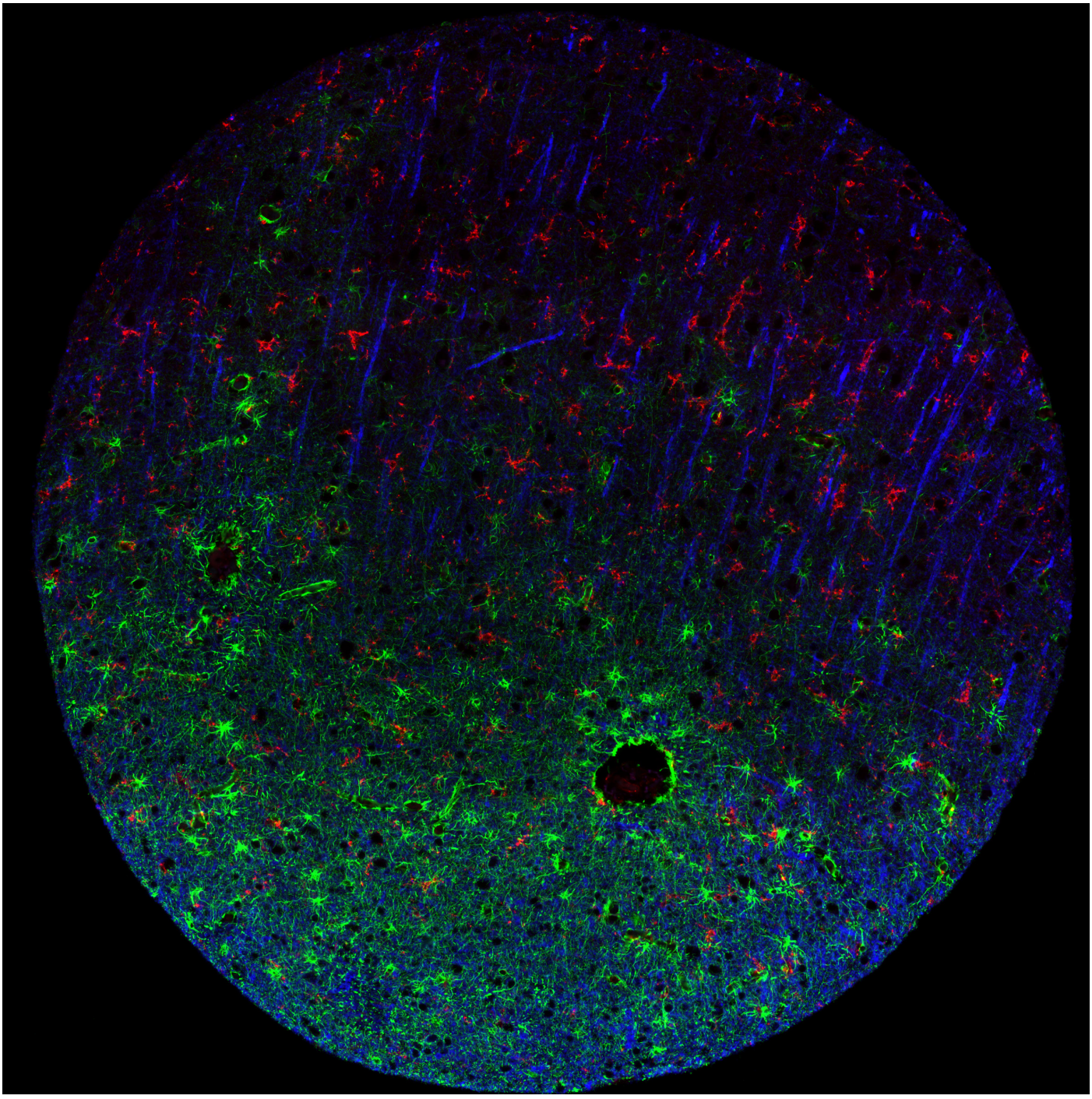


NEUROINFLAMMATION

ATLAS ANTIBODIES

Our understanding of the molecular pathogenesis of neuroinflammation is growing steadily. Progress in different areas of basic research, new animal models, and the generation of specific antibody markers to target essential proteins help to find and improve the treatment of patients with neuroimmunological diseases. In this white paper, we discuss the role of microglia, oligodendrocytes, and astrocytes in the neuroinflammatory processes highlighting the relevant antibody markers with a particular focus on multiple sclerosis.

Neuroinflammation broadly defines the collective reactive immune response in the brain and spinal cord in response to injury and disease. Inflammation in the central nervous system (CNS) is commonly associated with various degrees of tissue damage, such as loss of myelin and neurons.

The neuroinflammatory process is complex and involves disruption of the blood-brain barrier (BBB), peripheral leukocyte infiltration, edema, and gliosis. The inflammatory response is characterized by a host of cellular and molecular aberrations within the brain.

Neuroinflammation arises within the CNS through phenotypic changes of different non-neuronal cell types in the brain, such as microglia, oligodendrocytes, and astrocytes, causing the release of different cytokines and chemokines, and finally recruitment and infiltration of peripheral blood cells, mainly T-and B-cells, into the brain parenchyma [1,2,3].

Neuroinflammatory processes are the key causative factors behind brain and spinal cord injury. This is true not only for acute brain trauma and hypoxic-ischemic brain damage following stroke, but also for chronic infection and neurodegenerative diseases, such as Alzheimer's disease, amyotrophic lateral sclerosis (ALS), Lewy body dementia, and leukoencephalopathies like multiple sclerosis (MS). In addition, local peritumoral inflammation plays a role in the clinical progression and malignancy of glioblastomas, the most aggressive primary brain tumors.

Recent studies have revealed unexpected insights by providing hints of a protective role of inflammation.

