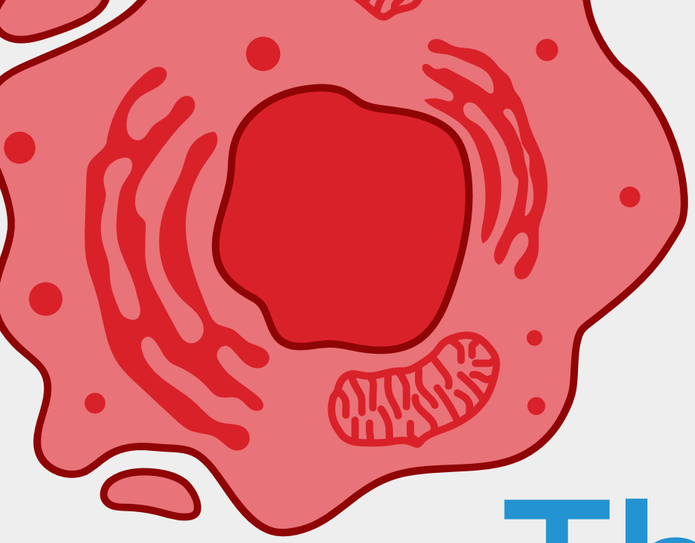
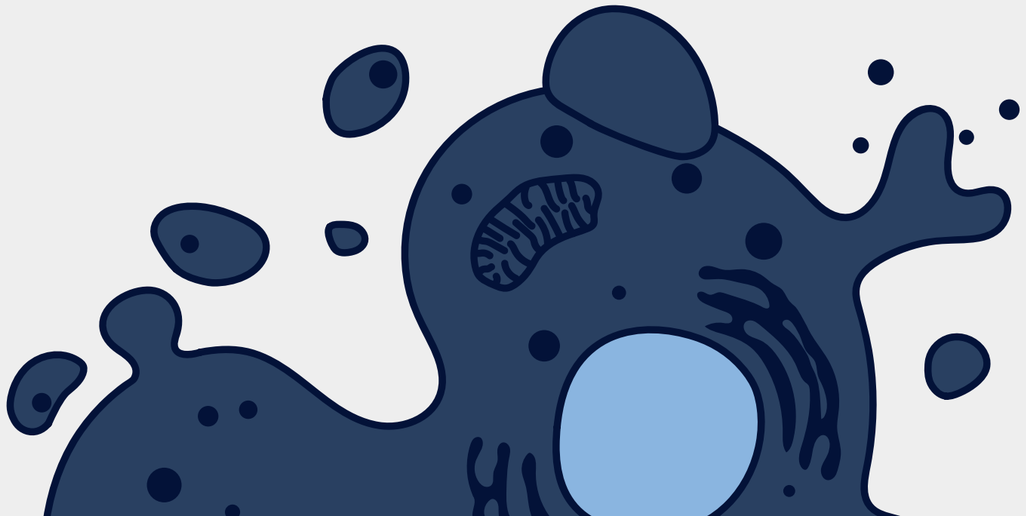


The Many Faces of Cell Death



ROCKLAND

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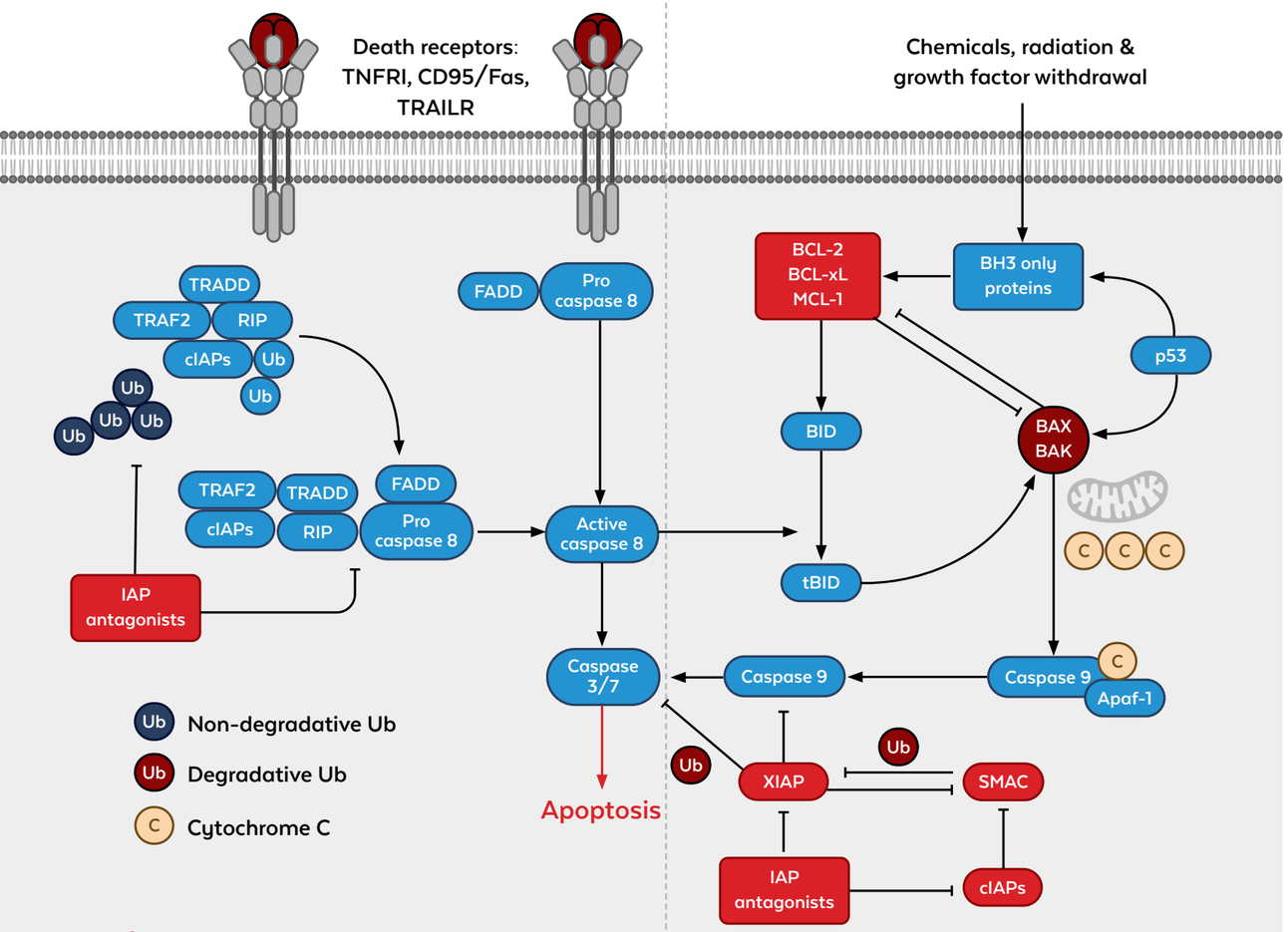
Apoptosis



In highly organized biological systems like multicellular organisms, tight regulation of growth and death is imminent. If cells are no longer required, they commit suicide by activating an intracellular death program. This method of programmed cell death is called **apoptosis**. ?

Continue: Apoptosis

Apoptosis is an energy-dependent biochemical process characterized by distinct morphological features including cell shrinkage, nuclear fragmentation, chromatin condensation, and membrane blebbing. It is a vital component of normal cell turnover, proper development and functioning of the immune system, hormone-dependent atrophy, embryonic development, and chemical-induced cell death, among others.

Because apoptosis cannot stop once it has begun, it is a highly regulated process. Apoptosis can be initiated through one of two pathways.

Caspase-6 Antibody
(600-401-AD7)

Caspases, a family of cysteine proteases, are major mediators of apoptosis. Caspase-6 is classified as an apoptotic effector and it mediates nuclear shrinkage during apoptosis, but it possesses unique activation and regulation mechanisms that differ from those of other effector caspases. Increasing evidence has shown that caspase-6 is highly involved in axon degeneration and neurodegenerative diseases, such as Huntington's disease and Alzheimer's disease.

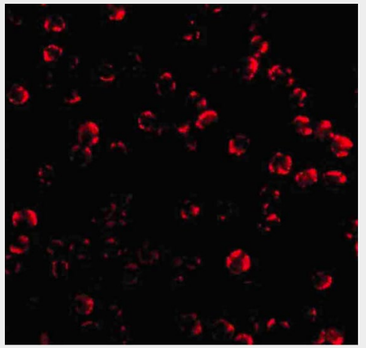


Fig. Immunofluorescence microscopy of anti-Caspase-6 antibody. Cell Type: MCF7 cells. Fixation: 0.5% PFA. Antigen retrieval: not required. Primary antibody: Caspase-6 antibody at 10 µg/mL for 1 h at RT. Secondary antibody: Fluorescein rabbit secondary antibody at 1:10,000 for 45 min at RT. Localization: Caspase-6 is cytoplasmic. Staining: Caspase-6 as red fluorescent signal.

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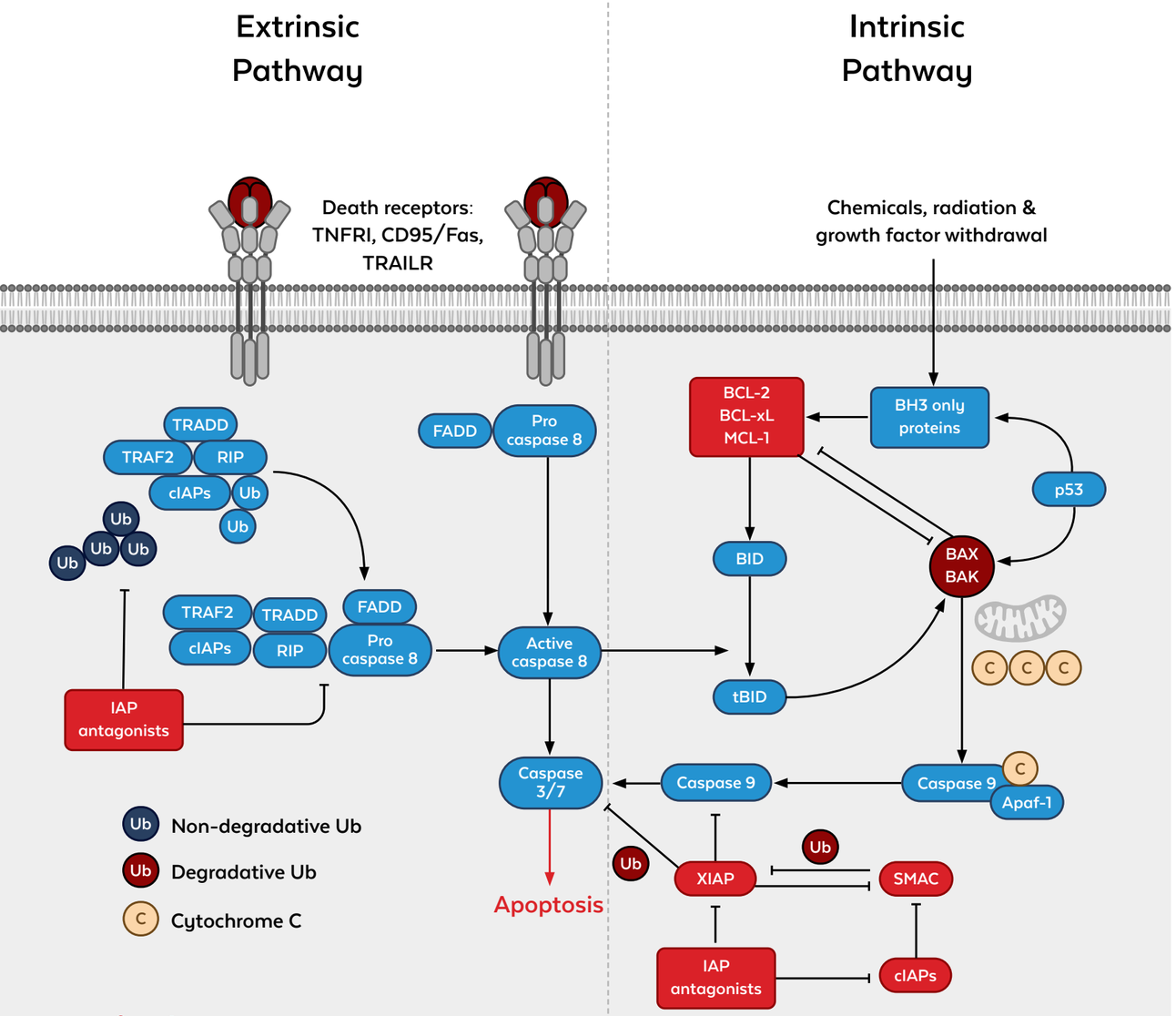
Continue: Apoptosis



In the **intrinsic pathway**, the cell kills itself because it senses cell stress that results in the activation of one or more members of the BH3-only family of proteins.

