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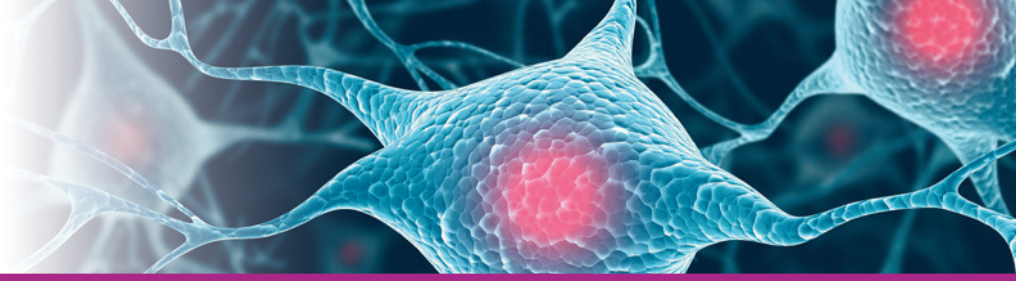

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2nd Edition

Neuroscience Research

Focus: Neuroinflammation & Neuronal Diseases

Microtubules and Neurodegeneration

Neurodegeneration refers to the progressive loss of structure and/or function of neurons often beginning at the synaptic distal ends of axons. Neurodegenerative diseases exhibit a broad range of clinical symptoms, which share several common pathological features. Prominent cellular features include the toxic aggregation of proteins that inhibit the protein quality control and the ubiquitin-proteasome machinery of the neuron, inflammatory responses, impaired ER calcium homeostasis, increased oxidative stress and microtubule defects.

In neurons, microtubules, actin filaments and neurofilaments compose the cytoskeleton, maintaining cell polarity, architecture and morphology. Microtubules (MTs) are highly dynamic polymers formed of the tubulin α and β heterodimers. The GTP bound to α-tubulin is stable and plays a structural function in the microtubule. The GTP bound to β-tubulin may be hydrolyzed to GDP shortly after assembly into MTs, with GDP-tubulin being more prone to depolymerization.

MTs are polar structures with a labile plus end (favored for assembly and disassembly) and a stable minus end (less favored for these dynamics). Regulation of MTs polymerization is controlled by microtubule associated proteins, post-translational modifications of tubulin α and β, microtubule destabilizers (severing enzymes of AAA-ATPase type) and signaling molecules.

Mounting evidence suggests that deregulation of neuronal cytoskeleton function constitutes a key insult during the pathogenesis of nervous system diseases. Microtubule mass is diminished and corruption of the microtubule polarity patterns (i.e. appearance of too many mal-oriented microtubules) and microtubule-mediated transport is observed during neurodegenerative diseases, including Amyotrophic Lateral Sclerosis, Alzheimer, Hereditary Spastic Paraplegia, Parkinson's disease and others.

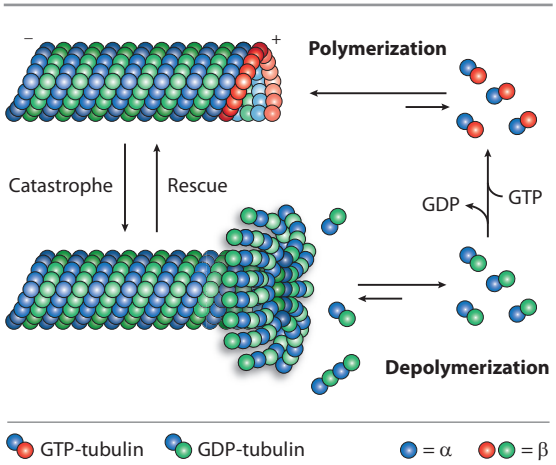

FIGURE: Microtubule dynamic instability. Polymerizing and rapidly depolymerizing polymers coexist at steady state.

SELECTED REVIEW ARTICLES

The tubulin code: molecular components, readout mechanisms, and functions: C. Janke; J. Cell. Biol. **206,** 461 (2014) • Microtubules in health and degenerative disease of the nervous system: A.J. Matamoros & P.W. Baas; Brain. Res. Bull. **(Epub ahead of print)** (2016) • The emerging role of the tubulin code: From the tubulin molecule to neuronal function and disease: S. Chakraborti, et al.; Cytoskeleton **(Epub ahead of print)** (2016)

CONTENTS

The Tubulin Code: Post-translational Modifications of Tubulins

Post-translational modifications (PTMs) are highly dynamic and often reversible processes where protein functional properties are altered by addition of a chemical group or another protein to its amino acid residues. As key cytoskeletal proteins with roles in neuronal development, growth, motility and intracellular trafficking, tubulins and microtubules (MTs) are major substrates for PTMs. They include tyrosination/detyrosination, Δ2-tubulin formation, **acetylation**, phosphorylation, polyamination, ubiquitination, **polyglutamylation** and glycylation (**see Figure**). Most of these PTMs preferentially take place on tubulin subunits already incorporated into microtubules.

