Elabscience®

The Cell Death Compendium

- Apoptosis
- Necrosis
- Pyroptosis
- Cuproptosis
- PANoptosis
- Autophagy
- Necroptosis
- Ferroptosis
- Disulfidptosis

Elabscience Bionovation Inc.

CONTENTS

Cell Death

Cell Death Introduction

Cell death refers to the irreversible cessation of cellular functions and the termination of a cell's biological processes, which commonly occurs in normal tissues. It is essential for maintaining tissue homeostasis and structural integrity. Cell death can be categorized into two main types: active forms (such as programmed cell death, including apoptosis) and passive forms (such as necrosis) .

Expertise in Cell Status Detection, Providing One-stop Solution

01 Apoptosis

Also known as programmed cell death or cell suicide, apoptosis is a genetically controlled process of active cell death. Apoptosis is an organism's self-protection against the release of inflammatory cell contents into the internal environment.

02 Autophagy

Refers to the process in which damaged, denatured or senescence proteins and organelles are transported to lysosomes under the regulation of Autophagy associated gene (Atg) , and the lysosomes digest and degrade them.

03 Necrosis

The death of cells induced by extreme physical, chemical or other serious pathological factors, which is pathological cell death.

04 Necroptosis

A process by which cells self-destruct when apoptosis is blocked and activated by extracellular signals (death receptor-ligand binding) or intracellular signals (foreign microbial nucleic acids) .

05 Pyroptosis

A type of regulated cell death characterized by inflammatory necrosis. It depends on Gasdermin family proteins forming plasma membrane pores, leading to cell swelling, membrane rupture, and the release of inflammatory factors, thereby triggering a strong immune response.

06 Ferroptosis

A non-regulatory cell death mode caused by iron-dependent oxidative damage, which was first proposed in 2012. Ferroptosis is closely related to the pathophysiological processes of various diseases such as tumors, metabolic diseases, nervous system diseases and kidney injury.

07 Cuproptosis

A mode of cell death in which copper binds directly to the fatty acylated component of the TCA cycle, resulting in the aggregation of fatty acylated proteins and the loss of iron-sulfur cluster proteins, leading to protein-toxic stress and ultimately cell death.

08 Disulfidptosis

A newly identified form of cell death triggered by disulfide stress due to excessive cystine accumulation.

09 PANoptosis

A regulated cell death pathway controlled by the PANoptosome complex, exhibiting features of pyroptosis (P) , apoptosis (A) , and/ necroptosis (N) . However, it cannot be fully explained by any of these three pathways alone.

Apoptosis

Definition of Apoptosis

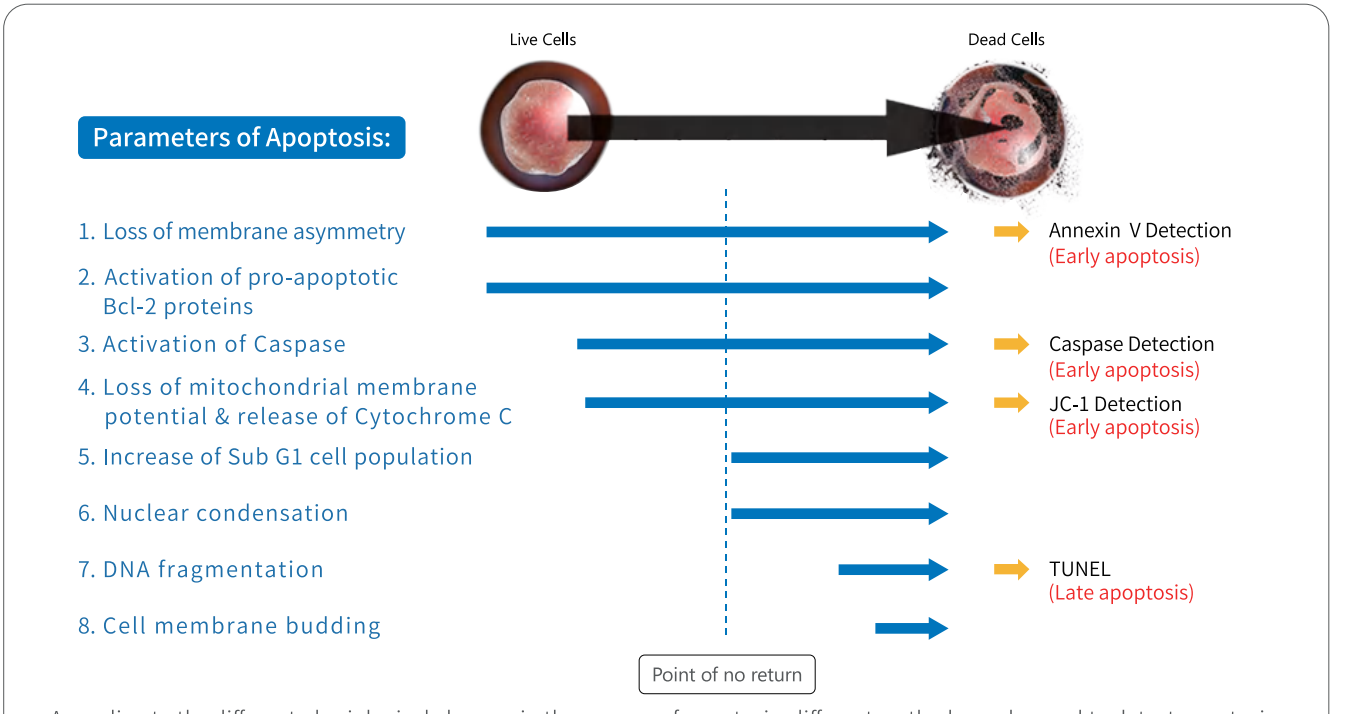
Apoptosis, also known as programmed cell death or cell suicide, is a genetically regulated process of active cell death. It serves as a protective mechanism to prevent the release of inflammatory cellular contents into the surrounding environment.

Methods for Apoptosis Detection

In the process of apoptosis, there are many symbolic physiological phenomena. With the further deepening of apoptosis, the phenomenon of apoptosis is also different.

Early apoptotic stage: Changes in cell membrane structure, Externalization of phosphatidylserine (PS) on the cell membrane, Activation of apoptosis-related proteins (e.g., Bcl-2) , Activation of intracellular caspase enzymes, Collapse of mitochondrial membrane potential.

- **Early Apoptotic Stage:** Changes in cell membrane structure, Externalization of phosphatidylserine (PS) on the cell membrane, Activation of apoptosis-related proteins (e.g., Bcl-2) , Activation of intracellular caspase enzymes, Collapse of mitochondrial membrane potential
- **Middle Apoptotic Stage:** Increase in sub-G1 phase cell population.
- **Late Apoptosis Stage:** Nucleus shrinks, DNA fragments, and the cell membrane budding to form apoptotic bodies.



According to the different physiological changes in the process of apoptosis, different methods can be used to detect apoptosis.

Fig. 1. Schematic Diagram Illustrating the Characteristics of the Apoptosis Process

Morphological Observation

The morphological changes of apoptosis can be observed under a microscope, which could be observed by HE staining, acridine orange staining, and trypan blue staining. These observations can be made through light microscope or fluorescence microscope, with apoptosis being detectable based on cellular coloration and nucle. ar/cytoplasmic alterations.

Annexin V Detection of PS Externalization

■ Detection Principle

Normal cells (living cells) have an intact cell membrane, with phosphotidylserine (PS) localized on the inner leaflet of the cell membrane. During apoptosis, the cell membrane structure undergoes changes, leading to PS externalization (flipping to the outer surface) . This phenomenon occurs in the early stages of apoptosis.

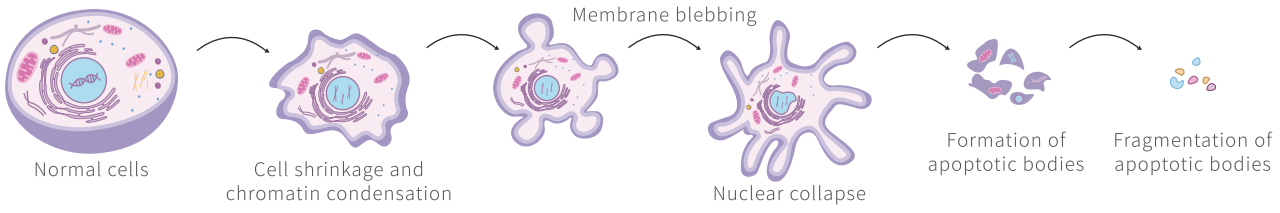


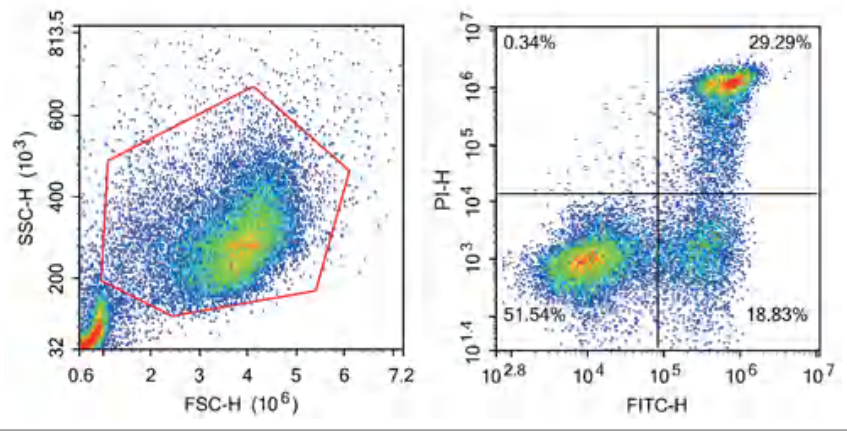
Fig. 2. Schematic diagram of the apoptosis process

Annexin V is a protein that specifically bind to phosphatidylserine (PS) . During apoptosis, fluorochrome-labeled Annexin V can be used to detect PS externalization by binding to exposed PS on the cell surface. The fluoro. chrome signal from Annexin V allows for the identification of apoptotic cells.

In the early stage of apoptosis, PS is externalized while the cell membrane remains intact. As a result, membrane-im. permeable nuclear dyes (e.g., propidium iodide, PI) cannot enter the cell, preventing nuclear staining.

In the late apoptotic stage or necrosis, the cell membrane loses integrity, permitting PI uptake and nuclear staining. Thus, dual staining with Annexin V and PI can distinguish early apoptosis (Annexin V^+/PI^-) from late apoptosis or necrosis (Annexin V^+/PI^+) .

Fig. 3. Annexin V experimental results (E-CK-A211)



Detection Instrument

Fluorescence Microscope / Flow Cytometer

Experimental Considerations

Precautions for Samples before Experiment

① Adherent cells should use EDTA-free pancreatic enzymes for digestion as much as possible. If EDTA containing pancreatic enzymes must be used, after the termination of digestion, the cells should be washed to remove EDTA as much as possible.

② Samples of cells and tissues that are difficult to digest can be digested in batches to avoid excessive digestion of the first digested cells,and avoid false positive results.

Precautions for Samples in the Experiment

Mechanical forces will damage the cells, and any violent behavior during manipulation increases the percentage of apoptosis in the cells themselves. During the experiment, blow the cells as little as possible.

Elabscience® Annexin V Featured Products

Cat. No.	Product Name	Fluorochrome	Size
E-CK-A211	Annexin V-FITC/PI Apoptosis Kit	FITC/PI	20/50/100/200 Assays
E-CK-A212	Annexin V-FITC/7-AAD Apoptosis Kit	FITC/7-AAD	20/50/100/200 Assays
E-CK-A216	Annexin V-PE/7-AAD Apoptosis Kit	PE/7-AAD	20/50/100/200 Assays
E-CK-A217	Annexin V-APC/PI Apoptosis Kit	APC/PI	20/50/100/200 Assays
E-CK-A218	Annexin V-APC/7-AAD Apoptosis Kit	APC/7-AAD	20/50/100/200 Assays
E-CK-A258	Annexin V-APC/DAPI Apoptosis Kit	APC/DAPI	20/50/100/200 Assays

* For more products, please visit www.elabscience.com or contact your local distributor.

Elabscience® Annexin V Product Citations

Title	Journal	Product Cited
MARCKSL1–2 reverses docetaxel-resistance of lung adenocarcinoma cells by recruiting SUZ12 to suppress HDAC1 and elevate miR-200b	*Molecular Cancer*	Annexin V-FITC/PI Apoptosis Kit (E-CK-A211)
Cell-in-Cell-Mediated Entosis Reveals A Progressive Mechanism in Pancreatic Cancer	*Gastroenterology*	Annexin V-Elab Fluor® 647/DAPI Apoptosis Kit (E-CK-A254)
Cell membrane-camouflaged bufalin targets NOD2 and overcomes multidrug resistance in pancreatic cancer	*Drug Resistance Updates*	Annexin V-APC/PI Apoptosis Kit (E-CK-A217)
Bacterial outer membrane vesicle based versatile nanosystem boosts the efferocytosis blockade triggered tumor-specific immunity	*Nature Communications*	Annexin V-FITC/DAPI Apoptosis Kit (E-CK-A252)
Targeting dual-specificity tyrosine phosphorylation-regulated kinase 2 with a highly selective inhibitor for the treatment of prostate cancer	*Nature Communications*	Annexin V-APC/PI Apoptosis Kit (E-CK-A217)

Title	Journal	Product Cited
Pyroptosis Remodeling Tumor Microenvironment to Enhance Pancreatic Cancer Immunotherapy Driven by Membrane Anchoring Photosensitizer	*Advanced Science*	Annexin V-APC/PI Apoptosis Kit (E-CK-A217)
Phytochemical natural killer cells reprogram tumor microenvironment for potent immunotherapy of solid tumors	*Biomaterials*	Annexin V-FITC/PI Apoptosis Kit (E-CK-A211)
VPS41-mediated incomplete autophagy aggravates cadmium-induced apoptosis in mouse hepatocytes	*Journal of Hazardous materials*	Annexin V-APC/PI Apoptosis Kit (E-CK-A217)
The translational landscape of human vascular smooth muscle cells identifies novel short open reading frame-encoded peptide regulators for phenotype alteration	*Cardiovascular Research*	Annexin V-FITC/7-AAD Apoptosis Kit (E-CK-A212)
Benzaldehyde-tethered fluorous tags for cytosolic delivery of bioactive peptides	*Journal of Controlled Release*	Annexin V-FITC/PI Apoptosis Kit (E-CK-A211)

* For more product citations, please visit www.elabscience.com.

Activation of Apoptosis-related Proteins and Related Detection

Apoptosis-related Protein

During apoptosis, some proteins in the cell are activated, and the occurrence of apoptosis can be determined by detecting these proteins.

Table 1. Apoptosis-activating Proteins

Marker	Full Name	Marker	Full Name
BAG3	Bcl2 Associated Athanogene 3	BAX	Bcl-2 Associated X Protein
Bcl-2	B-cell Leukemia/Lymphoma 2	CASP1	Caspase 1
CASP3	Caspase 3	CASP4	Caspase 4
CASP8	Caspase 8	CASP9	Caspase 9
FAS/CD95	Factor Related Apoptosis	FASL/TNFSF6	Factor Related Apoptosis Ligand
SIRT1	Sirtuin 1	Surv	Survivin
TNFRSF1A	Tumor Necrosis Factor Receptor Superfamily, Member 1A	TNF-α	Tumor Necrosis Factor Alpha
TRAIL/TNFSF10	Tumor Necrosis Factor Related Apoptosis Inducing Ligand	WISP1	WNT1 Inducible Signaling Pathway Protein 1

ELISA Method

Some apoptotic proteins can be detected by ELISA. Elabscience® apoptosis-related proteins can be detected by ELISA products as follows.

Cat. No.	Product Name
E-EL-H0562	Human BAX ELISA Kit
E-EL-R0098	Rat BAX ELISA Kit
E-EL-H0114	Human Bcl-2 ELISA Kit
E-EL-R0096	Rat Bcl-2 ELISA Kit
E-EL-H0016	Human CASP1 (Caspase 1) ELISA Kit
E-EL-M0201	Mouse CASP1 (Caspase 1) ELISA Kit
E-EL-R0371	Rat CASP1 (Caspase 1) ELISA Kit
E-EL-H0017	Human CASP3 (Caspase 3) ELISA Kit
E-EL-M0238	Mouse CASP3 (Caspase 3) ELISA Kit
E-EL-R0160	Rat CASP3 (Caspase 3) ELISA Kit
E-EL-H0660	Human CASP4 (Caspase 4) ELISA Kit
E-EL-H0659	Human CASP8 (Caspase 8) ELISA Kit
E-EL-M0063	Mouse CASP8 (Caspase 8) ELISA Kit
E-EL-H0663	Human CASP9 (Caspase 9) ELISA Kit
E-EL-H0067	Human FAS/CD95ELISA Kit
E-EL-M3061	Mouse FAS/CD95ELISA Kit
E-UNEL-H0143	Uncoated Human FAS/CD95 ELISA Kit
E-UNEL-R0027	Uncoated Rat FAS/CD95 ELISA Kit
E-EL-H0068	Human FASL/TNFSF6 ELISA Kit
E-EL-M0028	Mouse FASL/TNFSF6 ELISA Kit

Cat. No.	Product Name
E-UNEL-H0144	Uncoated Human FASL/TNFSF6 ELISA Kit
E-EL-H1546	Human SIRT1 (Sirtuin 1) ELISA Kit
E-EL-M0350	Mouse SIRT1 (Sirtuin 1) ELISA Kit
E-EL-R1102	Rat SIRT1 (Sirtuin 1) ELISA Kit
E-EL-H0217	Human TNFRSF1A ELISA Kit
E-UNEL-H0151	Uncoated Human TNFRSF1A ELISA Kit
E-UNEL-M0040	Uncoated Mouse TNFRSF1A ELISA Kit
E-EL-H0109	Human TNF-α ELISA Kit
E-EL-M3063	Mouse TNF-α ELISA Kit
E-EL-R2856	Rat TNF-α ELISA Kit
E-EL-P0010	Porcine TNF-α ELISA Kit
E-MSEL-M0002	MS Mouse TNF-α ELISA Kit
E-UNEL-H0175	Uncoated Human TNF-α ELISA Kit
E-UNEL-M0103	Uncoated Mouse TNF-α ELISA Kit
E-UNEL-R0057	Uncoated Rat TNF-α ELISA Kit
E-EL-H1593	Human TRAIL/TNFSF10 ELISA Kit
E-EL-M1084	Mouse TRAIL/TNFSF10 ELISA Kit
E-UNEL-H0219	Uncoated Human TRAIL/TNFSF10 ELISA Kit
E-EL-H5542	Human WISP1 ELISA Kit

WB Method

Some proteins can be detected using WB. Elabscience® apoptosis-related protein detection products are as follows.