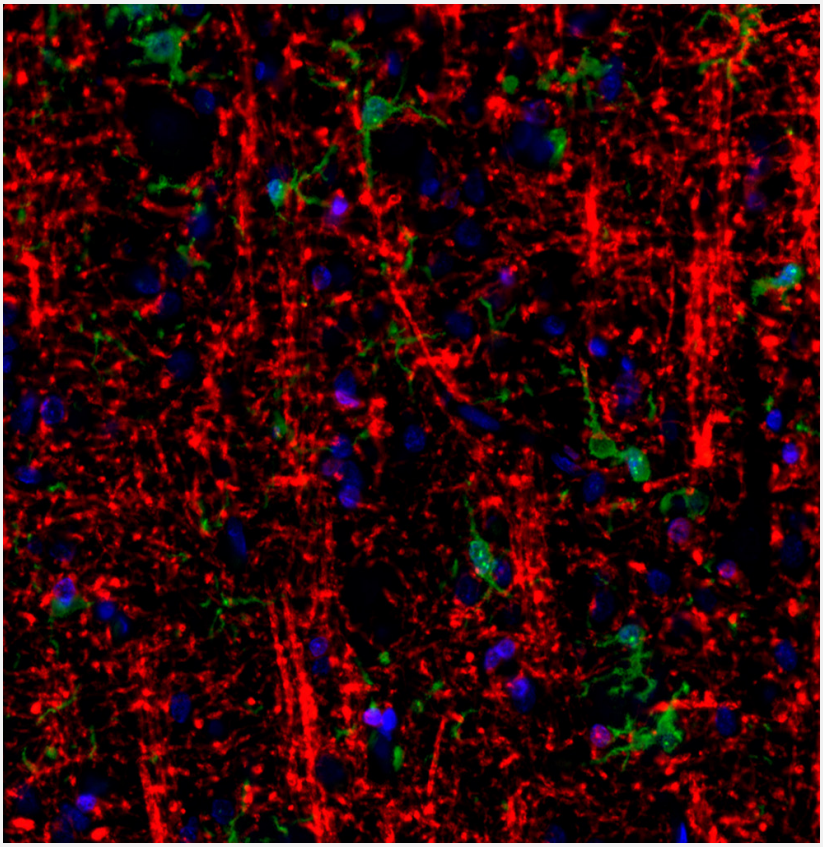


Figure 1
ICC-IF of myelinated axons and microglia.
Multiplexed immunofluorescence staining of human cerebral cortex. Microglial cells are visualized using the Anti-AIF1 polyclonal antibody (HPA049234, in green). Myelinated processes are shown using the Anti-MBP monoclonal antibody (AMAb91062, in red). Nuclei are counterstained by DAPI, in blue.

Cover image: Multiplexed immunofluorescence staining of human cerebral cortex (normal control) visualizing microglial cells in red (Anti-P2RY12, HPA014518), astrocytes in green (Anti-GFAP, AMAb91033) and myelinated processes in blue (Anti-MBP, AMAb91062).

Activated Microglia: the Hallmark of Neuroinflammation.

Glial cells, comprising astrocytes, oligodendrocytes, and microglia, are the non-neuronal cell population of the central nervous system (CNS). Glial cells do not produce electrical impulses. They maintain homeostasis, form myelin, and provide support and protection for neurons.

Microglia accounts for 10-15% of all cells found within the brain. They are the first-line defense innate immune cells, commonly regarded as “brain resident macrophages” and the sole resident immune cell type in the CNS [4].

The microglia cells are multitasking and instrumental for maintaining essential regulatory and homeostatic functions of the brain and spinal cord.

Naïve resting microglia cells are essentially immobile and continuously scan the CNS microenvironment for “danger signals” with their highly motile pseudopodial extensions.

When transforming functional phenotype from “surveying” into “primed” phenotypes, microglia undergo considerable molecular changes. Hence, during pathology, microglia change their morphology and deregulate their homeostatic gene profile.

Different cues produced within the CNS tissue microenvironment choreograph the microglia specification to enable the dramatic changes of the activation state of these cells.

Single-cell molecular RNA-profiling of microglia isolated from the brains of patients with MS and Alzheimer´s disease has enabled the discovery of novel microglia subtypes with unusual properties.

Microglial cells are very plastic and readily adopt into distinct phenotypes, including the classical activated “pro-inflammatory” (M1) state and the alternatively activated “immunomodulatory” (M2) state in response to various stimulations [5].

Activated M1 microglia upregulate cell surface markers, like HLA-DRA and CD86, to promote neuroinflammation and oligodendrocyte damage. In fact, as antigen-presenting cells (APC), microglia are endowed with the ability to release proinflammatory cytokines and chemokines into the CNS tissue microenvironment. The alternatively activated M2 microglia cells, on the other hand, are neuroprotective.

Activated microglia are the primary regulators of the pro-and anti-inflammatory microenvironment of the CNS. In the pathogenesis of Alzheimer´s disease, microglia participate in amyloid Aβ-plaques formation and the associated pathology.

A substantial buildup of amyloid Aβ in AD-patients induces inflammation and glial cell activation leading to a vicious cycle and increased Aβ-formation. Microglial phagocytosis of Aβ fibrils and the pro-inflammatory activation that follows involve a complex pro-inflammatory pathway mediated by CD36, CD49 and TLR4, among others. Also, cytokine-mediated activation of microglia induces neuroinflammation per se and subsequent neuronal loss.

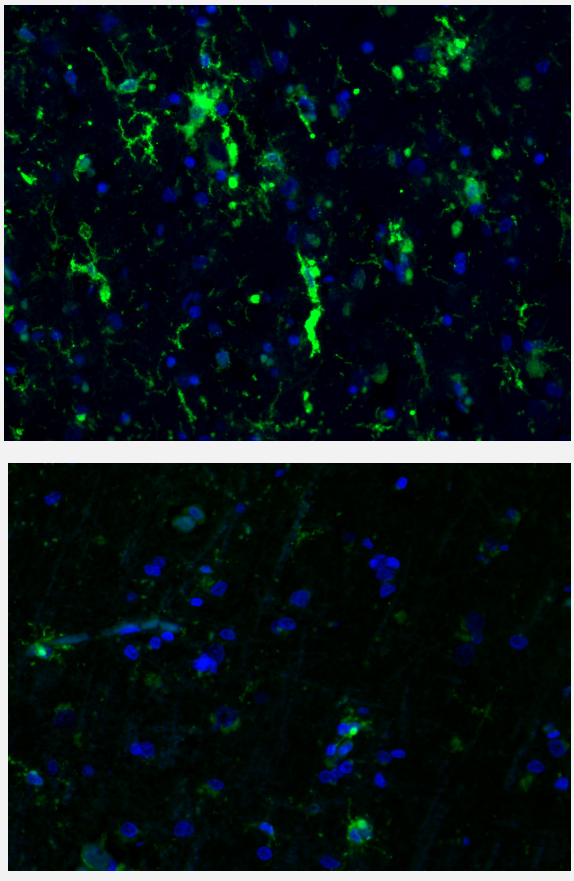


Figure 2
ICC-IF of activated microglia in AD.
Immunofluorescence staining of human cerebral cortex from an Alzheimer’s disease patient (upper image) and a normal control (lower image) using the Anti-HLA-DRA antibody (AMAb91674) showing positivity in microglial cells (in green). Nuclei counterstained by DAPI, in blue.