PTMs are involved in fine-tuning of interactions between microtubules and different MT-interacting proteins. Most axonal microtubules are detyrosinated and further labeled with acetate and polyglutamate marks. By contrast, the unstable microtubules are enriched in carboxy-terminal tyrosination and devoid of glutamate tails. Detyrosination and polyglutamylation of MTs can selectively modulate the affinities and motility of molecular motors. Acetylation seems to control intracellular transport by regulating the traffic of kinesin motors. Microtubules PTMs deregulation have impact on neuronal development and diseases.

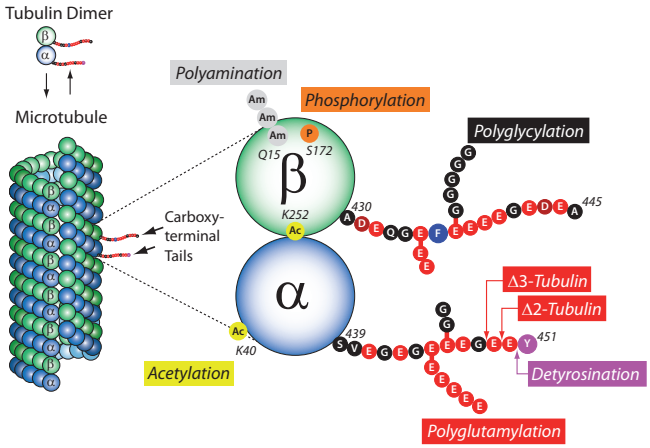

FIGURE: Tubulin PTM Overview. *Adapted from C. Janke; J. Cell. Biol.* ***206,*** *461 (2014)*

UNIQUE

Validated Post-translational Modification-specific Antibodies

ANTIBODIES	PID	SIZE	ISOTYPE/SOURCE	APPLICATION
anti-α-Tubulin (acetylated), mAb (TEU318)	AG-20B-0068	100 µg	Mouse IgG1	ICC, WB
anti-Polyglutamylation Modification, mAb (GT335)	AG-20B-0020	100 µg	Mouse IgG1κ	EM, ICC, IP, WB
anti-Polyglutamylation Modification, mAb (GT335) (Biotin)	AG-20B-0020B	100 µg	Mouse IgG1κ	ICC, IP, WB
anti-Polyglutamate chain (polyE), pAb (IN105)	AG-25B-0030	50 µg	Rabbit	ICC, WB

Recombinant Microtubule-target Antibodies

ANTIBODIES	PID	SIZE	ISOTYPE/SOURCE	APPLICATION	SPECIES
anti-Tubulin-GTP, mAb (rec.) (MB11) UNIQUE	AG-27B-0009	100 µg	Human IgG2λ	ICC	Hu, Ms, Rt, Dr
anti-α-Tubulin, mAb (rec.) (F2C)	AG-27B-0005	100 µg	Human IgG2λ	ICC, WB	Hu, Ms, Bv
anti-α-Tubulin, mAb (rec.) (F2C) (ATTO 488)	AG-27B-0005TD	100 µg	Human IgG2λ	ICC	Hu, Ms, Bv
anti-β-Tubulin, mAb (rec.) (S11B)	AG-27B-0008	100 µg	Rabbit	ELISA, ICC, WB	Hu, Ms, Rt, Pg, Dr, Mk

Rab1-GTP and Rab6-GTP Specific Antibodies

Rab proteins, members of the small GTPase superfamily, are important regulators of vesicle transport via interactions with effector proteins and motor proteins. Rab1 and 6 are implicated in anterograde and retrograde trafficking in the secretory pathway. Recently, Rab1 has been shown to be involved in **autophagy** by helping the formation of the pre-autophagosomal isolation membrane (phagophore). Rab6 also functions as modulator of the unfolded protein response (UPR), helping the recovery from an ER stress insult. Rab6 is upregulated in Alzheimer's disease brain.

ANTIBODIES	PID	SIZE	ISOTYPE/SOURCE	APPLICATION	SPECIES
anti-Rab1-GTP, mAb (rec.) (ROF7)	AG-27B-0006	100 µg	Human IgG2λ	ICC, IP	Hu, Ms, Rt, Dg
anti-Rab6-GTP, mAb (rec.) (AA2)	AG-27B-0004	100 µg	Human IgG2λ	ICC, WB	Hu, Ms, Dr
anti-Rab6-GTP, mAb (rec.) (AA2) (ATTO 488)	AG-27B-0004TD	100 µg	Human IgG2λ	ICC	Hu, Ms, Dr

