

AIRE Antibody (Center) Blocking peptide
Synthetic peptide
Catalog # BP11079c**Specification**

AIRE Antibody (Center) Blocking peptide - Product InformationPrimary Accession [O43918](#)**AIRE Antibody (Center) Blocking peptide - Additional Information****Gene ID** 326**Other Names**

Autoimmune regulator, Autoimmune polyendocrinopathy candidiasis ectodermal dystrophy protein, APECED protein, AIRE, APECED

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

AIRE Antibody (Center) Blocking peptide - Protein Information**Name** AIRE**Synonyms** APECED**Function**

Transcription factor playing an essential role to promote self-tolerance in the thymus by regulating the expression of a wide array of self-antigens that have the commonality of being tissue-restricted in their expression pattern in the periphery, called tissue restricted antigens (TRA) (PubMed:26084028). Binds to G-doublets in an A/T-rich environment; the preferred motif is a tandem repeat of 5'-ATTGGTTA-3' combined with a 5'-TTATTA-3' box. Binds to nucleosomes (By similarity). Binds to chromatin and interacts selectively with histone H3 that is not methylated at 'Lys-4', not phosphorylated at 'Thr-3' and not methylated at 'Arg-2'. Functions as a sensor of histone H3 modifications that are important for the epigenetic regulation of gene expression. Mainly expressed by medullary thymic epithelial cells (mTECs), induces the expression of thousands of tissue-restricted proteins, which are presented on major histocompatibility complex class I (MHC-I) and MHC-II molecules to developing T-cells percolating through the thymic medulla (PubMed:26084028). Also induces self-tolerance through other mechanisms such as the regulation of the mTEC differentiation program. Controls the medullary accumulation of thymic dendritic cells and the development of

regulatory T-cell through the regulation of XCL1 expression. Regulates the production of CCR4 and CCR7 ligands in medullary thymic epithelial cells and alters the coordinated maturation and migration of thymocytes. In thymic B-cells, allows the presentation of licensing-dependent endogenous self-antigen for negative selection. In secondary lymphoid organs, induces functional inactivation of CD4(+) T-cells. Expressed by a distinct bone marrow-derived population, induces self-tolerance through a mechanism that does not require regulatory T-cells and is resistant to innate inflammatory stimuli (By similarity).

Cellular Location

Nucleus. Cytoplasm. Note=Predominantly nuclear but also cytoplasmic (PubMed:11274163, PubMed:14974083). Found in nuclear body-like structures (dots) and in a filamentous vimentin-like pattern (PubMed:11274163, PubMed:14974083, PubMed:26084028). Associated with tubular structures (PubMed:11274163, PubMed:14974083)

Tissue Location

Widely expressed. Expressed at higher level in thymus (medullary epithelial cells and monocyte-dendritic cells), pancreas, adrenal cortex and testis. Expressed at lower level in the spleen, fetal liver and lymph nodes. In secondary lymphoid organs, expressed in a discrete population of bone marrow-derived tolerogenic antigen presenting cells (APCs) called extrathymic AIRE expressing cells (eTAC)(at protein level) (PubMed:23993652). Isoform 2 and isoform 3 seem to be less frequently expressed than isoform 1, if at all

AIRE Antibody (Center) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

AIRE Antibody (Center) Blocking peptide - Images

AIRE Antibody (Center) Blocking peptide - Background

This gene encodes a transcriptional regulator that forms nuclear bodies and interacts with the transcriptional coactivator CBP. At least three splice variant mRNAs products have been described including one which results in a premature stop codon and a transcript predicted to be a candidate for nuclear-mediated decay (NMD). Defects in this gene cause the rare autosomal-recessive systemic autoimmune disease termed autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED).

AIRE Antibody (Center) Blocking peptide - References

Cervato, S., et al. Clin. Endocrinol. (Oxf) 73(5):630-636(2010) Ruano, G., et al. Pharmacogenomics 11(7):959-971(2010) Conteduca, G., et al. Clin. Immunol. 136(1):96-104(2010) Colome, N., et al. J. Proteome Res. 9(5):2600-2609(2010) Gu, B., et al. Biochem. Biophys. Res. Commun. 394(2):418-423(2010)