In the **extrinsic pathway**, the cell kills itself because of signals from other cells as a result of binding extracellular death ligands (such as FasL or tumor necrosis factor-alpha, TNF alpha).

Both induce cell death by activating caspases, which are proteases or enzymes that degrade proteins. The two pathways activate initiator caspases (caspase 8, caspase 9, and caspase 10), which then activate executioner caspase (caspase 3, caspase 6, and caspase 7) that will kill the cell by degrading proteins indiscriminately.

Continue: Apoptosis

Defective apoptosis pathways can result in a wide variety of diseases including autoimmune disorders, neurodegenerative diseases, and many types of cancer.

Jump to:

Parthanatos Pathway

Apoptosis Antibodies

Product	Reactivity	Applications	Item No.
APAF1 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-Y26
APAF1 Antibody	Human, Mouse, Rat	WB, IHC, ELISA	600-401-Y27
APAF1 Antibody [2E10]	Human, Mouse, Rat	WB, IHC, ELISA	200-301-A35
APAF1 Antibody [5E11]	Human, Mouse, Rat	IHC, ELISA	200-301-Y28
BAK Antibody	Human, Mouse	WB, IHC, ELISA	200-401-Z23
BAX Antibody	Human	IHC	200-C01-B34
BCL2 Antibody	Human, Mouse	WB, IHC, IF, ELISA	200-401-Z43
BCL2 Antibody	Human, Mouse, Rat, Bovine	WB, ELISA	200-401-222
BCL-xL Antibody	Human	WB, ELISA	200-401-Z50
BID Antibody	Human, Mouse	WB, IHC, IF, ELISA	200-401-Z65
BID Antibody	Human, Mouse	WB, ELISA	200-401-Z66
Caspase-3 Antibody	Human	IHC, ELISA	600-401-AD2

Continue: Apoptosis Antibodies

Caspase-3 Antibody	Human	WB, IHC, IF, FC	200-301-H63
Caspase-6 Antibody	Human	WB, IHC, IF, ELISA	600-401-AD7
Caspase-6 Antibody	Human	WB, IHC, ELISA	600-401-AD8
Caspase-7 Antibody	Human, Mouse, Rat	WB, IHC, ELISA	600-401-AD9
Caspase-7 Antibody	Human, Mouse, Rat	WB, IHC, ELISA	600-401-AE0
Caspase-8 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-AE1
Caspase-8 Antibody	Human	WB, IHC, IF	200-301-H64
Caspase-9 Antibody	Human, Mouse, Rat	WB, ELISA	600-401-AE3
Caspase-9 Antibody	Human	WB, IHC, IF, ELISA, IP	600-401-AE4
Caspase-10 Antibody	Human	WB, IHC, IF, ELISA	200-401-AC6
CIAP Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-AK2
DcR1 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-AT5
DcR1 Antibody	Human, Mouse, Rat	WB, IF, ELISA	600-401-AT6
DcR2 Antibody	Human, Mouse, Rat	ELISA, IF, IHC, WB	600-401-G93
DR4 Antibody	Human	WB, IHC, IF, ELISA	200-401-AX5
Death Receptor 4 Antibody	Human	WB, IHC, IF, ELISA	600-401-982
DR5 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-G96
DR5 Antibody	Human	WB, IHC, IF, FC	200-401-H72
Mcl-1 Antibody	Human	WB, IHC, IF, ELISA	200-401-C50
RIP1 Antibody	Human	IHC, ELISA	600-401-EA2
Smac Antibody	Human, Mouse, Rat	WB, IHC, IF, IP, ELISA	600-401-ER4
TNF p55 Receptor Antibody	Human, Primate	WB, ELISA	109-401-308
TNF p55 Receptor Antibody	Human	WB, IP, ELISA	209-401-308
TRAF2 Antibody	Human	WB, ELISA	600-401-B27
TRAF2 Antibody	Human, Mouse, Rat	WB, IHC, IP, ELISA	600-401-FM5

Continue: Apoptosis Reagents

Apoptosis Reagents

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Product	Activity	CAS	Item No.
Actinomycin D	Transcription inhibitor	50-76-0	10-2054
Brefeldin A	Inhibits intracellular transport	20350-15-6	10-1071
Cytochalasin D	Disrupts actin filaments	22144-77-0	10-2071
Forskolin	Adenylate cyclase activator	66575-29-9	10-2073
Ionomycin Ca	Calcium ionophore	56092-82-1	10-2078
Mitomycin C	DNA crosslinking and damaging agent	20-07-7	10-1170
Nigericin Na	Induces rapid intracellular acidification	28643-80-3	10-2089
Okadaic acid	Protein phosphatase inhibitor	78111-17-8	10-2091
Puromycin	Translation inhibitor	58-58-2	10-2100
Staurosporine	Kinase inhibitor	62996-74-1	10-2104

References

1. Bertheloot, D., Latz, E., & Franklin, B. S. (2021). Necroptosis, pyroptosis and apoptosis: an intricate game of cell death. Cellular & molecular immunology, 18(5), 1106–1121.
2. Carneiro, B. A., & El-Deiry, W. S. (2020). Targeting apoptosis in cancer therapy. Nature reviews. Clinical oncology, 17(7), 395–417.
3. Van Opdenbosch, N., & Lamkanfi, M. (2019). Caspases in Cell Death, Inflammation, and Disease. Immunity, 50(6), 1352–1364.

Continue: Parthanatos

Parthanatos

Poly (ADP-ribose) polymerase-1 (PARP1) is a chromatin-associated, ADP-ribosylating enzyme essential for multiple cellular functions, including cardiac remodeling, vasoconstriction, regulation of astrocyte and microglial function, long-term memory, aging, transcription regulation, and DNA repair. It has also been implicated in a form of cell death termed **parthanatos** (?), which is distinct from apoptosis because it is caspase-independent, does not form apoptotic bodies, and does not lead to membrane blebbing. Apoptosis and parthanatos still share some features including the involvement of the mitochondrial-associated apoptosis-inducing factor (AIF).

Parthanatos Antibodies

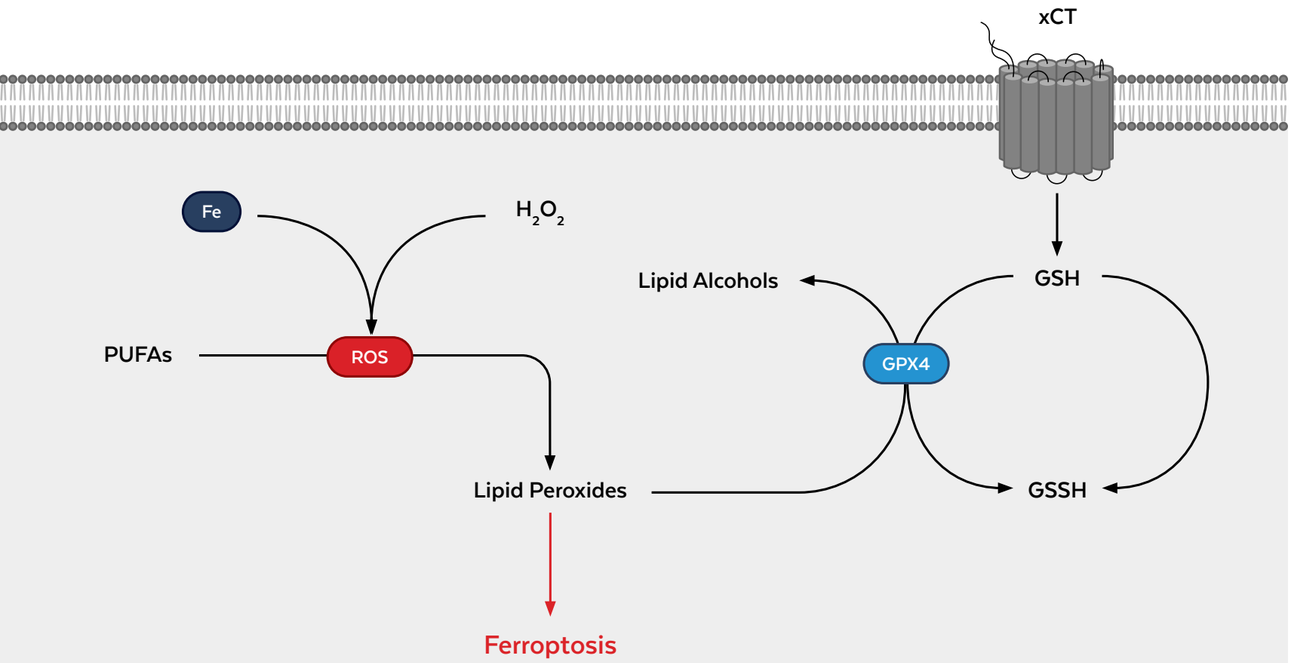
Product	Reactivity	Applications	Item No.
AIF Antibody	Human, Mouse, Rat	WB, IHC, ELISA	200-401-985
AIF Antibody	Human	WB, IHC, IF, ELISA	200-401-X83
PARP1 (N-term ZF1) Antibody	Human	WB, IHC, 2D-PAGE	200-401-GM8
PARP1 (internal) Antibody	Human	WB, IP, 2D-PAGE	200-401-X51

References

1. Fatokun, A. A., Dawson, V. L., & Dawson, T. M. (2014). Parthanatos: mitochondrial-linked mechanisms and therapeutic opportunities. British journal of pharmacology, 171(8), 2000–2016.

Continue: Ferroptosis

Ferroptosis



Death is part of the natural process of all living things, including individual cells. This process can occur in a variety of ways, and new pathways of cell death continue to be discovered. One of them, named **ferroptosis** (?), was first described in 2012 as a non-apoptotic, iron-dependent form of cell death.[1]

Continue: Ferroptosis

Years earlier, in the search for compounds that are selectively lethal to RAS-mutated tumor cells, researchers already identified two structurally independent small molecules named erastin and RSL3 that were able to induce a unique form of cell death.[2]

Further investigation revealed that this type of cell death does not share classic features of apoptosis, such as caspase activation and chromatin fragmentation, and is characterized by the iron-dependent accumulation of lipid hydroperoxides to lethal levels. In contrast, cells that undergo ferroptosis seem to exhibit distinct morphological characteristics such as shrunken and damaged mitochondria.[3]

GPX4 Antibody
(600-401-972)

Glutathione peroxidase 4 (GPX4) is a main regulator of the ferroptosis process. Its unique function is to interrupt the lipid peroxidation chain reaction by reducing complex hydroperoxides by converting them into non-toxic lipid alcohols.

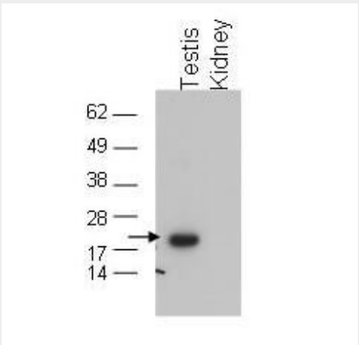


Fig. Western blot using anti-GPX4 antibody. Rockland's affinity purified anti-GPX4 antibody was used to detect GPX4 in testis extract (arrow). Tissue extract (40 µg) was electrophoresed and transferred to nitrocellulose. The membrane was probed with the primary antibody at a 1:1,000 dilution. Personal communication, Dolph Hatfield, CCR-NCI, Bethesda, MD.