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APPLICATIONS: FACS: Flow Cytometry; **FUNC:** Functional Application; **ICC:** Immunocytochemistry; **IHC:** Immunohistochemistry **IP:** Immunoprecipitation; **WB:** Western blot
SPECIES: Bv = Bovine; **Dg** = Dog; **Dr** = Drosophila; **Hu** = Human; **Mk** = Monkey; **Ms** = Mouse; **Pg** = Pig; **Rt** = Rat; **Rb** = Rabbit; **Prm** = Primate

Microtubule Stabilization – Notch & Small Molecule Modulators

Several studies show that the morphology of the neuron can be influenced by **microtubule** and **actin filament cytoskeleton** dynamics, and that neurite outgrowth can be modulated with stabilizing and destabilizing agents. Activation of the **Notch signaling pathway** results in stabilization of microtubules leading to regulation of axonal morphology, with thicker neurites, fewer branches and loss of synaptic varicosity. This Notch-dependent stabilization of microtubules is likely due to increase in acetylation and polyglutamylation of α-tubulins, both of which are markers of stable microtubules.

LIT: Notch signalling in adult neurons: a potential target for microtubule stabilization: S.A. Bonini, et al.; Ther. Adv. Neurol. Disord. **6,** 375 (2013) • Microtubule-stabilizing agents as potential therapeutics for neurodegenerative disease: K.R. Brunden, et al.; Bioorg. Med. Chem. **22,** 5040 (2014)

BULK

Ferulenol (Stimulator of tubulin polymerization)
AG-CN2-0011 — 1 mg | 5 mg | 10 mg

Paclitaxel (Microtubule assembly stabilizer)
AG-CN2-0045 — 1 mg | 5 mg | 25 mg | 100 mg

Jasplakinolide (high purity)
(Potent inducer of actin polymerization and stabilization)
AG-CN2-0037 — 50 µg | 100 µg

Visit www.adipogen.com for a Comprehensive Panel of Small Molecule Microtubule Modulators & Validated Notch Pathway Reagents!

LATEST INSIGHT

Angiopoietin-2 in Cerebral Cavernous Malformations (CCMs)

H.J. Zhou, et al. (2016) recently found that enhanced secretion of ANGPT2 in endothelial cells contributes to the progression of CCM disease and is associated with destabilized endothelial cell junctions, enlarged lumen formation and endothelial cell pericyte dissociation. **Treatment with an ANGPT2-neutralizing antibody** normalizes the defects in the brain and retina caused by endothelial-cell-specific CCM3 deficiency.

LIT: Endothelial exocytosis of angiopoietin-2 resulting from CCM3 deficiency contributes to cerebral cavernous malformation: H.J. Zhou, et al.; Nat. Med. **22,** 1033 (2016)

NEW

Potent ANGPT2 Blocking Antibodies

anti-Angiopoietin-2, mAb (rec.) (blocking) (Angy-2-1) (preservative free)
AG-27B-0016PF — 100 µg | 500 µg | 1mg

anti-Angiopoietin-2 (human), mAb (rec.) (blocking) (Angy-1-4) (preservative free)
AG-27B-0015PF — 100 µg | 500 µg | 1mg

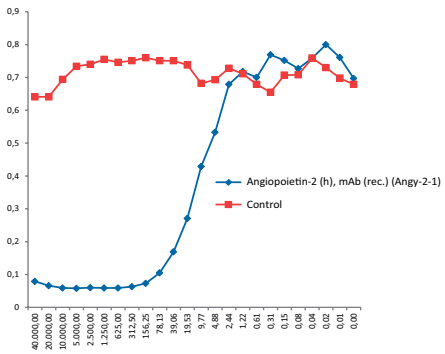

FIGURE: Binding of human Angiopoietin-2 to Tie-2 (human):Fc is inhibited by the antibody anti-Angiopoietin-2, mAb (rec.) (blocking) (Angy-2-1) (PF) (Prod. No. AG-27B-0016PF).Tie-2 (human):Fc was coated on an ELISA plate at 1µg/ml. Angy-2-1 or an unrelated mAb (recombinant) (Control) were added (starting at 40µg/ml with a twofold serial dilution) together with 20ng/µl of Angiopoietin-2 (human) (Prod. No. AG-40B-0114). After incubation for 1h at RT, the binding was detected using an anti-FLAG antibody (HRP).

Also Available:

Angiopoietin-2 (human) (rec.) — AG-40B-0114
Angiopoietin-2 (mouse) (rec.) — AG-40B-0131

