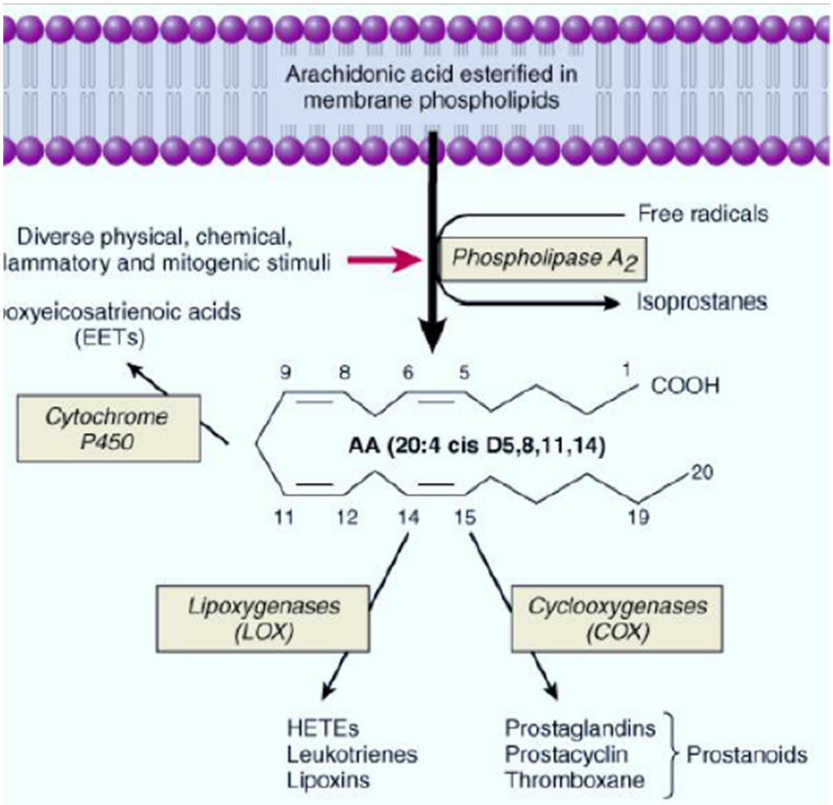
Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

- Las prostaglandinas se sintetizan desde el ácido araquidónico mediante las COX-1 Y COX-2.
- La producción constitutiva de prostaglandinas es un prerrequisitio para los procesos fisiológicos normales como la remodelación ósea, la vasodilatación y vasoconstricción , la permeabilidad vascular, la agregación plaquetaria o la respuesta de las células tendinosas al movimiento.



El ácido linoléico (omega 6) tiene la última insaturación a seis posiciones del final , produce el ácido araquidónico y pueden formar directamente las porstaglandinas proinflamatoiras (Pglandina G2) . El ácido linolénico (omega 3) tiene la última insaturación a 3 posiciones del final lo que impide que produza por sí mismo araquidónico, producen la PG g3 antiinflmatoira).

LA OXIDACIÓN CATALÍTICA DE LOS ÁCIDOS GRASOS ES UN REQUISITO PARA LA PRODUCCIÓN DE EICOSANOIDES QUE TIENE LUGAR GRACIAS A DOS FAMILIAS DE ENZIMAS (COX1 Y 2) Y LIPOOXIGENASAS. EL PRIMER PASO DE LA SÍNTESIS DE EICOSANOIDES OCURRE CUANDO LA CÉLULA ES ACTIVADA POR LESIÓN CELULAR, CITOQUINAS, FACTOREES DE CRECIMIENTO U OTROS ESTÍMULOS.PGD2 is degraded to 15d-PGJ2 (through COX-2), a strong ligand to PPARγ and has strong anti-inflammatory

Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

Properties Numerosos estudios han demostrado que los receptores activados por proliferadores de peroxisomas (PPARs) están implicados en varias vías metabólicas tales como la homeostasis glucídica y de lípidos y entre otras en el control de la proliferación y diferenciación celular1-5.

Los PPARs son factores de transcripción dependientes de ligando que pertenecen a la superfamilia de receptores nucleares hormonales6,7. El nombre de PPAR deriva en los roedores8 de su capacidad para estimular la proliferación de peroxisomas, organelas que intervienen en la β-oxidación de los ácidos grasos de cadena larga.

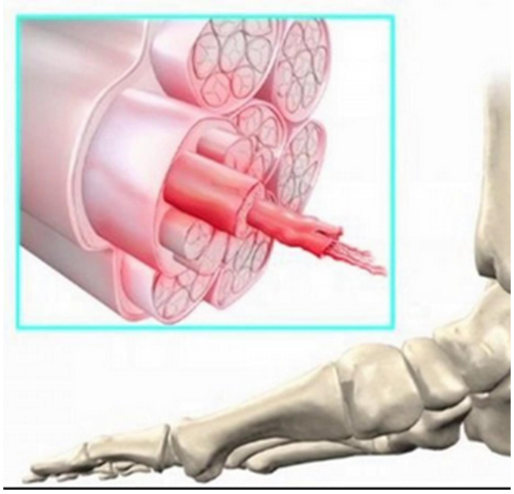
PPAR-α se expresa fundamentalmente en el hígado, riñón y músculo esquelético e interviene en la oxidación de los ácidos grasos6,10. PPAR- b/d interviene, entre otros procesos, en el desarrollo, implantación del embrión, mielinización del cuerpo calloso, etc11. PPAR-g se expresa principalmente en el tejido adiposo donde constituye un regulador principal de la diferenciación adipocitaria y participa en la homeostasis glucídica6,7. Sin embargo, también se expresa en otros territorios como en el riñón y en los vasos sanguíneos por lo que se le ha supuesto un papel en la regulación del tono vascular12. Algunas de las variantes alélicas y mutaciones caracterizadas en el gen PPAR-g humano se han asociado con la obesidad, la hipertensión arterial y la hipertensión arterial relacionada con la obesidad13-18.

