



LICENCIATURA EN NUTRICIÓN

FISIOPATOLOGÍA 1

CUADRO SINÓPTICO: MUERTE CELULAR

DOCENTE: MIGUEL BASILIO ROBLEDO

ALUMNA: XOCHITL PÉREZ PASCUAL

TERCER CUATRIMESTRE

GRUPO "A"

TAPACHULA CHIAPAS

08 DE MAYO DE 2020

M  
U  
E  
R  
T  
E  
  
C  
E  
L  
U  
L  
A  
R

APOPTOSIS

Muerte celular programada que elimina células lesionadas y envejecidas. Responsable de procesos fisiológicos como:

- Destrucción celular durante el desarrollo embrionario.
- Involución de tejidos dependientes de hormonas.
- Muerte de células inmunitarias.
- Separa los dedos de manos y pies.

Vías básicas

extrínseca

Comprende la activación de receptores de (FNT) y receptor ligando Fas.

Cuando se fija a su receptor, se congregan las proteínas al extremo citoplasmático del receptor Fas para formar un complejo de inicio de muerte.

El complejo convierte la procaspasa-8 en caspasa-8. Activa una cascada de caspasas que ejecutan el proceso de apoptosis.

intrínseca

Se activa por daño del ADN, EOR, hipoxia, disminución de ATP, senescencia celular y activación de la proteína p53 por daño al ADN32.

Implica apertura de los poros de permeabilidad de la membrana mitocondrial con la liberación de cromo C desde las mitocondrias hasta el citoplasma.

Activa la caspasa-3, aumento de proteína proapoptósica, como Bid y Bax, activación de la caspasa 8 en la vía intrínseca.

NECROSIS

Muerte celular en un órgano o tejido que sigue siendo parte de un órgano viviente.

Causa pérdida de la integridad de la membrana celular y desencadena el proceso inflamatorio.

Mecanismos

Necrosis por licuefacción

Ocurre cuando algunas células mueren, pero sus enzimas catalíticas no se destruyen.

Necrosis por coagulación

Desarrolla acidosis y desnaturaliza las proteínas enzimáticas y estructurales de las células.

Necrosis por caseosa

Característica de necrosis por coagulación en la que las células muertas persisten de manera indefinida.

## bibliográfia

1. Rubin R., Strayer, D. (Eds.). (2012). Rubin's pathology: Clinicopathologic foundations of medicine (6th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
2. Glass D. J. (2010). Signaling pathways perturbing muscle mass. *Current Opinion in Clinical Nutrition and Metabolic Care* 13(3), 225–229.
3. Tang S. C., Lai K. N. (2009). The ubiquitin—proteasome pathway and IgA nephropathy: A novel link? *Kidney International* 75(5), 457–459.
4. Luptak I., Balschi J. A., Xing Y., et al. (2005). Decreased contractile and metabolic reserve in peroxisome proliferator—activated receptor-alpha-null hearts can be rescued by increasing glucose transport and utilization. *Circulation* 112(15), 2339–2346.
5. Heineke J., Molkentin J. D. (2006). Regulation of cardiac hypertrophy by intracellular signalling pathways. *Nature Reviews: Molecular Cell Biology* 7, 589–600.
6. DynaMed Corporation. (2011). Endometrial hyperplasia. *Dyna Med* 8/23.
7. Lee J. Y., Foster H. E., McVary K. T., et al. (2011). Recruitment of participants to a clinical trial of botanical therapy for benign prostate hyperplasia. *Journal of Alternative and Complementary Medicine* 17(5), 469–472.
8. Boughey J. C., Hartmann L. C., Anderson S. S., et al. (2010). Evaluation of the tyrrer-cuzick model for breast cancer risk prediction in women with atypical hyperplasia. *Journal of Clinical Oncology* 28(22), 3591–3596.
9. Barbera, M., Fitzgerald R. C. (2009). Cellular mechanisms of Barrett's esophagus development. *Surgical Oncology Clinics of North America* 18(3), 393–410.
10. Wright C. J., Kirpalani H. (2011). Targeting inflammation to prevent bronchopulmonary dysplasia: Can new insights be translated into therapies? *Pediatrics* 128(1), 111–126.
11. Lopez J. K., Shtifman A. (2010). Intracellular [beta] amyloid accumulation leads to age-dependent progression of Ca (2+) dysregulation in skeletal muscle. *Muscle and Nerves* 42(5), 731–738.
12. Miedlinger D., Dalkeler T. (2008). Images in clinical medicine: Chronic venous insufficiency and dystrophic subcutaneous calcification. *New England Journal of Medicine* 358(9), e10.
13. Sencimen M., Gulses A., Ogretir O., et al. (2010). Dystrophic calcifications arising in the masseter muscle: A case report—2010. *Quintessence International* 41(4), 295–297.
14. Catavid J. C., Divertro M. L., Torres E. A, et al. Warfarin-induced pulmonary metastatic calcification and calciphylaxis in a patient with end-stage renal disease. *Chest* 139(6), 1503–1506.
15. McConnell T. H., Hull K. L. (2011). Human form human function: Essentials of anatomy & physiology. Philadelphia, PA: Lippincott Williams & Wilkins.
16. Koda S. (2010). A study of general practitioner's knowledge of ionizing radiation from diagnostic imaging examinations. *Quality in Primary Care* 18(6), 391–397.
17. Gray J., Evans N., Taylor B., et al. (2009). State of the evidence: The connection between breast cancer and the environment. *International Journal of Occupational and Environmental Health* 15(1), 43–78.
18. Autler P., Dori J. F., Eggermont A. M., et al. (2011). Epidemiological evidence that UVA radiation is involved in the genesis of cutaneous melanoma. *Current Opinions in Oncology* 23(2), 189–196.
19. Grampurchit V. J., Dinesh U. S., Rao R. (2011). Multiple cutaneous malignancies in a patient of xeroderma pigmentosum. *Journal of Cancer Research and Therapeutics* 7(2), 205–207.
20. Takayanagi T., Sasaki H., Kawashima A., et al.