Progranulin – Marker of Neuroinflammation

Progranulin (PGRN) is a cysteine-rich protein, that shows multifunctional biological activities, including major roles in cancer, inflammation, metabolic disease and neurodegeneration, especially as a valuable biomarker for Frontotemporal Lobar Degeneration (FTLD). In the brain, PGRN is primarily expressed in mature neurons and microglia. Absence of progranulin in microglia causes increased production and release of multiple cytokines, suggesting that PGRN regulates microglia activation. It is anticipated that PGRN affects microglial proliferation, recruitment, differentiation, activation and phagocytosis, suggesting that PGRN plays a central role in the regulation of neuroinflammatory responses. PGRN serves as an important "brake" to suppress excessive microglia activation in the aging brain by facilitating phagocytosis and endolysosomal trafficking in these cells. In neurons, PGRN i) enhances survival and neurite outgrowth through modulation of GSK-3β, ii) co-localizes in late endosomes and early lysosomes with the transmembrane protein TMeM106B, iii) co-localizes with markers such as BDNF along axons, iv) influences synaptic structure and function at synaptic and extra-synaptic sites, where it is secreted in an activity-dependent manner, and v) extracellular PGRN is endocytosed through the sortilin receptor and delivered to lysosomes. PGRN has also anti-inflammatory roles through inhibition of two Tumor Necrosis Factor Receptor family members (TNFR and DR3).

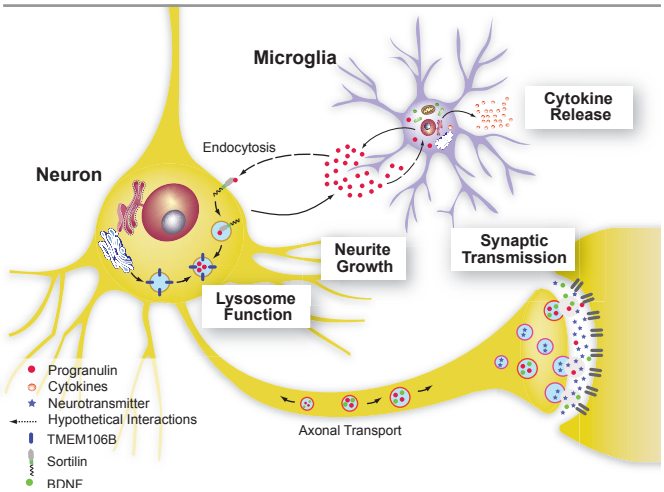

FIGURE: Potential functions of progranulin in the brain.

SELECTED REVIEWS: Progranulin: at the interface of neurodegenerative and metabolic diseases: A.D. Nguyen, et al.; Trends Endocrinol. Metab. **24,** 597 (2013) • Progranulin in neurodegenerative disease: T.L. Petkau & B.R. Leavitt; TINS **37,** 388 (2014) • Progranulin deficiency promotes circuit-specific synaptic pruning by microglia via complement activation: H. Lui, et al.; Cell **165,** 921 (2016)

Standard Progranulin ELISA Kits

Progranulin (human) ELISA Kit	AG-45A-0018Y
Progranulin (mouse) ELISA Kit	AG-45A-0019Y
Progranulin (rat) ELISA Kit	AG-45A-0043Y

Tag-free Progranulins

- **Higher activity compared to tagged Progranulins**
- **Suitable for *in vitro* and *in vivo* studies**
- **Reflects the native sequence with no additional amino acids**
- **Affinity purified**
- **Low endotoxin levels (<0.01EU/µg)**

Progranulin (human) (rec.) (untagged)
AG-40A-0188Y — 10 µg | 50 µg | BULK

Progranulin (mouse) (rec.) (untagged)
AG-40A-0189Y — 10 µg | 50 µg | BULK

Progranulin Antibodies & Tagged Proteins

ANTIBODIES	PID	SIZE	ISOTYPE/SOURCE	APPLICATION	SPECIES
anti-Progranulin (human), mAb (PG359-7)	AG-20A-0052	100 µg	Mouse IgG1κ	IHC, IP, WB	Hu
anti-Progranulin (human), pAb	AG-25A-0112	100 µg	Guinea pig	IHC, WB	Hu
anti-Progranulin (mouse), mAb (PG319-1)	AG-20A-0077	50 µg | 100 µg	Rat IgG2a κ	WB	Ms
anti-Progranulin (mouse), pAb	AG-25A-0093	100 µg	Rat	WB	Ms

PROTEINS	PID	SIZE	SOURCE	ENDOTOXIN	SPECIES
Progranulin (human) (rec.)	AG-40A-0068Y	10 µg | 50 µg	HEK293 Cells	<0.01EU/µg	Hu
Progranulin (rat) (rec.)	AG-40A-0194	10 µg | 50 µg	HEK293 Cells	<0.1EU/µg	Rt

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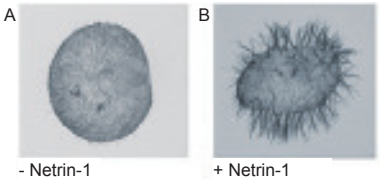
APPLICATIONS: FACS: Flow Cytometry; **FUNC:** Functional Application; **ICC:** Immunocytochemistry; **IHC:** Immunohistochemistry **IP:** Immunoprecipitation; **WB:** Western blot
SPECIES: Bv = Bovine; **Dg** = Dog; **Dr** = Drosophila; **Hu** = Human; **Mk** = Monkey; **Ms** = Mouse; **Pg** = Pig; **Rt** = Rat; **Rb** = Rabbit; **Prm** = Primate

