Molecular and Cellular Biomedical Sciences

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REVIEW ARTICLES

RESEARCH ARTICLES

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Molecular and Cellular Biomedical Sciences

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Article:

Sandra F, Esposti MD, Ndebele K, Gona P, Knight D, Rosenquist M, et al. Tumor Necrosis Factor-Related Apoptosis-Inducing Ligand Alters Mitochondrial Membrane Lipids. Cancer Res. 2005; 65(18): 8286-97.

Book

Murray PR, Rosenthal KS, Kobayashi GS, Pfaller MA. Medical microbiology. 4th ed. St. Louis: Mosby; 2002.

Chapter in a book:

Rosenberg GA. Matrix metalloproteinase and proteolytic opening of the blood-brain-barrier in neuroinflammation. In: deVries E, Prat A, editors. The Blood-brain Barrier and Its Microenvironment Basic Physiology To Neurological Disease. New York: Taylor and Francis Group; 2005. p.335-58.

Dissertation/Thesis/Essay:

Arlauckas SP. Near infrared fluorescent choline kinase alpha inhibitors for cancer imaging and therapy [Dissertation]. Philadelphia: University of Pennsylvania; 2015.

Part of Website/Monograph:

Medline Plus [Internet]. Bethesda: US National Library of Medicine; ©2009. Diabetic Kidney Problems [update 2015 Nov 2; cited 2015 Nov 16]. Available from: https://www.nlm.nih.gov/medlineplus/diabetickidneyproblems.html/.

Conference Paper:

Fledelius HS. Myopia and significant visual impairment: global aspects. In: Lin LLK, Shin YF, Hung PT, editors. Myopia Updates II: Proceedings of the 7th International Conference on Myopia 1998 Nov 17-20, Taipei. Tokyo: Springer; 2000. p.3-17.

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CONTENT

REVIEW ARTICLES

RESEARCH ARTICLES

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Abstract

DDC 362.19897
Aryana IGPS, Hapsari AAAR, Kuswardhani RAT (Geriatric Division, Internal Medicine Department, Faculty of Medicine, Udayana University, Denpasar, Indonesia)

Myokine Regulation as Marker of Sarcopenia in Elderly

Mol Cell Biomed Sci. 2018; 2(2): 38-47

Abstract (English)

The elderly population will increase as well as increasing life expectancy. Health problems in elderly will be more complex and need a comprehensive management. One of the problems that arise from the aging process is sarcopenia. Sarcopenia is a decreasing in muscle mass and muscle strength or muscle function caused by multifactorial not only due to aging process, but also nutrition, immobilization, genetics and others risk factors. Muscle is an endogen organ that produces various proteins that can affect the health system. This protein is referred to as myokine. Myokine is anti-inflammation cytokine and peptide produced by striated muscles. Physical activity results in myokine secretion that can reduce inflammation due to a sedentary lifestyle. Inflammation can lead to worsening sarcopenia and fat accumulation in striated muscles, thus reducing muscle mass, muscle strength and causing physical inactivity. The most of this type myokine have anti-inflammation effect have work as autocrine, paracrine and endocrine. Chronic inflammation is a contributor that plays a role in the pathophysiology of various diseases including sarcopenia, it will protected by myokine. Myokine can affect the metabolism of glucose, fatty acids, angiogenesis, myogenesis, neurogenesis, and can explain the relationship between muscle, liver, fat, tissue and brain. Some knewn myokines include interleukin (IL)-6, IL-8, IL-5, brain-derived neurotrophic factor (BDNF), fibroblast growth factor 21 (FGF-21), leukemia Inhibitory factor (LIF), irisin and secreted protein acidic and rich in cysteine (SPARC). Physical exercise can induce myokine secretion from striated muscle to circulation. Through these mechanisms, myokine is expected to improve metabolism of glucose, fat and protein muscle, liver, fat, tissue, brain and reduce the incidence some comorbidity especially sarcopenia. Finally, it's will be decreasing of disability, morbidity and mortality rate in elderly

Keywords: myokine, sarcopenia, elderly

DDC 616.02774
Chouw A, Triana R, Dewi NM, Darmayanti S, Rahman MN, Susanto A, Putera BW, Sartika CR (Magister Program of Clinical Pharmacy, Faculty of Pharmacy, Padjajaran University, Bandung, Indonesia)

Ischemic Stroke: New Neuron Recovery Approach with Mesenchymal and Neural Stem Cells

Mol Cell Biomed Sci. 2018; 2(2): 48-54

Abstract (English)

Stroke is a leading cause of death and long-term disability. This due to the ischemic event that cause by embolism of blockage blood flow. Thrombolytic agent plasminogen activator (tPA) is the only treatment approved by FDA. However, the used of tPA is limited to the short time window period. Neural stem cells (NSCs) show the potential to repair neuronal damage naturally after stroke. However, isolating NSCs is a challenging process due to the limitations of the method and its invasiveness. Some studies that had used mesenchymal stem cell (MSCs) as the main source of stem cell for therapy show that MSCs have the potency to differentiate into NSCs. *in vitro*, a differentiation process from MSC to NSC has been developed by combining the supplement or growth factor needed in the culture media.