Cat. No.	Product Name	Reactivity
E-AB-40660	BAG3 Polyclonal Antibody	Human, Rat
E-AB-70300	CASP1 Polyclonal Antibody	Human, Rat
E-AB-22115	Active CASP3 Monoclonal Antibody	Human, Rat, Mouse

Cat. No.	Product Name	Reactivity
E-AB-60646	(KO Validated) Caspase-3 Polyclonal Antibody	Human, Rat
E-AB-60017	Caspase-3 Polyclonal Antibody	Human, Mouse
E-AB-22035	CASP9 Monoclonal Antibody	Human
E-AB-14381	Survivin Polyclonal Antibody	Human
E-AB-33121	TNF alpha Polyclonal Antibody	Human
E-AB-70316	TRAIL Polyclonal Antibody	Human, Rat, Mouse

IHC Method

Some proteins can be detected by IHC method. Elabscience® apoptosis-related proteins can be detected by IHC method as follows.

Cat. No.	Product Name	Reactivity
E-AB-22128	BAX Monoclonal Antibody	Human, Rat, Mouse
E-AB-22213	CASP3 Monoclonal Antibody	Human
E-AB-22004	BCL2 Monoclonal Antibody	Human
E-AB-40521	BAX Polyclonal Antibody	Zebrafish
E-AB-60012	Bcl-2 Polyclonal Antibody	Human, Rat, Mouse
E-AB-60646	(KO Validated) Caspase-3 Polyclonal Antibody	Human, Rat
E-AB-53537	CASP4 Polyclonal Antibody	Human
E-AB-22107	CASP8 Monoclonal Antibody	Human, Rat, Mouse
E-AB-19664	CASP8 Polyclonal Antibody	Human
E-AB-22035	CASP9 Monoclonal Antibody	Human
E-AB-12941	CASP9 (active) Polyclonal Antibody	Human
E-AB-60760	Caspase-9 Polyclonal Antibody	Human, Rat, Mouse
E-AB-70174	FAS Monoclonal Antibody	Human, Mouse
E-AB-40063	FAS Polyclonal Antibody	Mouse
E-AB-70071	SIRT1 Polyclonal Antibody	Human, Rat, Mouse
E-AB-22112	Survivin Monoclonal Antibody	Mouse
E-AB-52065	TNF Polyclonal Antibody	Human

Elabscience® Apoptosis-related Protein Product Citations

Title	Journal	Product Cited
Novel combination strategy of high intensity focused ultrasound (HIFU) and checkpoint blockade boosted by bioinspired and oxygen-supplied nanoprobe for multimodal imaging-guided cancer ther	*Journal for ImmunoTherapy of Cancer*	Mouse TNF-α (Tumor Necrosis Factor Alpha) ELISA Kit (E-EL-M3063)
Exploring Cardiac Impact of Oral Nicotine Exposure in a Transplantable Neoplasm Mice Model: Insights from Biochemical Analysis, Morphometry, and Molecular Docking: Chlorella vulgaris Green Algae Support	*Toxicology*	Uncoated Mouse TNF-α (Tumor Necrosis Factor Alpha) ELISA Kit (E-UNEL-M0103)
Cycas pectinata stimulates germ cell proliferation in mouse testes	*Process Biochemistry*	Active CASP3 Monoclonal Antibody (E-AB-22115)
Investigation of the efficacy of diosmin against organ damage caused by bendiocarb in male Wistar albino rats	*Environmental Science and Pollution Research*	Caspase-3 Polyclonal Antibody (E-AB-60017)
Quinacrine inhibits HIF-1α/VEGF-A mediated angiogenesis by disrupting the interaction between cMET and ABCG2 in patient-derived breast cancer stem cells	*Phytomedicine*	TNF alpha Polyclonal Antibody (E-AB-33121)
Royal jelly protects brain tissue against fluoride-induced damage by activating Bcl-2/NF-κB/caspase-3/caspase-6/Bax and Erk signaling pathways in rats	*Environmental Science and Pollution Research*	BAX Monoclonal Antibody (E-AB-22128)
The petroleum ether extract of Brassica rapa L. induces apoptosis of lung adenocarcinoma cells via the mitochondria-dependent pathway	*Food & Function*	CASP9 Monoclonal Antibody (E-AB-22035)
Suppression of EGFR/PKC-δ/NF-κB Signaling Associated With Imipramine-Inhibited Progression of Non-Small Cell Lung Cancer	*Frontiers in Oncology*	FAS Polyclonal Antibody (E-AB-40063)
SIRT1 Activation Attenuates Bone Cancer Pain by Inhibiting mGluR1/5	*Cellular and Molecular Neurobiology*	SIRT1 Polyclonal Antibody (E-AB-70071)
The rejuvenating influence of young plasma on aged intestine	*Journal of Cellular and Molecular Medicine*	TNF Polyclonal Antibody (E-AB-52065)

* For more product citations, please visit www.elabscience.com.

Caspases Activation and Detection

Detection Principle

Caspases (Cysteinyl-aspartic acid proteases) are a family of proteases that play a central role in apoptosis. Normally existing as inactive zymogens in the cytoplasm, caspases become activated by various stimuli. Once activated, they cleave critical cellular proteins, triggering apoptosis through a cascade of sequential caspase activation.

During early apoptosis, activated caspases initiate a series of apoptotic cascade reactions. Their activity can be measured using synthetic substrates containing caspase-specific peptide sequences linked to a chromogenic group (e.g., p-nitroaniline, pNA) . When cleaved by active caspases, the liberated pNA produces a yellow color detectable at 405 nm using a spectrophotometer or microplate reader, allowing quantification of caspase activity.

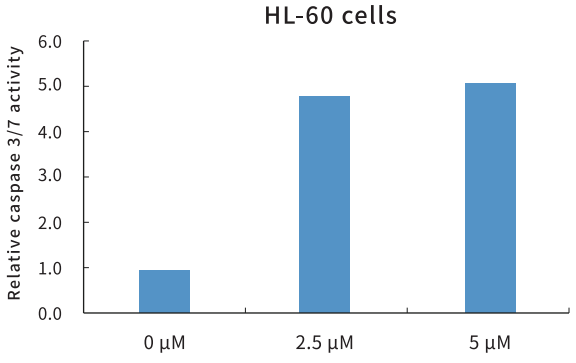
By measuring caspase activation levels, researchers can:

① Study apoptotic regulatory mechanisms

② Analyze cellular responses to various treatments

Fig. 4. Results of Caspase Experiment

HL-60 cells treated with camptothecin at different concentrations were cultured for 4 h and the activity of Caspase 3/7 was detected by E-CK-A373 (Caspase 3/7) .



Detection Instrument

Spectrophotometer, Enzyme-linked immunosorbent assay (ELISA) reader

Cautions

Cautions before Experiment

① The activation of Caspase could not be effectively detected when apoptosis was not obvious or when induction was excessive to cell death. It was suggested to set the gradient induction concentration and induction time before the experiment to select the best induction scheme.

② Before the experiment, the optimal induction density should be explored, and the recommended induction density is $5\sim10\times10^5$ /mL.

③ Before the experiment, pre-experimental detection can be performed, and the total protein detected is not less than 50 μg. Concentration can be measured using the Bradford method to increase the total amount of protein detected.

Cautions during Experiment

① Cell Lysis Buffer and cell lysis are performed on ice by swirling and mixing every 10 min to fully ensure enzyme activity.

② The optimal detection wavelength is 405 nm, if the instrument does not meet, 405±20 nm range can be selected.

③ The sample needs to be tested after removing bubbles to ensure the accuracy of the results.

Elabscience® Caspase Activity Detection Products

Cat. No.	Product Name	Size	Sample
E-CK-A381	Caspase 1 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue
E-CK-A382	Caspase 2 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue
E-CK-A383	Caspase 3/7 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue
E-CK-A384	Caspase 4 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue
E-CK-A386	Caspase 6 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue
E-CK-A388	Caspase 8 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue
E-CK-A389	Caspase 9 Activity Assay Kit (Colorimetric Method)	20/50 /100 Assays	Cell, Tissue

* For more products, please visit www.elabscience.com or contact your local distributor.

Elabscience® Caspase Activity Detection Product Citations

Title	Journal	Product Cited
Combined Donepezil with Astaxanthin via Nanostructured Lipid Carriers Effective Delivery to Brain for Alzheimer' s Disease in Rat Model	*International Journal of Nanomedicine*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Ozone fumigation promotes antioxidant activities to retard chlorophyll degradation and cell death in 'Jinda' chili during storage	*Gastroenterology*	Caspase 8 Activity Assay Kit (Colorimetric Method) (E-CK-A388) Caspase 9 Activity Assay Kit (Colorimetric Method) (E-CK-A389)
Isolation and characterization of N- (2-Hydroxyethyl) hexadecanamide from Colletotrichum gloeosporioides with apoptosis-inducing potential in breast cancer cells	*Biofactors*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Surface-Modified Polypyrrole-Coated PLCL and PLGA Nerve Guide Conduits Fabricated by 3D Printing and Electrospinning	*Biomacromolecules*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Tin oxide nanoparticles trigger the formation of amyloid β oligomers/protofibrils and underlying neurotoxicity as a marker of Alzheimer's diseases	*International Journal of Biological Macromolecules*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Methyl salicylate retards mitochondria-mediated programmed cell death in peel spotting of 'Sucrier' banana during storage	*Postharvest Biology and Technology*	Caspase 9 Activity Assay Kit (Colorimetric Method) (E-CK-A389)
Safety and Efficacy of Dietary Epigallocatechin Gallate Supplemen. tation in Attenuating Hypertension via Its Modulatory Activities on the Intrarenal Renin–Angiotensin System in Spontaneously Hypertensive Rats	*Nutrients*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Trimetazidine Modulates Mitochondrial Redox Status and Disrupted Glutamate Homeostasis in a Rat Model of Epilepsy	*Frontiers in Pharmacology*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
In vitro and in vivo anti-tumor activity of Antrodia salmonea against twist-overexpressing HNSCC cells: Induction of ROS-mediated autophagic and apoptotic cell death	*Food and Chemical Toxicology*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)

* Caspase series products will be upgraded in 2024, and the catalogue number will be changed. This table shows the new item number after the change.
* For more product citations, please visit www.elabscience.com.

Mitochondrial Membrane Potential Change Detection

Detection Principle

The decline of mitochondrial membrane potential (ΔΨm) is a hallmark event in early apoptosis, occurring prior to nuclear apoptotic features (chromatin condensation, DNA fragmentation) . Once ΔΨm collapses, apoptosis becomes irreversible. Monitoring ΔΨm changes allows for apoptosis detection.

JC-1, an ideal fluorescent probe for assessing ΔΨm in cells, tissues, or isolated mitochondria, exists in two forms with distinct emission spectra:
Normal cells (high ΔΨm) : JC-1 accumulates in mitochondria as aggregates, emitting red fluorescence (590 nm).
Early apoptosis (low ΔΨm) : JC-1 remains as monomers in the cytoplasm, emitting green fluorescence (530 nm).
The shift from red to green fluorescence indicates ΔΨm dissipation, enabling apoptosis determination.

Detection Instrument

Flow Cytometer, Fluorescence Microscopy, Laser Confocal Microscopy.

Cautions

Cautions before Experiment

① Good cell state is the key to the experiment. When the adherent cells are used for apoptosis detection, cell necrosis or apoptosis may be caused by pancreatic enzyme digestion, blowing and other treatments, which may have uncontrollable effects on the experimental results. Please handle with caution.

② The culture temperature is related to the cell type, 37°C is recommended for mammalian cells, and the appropriate temperature is selected according to the cell culture conditions for other species.

Cautions during Experiment

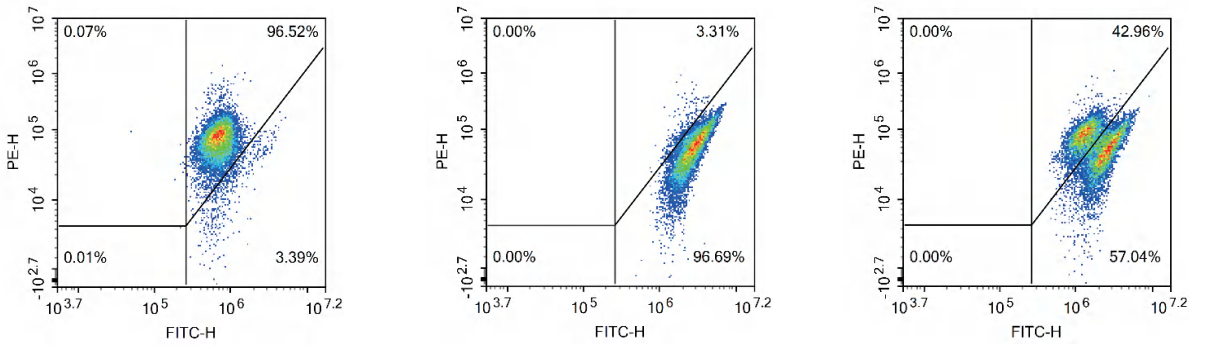
① In the case of fluorescence microscopy, the detection of JC-1 monomer can refer to the setting for observing other green fluorescence, such as GFP or FITC.

② The detection of JC-1 polymers can refer to the observation of other red fluorescence Settings, such as PI.

③ The presence of green fluorescence indicates a decreased mitochondrial membrane potential, and the cell is likely in the early stages of apoptosis. The presence of red fluorescence indicates that the mitochondrial membrane potential is relatively normal, and the cell state is also relatively normal.

Elabscience® JC-1 Product Introduction

▶Product Nmae: Mitochondrial Membrane Potential Assay Kit (with JC-1) ▶Catelogue：E-CK-A301 ▶Sample: Cell



Normal group Positive control (CCCP treatment) Experimental group (camptothecin induction 24 h)

Fig. 5. Flow Cytometry detection results

In the normal group, there were a few apoptotic cells and only a small amount of green fluorescence. After CCCP treatment, mitochondria lost membrane potential, and basically all cells showed green fluorescence. Camptothecin induced apoptosis of some cells.

Elabscience® JC-1 Product Citations

Title	Journal	Product Cited
Therapeutic targeting of P2X4 receptor and mitochondrial metabolism in clear cell renal carcinoma models	*Journal of Experimental & Clinical Cancer Research*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Dual Stimuli-Responsive Micelles for Imaging-Guided Mitochondri. on-Targeted Photothermal/Photodynamic/Chemo Combination Therapy-Induced Immunogenic Cell Death	*International Journal of Nanomedicine*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
A Second Near-Infrared Ru (II) Polypyridyl Complex for Synergistic Chemo-Photothermal Therapy	*Journal of Medicinal Chemistry*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Alpinetin inhibits neuroinflammation and neuronal apoptosis via targeting the JAK2/STAT3 signaling pathway in spinal cord injury	*CNS Neuroscience & Therapeutics*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Mitochondrial stress induces hepatic stellate cell activation in response to the ATF4/TRIB3 pathway stimulation	*Journal of Gastroenterology*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
The Novel Curcumin Derivative 1g Induces Mitochondrial and ER-Stress-Dependent Apoptosis in Colon Cancer Cells by Induction of ROS Production	*Frontiers in Oncology*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Ferroptosis-related signature and immune infiltration characterization in acute lung injury/acute respiratory distress syndrome	*Respiratory Research*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Resveratrol Protects Rat Ovarian Luteinized Granulosa Cells from H2O2-Induced Dysfunction by Activating Autophagy	*International Journal of Molecular Sciences*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Comparative physiological and transcriptome analysis provide insights into the inhibitory effect of 6-pentyl-2H-pyran-2-one on Clarireedia jacksonii	*Pesticide Biochemistry and Physiology*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)
Norlignans and phenolics from genus Curculigo protect corticoste. rone-injured neuroblastoma cells SH-SY5Y by inhibiting endoplasmic reticulum stress-mitochondria pathway	*Journal of Ethnopharmacology*	Mitochondrial Membrane Potential Assay Kit (with JC-1) (E-CK-A301)

* For more product citations, please visit www.elabscience.com.