Netrin-1 – Neuron Guidance Factor Involved in iPS Regulation

Netrin-1 is a guidance molecule that triggers either attraction or repulsion effects on migrating axons of neurons, interacting with the receptors **DCC** or **UNC5** (A to D). It has been proposed that DCC and UNC5 are dependence receptors that, in the absence of netrin-1, promote apoptosis. This pro-apoptotic activity requires initial caspase cleavage of the receptor's intracellular domain. Netrin-1 is therefore a pro-survival factor acting by blocking cell death induced by its unbound receptors. Netrin-1 protects neurons from death during development and favors tumor epithelial cells survival in some types of cancers. It interacts with the orphan amyloid precursor protein (APP), a protein component of the amyloid plaques that are associated with Alzheimer's disease (AD). Netrin-1 also inhibits remyelination of neurons in Multiple Sclerosis (MS) (and other progressive demyelinating diseases) by inhibiting oligodendrocyte precursor migration. Recently, Netrin-1 has been described to be the **5th Element of classical iPS cell factors**. Netrin-1 functions in protecting embryonic stem cells from apoptosis and addition of recombinant Netrin-1 improves the generation of mouse and human iPS cells (induced Pluripotent Stem Cells).

REVIEWS: Netrin-1 in the developing enteric nervous system and colorectal cancer: S.Y. Ko, et al.; Trends Mol. Med. **18,** 544 (2012) • Netrin-1 regulates somatic cell reprogramming and pluripotency maintenance: D. Ozmadenci, et al.; Nat. Commun. **6,** ID7398 (2015)



Picture courtesy of Dr. Véronique Corset, Prof. Patrick Mehlen lab, Centre Léon Bérard, Lyon

FIGURE: Netrin-1 (human):Fc (human) (rec.) (Prod. No. AG-40B-0075) induces outgrowth of the commisural axon.

METHOD: Dorsal spinal cords were dissected out from E13 rat embryos and cultured in collagen matrix in the presence or absence of netrin-1 (250ng/ml). Axons were then stained with an anti-β-tubulin antibody.

UNIQUE Biologically Active Human Netrin-1

PROTEINS	PID	SIZE	SOURCE	ENDOTOXIN	SPECIES
Netrin-1 (human) (rec.)	AG-40B-0040	10 µg | 3 x 10 µg | 100 µg	HEK293 Cells	<0.01EU/µg	Hu, Ms, Rt
Netrin-1 (human):Fc (human) (rec.)	AG-40B-0075	10 µg | 3 x 10 µg | 100 µg	HEK293 Cells	<0.1EU/µg	Hu, Ms, Rt
UNC5B (human):Fc (human) (rec.)	AG-40B-0037	50 µg | 3 x 50 µg	HEK293 Cells	<0.1EU/µg	Hu, Ms

NEW Potent Netrin-1 Blocking Antibody

ANTIBODY	PID	SIZE	ISOTYPE/SOURCE	APPLICATION	SPECIES
anti-Netrin-1 (human), mAb (rec.) (blocking) (2F5) (preservative free)	AG-27B-0018PF	100 µg | 500 µg	Human IgG2	ELISA, FUNC	Hu, Ms

LIT: Epidermal Growth Factor Receptor-Dependent Mutual Amplification between Netrin-1 and the Hepatitis C Virus: M.L. Plissonnier, et al.; PLoS Biol. **14,** e1002421 (2016) • Targeting netrin-1/DCC interaction in diffuse large B-cell and mantle cell lymphomas: T. Broutier, et al.; EMBO Mol. Med. **8,** 96 (2016)

LATEST INSIGHT

Pathological α-Synuclein Transmission is initiated by the Receptor LAG-3

Parkinson's Disease (PD) is partially caused by amplification of a pathological α-synuclein that spreads from cells to cells in the brain. Recently, X. Mao, et al. (2016) reported that α-synuclein transmission and toxicity is initiated by binding to LAG-3 followed by endocytosis. Blocking this binding with an antibody to LAG-3 can reduce the toxicity of the α-synuclein. This new discovery could help the future development of therapeutic drugs to slow down PD.