Rossi, A. G. & Sawatzky, D. A. *The resolution of inflammation (Birkhäuser, Basel ,2008), p. 7.*

PAPEL DE LAS PROSTAGLANDINAS EN LA TENDINOPATÍA:

- La prostaglandina E2 (PGE2) aumenta en la región peritendinosa aquílea y rotuliana en humanos y ratones sanos, respectivamente, tras el ejercicio in vivo , in vitro o tras lesión.

(Langberg et al., 1999) (Zhang and Wang, 2010)(Almekinders et al., 1993, 1995; Wang et al., 2004)(Tilley et al., 2001)(Tsuzaki et al., 2003; Yang et al., 2005).



Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

PAPEL DE LAS PROSTAGLANDINAS EN LA TENDINOPATÍA:

- Complejo y diverso con evidencia a favor de efectos deletéreos (PGE_1 →muerte o destrucción celular) y a favor también de efectos beneficiosos.
- EFECTOS DELETEREOS: La inyección peritendinosa de PGE_1 produce cambios degenerativos similares a los observados en las tendinopatías humanas tras 5 semanas de lesión (Sullo et al., 2001).
- EFECTOS BENEFICIOSOS: Parece existir evidencia sobre efectos positivos de la PGE_2 en la curación del tendón. La inyección peritendinosa de 800 ng PGE_2 en tendones rotulianos de ratas mejoró las propiedades mecánicas de los tendones comparado con los controles y con el grupo de inyección de suero salino (Ferry et al., 2012).

Deletéreo = Perjudicial

PGE2 VERSUS AINES:

- El papel de la PGE_2 EN ALTAS DOSIS PARECE TENER EFECTOS ANTICATABÓLICOS que antagonizan los efectos catabólicos de la IL-1β (Dakin et al., 2012)
- Los AINES inhiben la producción de prostaglandinas y son agentes farmacológicos ampliamente usados para el tratamiento de las enfermedades inflamatorias y para aliviar el dolor asociado con la tendinopatía.
- A pesar de la evidencia que sugiere que la inflamación tiene efectos perjudiciales para la curación del tejido conectivo, el bloqueo no específico de la respuesta inflamatoria no parece ser beneficioso para el proceso de curación (Magra and Maffulli, 2006; Marsolais et al., 2003).

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Prostaglandinas mediadoras en la tendinopatía:

Prostaglandin E2 affects proliferation and collagen synthesis by human patellar tendon fibroblasts.

Cilli F[1], Khan M, Fu F, Wang JH.

Author information

LA PROSTAGLANDINA E2 AFECTA A LA PROLIFERACIÓN Y PRODUCCIÓN DE COLÁGENO DE LOS FIBROPLASTOS DE MODO DOSIS DEPENDIENTE.

Abstract

OBJECTIVE: To determine the effect of prostaglandin E2 on proliferation and collagen synthesis by human patellar tendon fibroblasts.

DESIGN AND SETTING: Controlled laboratory study.

METHODS: Human patellar tendon fibroblasts were treated with different concentrations (1, 10, 100 ng/mL) of prostaglandin E2 in cultures. Fibroblasts without prostaglandin E2 treatment were used as the control group. The fibroblast proliferation and collagen synthesis were measured using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazolium bromide assay and Sircol collagen assay, respectively.

MAIN OUTCOME MEASURED: Changes in proliferation and collagen production of human patellar tendon fibroblasts.

RESULTS: : At 1 ng/mL of prostaglandin E2, there was no significant effect on fibroblast proliferation compared with the control group. At concentrations of 10 ng/mL and 100 ng/mL prostaglandin E2, however, fibroblast proliferation significantly decreased, by 7.3% ($P = 0.002$) and 10.8% ($P < 0.0001$), respectively, compared with the control group. At 1 ng/mL of prostaglandin E2, collagen production of the tendon fibroblasts was unaffected. However, at both 10 ng/mL and 100 ng/mL prostaglandin E2, collagen production was significantly decreased, by 45.2% ($P < 0.0001$) and 45.7% ($P < 0.0001$), respectively, compared with the control group. The levels of collagen production between these 2 dosages did not differ significantly.

CONCLUSIONS: Prostaglandin E2 affects the proliferation of and collagen production by human patellar tendon fibroblasts in a dosage-dependent manner.

CLINICAL RELEVANCE: Based on these in vitro findings, we speculate that production of prostaglandin E2 in tendons might play some role in the acellularity and matrix disorganization seen in exercise-induced tendinopathy

LA PROSTAGLANDINA E2 AFECTA A LA PROLIFERACIÓN Y PRODUCCIÓN DE COLÁGENO DE LOS FIBROPLASTOS DE MODO DOSIS DEPENDIENTE.

