represent a mechanism responsible for muscle decline. Furthermore, increased levels of apoptosis have also been reported in old rats undergoing muscle atrophy. We have previously demonstrated that 17β-estradiol (E2) inhibits apoptosis in skeletal muscle cells. Here, we show that the proapoptotic protein Bax binds to the cytoplasmic protein 14-3-3 and ERK. Upon E2 treatment Bax/14-3-3 interaction is increased while Bax/ERK complex is diminished in agreement with E2 induced-ERK translocation to mitochondria. Apoptotic stimulation with hydrogen peroxide disrupts Bax/14-3-3 association and increases Bax/ERK interaction and these effects are reversed by E2 pretreatment. Moreover, flow cytometry studies show that the apoptotic stimulus induces a decrease in mitochondrial membrane potential, a change which is prevented by preincubation with E2. These findings suggest that Bax is involved in negative regulation of muscle apoptosis by E2 affecting mitochondrial function.

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Role of testosterone in the intrinsic apoptotic pathway of skeletal muscle

L. Pronsato, R. Boland, L. Milanesi
Departamento de Biología, Bioquímica y Farmacia, Universidad Nacional del Sur, 8000 Bahía Blanca, Argentina

Apoptosis is a systematic set of events that results in cellular self-destruction without inflammation or damage to the surrounding tissue. Experimental animal data indicate that apoptosis is activated in the aged skeletal muscle, contributing to the pathogenesis of sarcopenia. Given the role played by mitochondria in the induction and regulation of programmed cell death, intensive investigations have focused on mitochondria-driven myonuclear apoptosis. We have previously demonstrated that testosterone protects against H_2O_2-induced apoptosis in C2C12 muscle cells. Typical changes of apoptosis such as nuclear fragmentation, cytoskeleton disorganization, mitochondrial reorganization/dysfunction and cytochrome c release induced by H_2O_2, are abolished when cells are previously exposed to the hormone. In the present work, we identified molecular events that occur during the anti-apoptotic effect of testosterone on C2C12 cells. At short times of exposure to H_2O_2, cells exhibit a defense response showing ERK2, Akt and Bad phosphorylation and an increase of HSP70 levels. At longer treatment times with the apoptotic agent, dephosphorylation of the proteins mentioned before, cytochrome c release, PARP cleavage and DNA fragmentation occur, but when cells are treated with testosterone prior to H_2O_2, Bad inactivation (phosphorylation), an increase in actin levels, translocation of HSP90 to mitochondria and a decrease in Bax levels are observed, revealing that, the steroid hormone regulates the apoptotic intrinsic pathway. Although further studies are required to establish the molecular basis of sarcopenia associated to states of testosterone deficit, the data presented allow us to begin to elucidate the mechanism by which the hormone prevents apoptosis in skeletal muscle.

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Frontal tomographic evaluation of mandibular condyle bone surfaces

S. Azcona, L. Giambartolomei, J. Pescio, D. Salica
Faculty of Dentistry, National University of Córdoba, Argentina
School of Medical Sciences, National University of Córdoba, Argentina
Health and Science Education Team, Argentina

This study aims to generate a technique to provide information about the surface structure of the mandibular condyle (MC) of the temporomandibular joint (TMJ). 40 TMJ were surveyed in 20 edentulous people with prosthesis (12 women and 8 men), then were quantified in 80 degrees of joint surfaces osteoarthritis by computed tomography (CT) in frontal coronal MC. Dentures were removed and replaced by a silicone block in order to standardize the separation of the jaws. To identify the surface, anatomic parameters were used, determining the medial and lateral articular surfaces (MAS and LAS). For quantification, the following were established: Grade I: cortical preserved and continuous bone marrow of normal image; Grade II: cortical interrupted without bone marrow involvement (mild osteoarthritis); Grade III: cortical loss, more loss of bone marrow (severe osteoarthritis). Among images, the most representative cut was selected, choosing the greatest dimension. **The results showed:** MAS: according to incidence, osteoarthritis was equally evident for Grades I and III (12) and higher for Grade II (16). LAS: according to incidence, osteoarthritis was the same for Grades I and III (11), and more in Grade II cases (18). **In conclusion:** The most frequent condition found at the articular bone surfaces of MC was osteoarthritis. The coincidence between MAS (28) and LAS (29) was reported in 21 cases (75.5%). For a statistical probability of 0.05: $x^2 = 0.589$; Fisher test = 0.704. Computed tomography proved to be the appropriate complement for diagnosis of diseases of the bone surfaces of the TMJ.