LIT: Pathological α-synuclein transmission initiated by binding lymphocyte-activation gene 3: X. Mao, et al.; Science **353,** 1513 (2016)

ANTIBODIES	PID	SIZE	ISOTYPE/SOURCE	APPLICATION	SPECIES
anti-LAG-3 (human), mAb (blocking) (17B4)	AG-20B-0012	100 µg	Mouse IgG1	FACS, FUNC, ICC, IHC, IP, WB	Hu
anti-LAG-3, mAb (blocking) (11E3)	AG-20B-0011	100 µg	Mouse IgG1	ELISA, FUNC, ICC, IHC, IP, WB	Hu, Mk

PROTEINS	PID	SIZE	SOURCE	ENDOTOXIN	SPECIES
LAG-3 (human):Fc (human) (rec.)	AG-40B-0031	50 µg	CHO cells	<0.1EU/µg	Hu
LAG-3 (mouse):Fc (mouse) (rec.)	AG-40B-0039	50 µg	CHO cells	<1EU/µg	Hu, Mk
Synuclein-α (human) (rec.) (His)	AG-40T-0388	500 µg	E.coli	n.a.	Hu

Inflammasomes & Neuroinflammation/Neurodegeneration

Neuroinflammation is an **innate immune response in the CNS** (Central Nervous System) against harmful and irritable stimuli such as pathogens, metabolic toxic waste or chronic mild stress that occurs in response to trauma, infections and/or neurodegenerative diseases. The main cell types contributing to the innate immune response are microglia, trafficking macrophages and astrocytes. These cells constantly survey the proximal environment through pattern-recognition receptors (PRRs) such as Toll-like receptors (TLRs), scavenger receptors (SRs) and **NOD-like receptors (NLRs)** (e.g. inflammasome complexes). These NLR receptors recognize not only exogenous pathogen-associated molecular patterns (PAMPs) but also endogenous modified molecules called damage-associated molecular patterns (DAMPs). After activation and release of immune molecules (e.g. cytokines), the innate immune system launches inflammatory and regulatory responses in order to counteract infection, injury and maintenance of tissue homeostasis. Although the evolutionary function is neuroprotective, innate immune responses can also promote immunopathology when they are excessive (e.g. chronic neuroinflammation). During chronic activation, the sustained exposure of neurons to pro-inflammatory mediators can cause neuronal dysfunction and contribute to cell death. As chronic neuroinflammation is observed at relatively early stages of neurodegenerative diseases, targeting the mechanisms that drive this process may be useful for diagnostic and therapeutic purposes.

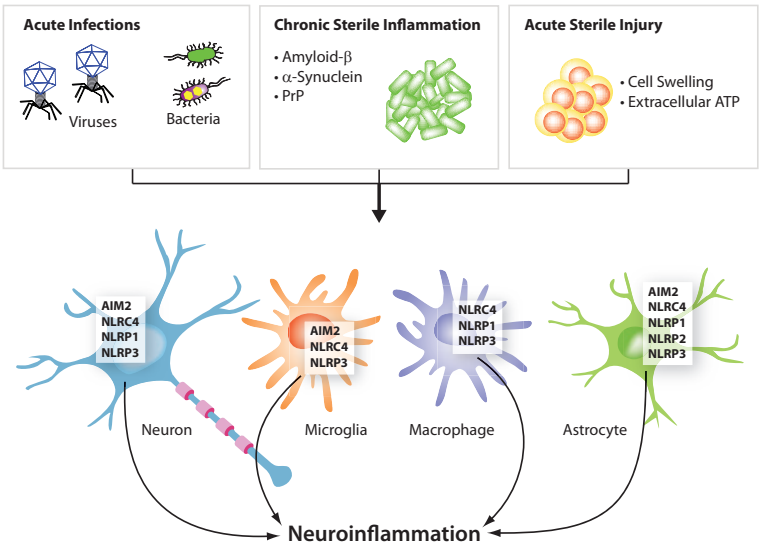


FIGURE: Selected activation factors, inflammasome complexes and target cells in the CNS.

Neuroinflammation is **mediated by protein complexes known as inflammasomes**. Inflammasomes function as intracellular sensors for infectious agents as well as for host-derived danger signals that are associated with neurological diseases, including meningitis, stroke and Alzheimer's disease (AD). The inflammasome can be activated in the CNS under diverse conditions that trigger inflammation, including acute infection (e.g. viruses, bacteria), chronic sterile inflammation (e.g. misfolded proteins such as amyloid-β, α-synuclein and prion protein) and acute sterile injury (ATP excess) (**see Figure**). Assembly of inflammasomes (NLRP1/2/3 and NLRC4/IPAF) activates pro-inflammatory caspase-1, which then cleaves the precursor forms of pro-inflammatory cytokines IL-1β and IL-18 into their active forms. These pro-inflammatory cytokines promote a variety of innate immune processes associated with infection, inflammation and autoimmunity, and play an instrumental role in the onset of neuroinflammation and subsequent occurrence of neurodegenerative diseases, cognitive impairment and dementia. NLRP1/2/3 and NLRC4/IPAF inflammasomes may also have a role in the etiologies of depression, Alzheimer's disease (AD) and in metabolic disorders, such as Type II diabetes, obesity and cardiovascular diseases that have been shown to be co-morbid with psychiatric illnesses.

