 0.30 |
| **Cranial nerves** | 1/15 (7%) | 0/4 (0%) | 7% (-6 / 19) | 0.61 |
| **Type of progression – N (%)** |  |  |  |  |
| **Chronic evolution without relapse** | 7/15 (47%) | 3/4 (75%) | 28% (-21 / 78) | 0.33 |
| **Chronic evolution with relapses** | 5/15 (33%) | 1/4 (25%) | 8% (-40 / 57) | 0.76 |
| **Only relapses** | 3/15 (20%) | 0/4 (0%) | 20% (0 / 40) | 0.34 |
| **Electroneuromyography – N (%)** |  |  |  |  |
| **Presence of conduction blocks** | 12/15 (80%) | 1/4 (25%) | 55% (8 / 102) | **0.04** |
| **Axonal loss** | 5/15 (33%) | 1/4 (25%) | 8% (-40 / 57) | 0.76 |
| **Biological data – N (%)** |  |  |  |  |
| **Abnormal CSF total proteins** | 10/12 (83%) | 1/1 (100%) | 17% (-4 / 38) | 0.67 |
| **Monoclonal gammopathy** | 3/15 (20%) | 0/4 (0%) | 20% (0 / 40) | 0.34 |
| **Presence of antinuclear antibodies** | 4/11 (36%) | 0/1 (0%) | 36% (8 / 65) | 0.48 |
| **Presence of other autoantibodies** | 3/11 (27%) | 1/1 (100%) | 73% (46 / 99) | 0.16 |
| **Co-morbidities – N (%)** |  |  |  |  |
| **Diabetes** | 2/11 (18%) | 1/1 (100%) | 82% (59 / 105) | 0.08 |
| **Cardiovascular risk factor or disease** | 7/11 (64%) | 1/1 (100%) | 36% (8 / 65) | 0.48 |
| **Other chronical disease** | 6/11 (55%) | 1/1 (100%) | 45% (16 / 75) | 0.40 |
| **Clinical scores before treatment – N (%)** |  |  |  |  |
| **INCAT ≥5** | 6/14 (43%) | 0/2 (0%) | 43% (17 / 69) | 0.26 |
| **mRS ≥4** | 6/15 (40%) | 0/3 (0%) | 40% (15 / 65) | 0.19 |

Abbreviations: CI = confident interval; CSF =cerebrospinal fluid; F = female; IVIg = intravenous immunoglobulins; LL = lower limbs; M = male; mRS = modified Rankin Score; MW = Mann-Whitney; N = number.

a The bolded values highlight the difference for which the 95% CIs exclude the zero.

b The bold p-values are statistically significant (≤0.05).

**Table e-3** Additional information on anchoring junction proteins associated with the plasma membrane or extracellular space.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Protein name** | **Gene name** | **Plasma membrane (GO)** | **Extracellular space (GO)** | **General Function (UniProt)** | **Known role in NS genral (CM)** | **Impact on Actin cytoskeleton** |
| 14-3-3 protein zeta/delta | YWHAZ | - | + | Adapter protein, signaling pathways | Regulates spine maturation (UniProt) | (+) |
| Actin-related protein 2/3 complex subunit 1B | ARPC1B | - | + | Actin polymerization | Initiation of neuronal dendrite branching1; spine synapse maturation2; process extension and axon ensheathment during myelination3; **in PNS:** 'potential effector protein in actin cytoskeleton regulation of DRG growth cones4 | + |
| Annexin A6 | ANXA6 | - | + | Regulation Ca2+ release from intracellular stores | **In PNS:** key membrane scaffolding protein during sensory neuron membrane biogenesis5; modulates Ca2+ and K+ conductances of spinal cord and dorsal root ganglion neurons6 | + |
| Band 4.1-like protein 2 | EPB41L2 | + | + | Required for dynein-dynactin complex during anaphase | **In PNS:** Axoglial organization and maintenance in myelinated axons7,8 | + |
| Brain-specific angiogenesis inhibitor 1-associated protein 2 | BAIAP2 | + | - | Adapter protein, links small G-proteins to cytoplasmic effector proteins | Formation of filopodia, neurite initiation, and neuronal dendritic branching9; synaptic transmission10 | + |
| Brain-specific angiogenesis inhibitor 1-associated protein 2-like protein 1 | BAIAP2L1 | + | + | Possible adapter protein involved in formation of actin bundle clusters | **In PNS:** Biomarker for mechanical nociceptor type of DRG neurons11 | + |
| Cadherin-15 | CDH15 | + | + | Calcium-dependent cell adhesion proteins | **In PNS:** Roles in axon/Schwann cell interactions and node of Ranvier structural maintenance12, neuromuscular axis development13 and Schwann cells14 | - |
| CD59 glycoprotein | CD59 | + | + | Complement inhibitor | Potential complement inhibitor protecting from autoimmune neurological disease16 and central nervous system-restricted lesions17; **In PNS:** 'deficiency might cause CIDP-like syndrome18 | - |
| Cdc42 effector protein 4 | CDC42EP4 | + | - | Actin cytoskeleton organization | Scaffold protein contributing to glia-neuron tripartite synapse configuration19 | + |
| Cell surface glycoprotein MUC18 | MCAM | + | + | Cell adhesion and cohesion of the endothelial monolayer at intercellular junctions | Role in and marker for neuroinflammation20; involved in neurite extension21 | + |
| Copine-3 | CPNE3 | + | + | Calcium- and growth factor-dependent phospholipid-binding protein |  | - |
| Coronin-1B | CORO1B | + | + | Regulates leading edge dynamics and cell motility |  | + |
| Cytohesin-1 | CYTH1 | + | - | Membrane trafficking, junctional remodeling and epithelial polarization | **In PNS:** Regulation of myelination22,23 | + |
| E3 ubiquitin-protein ligase CBL | CBL | + | - | Adapter protein, regulator of signaling pathways triggered by cell surface receptors | Role in microglia-mediated neuroinflammation24; neuroprotective role25 | + |
| Epidermal growth factor receptor | EGFR | + | + | Receptor tyrosine kinase activating several signaling cascades | Regulation of Myelination via oligodendrocyte’ maturation; Astrocyte differentiation, morphology and maturation, Regulation of neurite outgrowth26; **in PNS:** 'Regulation of neurite outgrowth, peripheral nociception26 | + |
| Ezrin | EZR | + | + | Involved in connecting cytoskeletal structures to the plasma membrane. | Mediation of neuritogenesis27; **in PNS:** node of Ranvier formation28,29; co-localizes with NF155 at the node of Ranvier30 | + |
| Membrane-associated guanylate kinase, WW and PDZ domain-containing protein 1 | MAGI1 | + | - | Probable scaffolding protein at cell-cell junctions | **In PNS:** 'Essential scaffold for ion transport in DRG neurons; important role in thermal nociception and acute inflammatory pain31 | + |
| Poly(rC)-binding protein 2 | PCBP2 | - | + | Adapter protein and single-stranded nucleic acid binding protein | Potential role in neuronal cell proliferation and apoptosis32; **in PNS:** potential role in Schwann cell proliferation after nerve injury33 | - |
| Protein disulfide-isomerase A3 | PDIA3 | - | + | Formation and breakage of disulfide bonds between cysteine residues | Neuroprotective role34; **in PNS:** peripheral nerve regeneration35 | - |
| Radixin | RDX | + | + | Binding of actin filaments to the plasma membrane | Role in 'in neuroblast proliferation and migration36; **in PNS**: node of Ranvier formation28,29 | + |
| Transducin-like enhancer protein 2 | TLE2 | - | + | Transcriptional corepressor that binds to a number of transcription factors | Regulation of neuronal differentiation37 | - |

