SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

[Primary Antibody]

Rabbit Anti-kir 6.1 antibody

Catalog Number: SL6468R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	kir 6.1
Immunogen:	KLH conjugated synthetic peptide derived from human kir 6.1: 61-160/424 <Extracellular>
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.
Applications:	WB=1:500-2000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Mouse,Rat(predicted:Human,Chicken,Pig,Cow,Rabbit)
Predicted MW.:	48kDa
Subcellular Location:	The cell membrane
Background:	This potassium channel is controlled by G proteins. Inward rectifier potassium channels are characterized by a greater tendency to allow potassium to flow into the cell rather than out of it. Their voltage dependence is regulated by the concentration of extracellular potassium; as external potassium is raised, the voltage range of the channel opening shifts to more positive voltages. The inward rectification is mainly due to the blockage of outward current by internal magnesium. Can be blocked by external barium. **Function:** This potassium channel is controlled by G proteins. Inward rectifier potassium channels are characterized by a greater tendency to allow potassium to flow into the cell rather than out of it. Their voltage dependence is regulated by the concentration of extracellular potassium; as external potassium is raised, the voltage range of the channel opening shifts to more positive voltages. The inward rectification is mainly due to the blockage of outward current by internal magnesium. Can be blocked by external barium (By

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	similarity). **Subcellular Location:** Membrane; Multi-pass membrane protein. **Tissue Specificity:** Predominantly detected in fetal and adult heart. DISEASE: Note=Defects in KCNJ8 may be associated with susceptibility to J-wave syndromes, a group of heart disorders characterized by early repolarization events as indicated by abnormal J-wave manifestation on electrocardiogram (ECG). The J point denotes the junction of the QRS complex and the ST segment on the ECG, marking the end of depolarization and the beginning of repolarization. An abnormal J wave is a deflection with a dome or hump morphology immediately following the QRS complex of the surface ECG. Examples of J-wave disorders are arrhythmias associated with an early repolarization pattern in the inferior or mid to lateral precordial leads, Brugada syndrome, some cases of idiopathic ventricular fibrillation (VF) with an early repolarization pattern in the inferior, inferolateral or global leads, as well as arrhythmias associated with hypothermia. Defects in KCNJ8 may be a cause of susceptibility to sudden infant death syndrome (SIDS) [MIM:272120]. SIDS is the sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of clinical history. Pathophysiologic mechanisms for SIDS may include respiratory dysfunction, cardiac dysrhythmias, cardiorespiratory instability, and inborn errors of metabolism, but definitive pathogenic mechanisms precipitating an infant sudden death remain elusive. **Similarity:** Belongs to the inward rectifier-type potassium channel (TC 1.A.2.1) family. KCNJ8 subfamily. **Database links:**

[VALIDATION IMAGES]

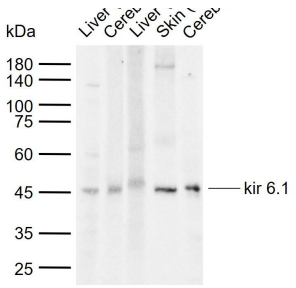


Figure 1. Sample: Lane 1: Mouse Liver tissue lysates Lane 2: Mouse Cerebrum tissue lysates Lane 3: Rat Liver tissue lysates Lane 4: Rat Skin tissue lysates Lane 5: Rat Cerebrum tissue lysates Primary: Anti-kir 6.1 (SL6468R) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 48 kDa Observed band size: 45 kDa

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