- Además la PGE_2 generada durante la inflamación activa mediadores lipídicos que estimulan la curación como las lipoxinas y resolvinas, que son esenciales en la restauración de la homeostais tisular tras la inflamación o herida (Levy et al., 2001; Serhan et al., 2000a, 2004).

- ***PROSTAGLANDINA E2 (PGE2)***

PGE2 VERSUS AINES:

- Por tanto, el bloqueo de las prostaglandinas mediante AINES es probable que tenga un impacto negativo en los mecanismos endógenos del cuerpo para regular la inflamación (Gilroy et al., 1999; Serhan, 2005).
- Las prostaglandinas tienen propiedades inmunomoduladores: por ejemplo PGE_2 regula a la baja la respuesta inflamatoria inhibiendo la producción de IL-12 proinflamatoria y ,a su vez , estimula la producción de la citokina IL-10 antiinflamatoria (van der Pouw Kraan et al., 1995).

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Prostaglandinas mediadoras en la tendinopatía:

Curr Sports Med Rep. 2011 Sep-Oct;10(5):255-70. doi: 10.1249/JSR.0b013e31822d4016.

Treatment options for patellar tendinopathy: critical review.

Gaida JE[1], Cook J.

Author information

Abstract

Patellar tendinopathy is a painful knee injury due to overuse common among jumping athletes. Because rest from sport is neither a feasible nor an effective treatment for patellar tendinopathy in elite athletes, active treatment options are needed. Treatment may be conservative, injection-based, or surgical. This review synthesizes findings from 32 studies of varying quality published between 2001 and 2011. Painful eccentric squats using a 25°-decline board is supported as a first-line treatment. Extracorporeal shock wave therapy is no more effective than placebo. Sclerosing injections seem to be effective, but the evidence is not definitive. Shaving of abnormal tissue via arthroscopic surgery with real-time ultrasound guidance is superior to sclerosing injections. Steroid injections are inferior to exercise interventions and are not recommended. Injections of autologous blood, platelet-rich plasma, and hyperosmolar dextrose are unproven and experimental. Clinicians need to have a comprehensive knowledge of the evidence in the literature, as well as training and experience, when treating patellar tendinopathy.

PMID: 23531972 DOI: 10.1249/JSR.0b013e31822d4016

Clin Sports Med. 2015 Apr;34(2):383-74. doi: 10.1016/j.csm.2014.12.006. Epub 2015 Jan 24.

Treating tendinopathy: perspective on anti-inflammatory intervention and therapeutic exercise.

Joseph MF[1], Denegar CR[2].

Author information

Abstract

Tendinopathy is a common and complex disorder. Once viewed as an inflammatory condition labeled tendinitis, it is now viewed along a continuum that can lead to tissue necrosis and risk of tendon rupture. Anti-inflammatory medications can alter symptoms but may also promote tissue degeneration. Loading of the tendon through exercise, especially exercise involving eccentric muscle contraction, has been shown to promote symptom resolution and functional recovery in many patients. This article reviews the pathoetiology of tendinopathy and the role anti-inflammatory interventions and therapeutic exercise in treatment of active patients.

KEYWORDS: Anti-inflammatory; Eccentric loading; Tendinopathy; Therapeutic exercise

PMID: 25818719 DOI: 10.1016/j.csm.2014.12.006

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Prostaglandinas mediadoras en la tendinopatía:

Unfallchirurg. 2017 Mar;120(3):199-204. doi: 10.1007/s00113-017-0310-9.

[Evidence-based therapy for tendinopathy of the knee joint : Which forms of therapy are scientifically proven?]

[Article in German]

Horstmann H[1], Clausen JD[2], Krettek C[2], Weber-Spickschen TS[3,4].

Author information

Abstract

Tendinopathy in the region of the knee joint is a common pathological disorder. People active in sports, in particular, have a high probability of suffering from tendinopathy. Despite its high clinical relevance, the level of evidence of therapy options for tendinopathy in the knee region differs greatly. This review gives an overview of current evidence levels for therapy options in tendinopathy of the quadriceps, patellar and pes anserinus insertion tendons as well as of the distal iliotibial tract tendon. The treatment with platelet-rich plasma showed a significantly better outcome when used correctly and treatment with shock waves, operative treatment and sclerotherapy have also shown positive effects. Treatment with corticosteroid injections and with oral non-steroidal anti-inflammatory drugs (NSAID) showed positive short-term effects (follow-up ±4 weeks). No reasonable data are available for the treatment of tendinopathy in the knee region by acupuncture, fascial therapy or cryotherapy. The use of kinesio taping showed no significant relief from complaints compared with standard conservative treatment. The use of multimodal therapy without evidence is, therefore, particularly common in elite athletes.

KEYWORDS: Jumpers knee; Level of evidence; Multimodal therapy approaches; Platelet rich plasma; Shock wave therapy

PMID: 28138766 DOI: 10.1007/s00113-017-0310-9

LA INFLALAMCIÓN ES NECESARIA PARA LA CURACIÓN:

- Identificación de mediadores pro resolución como las lipoxinas A_4 (LXA_4) (Serhan et al., 1984) que actúan sobre el receptor de resolvinas FPR2/ALX (Ye et al., 2009) y más recientemente las resolvinas and maresinas (Serhan et al., 2002, 2009). FPR2/ALX se expresa por monocitos y macrófagos (Yang et al., 2001) y es fundamental para controlar la duración y magnitud de la respuesta inflamatoria y provee señales endógenas de stop para la inflamación (Serhan et al., 2000b).