SELECTED REVIEWS: Inflammasomes in the CNS: J.G. Walsh, et al.; Nat. Rev. Neurosci. **15,** 84 (2014) • Innate immune activation in neurodegenerative disease: M.T. Heneka, et al.; Nat. Rev. Immunol. **14,** 463 (2014) • Inflammation in neurodegenerative diseases-an update: S. Amor, et al.; Immunol. **142,** 151 (2014)

THE STANDARD

NLRP3 Antibody

anti-NLRP3/NALP3, mAb (Cryo-2)

AG-20B-0014-C100 100 µg

Clone	Cryo-2
Isotype	Mouse IgG2b
Immunogen	Recombinant mouse NLRP3/NALP3 (pyrin domain/aa 1-93)
Application	ICC, IHC, IP, WB (1µg/ml) (see online protocol)
Specificity	Recognizes human and mouse NLRP3/NALP3.

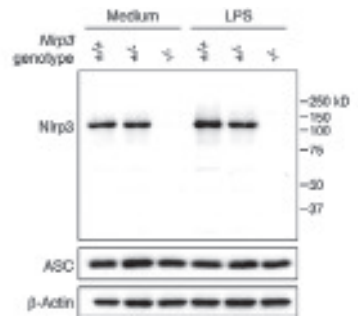


FIGURE: Mouse NLRP3 is detected in mouse macrophages using the monoclonal antibody to NLRP3 (Cryo-2) (Prod. No. AG-20B-0014).

METHOD: Cell extracts from mouse macrophages (BMDMs) WT (+/+) (lane 1), NLRP3 +/- (lane 2) or NLRP3 -/- (lane 3) with or without treatment with LPS (50ng/ml) for 3h, were separated by SDS-PAGE under reducing conditions, transferred to nitrocellulose and incubated with the mAb to NLRP3 (Cryo-2) (1µg/ml). Proteins are visualized by a chemiluminescence detection system.

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APPLICATIONS: FACS: Flow Cytometry; **FUNC:** Functional Application; **ICC:** Immunocytochemistry; **IHC:** Immunohistochemistry **IP:** Immunoprecipitation; **WB:** Western blot
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THE STANDARDS FROM THE EXPERTS & VALIDATED BY KEY LABORATORIES!

Immunoblotting for Activated/Cleaved Caspase-1

anti-Caspase-1 (p20) (mouse), mAb (Casper-1)

AG-20B-0042-C100		100 µg
AG-20B-0042B-C100	Biotin	100 µg

Clone Casper-1
Isotype Mouse IgG1
Immunogen Recombinant mouse caspase-1
Application WB (1µg/ml) (see online protocol), IHC (PS), IP
Specificity Recognizes endogenous full-length and activated (p20 fragment) mouse caspase-1.

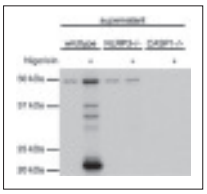


FIGURE: Mouse caspase-1 (p20) is detected by immunoblotting using anti-Caspase-1 (p20) (mouse), mAb (Casper-1) (Prod. No. AG-20B-0042).

METHOD: Caspase-1 was analyzed by Western blot in cell extracts and supernatants of differentiated bone marrow-derived dendritic cells (BMDCs) from wild-type, NLRP3-/- and caspase-1-/- mice activated or not by 5µM nigericin (Prod. No. AG-CN2-0020) for 30 min. Cell extracts and supernatants were separated by SDS-PAGE under reducing conditions, transferred to nitrocellulose and incubated with anti-Caspase-1 (p20) (mouse), mAb (Casper-1) (1µg/ml). Proteins were visualized by a chemiluminescence detection system.

anti-Caspase-1 (p20) (human), mAb (Bally-1)

AG-20B-0048-C100		100 µg
AG-20B-0048B-C100	Biotin	100 µg

Clone Bally-1
Isotype Mouse IgG1
Immunogen Recombinant human caspase-1
Application WB (1µg/ml) (see online protocol)
Specificity Recognizes endogenous full-length and activated (p20 fragment) human caspase-1.

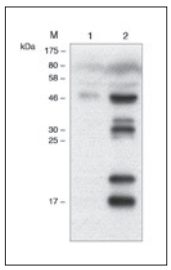


FIGURE: Human Caspase-1 (p20) is detected by immunoblotting using anti-Caspase-1 (p20) (human), mAb (Bally-1) (Prod. No AG-20B-0048).

METHOD: Caspase-1 was analyzed by Western blot in supernatants of THP1 cells differentiated for 3h with 0.5 µM PMA (Prod. No. AG-CN2-0010) and activated (lane 2) or not (lane 1) by 5 µM Nigericin for 1h (Prod. No. AG-CN2-0020). Supernatants (30µl) were separated by SDS-PAGE under reducing conditions, transferred to nitrocellulose and incubated with anti-Caspase-1 (p20) (human), mAb (Bally-1) (1µg/ml). Proteins were visualized by a chemiluminescence detection system.