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Bone fluoride (F) uptake rate determination

M. Lupo, B. García, A. Rigalli
Bone Biology Laboratory, Rosario, Argentina
E-mail address: arigalli@unr.edu.ar

F is a drug used to prevent bone mass loss and to measure bone resorption. A mathematical model has been developed to measure bone formation and resorption in rats. This method uses data from plasma and urinary F (Fu) concentration after a non toxic sodium fluoride (NaF) dose. It has been validated in different animal models with modified bone resorption by drugs, surgery and nutrition. However unexpected results might be a consequence of the assumptions made. For instance, 33% of bone F uptake constant (Ko) values are negative. This affects F blood elimination (Ke) and urinary depuration (Ku) constants. Another disadvantage is that plasma F determination is not as accurate as Fu measurement. The main objective of this essay was to obtain the rate constants of F in the rat, based on Fu measurements after an intravenous dose of NaF. Sprague–Dawley-adult rats were maintained under the effects of general anesthesia and rectal hydration. Urine samples were obtained every 15 min through urethral catheterism for 60 min (To). Afterwards 1 μmol F/100 g body weight was injected intravenously (Do) and continued collecting urine for 240 min. Fu was determined and basal excretion calculated (Uo, moles) and adjusted by: Uo = a.t, a = Fu basal elimination rate. Fu excretion after Do was adjusted by: $U = (ku.Do/Ke).(1 - \exp(-Ke.(t - to))) + Uo$, therefore ke and ku were obtained. The difference between them resulted in Ko. The values of the constants are expressed in min^{-1} and shown as media ± sem: Ke = 0.013 ± 0.002, Ku = 0.0013 ± 0.0006, and Ko = 0.012 ± 0.003. Ko values were as expected. These values permit a more accurate evaluation of F bone uptake, consequently an accurate estimation of bone resorption. The method has the advantages of obtaining pharmacokinetics values immediately and a more accurate F determination, and requires low complexity interventions.

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Fractures due to femoral insufficiency: Use of teriparatide

L. Plantalech, F. Comba, A. Morandi
Servicios de Endocrinología, Traumatología y Anatomía Patológica del Hospital Italiano de Buenos Aires, Argentina

In recent years, shaft and unilateral or bilateral subtrochanteric femoral and other bone fractures (Fx) have been described linked to prolonged use of Bisphosphonates (BP). We present the case of a patient with bilateral fractures of both femoral necks and the response to treatment with teriparatide. A 65-year-old woman consulted with right hip pain of 9 month duration. Incomplete fracture of femoral neck on the left hip and incipient fracture of right hip were observed. An intramedullary nail was placed at left and inadequate bone consolidation was observed. She has diagnosis of osteoporosis four years earlier and treated for the last two with BP. X rays show complete subcervical Fx of left femur, incomplete Fx of right and thickening of femoral cortical bone. Spine BMD: T −2.0/Total Femur: T: −0.1. Markers of bone turnover in normal range, hyperparathyroidism with hypovitaminosis D was seen in biochemical test. Adynamic bone disease: absence of osteoid tissue and decreased bone resorption surface, scarce double tetracycline label and delayed mineralization rate, was observed in bone biopsy. Treatment with calcium, cholecalciferol, thiazides and teriparatide was started. At 6 months, X-ray showed improvement of both Fx and callus in the left femur. Atypical Fxs in both femoral necks were observed in a patient with short exposure to BP associated to adynamic bone disease. Linking atypical femur Fx with chronic use of BP is controversial and a predisposition to this type of fracture in patients with pre-treatment low bone turnover is postulated. The importance of assessing the type of bone biology prior to indicating BP is stressed. Teriparatide activates bone remodelling units and has proved a useful treatment for this type of Fx with slow bone healing.

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Familiar hypophosphatemic rickets: Molecular findings in thirteen Argentinean families

G. Alonso, L. Plantalech, R. Guelman, S. Gonzalez, M.A. Redal, H. Cassinelli, T. Pasqualini
Sección Endocrinología, Departamento de Pediatría,
Servicio de Endocrinología, Unidad de Medicina Molecular (ICBME), Hospital Italiano, Argentina
División Endocrinología, Hospital de Niños R Gutiérrez, Buenos Aires, Argentina

Human disorders of phosphate (Pi) handling and skeletal mineralization can result from inactivating mutations in PHEX, an X linked dominant disorder (XLH). Autosomal-dominant hypophosphatemic rickets (ADHR) is a rare disorder caused by mutation in FGF23 which in