GMR Genetics and Molecular Research
Online Journal
ISSN:1676-5680

Methylation regulation of liver-specific microRNA-122 expression and its effects on the proliferation and apoptosis of hepatocellular carcinoma cells

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Genet. Mol. Res. 12 (3): 3588-3597 (2013)
Received October 2, 2012
Accepted February 28, 2013
Published September 13, 2013
DOI http://dx.doi.org/10.4238/2013.September.13.3

ABSTRACT. The regulation mechanism and significance of microRNA-122 (miRNA-122) expression are unclear. The aim of this study was to investigate the effects of DNA methylation on liver-specific miRNA-122 expression, cell proliferation, and apoptosis in hepatocellular carcinoma. Methylation of the miRNA-122 promoter region was detected through methylation sequencing. The level of miRNA-122 expression was measured using real-time quantitative polymerase chain reaction. The proliferation and apoptosis of hepatocellular cell lines were detected using flow cytometry and Cell Counting Kit-8 assays. Compared with those in human primary hepatocytes, methylation levels of the miRNA-122 promoter in the Huh7, HepG2, and QSG-7701 cell lines were significantly increased ($P = 0.000$). Similarly, levels of miRNA-122 expression in these cell lines significantly decreased ($P = 0.007$). After treatment with 5-aza-2-deoxycytidine, the Huh7 and HepG2 cell lines displayed a significantly lower degree of methylation ($P = 0.038$ and 0.025), and the levels

of miRNA-122 expression were significantly higher (P = 0.008 and 0.003) than those in the blank group. Compared with the blank group, apoptosis of Huh7 and HepG2 cells was significantly increased (P = 0.001 and 0.027). We concluded that the expression of miRNA-122 is regulated by DNA methylation and correlated with apoptosis of liver cancer cells. Methylation regulation of miRNA-122 expression might be involved in the development of hepatocellular carcinoma.

Key words: MicroRNA-122; Methylation regulation; Apoptosis; Hepatocellular carcinoma