PROBLEMAS DE ESTEROIDES Y AINES:

- El uso de esteroides y AINES para aliviar el dolor asociado a la lesión tendinosa es controvertido.
- Mientras que el uso a corto plazo de estas drogas a nivel sistémico no es probable que se asocie con efectos deletéreos , el uso prolongado de aines o la inyección intratendinosa de esteroides se asocia con efectos adversos en las propiedades mecánicas del tendón y propensión a la ruptura (Virchenko et al., 2004) (Shrier et al., 1996).

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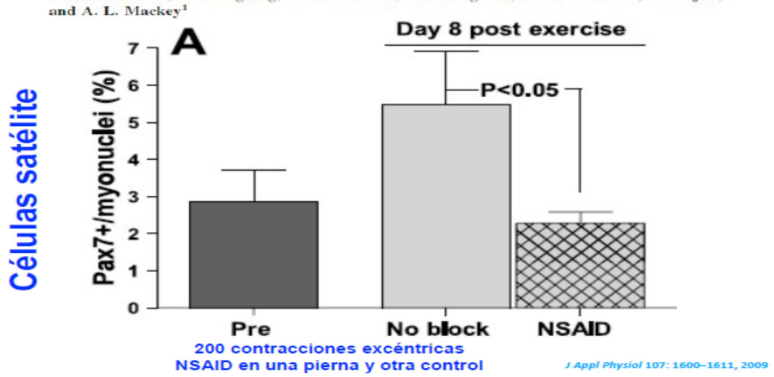
Prostaglandinas mediadoras en la tendinopatía:

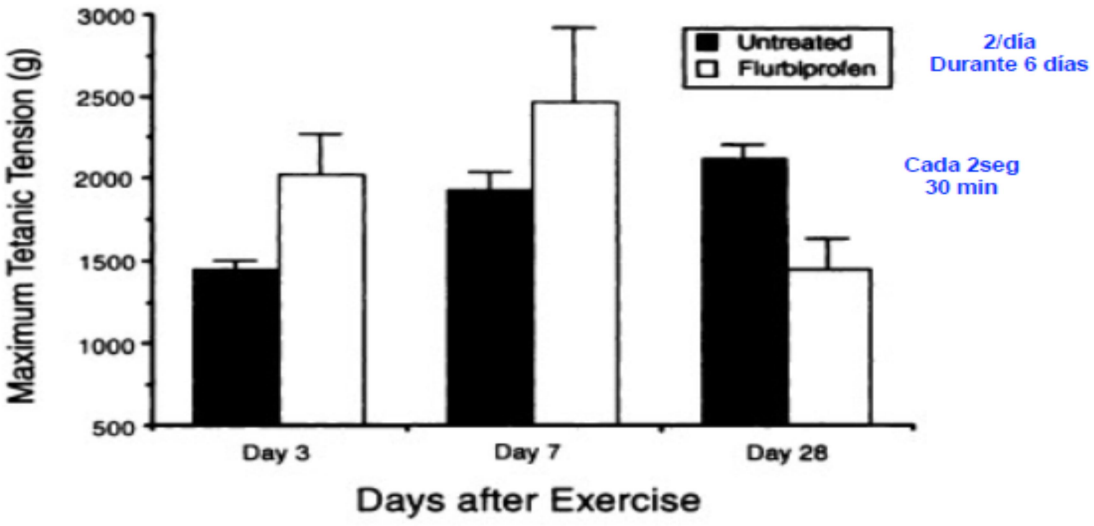
Local NSAID infusion inhibits satellite cell proliferation in human skeletal muscle after eccentric exercise

U. R. Mikkelsen,[1] H. Langberg,[1] I. C. Helmark,[1] D. Skovgaard,[1] L. L. Andersen,[2] M. Kjær,[1] and A. L. Mackey[1]



Anti-inflammatory medication after muscle injury. A treatment resulting in short-term improvement but subsequent loss of muscle function

DK Mishra, J Friden, MC Schmitz and RL Lieber
J Bone Joint Surg Am. 1995;77:1510-1519.



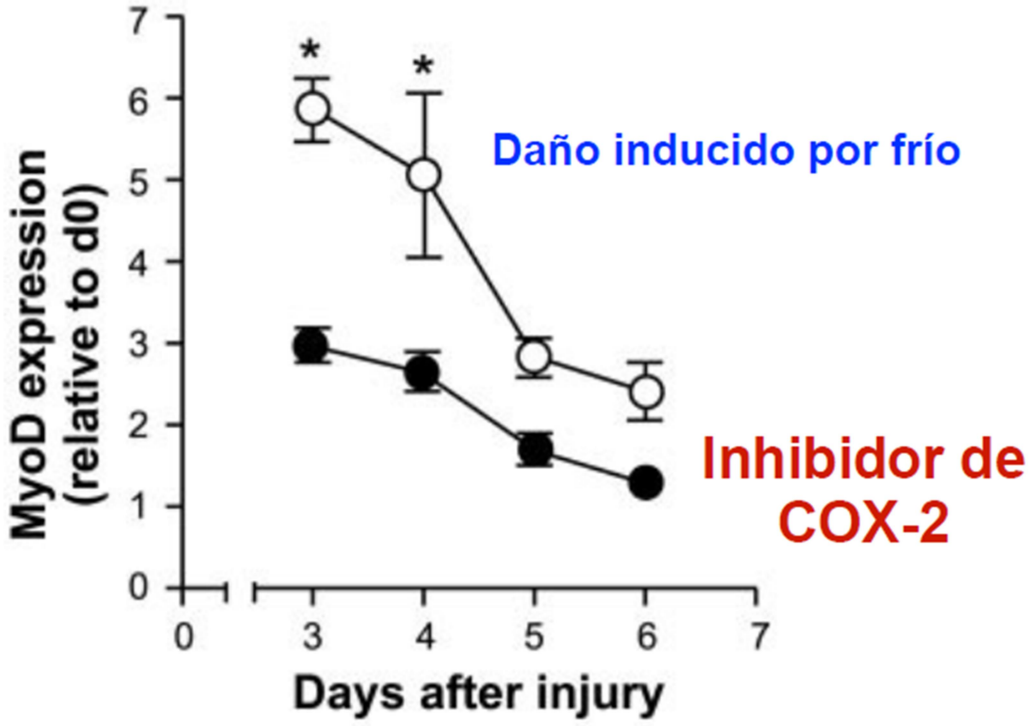
Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

