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#4769

IRAK Isoform Antibody Sampler Kit

1 Kit (4 x 20 microliters)

For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
IRAK1 (D51G7) Rabbit mAb	4504	20 µl	78-105 kDa	Rabbit IgG
IRAK2 Antibody	4367	20 µl	62 kDa	Rabbit
IRAK-M Antibody	4369	20 µl	68 kDa	Rabbit
IRAK4 Antibody	4363	20 µl	55 kDa	Rabbit
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 IRAK Isoform Antibody Sampler Kit provides an economical means to examine total protein levels of the four Interleukin-1 Receptor Associated Kinase family members: IRAK1, IRAK2, IRAK3/IRAK-M, and IRAK4.

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 antibody.

Background

Interleukin-1 (IL-1) receptor-associated kinase (IRAK) is a serine/threonine-specific kinase that can be coprecipitated in an IL-1-inducible manner with the IL-1 receptor (1). The mammalian family of IRAK molecules contains four members (IRAK1, IRAK2, IRAK3/IRAK-M, and IRAK4). The binding of IL-1 to IL-1 receptor type I (IL-1RI) initiates the formation of a complex that includes IL-1RI, AcP, MyD88, and IRAKs (2). IRAK undergoes autophosphorylation shortly after IL-1 stimulation. The subsequent events involve IRAK dissociation from the IL-1RI complex, its ubiquitination, and its association with two membrane-bound proteins: TAB2 and TRAF6. The resulting IRAK-TRAF6-TAB2 complex is then released into the cytoplasm where it activates protein kinase cascades, including TAK1, IKKs, and the stress-activated kinases (3).

Upon IL-1R/TLR (Toll-Like Receptor) ligation, IRAK1 and IRAK4 are rapidly recruited to the receptor by the adaptor MyD88 (4). IRAK1 is phosphorylated by IRAK4 at Thr209 and Thr387 (5), followed by sequential autohyperphosphorylation in various domains. Unlike IRAK1 and IRAK4, IRAK2 and IRAK-M do not have significant kinase activity although they can still activate NF-κB when overexpressed (6,7). Antisense oligonucleotide depletion of IRAK2 can inhibit IL-1 mediated NF-κB activation (8). Expression of IRAK-M is more restricted compared to other family members with highest levels of expression occurring in monocytes/macrophages (6). Studies from IRAK-M knockout mice suggest that it may play a role as a negative regulator of TLR signaling and innate immune responses by preventing the dissociation of IRAK1 and IRAK4 from MyD88 and the subsequent formation of its complex with TRAF6 (9).

Background References

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