Critical Role of Macrophages, Oligodendrocytes, and Astrocytes in Neuroinflammation

Macrophages play definite roles in neuroinflammation, where they participate in the initiation, maintenance, and resolution of inflammation. This lineage of non-brain parenchymal monocytes derives from hematopoietic precursors in the bone marrow, whereas, as mentioned above, microglia are parenchymal cells of the CNS and originate from a myeloid precursor in the fetal yolk sac.

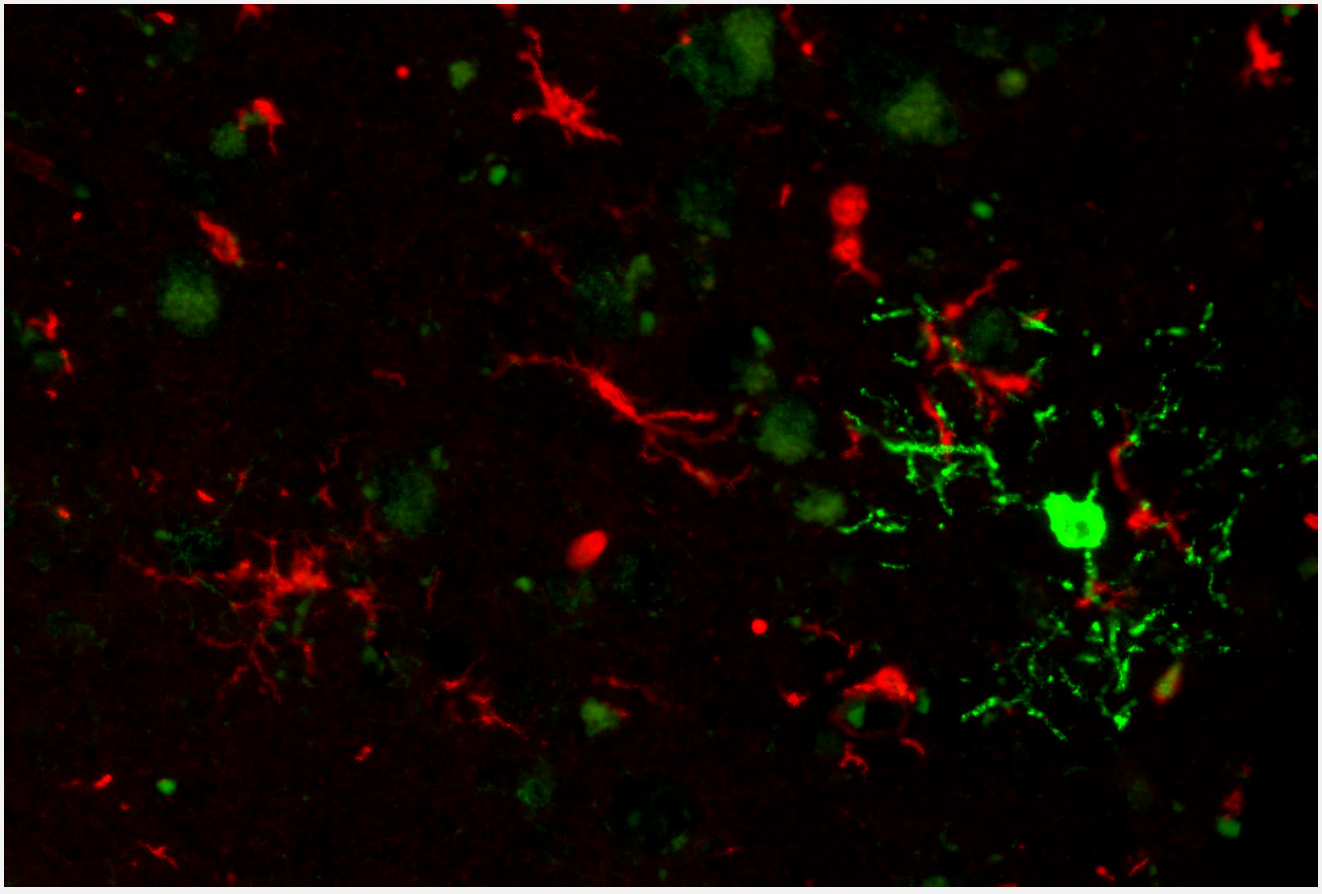
Both macrophages and microglia share CD68 and CD163 expression; however, macrophages display a higher constitutional expression of HLA-DR than microglia.

Like microglia, macrophages come as pro-inflammatory (M1) and neuroprotective (M2). M1 macrophages express high MHC Class II (HLA-DR) on their surface and produce tissue-damaging pro-inflammatory cytokines like IL1β, TNFβ, and IL-6 [6]. Neuroprotective and immunomodulatory (M2) macrophages are not antigen-presenting and instead produce anti-inflammatory cytokines and mediators like TGFβ and IL-10 that facilitate tissue repair during the remission phase in, for example, MS patients.

Oligodendrocytes are responsible for axonal myelination in the CNS white matter but are not generally thought of as being directly engaged in neuroinflammation.

