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Impact of co-transfection of livin and survivin shRNA expression vector on biological behavior of HepG2 cells

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Objective: To construct a short hairpin RNA (shRNA) eukaryotic expression vector targeting Livin or Survivin gene, and explore the impact of Co-transfection of Livin and Survivin shRNA expression vector on biological behavior of HepG2 cells.

Methods: shRNA eukaryotic expression vector pSD11-Livin and pSD11-Survivin were designed, constructed, and transfected into HepG2 cells in combination with liposome. Cells were divided into blank control group, negative control group, Survivin group, Livin group and co-transfection group. mRNA relative expression was detected by fluorescence quantitative PCR. Western blot were used to detect Livin, Survivin protein respectively. Changes of cell proliferation were detected by MTT, and TUNEL was used to detect apoptosis rate.

Results: The Livin and Survivin shRNA eukaryotic expression vectors were successfully constructed. Livin, Survivin mRNA relative expression quantity in HepG2 cells of co-transfection group was 0.120± 0.022 and 0.325 ±0.125 respectively. Compared with Survivin group or Livin group, mRNA relative expression quantity of co-transfection group was decreased significantly ($P < 0.05$). Livin, Survivin protein relative expression quantity in HepG2 cells of co-transfection group was 0.412± 0.099 and 0.473 ±0.051. Co-transfection inhibited protein expression more efficiently than single-transfection ($P < 0.05$). Cell growth inhibition rate in co-transfection group were higher than those in single-transfection group on 48h, 60h, 72h after transfection ($P < 0.05$). Apoptosis rate increased more significantly in co-transfection group than any other groups ($P < 0.05$).

Conclusion: Livin, Survivin shRNA eukaryotic expression vector was successfully constructed. Co-transfection of pSD11-Livin and pSD11-Survivin reduced the expression of Livin and Survivin gene in HepG2 cells more effectively, inhibited the proliferation of hepatoma cells more significantly, and induced the apoptosis of HepG2 cells more effectively.

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