TUNEL Detects Late Marcescent DNA Breaks

TUNEL Detection Principle

Late in apoptosis, DNA fragments, and the 3 '-OH exposed by the broken DNA can be catalyzed by terminal deoxynucleotide transferase (TdT) to bind to luciferin-labeled dUTP. Through the phenomenon of DNA breakage, cells in different apoptotic stages can be distinguished, and the effects of various treatments on cells can be analyzed to study the apoptotic mechanism. DNA fragmentation can be detected by a TUNEL kit, and apoptosis can be determined.

Detection Methods and Instruments

- Flow Cytometry detection: It is suitable for apoptosis detection of suspension cells and adherent cells, and the detection results can be analyzed by Flow Cytometry.
- Imaging detection: It is suitable for in situ apoptosis detection of tissue samples (paraffin embedded, frozen section) and cell samples (cell smear, climbing film) , and the detection results can be directly observed by fluorescence microscopy.
- DAB detection: It is suitable for tissue samples (paraffin section, frozen section) and cell samples (cell smear, cell creep) . Apoptotic cells can be observed under ordinary optical microscopy after biotin labeling and subsequent DAB color development.

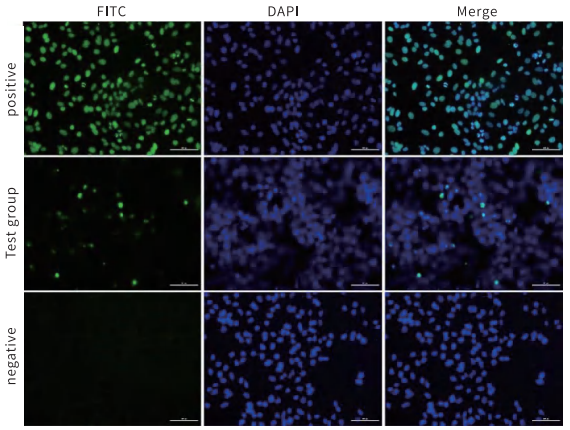


Fig. 6. TUNEL experiment results (E-CK-A320)

Cautions during Experiment

Fully dewaxed and hydrated

Before dewaxing, the slices can be roasted at 60°C for 20 min, and then dewaxed with xylene twice, for 5 to 10 min each time. It is generally recommended to use gradient ethanol to soak from high concentration to low concentration for hydration, so that the reagent can be fully and evenly combined in the later stage.

Grasp the cell permeability time

Generally, the incubation time of protease k is selected according to the thickness of the section, which is usually 10-30 min, and the section with a few μm thickness is used for a short time. Tens of μm thick sections are used for a long time, and conditions are reached by exploring so that the experimental process does not come off, but also enables the enzymes or antibodies behind to enter the cell.

Appropriate extension of TUNEL reaction liquid time

It is generally incubated at 37°C for 1 h, or a longer time can be selected according to the degree of apoptotic damage of cells, up to 2 h, but it should be determined by combining the final background coloring of the experiment.

Selection of DAB color developing conditions

If DAB is used for color development, the DAB reaction generally does not exceed 10 min, and the background color can be controlled in combination with microscope observation, and the color can be developed in tens of seconds.

Closure of endogenous POD

For tissues with large blood cell content such as liver and kidney, the sealing time can be extended appropriately and the concentration of hydrogen peroxide can be increased to achieve a better sealing effect without affecting the final specific staining.

Elabscience® TUNEL Products

Cat. No.	Product Name	Detection Instrument
E-CK-A320	One-step TUNEL In Situ Apoptosis Kit (Green, FITC)	Fluorescence Microscope
E-CK-A321	One-step TUNEL In Situ Apoptosis Kit (Green, Elab Fluor® 488)	Fluorescence Microscope
E-CK-A322	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 594)	Fluorescence Microscope
E-CK-A324	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 647)	Fluorescence Microscope
E-CK-A325	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 555)	Fluorescence Microscope
E-CK-A420	One-step TUNEL Flow Cytometry Apoptosis Kit (Green, FITC)	Flow Cytometry
E-CK-A421	One-step TUNEL Flow Cytometry Apoptosis Kit (Green, Elab Fluor® 488)	Flow Cytometry
E-CK-A422	One-step TUNEL Flow Cytometry Apoptosis Kit (Red, Elab Fluor® 594)	Flow Cytometry
E-CK-A423	One-step TUNEL Flow Cytometry Apoptosis Kit (Blue, Elab Fluor® Violet 450)	Flow Cytometry
E-CK-A424	One-step TUNEL Flow Cytometry Apoptosis Kit (Red, Elab Fluor® 647)	Flow Cytometry
E-CK-A425	One-step TUNEL Flow Cytometry Apoptosis Kit (Red, Elab Fluor® 555)	Flow Cytometry
E-CK-A331	TUNEL In Situ Apoptosis Kit (HRP-DAB Method)	Light microscope

Elabscience® TUNEL Product Citations

Title	Journal	Product Cited
Cellular plasticity of the bone marrow niche promotes hematopoietic stem cell regeneration	*Nature Genetics*	One-step TUNEL In Situ Apoptosis Kit (Green, FITC) (E-CK-A320)
Stem cell microencapsulation maintains stemness in inflammatory microenvironment	*International Journal of Oral Science*	One-step TUNEL In Situ Apoptosis Kit (Green, FITC) (E-CK-A320)
Activation of regulatory dendritic cells by Mertk coincides with a temporal wave of apoptosis in neonatal lungs	*Science Immunology*	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 555) (E-CK-A325)
Procyanidins Boost the Neuroprotective Effect of Minocycline for Intracerebral Haemorrhage	*Advanced Functional Materials*	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 594) (E-CK-A322)
Electrical stimulation induces anti-tumor immunomodulation via a flexible microneedle-array-integrated interdigital electrode	*Science Bulletin*	One-step TUNEL In Situ Apoptosis Kit (Green, FITC) (E-CK-A320)
Supramolecular Hydrogel Microspheres of Platelet-Derived Growth Factor Mimetic Peptide Promote Recovery from Spinal Cord Injury	*ACS Nano*	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 594) (E-CK-A322)
Long-term monitoring of intravital biological processes using fluorescent protein-assisted NIR-II imaging	*Nature Communications*	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 594) (E-CK-A322)
IGF1c mimetic peptide-based supramolecular hydrogel microspheres synergize with neural stem cells to promote functional recovery from spinal cord injury	*Nano Today*	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 594) (E-CK-A322)
Robust and Multifunctional Nanoparticles Assembled from Natural Polyphenols and Metformin for Efficient Spinal Cord Regeneration	*ACS Nano*	One-step TUNEL In Situ Apoptosis Kit (Red, Elab Fluor® 594) (E-CK-A322)
Cardiac-targeted delivery of nuclear receptor RORα via ultrasound targeted microbubble destruction optimizes the benefits of regular dose of melatonin on sepsis-induced cardiomyopathy	*Biomaterials Research*	One-step TUNEL In Situ Apoptosis Kit (Green, Elab Fluor® 488) (E-CK-A321)

* For more product citations, please visit www.elabscience.com.

Autophagy

Definition of Autophagy

Autophagy refers to the process in which damaged, denatured or senescent proteins and organelles are trans. ported to lysosomes under the regulation of Autophagy-associated gene (Atg) , and the lysosomes digest and degrade them. In order to maintain the homeostasis of the intracellular environment, normal animal cells need to continuously degrade dysfunctional or unwanted cell structures. Usually short-lived proteins are degraded through the ubiquitin-proteasome pathway, while long-lived proteins and cell structures are degraded by lysosomes through the autophagy pathway.

Characteristics of Autophagy

The current autophagy can be divided into three categories, including macroautophagy, microautophagy and chaperone-mediated autophagy (CMA) , of which macroautophagy is the most important and most studied form. The process of autophagy mainly includes the formation and expansion of phagophore, the formation of auto. phagosome, and the fusion of autophagosome and lysosome to form Autophagolysosome. After the auto. phagolysosome is formed, the inclusions are digested by hydrolases in the lysosome.

Process of Autophagy

The process of autophagy is not a completely passive cytological process, but an active biological process in which cells themselves maintain the stability of their internal environment through a series of intracellular signal transduction triggered by external stimuli. The complete process of autophagy can be divided into the following four stages: **Initiation → Nucleation-Elongation-Maturation → Fusion → Degradation**.

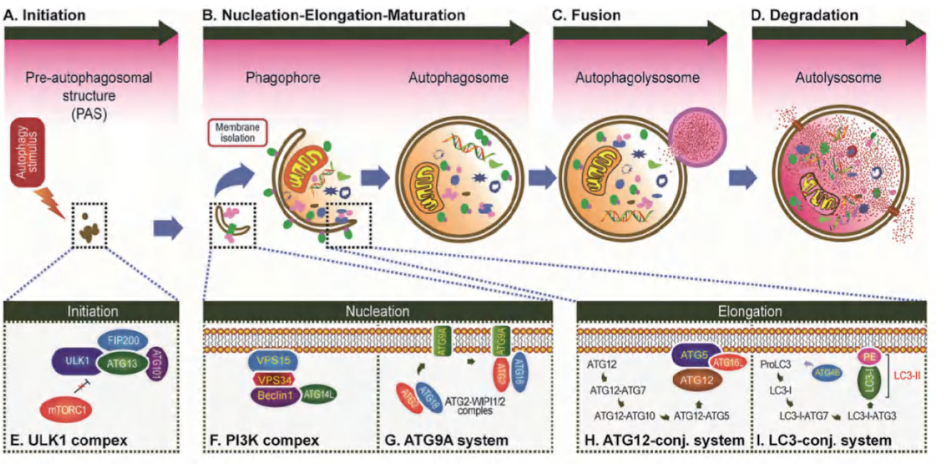
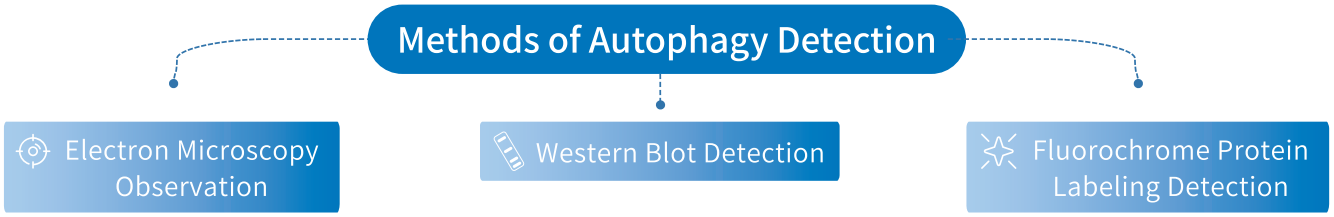


Fig. 7. Autophagy process
(Li et al. Molecular Cancer, 2020)

Common Methods for Autophagy Detection



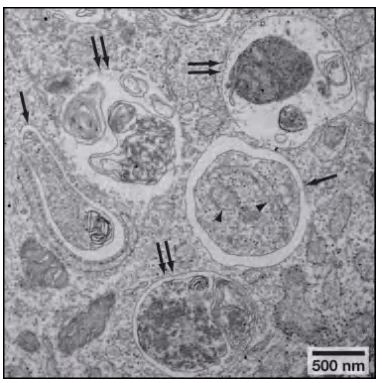
Electron Microscope Observation

◎ Transmission electron microscopy (TEM) is the most direct and classical method for observing autophagy based on the identification of autophagosome structure.

Table 2. Autophagy markers and their characteristics under transmission electron microscopy

Autophagy stage	Autophagy marker	Morphological feature
Early autophagy	Isolation membrane formation and expansion	Crescent-shaped or cup-shaped, bilayer or multilayer membrane, with a tendency to surround the cytoplasmic components.
Autophagy metaphase	The formation of autophagosomes	A vacuole-like structure of a double or multilayer membrane containing cytoplasmic components such as mitochondria, endoplasmic reticulum, and ribosomes.
Postautophagy	Formation of autophagy lysosomes	Monolayer with degraded cytoplasmic components

Fig.8. Morphology of autophagosome (single arrow) and autophagolysosome (double arrow) under transmission electron microscope

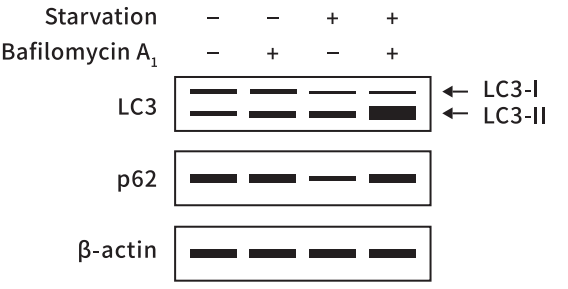


Western Blot Detection

◎ During autophagy, the C-terminal of LC3 was cleaved to LC3-I after synthesis and dispersed in the cytoplasm. After the autophagosome forms LC3-I, it is coupled with phosphatidylethanolamine to form LC3-II, which locates in the inner and outer membranes of the autophagy and remains stable until it is fused with the lysosome. Therefore, WB can be used to detect the change of LC3- II / I ratio to evaluate the strength of autophagy.

◎ As an important receptor for autophagy, p62 is mainly dependent on the autophagy pathway for degradation. Therefore, the protein level of p62 is also often used to indicate the degradation of autophagy.

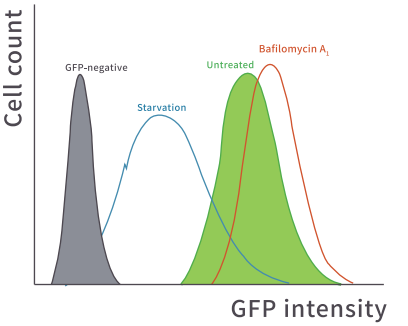
Fig. 9. The expression of LC3 and p62 protein was detected by Western Blot



Fluorochrome Protein Labeling Detection

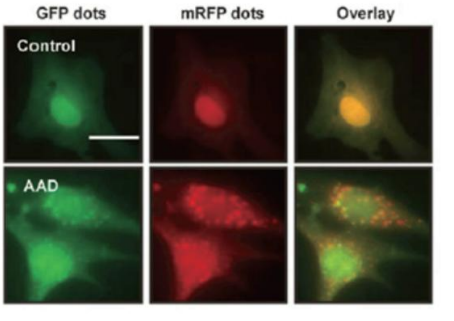
◎ **GFP-LC3:** In the absence of autophagy, GFP-LC3 was dispersed in the cytoplasm. During autophagy formation, GFP-LC3 accumulates on the autophagosome membrane. Multiple bright green fluorescent spots are formed under a fluorescence microscope, and the number of observed dots is the number of autophagosomes. LC3 on the membrane of autophagy is degraded in lysosomes; therefore, the overall degradation of LC3 can be quantified by flow cytometry through the reduction of GFP-LC3 fluorescence.

Fig. 10. GFP intensity decreased due to nutritional starvation, while autophagy or lysosome inhibitors increased GFP intensity



◎ **RFP-GFP-LC3 Dual Fluorochrome Indicator System:** The acidic environment in lysozyme led to the quenching of GFP fluorescence signal, and the red fluorescent protein had a good tolerance to the acidic environment and could construct RFP-GFP-LC3 tandem. When autophagy is formed, the autophagosome is marked yellow (green and red images coincide) and the autophagolysosome is marked red.

Fig. 11. RFP-GFP-LC3 dual fluorescent indicator system



Elabscience® Autophagy Related Products

Product Type	Product Name	Cat. No.
ELISA	Human Bcl-2 ELISA Kit	E-EL-H0114
	Rat Bcl-2 ELISA Kit	E-EL-R0096
	Human BAX ELISA Kit	E-EL-H0562
	Rat BAX ELISA Kit	E-EL-R0098
	Human BECN1 (Beclin 1) ELISA Kit	E-EL-H0564
	Human mTOR ELISA Kit	E-EL-H1655
	Human SIRT1 (Sirtuin 1) ELISA Kit	E-EL-H1546
	Mouse SIRT1 (Sirtuin 1) ELISA Kit	E-EL-M0350
	Rat SIRT1 (Sirtuin 1) ELISA Kit	E-EL-R1102

Product Type	Product Name	Cat. No.
Antibody	AKT1S1 Polyclonal Antibody	E-AB-40313
	AMPK alpha1 Polyclonal Antibody	E-AB-12968
	AMPK alpha1 Monoclonal Antibody	E-AB-22248
	AMPK gamma1 Polyclonal Antibody	E-AB-14562
	ATG7 Monoclonal Antibody	E-AB-22151
	ATG7 Polyclonal Antibody	E-AB-70288
	ATG10 Polyclonal Antibody	E-AB-10812
	ATG7 Monoclonal Antibody	E-AB-22151
	ATG12 Polyclonal Antibody	E-AB-10815
	ATG4B Polyclonal Antibody	E-AB-15507
	ATG5 Monoclonal Antibody	E-AB-22276
	ATG5 Monoclonal Antibody	E-AB-22149
	BCL2 Monoclonal Antibody	E-AB-22004
	Bcl-2 Polyclonal Antibody	E-AB-60012
	BAD Polyclonal Antibody	E-AB-70084
	BAX Monoclonal Antibody	E-AB-22128
	BAX Polyclonal Antibody	E-AB-40521
	BECN1 Monoclonal Antibody	E-AB-22139

Product Type	Product Name	Cat. No.
Antibody	BECN1 Polyclonal Antibody	E-AB-53242
	BNIP3 Polyclonal Antibody	E-AB-61061
	GABARAPL2 Polyclonal Antibody	E-AB-19104
	MLST8 Polyclonal Antibody	E-AB-18682
	LC3A/LC3B Polyclonal Antibody	E-AB-61027
	MAP1LC3A Monoclonal Antibody	E-AB-22136
	MLST8 Polyclonal Antibody	E-AB-18682
	MTOR Polyclonal Antibody	E-AB-15789
	CDKN1B Polyclonal Antibody	E-AB-52142
	P62/SQSTM1 Polyclonal Antibody	E-AB-70325
	PFKFB3 Polyclonal Antibody	E-AB-19543
	RAB25 Polyclonal Antibody	E-AB-12234
	RICTOR Polyclonal Antibody	E-AB-10904
	SIRT1 Polyclonal Antibody	E-AB-70071
	SMPD2 Polyclonal Antibody	E-AB-19030
	VCP Polyclonal Antibody	E-AB-18489

Elabscience® Autophagy Related Product Citations

Publication Name	Journal	Product Cited
Multi-Enzyme Cascade-Triggered Nitric Oxide Release Nanoplatform Combined with Chemo Starvation-like Therapy for Multidrug-Resistant Cancers	*ACS Applied Materials & Interfaces*	Human BAX (Bcl-2 Associated X Protein) ELISA Kit (E-EL-H0562)
The Antioxidant and Proapoptotic Effects of Sternbergia clusiana Bulb Ethanolic Extract on Triple-Negative and Estrogen-Dependent Breast Cancer Cells In Vitro	*Plants-Basel*	BCL2 Monoclonal Antibody (E-AB-22004)
Dietary phytoestrogen diosgenin interrupts metabolism, physiology, and reproduction of Swiss albino mice: Possible mode of action as an emerging environmental contaminant, endocrine disruptor and reproductive toxicant	*Food and Chemical Toxicology*	Bcl-2 Polyclonal Antibody (E-AB-60012)
Royal jelly protects brain tissue against fluoride-induced damage by activating Bcl-2/NF-κB/caspase-3/caspase-6/Bax and Erk signaling pathways in rats	*Environmental Science and Pollution Research*	BAX Monoclonal Antibody (E-AB-22128)
L-carnitine reverses methotrexate-induced nephrotoxicity in experimental rat model: Insight on SIRT1/PGC-1α/Nrf2/HO-1 axis	*Journal of Applied Toxicology*	SIRT1 Polyclonal Antibody (E-AB-70071)

*For more product citations, please visit www.elabscience.com.

