# Apoptosis

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## Description

This lecture note on "**Apoptosis**" were prepared and delivered to my BVSc.&A.H students studying Veterinary Pathology courses. This course was offered during the academic year 2022-23 in the second professional year at College of Veterinary & Animal Sciences, Sardar Vallabhbhai Patel University of Agriculture and Technology, Meerut, Uttar Pradesh, India. I had tried my level best to extract the contents simplify the facts in easy to memories in very short time. Further constructive suggestions to improve this lecture note are always welcome its users on my email and whatsapp.

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# What is Apoptosis

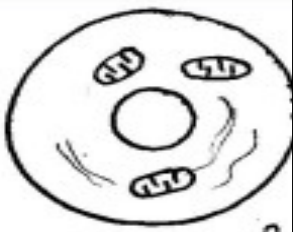
- Programmed cell death (PCD)
- “falling off,”
- Cellular suicide
- Condensation of nuclear chromatin is a hallmark
- Active process involves active production of enzymes (caspases)

1. Physiological: Important process to remove unwanted cells of body

- During embryogenesis
- Harmonal (eg menstrual /oestrus cycle)
- Deletion of autoreactive immunocytes

2. Pathological: Sub lethal /mild pathological injury may progress to apoptosis

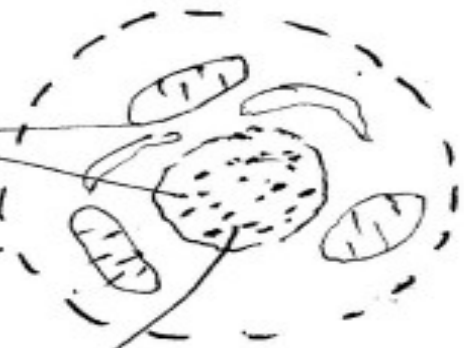
❖ Failure of Apoptosis → Cancer/Tumor



Normal

Necrosis

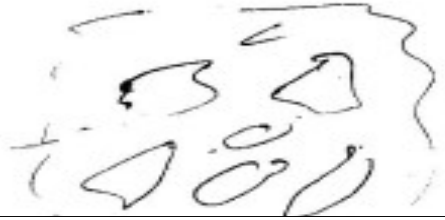
Cell injury / Hypoxia
free radical, toxins