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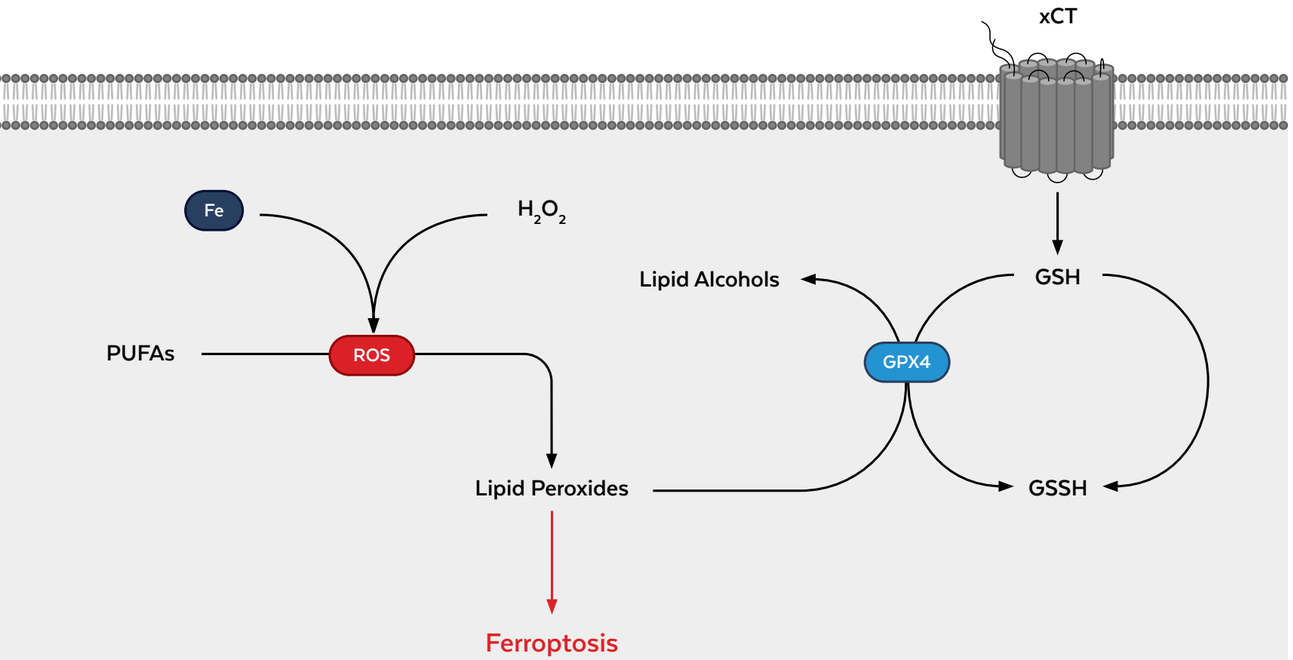
Continue: Ferroptosis



While several proteins have been shown to regulate ferroptosis, glutathione peroxidase 4 (**GPX4**) is the central enzyme of this pathway. GPX4 effectively inhibits ferroptosis by reducing and thus limiting lipid peroxides and reactive oxygen species (**ROS**).[4] This process requires the substrate glutathione (**GSH**), which is provided by the enzyme **xCT** via an intermediate step.

Continue: Ferroptosis

Since its initial discovery, ferroptosis has attracted great interest in its process and function. According to PubGrade, the number of publications has increased exponentially in past years, from 405 in 2019, 849 in 2020, to 1670 in 2021.

Go to products:

Cystein Metabolism
DNA Damage Pathway
Epithelial-Mesenchymal Pathway
ER Stress
Glutamine Metabolism
Iron Metabolism
KRAS Pathway
Lipid Metabolism
Lysosome & Autophagy
Mitochondrial Function
NRF2 Pathway
NOX Pathway
RNS Pathway
Ferroptosis Reagents

Jump to:

Necroptosis Pathway

Calcium Pathway

Dysregulated ORAI1-mediated Ca2+ influx contributes to ferroptosis induced by GSH depletion.[7]

Product	Reactivity	Applications	Item No.
ORAI1 Antibody	Human	WB	100-401-P17
ORAI1 Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-C00
ORAI1 Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-DG9
ORAI1 Antibody [3F6H5]	Human, Mouse, Rat	WB, IHC, IF, ELISA	200-301-DH0
ORAI1 Antibody [6D11A11]	Human, Mouse, Rat	WB, IHC, IF, ELISA	200-301-DH1

Cell Adhesion

Cadherin-mediated intercellular interactions suppress ferroptosis by activating intracellular NF2.[8]

Product	Reactivity	Applications	Item No.
NF2 phospho S518 Antibody	Mouse	WB, IF, ELISA	600-401-414

Continue: Ferroptosis Antibodies

Cysteine Metabolism

The availability of free cysteine determines the extent of GSH synthesis and protection against ferroptosis.[9]

Product	Reactivity	Applications	Item No.
ATF3 Antibody	Human	WB, ELISA	600-401-493
CD44 Antibody	Human	WB, ELISA	600-401-GT4
MUC1 Antibody	Human, Mouse	WB, ELISA	600-401-CW0
xCT Antibody	Human	WB, FC, IF, ELISA	600-401-GU3

Epithelial–Mesenchymal Transition Pathway

ZEB1 provides a bridge between mesenchymal gene expression and lipid peroxide susceptibility.[11]

Product	Reactivity	Applications	Item No.
ZEB1 Antibody	Human	WB, IHC, IF, ELISA	600-401-GD0

DNA Damage Pathway

ATM has been identified as a target for tumor cell ferroptosis, as it can be activated by radiotherapy and increases lipid oxidative damage.[10]

Product	Reactivity	Applications	Item No.
ATM Protein Kinase S1981 Antibody	Human, Mouse	WB, IHC, IF, FC, ELISA	600-401-398
ATM phospho S1981 Antibody	Human	WB, ELISA	600-601-400
ATM phospho S1981 Antibody	Human, Mouse, Rat	WB, IHC, IF, ChIP, IP, FC, FISH, ELISA	200-301-400
ATM phospho S1981 Antibody	Human, Mouse	WB, IHC, IF, IP, ELISA	200-301-500
ATM phospho S1981 Biotin Conjugated Antibody	Human, Mouse, Rat	WB, ELISA	200-306-400
ATM phospho S1981 Peroxidase Conjugated Antibody	Human, Mouse, Rat	WB, ELISA	200-303-400
TFAM Antibody	Human, Mouse, Rat	WB	100-401-X30

Continue: Ferroptosis Antibodies

ER Stress

Ferroptosis is associated with increased ER stress. The chaperone GRP78 (through activation of ATF4) inhibits GPX4 degradation and promotes oxidative stress resistance.[12]

Product	Reactivity	Applications	Item No.
ATF4 Antibody	Human, Rat	WB, IHC, IF	200-301-W61
GRP78 Antibody	Broad	WB, IF	100-401-F38
GRP78 Antibody	Broad	WB, IF	200-301-F37
GRP78 Antibody	Broad	WB, IF	200-301-F36

Glutamine Metabolism

GLS2-mediated glutamate production is required for erastin-induced ferroptosis.[13]

Product	Reactivity	Applications	Item No.
GLS2 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-BL8

Iron Metabolism

Iron is required for the accumulation of lipid peroxides. In this context, the iron carrier protein transferrin plays a key role in the import of iron into the cell.[2]

Product	Reactivity	Applications	Item No.
CISD2 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-AL1
HO-1 Antibody	Human, Mouse, Rat, Dog	WB	600-401-F48
HO-1 Antibody	Broad	WB, IHC, IF, IP	200-301-F47
HSPB2 (MKBP) Antibody	Human, Mouse, Rat	WB, IF	600-401-F76
Hsp25/Hsp27 Antibody	Broad	WB, IHC, IF, IP, FC	200-301-F55
HSP27 Antibody	Human	WB, ELISA	200-301-243
SLC40A1 Antibody	Human	WB, IF, ELISA	600-401-MG1
Mouse Transferrin Antibody	Mouse	EM, ELISA	600-401-255
Transferrin Antibody	Human	WB, ELISA	109-4134
Transferrin Antibody	Human	WB, IHC, ELISA	209-4134

Continue: Ferroptosis Antibodies

KRAS Pathway

Mutations in the oncogene B-raf render cells more susceptible to erastin-induced ferroptosis.[14]

Product	Reactivity	Applications	Item No.
B-raf Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-Z87
B-raf Antibody	Human, Mouse, Rat	WB, ELISA	200-901-Z86

Lipid Metabolism

Glutathione peroxidase 4 (GPX4) is the central enzyme of the ferroptosis pathway. GPX4 effectively inhibits ferroptosis by reducing and thus limiting lipid peroxides and reactive oxygen species.[4]

Product	Reactivity	Applications	Item No.
Glutathione Peroxidase 4 Antibody	Guinea Pig, Mouse, Rat	WB, ELISA	600-401-972
HIF-1-alpha Antibody	Bovine, Human, Mouse, Rat	WB, IHC, IF, ELISA	200-301-F45
HIF-1-alpha hydroxy P564 Antibody	Human	WB, ELISA	100-401-A25
HIF2 alpha Antibody	Human	WB, IHC	209-301-F46
MDM2 Antibody	Mouse	WB, ELISA	600-401-927
Mdm2 phospho S185 Antibody	Human, Mouse	WB, ELISA	600-401-423

Continue: Ferroptosis Antibodies

Lysosome & Autophagy

Several autophagy-related genes modulate ferroptosis by autophagic degradation of cellular iron storage proteins.[15]

Product	Reactivity	Applications	Item No.
ATG3 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-Y81
ATG5 Antibody	Human, Mouse	WB, IHC, ELISA	200-901-Y86
ATG5 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-Y87
ATG8 Antibody	Human, Mouse, Rat	WB, IHC	200-401-H57
ATG13 Antibody	Human	WB, ELISA	600-401-C50
ATG13 phospho S318 Antibody	Human	WB, IF, FC, ELISA, Dot Blot	600-401-C49
BECLIN1 Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-Z53
Beclin 1 Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-MG4
HSP90 total Antibody	Human, Mouse, Rat	WB, IHC, IF, IP	200-301-F74
PINK1 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-DN9
PINK1 truncated Antibody	Human, Mouse	WB, IF, ELISA	600-401-GU5
PINK1 Antibody	Human, Mouse, Rat	WB, IHC, IF	200-301-W64
RAB7 Antibody	Human, Mouse	WB, IHC, IF	600-401-I05
SQSTM1 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-EU6
SQSTM1/p62 Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-HB8
STAT3 (Internal) Antibody	Human	Dot Blot	600-401-GH6
STAT3 R31-Me2a Antibody	Human	Dot Blot	600-401-GH3
STAT3 phospho Y705 Antibody	Human	WB, IHC, ELISA	600-401-C64
ULK1 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-FU4
ULK2 Antibody	Human	WB, IHC, IF, ELISA	600-401-FU5

Continue: Ferroptosis Antibodies

Mitochondrial Function

The ferroptotic small molecules, erastin and artesunate, induce pro-apoptotic PUMA expression.[16]

Product	Reactivity	Applications	Item No.
BID Antibody	Human, Mouse	WB, IHC, IF, ELISA	200-401-Z65
BID Antibody	Human, Mouse	WB, ELISA	200-401-Z66
NEDD4 Antibody	Human	WB, IF, ELISA	600-401-B05
PUMA Antibody	Human	WB, IHC, IF, ELISA	600-401-DV0
PUMA Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-987
PUMA Antibody [10D4G7]	Human, Rat	WB, ELISA	200-301-DV2
PUMA Antibody [2A9G5]	Human, Mouse, Rat	WB, ELISA	200-301-DV4
PUMA Antibody [2A8F6]	Human, Rat	WB, ELISA	200-301-DV3
PUMA Antibody [10C5G1]	Human, Rat	WB, ELISA	200-301-DV1

NRF2 Pathway

NRF2 is an important transcriptional regulator of anti-ferroptotic genes and is itself regulated by enzymes such as KEAP1.[5]

Product	Reactivity	Applications	Item No.
ACVR1B Antibody	Human, Mouse	WB, ELISA	600-401-X67
CDKN2A Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-AJ9
Nrf2 Antibody	Human, Mouse	WB, ELISA	600-401-GT6
PKR Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-DP7
PKR Antibody	Human, Rat	WB, IHC, ELISA	600-401-DP8
KEAP1 Antibody	Human, Mouse, Rat	WB, IF, ELISA	600-401-CE1
TGF Beta Receptor 1 Antibody	Human, Mouse	WB, IHC, FC, ELISA	600-401-MG6

Continue: Ferroptosis Antibodies

NOX Pathway

The NOX family of proteins promote lipid peroxidation in ferroptosis via ROS production.[6]

Product	Reactivity	Applications	Item No.
Nox1 Antibody	Mouse, Rat	WB, IHC	600-401-R15
NOX1 Antibody	Human	WB, IHC, IF, ELISA	600-401-DD8
NOX2 Antibody	Human, Mouse, Rat	WB, IHC	600-401-R16
NOX2 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-DD9
NOX4 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-DE1

RNS Pathway

Scaffolding protein Cav-1 is involved in erastin-induced ferroptosis and links reactive nitrogen species (RNS) to ferroptosis.[17]