Necrosis

Definition of Necrosis

Necrosis is the death of cells induced by extreme physical, chemical or other serious pathological factors, a form of pathological cell death.

There is a "program" in the cell itself, and if the cell runs until it dies according to the set work of the "program", then we call its death programmed death. However, if there is a small accident in the cell midway and it is out of the "programmed" control, then the cell that dies in this way will die unprogrammed, that is, by cell necrosis.

For a long time, cell necrosis was considered a passive death due to pathology, but recent research suggests that cell necrosis may be another form of programmed cell death, which has important physiological functions including triggering inflammatory responses. When apoptosis does not occur normally and cells must die, necrosis is used as a "substitute" for apoptosis.

Features of Necrosis

◎ **Nuclear Change:**This is the main morphological sign of cell necrosis, manifested by nuclear enrichment, nuclear fragmentation, and nucleolysis.

◎ **Cytoplasmic Changes:** Due to coagulation or dissolution of the cytoplasm, HE staining is dark red granular, such as eosinophilic bodies that appear in liver cell necrosis.

◎ **Mesenchymal Changes:** Due to the action of various lyolytic enzymes, the matrix disintegrates, the collagen fibers swell, break or liquefy, and the necrotic cells fuse into a piece, which is a red granular unstructured substance.

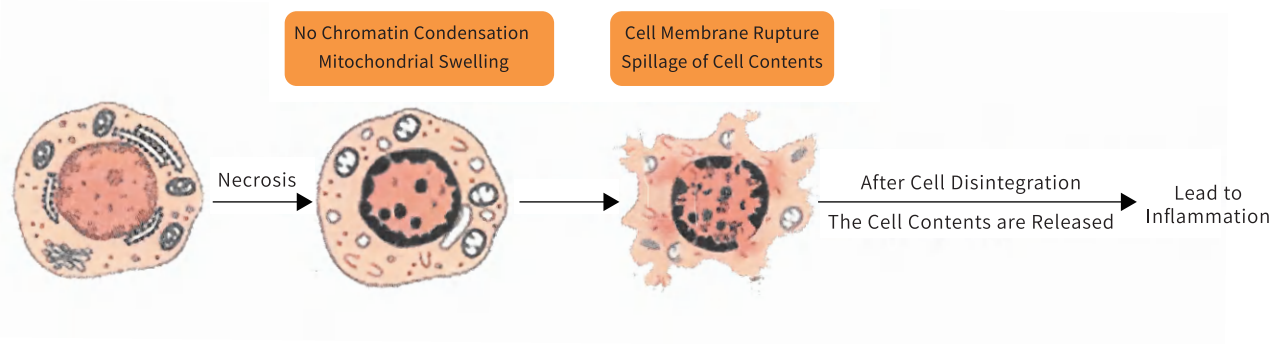


Fig. 12. Schematic diagram of the process of cell necrosis

Classification of Necrosis

Cell necrosis can be divided into the following types according to the protein degeneration in the tissue

◎ **Coagulative Necrosis:** coagulative necrosis refers to the dry state of local tissue cells after death, also known as ischemic necrosis, these tissue cells due to water loss, protein coagulation, and the formation of dry coagulation. There are also some specific types of coagulated necrosis, such as caseous necrosis, which is often caused by infection with the bacterium tuberculosis, resulting in a yellow, soft caseous state after tissue necrosis.

◎ **Liquefaction Necrosis:** It shows a liquefaction state after the death of tissue cells, called liquefaction necrosis, which is more common in tissues containing less protein, fat or water, and is prone to dissolu. tion and liquefaction after necrosis. Fat necrosis is also a kind of liquefaction necrosis, mainly due to the disintegration of fat cells, necrosis and liquid.

◎ **Cellulose-like Necrosis:** This kind of necrosis can only be seen under a medical microscope, which is manifested as small strips or small clumps of unstructured tissue after necrosis, and the character is like fibrin, so it is called cellulose-like necrosis.

◎ **Gangrene:** Necrotic tissue cells are secondary to bacterial infection, resulting in a black, dark green shape of these necrotic tissues. Gangrene includes dry gangrene, which is coagulated necrosis, and wet gangrene, which is similar to liquefaction necrosis. Some gangrene will produce a lot of gas, called gas gangrene, this gas gangrene is often formed on the basis of liquefaction necrosis, belongs to the type of special wet gangrene.

Difference between Cell Necrosis and Apoptosis

Although the end results of apoptosis, autophagy, and necrosis are very similar, their processes and mani. festations are very different.

As a type of cell ending life actively, apoptosis plays a very important role in the process of maintaining organism stability. Its occurrence is controlled by the program, and it is caused by the gradual activation of apoptosis pathway, and does not produce inflammation. Autophagy is the phagocytosis of damaged cellular organelles or aging proteins by intracellular lysosomes, which is a kind of self-protection of cells and usually leads to inflammation. Cell necrosis is often a type of acute cell end of life, with the release of cellular contents outside the cell, which leads to inflammation. All three are the ways of cell death, but there are great differences in the causes, mechanisms, and morphology of each part of the cell.

Table 3. Differences between cell necrosis and apoptosis and autophagy

	Apoptosis	Autophagy	Necrosis
Cause	Induced by physiological or minor pathological stimuli	Nutritional deficiency or hormone induction	Pathological stimulator induced or severe injury
Cell membrane	Membrane structure integrity	Membrane structure integrity	Cell membrane rupture
Cell morphology	Decrease and shrink	Cavitation	The cells swell and deform
DNA	Fragments degraded to 180~200 bp or integer multiples	Random degradation	Random degradation
Organelle	Remains intact and undisintegrated	It is extracted by autophagy and eventually digested by lysosomes	Deformity or swelling
Lysosome	It stays intact, the enzyme doesn't spill	Autophagy lysosomes are formed in the late stage of autophagy	Destruction, enzyme spill

	Apoptosis	Autophagy	Necrosis
Ending	Membrane budding forms apoptotic bodies, which are engulfed by macrophages	Cytoplasmic vacuolation occurs, autophagosomes are formed, and substances are removed by lysosomes	The cells break, dissolve, and the debris is devoured by macrophages
Inflammatory response	It does not cause inflammation in surrounding tissues	Can lead to inflammation, which increases the risk of bowel disease	Causing surrounding tissue to be engulfed by macrophages
Molecular mechanism	Related to the protease Caspase gene family	It occurs under the action of lysosomes	It is related to the expression of protein kinase RIP3

Methods for Necrosis Detection

To detect cell necrosis, it is first necessary to determine whether the cell membrane is broken. Detection methods include:

◎ **Morphological Observation:** transmission electron microscope or scanning electron microscope observation, if there is a membrane complete body, it is apoptosis; if the cell membrane bursts, it's necrosis.

◎ **Immunofluorescence or Flow Method:** PI or 7-AAD staining, because PI, 7-AAD can bind to DNA, if the cell membrane is broken, the dye can enter the cell and fluoresce.

And then the trigger determines whether it's programmed necrosis or passive cell necrosis, and if it's a physical factor, like an extreme environment, it's passive cell necrosis.

Elabscience® Necrosis Related Products

Cat. No.	Product Name	Size
E-CK-A161	PI Reagent (50 μg/mL)	50/100/200/500 Tests
E-CK-A165	Propidium Iodide (PI) Solution (750 μM)	100/500 Tests /500 Tests×10

Elabscience® Necrosis Related Products Citations

Title	Journal	Product Cited
PP2Acα promotes macrophage accumulation and activation to exacerbate tubular cell death and kidney fibrosis through activating Rap1 and TNFα production	*Cell Death and Differentiation*	PI Reagent (E-CK-A161)
Triptolide inhibits epithelial-mesenchymal transition phenotype through the p70S6k/GSK3/β-catenin signaling pathway in taxol-resistant human lung adenocarcinoma	*Translational Lung Cancer Research*	PI Reagent (E-CK-A161)

* For more product citations, please visit www.elabscience.com.

Necroptosis

Definition and Characteristics of Necroptosis

Necroptosis is the process by which cells self-destruct when apoptosis is blocked and they are activated by extracellular signals (death receptor-ligand binding) or intracellular signals (foreign microbial nucleic acids). During necroptosis, organelle swelling, cell membrane rupture, and breakdown of cytoplasm and nucleus can be observed.

When cells fail to undergo normal apoptosis after inflammatory, oxidative, or ischemic stress, necroptosis is used as a "substitute" for apoptosis. Necroptosis clears damaged cells without the activation of caspases.

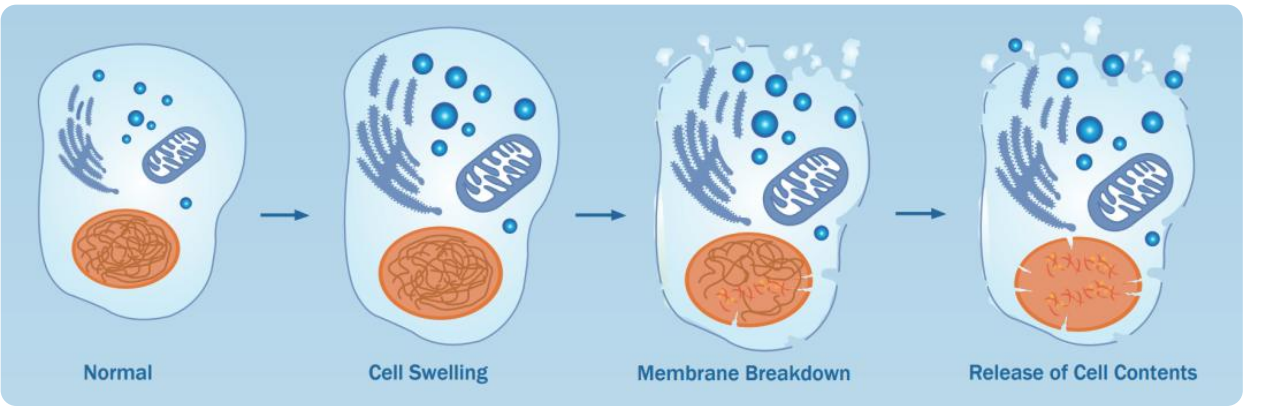


Fig. 13. The process of Necroptosis

Difference between Necrosis, Apoptosis and Necroptosis

Necrosis is an unregulated form of cell death caused by external physical and chemical stress. Necroptosis is highly regulated as a defense mechanism or escape route for cells facing viral infection. When a viral caspase inhibitor is present, the cell can only choose to commit suicide in a way that does not rely on caspases.

If apoptosis is a silent death in an orderly fashion, necroptosis opts for a messier, more violent form that uses the immune system to attack and kill the body's own cells. On the one hand, cells undergoing necroptosis have a typical necrotic morphology: cell membranes are destroyed, and cells and organelles are swollen and even disintegrated; on the other hand, necroptosis causes a significant inflammatory response, manifested by a large number of inflammatory cell infiltration and activation.

- Compared with apoptosis, necroptosis does not form apoptotic bodies, chromatin does not agglomerate, and its occurrence does not depend on Caspase activation, but on the formation of the necrosome.
- Compared with necrosis, the necroptosis process is regulated by multiple genes and is a more regular mode of death than necrosis.

Table 4. Differences between necrosis, apoptosis and necroptosis

	Necrosis	Apoptosis	Necroptosis
Procedural Type	Nonprocedural	Procedural	Procedural
Cell morphology	Cell swelling	Cell shrinkage	Cell swelling
Ending	Release cell debris	Phagocytes/macrophages devour cells	Release cell debris
Inflammation	Inflammatory	Non-inflammatory	Inflammatory

Pathogenesis of Necroptosis

Apoptosis is an active, intracellular signal transduction process triggered by the activation of death recep. tors by ligands. Currently, it is believed that the occurrence and regulation of necroptosis are mainly related to TNFα (tumor necrosis factor) , caspase-8, RIPK1 (receptor-interacting protein kinase) , RIPK3 and MLKL (mixed lineage kinase domain-like protein) .

Among them, TNF-α is the most important upstream signaling element of necroptosis. Normally, signaling by TNF-α, FASL (FAS ligand) , and TRAIL (tumor necrosis factor-associated apoptosis-inducing ligand) recruits RIPK1 kinase into a complex of apoptosis inhibitors (cIAPs) on the plasma membrane. This leads to a cascade reaction that activates NF-κB and promotes cell survival.

When this process is blocked, RIPK1 forms a secondary complex with activated caspase-8 and other factors, inducing apoptosis. Under conditions such as cellular stress, caspase-8 activity is inhibited, resulting in the formation of necrotic bodies, and the regulatory function of RIPK1 switches from apoptosis to necroptosis.

This leads to a series of autophosphorylation and cross-phosphorylation between RIPK1 and RIPK3, with the phosphorylation of RIPK3 leading to the recruitment and subsequent phosphorylation of MLKL and its trans. fer to the plasma membrane to form pore complexes. When the cell membrane ruptures, cell contents over. flow into the extracellular space, resulting in the emergence of inflammatory phenotypes and the release of damage-associated molecular patterns (DAMP) , such as IL-1α, IL-1β, and IL-33, which trigger an immune response and ultimately lead to cell death.

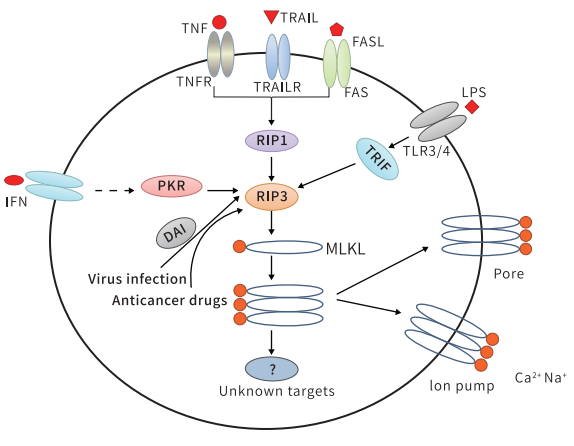


Fig. 14. Schematic diagram of the molecular mechanism of Necroptosis

Methods of Necroptosis Detection

◎ **Morphological Change:** The morphological change of cell death is a dynamic process, so it is necessary to "monitor" the whole process in real time to confirm the occurrence of necroptosis. Time-lapse video microscopy can detect necroptosis by correlating individual cell morphological changes to molecular, subcellular, and biochemical events (but this method is expensive and labor-intensive) .

◎ **Inhibition and Knockout:** RIPK1 inhibitor Necrostatin-1 and MLKL inhibitor necrosulfonamide are used to block the necroptosis, determine the cell survival rate, and verify the occurrence of necroptosis in the opposite direction. However, inhibitors can induce apoptosis in some cells and are not specific enough to distinguish between necrotic and apoptosis. Therefore, evidence for inhibition experiments needs to be supplemented by knocking out key proteins such as RIPK3 or MLKL to block necroptosis.

◎ **Key Protein Detection:** WB, IHC or flow cytometry were used to detect changes in key proteins in the necrotic apoptotic pathway, such as RIPK3, RIPK1 and MLKL. Phosphorylation of MLKL (Ser358 and Thr357) is the most commonly detected protein that confirms necroptosis through MLKL activation mediated by RIPK3. In addition, the high ratio of RIPK1/ pro-caspase-8 also suggests that the environment is conducive to necroptosis.

