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Dexamethasone Alters the Expression of Scar-Related Genes in Biliary Fibroblasts Induced by TNF-α

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***SUMMARY*. The purpose of this work is to investigate the effects of dexamethasone (Dex) on rabbit bile duct-derived fibroblasts treated with tumor necrosis factor (TNF)-α. Rabbit bile duct-derived fibroblasts were cultured, characterized, and divided into four groups: control, 30 ng/mL TNF-α, 30 ng/mL TNF-α + 0.02 mg/mL Dex, and 30 ng/mL TNF-α + 0.1 mg/mL Dex. After treatment 48 h, cell proliferation was assessed by cell counting kit-8. Relative mRNA expression levels of transforming growth factor (TGF)-β1, α-smooth muscle actin (α-SMA) and neuronal regeneration related protein (P311) were assessed by quantitative PCR. Relative protein expression levels of TGF-β1 and α-SMA were measured by western blotting. TNF-α significantly up-regulated mRNA/protein expression levels of TGF-β1, α-SMA, P311 (all P < 0.01), and significantly enhanced proliferation of bile duct fibroblasts (P < 0.01). However, Dex inhibited TNF-α-mediated cells proliferation in a dose-dependent manner (all P < 0.01). Dex inhibited TNF-α-mediated up-regulation mRNA/protein expression levels of TGF-β1, α-SMA, and P311 (all P < 0.01). TNF-α up-regulated of scar-related genes and induced proliferation of bile duct fibroblasts. Dex was a potential inhibitor of scar-related genes expression in bile duct fibroblasts and may be useful in the treatment of benign biliary stricture.**

RESUMEN. El objetivo de este trabajo es investigar los efectos de la dexametasona (Dex) sobre fibroblastos de conductos biliares de conejo tratados con factor de necrosis tumoral (TNF)-α. Los fibroblastos se cultivaron, caracterizaron y dividieron en cuatro grupos: control, TNF-α de 30 ng/mL, TNF-α de 30 ng/mL + 0,02 mg/mL de Dex y TNF-α de 30 ng/mL + 0,1 mg/mL Dex. Después de 48 h, la proliferación celular se evaluó mediante el recuento de células kit-8. Los niveles relativos de expresión del ARNm del factor de crecimiento transformante (TGF)-β1, α-actina del músculo liso (α-SMA) y proteína relacionada con la regeneración neuronal (P311) se evaluaron cuantitativamente mediante PCR. Los niveles relativos de expresión proteica de TGF-β1 y α-SMA se midieron mediante Western blot. TNF-α aumentó significativamente los niveles de expresión de RNAm/proteína de TGF-β1, α-SMA, P311 (todos P <0,01) y aumentó significativamente la proliferación de fibroblastos de vía biliar (P < 0,01). Sin embargo, Dex inhibió la proliferación de células mediadas por TNF-α de una manera dependiente de la dosis (todas P < 0,01). Dex inhibió los niveles de expresión de ARNm/proteína mediados por TNF-α de TGF-β1, α-SMA y P311 (Todos P < 0,01). TNF-α sobrerreguló los genes relacionados con la cicatrización y la proliferación inducida de los fibroblastos del conducto biliar. Dex fue un potencial inhibidor de la expresión de genes relacionados con cicatrización en fibroblastos del conducto biliar y puede ser útil en el tratamiento de la estenosis biliar benigna.

KEY WORDS: bile duct fibroblasts; scar-related genes; Tumor necrosis factor-α; benign biliary stricture; dexamethasone.

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