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# hTERT 启动子驱动的肿瘤抑素靶向肝癌细胞表达及其抗血管形成的作用 点此下载全文

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**摘要：**

目的：观察人端粒酶逆转录酶基因（human telomerase reverse transcriptase， hTERT ）启动子驱动的肿瘤抑素(tamstatin)基因在肝癌细胞HepG2内特异性表达及其体外抗血管形成的作用。方法: 构建phTERT-tumstatin、pCMV-tumstatin（阳性对照）、phTERT-EGFP（阴性对照）质粒，脂质体介导转染HepG2肝癌细胞、L-02正常肝细胞，荧光显微镜检测EGFP的表达。Western blotting检测tumstatin在HepG2细胞内的表达，MTS法检测稳定转染后HepG2细胞的增殖以及含或不含tumstatin蛋白的条件培养基对人脐静脉内皮细胞(human umbilical vascular endothelial cell, HUVEC)增殖的影响。通过计数管样分支数检测tumstatin蛋白对HUVEC管道结构形成的影响。结果: 成功构建phTERT-tumstatin、pCMV-tumstatin和phTERT-EGFP质粒，质粒转染后tumstatin基因在肝癌细胞HepG2中特异地表达，在正常肝细胞L-02中无表达。phTERT-tumstatin和phTERT-EGFP转染均不影响HepG2细胞的增殖。含tumstatin蛋白的条件培养基CM-T抑制HUVEC的增殖\[抑制率为(56.49±0 33)%\]；CM-T与不含tumstatin蛋白的条件培养基CM-N、CM-NT相比显著抑制了HUVEC血管结构的形成\[(3.33±1 53)% vs (24.44±3.11)%、(23.94±2.92)%， P <0.01)\]。结论： phTERT 基因启动子可驱动tumstatin靶向表达于肝癌细胞，并抑制HUVEC血管管道结构形成。

**关键词：**肿瘤抑素 肝肿瘤 hTERT启动子 基因治疗 抗血管生成

# hTERT promoter-induced targeting expression of tumstatin in hepatocarcinoma cells and its antiangiogenic effect Download Fulltext

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Abstract:

Objective:To observe the specific expression of tumstatin in hepatocarcinoma HepG2 cells induced by the human telomerase reverse transcriptase ( hTERT ) gene promoter and its antiangiogenic effect in vitro . Methods: phTERT-tumstatin, pCMV-tumstatin (positive control), phTERT-EGFP (negative control) plasmids were constructed and transfected into HepG2 and L-02 normal liver cells. The expression of EGFP was examined by fluorescence microscope. The expression of tumstatin protein in HepG2 cells was detected by Western blotting analysis; the proliferation of HepG2 cells after stably transfected with plasmids was measured by MTS assay; the effect of conditioned medium (containing tumstatin protein or not) on proliferation of human umbilical vascular endothelial cell (HUVEC) was detected by MTS assay. The effect of tumstatin protein on cellular tube structure formation of HUVEC was examined through counting the number of tube branches. Results: phTERT-tumstatin, pCMV-tumstatin, and phTERT-EGFP plasmids were successfully constructed. The specific expression of tumstatin was only observed in hepatocarcinoma HepG2, not in normal liver L-02 cells. phTERT-tumstatin and phTERT-EGFP transfection did not affect the proliferation of HepG2 cells; conditioned medium (CM) containing tumstatin protein (CM-T) inhibited the proliferation of HUVEC cells, with the inhibition rate being (56.49±0.33)%. The cellular tube structure formation of HUVEC cells on matrigel-coated plates supplemented with CM-T was significantly inhibited compared with conditioned medium CM-N and CM-NT (\[3.33±1.53\]% vs \[24.44±3.11\]%，\[23.94±2.92\]%， P <0.01). Conclusion: phTERT gene promoter can induce targeting expression of tumstatin in hepatocarcinoma cells and inhibit cellular tube structure formation of HUVEC cells.

Keywords:tumstatin hepatocellular carcinoma hTERT promoter gene therapy antiangiogenesis

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