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Pharmacognosy?.Cytotoxicity of benzoic acid
related to glucose metabolism. Benzoic acid (BA)
is a naturally occurring plant product and widely
used chemical, which is known to induce glucose
metabolism disorder in animals. In the present
study, we investigated effects of BA on human cell
growth and glucose metabolism in relation to
different molecular mechanisms. BA suppressed
the growth of human normal fibroblast MRC-5
cells as well as human cancerous KB and KB-VIN
cells. The antiproliferative activities of BA were
abolished by actinomycin D and cycloheximide
but not by 5,5-dimethyl-1-pyrroline-N-oxide and
sodium azide, suggesting the involvement of both

de novo protein synthesis and protein formation. In addition, BA induced expression of the glucose transporter GLUT4 at the mRNA and protein levels but markedly inhibited the activities of hexokinase (HK), and pyruvate kinase (PK). Transfection of human GLUT4 complementary DNA in human KB cells significantly prevented the growth inhibition and inhibition of HK and PK activities caused by BA. Among various serine/threonine protein kinases, cyclin-dependent protein kinase-2 (Cdk-2), which regulates the cell cycle, was significantly activated by BA and exhibited dose-dependent increase. In normal and cancerous KB and KB-VIN cells, BA markedly increased activity and expression of Cdk-2 whereas benzyl isonicotinic acid (BIA), an inactive analog of BA, failed to do so. Inhibition of Cdk-2 activity by Roscovitine, a specific inhibitor, significantly blocked BA-induced growth inhibition, GLUT4 expression, HK activity, and PK activity.

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