Elabscience® Necroptosis Related Products

Product Name	Cat. No.	Reactivity	Application
RIPK1 Polyclonal Antibody	E-AB-18284	Human	WB,IHC,ELISA
MLKL Polyclonal Antibody	E-AB-18957	Human	IHC,ELISA
MLKL Monoclonal Antibody	E-AB-22249	Human	IHC-p
Phospho-MLKL (Ser358) Monoclonal Antibody	E-AB-21338	Human	IHC-p
CASP8 Monoclonal Antibody	E-AB-22107	Human,Mouse,Rat	WB,IHC-p,IF
Caspase 8 Activity Colorimetric Assay Kit	E-CK-A388	——	FCM

Elabscience® Necroptosis Related Products Citations

Title	Journal	Product Citation
Selective pks+ Escherichia coli strains induce cell cycle arrest and apoptosis in colon cancer cell line	*World Journal Microbiology & Biotechnology*	Caspase 8 Activity Colorimetric Assay Kit (E-CK-A388)
Suppression of EGFR/PKC-δ/NF-κB Signaling Associated With Imipra. mine-Inhibited Progression of Non-Small Cell Lung Cancer	*Frontiers in Oncology*	CASP8 Monoclonal Antibody (E-AB-22107)
Allyl Isothiocyanate (AITC) Induces Apoptotic Cell Death In Vitro and Exhibits Anti-Tumor Activity in a Human Glioblastoma GBM8401/luc2 Model	*International Journal of Molecular Sciences*	CASP8 Monoclonal Antibody (E-AB-22107)
Increased autophagy in EOC re-ascites cells can inhibit cell death and promote drug resistance	*Cell Death & Disease*	CASP8 Monoclonal Antibody (E-AB-22107)

* For more product citations, please visit www.elabscience.com.

Pyroptosis

Definition of Pyroptosis

Pyroptosis: Inflammatory cell necrosis is a regulated cell death that depends on Gasdermin family proteins to form pores in the plasma membrane, manifested by the continuous expansion of cells until the membrane bursts, resulting in the release of a large number of inflammatory factors from the cell contents, which activates a strong inflammatory response. Pyroptosis is an important natural immune response of the body, which plays an important role in fighting infection.

Characteristics of Pyroptosis

Under light microscope, pyroptosis appears as swelling of cells with many bubble-like protrusions (pyroptosis bodies) .

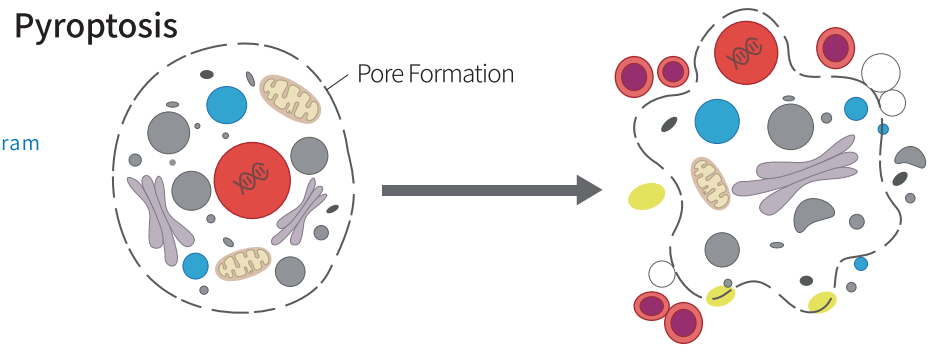


Fig. 15. Schematic diagram of pyroptosis

Pyroptosis Pathway

◎ **Classical Pathway:** also known as Caspase-1-dependent pathway, in this pathway, after the activation of NLRP3, NLRC4, AIM2, Pyrin and other inflammatory bodies, Pro-Caspase-1 will be activated and cleaved to form active Caspase-1. Activated Caspase-1, on the one hand, cleaved GSDMD to form peptides containing the active domain of GSDM-NT, induced cell membrane perforation, cell rupture, release of contents, and caused inflammation. On the other hand, activated Caspase-1 cuts IL-1β and IL-18 precursors to form active IL-1β and IL-18, which are released into the extracellular, recruiting inflammatory cells to gather, and expanding the inflammatory response.

◎ **Non-classical Pathway:** After being stimulated by lipopolysaccharide (LPS) , Caspase-4, Caspase-5 and Caspase-11 can directly bind to LPS and initiate pyrodeath process. On the one hand, activated Caspase-4/5/11 can lysate GSDMD protein, and the N-terminal of GSDMD protein can mediate both cell membrane lysis and pyrodeath. On the other hand, activated Caspase-4/5/11 activates the NLRP3 inflammasome to activate Caspase-1 and ultimately produce and release IL-1β and IL-18.

Methods for Pyroptosis Detection

◎ **Morphological Changes:** Scanning electron microscopy, TUNEL staining, immunofluorescence (GSDMD/GSDME) .

◎ **Detection of Pyrodeath Related Proteins**

① **q-PCR/WB/IHC Detection:** Detected the expression levels of the genes or proteins associated with pyro death: Caspase 1, 4, 5, 11, GSDMD, Cleaved CASP-3, and other pathway proteins (such as AIM2).

② **ELISA Kit Detection:** ELISA Kit was used to detect the expression levels of inflammatory factors such as IL-1β and IL-18.

③ **Caspase Kit Detection:** Related Caspase enzymes (Caspase 1, Caspase 4, Caspase 5, Caspase 11) detection.

Elabscience® Cell Pyroptosis Related Products

Sort	Product Name	Cat. No.
Activity Assay Kit	Caspase 1 Activity Assay Kit	E-CK-A381
	Caspase 4 Activity Assay Kit	E-CK-A384
ELISA	DHVD3ELISA Kit	E-EL-0016
	Human CASP1 (Caspase 1) ELISA Kit	E-EL-H0016
	Mouse CASP1 (Caspase 1) ELISA Kit	E-EL-M0201
	Rat CASP1 (Caspase 1) ELISA Kit	E-EL-R0371
	Human CASP3 (Caspase 3) ELISA Kit	E-EL-H0017
	Mouse CASP3 (Caspase 3) ELISA Kit	E-EL-M0238
	Rat CASP3 (Caspase 3) ELISA Kit	E-EL-R0160
	Human CASP4 (Caspase 4) ELISA Kit	E-EL-H0660
	Human CASP8 (Caspase 8) ELISA Kit	E-EL-H0659
	Mouse CASP8 (Caspase 8) ELISA Kit	E-EL-M0063
	Human CASP9 (Caspase 9) ELISA Kit	E-EL-H0663
	Human IL-18 ELISA Kit	E-EL-H0253
	Mouse IL-18 ELISA Kit	E-EL-M0730
	Rat IL-18 ELISA Kit	E-EL-R0567
	HS Mouse IL-18 ELISA Kit	E-HSEL-M0006
	Uncoated Human IL-18 ELISA Kit	E-UNEL-H0088
	Mouse NFKB-p65 ELISA Kit	E-EL-M0838
	Rat NFKB-p65 ELISA Kit	E-EL-R0674
	Human NF-κB p105 ELISA Kit	E-EL-H1386

Sort	Product Name	Cat. No.
ELISA	Uncoated Rat IL-18 ELISA Kit	E-UNEL-R0024
	Human IL-1β ELISA Kit	E-EL-H0149
	Mouse IL-1β ELISA Kit	E-EL-M0037
	Rat IL-1β ELISA Kit	E-EL-R0012
	HS Human IL-1β ELISA Kit	E-HSEL-H0001
	HS Mouse IL-1β ELISA Kit	E-HSEL-M0001
	HS Rat IL-1β ELISA Kit	E-HSEL-R0002
	MS Mouse IL-1β ELISA Kit	E-MSEL-M0003
	Uncoated Human IL-1β ELISA Kit	E-UNEL-H0091
	Uncoated Mouse IL-1β ELISA Kit	E-UNEL-M0064
	Uncoated Rat IL-1β ELISA Kit	E-UNEL-R0028
	Human MCP-1 ELISA Kit	E-EL-H6005
	Mouse MCP- ELISA Kit	E-EL-M3001
	Rat MCP-1 ELISA Kit	E-EL-R0633
	MS Mouse MCP-1 ELISA Kit	E-MSEL-M0012
	Uncoated Human MCP-1 ELISA Kit	E-UNEL-H0112
	Uncoated Mouse MCP-1 ELISA Kit	E-UNEL-M0077
	Human NFKB-p65 ELISA Kit	E-EL-H1388
	Mouse TNF-α ELISA Kit	E-EL-M3063
	Rat TNF-α ELISA Kit	E-EL-R2856
	Porcine TNF-α ELISA Kit	E-EL-P0010

Sort	Product Name	Cat. No.
ELISA	Mouse NFKB-p105 ELISA Kit	E-EL-M0836
	Rat NFKB-p105 ELISA Kit	E-EL-R0673
	Human TLR4 ELISA Kit	E-EL-H5820
	Mouse TLR4 ELISA Kit	E-EL-M2417
	Rat TLR4 ELISA Kit	E-EL-R0990
	Human TNF-α ELISA Kit	E-EL-H0109
Antibody	AIM2 Polyclonal Antibody	E-AB-10974
	Active CASP3 Monoclonal Antibody	E-AB-22115
	CASP3 Monoclonal Antibody	E-AB-22213
	(KO) Caspase-3 Polyclonal Antibody	E-AB-60646
	Caspase-3 Polyclonal Antibody	E-AB-60017
	CASP4 Polyclonal Antibody	E-AB-53537
	CASP8 Monoclonal Antibody	E-AB-22107
	CASP8 Polyclonal Antibody	E-AB-19664
	CASP9 Monoclonal Antibody	E-AB-22035
	CASP9 (active) Polyclonal Antibody	E-AB-12941
	Caspase-9 Polyclonal Antibody	E-AB-60760
	ELAVL1 Polyclonal Antibody	E-AB-14059
	(KO) HuR / ELAVL1 Antibody	E-AB-60367
	GSDMD Monoclonal Antibody	E-AB-81473

Sort	Product Name	Cat. No.
ELISA	MS Mouse TNF-α ELISA Kit	E-MSEL-M0002
	Uncoated Human TNF-α ELISA Kit	E-UNEL-H0175
	Uncoated Mouse TNF-α ELISA Kit	E-UNEL-M0103
	Uncoated Rat TNF-α ELISA Kit	E-UNEL-R0057
	Human TBP2 ELISA Kit	E-EL-H1729
Antibody	CCL2 Polyclonal Antibody	E-AB-40274
	NFκB-p65 Polyclonal Antibody	E-AB-32233
	NFκB-p65 Monoclonal Antibody	E-AB-22066
	Zebrafish p65 Polyclonal Antibody	E-AB-40558
	NFKB1 Polyclonal Antibody	E-AB-52131
	NLRC4 Polyclonal Antibody	E-AB-52183
	NLRP6 Polyclonal Antibody	E-AB-13438
	PYCARD Polyclonal Antibody	E-AB-30582
	TNF alpha Polyclonal Antibody	E-AB-33121
	TNF Polyclonal Antibody	E-AB-52065
	TXNIP Polyclonal Antibody	E-AB-17495
	IkB alpha Polyclonal Antibody	E-AB-40093
	P-IkB alpha (Ser32/S36) Antibody	E-AB-20911
	IL18 Polyclonal Antibody	E-AB-14154
	IL1 beta Polyclonal Antibody	E-AB-40530

Elabscience® Cell Pyroptosis Related Products Citations

Title	Journal	Product Cited
Dentatin triggers ROS-mediated apoptosis, G0/G1 cell cycle arrest and release of Th1-related cytokines in colorectal carcinoma cells	*Journal of Taibah University for Science*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Improvement of Antialveolar echinococcosis efficacy of novel Albenda. zole-Bile acids Derivatives with Enhanced Oral Bioavailability	*PLoS Neglected Tropical Diseases*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Isolation and characterization of N- (2-Hydroxyethyl) hexadecanamide from Colletotrichum gloeosporioides with apoptosis-inducing potential in breast cancer cells	*Biofactors*	Caspase 3/7 Activity Assay Kit (Colorimetric Method) (E-CK-A383)
Blood-brain barrier Permeable nanoparticles for Alzheimer's disease treatment by selective mitophagy of microglia	*Biomaterials*	MS Mouse IL-1β (Interleukin 1 Beta) ELISA Kit (E-MSEL-M0003)
The role of reactive astrocytes in neurotoxicity induced by ultrafine particulate matter	*Science of The Total Environment*	MS Mouse IL-1β (Interleukin 1 Beta) ELISA Kit (E-MSEL-M0003)
Safety and Effects of Lactobacillus paracasei TISTR 2593 Supplementa. tion on Improving Cholesterol Metabolism and Atherosclerosis-Related Parameters in Subjects with Hypercholesterolemia: A Randomized, Double-Blind, Placebo-Controlled Clinical Trial	*Nutrients*	Human MCP-1 (Monocyte Chemotactic Protein 1) ELISA Kit (E-EL-H6005)
Secretions from hypochlorous acid-treated tumor cells delivered in a melittin hydrogel potentiate cancer immunotherapy	*Bioactive Materials*	Mouse MCP-1 (Monocyte Chemotactic Protein 1) ELISA Kit (E-EL-M3001)
Novel combination strategy of high intensity focused ultrasound (HIFU) and checkpoint blockade boosted by bioinspired and oxygen-supplied nanoprobe for multimodal imaging-guided cancer therapy	*Journal for ImmunoTherapy of Cancer*	Mouse TNF-α (Tumor Necrosis Factor Alpha) ELISA Kit (E-EL-M3063)
Exploring Cardiac Impact of Oral Nicotine Exposure in a Transplantable Neoplasm Mice Model: Insights from Biochemical Analysis, Morphome. try, and Molecular Docking: Chlorella vulgaris Green Algae Support	*Toxicology*	Uncoated Mouse TNF-α (Tumor Necrosis Factor Alpha) ELISA Kit (E-UNEL-M0103)
Cycas pectinata stimulates germ cell proliferation in mouse testes	*Process Biochemistry*	Active CASP3 Monoclonal Antibody (E-AB-22115)

* For more product citations, please visit www.elabscience.com.

Ferroptosis

Definition of Ferroptosis

Ferroptosis is a non-regulated cell death mode caused by iron-dependent oxidative damage, which was first proposed in 2012. Ferroptosis is closely related to the pathophysiological processes of various diseases including tumors, metabolic diseases, nervous system diseases, and kidney injury, and is currently a hot spot research.

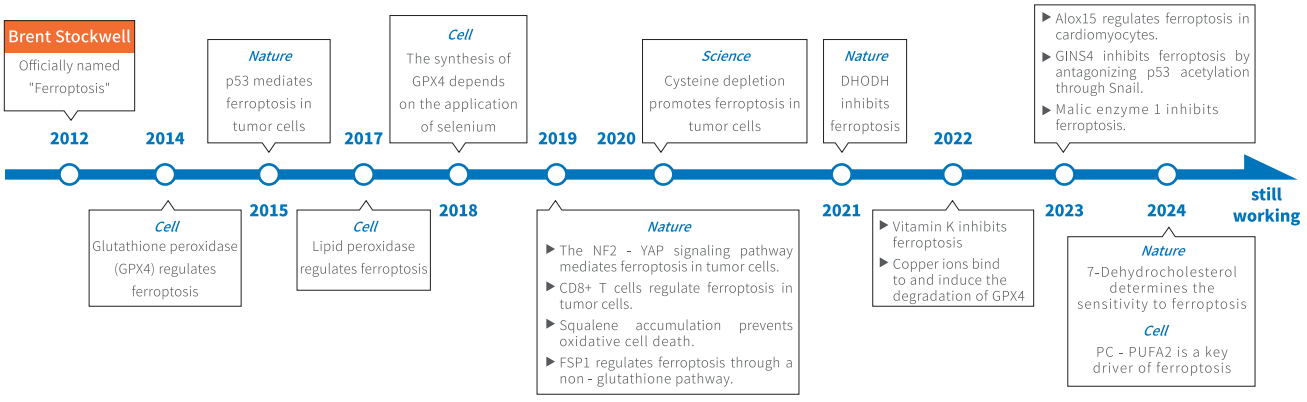
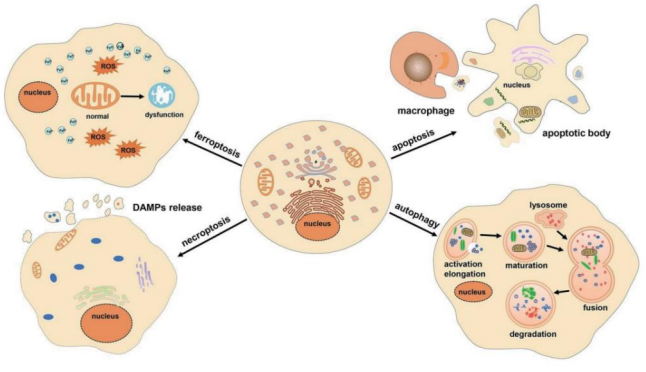


Fig. 16. History of Ferroptosis research

Characteristic of Ferroptosis

Ferroptosis is a form of cell death completely different from apoptosis, necrosis and autophagy in morphology, biochemistry and genetics. Ferroptosis cannot be inhibited by inhibitors of apoptosis, pyroptosis, and autophagy, but can be inhibited by iron chelators and antioxidants, among others. Therefore, the main characteristics of ferroptosis are the **increase of lipid reactive oxygen species and the accumulation of ferrous ions in cells**.

