[1]陈书梅,杨淑敏,张文龙,等.TNF-α促进HepG2肝细胞脂质积聚及其机制的初步研究[J].第三军医大学学报,2013,35(11):1088-1092.

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TNF-α促进HepG2肝细胞脂质积聚及其机制

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Title: TNF-α promotes HepG2 hepatocytes lipid accumulation

作者: 陈书梅; 杨淑敏; 张文龙; 吕琼; 叶鹏; 高茹菲; 梅玫; 汪志红; 李启富

重庆医科大学附属第一医院内分泌科

Author(s): Chen Shumei; Yang Shumin; Zhang Wenlong; Lyu Qiong; Ye Peng; Gao Rufei; Mei Mei; Wang Zhihong; Li Qifu

Department of Endocrinology, First Affiliated Hospital, Chongqing Medical University, Chongqing, 400016, China

关键词: TNF-α; 软脂酸; SREBP-1; HepG2肝细胞; 脂质积聚

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摘要: 目的 探讨肿瘤坏死因子-α（tumor necrosis factor-α，TNF-α）是否能够促进肝细胞脂质积聚，并对其机制进行初步探讨。 方法 将HepG2肝细胞分为空白对照组、单纯TNF-α组（TNF-α 2 ng/mL或20 ng/mL）、软脂酸组（软脂酸0.08 mmol/L或0.2 mmol/L）及联合组（TNF-α 2 ng/mL联合软脂酸0.08 mmol/L、TNF-α 2 ng/mL联合软脂酸 0.2 mmol/L、TNF-α 20 ng/mL联合软脂酸 0.08 mmol/L、TNF-α 20 ng/mL联合软脂酸 0.2 mmol/L），处理24 h，应用化学酶促-比色法定量检测细胞内TG含量。进一步选取TNF-α 20 ng/mL和软脂酸0.08 mmol/L，通过油红O染色观察HepG2细胞内脂质积聚情况；实时荧光定量PCR和Western blot检测HepG2细胞SREBP-1、FAS、ACCα的表达水平。 结果 ①单纯TNF-α组TG含量[TNF-α 2 ng/mL组（0.344±0.093）μg/μg、TNF-α 20 ng/mL组（0.329±0.068）μg/μg]分别较空白对照组[（0.192±0.048）μg/μg]显著升高（$P<0.05$）；联合组

[TNF-α 2 ng/mL联合软脂酸 0.08 mmol/L组（0.451±0.096）μg/μg、TNF-α 2 ng/mL联合软脂酸 0.2 mmol/L组（0.821±0.257）μg/μg、TNF-α 20 ng/mL联合软脂酸 0.08 mmol/L组（1.032±0.286）μg/μg、TNF-α 20 ng/mL联合软脂酸 0.2 mmol/L组（2.134±1.049）μg/μg]分别较软脂酸组[软脂酸 0.08 mmol/L组（0.247±0.069）μg/μg、软脂酸 0.2 mmol/L组（0.341±0.031）μg/μg]显著升高（*P*<0.05）；②油红O染色进一步显示，TNF-α促进肝细胞内脂质积聚。③实时荧光定量PCR和Western blot检测结果显示单纯TNF-α组与空白对照组相比，HepG2细胞SREBP-1、FAS、ACCα的表达均增加（*P*<0.05）；联合组与软脂酸组相比，肝细胞内SREBP-1、FAS、ACCα的表达水平明显上调（*P*<0.05）。 结论 TNF-α促进HepG2肝细胞内脂质积聚，增加SREBP-1、FAS、ACCα的表达。

Abstract: Objective To determine the effect of tumor necrosis factor-α (TNF-α) on lipid accumulation in HepG2 cells and its underlying possible mechanism. Methods HepG2 cells were treated with TNF-α (2 or 20 ng/mL), palmitate (PA, 0.08 or 0.2 mmol/L), and TNF-α plus palmitate (combination of the 2 doses of 2 agents) for 24 h, respectively. The intracellular triglyceride (TG) was measured by enzymatic colorimetric method. Then TNF-α of 20 ng/mL and palmitate of 0.08 mmol/L was chosen for the further experiment. Lipid accumulation in the HepG2 cells was observed with Oil Red O staining. Real-time PCR and Western blot analysis were used to detect the expression of SREBP-1, FAS and ACCα at mRNA and protein levels. Results TG level was significantly higher in TNF-α treated cells (0.344±0.093 and 0.329±0.068 μg/μg for the doses of 2 and 20 ng/mL) than in control cells (0.192±0.048 μg/μg, P<0.05). And that of the combination treatment cells (TNF-α 2 ng/mL plus PA 0.08 mmol/L: 0.451±0.096, TNF-α 2 ng/mL plus PA 0.2 mmol/L: 0.821±0.257, TNF-α 20 ng/mL plus PA 0.08 mmol/L: 1.032±0.286, TNF-α 20 ng/mL plus PA 0.2 mmol/L: 2.134±1.049 μg/μg) was significant higher than the cells treated by PA alone (PA 0.08 mmol/L: 0.247±0.069, PA 0.2 mmol/L: 0.341±0.031 μg/μg, all *P*<0.05). Oil red O staining also showed that TNF-α promoted lipid accumulation in HepG2 cells. The expression of SREBP-1, FAS and ACCα at mRNA and protein levels was significantly higher in TNF-α treatment and the TNF-α plus PA treatment cells than in control cells (*P*<0.05). Conclusion TNF-α promotes lipid accumulation, and enhances the expression of SREBP-1, FAS and ACCα in HepG2 hepatocytes.

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