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白介素22活化STAT3信号通路促进结直肠癌细胞的增殖及其可能机制 点此下载全文

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摘要：

目的： 探究白介素22（interleukin-22，IL-22）对结直肠癌细胞增殖的影响，并阐明其信号通路机制。 方法 ： Real-ti
0和SW620中IL-22受体1（IL-22 receptor 1， IL-22R1 ）mRNA的表达；采用不同质量浓度IL-22(0、1、5、10 ng/ml)
形成实验检测IL-22对SW480和SW620细胞增殖的影响；IL-22处理SW480和SW620细胞不同时间后，采用Western blotting
K、P38蛋白磷酸化的情况；观察特异性STAT3磷酸化抑制剂LLL12处理是否影响IL-22诱导的结直肠癌细胞的促增殖效应。 结果：
癌SW480、SW620细胞中均有表达。IL-22剂量依赖性地促进SW480和SW620细胞增殖，10 ng/ml IL-22作用后，SW480与
\[（5.18±0.212） vs （2.64±0.27），（8.14±0.61） vs （6.08±0.096）；均 P <0 01\]；且IL-22可提
能力，细胞克隆数明显多于空白对照组\[（1 680.67±124.05） vs （730±64.29），(2 668± 116.37） vs （
0.01\]。Western blotting证实IL-22能显著活化STAT3通路，而对AKT、ERK、JNK、P-38通路影响不明显；STAT3抑制剂LLL
620细胞的促增殖效应明显减弱。 结论 ： IL-22通过活化STAT3信号通路促进结直肠癌细胞SW480和SW620的增殖。

关键词：结直肠癌 白介素22 STAT3通路 增殖

Interleukin-22 promotes proliferation of colorectal cancer cells via STAT3 signaling activation and mechanism Download Fulltext

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Abstract:

Objective:To explore the effect of IL-22 on the proliferation of colorectal cancer cells and to illustrate its u
mechanism. Methods: The relative expression levels of IL-22 receptor 1 (IL-22R1) mRNA in colorectal cancer
were detected by real-time PCR. The different mass concentrations of IL-22 (0, 1, 5, 10 ng/ml) were used to tre
The effect of IL-22 on the proliferation of SW480 and SW620 cells was analyzed by MTT assay and colony form
SW620 cells were stimulated with IL-22 for various time points and were subjected to Western blotting for the
phosphorylations, including STAT3, AKT, ERK, JNK and P38. Whether the specific inhibitor LLL12 blocking phosph
promote the proliferation of colorectal cancer cells induced by IL-22 was observed. Results: IL-22R1 mRNA w
cancer cells (SW480, SW620). IL-22 can promote the proliferation of SW480 and SW620 cells in a dose-depend
treatment with IL-22 (10 ng/ml), the cell proliferation folds were significantly increased (SW480: \[5.18±0.2
SW620: \[8 14±0.61\] vs \[6.08±0.096\], P <0.01). Moreover, IL-22 enanced the colony formation abil
cells. The colony numbers of IL-22 treating group were significantly higher compared with the control group
\[730± 64.29\], \[2 668±116.37\] vs \[1 294±171.61\], P <0.01). Western blotting demonstrated th
activated after the treatment of IL-22. However, there was no significant alteration in AKT, ERK, JNK, P38 pathv
of IL-22 can be attenuated after the treatment of STAT3 phosphorylation inhibitor LLL12. Conclusion: IL-22 ma
colon cancer SW480 and SW620 cells via STAT3 signaling pathway.

Keywords:colorectal cancer interleukin-22（IL-22） STAT3 pathway proliferation

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