Fig. 17. Distinct forms of cell death and their morphological characteristics



Morphologically, the main characteristics of ferroptosis in cells are reflected in the morphological changes of mitochondria. The plasma membrane peroxidation induced by excess iron load changes the fluidity and integrity of the plasma membrane, ruptures the mitochondrial outer membrane, shrinks the volume, reduces or elimi. nates the cristae, and lead to the loss of mitochondrial function.

At the metabolic level, the process of ferroptosis, in addition to the accumulation of iron and ferrous ions in the cell and the increase of reactive oxygen species (ROS) , is usually accompanied by a decrease in the level of gluta. thione metabolism and other changes.

Ferroptosis Pathway

Previous studies have shown that ferroptosis can be regulated by a variety of metabolic processes, involving lipid, iron, amino acids, and glutathione metabolism, can be divided into GPX4-dependent metabolic pathways and GPX4-independent regulatory pathways. GPX4 is a key enzyme in the reduction of toxic peroxides. Through its enzymatic activity, it can prevent the toxicity of lipid peroxides and maintain the homeostasis of membrane lipid bilayers. It plays a key role in the process of ferroptosis. When GPX4 activity is hindered, lipid peroxides accumulate and eventually lead to cell death.

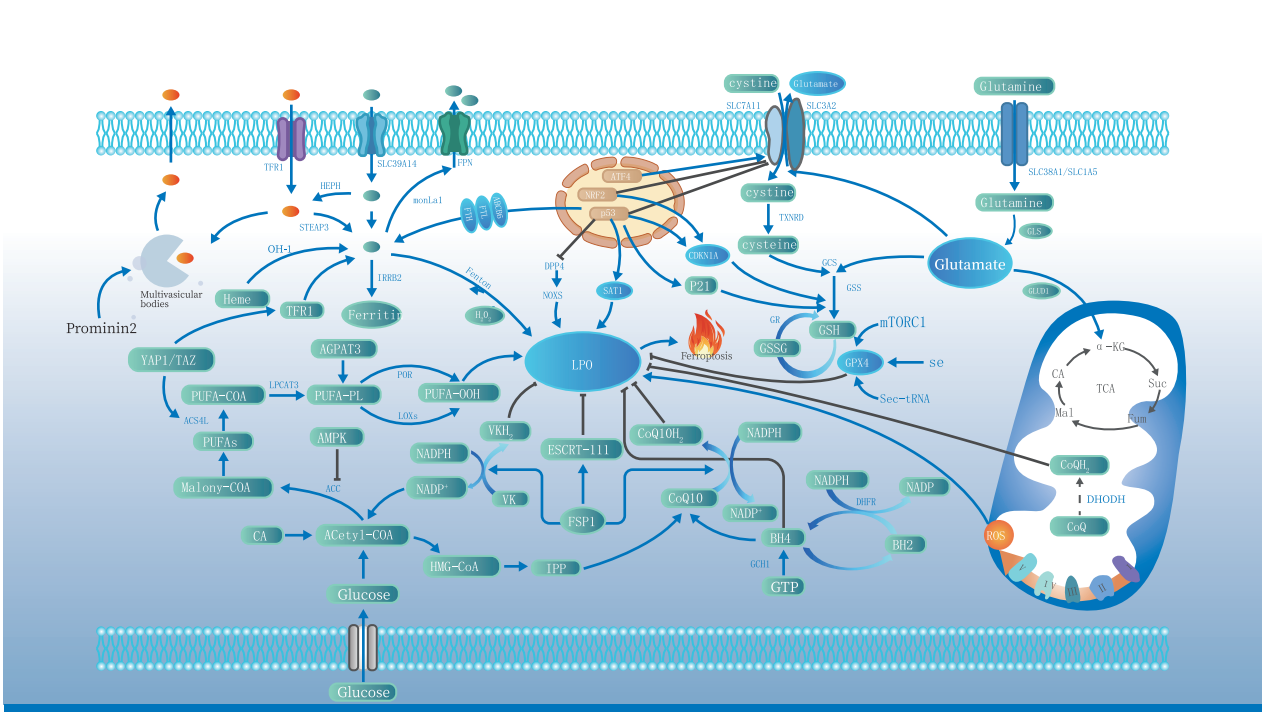


Fig. 18. Schematic pathway of ferroptosis

Methods for Ferroptosis Detection

The determination and detection of ferroptosis can be carried out through four aspects: related gene detection, related protein detection, cell morphology observation, and metabolism detection. These indicators include key genes of various regulatory pathways and characteristic indicators of ferroptosis.

◎ **Cell Morphology Observation:** The morphological changes of mitochondria and the integrity of cell membrane were detected mainly through electron microscopy.

◎ **Related Genes Detection:** Such as GSH homeostasis regulation gene, up-regulation of CHAC1, activation of NFE2L2 gene, etc.

◎ **Related Proteins Detection:** Such as the increase of CHAC1 protein, the decrease of GPX4 protein, etc.

◎ **Related Metabolism Detection:** Such as changes in cell activity, intracellular changes in iron/ferrous ions, increases in oxides such as ROS and hydrogen peroxide, and increases in lipid peroxidation products (such as MDA and LPO) .

Elabscience® Ferroptosis Related Products

◆ Full Stage Detection

Product Name	Cat. No.
Enhanced Cell Counting Kit 8 (WST-8/CCK8)	E-CK-A362
Lactate Dehydrogenase (LDH) Cytotoxicity Colorimetric Assay Kit	E-BC-K771-M
Cell Total Iron Colorimetric Assay Kit	E-BC-K880-M
Total Iron Colorimetric Assay Kit	E-BC-K772-M
Cell Ferrous Iron Colorimetric Assay Kit	E-BC-K881-M
Ferrous Iron Colorimetric Assay Kit	E-BC-K773-M
Malondialdehyde (MDA) Colorimetric Assay Kit (Cell Samples)	E-BC-K028-M
Malondialdehyde (MDA) Colorimetric Assay Kit(TBA Method)	E-BC-K025-M
Lipid Peroxide (LPO) Fluorometric Assay Kit	E-BC-F003
Lipid Peroxide (LPO) Colorimetric Assay Kit	E-BC-K176-M

Product Name	Cat. No.
Reactive Oxygen Species (ROS) Fluorometric Assay Kit	E-BC-K138-F
Hydrogen Peroxide (H_2O_2) Fluorometric Assay Kit	E-BC-F001
Total Superoxide Dismutase (T-SOD) Activity Assay Kit (WST-1 Method)	E-BC-K020-M
Reduced Glutathione (GSH) Colorimetric Assay Kit	E-BC-K030-M
Total Glutathione (T-GSH) /Oxidized Glutathione (GSSG) Colorimetric Assay Kit	E-BC-K097-M
Glutathione Peroxidase (GSH-Px) Activity Assay Kit	E-BC-K096-M
Glutathione Peroxidase 4 (GPX4) Activity Assay Kit	E-BC-K883-M
Glutamic Acid (Glu) Colorimetric Assay Kit	E-BC-K903-M
Cysteine (Cys) Colorimetric Assay Kit	E-BC-K352-M

◆ Ferroptosis Occurrence Detection

Product Name	Cat. No.
Human HSP-27/HSPB1 (Heat Shock Protein 27) ELISA Kit	E-EL-H1860
Human NFE2L2 (NuclearFactor, Erythroid Derived 2 Like 2) ELISA Kit	E-EL-H1564
Mouse NFE2L2 (Nuclear Factor, Erythroid Derived 2, Like 2) ELISA Kit	E-EL-M2607
Human FPN (Ferroportin) ELISA Kit	E-EL-H2355
Human TF (Transferrin) ELISA Kit	E-EL-H6028
Rat TF (Transferrin) ELISA Kit	E-EL-R3028

Product Name	Cat. No.
Rat NFE2L2 (Nuclear Factor, Erythroid Derived 2, Like 2) ELISA Kit	E-EL-R1052
Human PTGS2/COX-2 (Prostaglandin Endoperoxide Synthase 2) ELISA Kit	E-EL-H1846
Mouse PTGS2/COX-2 (Prostaglandin Endoperoxide Synthase 2) ELISA Kit	E-EL-M0959
Mouse TF (Transferrin) ELISA Kit	E-EL-M1184
Uncoated Human TF (Transferrin) ELISA Kit	E-UNEL-H0165
QuicKey Human TF (Transferrin) ELISA Kit	E-TSEL-H0001

Product Name	Cat. No.
Rat PTGS2/COX-2 (Prostaglandin Endoperoxide Synthase 2) ELISA Kit	E-EL-R0792
Ferritin Heavy Polyclonal Antibody	E-AB-40633
HSP27 Monoclonal Antibody	E-AB-22157
HSP27 Polyclonal Antibody	E-AB-31748
COX2 Polyclonal Antibody	E-AB-17010

Product Name	Cat. No.
Human sTfR1 (Soluble Transferrin Receptor 1) ELISA Kit	E-EL-H6085
SLC40A1 Polyclonal Antibody	E-AB-17960
Tf Polyclonal Antibody	E-AB-40476
Transferrin Monoclonal Antibody	E-AB-22178

* For more information, please visit www.elabscience.com or contact your local distributor.

Elabscience® Ferroptosis Related Product Citations

Title	Journal	Product Citation
A novel polypeptide CAPG-171aa encoded by circCAPG plays a critical role in triple-negative breast cancer	*Molecular Cancer*	Enhanced Cell Counting Kit 8 (WST-8/CCK8) (E-CK-A362)
Ultra-small radiosensitizers deliver epigenetic drugs to induce pyroptosis and boost triple-negative breast cancer radiotherapy	*Nano Today*	Lactate Dehydrogenase (LDH) Cytotoxicity Colorimetric Assay Kit (E-BC-K771-M)
A biomimetic mineralization nanosystem based on glycolysis-oxidative stress-autophagy regulation for the suppression of malignant tumor and lung metastasis	*Chemical Engineering Journal*	Total Iron Colorimetric Assay Kit (E-BC-K772-M)
Natural flavonoids disrupt bacterial iron homeostasis to potentiate colistin efficacy	*Science Advances*	Cell Ferrous Iron Colorimetric Assay Kit (E-BC-K881-M)
VDAC2 malonylation participates in sepsis-induced myocardial dysfunction via mitochondrial-related ferroptosis	*International Journal of Biological Sciences*	Ferrous Iron Colorimetric Assay Kit (E-BC-K773-M)
17β-oestradiol inhibits ferroptosis in the hippocampus by upregulat. ing DHODH and further improves memory decline after ovariectom	*Redox Biology*	Malondialdehyde (MDA) Colorimetric Assay Kit(Cell Samples) (E-BC-K028-M)
Mangiferin Protects DNase 2 Abundance via Nrf2 Activation to Prevent Cytosolic mtDNA Accumulation during Liver Injury	*Molecular Nutrition & Food Research*	Reactive Oxygen Species (ROS) Fluorometric Assay Kit (E-BC-K138-F)
The Anti-Inflammatory Effect of a Combination of Five Compounds From Five Chinese Herbal Medicines Used in the Treatment of CO	*Frontiers in Pharmacology*	Lipid Peroxide (LPO) Colorimetric Assay Kit (E-BC-K176-M)
Tumor microenvironment activated nanoreactors for chemilumines. cence imaging-guided simultaneous elimination of breast tumors and tumor-resident intracellular pathogens	*Chemical Engineering Journal*	Reduced Glutathione (GSH) Colorimetric Assay Kit (E-BC-K030-M)
Integrative analysis of multiomics data identifies selenium-related gene ALAD associating with keshan disease	*Free Radical Biology and Medicine*	Glutathione Peroxidase (GSH-Px) Activity Assay Kit (E-BC-K096-M)
Tumor microenvironment activated nanoreactors for chemilumines. cence imaging-guided simultaneous elimination of breast tumors and tumor-resident intracellular pathogens	*Chemical Engineering Journal*	Total Glutathione (T-GSH) /Oxidized Glutathione (GSSG) Colorimetric Assay Kit (E-BC-K097-S)

* For more product citations, please visit www.elabscience.com.

Cuproptosis

Definition of Cuproptosis

Cuproptosis was first proposed in the literature **"Copper induces cell death by targeting lipoylated TCA cycle proteins"** published in the journal ***Science*** in 2022. Copper directly binds to the lipoylated components of the tricarboxylic acid (TCA) cycle, leading to the aggregation of lipoylated proteins and the loss of iron-sulfur cluster proteins. This, in turn, triggers proteotoxic stress and ultimately results in cell death.

Characteristics of Cuproptosis

Cuproptosis is a form of regulated cell death with a mechanism that is significantly different from other known forms of cell death such as apoptosis, pyroptosis, necroptosis, and ferroptosis. Its main characteristic is the increase in cellular copper content. The cell death induced by cuproptosis can be triggered by copper ionophores and inhibited by copper chelators, but cannot be inhibited by other cell death inhibitors.

Table 5. Comparison of characteristics among different forms of cell death

Mode	Morphological Characteristics	Biological Characteristics	Impact of Occurrence
Cuproptosis	◎ Rupture of mitochondrial membrane	◎ Increase in copper, pyruvate, α-keto. glutarate, and HSP70 ◎ Decrease in Fe - S	◎ Trigger an immune response
Ferroptosis	◎ Mitochondrial shrinkage, reduction of cristae, and rupture of outer membrane	◎ Accumulation of ferrous iron and lipid peroxidation ◎ Decrease in cystine intake	◎ Trigger an immune response
Apoptosis	◎ Cell shrinkage and detachment ◎ Disappearance of nuclear structure and uniform degradation of DNA ◎ Formation of apoptotic bodies	◎ Release of cytochrome C and activation of Caspase ◎ Increase in intracellular calcium ions ◎ Decrease in mitochondrial membrane potential	◎ Asynchronous loss of individual cells ◎ Do not trigger inflammation
Autophagy	◎ Formation of autolysosomes ◎ Formation of vacuoles inside the cell	◎ Increase in the ratio of LC3 - II/LC3 - I	◎ Asynchronous occurrence in individual cells ◎ Maintain tissue homeostasis
Pyroptosis	◎ Cell swelling and deformation ◎ Deformation of organelles ◎ Nuclear pyknosis	◎ Cleavage and activation of Gasdermin - D ◎ Release of pro - inflammatory factors	◎ Trigger an inflammatory response

Cuproptosis Pathway

As a newly discovered form of cell death, studies shows that the regulation of some cases of cuproptosis involves copper metabolism, mitochondrial function, and protein modification. Among them, the lipoylation of FDX1 and DLAT proteins is a key factor in inducing cuproptosis. Excessive copper promotes the aggregation and loss of function of lipoylated proteins, triggers the instability of iron-sulfur cluster proteins, leads to proteotoxic stress, and ultimately causes cell death.

Other studies on cuproptosis primarily rely on bioinformatics. Through date from databases such as TCGA and bioinformatics analysis, researchers screen for cuproptosis-ralated genes and possible regulatory pathways are screened, and predict their relationship with diseases. For example, studies on breast cancer, acute myeloid leukemia, and diabetes have shown significant changes in the expression of cuproptosis-related genes including SLC31A1, DLAT, ATP7A, and ATP7B. The regulation of cuproptosis and its associated regulatory pathways under different pathological condition require further investigation.

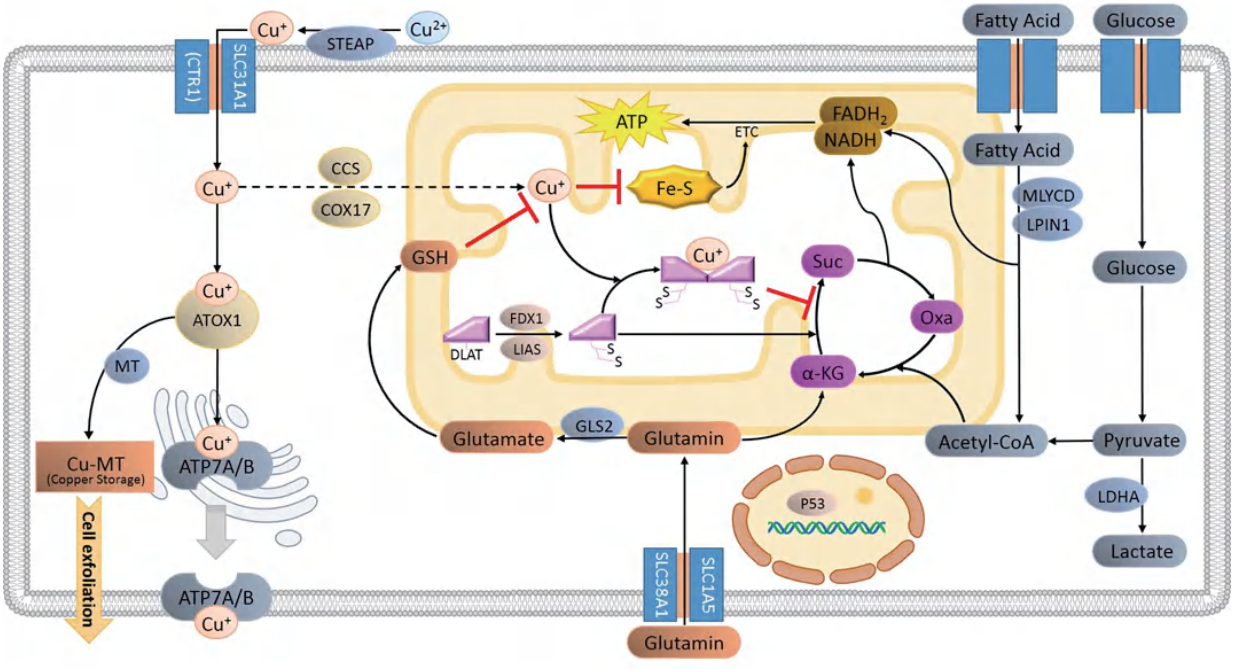


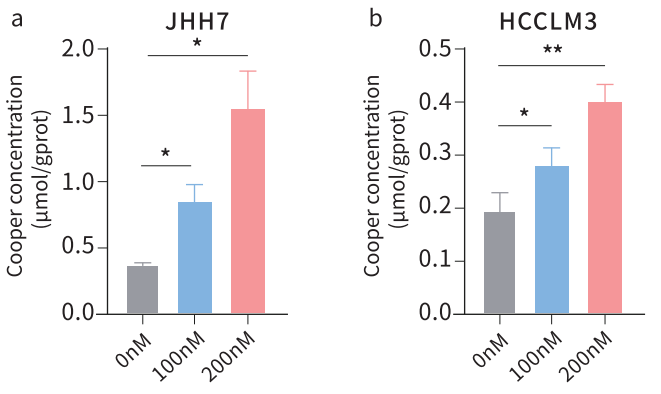
Fig. 19. Schematic pathway of cuproptosis

Methods for Cuproptosis Detection

The assessment and detection of cuproptosis can be carried out through four aspects: cell morphology observation, genes expression analysis, proteins detection, and metabolic profiling. These detection indicators include key genes and characteristic indicators of each regulatory pathway.