Product	Reactivity	Applications	Item No.
Caveolin-1 Antibody	Human	WB	600-401-J62
Caveolin-1 Antibody	Human	WB	600-401-J63
Caveolin-1 phospho S168 Antibody	Human	WB	600-401-J64
NOS2 Antibody	Human, Mouse, Rat	WB	600-401-P89

Continue: Ferroptosis Reagents

Ferroptosis Reagents

Provided by FOCUS BIOMOLECULES

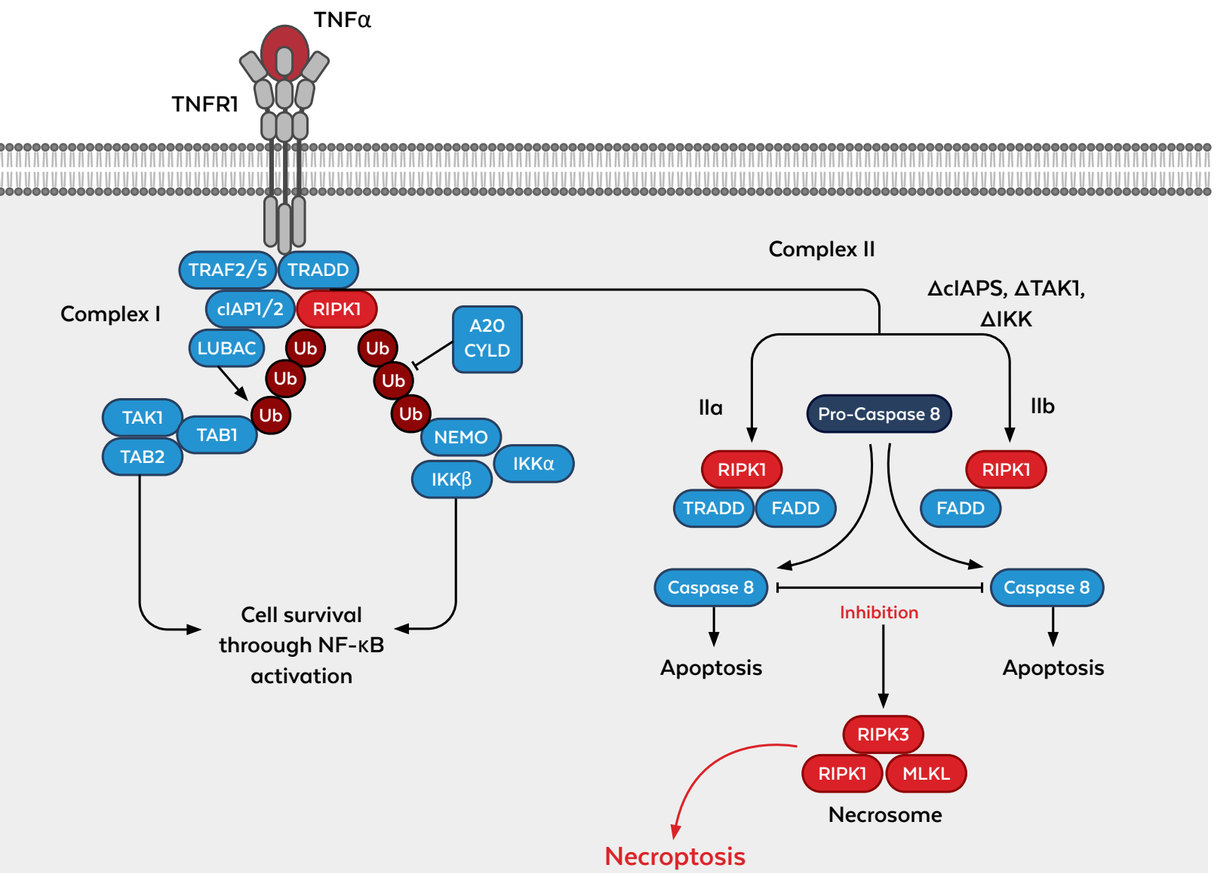
Product	Activity	CAS	Item No.
Artesunate	Ferroptosis inducer	88495-63-0	10-2411
Erastin	Ferroptosis inducer	571203-78-6	10-3406
Ferrostatin-1	Ferroptosis inhibitor	347174-05-4	10-1380
FIN56	Ferroptosis inducer	1083162-61-1	10-4666
Liproxstatin-1	Ferroptosis inhibitor	950455-15-9	10-4670
ML210	GPX4 inhibitor, Ferroptosis inducer	1360705-96-9	10-4002
Nocardamine	Iron chelator, Ferroptosis inhibitor	26605-16-3	10-2775
RSL3	GPX4 inhibitor, Ferroptosis inducer	1219810-16-8	10-3417
SRS16-86	Ferroptosis inhibitor	1793052-96-6	10-4668

References

1. Dixon SJ, Lemberg KM, Lamprecht MR, Skouta R, Zaitsev EM, Gleason CE, Patel DN, Bauer AJ, Cantley AM, Yang WS, Morrison B 3rd, Stockwell BR. Ferroptosis: an iron-dependent form of nonapoptotic cell death. Cell. 2012 May 25;149(5):1060-72.
2. Yang WS, Stockwell BR. Synthetic lethal screening identifies compounds activating iron-dependent, nonapoptotic cell death in oncogenic-RAS-harboring cancer cells. Chem Biol. 2008 Mar;15(3):234-45.
3. Stockwell BR, Friedmann Angeli JP, Bayir H, et al. Ferroptosis: A Regulated Cell Death Nexus Linking Metabolism, Redox Biology, and Disease. Cell. 2017;171(2):273-285.
4. Yang WS, SriRamaratnam R, Welsch ME, Shimada K, Skouta R, Viswanathan VS, Cheah JH, Clemons PA, Shamji AF, Clish CB, Brown LM, Girotti AW, Cornish VW, Schreiber SL, Stockwell BR. Regulation of ferroptotic cancer cell death by GPX4. Cell. 2014 Jan 16;156(1-2):317-331.
5. Dodson M, Castro-Portuguez R, Zhang DD. NRF2 plays a critical role in mitigating lipid peroxidation and ferroptosis. Redox Biol. 2019 May;23:101107.
6. Tang D, Chen X, Kang R, Kroemer G. Ferroptosis: molecular mechanisms and health implications. Cell Res. 2021 Feb;31(2):107-125.
7. Henke N, Albrecht P, Bouchachia I, Ryazantseva M, Knoll K, Lewerenz J, Kaznacheyeva E, Maher P, Methner A. The plasma membrane channel ORAI1 mediates detrimental calcium influx caused by endogenous oxidative stress. Cell Death Dis. 2013 Jan 24;4(1):e470.
8. Wu J, Minikes AM, Gao M, Bian H, Li Y, Stockwell BR, Chen ZN, Jiang X. Intercellular interaction dictates cancer cell ferroptosis via NF2-YAP signalling. Nature. 2019 Aug;572(7769):402-406.
9. Fujii J, Homma T, Kobayashi S. Ferroptosis caused by cysteine insufficiency and oxidative insult. Free Radic Res. 2020 Dec;54(11-12):969-980.
10. Lang X, Green MD, Wang W, Yu J, Choi JE, Jiang L, Liao P, Zhou J, Zhang Q, Dow A, Saripalli AL, Kryczek I, Wei S, Szeliga W, Vatan L, Stone EM, Georgiou G, Cieslik M, Wahl DR, Morgan MA, Chinnaiyan AM, Lawrence TS, Zou W. Radiotherapy and Immunotherapy Promote Tumoral Lipid Oxidation and Ferroptosis via Synergistic Repression of SLC7A11. Cancer Discov. 2019 Dec;9(12):1673-1685.
11. Viswanathan, V., Ryan, M., Dhruv, H. et al. Dependency of a therapy-resistant state of cancer cells on a lipid peroxidase pathway. Nature 547, 453–457 (2017).
12. Zhu S, Zhang Q, Sun X, Zeh HJ 3rd, Lotze MT, Kang R, Tang D. HSPA5 Regulates Ferroptotic Cell Death in Cancer Cells. Cancer Res. 2017 Apr 15;77(8):2064-2077.
13. Gao M, Monian P, Quadri N, Ramasamy R, Jiang X. Glutaminolysis and Transferrin Regulate Ferroptosis. Mol Cell. 2015 Jul 16;59(2):298-308.
14. Yagoda N, von Rechenberg M, Zaganjor E, Bauer AJ, Yang WS, Fridman DJ, Wolpaw AJ, Smukste I, Peltier JM, Boniface JJ, Smith R, Lessnick SL, Sahasrabudhe S, Stockwell BR. RAS-RAF-MEK-dependent oxidative cell death involving voltage-dependent anion channels. Nature. 2007 Jun 14;447(7146):864-8.
15. Gao M, Monian P, Pan Q, Zhang W, Xiang J, Jiang X. Ferroptosis is an autophagic cell death process. Cell Res. 2016 Sep;26(9):1021-32.
16. Hong SH, Lee DH, Lee YS, Jo MJ, Jeong YA, Kwon WT, Choudry HA, Bartlett DL, Lee YJ. Molecular crosstalk between ferroptosis and apoptosis: emerging role of ER stress-induced p53-independent PUMA expression. Oncotarget. 2017 Dec 8;8(70):115164-115178.
17. Deng G, Li Y, Ma S, Gao Z, Zeng T, Chen L, Ye H, Yang M, Shi H, Yao X, Zeng Z, Chen Y, Song Y, Liu B, Gao L. Caveolin-1 dictates ferroptosis in the execution of acute immune-mediated hepatic damage by attenuating nitrogen stress. Free Radic Biol Med. 2020 Feb 20;148:151-161.

Continue: Necroptosis

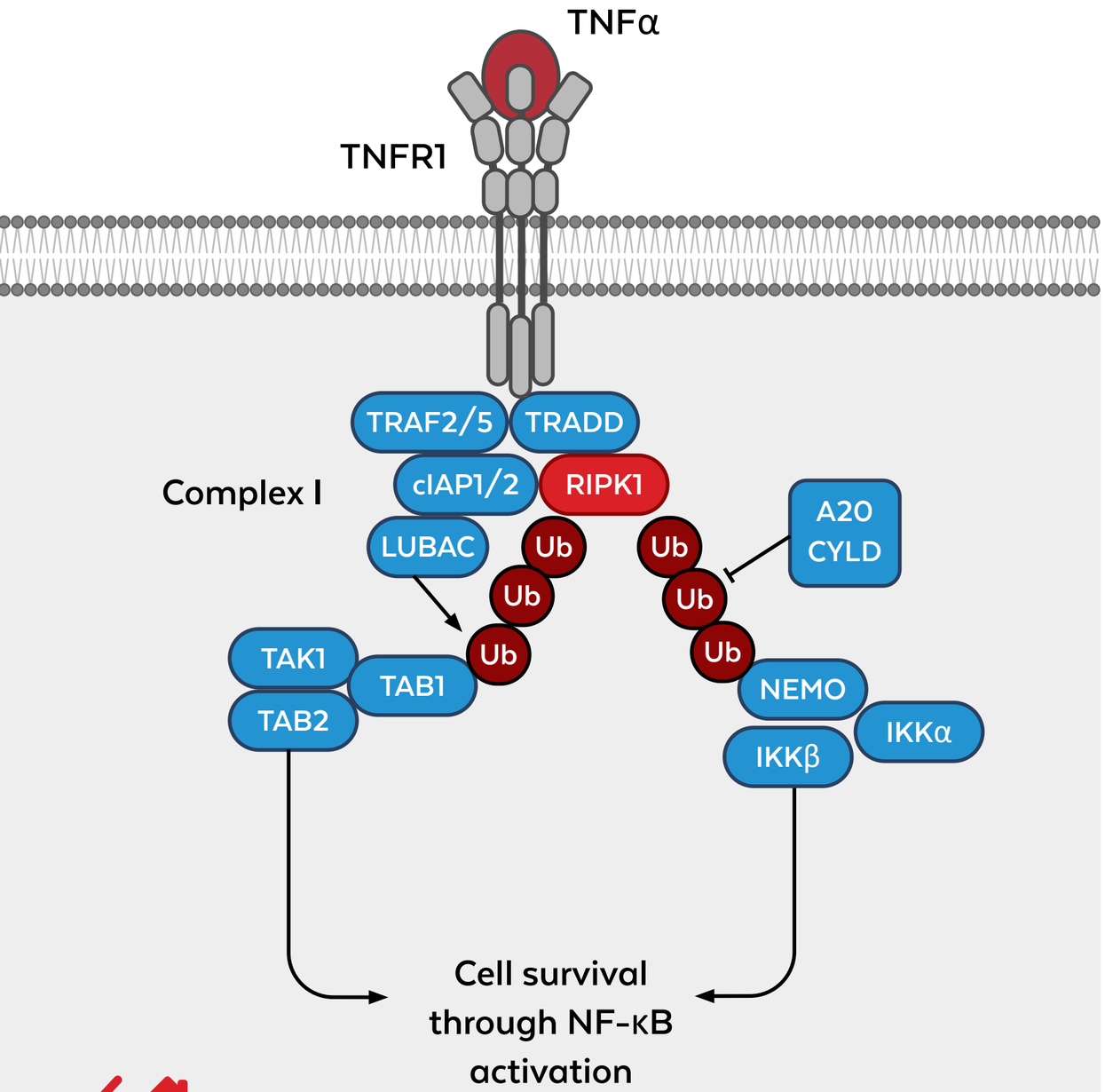
Necroptosis



Among the many ways in which cells can die, **necroptosis** ? is a caspase-independent form of programmed cell death induced by certain changes in cellular homeostasis or when apoptosis is blocked.[1]

It has roles in normal biological processes like inflammation, wound healing, combating infectious disease, as well as in disease states like cancer, and chronic inflammation.[2] In fact, necroptosis can protect or kill tumor cells, depending on the context.[3]

Continue: Necroptosis



Necroptosis can be viewed as a combination of apoptosis and necrosis.[4] It begins with external or internal triggers such as TNFα, TRAIL, interferon γ, genotoxic stress, viral DNA/RNA, bacterial LPS, or caspase 8 inhibition.