**Supplementary Methods**

**Protein microarrays data analysis and definition of repertoires**

Data analysis of protein microarrays included local background subtraction (normalizes for serum-specific background noise similar to improved ELISA approaches),38 deriving average and coefficient of variation (CV) of duplicate spots, and subtraction of unspecific signal resulting from secondary antibody (assessed by serum-free control array). Proteins with CV > 25% in their duplicate spots (technical replicates) were excluded from the analysis if the corresponding signal was above noise (≥1.5 SDs above the arrays total mean signal intensities; without this restriction we would have lost many low signals in the noise). An *intra*-sample standard normalization (*intra*-z-score) was used to normalize for systematic biases between the samples (e.g., systematic labelling efficiency differences). In detail, every signal intensity was expressed as the number of SDs above or below the arithmetical mean of each array, resulting in average intra-z-scores of zero for each single array. *Inter*-sample standard normalization (*inter*-z-score) was used to compare the reactivities of each antigen among the samples. In detail, for each antigen and for each sample we expressed an inter-z-score indicating how many SDs does each intra-z-score lie above or below the arithmetical mean of the corresponding intra-z-scores of the other two groups (ONP+HC for defining the CIDP repertoire, CIDP+HC for the ONP repertoire, CIDP+ONP for the HC repertoire).

To define the antigen repertoires comprising group-specific and significantly targeted antigens, all of the following criteria must be fulfilled: 1) signals must be far above noise, i.e., *intra*-z-score ≥2; 2) antigens must be in ≥1 sample of the group-of-interest, but in none of the other two groups, ≥ 3 SDs above the mean of the corresponding two sample groups (*inter*-z-score ≥3); 3) the sample with the strongest signal of the group-of-interest must be ≥0.6 SDs above the sample with the strongest signal of the other two groups, in order to exclude borderline candidates.

**PANTHER analysis**

For the PANTHER analysis, we took only those antigens into account that have at least one GO annotation in order to avoid potential bias by yet undescribed proteins. PANTHER version 14.0 (released 2018-12-03) online software (<http://www.pantherdb.org/>) was applied to identify the Gene Ontology (GO) categories (*Cellular Component* and *Biological Process*) and the Reactome categories covered by the repertoires of targeted antigens.39 The Cellular Component category of GO refers to “the place in the cell where a gene product is active.”40 The Biological Process category of GO refers to “a biological objective to which the gene or gene product contributes”.40 Reactome pathways represents a network of molecular reactions that are systematically described in molecular detail to generate an ordered matrix of molecular transformations.41 We made the repertoire lists software-readable, removed duplicate entries, and uploaded them in Panther, performing a statistical overrepresentation test (PANTHER Overrepresentation Test, Released 20181113) using “homo sapiens” as organism and GO categories “GO Cellular Component complete” and “GO Biological Process complete” (GO database released 2019-01-01), as well as “Reactome pathways” (Reactome version 65 Released 2018-06-12) as the annotation data set. The list of all spotted HuProt™ proteins were applied as reference list.

For the second PANTHER analysis normalizing the number of targeted proteins of each category with the category size instead of the repertoire size we compared only the CIDP-repertoire (22 samples) with the corresponding and equally sized joint control repertoires (ONP+HC, 21 samples) in order to avoid a bias of different sample sizes in the three study groups.

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