The COX-2 pathway is essential during early stages of skeletal muscle regeneration

Am J Physiol Cell Physiol 287: C475–C483, 2004

Brenda A. Bondesen,[1,2] Stephen T. Mills,[1] Kristy M. Kegley,[1] and Grace K. Pavlath[1]



La proteína 1 de diferenciación miogénica es una proteína importante en la regulación de la diferenciación muscular, es un Factor regulador miogénico o factor de transcripción. Es uno de los marcadores más tempranos de la miogénesis y es expresada en las células satélite activadas.

LA CRIOTERAPIA ANTES DE UNA ACTIVIDAD DEPORTIVA TIENE MÁS RIESGO DE LESIÓN
PONER HIELO A TODAS HORAS TAMPOCO ES BUENO.

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Prostaglandinas mediadoras en la tendinopatía:

Cell Physiol Biochem. 2018;50(6):2046-2059. doi: 10.1159/000495050. Epub 2018 Nov 9.

High Concentration of Aspirin Induces Apoptosis in Rat Tendon Stem Cells via Inhibition of the Wnt/β-Catenin Pathway.

Wang Y[1], Tang H[1], He G[1], Shi Y[1], Kang X[1], Lyu J[1], Zhou M[1], Zhu M[1], Zhang J[2], Tang K[3].

Author information

Abstract

BACKGROUND/AIMS: Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used in clinical practice to relieve fever and pain. Aspirin, as a representative NSAID, has been widely used in the treatment of tendinopathy. Some reports have demonstrated that aspirin can induce apoptosis in cancer cells. However, evidence regarding aspirin treatment for tendinopathy, especially the effect of this treatment on tendon stem cells (TSCs), is lacking. Understanding the effect of aspirin on tendinopathy may provide a basis for the rational use of NSAIDs in clinical practice. The aim of our study was to determine whether aspirin induces apoptosis in rat TSCs via the Wnt/β-catenin pathway.

METHODS: First, we used flow cytometry and fluorescence to detect TSC apoptosis. Protein expression of the apoptosis-related caspase-3 pathway was investigated via western blot analysis. Next, we used western blotting to determine the effect of aspirin on the Wnt/β-catenin pathway. We used immunostaining to detect the levels of Bcl2, cleaved caspase-3, and P-β-catenin in the Achilles tendon. Finally, we used flow cytometry, fluorescence, and western blotting to investigate the aspirin-induced apoptosis of TSCs via the Wnt/β-catenin pathway.

RESULTS: Aspirin induced morphological apoptosis in rat TSCs via the mitochondrial/caspase-3 pathway and induced cellular apoptosis in the Achilles tendon. Apoptosis was partly reversed after adding the Wnt signaling activator Wnt3a and lithium chloride (LiCl, a GSK-3β inhibitor). Aspirin administration led to a dose-dependent increase in COX-2 expression. Apoptosis was promoted after adding the COX-2 inhibitor NS398.

CONCLUSION: The Wnt/β-catenin pathway plays a vital role in aspirin-induced apoptosis by regulating mitochondrial/caspase-3 function. Elevating COX-2 levels may protect cells against apoptosis. More importantly, the results remind us to consider the apoptotic effect of aspirin on TSCs and tendon cells when aspirin is administered to treat tendinopathy. The relationship between the positive and negative effects of aspirin remains a subject for future study.

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Prostaglandinas mediadoras en la tendinopatía:

ROLE OF CYCLOOXYGENASE-1 AND -2 IN SATELLITE CELL PROLIFERATION, DIFFERENTIATION, AND FUSION

CHRISTOPHER L. MENDIAS, MS,[1,2] RYUICHI TATSUMI, PhD,[3] and RONALD E. ALLEN, PhD[2]

- Células en cultivo 96 h
- Inhibición selectiva de COX-2 disminuye proliferación de células satélite
- Inhibición de COX-1 y COX-2 reduce diferenciación y fusión de células satélite

Muscle Nerve 30: 497–500, 2004

Cell Prolif. 2009 Dec;42(6):721-30. doi: 10.1111/j.1365-2184.2009.00639.x. Epub 2009 Aug 25.

Aspirin induces apoptosis in mesenchymal stem cells requiring Wnt/beta-catenin pathway.