These signals are transduced by receptors and binding proteins such as the toll-like receptor (TLR), tumor necrosis factor receptor 1, FAS, or Z-DNA binding protein 1 (ZBP1).[2]

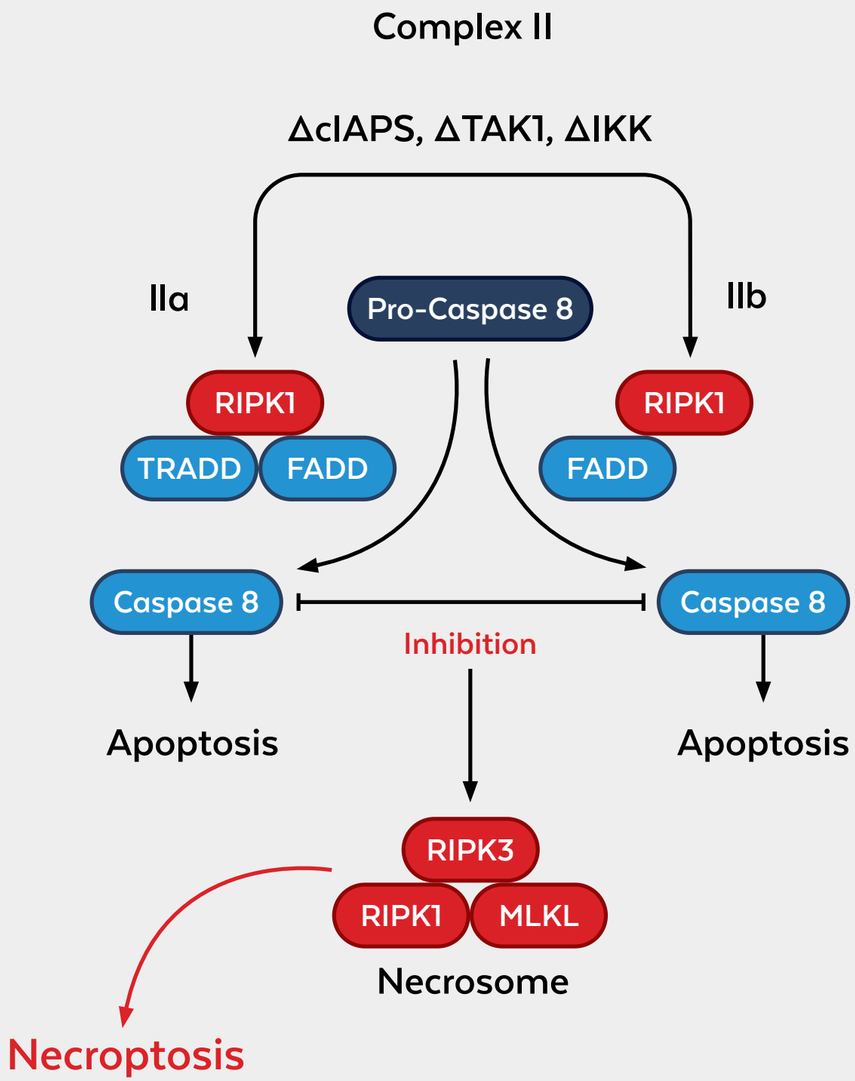
The most understood pathway is the one beginning with TNF-α binding to its receptor, TNFR1. This results in the formation of **complex I**, which comprises **RIPK1**, **TRADD**, **TRAF2** & **-5**, **cIAP1** & **-2**, and **LUBAC**. If RIPK1 is polyubiquitinated by cIAP1/2 and LUBAC, cell survival is achieved by activation of the NF-κB pathway.[4]

Continue: Necroptosis

If instead RIPK1 is deubiquitinated by CYLD or A20, TRADD and RIPK1 are released and form either complex **IIa** (TRADD, FADD, and RIPK1) or complex **IIb** (FADD, RIPK1).

If **caspase 8** is present and active, apoptosis ensues via complex IIa or IIb. However, if caspase 8 is inhibited or absent, **RIPK3** is recruited, causing RIPK3 oligomerization and autophosphorylation.[5] RIPK3 then phosphorylates **MLKL**, causing its oligomerization, which induces (among other things) Ca2+ influx via TRPM7[6], promoting cell membrane perforation and eventually necroptosis.[5]

Alternatively, in the presence of reactive oxygen species (ROS) RIPK1 autophosphorylation recruits RIPK3 to form the necrosome, again leading to necroptosis.[7]



Continue: Necroptosis

RIPK1 Antibody
(600-401-EA3)

RIPK1 plays a key role in apoptosis and necroptosis. RIPK1 interacts with RIPK3 through receptor homology domain (RHD) leading to formation of necrosome which further initiates the downstream signaling resulting in necroptosis.

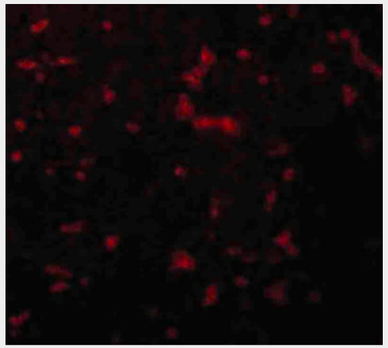


Fig. Immunofluorescence microscopy of anti-RIPK1 antibody. Tissue: mouse kidney cells. Fixation: 0.5% PFA. Antigen retrieval: not required. Primary antibody: RIPK1 antibody at 20 µg/mL for 1 h at RT. Secondary antibody: Fluorescein rabbit secondary antibody at 1:10,000 for 45 min at RT. Localization: RIPK1 is cytoplasmic. Staining: RIPK1 as red fluorescent signal.

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Components of necroptosis can overlap with other forms of cell death.

For example:

a. Parthanatotic death is driven by DNA damage and its pathways can involve RIPK1 and RIPK3 stimulation of PARP1[8]

b. NETotic cell death can be blocked by RIPK1 or MLKL small molecule inhibitors[9]

c. Autophagy can mediate necroptosis via formation of necrosomes on autophagosomes[10]

Continue: Necroptosis

Necroptosis is a key player in pathologies such as neurodegeneration, inflammation, kidney damage, and cancer (proliferation, invasion, angiogenesis, metastasis)[2], thus many small molecule modulators of necroptotic pathways have been developed for use as research tools and therapeutics.[4]

For example:

a. Necrostatin-1 (Nec-1) and RIPA-56 are potent and selective inhibitors of RIPK1
b. Ponatinib inhibits both RIPK1 and RIPK3
c. Necrosulfonamide has a different mechanism of action, specifically blocking the interaction of MLKL with RIPK3

Compounds like these are important tools for necroptosis research, and many are currently in clinical trials for cancer, colitis, arthritis, psoriasis, Alzheimer's, and ALS4.

Go to products:

Necroptosis Reagents
Necroptosis Antibodies

Jump to:

Cuproptosis Pathway

Continue: Necroptosis Reagents

Necroptosis Reagents

Provided by FOCUS BIOMOLECULES

Product	Activity	CAS	Item No.
7-Cl-O-Nec1	RIP1 inhibitor	852391-15-2	10-4544
GSK872	Necroptosis inhibitor, RIP3 inhibitor	1346546-69-7	10-4861
Matrine	Necroptosis inducer	519-02-8	10-4612
Necrostatin-1	RIP1 inhibitor	4311-88-0	10-1162
Necrosulfonamide	MLKL inhibitor	1360614-48-7	10-4860
Ponatinib	Multikinase inhibitor	943319-70-8	10-5064
RIPA-56	RIPK1 inhibitor	1956370-21-0	10-4611

Necroptosis Antibodies

Product	Reactivity	Applications	Item No..
A20 Antibody	Human	WB, IHC, IF, IP, FC	200-301-H52
CIAP Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-AK2
FLIP Antibody	Human, Mouse, Rat	WB, IHC, IF, FC, ELISA	600-401-BF3
IKB alpha Antibody	Human, Mouse, Rat	WB, EMSA	100-4167C
IKK alpha Antibody	Human	WB, IHC, IF, ELISA	600-401-BT9
IKK alpha Antibody	Human	WB, IHC, IF, ChIP, IP, FC	200-301-H82
IKK beta Antibody	Human, Mouse	WB, IHC, IF, IP, FC	200-301-H83
IKK beta Antibody	Human, Mouse, Rat	WB, IHC	100-401-220
NEMO/IKK-gamma Antibody	Human	WB, IP	200-401-GM7
NFkB p65 Antibody	Human	WB, IHC, IF, EMSA, ELISA	600-401-271

Continue: Necroptosis Antibodies

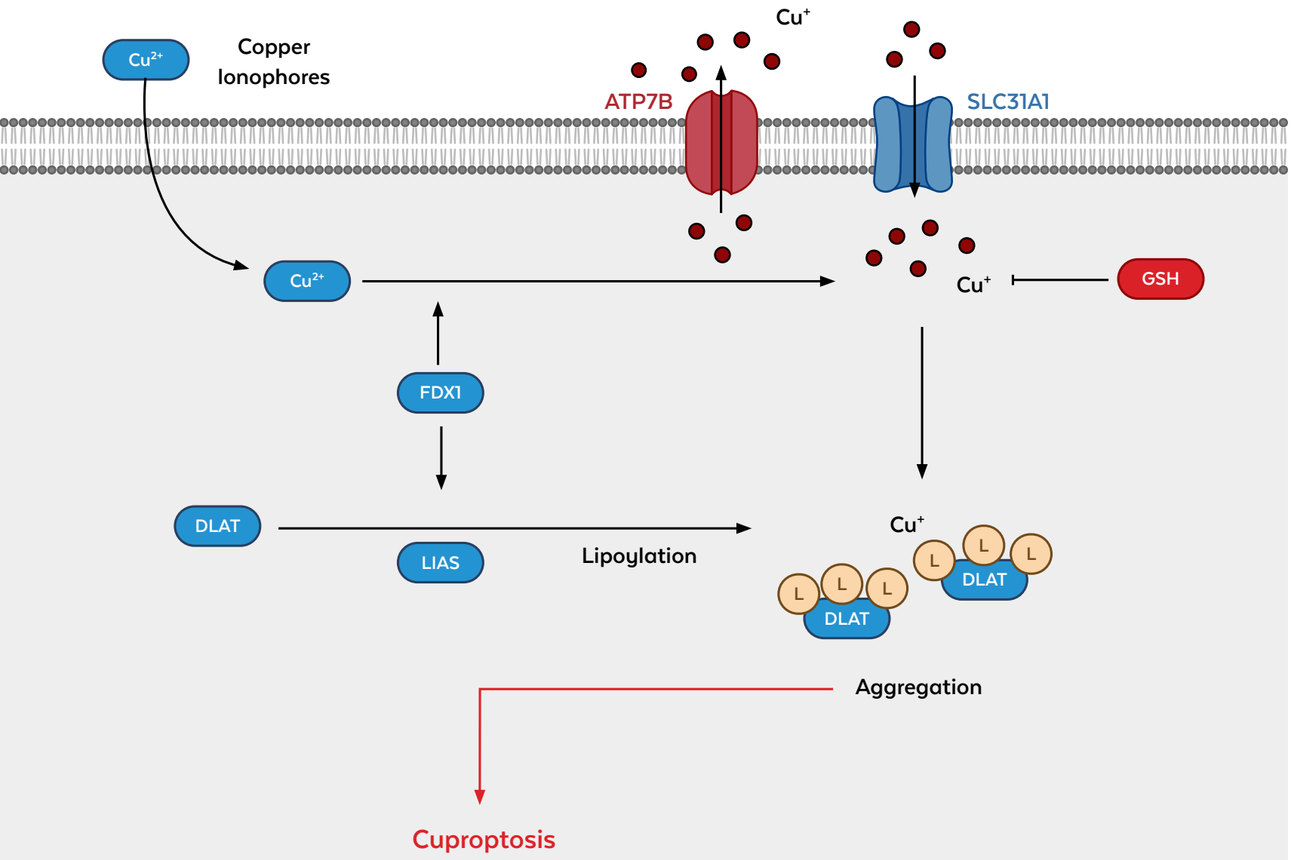
NFkB p65 Antibody	Human, Mouse	WB, IHC, IF, ChIP, IP, EMSA, ELISA	100-4165
Recombinant Anti-TNF alpha Fab Antibody	Human	WB, ELISA	400-001-MT3
RIPK1 Antibody	Human, Mouse, Rat	WB, IHC, IF, ELISA	600-401-EA3
RIP3 Antibody	Human, Mouse, Rat	WB, IHC, IF, IP, ELISA	600-401-H16
TAB1 Antibody	Human, Mouse	WB, IHC, IF, ELISA	600-401-EZ1
TAB2 Antibody	Human	IHC, ELISA	600-401-EZ2
TAK1 Antibody	Human, Mouse, Rat	WB, IF, ELISA	600-401-EZ6
TLR3 Antibody	Human, Mouse	WB, IHC, IF, ELISA	200-401-FF9
TLR3 Antibody	Human	WB, IHC, IF, IP, FC	200-301-I24
TLR4 Antibody	Human	WB, IHC, ELISA	600-401-MK3
TLR4 Antibody	Human, Mouse, Rat	WB, IHC, IF, ChIP, FC, EMSA, ELISA	200-301-I25
TNF alpha Antibody	Human	WB, IHC, IF	209-401-306
TNF alpha Antibody	Mouse	WB, IHC	210-401-321
TRAF2 Antibody	Human, Mouse, Rat	WB, IHC, IP, ELISA	600-401-FM5

