

**bs-9051R****[ Primary Antibody ]****Bioss**  
**ANTIBODIES**

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**TRPM4 Rabbit pAb****DATASHEET****Host:** Rabbit**Isotype:** IgG**Clonality:** Polyclonal**GeneID:** 54795**SWISS:** Q8TD43**Target:** TRPM4**Immunogen:** KLH conjugated synthetic peptide derived from human TRPM4: 751-850/1214.**Purification:** affinity purified by Protein A**Concentration:** 1mg/ml**Storage:** 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.

Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

**Background:** Calcium-activated non selective (CAN) cation channel that mediates membrane depolarization. While it is activated by increase in intracellular Ca(2+), it is impermeable to it. Mediates transport of monovalent cations (Na(+) > K(+) > Cs(+) > Li(+)), leading to depolarize the membrane. It thereby plays a central role in cardiomyocytes, neurons from entorhinal cortex, dorsal root and vomeronasal neurons, endocrine pancreas cells, kidney epithelial cells, cochlea hair cells etc. Participates in T-cell activation by modulating Ca(2+) oscillations after T lymphocyte activation, which is required for NFAT-dependent IL2 production. Involved in myogenic constriction of cerebral arteries. Controls insulin secretion in pancreatic beta-cells. May also be involved in pacemaking or could cause irregular electrical activity under conditions of Ca(2+) overload. Affects T-helper 1 (Th1) and T-helper 2 (Th2) cell motility and cytokine production through differential regulation of calcium signaling and NFATC1 localization. Enhances cell proliferation through up-regulation of the beta-catenin signaling pathway.

Involvement in disease:

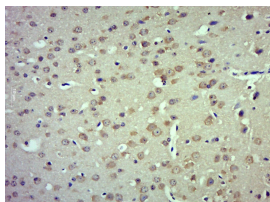
Defects in TRPM4 are the cause of progressive familial heart block type 1B (PFHB1B) [MIM:604559]. It is a cardiac bundle branch disorder characterized by progressive alteration of cardiac conduction through the His-Purkinje system, with a pattern of a right bundle-branch block and/or left anterior hemiblock occurring individually or together. It leads to complete atrio-ventricular block causing syncope and sudden death.

**Applications:** IHC-P (1:100-500)

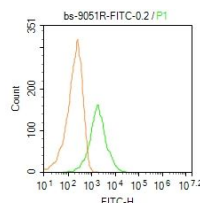
IHC-F (1:100-500)

IF (1:50-200)

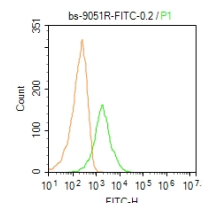
Flow-Cyt (0.2ug/test)

**Reactivity:** Mouse (predicted: Human, Rat)**Predicted MW.:** 134 kDa**Subcellular Location:** Cell membrane ,Cytoplasm**VALIDATION IMAGES**

Paraformaldehyde-fixed, paraffin embedded (Mouse brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (TRPM4) Polyclonal Antibody, Unconjugated (bs-9051R) at 1:500 overnight at 4°C, followed by a conjugated secondary (sp-0023) for 20 minutes and DAB staining.



Blank control: Mouse spleen. Primary Antibody (green line): Rabbit Anti-TRPM4/FITC Conjugated antibody (bs-9051R-FITC) Dilution: 0.2µg /10<sup>6</sup> cells; Isotype Control Antibody (orange line): Rabbit IgG-FITC . Protocol The cells were fixed with 4% PFA (10min at room temperature) and then permeabilized with 0.1% PBST for 20 min at -20°C. The cells were then incubated in 5% BSA to block non-specific protein-protein interactions for 30 min at room temperature. The cells were stained with Primary Antibody for



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**Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

30 min at room temperature. Acquisition of  
20,000 events was performed.

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## — SELECTED CITATIONS —

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- **[IF=4.8]** Xiaoyu Zhu. et al. Chaihu Guizhi Decoction Prevents Cognitive, Memory Impairments and Sensorimotor Gating Deficit Induced by N-methyl-d-aspartate Receptor Antibody in Mice. J ETHNOPHARMACOL. 2024 Sep;;118806 WB ;Mouse. 39278296