P53. Ácido fítico ou mio-inositol-hexafosfato ou di-hidrogênio fosfato provoca apoptose e diminuição da proliferação em células do carcinoma gástrico possivelmente por diminuição da atividade do p53

16/09/10

Primeiro trabalho que encontramos na literatura onde a diminuição da expressão do p53 provoca apoptose.

O gene p53 é um gene supressor de tumor e a sua ativação provoca apoptose e diminuição da proliferação celular. Ele sofre mutação com a consequente inativação em mais de 50% dos tumores sólidos e a meta terapêutica habitual é ativá-lo e não inibi-lo.


Ácido fítico = mio-inositol-hexafosfato = di-hidrogênio fosfato = C₆H₁₄O₂₄P₆

José de Felippe Junior

Growth inhibition and apoptosis-inducing effects of phytic acid in human gastric carcinoma cells

[Article in Chinese]
Harbin Institute of Technology, Harbin 150090, China. wanglu_tianfeng@yahoo.com.cn

Abstract

OBJECTIVE: To explore the growth inhibition and apoptosis-inducing effects of phytic acid in human gastric cancer SGC-7901 cells.

METHODS: The growth inhibition action of phytic acid on SGC-7901 cells was examined by MTT assay. AO/EB fluorescence staining and DNA ladder assay were applied to study the proapoptosis effects of phytic acid. The expression of apoptosis relative proteins, P53, were analyzed by using immune histochemistry method. RESULTS: Phytic acid treatment significantly inhibited the growth of human gastric cancer cell SGC-7901 and markedly caused their apoptosis following downregulation of P53 protein expression. CONCLUSION: The downregulation of apoptosis relative protein P53 expression was the possible mechanism of phytic acid induced growth inhibition and apoptosis in SGC-7901 cells.

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[Blocking effect of phytic acid on cell proliferation in human gastric carcinoma]

[Article in Chinese]
Department of Food Science and Engineering, Harbin Institute of Technology, Harbin 150090, China.

Abstract

OBJECTIVE: To explore the bcl-2 and the bax protein expression, the effect and possible mechanism of phytic acid (IP6) on cell proliferation in human gastric carcinoma. METHODS: The inhibiting action of IP6 on human gastric carcinoma was examd by MTT assay. The morphological observation by reverse discrepancy microscope. The apoptosis of SGC-7901 cells exposed to IP6 were not well. The DNA damage rates of SGC-7901 cells treated with IP6 were higher than those of control groups in dose and time dependent manners. The bcl-2 protein expressions treated with IP6 were reduced, and the bax protein expressions treated with IP6 were more than those of control groups in dose and time dependent manners. CONCLUSION: The proliferation of gastric carcinoma SGC-7901 cells inhibited by IP6 could be associated with apoptosis of gene bax and bcl-2.

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