In MS and Experimental Allergic Encephalitis (EAE) model, however, oligodendroglia precursor cells (OPCs) develop a distinctly disease-specific immune phenotype, “immune-OPCs” [7]. This specific immune-OPC phenotype is antigen-presenting and phagocytosing and can support T-cell proliferation and survival, actively contributing to the neuroinflammation occurring in EAE and MS. OPCs isolated from white matter of MS-patients have been defined by single-cell epigenomic analysis by their increased chromatin accessibility at immune genes.

Figure 3
ICC-IF of oligodendrocytes and microglia in AD.
Multiplexed immunofluorescence staining of human cerebral cortex (Alzheimer’s disease) visualizing oligodendrocyte precursor cells in green (Anti-GPR17, AMAb91624) and microglia cells in red (Anti-AIF1, HPA049234).



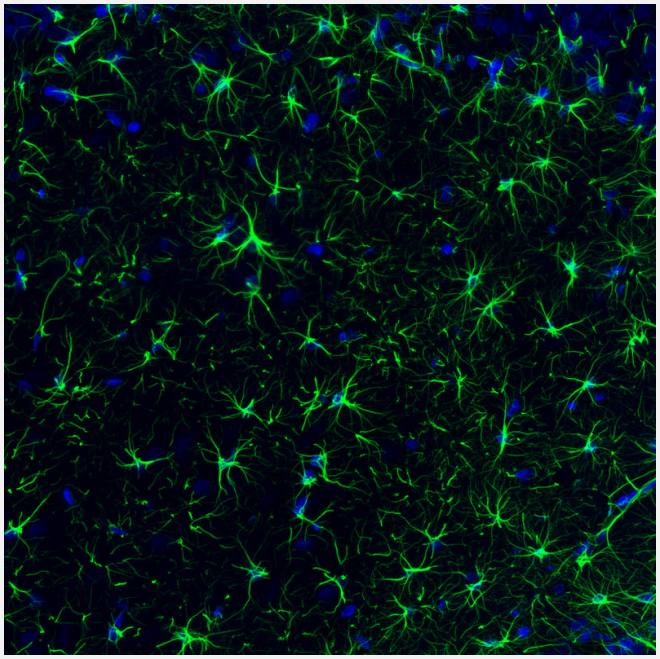


Figure 4
ICC-IF of astrocytes in rat brain tissue.
Immunofluorescence staining of rat hippocampus with the Anti-GFAP monoclonal antibody (AMAb91033, in green) shows strong positivity in astrocytes. Nuclei counterstained by DAPI, in blue.

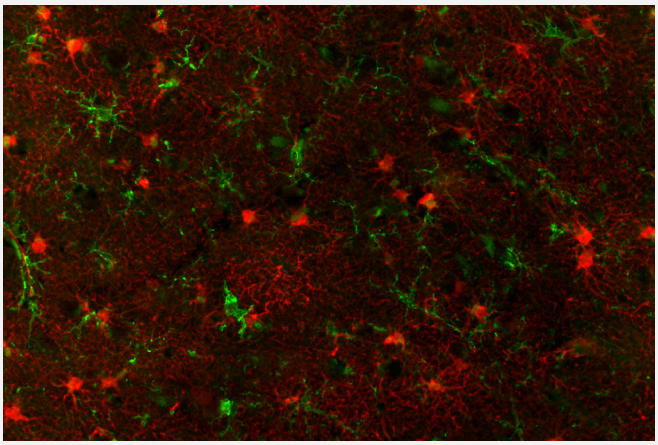


Figure 5
ICC-IF of astrocytes and microglia in human brain tissue.
Multiplexed immunofluorescence staining of human cerebral cortex showing microglia in green (Anti-TMEM119, AMAb91528) and astrocytes in red (Anti-MT3, HPA004011).

Astrocytes are the star-shaped glial cells that constitute one-third of the cell population in the CNS. They regulate neuroprotective homeostasis in the CNS but also become reactive with pro-inflammatory properties during neuroinflammation [8].

Under inflammatory conditions, astrocytes in the CNS proliferate massively and become hypertrophic. This growth is associated with strong upregulation of glial fibrillary acidic protein (GFAP), leading to the so-called astroglial scarring (the counterpart to scarring in peripheral tissues).

Proinflammatory astrocytes actively secrete inflammatory mediators, including cytokines. Transcriptomic profiling of reactive astrocytes in neuroinflammation and ischemia has revealed two distinct forms of astrocytes: A1 and A2. The A1 astrocytes display enhanced levels of neuron-damaging complement cascade proteins, whereas the A2 phenotype is prevalently anti-inflammatory and neuroprotective.

It appears that a neurotoxic factor secreted from A1 astrocytes is detrimental to motor neurons in ALS, but not in healthy individuals. The proinflammatory astrocyte state also contributes to the pathogenesis of ALS.

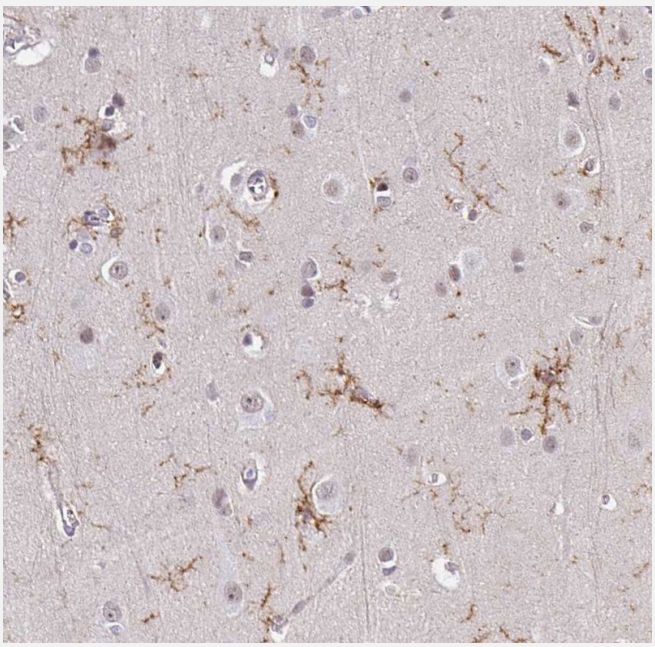


Figure 6
IHC staining of microglia in human cortical tissue.
IHC staining in human cerebral cortex with Anti-TMEM119 polyclonal antibody (HPA051870) shows moderate membranous positivity in microglia, in brown.