- **Cell Morphology Observation:** Detect the morphological changes of mitochondria and the integrity of the cell membrane.
- **Related Genes Detection:** Such as FDX1, LIAS, SLC31A1.
- **Related Proteins Detection:** Such as DLAT, Fe - S, HSP70.
- **Related Metabolism Detection:** Such as changes in cell viability; changes in intracellular indicators of copper ions, such as OCR, α - ketoglutarate, pyruvate, etc.

Fig. 20. Detection of cellular copper in cuproptosis research (Cell Copper (Cu2+) Colorimetric Assay Kit) (Cat. No: E-BC-K775)



Elabscience® Cuproptosis Related Products

Product Name	Cat. No.
Reduced Glutathione (GSH) Colorimetric Assay Kit	E-BC-K030-M
Total Glutathione (T-GSH) /Oxidized Glutathione (GSSG) Colorimetric Assay Kit	E-BC-K097-M
Pyruvic Acid Colorimetric Assay Kit	E-BC-K130-M
Lactate Dehydrogenase (LDH) Cytotoxicity Colorimetric Assay Kit	E-BC-K771-M
NADP+/NADPH Colorimetric Assay Kit (WST-8)	E-BC-K803-M
Glutamine (Gln) Colorimetric Assay Kit	E-BC-K853-M
α-Ketoglutarate (α-KG) Fluorometric Assay Kit	E-BC-F047
Enhanced Cell Counting Kit 8 (WST-8/CCK8)	E-CK-A362
GSH (Glutathione) ELISA Kit	E-EL-0026
Human HIF-1α (Hypoxia Inducible Factor 1 Alpha) ELISA Kit	E-EL-H6066
Mouse HIF-1α (Hypoxia Inducible Factor 1 Alpha) ELISA Kit	E-EL-M0687
Rat HIF-1α (Hypoxia Inducible Factor 1 Alpha) ELISA Kit	E-EL-R0513
Human HSP-70/HSPA9 (Heat Shock 70 kDa Protein 9) ELISA Kit	E-EL-H1863

Product Name	Cat. No.
Mitochondrial Complex II Activity Assay Kit	E-BC-K150-M
Mitochondrial Complex IV (Cytochrome C Oxidase) Activity Assay Kit	E-BC-K152-M
Cell Copper (Cu2+) Colorimetric Assay Kit (Complexing Method)	E-BC-K775-M
Mitochondrial Complex I (NADH-CoQ Reductase) Activity Assay Kit	E-BC-K149-M
Mitochondrial Complex III (Coenzyme Q-Cytochrome c Reductase) Activity Assay Kit	E-BC-K151-M
Mitochondrial Complex V (F0F1-ATPase/ATP Synthase) Activity Assay Kit	E-BC-K153-M
HSP70 Monoclonal Antibody	E-AB-22005
HSP70 Polyclonal Antibody	E-AB-40490
CDKN2A Polyclonal Antibody	E-AB-19334
PDHA1 Polyclonal Antibody	E-AB-19017
PDHA1 Polyclonal Antibody	E-AB-52930
COX7B Polyclonal Antibody	E-AB-10073
GLS Polyclonal Antibody	E-AB-19340

Elabscience®Cuproptosis Related Product Citations

Title	Journal	Product Citation
Polystyrene nanoplastics lead to ferroptosis in the lungs	*Journal of Advanced Research*	Reduced Glutathione (GSH) Colorimetric Assay Kit (E-BC-K030-M)
17β-oestradiol inhibits ferroptosis in the hippocampus by upregulating DHODH and further improves memory decline after ovariectomy	*Redox Biology*	Reduced Glutathione (GSH) Colorimetric Assay Kit (E-BC-K030-M)
Synergized photothermal therapy and magnetic field induced hyperthermia via bismuthene for lung cancer combinatorial treatment	*Materials Today Bio*	Total Glutathione (T-GSH) /Oxidized Glutathione (GSSG) Colorimetric Assay Kit (E-BC-K097-M)
Ultra-small radiosensitizers deliver epigenetic drugs to induce pyroptosis and boost triple-negative breast cancer radiotherapy	*Nano Today*	Lactate Dehydrogenase (LDH) Cytotoxicity Colorimetric Assay Kit (E-BC-K771-M)
HTR2A agonists play a therapeutic role by restricting ILC2 activation in papain-induced lung inflammation	*Cellular & Molecular Immunology*	Enhanced Cell Counting Kit 8 (WST-8/CCK8) (E-CK-A362)
Hyperbaric Oxygen Therapy Reduces Oxidative Stress and Inflammation, and Increases Growth Factors Favouring the Healing Process of Diabetic Wounds	*International Journal of Molecular Sciences*	Human HIF-1α (Hypoxia Inducible Factor 1 Alpha) ELISA Kit (E-EL-H6066)
Identification and development of a novel risk model based on cuproptosis-associated RNA methylation regulators for predicting prognosis and characterizing immune status in hepatocellular carcinoma	*Hepatology International*	Cell Copper (Cu2+) Colorimetric Assay Kit (Complexing Method) (E-BC-K775-M)
Single-cell RNA sequencing reveals XBP1-SLC38A2 axis as a metabolic regulator in cytotoxic T lymphocytes in multiple myeloma	*Cancer Letters*	Glutamine (Gln) Colorimetric Assay Kit (E-BC-K853-M)
Gemcitabine nano-prodrug reprograms intratumoral metabolism and alleviates immunosuppression for hepatocellular carcinoma therapy	*Nano Today*	GLS Polyclonal Antibody (E-AB-19340)

* For more product citations, please visit www.elabscience.com.

Disulfidptosis

Definition of Disulfidptosis

Disulfidptosis is a new type of cell death caused by disulfide stress induced by excessive accumulation of cystine. It was first reported in the research paper "**Actin cytoskeleton vulnerability to disulfide stress mediates disulfidptosis**" published in the journal *Nature Cell Biology* in March 2023.

The research found that the cell death of cancer cells with high-expression of SLC7A11 induced by glucose deprivation does not belong to any known type of cell death. This new type of cell death cannot be inhibited by drugs commonly used to suppress cell death, nor can it be prevented by knocking out key genes related to ferroptosis or apoptosis. However, thiol oxidants (such as diamide and diethyl maleate) can significantly enhance this type of cell death. Therefore, this form of cell death was named disulfidptosis.

Further research revealed that the main cause of disulfidptosis is that the supply of NADPH fails to meet the requirements for reducing cystine to cysteine in cells, resulting in disulfide stress. This stress induces the formation of disulfide bonds in actin cytoskeletal proteins, contraction of the cytoskeleton, and detachment from the plasma membrane, ultimately leading to cell death. Insufficient glucose intake and excessive cystine intake in cells may both induce disulfidptosis.

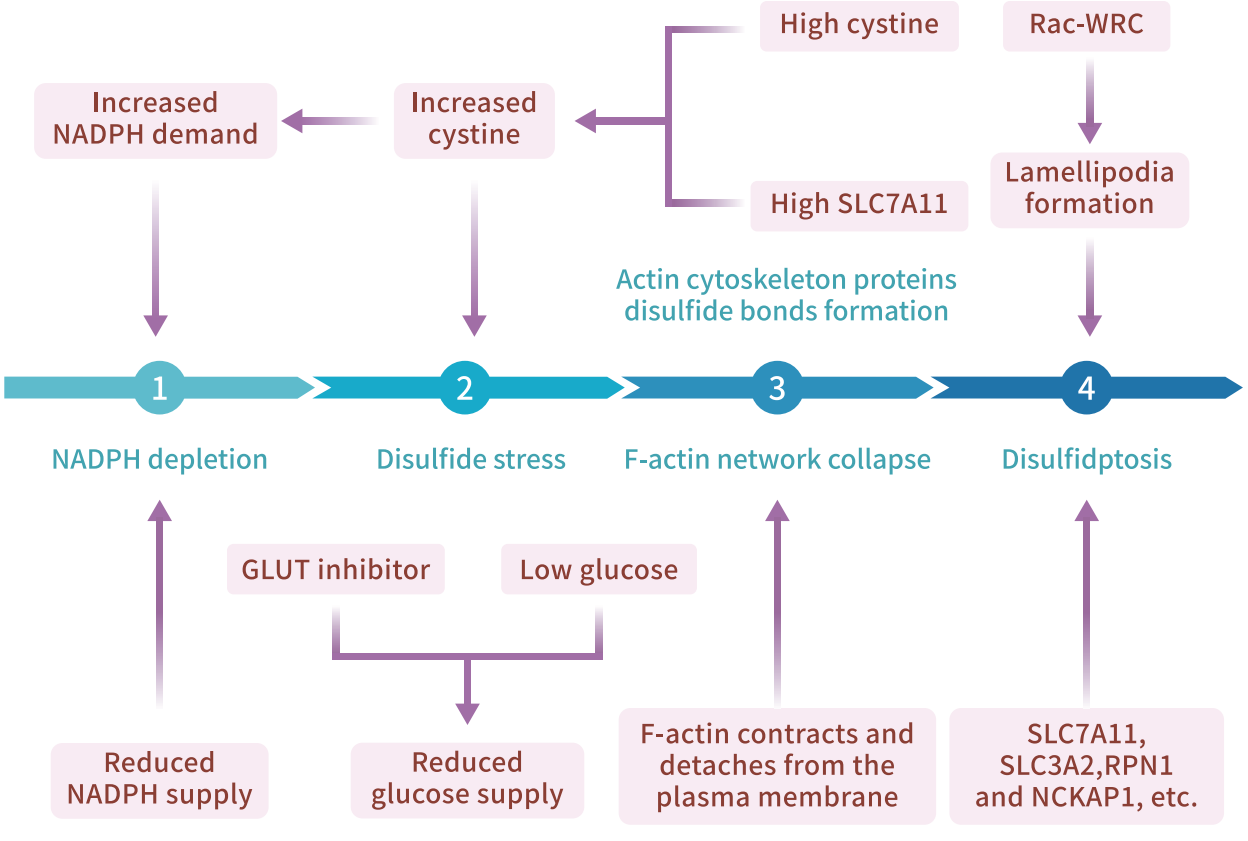


Fig. 21. The process of disulfidptosis

Characteristics of Disulfidptosis

The characteristics of disulfidptosis include an increase in cystine, an increase in disulfide molecules, cell contraction, and contraction of F-actin, among others.

Mechanism of Disulfidptosis

Disulfidptosis is a form of programmed cell death that triggers cell death through redox (oxidation-reduction) reactions and disulfide bond formation. According to existing literature, the regulatory pathways involved in disulfidptosis include the cystine uptake and glucose metabolism pathways, which can be regulated by proteins such as the cystine transporter solute carrier family 7 member 11 (SLC7A11) (also known as xCT) , NCKAP1, and compounds such as disulfide reducing agents and 2-DG. The main key factor is SLC7A11.

The function of SLC7A11 is to transport extracellular cystine into the cell. Cystine is one of the important raw materials for synthesizing glutathione and inhibiting cellular oxidative stress, but it also has certain cytotoxicity. It is an insoluble amino acid. To prevent the toxic accumulation of highly insoluble cystine in the cell, the cell needs to rapidly reduce cystine to cysteine. This process requires a large amount of NADPH from the glucose-pentose phosphate pathway (PPP) , which will cause significant depletion of the cellular NADPH pool and make such cells dependent on glucose and the pentose phosphate pathway (PPP) . When there is high expression of SLC7A11 in the cell, it will lead to an increase in cystine uptake. When the supply of NADPH is insuf. ficient, it may result in cell death.

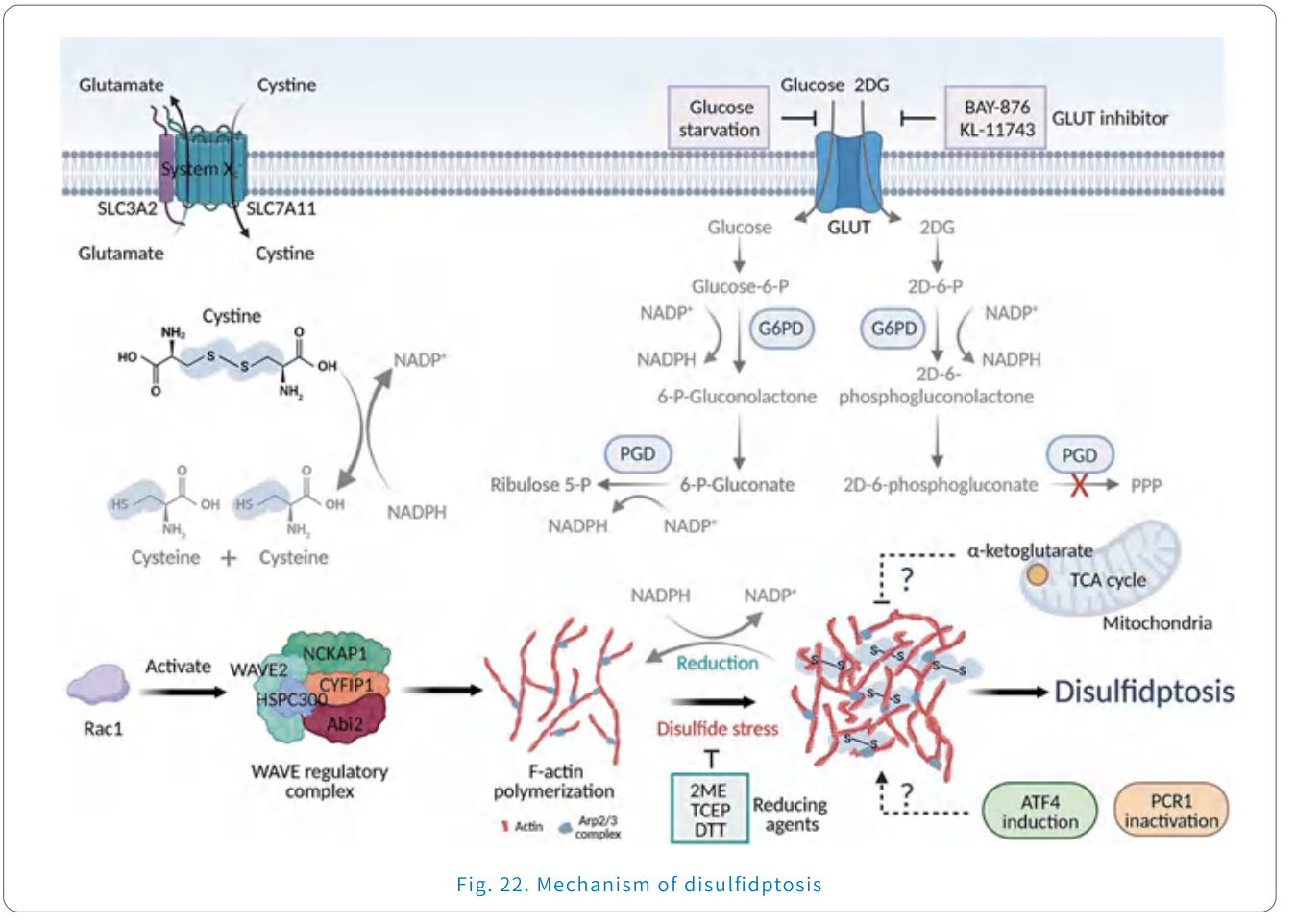


Fig. 22. Mechanism of disulfidptosis

Methods for Disulfidptosis Detection

Based on its characteristics, the detection of disulfidptosis can be classified as follows.

◎ **Cell Morphology Observation:** Staining to detect the morphology of actin filaments.

◎ **Metabolic Detection:** NADPH, ATP, cystine uptake, glucose, Trx, etc.

◎ **Protein Detection:** GLUT1, GLUT3, HK2, LDHA, SLC7A11.

◎ **Gene Detection:** GSTT1, MYH9, NCKAP1, RPN1.