Deng L[1], Hu S, Baydoun AR, Chen J, Chen X, Cong X.

Author information

Abstract

BACKGROUND AND OBJECTIVES: Mesenchymal stem cells (MSC) are multipotent progenitor cells that are have found use in regenerative medicine. We have previously observed that aspirin, a widely used anti-inflammatory drug, inhibits MSC proliferation. Here we have aimed to elucidate whether aspirin induces MSC apoptosis and whether this is modulated through the Wnt/beta-catenin pathway.

MATERIALS AND METHODS: Apoptosis of MSCs was assessed using Hoechst 33342 dye and an Annexin V-FITC/PI Apoptosis Kit. Expression of protein and protein phosphorylation were investigated using Western blot analysis. Caspase-3 activity was detected by applying a caspase-3/CPP32 Colorimetric Assay Kit.

RESULTS: In these MSCs, aspirin induced morphological changes characteristic of apoptosis, cytochrome c release from mitochondria, and caspase-3 activation. Stimulating the Wnt/beta-catenin pathway by both Wnt 3a and GSK-3beta inhibitors (LiCl and SB 216763), blocked aspirin-induced apoptosis and protected mitochondrial function, as demonstrated by decreased cytochrome c release and caspase-3 activity. Aspirin initially caused a time-dependent decrease in COX-2 expression but subsequently, and unexpectedly, elevated the latter. Stimulation of COX-2 expression by aspirin was further enhanced following stimulation of the Wnt/beta-catenin pathway. Application of the COX-2 inhibitor NS-398 suppressed elevated COX-2 expression and promoted aspirin-induced apoptosis.

CONCLUSION: These results demonstrate that the Wnt/beta-catenin pathway is a key modulator of aspirin-induced apoptosis in MSCs by regulation of mitochrondrial/caspase-3 function. More importantly, our findings suggest that aspirin may influence MSC survival under certain conditions; therefore, it should be used with caution when considering regenerative MSC transplantation in patients with concomitant chronic inflammatory diseases such as arthritis.

PMID: 19706045 DOI: 10.1111/j.1365-2184.2009.00639.x

Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

¿ Qué ofrece la Osteopatía para la reparación tendinosa?

Microsc Res Tech. 2007 Apr;70(4):310-24.

Studies on the importance of sympathetic innervation, adrenergic receptors, and a possible local catecholamine production in the development of patellar tendinopathy (tendinosis) in man.

Danielson P[1], Alfredson H, Forsgren S.

Author information

Abstract

Changes in the patterns of production and in the effects of signal substances may be involved in the development of tendinosis, a chronic condition of pain in human tendons. There is no previous information concerning the patterns of sympathetic innervation in the human patellar tendon. In this study, biopsies of normal and tendinosis patellar tendons were investigated with immunohistochemical methods, including the use of antibodies against tyrosine hydroxylase (TH) and neuropeptide Y, and against alpha1-, alpha2A-, and beta1-adrenoreceptors. It was noticed that most of the sympathetic innervation was detected in the walls of the blood vessels entering the tendon through the paratendinous tissue, and that the tendon tissue proper of the normal and tendinosis tendons was very scarcely innervated. Immunoreactions for adrenergic receptors were noticed in nerve fascicles containing both sensory and sympathetic nerve fibers. High levels of these receptors were also detected in the blood vessel walls; alpha1-adrenoreceptor immunoreactions being clearly more pronounced in the tendinosis tendons than in the tendons of controls. Interestingly, immunoreactions for adrenergic receptors and TH were noted for the tendon cells (tenocytes), especially in tendinosis tendons. The findings give a morphological correlate for the occurrence of sympathetically mediated effects in the patellar tendon and autocrine/paracrine catecholamine mechanisms for the tenocytes, particularly, in tendinosis. The observation of adrenergic receptors on tenocytes is interesting, as stimulation of these receptors can lead to cell proliferation, degeneration, and apoptosis, events which are all known to occur in tendinosis. Furthermore, the results imply that a possible source of catecholamine production might be the tenocytes themselves.

PMID: 17206652 DOI: 10.1002/jemt.20413

Histol Histopathol. 2008 Feb;23(2):197-208. doi: 10.14670/HH-23.197.

Immunohistochemical and in situ hybridization observations favor a local catecholamine production in the human Achilles tendon.

Bjur D[1], Danielson P, Alfredson H, Forsgren S.

Author information

Abstract

Results of recent studies using immunohistochemistry show evidence of an occurrence of catecholamine production in the cells (tenocytes) of patellar tendons exhibiting tendinopathy (tendinosis). In the present study, antibodies against the catecholamine-synthesizing enzyme tyrosine hydroxylase (TH) and alpha1-adrenoreceptors were applied to sections of specimens of normal and tendinosis Achilles tendons. In situ hybridization using a probe detecting human TH mRNA was also utilized. It was found that sympathetic innervation was very scarce. On the other hand, there were distinct alpha1-adrenoreceptor immunoreactions in blood vessel walls. Interestingly, tenocytes, particularly from tendinosis samples in which the tenocytes showed an abnormal shape (not the typical slender appearance), displayed TH immunoreactions and reactions for TH mRNA. Of further interest was the finding of alpha1-adrenoreceptor immunoreactions in tenocytes. The observations show not only evidence of local catecholamine production at the protein level, which was the case in recent studies for the patellar tendon, but also at the mRNA level. The observations suggest that the tenocytes, especially those with disfigured appearances in tendinosis, can produce catecholamines and also that they can respond to sympathetic transmitters. This is of interest as adrenergic stimulation in other parts of the body is known to induce degenerative/apoptotic and proliferative events, features which are seen in Achilles tendinosis. These observations are completely new findings concerning the human Achilles tendon. It is likely that locally produced catecholamines and the occurrence of autocrine/paracrine effects of these substances are of great relevance during the process of tendinosis.

