Arch Bronconeumol. 2009;**45(10)**:529

ARCHIVOS DE BRONCONEUMOLOGIA

www.archbronconeumol.org

Erratum

Erratum of "Implications of cyclooxygenese-2 regulation of myofibroblast formation in pulmonary fibrogenesis"

E. Cano-Jiménez,[a,d] J. Pereda,[a,d] D. Royo,[a,d] M. Molina-Molina,[c,d] M. Gabasa,[a] J. Roca,[a,d] L. Pujols,[a,d] J. Ramírez,[b,d] C. Picado,[a,d] and A. Xaubet[a,d,*]

[a] *Clinical Hospital: Pneumology Department. IDIBAPS, Barcelona, Spain*
[b] *Clinical Hospital: Anatomical Pathology Department. IDIBAPS*
[c] *Hospital of Bellvitge: Pneumology Department. IDIBELL*
[d] *CibeRes*

In the speech: "Implications of cyclooxygenese-2 regulation of myofibroblast formation in lung fibrogenesis." by E. Cano-Jiménez, J. Pereda, D. Royo, M. Molina-Molina, M. Gabasa, J. Roca, L. Pujols, J. Ramírez, C. Picado and A. Xaubet, certain errors have been detected: the list of authors and the summary are not correct, therefore, we shall reprint the information as it should have appeared.

Abstract

Introduction: A decrease has been described in cyclooxygenese-2 (COX-2) and prostaglandine-E2 (PGE-2) expression in lung fibrogenesis. PGE-2 is an antifibrotic mediator. Myofibroblasts play an important role in fibrogenesis; they could form through fibroblasts (fibroblast-myofibroblast transition) and epithelial cells (epithelial-mesenchymal transition). The aim is to study COX-2 expression and PGE2 synthesis in induction of myofibroblast formation.
Methods: Normal fibroblasts were obtained by lung biopsy performed on subjects with pneumothorax (n = 5) and fibroblasts from patients with idiopathic pulmonary fibrosis (IPF) (n = 5). Immortal A549 epithelial cells were used in the epithelial function study. Immunohistochemical staining (IHC) and a Western blot were carried out in order to determine COX-2 and α-SMA (smooth muscle actin, a myofibroblast marker). PGE2 synthesis was studied using ELISA. Cell proliferation was evaluated through nuclear incorporation of a nucleoside analogue.
Results: The IPF fibroblasts showed greater levels of β-SMA compared with fibroblast controls. Both showed an undectable COX-2 expression. Following stimulation with interleukin 1β (IL-1β), the fibroblast controls showed higher levels of COX-2 than fibrotic fibroblasts. The myofibroblasts detected with IHC showed no COX-2. The fibroblasts treated with TGF-β1 showed α-SMA, in shape, dose and time, depending on fibre-optic fibroblasts and fibroblast controls. A549 cells treated with TGF-β1 changed their phenotype, showing stress fibres related with the forming of myofibroblasts (α-SMA+). The myofibroblasts obtained through treating fibroblasts and A549 with TGF-β1 showed a decrease in levels of COX-2 and PGE-2 following IL-1β stimulation. There were no variations in cell proliferation.
Conclusions: Myofibroblasts are characterized by an alteration in COX-2 expression regulation and PGE-2 synthesis, in those transformed through fibroblasts as well as through epithelial cells.
Financed by SEPAR-Fundación Respira FIS PI060064.

* Corresponding author.
E-mail address: axaubet@clinic.ub.es (A.Xaubet).