Elabscience® Disulfidptosis Related Products

Product Name	Cat. No.
Enhanced Cell Counting Kit 8 (WST-8/CCK8)	E-CK-A362
Lactate Dehydrogenase (LDH) Cytotoxicity Colorimetric Assay Kit	E-BC-K771-M
Lipid Peroxide (LPO) Fluorometric Assay Kit	E-BC-F003
Lipid Peroxide (LPO) Colorimetric Assay Kit	E-BC-K176-M
NADP+/NADPH Colorimetric Assay Kit (WST-8)	E-BC-K803-M
Glucose (Glu) Colorimetric Assay Kit (GOD-POD Method)	E-BC-K234-M
Glucose (GLU) Fluorometric Assay Kit	E-BC-F037
Glucose Uptake Fluorometric Assay Kit	E-BC-F041
ACTA2 Monoclonal Antibody	E-AB-22106
ACTA2 Polyclonal Antibody	E-AB-16235
FLNA Polyclonal Antibody	E-AB-31433
beta actin Monoclonal Antibody	E-AB-48018
beta actin Polyclonal Antibody	E-AB-40517
SLC3A2 Polyclonal Antibody	E-AB-18594

Product Name	Cat. No.
Cystine Uptake Fluorometric Assay Kit	E-BC-F066
ATP Colorimetric Assay Kit	E-BC-K157-M
ATP Chemiluminescence Assay Kit	E-BC-F002
Reactive Oxygen Species (ROS) Fluorometric Assay Kit (Green)	E-BC-K138-F
Glucose-6-phosphate (G6P) Colorimetric Assay Kit	E-BC-K011-M
Glucose-6-Phosphate Dehydrogenase (G-6-PD) Activity Assay Kit	E-BC-K056-M
Mouse ACTα2 (Actin Alpha 2, Smooth Muscle) ELISA Kit	E-EL-M2434
IQGAP1 Polyclonal Antibody	E-AB-53496
PRDX1 Monoclonal Antibody	E-AB-22067
RPN1 Polyclonal Antibody	E-AB-14657
INF2 Polyclonal Antibody	E-AB-18555
CD2AP Polyclonal Antibody	E-AB-16309
CLP36 Polyclonal Antibody	E-AB-15288
ACTN4 Polyclonal Antibody	E-AB-14759

Elabscience® Disulfidptosis Related Product Citations

Title	Journal	Product Citation
HTR2A agonists play a therapeutic role by restricting ILC2 activation in papain-induced lung inflammation	*Cellular & Molecular Immunology*	Enhanced Cell Counting Kit 8 (WST-8/CCK8) (E-CK-A362)
Abiotic Synthetic Antibody Inhibitor with Broad-Spectrum Neutral. ization and Antiviral Efficacy against Escaping SARS-CoV-2 Variants	*ACS Nano*	Enhanced Cell Counting Kit 8 (WST-8/CCK8) (E-CK-A362)
Ultra-small radiosensitizers deliver epigenetic drugs to induce pyroptosis and boost triple-negative breast cancer radiotherapy	*Nano Today*	Lactate Dehydrogenase (LDH) Cytotoxicity Colorimetric Assay Kit (E-BC-K771-M)
Docetaxel-loaded pH/ROS dual-responsive nanoparticles with self-supplied ROS for inhibiting metastasis and enhancing immunotherapy of breast cancer	*Journal of Nanobiotechnology*	ATP Colorimetric Assay Kit (E-BC-K157-M)
Interferon-γ regulates immunosuppression in septic mice by promoting the Warburg effect through the PI3K/AKT/mTOR pathway	*Molecular Medicine*	ATP Chemiluminescence Assay Kit (E-BC-F002)
Compound Danshen Dripping Pill inhibits hypercholesterolemia/ atherosclerosis-induced heart failure in ApoE and LDLR dual deficient mice via multiple mechanisms	*Acta Pharmaceutica Sinica B*	Reactive Oxygen Species (ROS) Fluorometric Assay Kit (Green) (E-BC-K138-F)
Targeting the Na+/K+ ATPase DR-region with DR-Ab improves doxorubicin-induced cardiotoxicity	*Free Radical Biology and Medicine*	Lipid Peroxide (LPO) Colorimetric Assay Kit (E-BC-K176-M)
Phosphatidylethanolamine aggravates Angiotensin II-induced atrial fibrosis by triggering ferroptosis in mice	*Frontiers in Pharmacology*	NADP+/NADPH Colorimetric Assay Kit (WST-8) (E-BC-K803-M)
A smartphone-assisted “all-in-one” paper chip for one-pot noninvasive detection of salivary glucose level	*Chemical Engineering Journal*	Glucose (Glu) Colorimetric Assay Kit (GOD-POD Method) (E-BC-K234-M)
Sodium-Glucose Cotransporter-2 (SGLT2) expression in diabetic and non-diabetic failing human cardiomyocytes	*Pharmacological Research*	Glucose Uptake Fluorometric Assay Kit (E-BC-F041)

* For more product citations, please visit www.elabscience.com.

PANoptosis

Definition of PANoptosis

PANoptosis is a brand new form of programmed cell death proposed by American scholar Malireddi et al. in 2019. PANoptosis is regulated by the PANoptosome complex and exhibits the key characteristics of pyroptosis, apoptosis, and/or necroptosis, which is also the origin of the "P", "A", and "N" in the term PANoptosis. However, PANoptosis cannot be solely explained by these three programmed cell death (PCD) pathways.

In previous studies, apoptosis, pyroptosis, and necroptosis generally operate independently. But as research deepened, more and more findings indicate that there is some crosstalk among these three, and they can regulate each other in a cross manner. For example, the apoptosis executioners caspase-3 and caspase-7 can inactivate GSDMD through N-terminal cleavage and can also cleave GSDME, transforming the morphology of cell death from apoptosis to pyroptosis. The execution of necroptosis and the formation of MLKL pores can activate apoptosis through membrane damage. These discoveries have contributed to the establishment of the concept of PANoptosis.

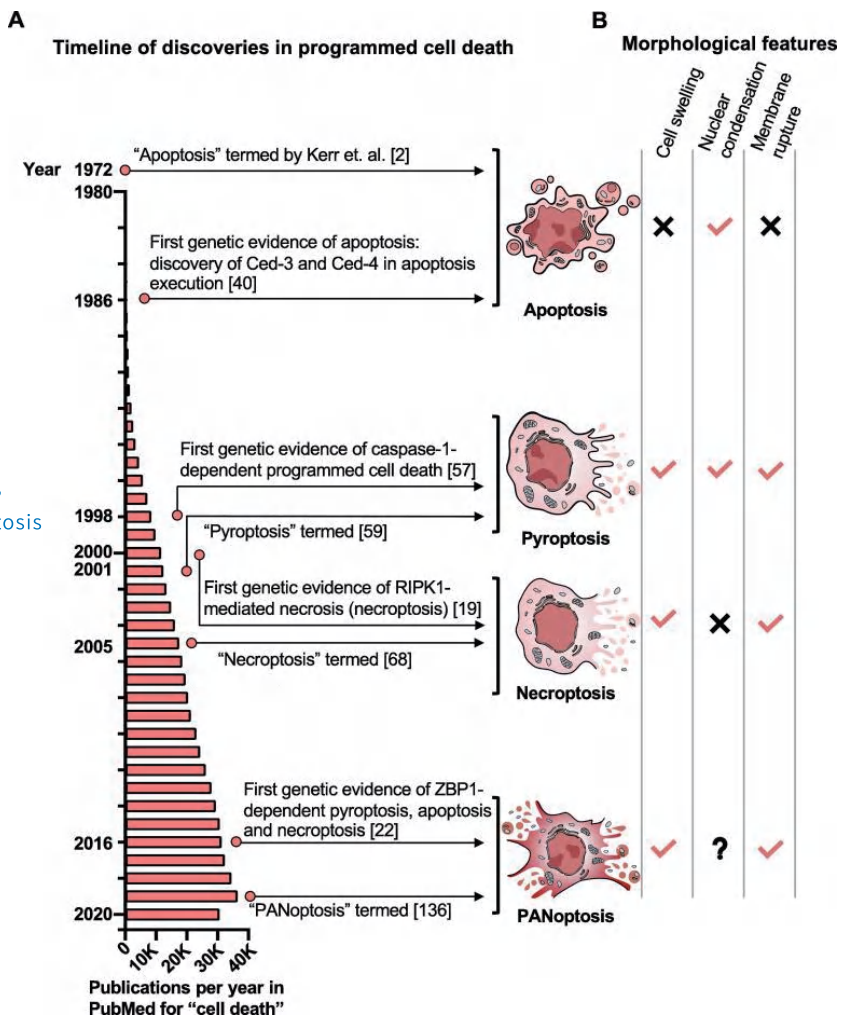


Fig. 23. Key milestones of apoptosis, pyroptosis, necroptosis, and PANoptosis

Regulation of PANoptosis

PANoptosis is regulated by a cascade of upstream sensors and molecular signals. These sensors and signal. ing cascades assemble into a multimeric complex, called the PANoptosome. As a platform for activating downstream molecules and the "master switch" for initiating the three PCD pathways, the PANoptosome and its upstream sensors provide attractive intervention targets for the treatment of human diseases. There are three well-characterized upstream molecules of PANoptosis, namely ZBP1, RIPK1, and AIM2. They can sense specific stimuli and trigger the assembly of the PANoptosome, resulting in three distinct PANoptosome types—ZBP1-PANoptosomes, AIM2-PANoptosomes, and RIPK1-PANoptosomes—each with unique sensors and regulators,forming three types of PANoptosomes with different sensors and regulators, namely ZBP1-PANoptosomes, AIM2-PANoptosomes, and RIPK1-PANoptosomes.

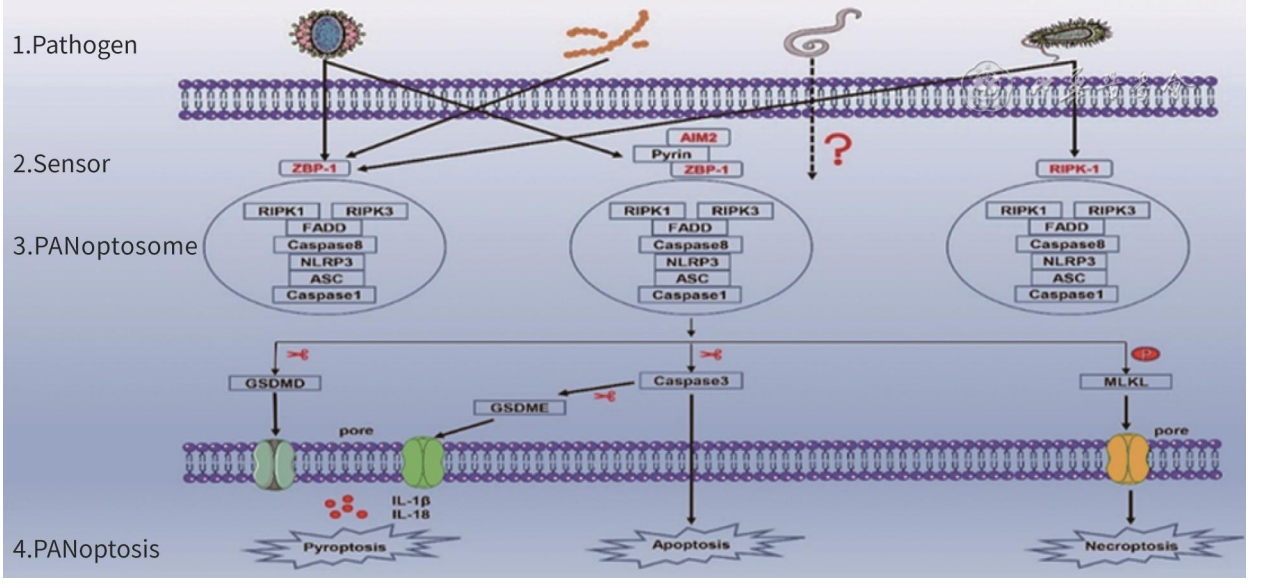


Fig. 24. PANoptosome and the pathways

Formation of the PANoptosome

Although the proximal sensors activated by different pathogen infections vary, these sensor molecules can all initiate the formation of the PANoptosomes. The PANoptosomes provides a molecular scaffold that allows the coupling and binding of proteins or molecules required for pyroptosis (NLRP-3, apoptosis-associated speck-like protein containing a CARD (ASC) , and Caspase-1, apoptosis (Caspase-8/3) , and necroptosis (RIPK3, p-MLKL) . It is worth mentioning that the specific mechanisms by which upstream sensors recognize pathogen infections and the interactions between these components remain unknown. To date, three upstream molecules of PANoptosis have been identified: ZBP1, RIPK1, and interferon-inducible protein 2 (AIM2) , which can sense specific stimuli and trigger the assembly of the PANoptosomes. However, it is likely that there are other upstream molecules that can initiate PANoptosis.

Methods for PANoptosis Detection

- Cell morphology Observation: PANoptosis, as a novel form of programmed cell death, simultaneously exhibits the typical morphological characteristics of pyroptosis, apoptosis, and necroptosis.
  - ◎Characteristics of pyroptosis: cell swelling, membrane blebbing, and formation of GSDMD pores.
  - ◎Characteristics of apoptosis: cell shrinkage, chromatin condensation, and formation of apoptotic bodies.
  - ◎Characteristics of necroptosis: Rupture of the cell membrane and swelling of organelles.

- Detect the key proteins of different types of programmed cell death (PCD) .
  - ◎Related to pyroptosis: Caspase-1, Caspase-4, GSDMD, AIM2/Pyrin/NLRP3, etc.
  - ◎Related to apoptosis: Caspase-3, Caspase-7, Caspase-8, PARP, Bax/Bcl, etc.
  - ◎Related to necroptosis: MLKL, RIPK1, RIPK3, ZBP1, etc.

Elabscience® PANoptosis Related Products

Product Name	Cat. No.
Caspase 1 Activity Assay Kit (Colorimetric Method)	E-CK-A381
Caspase 3/7 Activity Assay Kit (Colorimetric Method)	E-CK-A383
Caspase 4 Activity Assay Kit (Colorimetric Method)	E-CK-A384
Caspase 8 Activity Assay Kit (Colorimetric Method)	E-CK-A388
AIM2 Polyclonal Antibody	E-AB-10974

Product Name	Cat. No.
PARP1 Monoclonal Antibody	E-AB-22073
BAX Polyclonal Antibody	E-AB-40521
MLKL Polyclonal Antibody	E-AB-18957
RIPK1 Polyclonal Antibody	E-AB-18284
ZBP1 Polyclonal Antibody	E-AB-53214

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To keep pace with the rapid advancements in research, we are dedicated to the continuous development of cutting-edge antibody and protein reagents, ensuring that our products evolve to meet the latest scientific needs. Our commitment extends beyond cell detection to include sophisticated cell isolation and characterization, empowering researchers to tackle the most complex challenges in cell biology. We pride ourselves on maintaining stringent quality control for every product, enhancing the accuracy and reliability of your experimental results. Our relentless pursuit of excellence since 2009 has established our presence in over 150 countries and regions worldwide.

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Citations	Cooperation Units	Countries Worldwide	Technical Platforms

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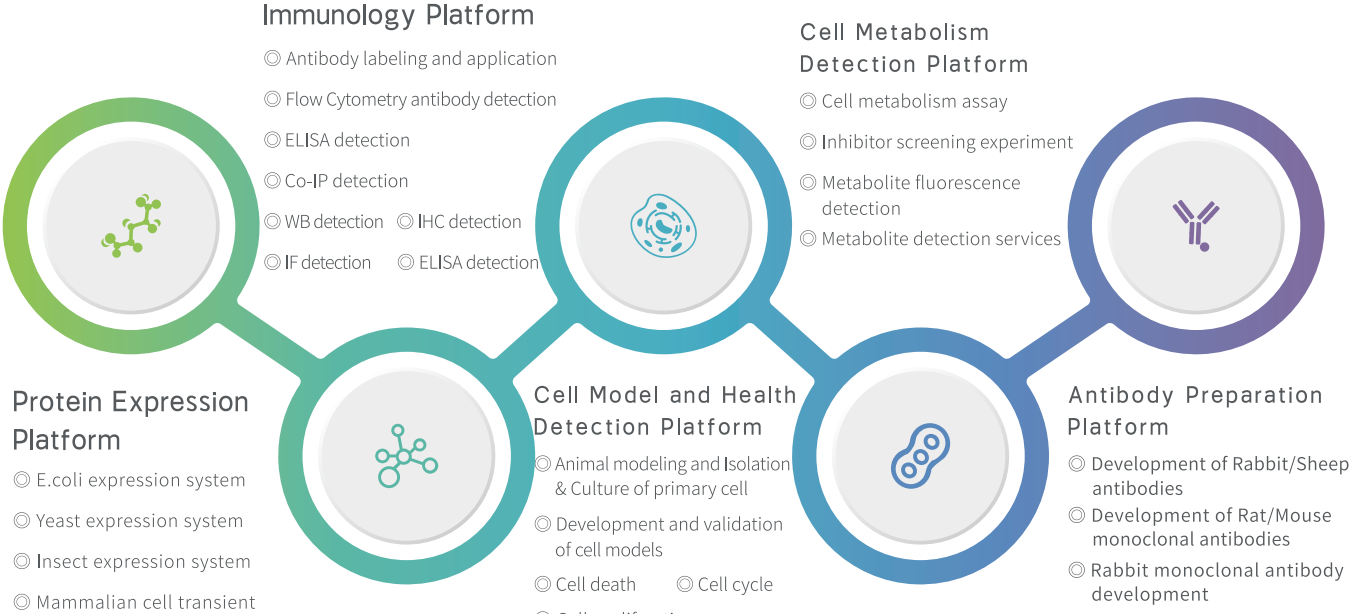

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