

IRGM Antibody
Catalog # ASC10669**Specification****IRGM Antibody - Product Information**

Application	WB, IHC-P, IF, E
Primary Accession	A1A4Y4
Other Accession	AAI28169 , 345611
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Application Notes	IRGM antibody can be used for detection of IRGM by Western blot at 1 - 2 µg/mL. Antibody can also be used for immunohistochemistry starting at 5 µg/mL. For immunofluorescence start at 20 µg/mL.

IRGM Antibody - Additional InformationGene ID **345611****Target/Specificity**

IRGM antibody was raised against a 17 amino acid synthetic peptide near the amino terminus of the human IRGM.

The immunogen is located within amino acids 30 - 80 of IRGM.

Reconstitution & Storage

IRGM antibody can be stored at 4°C for three months and -20°C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

Precautions

IRGM Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

IRGM Antibody - Protein Information**Name** IRGM {ECO:0000303|PubMed:19266026, ECO:0000312|HGNC:HGNC:29597}**Function**

Immunity-related GTPase that plays important roles in innate immunity and inflammatory response (PubMed:16888103, PubMed:19165925, PubMed:25891078). Acts as a dynamin-like protein that binds to intracellular membranes and promotes remodeling and trafficking of those membranes (By similarity). Required for clearance of acute protozoan and bacterial infections by interacting with autophagy and lysosome regulatory proteins, thereby promoting the fusion of phagosomes with lysosomes for efficient degradation of cargo including microbes (PubMed:16888103, PubMed:16888103, PubMed:19165925, PubMed:25891078).

[25891078](http://www.uniprot.org/citations/25891078), PubMed: [29420192](http://www.uniprot.org/citations/29420192), PubMed: [32939830](http://www.uniprot.org/citations/32939830)). Regulates selective autophagy, including xenophagy and mitophagy, both directly and indirectly (PubMed: [16888103](http://www.uniprot.org/citations/16888103), PubMed: [25891078](http://www.uniprot.org/citations/25891078), PubMed: [29420192](http://www.uniprot.org/citations/29420192), PubMed: [32939830](http://www.uniprot.org/citations/32939830)). Directly regulates autophagy by acting as a molecular adapter that promotes the coassembly of the core autophagy machinery to mediate antimicrobial defense: IRGM (1) activates AMPK, which in turn phosphorylates ULK1 and BECN1 to induce autophagy, (2) promotes the coassembly of ULK1 and BECN1, enhancing BECN1-interacting partners and (3) influences the composition of the BECN1 complex, by competing with the negative regulators BCL2 and RUBCN, to trigger autophagy (PubMed: [25891078](http://www.uniprot.org/citations/25891078)). Also activates autophagy by promoting recruitment of STX17 to autophagosomes (PubMed: [29420192](http://www.uniprot.org/citations/29420192)). In collaboration with ATG8 proteins, regulate lysosomal biogenesis, a fundamental process for any autophagic pathway, by promoting TFEB dephosphorylation (PubMed: [32753672](http://www.uniprot.org/citations/32753672)). Also modulates autophagy by assisting with autophagosome formation and preventing lysosomal deacidification (By similarity). While activating autophagy, acts as a key negative regulator of the inflammatory and interferon responses both by (1) promoting mitophagy and (2) mediating autophagy-dependent degradation of effectors of the inflammatory response (PubMed: [30612879](http://www.uniprot.org/citations/30612879), PubMed: [32715615](http://www.uniprot.org/citations/32715615), PubMed: [36221902](http://www.uniprot.org/citations/36221902)). Promotes degradation of damaged and IFNG/IFN-gamma-stressed mitochondria via mitophagy, preventing cytosolic release of ligands that activate inflammation (PubMed: [32715615](http://www.uniprot.org/citations/32715615)). Acts as a suppressor of inflammation by promoting recruitment of inflammation effectors, such as CGAS, RIGI/RIG-I and NLRP3, to autophagosome membranes, leading to their SQSTM1/p62-dependent autophagic degradation (PubMed: [30612879](http://www.uniprot.org/citations/30612879), PubMed: [32715615](http://www.uniprot.org/citations/32715615)). Also directly inhibits assembly of the NLRP3 inflammasome by preventing the association between NLRP3 and PYCARD (PubMed: [30612879](http://www.uniprot.org/citations/30612879)). Acts as a negative regulator of antiviral innate immune response by suppressing the RIPK2-dependent pro-inflammatory response: mediates recruitment of RIPosomes, composed of RIPK2 and NOD1 or NOD2, to autophagosome membranes, promoting their SQSTM1/p62-dependent autophagic degradation (PubMed: [34467632](http://www.uniprot.org/citations/34467632), PubMed: [36221902](http://www.uniprot.org/citations/36221902)).

