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UCP2 Antibody (Internal)
Goat Polyclonal Antibody
Catalog # ALS12723

Specification

UCP2 Antibody (Internal) - Product Information

Application	**WB, IHC-P, E**
Primary Accession	P55851
Reactivity	**Human, Mouse, Rat, Rabbit, Hamster, Monkey, Pig, Horse, Xenopus, Bovine, Dog**
Host	**Goat**
Clonality	**Polyclonal**
Calculated MW	**33kDa KDa**
Dilution	**WB~~1:1000** **IHC-P~~N/A** **E~~N/A**

UCP2 Antibody (Internal) - Additional Information

Gene ID 7351

Other Names
Mitochondrial uncoupling protein 2, UCP 2, Solute carrier family 25 member 8, UCPH, UCP2, SLC25A8

Target/Specificity
Human UCP2.

Reconstitution & Storage
Store at -20°C. Minimize freezing and thawing.

Precautions
UCP2 Antibody (Internal) is for research use only and not for use in diagnostic or therapeutic procedures.

UCP2 Antibody (Internal) - Protein Information

Name UCP2

Synonyms SLC25A8 {ECO:0000303|PubMed:33798544}

Function
Antiporter that exports dicarboxylate intermediates of the Krebs cycle in exchange for phosphate plus a proton across the inner membrane of mitochondria, a process driven by mitochondrial motive force with an overall impact on glycolysis, glutaminolysis and glutathione-dependent redox balance. Continuous export of oxaloacetate and related four-carbon dicarboxylates from mitochondrial matrix into the cytosol negatively regulates the oxidation of acetyl-CoA substrates via the Krebs cycle, lowering the ATP/ADP ratio and reactive oxygen species (ROS) production

(PubMed:24395786). May mediate inducible proton entry into the mitochondrial matrix affecting ATP turnover as a protection mechanism against oxidative stress. The proton currents are most likely associated with fatty acid flipping across the inner membrane of mitochondria in a metabolic process regulated by free fatty acids and purine nucleotides (By similarity) (PubMed:11171965, PubMed:11278935, PubMed:22524567, PubMed:26182433, PubMed:33373220). Regulates the use of glucose as a source of energy. Required for glucose-induced DRP1-dependent mitochondrial fission and neuron activation in the ventromedial nucleus of the hypothalamus (VMH). This mitochondrial adaptation mechanism modulates the VMH pool of glucose- excited neurons with an impact on systemic glucose homeostasis (By similarity). Regulates ROS levels and metabolic reprogramming of macrophages during the resolution phase of inflammation. Attenuates ROS production in response to IL33 to preserve the integrity of the Krebs cycle required for persistent production of itaconate and subsequent GATA3-dependent differentiation of inflammation-resolving alternatively activated macrophages (By similarity). Can unidirectionally transport anions including L-malate, L-aspartate, phosphate and chloride ions (PubMed:22524567, PubMed:24395786, PubMed:26182433). Does not mediate adaptive thermogenesis (By similarity).

Cellular Location
Mitochondrion inner membrane {ECO:0000250|UniProtKB:P70406}; Multi-pass membrane protein

Tissue Location
Widely expressed in adult human tissues, including tissues rich in macrophages. Most expressed in white adipose tissue and skeletal muscle.

UCP2 Antibody (Internal) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

UCP2 Antibody (Internal) - Images

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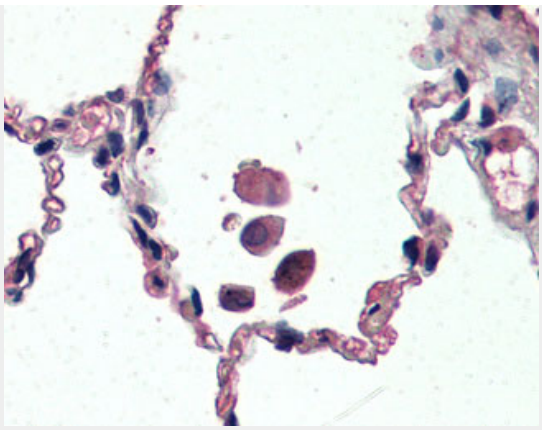
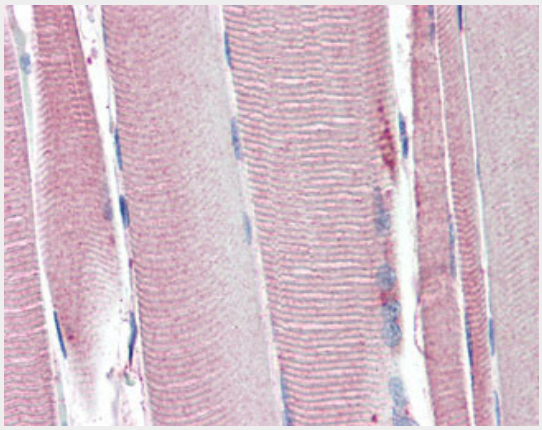


Anti-UCP2 antibody IHC of human lung.



Anti-UCP2 antibody IHC of human skeletal muscle.

UCP2 Antibody (Internal) - Background

UCP are mitochondrial transporter proteins that create proton leaks across the inner mitochondrial membrane, thus uncoupling oxidative phosphorylation from ATP synthesis. As a result, energy is dissipated in the form of heat.

UCP2 Antibody (Internal) - References

Boss O.,et al.FEBS Lett. 408:39-42(1997).
Fleury C.,et al.Nat. Genet. 15:269-272(1997).
Gimeno R.E.,et al.Diabetes 46:900-906(1997).
Klannemark M.,et al.Submitted (JAN-1998) to the EMBL/GenBank/DDBJ databases.
Argyropoulos G.,et al.Diabetes 47:685-687(1998).