PMID: 17999376 DOI: 10.14670/HH-23.197

Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

Front Aging Neurosci. 2016 Aug 9;8:186. doi: 10.3389/fnagi.2016.00186. eCollection 2016.

Role of VEGF, Nitric Oxide, and Sympathetic Neurotransmitters in the Pathogenesis of Tendinopathy: A Review of the Current Evidences.

Vasta S[1], Di Martino A[1], Zampogna B[1], Torre G[1], Papalia R[1], Denaro V[1].

Author information

Abstract

Chronic tendinopathy is a painful common condition affecting athletes as well as the general population undergoing to tendon overuse. Although its huge prevalence, little is known about tendinopathy pathogenesis, and even cloudier is its treatment. Traditionally, tendinopathy has been defined as a lack of tendon ability to overcome stressing stimuli with appropriate adaptive changes. Histologic studies have demonstrated the absence of inflammatory infiltrates, as a consequence conventional antinflammatory drugs have shown little or no effectiveness in treating tendinopathies. New strategies should be therefore identified to address chronic tendon disorders. Angiofibroblastic changes have been highlighted as the main feature of tendinopathy, and vascular endothelial growth factor (VEGF) has been demonstrated as one of the key molecules involved in vascular hyperplasia. More recently, attention has been focused on new peptides such as Substance P, nitric oxide, and calcitonin gene-related peptide (CGRP). Those new findings support the idea of a nerve-mediated disregulation of tendon metabolism. Each of those molecules could be a target for new treatment options. This study aimed to systematically review the current available clinical and basic science in order to summarize the latest evidences on the pathophysiology and its effect on treatment of chronic tendinopathy, and to spread suggestions for future research on its treatment.

KEYWORDS: VEGF; neurotransmitter agents; nitric oxide (NO); nociceptive substance P (SP); tendinopathy; tendons

Comment in

Commentary: Role of VEGF, Nitric Oxide, and Sympathetic Neurotransmitters in the Pathogenesis of Tendinopathy: A Review of the Current Evidences. [Front Aging Neurosci. 2017]

PMID: 27555817 PMCID: PMC4977280 DOI: 10.3389/fnagi.2016.00186

FAVORECER LA REGENERACIÓN TENDINOSA:

Jewson et al, Alfredson et al y Beccati et al (2019)
Se han observado alteraciones neurovegetativas en la génesis de las alteraciones tendinosas.
El SNV está implicado en la degeneración tisular y tendinosa.

Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

LA OSTEOPATÍA ACTIVA EL PARASIMPÁTICO Y ESTO ES BUENO PARA LA REGENERACIÓN TENDINOSA

Effects of Upper and Lower Cervical Spinal Manipulative Therapy on Blood Pressure and Heart Rate Variability in Volunteers and Patients With Neck Pain: A Randomized Controlled, Cross-Over, Preliminary Study.

Win NN[1], Jorgensen AM[1], Chen YS[2], Haneline MT[3].

⊕ Author information

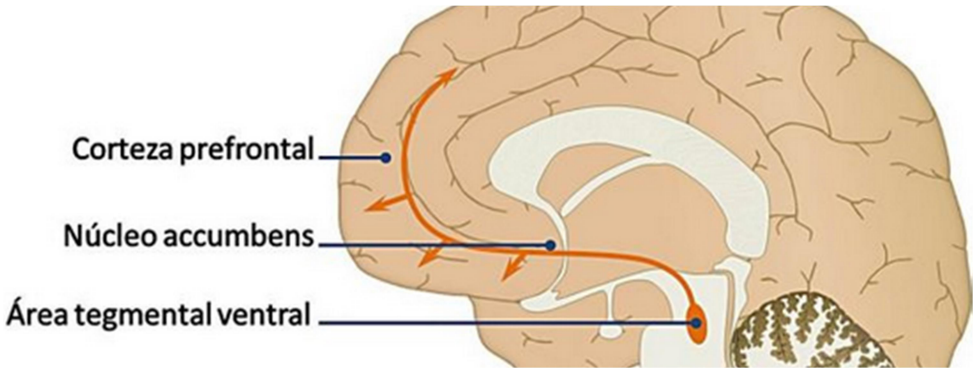
Abstract

OBJECTIVE: The aims of this study were to examine autonomic nervous system responses by using heart rate variability analysis (HRV), hemodynamic parameters and numeric pain scale (NPS) when either upper (C1 and C2) or lower (C6 and C7) cervical segments were manipulated in volunteers, and whether such response would be altered in acute mechanical neck pain patients after spinal manipulative therapy (SMT).

METHODS: A randomized controlled, cross-over, preliminary study was conducted on 10 asymptomatic normotensive volunteers and 10 normotensive patients complaining of acute neck pain. HRV, blood pressure (BP) and heart rate (HR), and NPS were recorded after upper cervical and lower cervical segments SMT in volunteer and patient groups.

