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Ginseng Saponin Induces Apoptosis and Selectively Elevates Protein Levels of p53 and p21 in MMS-Treated NIH3T3 Cells

> Sung Jin Hwang and Jong Kun Park Division of Life Science, Wonkwang University

Is this study we have investigated the effect of ginseng saponin on the p53-dependent apoptosis in NIH3T3 cells exposed to methylmethane sulfonate(MMS), an alkylating agent. Cell morphology studies and FACS analysis showed that diol- and triol-type ginseng saponins increases the MMS-induced apoptosis. Western blot analysis indicated that the post-incubation of saponins potentiated the MMS-induced p53 and p21 expression but decreased those of cdk2, cyclinE and D1, and PCNA. These results suggest that ginseng saponins contain components potentiating MMS-induced apoptosis of NIH3T3 cells via p53 and p21 activation, followed by down-regulation of cell cycle related protein expression.

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Apoptosis of HL-60 Cells Treated with PARP Inhibitor and Base-Damaging Agent

Hye Souk Lee, Sung Jin Hwang, Wan Ju Kim, and Jong Kun Park Division of Life Science, Wonkwang University

It is well established that MMS induces alkylation of bases and SIN-1, via formation of peroxynitrite induces base modification and DNA strand breaks. p53, activated by DNA damaging agents, transactivate genes such as GADD45, p21 and MDM2. PARP is involved in cell cycle progression and DNA repair, and cleaved by caspase during early stage of apoptosis. In the present study, we have investigated the effect of 3AB, and inhibitor of PARP, of the apoptosis induced by MMS or SIN-1. FACS analysis showed that 3AB cotreatment delayed the MMS-induced apoptosis. Western and Northern blot analysis indicated that the MMS-induced expression of p53-dependent pathway genes including p53, p21 and MDM2 is decreased and delayed by 3AB cotreatment. Similar 3AB effect was observed in SIN-1-induced apoptosis. By contrast, the MMS-induced expression of GADD45 protein was increased by 3AB cotreatment. These results suggest that the inhibition of PARP decrease or delay the apoptosis of HL-60 cells induced by base-damaging agent.