References

1. Galluzzi L, Vitale L, Aaronson SA et al. Molecular mechanisms of cell death: recommendations of the Nomenclature Committee on Cell Death 2018. Cell Death Differ. 2018 Mar;25(3):486-541.
2. He R, Wang Z, Dong S, Chen Z, Zhou W. Understanding Necroptosis in Pancreatic Diseases. Biomolecules. 2022 Jun 13;12(6):828.
3. Qin X, Ma D, Tan Y.-X, Wang H.-Y, Cai Z. The role of necroptosis in cancer: A double-edged sword? Biochim Biophys Acta Rev Cancer. 2019 Apr;1871(2):259-266.
4. Chen J, Kos R, Garssen J, Redegeld F. Molecular Insights into the Mechanism of Necroptosis: The Necrosome as Potential Therapeutic Target. Cells. 2019 Nov 21;8(12):1486.
5. Wang Q, Fan D, Xia Y, Ye Q, Xi X, Zhang G, Xiao C. The latest information on the RIPK1 post-translational modifications and functions. Biomed Pharmacother. 2021 Oct;142:112082.
6. Cai Z, Jitkaew S, Zhao J, Chiang H-C, Choksi S, Liu J, Ward Y, Wu L-G, Liu Z-G. Plasma membrane translocation of trimerized MLKL protein is required for TNF-induced necroptosis. Nat Cell Biol. 2014 Jan;16(1):55-65.
7. Seo J, Nam YW, Kim S, Oh D-B, Song J. Necroptosis molecular mechanisms: Recent findings regarding novel necroptosis regulators. Exp Mol Med. 2021 Jun;53(6):1007-1017.
8. Jouan-Lanhouet S, Arshad MI, Piquet-Pellorce C, Martin-Chouly C, Le Moigne-Muller G, Van Herreweghe F, Takahashi N, Sergent O, Lagadic-Gossmann D, Vandenabeele P, Samson M, Dimanche-Boitrel M-T. TRAIL induces necroptosis involving RIPK1/RIPK3-dependent PARP-1 activation. Cell Death Differ. 2012 Dec;19(12):2003-14.
9. Desai J, Kumar SV, Mulay SR, Konrad L, Romoli S, Schauer C, Herrmann M, Bilyy M, Müller S, Popper B, Nakazawa D, Weidenbusch M, Thomasova D, Krautwald S, Linkermann A, Anders H-A. PMA and crystal-induced neutrophil extracellular trap formation involves RIPK1-RIPK3-MLKL signaling. Eur J Immunol. 2016 Jan;46(1):223-9.
10. Basit F, Cristofanon S, Fulda S. Obatoclax (GX15-070) triggers necroptosis by promoting the assembly of the necrosome on autophagosomal membranes. Cell Death Differ. 2013;20:1161-73.

Continue: Cuproptosis

Cuproptosis



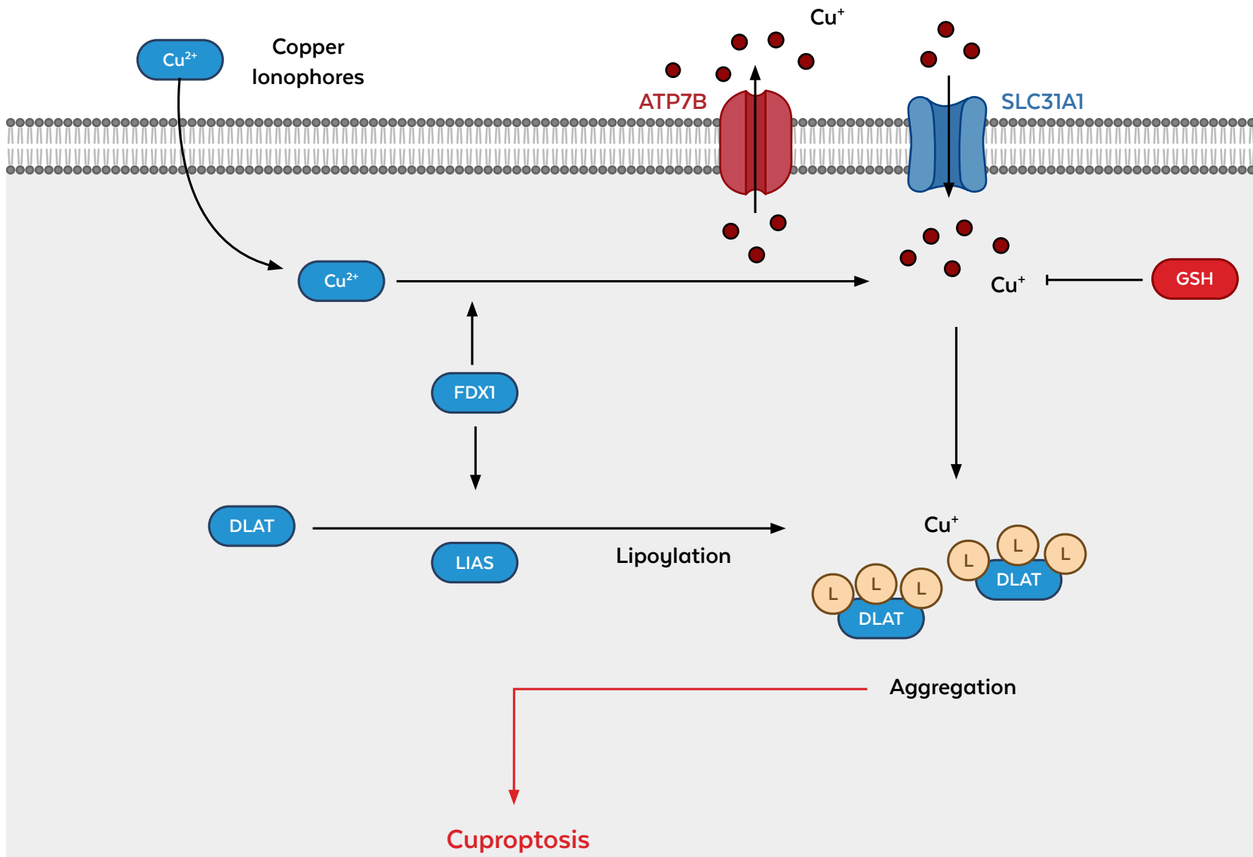
Although it has long been known that copper has a lethal effect on cells at higher doses, only recently has a possible explanation been found.

In the March 2022 issue of Science, Tsevtkov *et al.* reported a copper-induced cell death associated with lipoylated tricarboxylic acid cycle proteins.[1] By analogy with other types of cell death, this newly discovered form has been termed **cuproptosis**. ?

Continue: Cuproptosis

The researchers used various copper ionophores as tools to shuttle copper ions into the cells in specific concentrations and observe the effects they triggered. By using copper-free, as well as copper-containing cell culture media and copper chelators as controls, the relationship between cell death and intracellular copper accumulation was proven. But could it be that the elevated copper concentration triggers an already known form of cell death?

To answer this question, the researchers led by Peter Tsvetkov investigated the effect of blocking known signaling pathways using knock-outs and inhibitors. It was clearly shown that cell death triggered by copper ionophores differs in its signaling pathway from apoptosis, necroptosis, pyroptosis, and ferroptosis.



Continue: Cuproptosis

FDX1 Antibody
(ABIN6140571)

FDX1 is considered a central regulator of cuproptosis as depletion of FDX1 has led to complete loss of protein lipoylation.

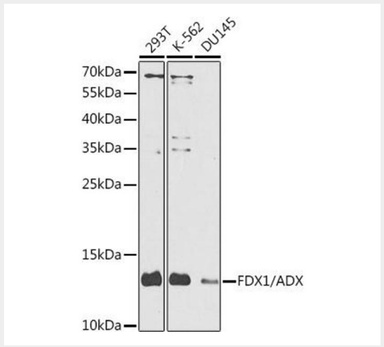


Fig. Western blot analysis of extracts of various cell lines, using anti-FDX1/ADX antibody at 1:1000 dilution. Secondary antibody: HRP Goat Anti-Rabbit (H&L) at 1:10000 dilution. Lysates/proteins: 25µg per lane. Blocking buffer: 3% nonfat dry milk in TBST. Detection: ECL Enhanced Kit (RM00021). Exposure time: 90s.

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The question of identifying the proteins involved in cuproptosis was subsequently investigated using CRISPR-CAS9 screens. Seven genes were identified that were able to escape copper-induced cell death. Surprisingly, some of them were found to be involved in a rare post-translational modification that has so far been documented for only five proteins. This modification, named lipoylation, is characterized by the covalent attachment of lipoamide to lysine residues.[2]

One of the candidates found in the initial screens attracted particular interest as deletion of FDX1 resulted in consistent resistance to cuproptosis. Further immunological experiments demonstrated that the knockout of FDX1 also leads to a complete loss of protein lipoylation, identifying FDX1 as a previously unknown regulator of this pathway.

Continue: Cuproptosis

Most interestingly is how copper-induced cell death and lipoylation are related. The paper strongly suggests that copper is bound to the lipoyl moiety of lipoylated proteins and leads to the aggregation of those proteins and subsequent HSP70 activation.

It remains to be seen whether exploiting these findings can provide new momentum for the research on copper-related disorders, such as Menkes disease, occipital horn syndrome, and Wilson disease, or if it will provide a new approach to treating certain types of tumors.

