[1]王权,戚华兵,王晓凤,等.阻断TGF-β/TGF-βRⅠ通路对ATDC5细胞增殖、分化及细胞外基质的影响[J].第三军医大学学报,2013,35(10):917-921.

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阻断TGF-β/TGF-βRⅠ通路对ATDC5细胞增殖、分化影响(PDF)

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Title: Effect of blocking TGF-β/TGF-βRI signaling pathway on proliferation, differentiation and extracellular matrix of ATDC5 cells

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关键词: 转化生长因子βⅠ型受体; SB-505124; ATDC5; 细胞增殖; 细胞外基质

Keywords: transforming growth factor beta type Ⅰ receptor; SB-505124; ATDC5; cell proliferation; extracellular matrix

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摘要: 目的 研究转化生长因子βⅠ型受体（transforming growth factor beta type Ⅰ receptor, TGF-βRⅠ)阻断剂SB-505124对软骨细胞增殖、分化及细胞外基质的影响与机制。 方法 利用Western blot检测不同浓度的SB-505124处理后，前软骨细胞系ATDC5中p-Smad2/3的变化。MTT法检测SB-505124处理对ATDC5细胞生长、增殖的影响及时间、浓度依赖性效应。Western blot检测c-Myc蛋白水平的变化。Real-time PCR检测SB-505124处理后软骨分化、细胞外基质合成及降解相关分子Collagen Ⅱ、Aggrecan及Adamts5表达与变化，并利用阿尔新蓝染色法检测SB-505124处理对ATDC5细胞中蛋白聚糖合成的影响。 结果 Western blot检测结果示：SB-505124处理ATDC5细胞明显抑制TGF-β1激活的p-Smad2/3，并呈浓度依赖性；MTT检测结果示：SB-505124浓度和时间依赖性的抑制ATDC5细胞增殖；Real-time PCR检测结果示：SB-505124处理明显抑制ATDC5细胞中Collagen Ⅱ、Aggrecan的表达，同时上调了Adamts5的表达；阿尔新蓝染色结果提示SB-505124处理明显抑制ATDC5细胞中蛋白聚糖的合成。 结论 利用SB-505124阻断内源性TGF-β信号，可抑制软骨细胞增殖、分化

及细胞外基质的合成，同时促进细胞外基质的降解。

Abstract: Objective To investigate the effect of transforming growth factor beta type Ⅰ receptor（TGF-βRⅠ） inhibitor SB-505124 on the proliferation, differentiation and extracellular matrix of chondrocytes. Methods The expression of p-Smad2/3 in ATDC5 cells that were treated with different concentrations of SB-505124 was analyzed by Western blotting. The effect of SB-505124 on proliferation of ATDC5 cells was detected by MTT assay. The effect of SB-505124 on the expression of c-Myc protein was analyzed by Western blotting. The effect of SB-505124 on the markers of cell differentiation and extracellular matrix, including collagen Ⅱ, aggrecan and ADAMTS5 were analyzed by real-time PCR. The effect of SB-505124 on expression of sulfated proteoglycans was measured by Alcian blue staining. Results Western blotting results showed that the treatment with SB-505124 induced a decline of phosphorylated Smad2/3 (p-Smad2/3), including carboxyl termini and linker region stimulated by TGF-β1 in ATDC5 cells. MTT assay results demonstrated that SB-505124 significantly inhibited ATDC5 cell proliferation in a dose-and time-dependent manner, and the expression of c-Myc protein was also inhibited. Real-time PCR showed that collagen Ⅱ and aggrecan were inhibited by SB-505124, while ADAMTS5 was elevated by SB-505124. Alcian blue staining showed that the staining intensity was reduced in SB-505124 treated cells. Conclusion SB-505124 can block endogenous TGF-β signaling pathway to inhibit cell proliferation, cell differentiation and synthesis of extracellular matrix, and to promote the degradation of extracellular matrix.

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