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Antiproliferative properties of probiotic *Pediococcus acidilactici* MTCC 5101

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Probiotics are live microorganisms, which when administered in adequate amounts confer one or more health benefits on the host by maintaining their intestinal microbial balance. A vast majority of studies in literature have dealt with the anticancer effects of probiotics on various types of cancers. The aim of present study was to investigate the antiproliferative effects of viable cells and cell-free supernatant of *Pediococcus acidilactici* 5101 against Human epithelial colorectal adenocarcinoma (Caco-2) and Hepatocellular carcinoma (HepG2) cell line models under *in vitro* conditions. MTT (3-(4, 5-dimethylthiazol-2-Yl)-2, 5-diphenyltetrazolium bromide) cytotoxicity assay was carried out for assessing metabolic activity of the cells. Morphological abnormalities in cell lines were visualized under Scanning Electron Microscope (SEM). In concordance with previous studies, present study also revealed a dose-dependent antiproliferative effect in presence of viable cells as well as culture supernatant of *P. acidilactici* MTCC 5101 on cancer cell lines. Supernatant (500 µl/ml) had strong inhibitory effects on the growth and proliferation of cancerous cells as compared to whole viable bacterial cells as detected by colorimetric MTT assay. SEM micrographs revealed abnormalities in cell morphology after co-culturing cell lines with bacterial cells. Extensive villi disruption was observed in co-cultured samples resulting in abnormal cell morphology as compared to untreated controls. The reduction in cell division of cancerous cells by probiotics may be attributed to production of antimicrobial compounds such as bacteriocin, inactivation and inhibition of carcinogenic compounds, etc. The results emphasize on the mechanism underlying the observed antiproliferative properties of probiotic strain.

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NF-κB: A new candidate in cigarette smoke-induced atherosclerosis

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Atherosclerosis is a chronic inflammatory disease characterized by gradual thickening and hardening of arteries. It is caused by the slow build up of plaque on the inner side of arterial walls that leads to the reduction of lumen diameter and restricts blood flow. This results in clinical conditions such as myocardial infarction, a leading cause of death all over the world. Epidemiological studies show that cigarette smoking is a major risk for developing this disease. Towards understanding the mechanism, we have established an animal model and found that exposure of guinea pigs to cigarette smoke (CS) causes an induction of apoptosis to aortal section. However, results showed that apoptosis is not involved in the initiation of atherosclerotic development. We also investigated the underlying mechanism of foam cell formation which is the hall mark event for the development of atherosclerosis. Since scavenger receptor CD36 plays an important role in foam cell formation, we have studied the effect of CS on CD36 expression. We observed an increased expression of CD36 in CSE treated macrophage cells. Our study revealed a new mechanism for this increased expression wherein NF-κB activity is involved. Currently, we are studying the effect of NF-κB on CS-induced foam cell formation which may provide an explanation for the rapid development of atherosclerosis in cigarette smokers.

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