

<b>Product name:</b>	S26A5 Rabbit Polyclonal Antibody
<b>Cat number:</b>	ABN17513
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100µL
<b>Clone:</b>	Polyclonal
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rabbit
<b>Isotype:</b>	IgG
<b>Immunogen:</b>	Synthesized peptide derived from human S26A5
<b>Reactivity:</b>	Human, Mouse, Rat
<b>Applications:</b>	WB 1:500-1:2000, IHC 1:100-1:500, ICC/IF 1:50-1:200
<b>Molecular Weight:</b>	81kDa
<b>Purification:</b>	Affinity purification
<b>Form:</b>	Liquid
<b>Buffer:</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
<b>Storage:</b>	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

**Background:**

This gene encodes a member of the SLC26A/SulP transporter family. The protein functions as a molecular motor in motile outer hair cells (OHCs) of the cochlea, inducing changes in cell length that act to amplify sound levels. The transmembrane protein is an incomplete anion transporter, and does not allow anions to cross the cell membrane but instead undergoes a conformational change in response to changes in intracellular Cl<sup>-</sup> levels that results in a change in cell length. The protein functions at microsecond rates, which is several orders of magnitude faster than conventional molecular motor proteins. Mutations in this gene are potential candidates for causing neurosensory deafness. Multiple transcript variants encoding different isoforms have been found for this gene.[provided by RefSeq, Nov 2009],disease:Defects in SLC26A5 are a cause of some forms of recessive non-syndromic deafness.,function:Motor protein that converts auditory stimuli to length changes in outer hair cells and mediates sound amplification in the mammalian hearing organ. Prestin is a bidirectional voltage-to-force converter, it can operate at microsecond rates. It uses cytoplasmic anions as extrinsic voltage sensors, probably chloride and bicarbonate. After binding to a site with millimolar affinity, these anions are translocated across the membrane in response to changes in the transmembrane voltage. They move towards the extracellular surface following hyperpolarization, and towards the cytoplasmic side in response to depolarization. As a consequence, this translocation triggers conformational changes in the protein that ultimately alter its surface area in the plane of the plasma membrane. The area decreases when the anion is near the cytoplasmic face of the membrane (short state), and increases when the ion has crossed the membrane to the outer surface (long state). So, it acts as an incomplete transporter. It swings anions across the membrane, but does not allow these anions to dissociate and escape to the extracellular space. Salicylate, an inhibitor of outer hair cell motility, acts as competitive antagonist at the prestin anion-binding site.,online information:Pump up the volume -Issue 22 of May 2002,similarity:Belongs to the SLC26A/SulP transporter (TC 2.A.53) family.,similarity:Contains 1 STAS domain.,subcellular location:Lateral wall of outer hair cells.,