RESULTS: The standard deviation of average normal to normal R-R intervals (SDNN) increased (83.54 ± 22 vs. 105.41 ± 20; P = .02) after upper cervical SMT. The normalized unit of high frequency (nuHF), which shows parasympathetic activity, was predominant (40.18 ± 9 vs. 46.08 ± 14) after upper cervical SMT (P = .03) with a significant decrease (109 ± 10 vs. 98 ± 5) in systolic BP (P = .002). Low frequency to high frequency (LF/HF) ratio, which shows predominance of sympathetic activity increased (1.05 ± 0.7 vs. 1.51 ± 0.5; P = .02) after lower cervical SMT in the healthy volunteers group. However, there was an increase in SDNN (70.48 ± 18 vs. 90.23 ± 20; P = .02 and 75.19 ± 16 vs 97.52 ± 22; P = .01), a decrease in LF/HF ratio (1.33 ± 0.3 vs. 0.81 ± 0.2; P = .001 and 1.22 ± 0.4 vs. 0.86 ± 0.3; P = .02), which was associated with decreased systolic BP (105 ± 10 vs. 95 ± 9; P = .01 and 102 ± 9 vs. 91 ± 10; P = .02) and NPS scores (3 ± 1 vs. 0; P = .01 and 3 ± 1 vs. 1 ± 1; P = .03) following both upper and lower cervical SMT in the patient's group. The baseline HR was 67 ± 9 vs 64 ± 5 (upper cervical) and 65 ± 7 vs 69 ± 11 (lower cervical) in both the healthy volunteer' and patient' groups.

CONCLUSION: Upper cervical SMT enhances dominance of parasympathetic and lower cervical SMT enhances dominance of sympathetic activity in this young volunteer group. However, dominance of parasympathetic activity was found in patients with neck pain that received both upper and lower cervical SMT.



ACTIVAR ÁREAS CORTICALES RELACCIONADAS CON LA MOTIVACIÓN Y LA RECOMPENSA.

Inami A, Ogura T, Watanuki S, Masud MM, Shibuya K, Miyake M, et al. Glucose Metabolic Changes in the Brain and Muscles of Patients with Nonspecific Neck Pain Treated by Spinal Manipulation Therapy: A [(18)F]FDG PET Study. Evid Based Complement Alternat Med. 2017;2017:4345703.

Escuela de Osteopatía de Madrid Internacional

Prostaglandinas mediadoras en la tendinopatía:

El núcleo accumbens y el tubérculo olfatorio constituyen la parte ventral del cuerpo estriado, parte de los ganglios basales. Es un núcleo importante del placer, la risa, recompensa, miedo, agresión y efecto placebo, implicado en el circuito de premio recompensa. El área tegmental ventral implicada en el circuito de refuerzo, placer y motivación.
POR TANTO EL EFECTO PLACEBO TB ES MUY BUENO PORQUE ENTRA EN CIRCUITOS DE RECOMPENSA Y MOTIVACIÓN

Evid Based Complement Alternat Med. 2017;2017:4345703. doi: 10.1155/2017/4345703. Epub 2017 Jan 12.

Glucose Metabolic Changes in the Brain and Muscles of Patients with Nonspecific Neck Pain Treated by Spinal Manipulation Therapy: A [^{18}F]FDG PET Study.

Inami A[1], Ogura T[2], Watanuki S[1], Masud MM[3], Shibuya K[4], Miyake M[1], Matsuda R[1], Hiraoka K[1], Itoh M[4], Fuhr AW[5], Yanai K[6], Tashiro M[1].

Author information

Abstract

Objective. The aim of this study was to investigate changes in brain and muscle glucose metabolism that are not yet known, using positron emission tomography with [^{18}F]fluorodeoxyglucose ([^{18}F]FDG PET). *Methods*. Twenty-one male volunteers were recruited for the present study. [^{18}F]FDG PET scanning was performed twice on each subject: once after the spinal manipulation therapy (SMT) intervention (treatment condition) and once after resting (control condition). We performed the SMT intervention using an adjustment device. Glucose metabolism of the brain and skeletal muscles was measured and compared between the two conditions. In addition, we measured salivary amylase level as an index of autonomic nervous system (ANS) activity, as well as muscle tension and subjective pain intensity in each subject. *Results*. Changes in brain activity after SMT included activation of the dorsal anterior cingulate cortex, cerebellar vermis, and somatosensory association cortex and deactivation of the prefrontal cortex and temporal sites. Glucose uptake in skeletal muscles showed a trend toward decreased metabolism after SMT, although the difference was not significant. Other measurements indicated relaxation of cervical muscle tension, decrease in salivary amylase level (suppression of sympathetic nerve activity), and pain relief after SMT. *Conclusion*. Brain processing after SMT may lead to physiological relaxation via a decrease in sympathetic nerve activity.

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CONCLUSIÓN:

Hasta ahora no se ha demostrado que ningún medicamento o tratamiento inyectable altere las propiedades del tejido;
Solo la carga CONTROLADA BASADA EN LA EVIDENCIA de LAS ESTRUCTURAS CONECTIVAS ASOCIADA A LA MODULACIÓN DE LA RESPUESTA NEUROVEGETATIVA puede estimular su remodelación.

The challenge of managing tendiopathy in competing athletes. Br J Sports Med. 2014;48:506-9.