Provided by

ANTIBODIES ONLINE

Cuproptosis Antibodies

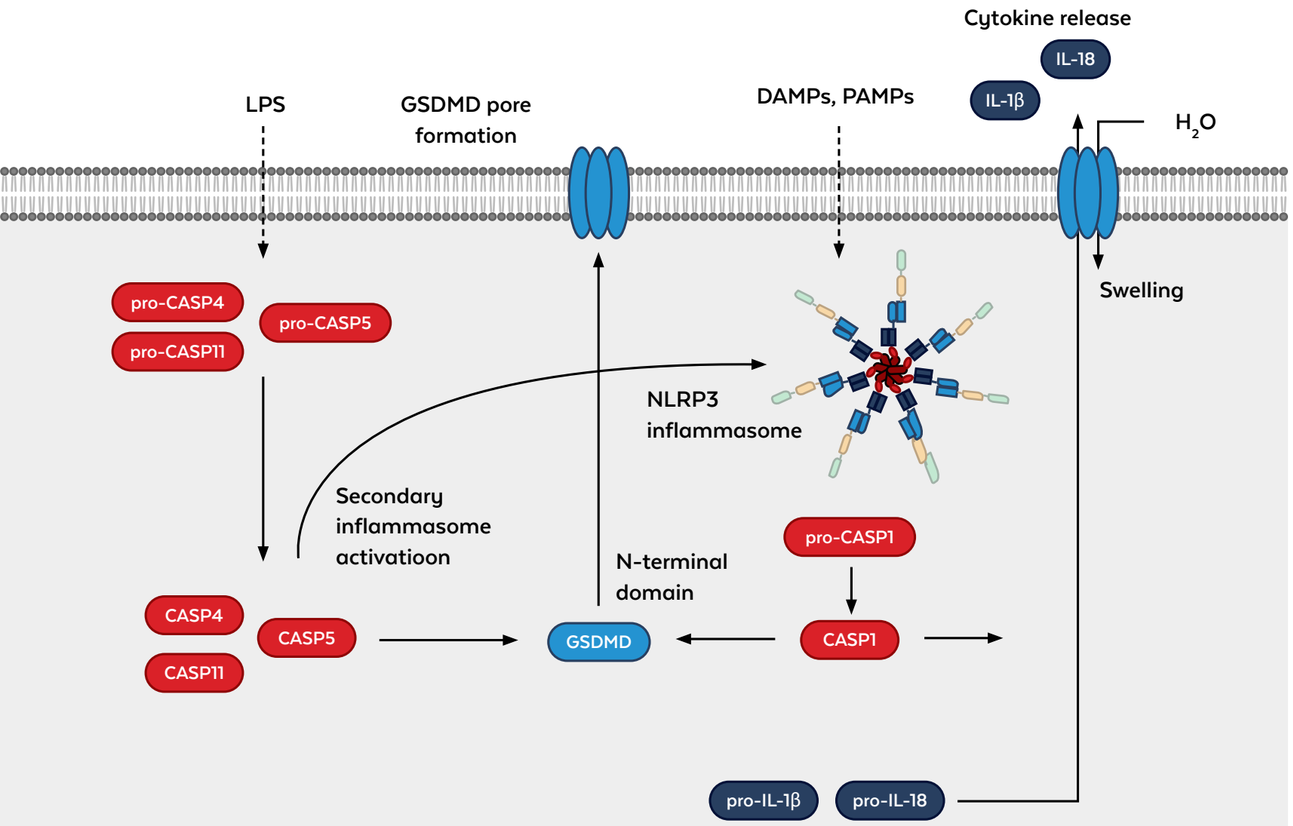
Product	Reactivity	Applications	Item No.
ATP7B Antibody	Human, Mouse, Rat	WB, IHC, IF, IP	ABIN1686618
DLAT Antibody	Human, Mouse, Rat, Dog	WB, IHC	ABIN629688
FDX1 Antibody	Human	WB, IHC	ABIN6140571
GSH Antibody	Broad	IHC, ELISA	ABIN6994369
Hsp70 Antibody	Broad	WB, IHC, FC, ELISA	ABIN6656050
LIAS Antibody	Human	WB, IHC, ELISA	ABIN2145837

References

1. Tsvetkov, P., Coy, S., Petrova, B., Dreishpoon, M., Verma, A., Abdusamad, M., Rossen, J., Joesch-Cohen, L., Humeidi, R., Spangler, R. D., Eaton, J. K., Frenkel, E., Kocak, M., Corsello, S. M., Lutsenko, S., Kanarek, N., Santagata, S., & Golub, T. R. (2022). Copper induces cell death by targeting lipoylated TCA cycle proteins. Science (New York, N.Y.), 375(6586), 1254–1261.
2. Rowland, E. A., Snowden, C. K., & Cristea, I. M. (2018). Protein lipoylation: an evolutionarily conserved metabolic regulator of health and disease. Current opinion in chemical biology, 42, 76–85.

Continue: Pyroptosis

Pyroptosis



Although already discovered in 1992 as a form of programmed cell death (PCD) caused by pathogen infection of macrophages[1], the term **pyroptosis** ? was only introduced in 2001 to describe the rapid release of inflammatory cytokines such as interleukin-1β (IL-1β) and IL-18 from dying cells.[2]

Continue: Pyroptosis

Pyroptosis thereby helps to combat intracellular infections by eliminating the affected cell and exposing the pathogen but is not limited to host defense. Some viruses such as SARS-CoV-2 can induce pyroptosis, which contributes to the development of an excessive immune response known as the “cytokine storm”.[3]

Over the years, several mediators of pyroptosis were identified. While initial studies showed a dependence on caspase-14, it is now clear that other caspases such as caspases 4, 5, and 11 can also mediate pyroptosis.[5]

Caspase-1 Antibody
(200-301-H62)

Pyroptosis is triggered by Caspase-1 after its activation by various inflammasomes and results in lysis of the affected cell. Caspase-1 (ICE, IL-1β converting enzyme) is similar to the cell death gene CED-3 of Caenorhabditilis elegans and regulates multiple proinflammatory cytokines, including interleukin-1β and interferon-gamma-inducing factor.

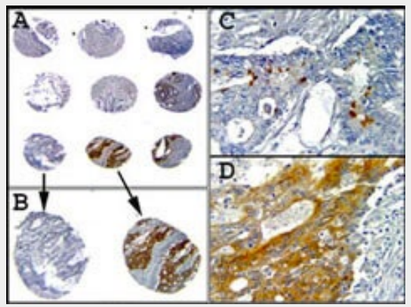


Fig. Immunohistochemistry of mouse anti-Caspase-1 antibody. Tissue: human colon cancer. Fixation: formalin fixed paraffin embedded. Antigen retrieval: not required. Primary antibody: Caspase-1 at 5 µg/mL for 1 h at RT. Secondary antibody: Peroxidase mouse secondary antibody at 1:10,000 for 45 min at RT. Localization: Caspase-1 is a cytoplasmic protein. Staining: Caspase-1 has a DAB chromogen and Hematoxylin counterstain. A). Only rare staining is observed. B). Abundant staining is observed. A and B). Two of these sections are shown at higher magnifications. C). Differential staining was observed.

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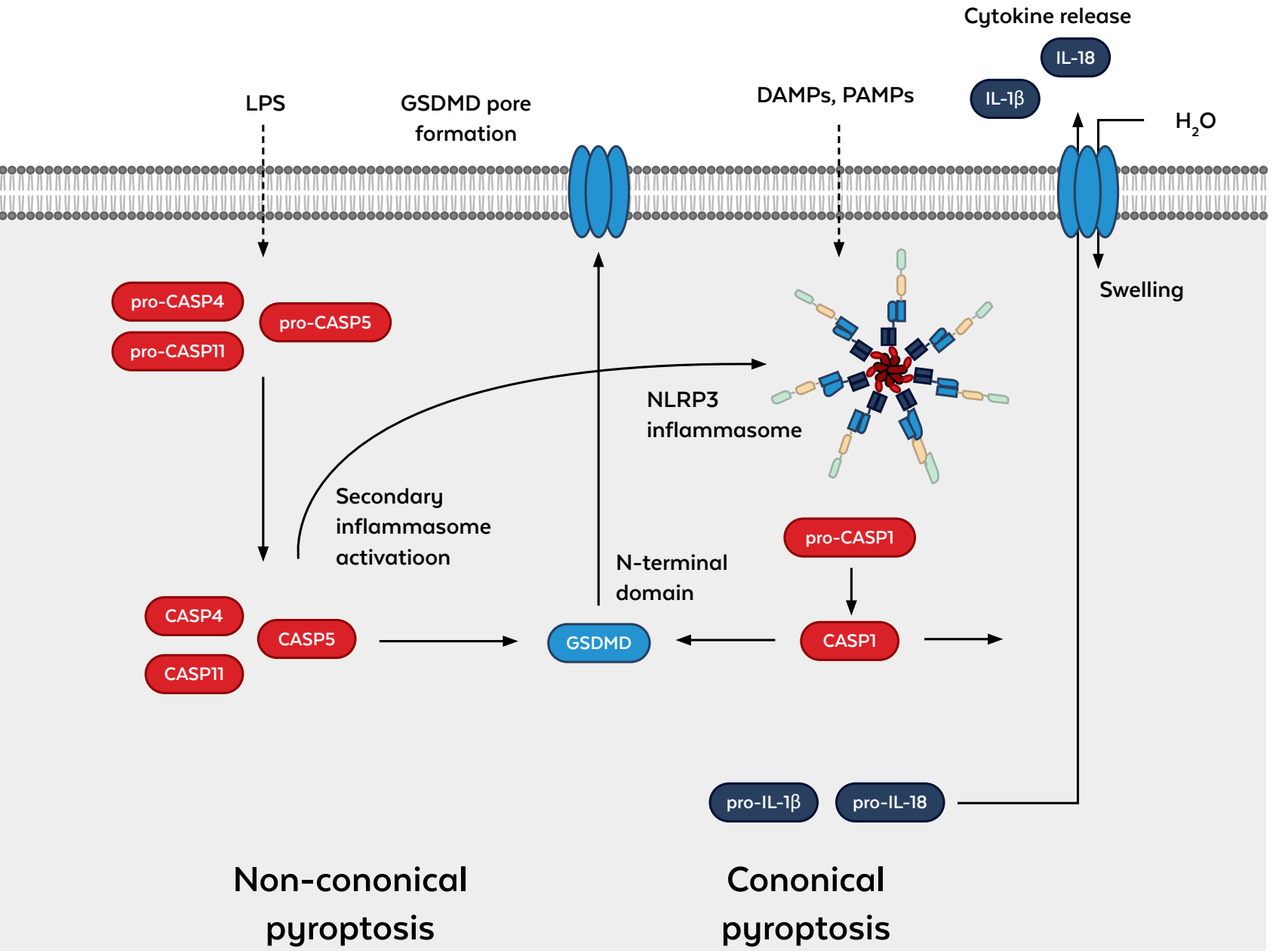
Continue: Pyroptosis



Based on the activating caspases, the signaling pathways can be divided into canonical (caspase-1) and non-canonical signaling pathways (caspase-4, 5, and 11).[6]

In the **canonical pathway**, caspase-1 is activated by inflammasomes such as the NLRP3 inflammasome, which are multimeric protein complexes reacting to different stimuli such as damage- or pathogen-associated molecular patterns (DAMPs or PAMPs respectively).

On the **non-canonical pathway**, the pyroptosis-triggering caspases directly serve as receptors for intracellular lipopolysaccharide (LPS) from Gram-negative bacteria, activating the NLRP3 inflammasome in a secondary step.[7]

Continue: Pyroptosis

The effector of pyroptosis that ultimately leads to cell death by membrane rupture was not identified until 2015.

Gasdermin D (GSDMD), as it is called, belongs to a protein family with a conserved structure. Gasdermin D is activated by proteolytic cleavage by caspases, releasing its n-terminal gasdermin domain and forming a pore in the plasma membrane. Caspase-1 cleaves pro-IL-1β and pro-IL-18 to generate mature cytokines which are then released through these pores prior to H_2O influx and membrane rupture.[8]

Since we are only beginning to understand the various components and interactions of this pathway, the future will hold many more insights.