Standard Inflammasomes Signaling Antibodies

PRODUCT NAME	PID	SIZE	SOURCE/ISOTYPE	APPLICATION	SPECIES
Nod-like Receptors (NLRs)					
anti-NAIP1/2/5 (mouse), mAb (Naipa-1)	AG-20B-0045	100 µg	Mouse IgG2bκ	WB	Ms
anti-NLRP1/NALP1 (human), pAb (AL176)	AG-25B-0005	100 µg	Rabbit	WB	Hu
Cytosolic DNA Sensor					
anti-AIM2 (human), mAb (3B10)	AG-20B-0040	100 µg	Mouse IgG1	ICC, WB	Hu
Signaling Antibodies					
anti-Asc [Pycard], pAb (AL177)	AG-25B-0006	100 µg	Rabbit	ICC, IHC (PS), IP, WB, FUNC (Blocking)	Hu, Ms
anti-Asc [Pycard], pAb (AL177) (preservative free)	AG-25B-0006PF	100 µg	Rabbit	ICC, IHC (PS), IP, WB, FUNC (Blocking)	Hu, Ms
anti-Asc, pAb (AL177) (ATTO 647N)	AG-25B-0006TS	100 µg	Rabbit	ICC, IHC (PS)	Hu, Ms
Cytosolic PAMPs Sensors					
anti-Caspase-4/11 (p20), mAb (Flamy-1)	AG-20B-0060	100 µg	Mouse IgG2bκ	IP, WB	Hu, Ms
anti-Caspase-4/11 (p20), mAb (Flamy-1) (Biotin)	AG-20B-0060B	100 µg	Mouse IgG2bκ	IP, WB	Hu, Ms

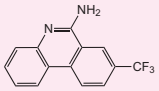
For a comprehensive Overview on Unique Inflammasome Reagents ask for AdipoGen®'s Inflammasome Signaling Brochure & Wallchart !

Lead Compounds for Neurodegenerative Diseases

Anti-Prion Agents – Protein Aggregation Inhibitors

6-Amino-8-trifluoromethylphenanthridine
AG-MR-C0031 — 1 mg | 5 mg | 25 mg

Formula: $C_{14}H_9F_3N_2$
MW: 262.2
CAS: 651055-83-3



6-Aminophenanthridine
AG-MR-C0029 — 1 mg | 5 mg | 25 mg

Chloroguanabenz . acetate
AG-MR-C0036 — 1 mg | 5 mg

Selective N- & P/Q-type Ca^{2+} Channel Agonist

GV-58
AG-MR-C0035 — 1 mg | 5 mg

Anti-Alzheimer's Disease (AD) Agent

Leucettine L41
AG-MR-C0023 — 1 mg | 5 mg | 25 mg

Alzheimer's Disease (AD) Accelerator

Aftin-4
AG-MR-C0014 — 1 mg | 5 mg | 25 mg

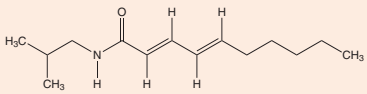
Aftin-5
AG-MR-C0015 — 1 mg | 5 mg | 25 mg

BULK

Model Compound for Sensory Studies

Pellitorine
AG-CN2-0009 — 1 mg | 5 mg

Formula: $C_{14}H_{25}NO$
MW: 223.4
CAS: 18836-52-7 **Source:** Synthetic



Tingling-inducing agent. Excellent stable model compound for sensory studies. Exerts same profile as the unstable compound hydroxy-α-sanshool.

Selected Receptor Agonists and Antagonists

PRODUCT NAME	ACTIVITY	PID	SIZE
epi-Aszonalenin A	Substance P inhibitor	AG-CN2-0163	1 mg | 5 mg
Bilobalide	GABA(A) receptor antagonist	AG-CN2-0026	10 mg | 50 mg
Cyclopenin	AChE inhibitor	AG-CN2-0134	1 mg | 5 mg
Debromohymenialdisine	Potential anti-Alzheimer's agent	AG-CN2-0068	100 µg
EM574 [Motilide]	Motilin receptor agonist	AG-CN2-0102	250 µg | 1 mg
Fulvic acid	Tau and Aβ aggregation inhibitor	AG-CN2-0135	1 mg | 5 mg
Hyperforin . DCHA	TRPC6 channel activator	AG-CN2-0008	500 µg | 1 mg
20-Hydroxyecdysone	GABA(A) receptor modulator	AG-CN2-0072	5 mg | 10 mg | 50 mg
MTEP	Potent mGluR5 antagonist	AG-CR1-0022	5 mg | 25 mg
NG 012	NGF potentiator	AG-CN2-0155	1 mg | 5 mg
Pseurotin D	Neuroleptic agent	BVT-0426	1 mg | 5 mg
Territrem B	AChE inhibitor	AG-CN2-0142	500 µg | 1 mg
SNC80	δ-Opioid receptor agonist	AG-CR1-0017	5 mg | 25 mg
Umbellulone	Selective TRPA1 activator	AG-CN2-0085	10 mg

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