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Ten.+38 0676323613
E-mail: uslıakova_g@ukr.net

3acT\nıınK TOJIOBH: nporj). He/nneuhKiıü B.C.
Tejı.+38 0955033215
E-mail: nedzvetskvviclorrgjukr.net

Address of Organising Comınittce
Dept. Biophysics and Biochemistry,
Oles' Honchar Dnipro National University
72 Gagarin Ave.. Dnipro, 49050 Ukraine

colorectal adenocarcinoma cells (p<0.0001). Furthennore, quercetin dramatically decıeased the Bcl-2 expression level vvhich inlıibited the apoptosis (p 0.0001).

Conchtsion.

Our results indicate that quercetin and 5-FU combination inhibits the grovvth of III -29 colorectal adenocarcinoma cells and induce the apoptosis. As a result, quercetin can minimize the cytotoxic effect of 5-FU and combined trcatments of 5-FU and quercetin may use as a new therapeutic approach for colorectal cancer therapy.

Key vvords: quercetin, colorectal cancer, apoptosis, 5-FU, synergistic

DETERMINATION OF PC3 GRONVN MEDİUM RESISTANT *Saccharomyces cerevisiue* OBTAINED BY INVERSE METABOLIC STRATEGY AlNI) TRANS-EFFECT OF ITS METABOLITS ON PC3

Musa Tartik", Mehmet Ciftci[l'], Giyasettin Baydas[l']
•'Department of Molecular Biology and Genetics, Faculty of Arts and Sciences, Bingöl University, 12000 Bingöl, Turkey
[h] Department of Chemistry, Faculty of Arts and Sciences, Bingöl University, 12000 Bingöl, Turkey
[c] Council of Higher Edııcation, 06539, Bilkent, Ankara, Turkey
musatartik(a;hotmail.com

The complexity of cancer is well-known majör problem for trealment of disease. The celi variety and the multiplicity of intracellular pathways make it difficult to find a single and definitive solution for cancer. in order to find a definite solution to cancer, it is necessary to knovv the entire metabolism in detail, but the current level of knovvledge and technology is not sufficient Therefore, it is need to find a natural solution that presents another way to slruggle vvith disease. Therefore, the present study is based on a natural approach to deal with cancer; that suggests using the metabolite of another organism produced against cancer environment. in this study; the mutant strain of *S.cerevisiae* with specific clıaracteristics, vvhich enable to grow in cancerous environment without difficulty, vvere successfully obtained by in vivo evolutionary engineering approach. The cıoss-resistance tests were applied to sclccl the best mutant. Furthermore, the counter effect of DMEMs (Dulbecco's Modified Eagle Medium) fermented with these individual mutants and WT (Wild fypc) ycasts in PC3 cells were examined according to several molecular assays such as celi gro\vth, oxidative injuty, celi migration and apoptosis related gene expressions. Evolutionary engineering was applied to WT yeast population wilh a chemical mutagen EMS (Ethyl Methane Sulfonate) to randomly generate a variety of genetic phenotypes. The best individual mutants with resistant to

PCM (DMEM cultured vvith PC3 cells) (MY2 and MP2) vvere determined vvith genetic stability methods such as MNP and Spot assays. Additionally, the selected mutants and WT vvere separately cultivated in DMEM until logarithmic phase to gain a fermented medium (VVT-DM, MP-DM and MY-DM). PC3 cells treated vvith each WT-DM, MP-DM and MY-DM to clarify its metabolite effects vvith measuring oxidative damage, apoptotic index, celi migration and gene expression assays.

According to the findings, the grovvlh fltness of mutant yeasts dramatically increased in PCM, vvhich compared to WT. Therefore, the randomly EMS-mutagenized population probably consists of the desired colonies that can normally grovv in PCM. This study further displayed that WT-DM, MP-DM and MY-DM significantly decıeased celi grovvth by inducing apoptosis in PC3 celi culture. Ilovvever, MP-DM increased apoptotic index vvhereas it vvas dovvnregulated apoptotic genes exprcssion. Unlike WT-DM and MP-DM, MY-DM simultaneously activatcd many molecular pathvvays, for instance elevated ROS production, suppressed celi migration and upregulation of apoptotic genes expression, to promote apoptosis in PC3 cells. As a conclusion, in order to alter situation that is the restricted grovvth of WT in PCM, the current study vvas successfully applied evolutionary engineering slrategies to obtain the desired phenotypes (MY2 and MP2). Moreover, the results indicated that WT-DM and MP-DM, MY-DM include various effective metabolites to induce apoptosis in PC3 cells.

Keywords: Evolutionary engineering, cancer, PC3, *Saccharomyces cerevisiae,* yeast, fermented medium.

SERUM LEVEL OF ADVANCED GLYCATION END PRODUCTS İN PATIENTS WIT1I POST INFARCTION CHRONIC HEART FAILURE

O. Kuryata, A. Zabida, V. Tkasheıika
"Ministry of Health Ukraine, Dnipropetrovsk Medical Academy, Department of internal medicine JVe2", Dnipro, Ukraine

Introduction: AGEs are end-products formed by oxidative and non-oxidative reactions betvveen sugars and proteins. AGEs formcross-links vvith long-living tissue proteins, vvhich cause them to accumulate in the body över time. AGEs can bindto the receptor of AGE (RAGE) and thereby induce cardiovascular dysfunction.RAGE has a C-truncatedsecretory isoform, soluble RAGE (sRAGE), that circulates in plasma.sRAGE has been proposed to have an athcrosclerotic-protective function.Ilovvever, AGE-RAGE interaction can also cause inflammation and increased AGE-accumulation.AGE-accumulation in turn can cause up regulation of RAGE.Through decıeased compliance of the

PURIFICATION AND CHARACTERIZATION OF GLUCOSE 6-PHOSPHATE DEHYDROGENASE FROM JAPANESE QUAIL ERYTHROCYTES

Yusuf Temel[1], İbrahim Haindi Shafeeq[2], Mehmet Çiftçi[2]
'Program of Medical Laboratory Techniques, Department of Medical Services and Teclıniques, Solhan Vocational School of Health Services. Bingöl University, Bingöl, Turkey, ytemel(ci;bingol.edu.tr.
[2]Department of Chemistry, Faculty of Arts and Sciences, Bingöl University, Bingöl, Turkey.
vtemel(a),bingol.edu.tr.

Glucose-6-phosphate delıydrogenase (G6PD. EC 1.1.1.49, P-D-glucose-6-phosphate; NADP oxidoreductase) is the initial and the key regulatory enzyme in the pentose phosphate pathvvay in carboiıydrate metabolism. Glucose 6-phosphatc delıydrogenase is catalyses the oxidation of glucose 6-phosphate to 6-phosphogluconolactone vvitlı the concomitant reduction of NADP[J] to NADPH "" 3l.

in this study, glucose 6-phosphate dehydrogenase (G6PD; EC 1.1.1.49) whiclı is an importance enzyme for the carboiıydrate metabolism, was purifıed fıom quaifs erythrocyte and characterized. The purification \vas performed by preparation of hemolysate and 2', 5'-ADP Sepharose-4B affinity chromatography. G6PD fıom quail's erythrocyte vvas obtaiııed vvitlı a yield of 77.17% having a specifıc activity of 60.40 EU/mg. protein. The overall purification fold vvas around 4137. 81 kDa by the SÜS PAGE method. Additionally, the optimum temperature. optimum pH. Vmax and KM of the enzyme vvas characterised in that study.

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Keyvvords: G6PD, enzyme, pentose phosphate pathvvay, purification, characterization.