Pyroptosis Antibodies

Product	Reactivity	Applications	Item No.
ASC Antibody	Human	WB, IHC, ELISA	600-401-Y67
Caspase-1 Antibody	Human	WB, IHC, ELISA	600-401-AC4
Caspase-1 Antibody	Human	WB, IF, IHC, ELISA	600-401-AC5
Caspase-1 Antibody	Human, Mouse	WB, IF, IHC	200-301-H62
Caspase-4 Antibody	Human, Mouse	WB, IF, IHC, ELISA	600-401-AD3
Caspase-4 Antibody	Human	WB, IF, IHC, ELISA	600-401-AD4
Caspase-5 Antibody	Human	WB, ELISA	600-401-AD5
Caspase-5 Antibody	Human	WB, IF, IHC, ELISA	600-401-AD6
IL-1 Beta Antibody	Mouse	WB, IF, IHC	210-401-319
IL-1 Beta Antibody	Human	WB, IHC, ELISA Functional Assay	209-401-301

Continue: Pyroptosis Antibodies

IL-1 Beta Antibody	Human, Dog, Primate	WB	209-401-B73
Mouse IL-18 Antibody	Mouse	WB, IF, IHC	210-401-323
NALP3 Antibody	Human, Mouse	WB, IF, IHC, ELISA	600-401-H02
NLRP3 Antibody	Human, Mouse, Rat	WB, IF, IHC, FC	600-401-R14

Pyroptosis Assays

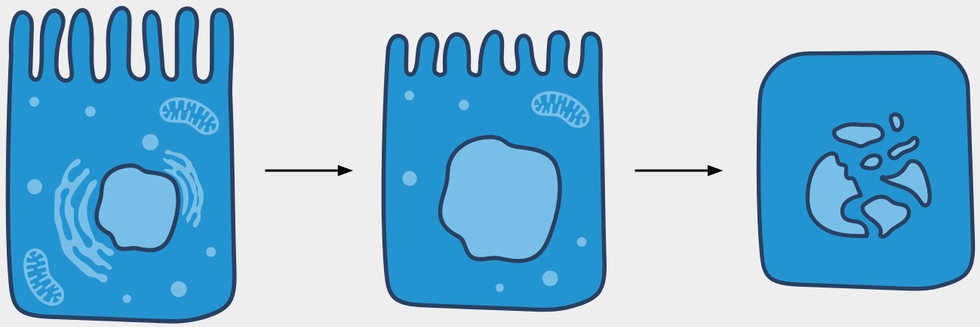
Product	Reactivity	Detection Range	Item No.
Human IL-1 beta ELISA Kit	Human	3.9 pg/mL - 250 pg/mL	KOA0209
Mouse IL-1 beta ELISA Kit	Mouse	12.5 pg/mL - 800 pg/mL	KOA0211
Rat IL-1 beta ELISA Kit	Rat	31.2 pg/mL - 2000 pg/mL	KOA0210
Human IL-18 ELISA Kit	Human	31.2 pg/mL - 2,000 pg/ml	KOA0523
Rat IL-18 ELISA Kit	Rat	15.6 pg/mL - 1000 pg/mL	KOA0362

References

1. Zychlinsky, A., Prevost, M. C., & Sansonetti, P. J. (1992). Shigella flexneri induces apoptosis in infected macrophages. Nature, 358(6382), 167-169.
2. D'Souza, C. A., & Heitman, J. (2001). Dismantling the Cryptococcus coat. Trends in microbiology, 9(3), 112-113.
3. Ferreira, A. C., Soares, V. C., de Azevedo-Quintanilha, I. G., Dias, S. D. S. G., Fintelman-Rodrigues, N., Sacramento, C. Q., Mattos, M., de Freitas, C. S., Temerozo, J. R., Teixeira, L., Damaceno Hottz, E., Barreto, E. A., Pão, C. R. R., Palhinha, L., Miranda, M., Bou-Habib, D. C., Bozza, F. A., Bozza, P. T., & Souza, T. M. L. (2021). SARS-CoV-2 engages inflammasome and pyroptosis in human primary monocytes. Cell death discovery, 7(1), 43.
4. Hilbi H, Moss JE, Hersh D, Chen Y, Arondel J, Banerjee S, Flavell RA, Yuan J, Sansonetti PJ, Zychlinsky A. Shigella-induced apoptosis is dependent on caspase-1 which binds to IpaB. J Biol Chem. 1998 Dec 4;273(49):32895-900.
5. Man SM, Kanneganti TD. Converging roles of caspases in inflammasome activation, cell death and innate immunity. Nat Rev Immunol. 2016 Jan;16(1):7-21.
6. Wei, X., Xie, F., Zhou, X., Wu, Y., Yan, H., Liu, T., Huang, J., Wang, F., Zhou, F., & Zhang, L. (2022). Role of pyroptosis in inflammation and cancer. Cellular & molecular immunology, 19(9), 971-992.
7. Downs, K. P., Nguyen, H., Dorfleutner, A., & Stehlik, C. (2020). An overview of the non-canonical inflammasome. Molecular aspects of medicine, 76, 100924.
8. Kovacs, S. B., & Miao, E. A. (2017). Gasdermins: Effectors of Pyroptosis. Trends in cell biology, 27(9), 673-684.

Continue: Erebosis

Erebosis



During erebosis, the cells lose important cytoskeletal proteins while the microvilli on the apical surface of the gut cell shrink. The nucleus initially enlarges, then flattens, shrinks, and finally fragments.

More and more cell death mechanisms are being discovered at an increasing rate. One of the newest members is erebosis, which was first described in the April 2022 issue of PLoS Biology by Ciesielski *et al.*[1] This novel form of cell death was found in the Drosophila intestine and was named **erebosis**. ?

Gut epithelial cells such as enterocytes are in a constant state of renewal. However, cells dying by apoptosis were always difficult to detect in this environment.

Continue: Erebosis

GFP Antibody
(600-901-215)

With new methods and the use of Rockland's anti-GFP antibody, researchers from Japan were able to discover an alternative signaling pathway featuring the accumulation of angiotensin-converting enzymes.

During their staining analysis with DAPI, Hoechst, GFP, and RFP, it emerged that DNA staining was occasionally weak, while staining with fluorescent proteins was gradually lost in this process.

The loss of signal started with cytoplasmic GFP, nuclear GFP, and lastly nuclear RFP. This observation raised the question of whether these proteins are denatured or degraded. Fortunately, this question could be easily answered with GFP and RFP antibodies that detect denatured but not degraded proteins.

Consistent with other experiments shown in the paper, this proved that the loss of signal was due to degradation.

In addition, flat nuclei and loss of cytoskeleton and cell organelles add to the features of erebosis. The authors speculate that erebosis is a coordinated cell death mechanism that enables the enterocyte flux under normal physiological conditions.

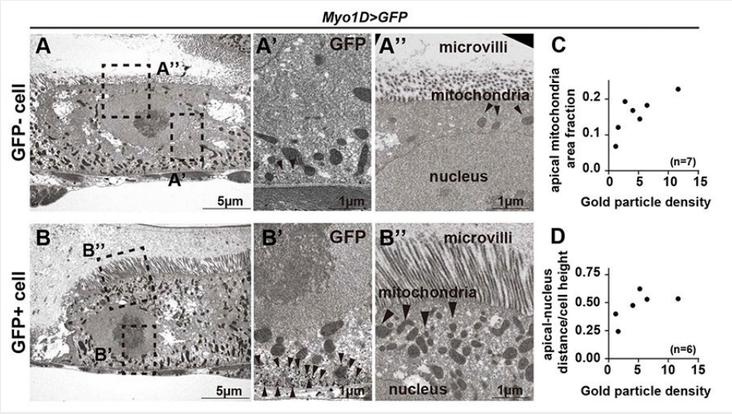


Fig. Immuno-electron microscopy of (A) GFP-negative and (B) GFP-positive cells using Rockland's anti-GFP antibody #600-901-215. Arrowheads in (A') and (B') indicate sparse GFP labeling in GFP- cells. (A'') Erebotic cells show short microvilli and fewer mitochondria when compared to (B'') GFP+ cells. (Image used under CC BY 4.0 from Ciesielsi *et al.*)

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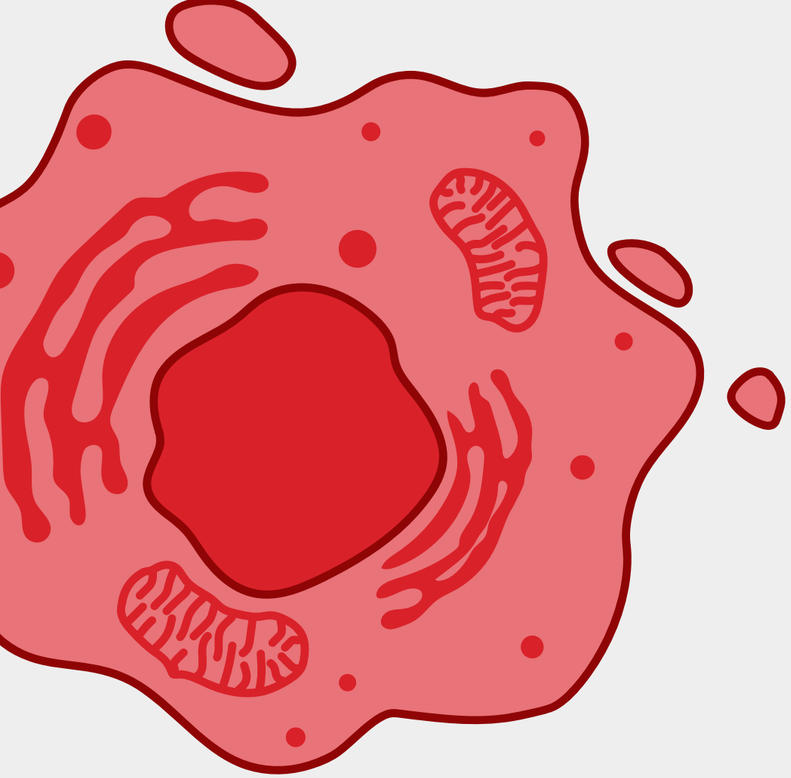
The observation of gradual GFP and RFP loss in erebosis makes fluorescent proteins and antibodies targeted against them versatile tools in the study of this new form of cell death. Utilizing these tools might also help to discover even more cell death pathways. As Andreas Bergmann aptly summarizes in his commentary on the first publication of erebosis: “Although there are over a dozen types of cell death known, there is clearly more to discover in this field.”[2]

Anti-GFP Antibodies

Product	Reactivity	Applications	Item No.
GFP Antibody	eGFP, rGFP, WT	WB, IHC, IF, Dot Blot, Purification, ELISA	600-901-215
GFP Antibody	eGFP, rGFP, WT	WB, IHC, IF, IP, EM, FC, FISH, Purification, ELISA	600-101-215
GFP Antibody	eGFP, RS-GFP, S65T-GFP, WT, YFP	WB, IHC, IF, IP EM, Purification, ELISA	600-401-215
GFP Monoclonal Antibody	eGFP, rGFP, WT	WB, IHC, IF, IP, ChIP, FC, Dot Blot, ELISA	600-301-215
GFP Antibody Dylight™ 488 Conjugated Pre-Adsorbed	eGFP, rGFP, WT	WB, IHC, IF, IP, FC, Dot Blot	600-141-215

References

1. Ciesielski, H. M., Nishida, H., Takano, T., Fukuhara, A., Otani, T., Ikegawa, Y., Okada, M., Nishimura, T., Furuse, M., & Yoo, S. K. (2022). Erebosis, a new cell death mechanism during homeostatic turnover of gut enterocytes. PLoS biology, 20(4), e3001586
2. Bergmann A. (2022). Erebosis is a new type of cell death for tissue homeostasis in the Drosophila intestine. PLoS biology, 20(4), e3001614.



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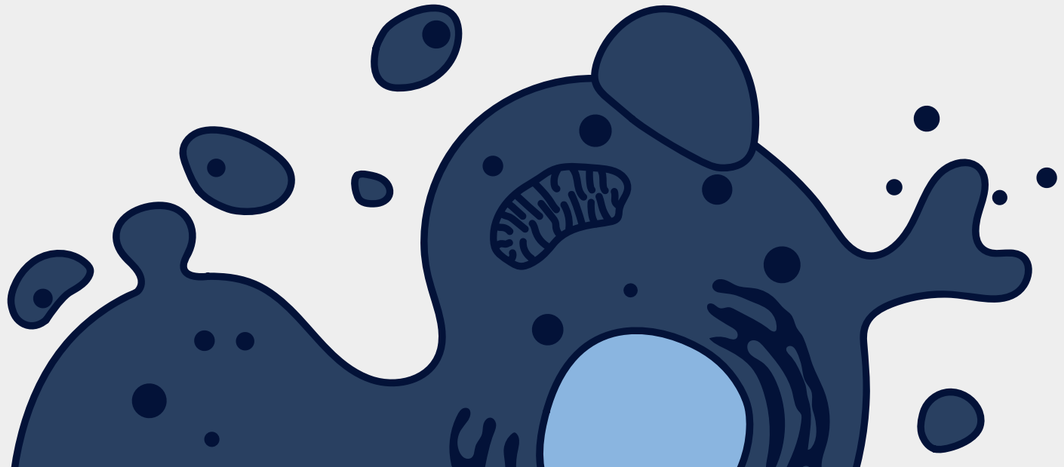

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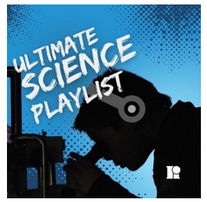

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