Table 1 lists a selection of antibody markers for neurons and the different types of non-neuronal cellular populations involved in neuroinflammation processes.

Microglia-specific markers include the purinergic P2RY12 receptor, the transmembrane protein 119 (TMEM119), HLA class II histocompatibility antigen (HLA-DRA), and the allograft inflammatory factor 1 (AIF-1), also known as ionized calcium-binding adapter molecule 1 (IBA1).

Examples of mixed microglia/macrophage markers are the clusters of differentiation CD68 and CD163.

Oligodendrocytes-specific markers include Olig2, GPR17, and MBP, while astrocytes-specific markers are GFAP, AQP4, and MT3.

Markers for MS include MBP, MOG, GFAP, NEFM, MAP2, CD3e, FoxP3, CD20, CD4, CD8.

Table 1

Atlas Antibodies' markers for different cells type involved in neuroinflammation processes.

Target cell	Product Name	Product Number	Clonality	Validated Applications	Seq Identity mouse / rat
Astrocytes	Anti-GFAP	AMAb91033	Monoclonal	IHC*, WB*	98 / 100%
Astrocytes	Anti-GFAP	HPA056030	Polyclonal	IHC*,WB, ICC-IF	98 / 100%
Astrocytes	Anti-GLUL	AMAb91101	Monoclonal	IHC, WB*	95 / 53%
Astrocytes	Anti-GLUL	AMAb91102	Monoclonal	IHC, WB*	95 / 92%
Astrocytes	Anti-GLUL	AMAb91103	Monoclonal	IHC, WB	95 / 92%
Astrocytes	Anti-GLUL	HPA007316	Polyclonal	IHC,WB*	95 / 53%
Astrocytes	Anti-S100B	AMAb91038	Monoclonal	IHC*, WB	99 / 98%
Astroglia	Anti-EZR	AMAb90976	Monoclonal	IHC*,WB*,ICC-IF	93 / 93%
Astroglia	Anti-EZR	HPA021616	Polyclonal	IHC*,WB*,ICC-IF	93 / 93%
Astroglia	Anti-MT3	HPA004011	Polyclonal	IHC*	86 / 83%
Microglia	Anti-AIF	HPA049234	Polyclonal	IHC*	84 / 84%
Microglia	Anti-CD68	AMAb90874	Monoclonal	IHC,WB*	76 / 76%
Microglia	Anti-CD68	HPA048982	Polyclonal	IHC*, WB*	76 / 76%
Microglia	Anti-ITGAM (CD11b)	AMAb90911	Monoclonal	IHC*, WB	67 / 68%
Microglia	Anti-PTPRC	AMAb90518	Monoclonal	IHC,WB	35 /37%
Microglia	Anti-PTPRC	HPA000440	Polyclonal	IHC*,WB	35 /37%
Microglia	Anti-TMEM119	AMAb91528	Monoclonal	IHC*,WB, ICC-IF	52 / 46%
Microglia	Anti-TMEM119	HPA051870	Polyclonal	IHC*	52 / 46%
Microglia	Anti-P2RY12	HPA014518	Polyclonal	IHC*	70 / 63%
Microglia	AIF1/Iba1	AMAb91671	Monoclonal	IHC	95 / 87%
Microglia	AIF1/Iba2	AMAb91672	Monoclonal	IHC	95 / 87%
Microglia	Anti-HLA-DRA	AMAb91674	Monoclonal	WB, IHC	0 / 90%
Neurons	Anti-NEFM (NF160)	AMAb91027	Monoclonal	IHC, WB	98 / 98%
Neurons	Anti-NEFM (NF160)	AMAb91028	Monoclonal	IHC, WB	98 / 98%
Neurons	Anti-NEFM (NF160)	AMAb91029	Monoclonal	IHC, WB	98 / 98%
Neurons	Anti-NEFM (NF160)	AMAb91030	Monoclonal	IHC, WB	98 / 98%
Neurons	Anti-NEFH (NF200)	AMAb91025	Monoclonal	IHC, WB	98 / 98%
Neurons	Anti-NEFH (NF200)	HPA061615	Polyclonal	IHC*	88 / 94%
Neurons	Anti-UCHL1	AMAb91145	Monoclonal	IHC, WB, ICC-IF	97 / 97%
Neurons	Anti-UCHL1	HPA005993	Polyclonal	IHC*,WB*,ICC-IF	97 / 97%
Neurons	Anti-VIM	AMAb90516	Monoclonal	IHC,WB*	99 / 99%
Neurons	Anti-VIM	HPA001762	Polyclonal	IHC*,WB*,ICC-IF	99 / 99%
Oligodendrocytes	Anti-MBP	AMAb91062	Monoclonal	IHC, WB, ICC-IF	97 / 97%
Oligodendrocytes	Anti-MBP	AMAb91063	Monoclonal	IHC, WB, ICC-IF	97 / 97%
Oligodendrocytes	Anti-MBP	AMAb91064	Monoclonal	IHC, WB	97 / 97%
Oligodendrocytes	Anti-MOG	AMAb91066	Monoclonal	IHC, WB	91 / 89%
Oligodendrocytes	Anti-MOG	AMAb91067	Monoclonal	IHC, WB	91 / 89%
Oligodendrocytes	Anti-MOG	HPA021873	Polyclonal	IHC	91 / 89%
Oligodendrocytes	Anti-CNP	AMAb91068	Monoclonal	IHC, WB*	76 / 77%
Oligodendrocytes	Anti-CNP	AMAb91069	Monoclonal	IHC, WB	76 / 77%
Oligodendrocytes	Anti-CNP	AMAb91072	Monoclonal	IHC, WB, ICC-IF	76 / 77%
Oligodendrocytes	Anti-CNP	HPA023280	Polyclonal	IHC*,WB*,ICC-IF	76 / 77%
Oligodendrocytes	Anti-GRP17	AMAb91624	Monoclonal	WB, IHC	95 / 95%
Oligodendrocytes	Anti-Olig2	HPA003254	Polyclonal	IHC*, WB	94 / 93%

* Products with enhanced validation for indicated application.

