

**bs-7855R****[ Primary Antibody ]****AICDA Rabbit pAb****BioSS**  
**ANTIBODIES**

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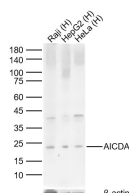
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**— DATASHEET —****Host:** Rabbit**Isotype:** IgG**Clonality:** Polyclonal**GeneID:** 57379**SWISS:** Q9GZX7**Target:** AICDA**Immunogen:** KLH conjugated synthetic peptide derived from human AICDA: 101-198/198.**Purification:** affinity purified by 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:** RNA-editing deaminase involved in somatic hypermutation, gene conversion, and class-switch recombination. Required for several crucial steps of B-cell terminal differentiation necessary for efficient antibody responses. Tissue specificity: Strongly expressed in lymph nodes and tonsils. Involvement in disease: Defects in AICDA are the cause of hyper-IgM immunodeficiency syndrome type 2 (HIGM2); also known as hyper-IgM syndrome 2. HIGM2 is an autosomal recessive disorder characterized by normal or elevated serum IgM levels with absence of IgG, IgA, and IgE, resulting in a profound susceptibility to bacterial infections. HIGM2 causes the absence of Ig class switch recombination (CSR), the lack of Ig somatic hypermutations, and lymph node hyperplasia caused by the presence of giant germinal centers.**Applications:** WB (1:500-2000)**IHC-P** (1:100-500)**IHC-F** (1:100-500)**IF** (1:100-500)**ELISA** (1:5000-10000)**Reactivity:** Human (predicted: Mouse, Rat)**Predicted MW.:** 24 kDa**Subcellular Location:** Nucleus**— VALIDATION IMAGES —**

Sample: Lane 1: Human Raji cell Lysates Lane 2: Human HepG2 cell Lysates Lane 3: Human HeLa cell Lysates Primary: Anti-AICDA (bs-7855R) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 24kDa Observed band size: 24kDa