Keywords: stem cells, neuron stem cell, mesenchymal stem cell, stroke, trans-differentiation

DDC 571.936
Sandra F (Department of Biochemistry and Molecular Biology, Division of Oral Biology, Faculty of Dentistry, Trisakti University, Jakarta, Indonesia)

Survivin Ser81 Plays An Important Role in PI3K/Akt/mTOR Signaling Pathway

Mol Cell Biomed Sci. 2018; 2(2): 55-9

Abstract (English)

Background: Survivin, a member of the inhibitor of apoptosis protein family, has been associated with protection from cell apoptosis and regulation of mitosis. Phosphorylated-Survivin at Ser81 was reported to provide cytoprotection against tumor necrosis factor (TNF)-related apoptosis-inducing ligand (TRAIL) in L929 cells by inducing a backloop activation of phosphatidylinositol 3-kinase (PI3K). Therefore Akt as a possible substrate of PI3K was investigated.

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Materials and Methods: L929 cells were pretreated with/without 50 mM LY294002 or 10 mM Perifosine, and infected with viral particle of Survivin, anti sense of Survivin, Ser81Ala mutated Survivin or vector only. Cells were then harvested, lysed and subjected to immunoblot assay to detect Akt, phosphorylated Akt (Ser473), mammalian target of rapamycin (mTOR), phosphorylated-mTOR (Ser2448).
Results: Survivin induced Akt and mTOR phosphorylations in a viral particle concentration dependent manner. Pretreatment of LY294002 or Perifosine prior to Survivin infection, attenuated Akt or mTOR phosphorylations, respectively. Low Akt or mTOR phosphorylations were observed when L929 cells were infected with Ser81Ala mutated Survivin.
Conclusions: Ser81 phosphorylation site of Survivin played an important role in activating Survivin/PKA/PI3K/Akt/mTOR signaling pathway.

Keywords: survivin, Ser81, Akt, mTOR, LY294002, perifosine

DDC 616.99449
Widowati W, Jasaputra DK, Sumitro SB, Widodo MA, Afifah E, Rizal, Rihibiha DD, Kusuma HSW, Murti H, Bachtiar I, Faried A (Medical Research Center, Faculty of Medicine, Maranatha Christian University, Bandung, Indonesia)
Direct and Indirect Effect of TNFα and IFNγ Toward Apoptosis in Breast Cancer Cells
Mol Cell Biomed Sci. 2018; 2(2): 60-9
Abstract (English)
Background: Breast cancer (BC) is the leading cause of death cancer in women. Cancer therapies using TNFα and IFNγ have been recently developed by direct effects and activation of immune responses. This study was performed to evaluate the effects of TNFα and IFNγ directly, and TNFα and IFNγ secreted by Conditioned Medium-human Wharton's Jelly Mesenchymal Stem Cells (CM-hWJMSCs) toward apoptosis of BC cells (MCF7).
Materials and Methods: BC cells were induced by TNFα and IFNγ in 175 and 350ng/mL, respectively. CM-hWJMSCs were produced by co-culture hWJMSCs and NK cells that secreted TNFα, IFNγ, perforin (Prf1), granzyme B (GzmB) for treating BC cells. The BC cells were treated with CM-hWJMSCs in 50%. The expression of apoptotic genes Bax, p53, and the anti-apoptotic gene Bcl-2 were determined using RT-PCR.
Results: TNFα and IFNγ at concentration of 350 ng/mL induced higher Bax expression compared to 175 ng/mL. TNFα and IFNγ 350 ng/mL, 175 ng/mL induced p53 expression, whilst TNFα and IFNγ at 350 ng/mL decreased Bcl-2 expression. Perf1, GzmB, TNFα and IFNγ-containing CM-hWJMSCs induced significantly apoptosis percentage, induced Bax expression, but did not effect p53, Bcl-2 expression.
Conclusion: TNFα and IFNγ directly induce Bax, p53, decrease Bcl-2 gene expression. The Prf1, GzmB, TNFα, IFNγ-containing CM-hWJMSCs induce apoptosis and Bax expression.

Keywords: breast cancer, Wharton's Jelly mesenchymal stem cells, TNFα, IFNγ

DDC 616.53
Nasution K, Putra IB, Jusuf NV (Department of Dermatology and Venereology, Faculty of Medicine, Universitas Sumatera Utara, Medan, Indonesia)
No Association Between Lipid Profiles and Acne Vulgaris
Mol Cell Biomed Sci. 2018; 2(2): 70-2
Abstract (English)
Background: Acne vulgaris is a chronic inflammation of pilosebaceous follicle that can spontaneously heal with clinical manifestations such as blackhead, papules, pustules, nodule, and cyst on the face, upper chest, arms, and back. Until now, the effect of lipid metabolism on sebaceous gland secretions in the pathogenesis of acne vulgaris is still under research.
Materials and Methods: An analytic observational study with cross sectional design involving 30 acne vulgaris and 30 control subjects was conducted. Blood samples were taken from subjects and lipid profile levels were measured. The data were then statistically analyzed.
Results: From this research, there was no significant association between lipid profiles with acne vulgaris. There was not any significant difference between the acne vulgaris and the control subjects for total cholesterol, High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL) and triglyceride levels (p>0.05).
Conclusion: There is no significant association between lipid profiles levels and acne vulgaris.

Keywords: acne vulgaris, lipid profiles, total cholesterol, HDL, LDL, triglyceride

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