Revision 2

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#30182

Mouse Reactive Alzheimer's Disease Model Microglia Phenotyping IF Antibody Sampler Kit

1 Kit (9 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Iba1/AIF-1 (E4O4W) XP® Rabbit mAb	17198	20 µl	17 kDa	Rabbit IgG
TMEM119 (E3E1O) Rabbit mAb	90840	20 µl		Rabbit IgG
β-Amyloid (D54D2) XP® Rabbit mAb	8243	20 µl	5 kDa	Rabbit IgG
GPNMB (E7U1Z) Rabbit mAb	90205	20 µl	90, 100 kDa	Rabbit IgG
CD11c (D1V9Y) Rabbit mAb	97585	20 µl	145 kDa	Rabbit IgG
HS1 (D5A9) XP® Rabbit mAb	3892	20 µl	80 kDa	Rabbit IgG
Cathepsin B (D1C7Y) XP® Rabbit mAb	31718	20 µl	44, 27, 24 kDa	Rabbit IgG
Cathepsin D (E179) Antibody	69854	20 µl	46, 43, 28 kDa	Rabbit
ASC/TMS1 (D2W8U) Rabbit mAb	67824	20 µl	22 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Mouse Reactive Alzheimer's Disease Model Microglia Phenotyping IF Antibody Sampler Kit provides an economical means of detecting microglia proteins in β-Amyloid mouse models of Alzheimer's disease (AD) by immunofluorescence and/or western blot. This kit includes enough primary antibodies to perform at least twenty IF-F tests or two western blot experiments per primary antibody.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. *Do not aliquot the antibodies.*

Background

Distinct microglial activation states have been identified using RNA-seq data from a vast array of neurological disease and aging models. In both mouse models of Alzheimer's disease (AD) and AD patients, unique microglia molecular signatures are associated with disease progression (1-3). AD progression is correlated with the extracellular deposition and accumulation of the released Aβ fragments, derived from the transmembrane glycoprotein Amyloid β (Aβ) precursor protein (APP), that form amyloid plaques, the pathological hallmark of AD (4). Microglia are the resident macrophages of the brain and contribute to neurodegenerative disease (5). Ionized calcium-binding adaptor molecule 1 (Iba1), also known as allograft inflammatory factor 1 (AIF-1), is uniquely expressed in cells of monocytic lineage and is, therefore, widely used as a marker for microglia/macrophages in the brain and other tissue (6,7). HS1 (HCLS1, LckBP1, p75) is a protein kinase substrate that is expressed only in tissues and cells of hematopoietic origin and is also expressed in microglia (8,9). Transmembrane protein 119 (TMEM119) is a cell-surface protein of unknown function, expressed exclusively by the microglia subset of myeloid and neural cells (10). Iba1+ microglia with both ramified and amoeboid morphologies express TMEM119, while Iba1+ macrophages are TMEM119 negative (11). *TMEM119* and other homeostatic genes have been shown to be downregulated in microglia. In addition to general markers of microglia, several microglia genes are upregulated during disease progression (12). CD11c (integrin αX, ITGAX) is a transmembrane glycoprotein that forms an α/β heterodimer with CD18 (integrin β2), which interacts with a variety of extracellular matrix molecules and cell surface proteins (13). CD11c-positive microglia transcriptionally correlate with amyloid plaques (14). In addition, other genes are upregulated in a similar manner. Glycoprotein non-metastatic gene B (GPNMB) is a type I transmembrane glycoprotein overexpressed in many types of cancer. The GPNMB glycoprotein is involved in many physiological processes, including mediating transport of late melanosomes to keratinocytes (9,15). Cathepsin B and D are widely expressed cysteine and aspartyl proteases, respectively, involved in the normal degradation of proteins (16,17). ASC/TMS1 has been found to be a critical component of inflammatory signaling where it associates with and activates caspase-1 in response to pro-inflammatory signals and may directly contribute to amyloid plaque formation (18,19).

Background References

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