

**bs-2087R****[ Primary Antibody ]**

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**TSHR Rabbit pAb****— DATASHEET —**

<b>Host:</b> Rabbit	<b>Isotype:</b> IgG	<b>Applications:</b> ELISA (1:5000-10000)
<b>Clonality:</b> Polyclonal		
<b>GeneID:</b> 7253	<b>SWISS:</b> P16473	
<b>Target:</b> TSHR		
<b>Immunogen:</b> KLH conjugated synthetic peptide derived from human TSHR: 201-300/764.		
<b>Purification:</b> affinity purified by Protein A		<b>Reactivity:</b> Human (predicted: Mouse, Rat, Dog, Horse)
<b>Concentration:</b> 1mg/ml		<b>Predicted MW.:</b> 86 kDa
<b>Storage:</b> 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.		<b>Subcellular Location:</b> Cell membrane
<b>Background:</b> The glycoprotein hormone receptor family consists of the luteinizing hormone receptor, the follicle-stimulating hormone receptor, and the thyroid stimulating hormone(TSH) receptor. TSH, which is released from the pituitary gland, binds to the TSH receptor on thyroid cells to control size and function of the thyroid gland (De Felice et al. 2004). The TSH receptor signals through Gs to elevate intracellular cAMP in the thyroid gland, which regulates iodide uptake, and transcription of thyroglobulin (Tg), thyroid peroxidase (TPO), and sodium-iodide symporter. The TSH receptor also signals Gq and phospholipase C to regulate iodide efflux, H2O2 production, and thyroglobulin iodination. Autoimmunity to the TSH receptor causes hyperthyroidism (Graves disease) or hypothyroidism (Hashimoto thyroiditis) when the autoantibodies function as agonists or antagonists, respectively, at the TSH receptor (Rapoport and McLachlan, 2001; Davies et al., 2002). Millipore's cloned human TSH receptor-expressing cell line is made in the Chem-10 host, which supports high levels of recombinant TSH receptor expression on the cell surface and contains high levels of the promiscuous G protein to couple the receptor to the calcium signaling pathway. Thus, the cell line is an ideal tool for screening for antagonists of interactions between TSH and its ligands.		