



Anti-AIF (ED2)

(Apoptosis inducing factor, *PDCD8*, Programmed cell death factor 8)

CATALOG NO.: 54068

BACKGROUND:

Apoptosis is characterized by several morphological nuclear changes including chromatin condensation and nuclear fragmentation. These changes are triggered by the activation of members of caspase family, caspase activated DNase, and several novel proteins (1). A novel gene, the product of which causes chromatin condensation and DNA fragmentation, was recently identified, cloned, and designated apoptosis inducing factor (AIF) (2). Like the critical molecules, cytochrome *c* and caspase-9, in apoptosis, AIF localizes in mitochondria. AIF translocates to the nucleus when apoptosis is induced and induces mitochondria to release the apoptogenic proteins cytochrome *c* and caspase-9. AIF induces chromatin condensation and DNA fragmentation, which are the hallmarks of apoptosis, of the isolated nucleus and the nucleus in live cells by microinjection. AIF is highly conserved between human and mouse and widely expressed (2).

SOURCE & REACTIVITY:

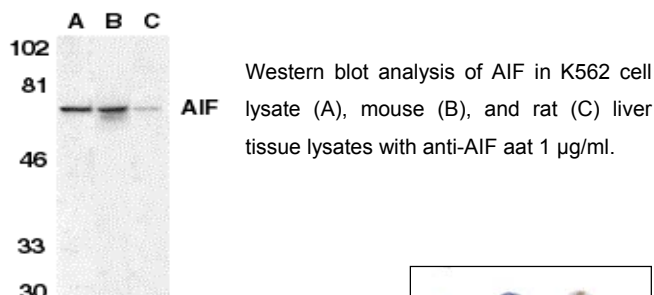
Rabbit anti-AIF polyclonal antibody was raised against a peptide near the C-terminus of human AIF. This sequence is identical to those of mouse and rat AIF. Anti-AIF reacts with AIF at the molecular weight of 67 kDa on western blot. Species reactivity includes human, mouse, and rat, while others are not tested.

APPLICATION:

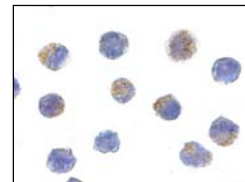
The following concentration ranges are recommended starting points for this product.

WB: 0.25 - 1 µg/ml.

Positive Control: K562 cell lysate



Immunocytochemistry of AIF in K562 cells with anti-AIF at 5 µg/ml.



This product is for in vitro research purposes only.

RELATED PRODUCTS:

K562 Cell Lysate, Catalog No. **29501**

Anti-AIF (IN), Catalog No. **54017**

Anti-AIF (NT), Catalog No. **53214**

Anti-Caspase-9 (IN1), Catalog No. **54040**

STORAGE:

The antibody is supplied as immunoaffinity chromatography purified IgG, in 1X PBS containing 0.02% Sodium Azide. Store at 4°C, stable for one year.

REFERENCES:

1. Zamzami N. et al. *Nature* **401**, 127 (1999).
2. Susin SA. Et al. *Nature* **397**, 44 (1999).