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TFAM (Transcription Factor A, motochondrial) Antibody
Rabbit Polyclonal Antibody
Catalog # AN1293

Specification

TFAM (Transcription Factor A, motochondrial) Antibody - Product Information

Application	**WB**
Primary Accession	Q00059
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**29097**

TFAM (Transcription Factor A, motochondrial) Antibody - Additional Information

Gene ID	**7019**
Gene Name	**TFAM**

Target/Specificity
Native recombinant human TFAM protein with c-terminal 6-his tag

Dilution
WB~~ 1:2000

Format
Neat Serum

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
TFAM (Transcription Factor A, motochondrial) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Shipping
Blue Ice

TFAM (Transcription Factor A, motochondrial) 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 Cytomety

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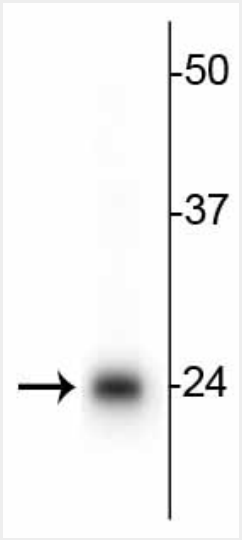
- Cell Culture

TFAM (Transcription Factor A, motochondrial) Antibody - Images



Western blot of HeLa lysate showing specific immunolabeling of the ~24 kDa TFAM protein.

TFAM (Transcription Factor A, motochondrial) Antibody - Background

Mitochondrial Transcription Factor A (TFAM) is a key activator of mitochondrial (mt) DNA transcription as well as a participant in mitochondrial genome replication. mtDNA is highly susceptible to oxidative stress leading to mitochondrial dysfunction. Overexpression of TFAM has been implicated in the amelioration of age dependent impairment of brain functions through the prevention of oxidative stress and mitochondrial dysfunction in microglia (Hayashi et al., 2008). More recently, TFAM overexpression has been shown to potentially reduce oxidative stress in motor neurons and delay onset of amyotrophic lateral sclerosis (ALS) in ALS model mice (Morimoto et al., 2012).