① Swelling
- Cell organale

② Membrane damage

③ Clumping of nuclear chromatin

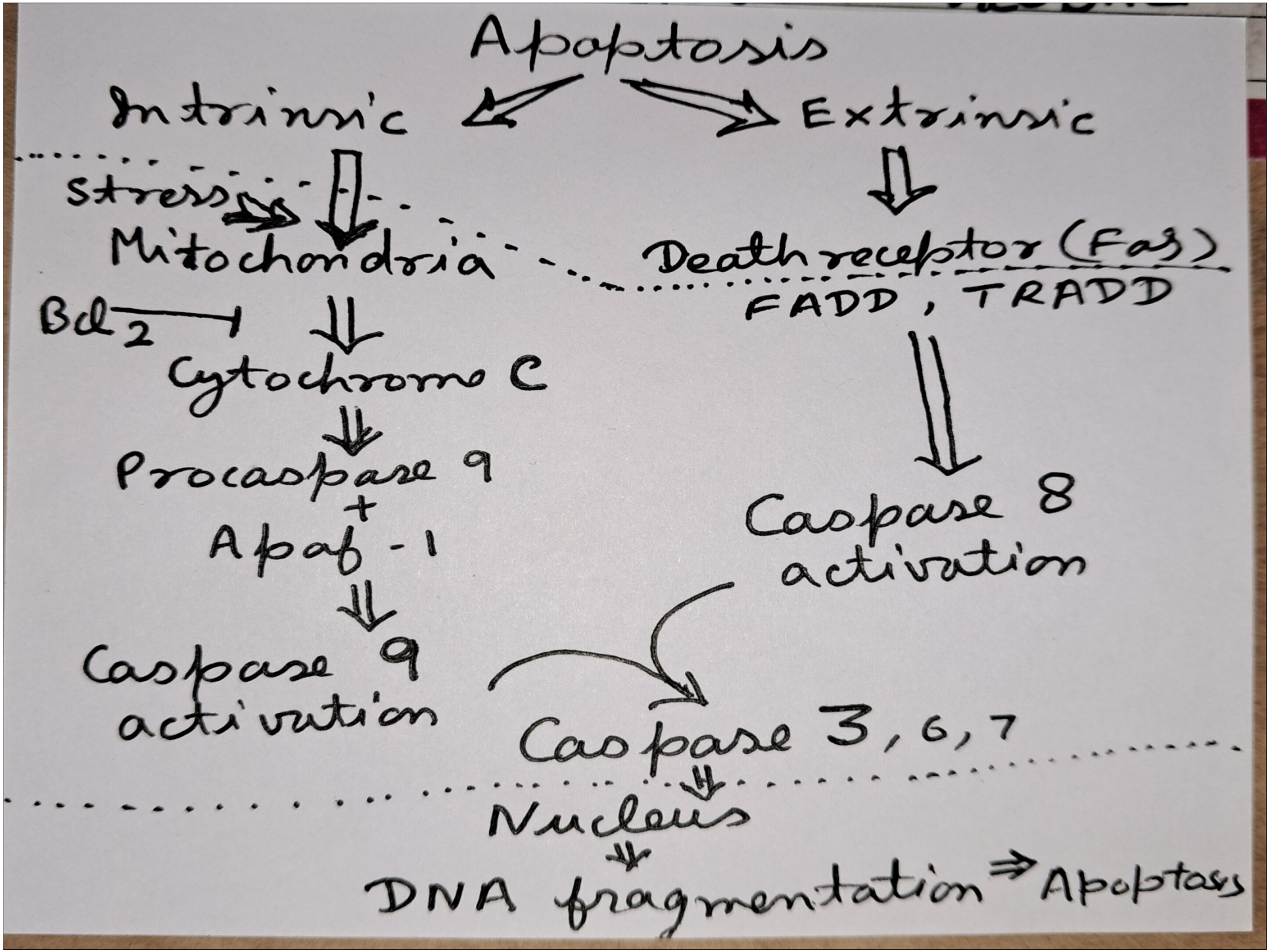


# Important molecules in execution of Apoptosis

1. Plasma membrane receptors of TNF family (FAS & TNF) have death domain (FADD & TRADD) → Activation of caspases → Apoptosis
2. BCL-2 family proteins well known for suppressing apoptosis by sparing Pro apoptotic proteases (Apaf-1). BCL-2+Apaf-1 →Caspases → Apoptosis
3. Caspases: Cysteine proteases responsible for protein hydrolysis

   Two types

1) Initiator: eg. Caspase 8, 9
2) Execution: eg. Caspase **3**, 6, 7


Apoptosis
Intrinsic
Extrinsic
Stress
Mitochondria
Death receptor (Fas)
FADD, TRADD
Bcl2
Cytochrome C
Procaspase 9
+
Apaf-1
Caspase 9 activation
Caspase 8 activation
Caspase 3, 6, 7
Nucleus
DNA fragmentation ⇒ Apoptosis

| Criteria | Necrosis |
| --- | --- |
| Stimulus | Cell injury/tissue damage<br>Pathological |
| Cell affected | Many |
| Process | Passive process,<br>Uncontrolled release of cellular contents |
| Inflammation | Present |
| Clearing of dead cell / content by | Infiltrating inflammatory cells (Macrophages) |
| Cellular alteration | Cellular swelling<br>Cell lysis |
| Nucleus | Pyknosis, karyorrhexis, karyolysis |

# Variants

1. Programmed cell death (PCD)
2. Accidental cell death (ACD)

- Apoptosis is a form of PCD that due to activation of caspases, leading to DNA condensation & fragmentation. Nonlytic cell death. No inflammatory.
- Pyroptosis : Lytic cell death. Inflammatory stimuli →caspase-1 activation →cytokines → Membrane perforation → leakage of cytosolic content.
- Necroptosis: Lytic cell death → DAMP release → Cytokines → Membrane perforation → leakage of cytosolic content.
- Ferroptosis: Mostly due to iron-dependent lipid peroxidation (can be prevented by glutathione peroxidase.
- Necrosis: Lytic cell death → DAMP release → propagation of inflammation.

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