

News & Comments

Mechanism of Curcumin Improving Cisplatin Resistance

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Breast cancer patients who test negative for ER, PR, and human epidermal growth factor receptor 2 are referred to as having triple-negative breast cancer (TNBC) (HER-2). As the most efficient methods of cancer treatment, chemotherapy, surgery, and radiotherapy are indispensable to the clinical management of cancer. It is crucial to understand the mechanism underlying TNBC resistance as well as the primary targets for producing TNBC resistance. Most tumours respond very well to the platinum-based medication cisplatin (DDP), which is used in chemotherapy regimens for cancer. The effects of curcumin combined with DDP on autophagy of drug-resistant breast cancer cells were examined in this study, as well as the mechanism by which curcumin reduces TNBC's resistance to DDP was explored.

The Liaoning University of Traditional Chinese Medicine in Shenyang, China, was the site of this investigation. Female naked mice weighing 20 g were acquired from Beijing HFK Bioscience Co., Ltd. (Reference No.: SCXK (Beijing) 2016-0002). They have grown adaptably for seven days, fed and hydrated properly, and all procedures adhered to ethical standards. Cisplatin was obtained from Qilu Pharmaceutical Ltd., Co. (H37021358), L-15 medium was purchased from Hyclone Ltd., Co., and curcumin was purchased from Shanghai Aladdin Biochemical Technology Ltd., Co. (CAS 458-37-7). Four groups of MDA-MB-231/DDP cells, comprising a blank control group, curcumin groups, DDP groups, and curcumin groups, were randomly assigned. The tumour-bearing mice were separated into four groups, one of which was the blank control group; at random once the subcutaneous tumour had grown to a diameter of 1 cm. Analysis was performed using SPSS 22.0.

The CCK-8 assay was used to measure each group's cell proliferation activity, and each group's absorbance value represented that activity. Each group's mice had their tumour tissues dissected, and the tumour's mass, diameter, and length were all calculated. The homeostasis of the intracellular environment depends heavily on autophagy. Normal activation of autophagy can hasten the breakdown of intracellular proteins. Both the MDA-MB-231/DDP cell line and the tumour-bearing animals used in this study demonstrated that curcumin had a potent anti-tumour impact. This agrees with earlier research. However, it has a good anti-tumour effect when combined with DDP. Curcumin had no proven anti-tumour impact, according to research on autophagy-related proteins and the TRAP1/Akt/p70S6K signalling pathway.

By reducing the level of TRAP1 protein expression, curcumin may prevent the overactivation of the Akt/p70S6K signalling pathway. Curcumin may lower the resistance of MDA-MB-231/DDP tumour-



bearing animals to DDP and block the overactivation of the Akt/p70S6K signalling pathway. It may also enhance autophagy in MDA-MB-231 cells and cause the autophagic death of tumour cells.

JOURNAL REFERENCE

Qian, M.Q., X.D. Ma and G.W. Pan, 2022. Curcumin improving drug resistance of MDA-MB-231/DDP tumor treatment by enhancing autophagy. *Int. J. Pharmacol.*, 18: 806-816.

KEYWORDS

Curcumin, autophagy, drug resistance, cisplatin, breast cancer

