

**TREM2-dependent mTOR Metabolic Fitness Antibody Sampler Kit**

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1 Kit (9 x 20 microliters)

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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
TREM2 (D8I4C) Rabbit mAb	91068	20 µl	28 kDa	Rabbit IgG
TREM2 (E7P8J) Rabbit mAb (Carboxy-terminal Antigen)	76765	20 µl	11, 28 kDa	Rabbit IgG
AMPKα (D5A2) Rabbit mAb	5831	20 µl	62 kDa	Rabbit IgG
Phospho-AMPKα (Thr172) (40H9) Rabbit mAb	2535	20 µl	62 kDa	Rabbit IgG
mTOR (7C10) Rabbit mAb	2983	20 µl	289 kDa	Rabbit IgG
Phospho-mTOR (Ser2448) (D9C2) XP® Rabbit mAb	5536	20 µl	289 kDa	Rabbit IgG
Akt (pan) (C67E7) Rabbit mAb	4691	20 µl	60 kDa	Rabbit IgG
Phospho-Akt (Ser473) (D9E) XP® Rabbit mAb	4060	20 µl	60 kDa	Rabbit IgG
LC3A/B (D3U4C) XP® Rabbit mAb	12741	20 µl	14, 16 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit [cellsignal.com](http://cellsignal.com) for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

**Description**

The TREM2-dependent mTOR Metabolic Fitness Antibody Sampler Kit provides an economical means of detecting metabolic signaling pathways downstream of TREM2 by western blot. The kit includes enough antibodies to perform at least two western blot experiments with each 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 antibody.

**Background**

The triggering receptor expressed on myeloid cells 2 (TREM2) protein is an innate immune receptor that is expressed on the cell surface of microglia, macrophages, osteoclasts, and immature dendritic cells (1). The TREM2 protein plays a role in innate immunity and a rare functional variant (R47H) of TREM2 is associated with the late-onset risk of Alzheimer's disease (AD) (1,2). Research studies using mouse models of AD indicate that deficiency and haploinsufficiency of TREM2 can lead to increased β-amyloid (Aβ) accumulation as a result of dysfunctional microglia response (3). Activation of TREM2 in mouse models of AD ameliorates several forms of AD pathology, likely through a microglia-specific mechanism (4,5). This mechanism is under intense investigation, but may involve TREM2-dependent maintenance microglia energetic and biosynthetic metabolism (6). Autophagy is one mechanism by which cellular metabolism is maintained and, in the absence of TREM2, several AMPK-dependent autophagy cell signaling pathways are enhanced. AMP-activated protein kinase (AMPK) is highly conserved from yeast to plants and animals and plays a key role in the regulation of energy homeostasis (7). The tumor suppressor LKB1, in association with accessory proteins STRAD and MO25, phosphorylates AMPKα at Thr172 in the activation loop, and this phosphorylation is required for AMPK activation (8-10). AMPK is further regulated by several proteins within a regulatory cell signaling pathway. The mammalian target of rapamycin (mTOR, FRAP, RAFT) is a Ser/Thr protein kinase (11) that functions as an ATP and amino acid sensor to balance nutrient availability and cell growth (12). mTOR is phosphorylated at Ser2448 via the PI3 kinase/Akt signaling pathway and autophosphorylated at Ser2481 (13). Akt, also referred to as PKB or Rac, is regulated by phosphorylation at Ser473 (14,15). The presence of autophagy marker Light Chain 3 (LC3) in autophagosomes and the conversion of LC3 to the lower migrating form, LC3-II, have been used as indicators of autophagy (16).

**Background References**

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