Multiple Sclerosis

Multiple sclerosis (MS) is a human-specific, chronic inflammatory, and neurodegenerative CNS disease and the single most prevalent chronic neurological disability in young adults.

Demyelination is the primary pathological event in MS, followed by reactive gliosis and edema. Neuroinflammation affects both white and grey matter, a process that engages activated microglia. It appears that stressed immune OPCs, reactive astrocytes, and activated microglia become most prevalent towards the rim of the MS lesions. The cumulative axon loss and grey matter atrophy seen in chronic MS ultimately results in neurodegeneration, progressive disability, and cognitive deficits.

A combination of environmental and genetic factors converges into disease progression. MS is an autoimmune disease in which aberrant T-cell and B-cell activation of the adaptive response triggers the tissue-damaging inflammatory response.

The inflammatory response primarily involves degradation of the myelin layers that wrap the axons of the white matter, pathogenic multifocal neuroinflammation, and build-up of demyelinated plaques. These alterations derail the neuronal communication within the CNS and, importantly, between the brain and the peripheral organs.

The specific neuroinflammation in MS is initiated and reinforced by humoral factors, mostly autoreactive antibodies and infiltration of myelin-autoreactive HLA-DR15 class II-restricted CD4-positive helper T-cells of the helper type 1 (Th1) and helper Type 2 (Th17) subclasses. At later stages, this is followed by the CD8-positive effector of the T-and B-cells involvement via the compromised BBB [9].

The neuroinflammatory response in MS is modulated by the local presence of immunosuppressive FoxP3-positive regulatory T-cells (Tregs) favoring the progression of the recovery phase in recurrent MS.

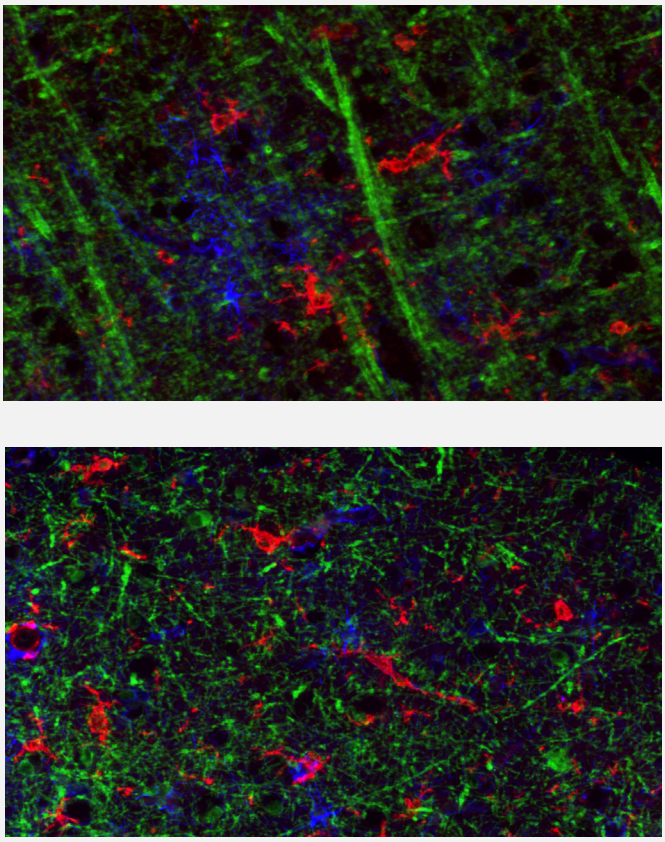


Figure 7
Myelinated processes in AD.
Multiplexed ICC-IF staining of human cerebral cortex from Alzheimer's disease patient (upper image) and a normal control (lower image). Microglial cells are visible in red (Anti-P2RY12, HPA014518), astrocytes in blue (Anti-GFAP, AMAb91033), and myelinated processes in green (Anti-MBP, AMAb91062).

The infiltrating B-cells have antigen-presenting capability and regulate the concerted immune responses by producing pro-and anti-inflammatory cytokines and local production of myelin autoreactive antibodies.

Moreover, in demyelinated brain lesions burdened by pathogenic B-cells, therapeutic, B-cell depleting antibodies against the B-cell marker CD20 has shown promising clinical results.

The absence of oligodendrocyte renewal in MS-patients suggests that remyelination is absent or very rare, and if so, regenerate by the action of mature pre-occurring oligodendrocytes.

EAE, the Animal Model to Study MS

Experimental autoimmune encephalomyelitis, sometimes referred to as experimental allergic encephalomyelitis (EAE), is the established and standardized experimental animal model for studying acute and remission-relapsing phases of human MS [10].

EAE is an antigen-dependent autoimmune model driven by myelin oligodendrocyte glycoprotein (MOG) -reactive Th1/Th17 cells, perivascular reactivation, and a subsequent inflammatory cascade leading to demyelination and chronic disease progression. The inflammatory cascade, in turn, leads to axonal degeneration and the development of neuropathological features similar to human MS, including optic nerve pathology.

Different mammalian species such as mice, rats, and primates are a suitable animal model to study EAE. Although EAE strongly mimics the human MS, it fails to cover all aspects of the human condition.

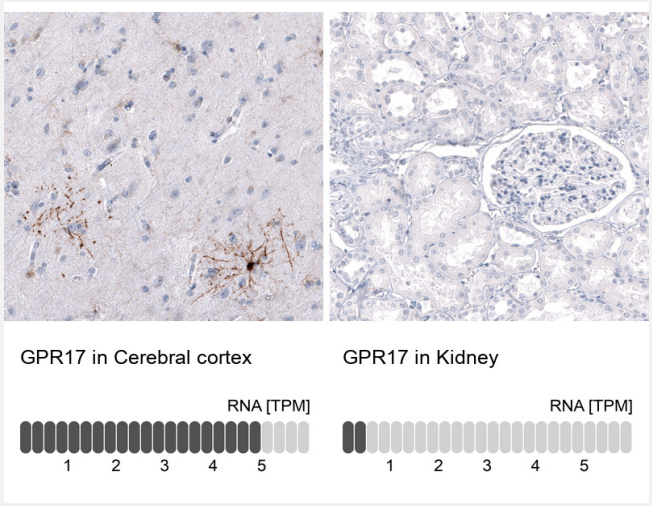


Figure 8
Oligodendrocytes in human cortical tissue.
IHC of human cerebral cortex (left) and kidney (right) tissues using Anti-GPR17 monoclonal antibody (AMAb91624), Oligodendrocytes in the cerebral cortex are visible in brown.