Cellular Location

Golgi apparatus membrane. Cell membrane {ECO:0000250|UniProtKB:Q60766}. Cytoplasmic vesicle, phagosome membrane {ECO:0000250|UniProtKB:Q60766}. Cytoplasmic vesicle, autophagosome membrane. Lysosome membrane {ECO:0000250|UniProtKB:Q60766}. Late endosome membrane {ECO:0000250|UniProtKB:Q60766}. Mitochondrion membrane {ECO:0000250|UniProtKB:Q60766}. Cell projection, phagocytic cup {ECO:0000250|UniProtKB:Q60766}. Note=Behaves like an integral membrane protein. Recruited to the plasma membrane around forming phagocytic cups, it remains associated with maturing phagosomes. Association with phagosomes is dependent on nucleotide-binding but is IFNG-independent Also detected in late endosomes and lysosomes {ECO:0000250|UniProtKB:Q60766}

Tissue Location

Widely expressed (at protein level) (PubMed:16888103). Expressed in several tissues including

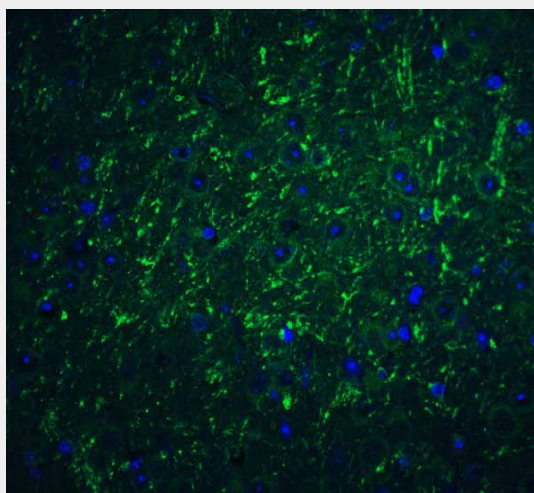
colon, small bowel and peripheral blood leukocytes (PubMed:17554261)

IRGM Antibody - Protocols

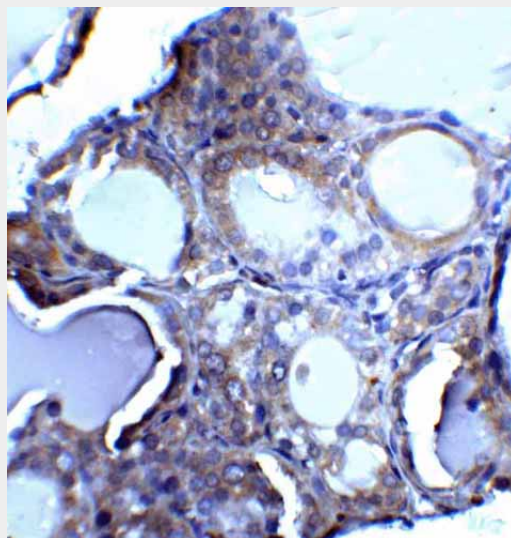
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

IRGM Antibody - Images



Immunofluorescence of PD-L2 in mouse brain tissue with PD-L2 antibody at 20 μ g/ml.



Immunohistochemistry of DNase II in human spleen tissue with DNase II antibody at 5 μ g/ml.

IRGM Antibody - Background

IRGM Antibody: Autophagy, the process of bulk degradation of cellular proteins through an autophagosomic-lysosomal pathway is important for normal growth control and may be defective in tumor cells. It is involved in the preservation of cellular nutrients under starvation conditions as well as the normal turnover of cytosolic components. Two of the strongest hits implicate genes IRGM and ATG16L1, which encode proteins thought to be critical to the autophagy pathway and being significantly associated with Crohn's disease. IRGM induces autophagy and generates large autolysosomal organelles as a mechanism for the elimination of intracellular Mycobacterium tuberculosis. In mouse, IRGM belongs to a family of gamma-interferon-induced GTP-binding proteins of approximately 48 kDa that also includes IRGM2 and IRGM3; this antibody may also recognize these proteins.

IRGM Antibody - References

Gozuacik D and Kimchi A. Autophagy as a cell death and tumor suppressor mechanism. *Oncogene*2004; 23:2891-906.
Massey DC and Parkes M. Genome-wide association scanning highlights two autophagy genes, ATG16L1 and IRGM, as being significantly associated with Crohn's disease. *Autophagy*2007; 3:649-51.
Fisher SA, Tremelling M, Anderson CA, et al. Genetic determinants of ulcerative colitis include the ECM1 locus and five loci implicated in Crohn's disease. *Nat. Genet.*2008; 40:710-2.
Singh SB, Davis AS, Taylor GA, et al. Human IRGM induces autophagy to eliminate intracellular mycobacteria. *Science*2006; 313:1438-41.