(2011). A new enteral diet, MHN-02, which contains abundant antioxidants and whey peptide, protects against carbon tetrachloride-induced hepatitis. *Journal of Parenteral and Enteral Nutrition* 35(4), 516–522. 21. Chandraw L., Cataldo R. (2010). Lead poisoning: Basics and new developments. *Pediatrics in Review* 31(10), 399–406. 22. Centers for Disease Control and Prevention. (2011). Lead. [Online]. Available: <http://www.cdc.gov/nceh/lead/>. Retrieved September 19, 2011. 23. Centers for Disease Control and Prevention. (2009). Lead prevention tips. [Online]. Available: <http://www.cdc.gov/nceh/lead/tips.htm>. Retrieved September 19, 2011. 24. Balmadrid C., Bono M. J. (2009). Recognizing and managing lead and mercury poisoning. *Emergency Medicine* 41(9), 35–43. 25. Ning M., Sarracino D. A., Kho A. T., et al. (2011). Proteomic temporal profile of human brain endothelium after oxidative stress. *Stroke* 42(1), 37–43. 26. Giustarini D., Dalle-Donne I., Tsikas D., et al. (2009). Oxidative stress and human diseases: Origin, link, measurement, mechanisms, and biomarkers. *Critical Reviews in Clinical Laboratory Sciences* 46(5–6), 241–281. 27. Hambali Z., Ahmad Z., Arab S., et al. (2011). Oxidative stress and its association with cardiovascular disease in chronic renal failure patients. *Indian Journal of Nephrology* 21(1), 21–25. 28. Martin I., Grotewiel M. S. (2006). Oxidative damage and age-related functional declines. *Mechanisms of Ageing and Development* 127, 411–423. 29. Shimoda L. A., Semenza G. L. (2011). HIF and the lung: Role of hypoxia-inducible factors in pulmonary development and disease. *American Journal of Respiratory and Critical Care Medicine* 183(2), 152–156. 30. Babashah S., Soleimani M. (2011). The oncogenic and tumour suppressive roles of micro-RNAs in cancer and apoptosis. *European Journal of Cancer* 47(8), 1127–1137. 31. Ji X., Jiang C., Liu Y., et al. (2011). Fas ligand gene transfer effectively induces apoptosis in head and neck cancer cells. *Acta Otolaryngologica* 131(8), 876–881. 32. Fulda S. (2011). Exploitation of apoptosis pathways for childhood Leukemia. *Current Pediatric Reviews* 7(4), 266–270. 33. Dorner T., Lipsky P. E. (2006). Signalling pathways in B cells: Implications for autoimmunity. *Current Topics in Microbiology and Immunology* 305, 213–240. 34. Sanz A., Caro P., Ayala V., et al. (2006). Methionine restriction decreases mitochondrial oxygen radical generation and leak as well as oxidative damage to mitochondrial DNA and proteins. *FASEB Journal* 20, 1064–1073. 35. Halaschek-Wiener J., Khattra J. S., Pouzyrev A., et al. (2005). Analysis of long-lived *C. elegans* daf-2 mutants using serial analysis of gene expression. *Genome Research* 15, 603–615.