The orthogonal enhanced validation of the Anti-GPR17 antibody shows the corresponding GPR17 RNA-seq data (RNA-TPM) for the same tissues. Note that no GPR17 staining is visible in the kidney tissue according to the RNA levels.

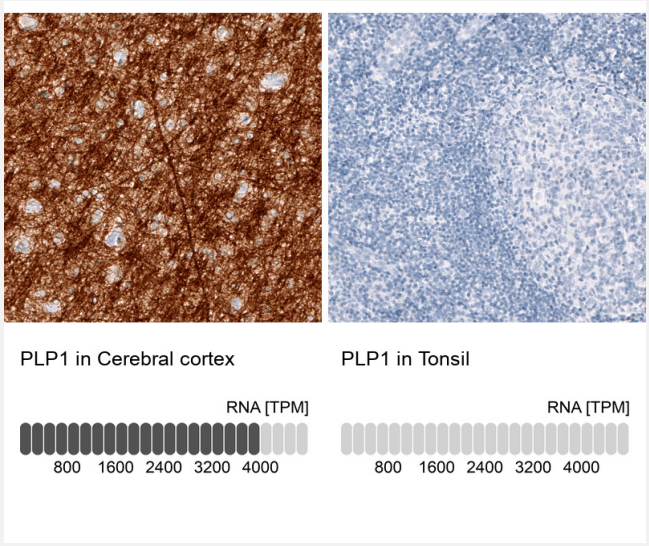


Figure 9
Myelinated axons in human cortical tissue.
IHC of human cerebral cortex (left) and tonsil (right) tissues using Anti-PLP1 monoclonal antibody (AMAb91639). Cortical myelinated axons are visible in brown.

The orthogonal enhanced validation of the Anti-PLP1 antibody shows the corresponding PLP1 RNA-seq data (RNA-TPM) for the same tissues. Note that no PLP1 staining is visible in the tonsil tissue according to the RNA levels.

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Table 2
PrecisA™ monoclonals

Product Name	Product Number	Validated Applications	Isotype
Anti-AGER	AMAb91634	IHC*	IgG2a
Anti-AGER	AMAb91635	IHC*	IgG1
Anti-AIF1	AMAb91671	IHC	IgG2b
Anti-AIF1	AMAb91672	IHC	IgG1
Anti-AMIGO3	AMAb91677	WB	IgG1
Anti-ANO2	AMAb91641	IHC	IgG1
Anti-ANO2	AMAb91642	IHC	IgG1
Anti-CD163	AMAb91646	IHC*,WB	IgG1
Anti-CD163	AMAb91648	IHC*	IgG1
Anti-CRYAB	AMAb91661	IHC*, WB, ICC-IF	IgG1
Anti-CDH1	AMAb90862	IHC*, WB*, ICC-IF	IgG2b
Anti-CRYAB	AMAb91662	IHC* WB	IgG2b
Anti-CXCL13	AMAb91629	IHC*	IgG1
Anti-DYSF	AMAb91667	IHC*, WB	IgG1
Anti-FUS	AMAb90549	IHC*, WB*, ICC-IF	IgG1
Anti-GAP43	AMAb91664	IHC*	IgG2b
Anti-GAP43	AMAb91665	IHC*	IgG1
Anti-GAP43	AMAb91666	IHC*	IgG2a
Anti-GPR17	AMAb91624	IHC*, WB	IgG1
Anti-GZMB	AMAb91650	IHC*	IgG1
Anti-HLA-DRA	AMAb91673	IHC*, WB	IgG1
Anti-HLA-DRA	AMAb91674	IHC*, WB	IgG2a
Anti-HLA-DRA	AMAb91675	IHC*, WB	IgG2b
Anti-IL17A	AMAb91615	IHC	IgG1
Anti-IL17RA	AMAb91617	WB	IgG2b
Anti-IL17RA	AMAb91619	WB	IgG1
Anti-IL7	AMAb91684	ICC-IF	IgG1
Anti-ITGA4	AMAb91699	WB	IgG2b
Anti-ITGB8	AMAb91467	WB, ICC-IF	IgG1
Anti-LAMC2	AMAb91098	IHC*, WB*, ICC-IF	IgG1
Anti-LAMA4	AMAb91134	IHC, WB	IgG1
Anti-MS4A1	AMAb91636	IHC*, WB	IgG2a
Anti-OPALIN	AMAb91685	IHC*	IgG1
Anti-OPALIN	AMAb91686	IHC*	IgG2a
Anti-PARP1	AMAb90959	IHC, WB*, ICC-IF	IgG1
Anti-PBRM1	AMAb90690	IHC*, WB*	IgG1
Anti-PLA2R1	AMAb90772	IHC, WB*, ICC-IF	IgG1
Anti-PLP1	AMAb91639	IHC*	IgG1
Anti-RGMA	AMAb91702	WB	IgG2b
Anti-S100A8	AMAb91689	IHC*	IgG1
Anti-S100A9	AMAb91690	IHC*	IgG2a
Anti-SERPINA3	AMAb91655	IHC*	IgG1
Anti-SNAI1	AMAb91215	IHC*, ICC-IF	IgG1
Anti-SATB2	AMAb90635	IHC*, WB	IgG1
Anti-SATB2	AMAb90682	IHC, WB, ICC-IF	IgG1
Anti-SOX4	AMAb91380	IHC, ICC-IF	IgG1
Anti-SPP1	AMAb91653	IHC*	IgG1
Anti-TBX19	AMAb91409	IHC	IgG1
Anti-TH	AMAb91112	IHC	IgG1
Anti-TLR2	AMAb91631	WB	IgG1
Anti-TRPM4	AMAb91693	IHC*, WB, ICC-IF	IgG1
Anti-VGAT	AMAb91043	IHC	IgG1
Anti-VGLUT1	AMAb91041	IHC*, WB	IgG2b

