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Effect of *p53* gene polymorphism on functions of prostate cancer cells

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ABSTRACT. Prostate cancer cells were transfected with plasmids [empty plasmids, wild-type pcDNA3.1-p53 (V/V), mutant type pcDNA3.1- p53 (G/G)] to analyze the effect of *p53* gene polymorphisms on the proliferation, cycle, and apoptosis of prostatic cancer cells. Empty plasmids containing wild-type pcDNA3.1-p53 (V/V) and mutant type pcDNA3.1- p53 (G/G) were used to transfect PC3 and LNCaP cells, respectively. Cell proliferation was detected at 0, 24, 48, and 72 h using the MTT method. Cells were collected at 24 and 72 h. The distribution of cell cycles in various groups was detected using flow cytometry (propidium iodide staining method) and the apoptosis rate was detected using annexin V + propidium iodide double staining. Compared with the control group, wild-type pcDNA3.1-p53 (V/V) and mutant type pcDNA3.1-p53 (G/G) showed a significant inhibitory effect on cell proliferation ($P < 0.05$); the inhibitory effect of the mutant type was stronger than that of the wild-type. There was no significant difference between PC3 cells and LNCaP cells. After transfection with wild-type pcDNA3.1-p53 (V/V) and mutant type pcDNA3.1-p53 (G/G), PC3

and LNCaP cells were arrested in the G0/G1 stage. Transfection with pcDNA3.1-p53 (G/G) showed a more significant effect than transfection with pcDNA3.1-p53 (V/V). Both the wild-type pcDNA3.1-p53 (V/V) and mutant-type pcDNA3.1-p53 (G/G) led to an increased apoptosis rate of PC3 and LNCaP cells. The *p53* gene polymorphism affects the proliferation, apoptosis, and cycle of prostate cancer cells and may serve as a reliable index for the diagnosis and treatment of prostate cancer.

Key words: Apoptosis; Cell proliferation; *p53* gene; Polymorphism; Prostatic cancer

