Microglia are the resident macrophages of the brain and act as the main form of active immune defence scavenging damaged neurons, debris and pathogens. Whilst microglia offer a protective function they can also have deleterious effects as a result of cytotoxic secretion of hydrogen peroxide, nitric oxide, proteases and cytokines.

Microglial cells are the only brain cells to express Iba-1 (ionised calcium binding adapter molecule 1). Calcium ions are important signal mediators and exert their signalling activity through association with various calcium binding proteins. Iba-1 is a 17kDa protein from the large EF hand family of proteins which contain the EF-hand motif.

Iba-1 expression is up-regulated in activated microglia enabling differentiation between cells engaged in routine surveillance and those which are activated in response to injury. For this reason Iba-1, also known as Allograft Inflammatory factor 1 (AIF-1), is often used in immunohistochemistry as a marker for microglia. Enhanced Iba-1 expression has been observed in traumatic brain injury, ischemia and inflammation.

The Wako Anti-Iba-1 antibodies for immunocytochemistry and Western Blotting have been raised against a synthetic peptide corresponding to the carboxyl-terminus of Iba-1, which is conserved amongst human, rat and mouse Iba-1 protein sequences. These antibodies are specific to microglia and macrophages and do not cross react with neurons or astrocytes.

The immunocytochemistry Iba-1 antibody (Cat. 019-19741) is well suited for double-immunostaining of brain tissue or cell cultures in combination with a monoclonal antibody specific to astrocytes, such as Glial Fibrillary Acid Protein (GFAP), as seen opposite.

The Wako anti-Iba1 antibody is widely referenced and according to literature review has also been used with tissues from a number of different species including: human, rat, mouse, dog, pig, macaque and red spotted newt.

<table>
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<tr>
<th>Cat. No.</th>
<th>Description</th>
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<tbody>
<tr>
<td>019-19741</td>
<td>Anti-Iba1 Rabbit IC</td>
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<td>016-20001</td>
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</table>

(Data was provided by Department of Neurochemistry, National Institute of Neuroscience (Japan))

Not for diagnostic use


Compensation of cATSCs-derived TGFβ1 and IL-10 expressions was effectively modulated atopic dermatitis. Jee, M.K., Im, Y.B., Choi, J.I., Kang, S.K. Cell Death Dis. (2013):4:e497