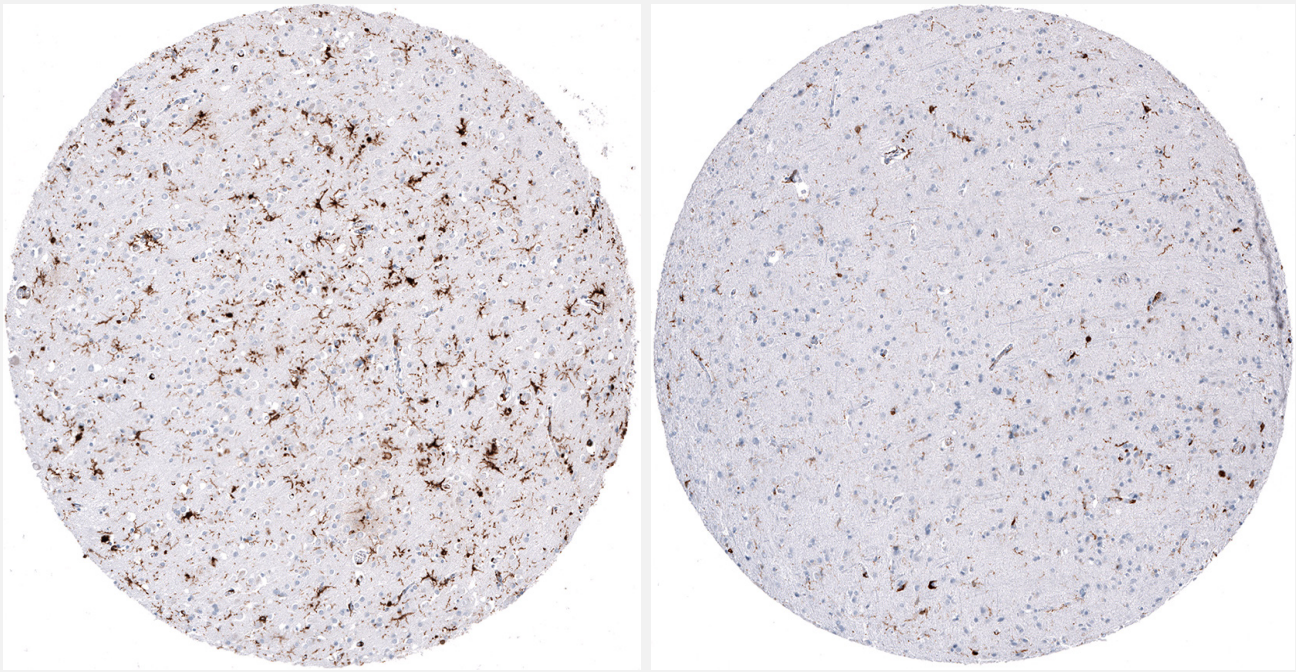
Table 3
Triple A™ polyclonals

Product Name	Product Number	Validated Applications	Seq Identity mouse / rat
Anti-AGER	HPA064436	WB, ICC-IF	41 / 41%
Anti-AIF1	HPA049234	IHC*	84 / 84%
Anti-AIF1	HPA062949	ICC-IF	86 / 83%
Anti-ANO2	HPA057499	IHC	84 / 85%
Anti-CD163	HPA046404	IHC*, ICC-IF	51 / 30%
Anti-CD163	HPA051974	IHC*	75 / 83%
Anti-CRYAB	HPA057100	IHC*	98 / 98%
Anti-DYSF	HPA017071	IHC*, ICC-IF	96 / 96%
Anti-DYSF	HPA021945	IHC*	88 / 88%
Anti-GAP43	HPA013392	IHC*, ICC-IF	88 / 86%
Anti-GAP43	HPA015600	IHC*, ICC-IF	71 / 70%
Anti-GPR17	HPA029766	IHC*	92 / 92%
Anti-GZMB	HPA003418	IHC*, WB*	63 / 63%
Anti-HLA-DRA	HPA050162	IHC*	54 / 69%
Anti-HLA-DRA	HPA053176	IHC*	41 / 83%
Anti-IL7	HPA019590	IHC	46 / 46%
Anti-ITGA4	HPA074961	ICC-IF	90 / 89%
Anti-MS4A1	HPA014341	IHC*, WB*, ICC-IF	73 / 77%
Anti-MS4A1	HPA014391	IHC*, WB*	61 / 64%
Anti-OPALIN	HPA014372	IHC	72 / 75%
Anti-PLP1	HPA004128	IHC*	100 / 100%
Anti-RGMA	HPA039880	ICC-IF	96 / 96%
Anti-RGMA	HPA044222	ICC-IF	87 / 87%
Anti-S100A8	HPA024372	IHC*, WB*, ICC-IF	56 / 60%
Anti-SERPINA3	HPA000893	IHC*, WB	61 / 61%
Anti-SERPINA3	HPA002560	IHC*, WB	60 / 59%
Anti-SPP1	HPA027541	IHC*, WB, ICC-IF	56 / 53%
Anti-TRPM4	HPA041169	IHC*, ICC-IF	93 / 91%

* Products with **enhanced validation** for indicated application.

Enhanced Validation

In addition to the extensive validation and characterization always performed for our antibodies, we conduct application-specific **enhanced validation** to secure the antibody specificity in a defined context.



Immunohistochemical staining of human cerebral cortex from Alzheimer's disease (left) and normal control (right) tissues using Anti-HLA-DRA monoclonal antibody (AMAb91674). A strong positivity for microglial cells is visible in brown in Alzheimer's disease but not healthy control tissues.

PrecisA™ Monoclonals are mouse monoclonal primary antibodies developed against a number of carefully selected targets. Clones are selected to recognize only unique non-overlapping epitopes and isotypes.

Triple A™ Polyclonals are rabbit polyclonal primary antibodies developed within the Human Protein Atlas project. IHC characterization data from 44 normal and 20 cancer tissues is available on the Human Protein Atlas portal.

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