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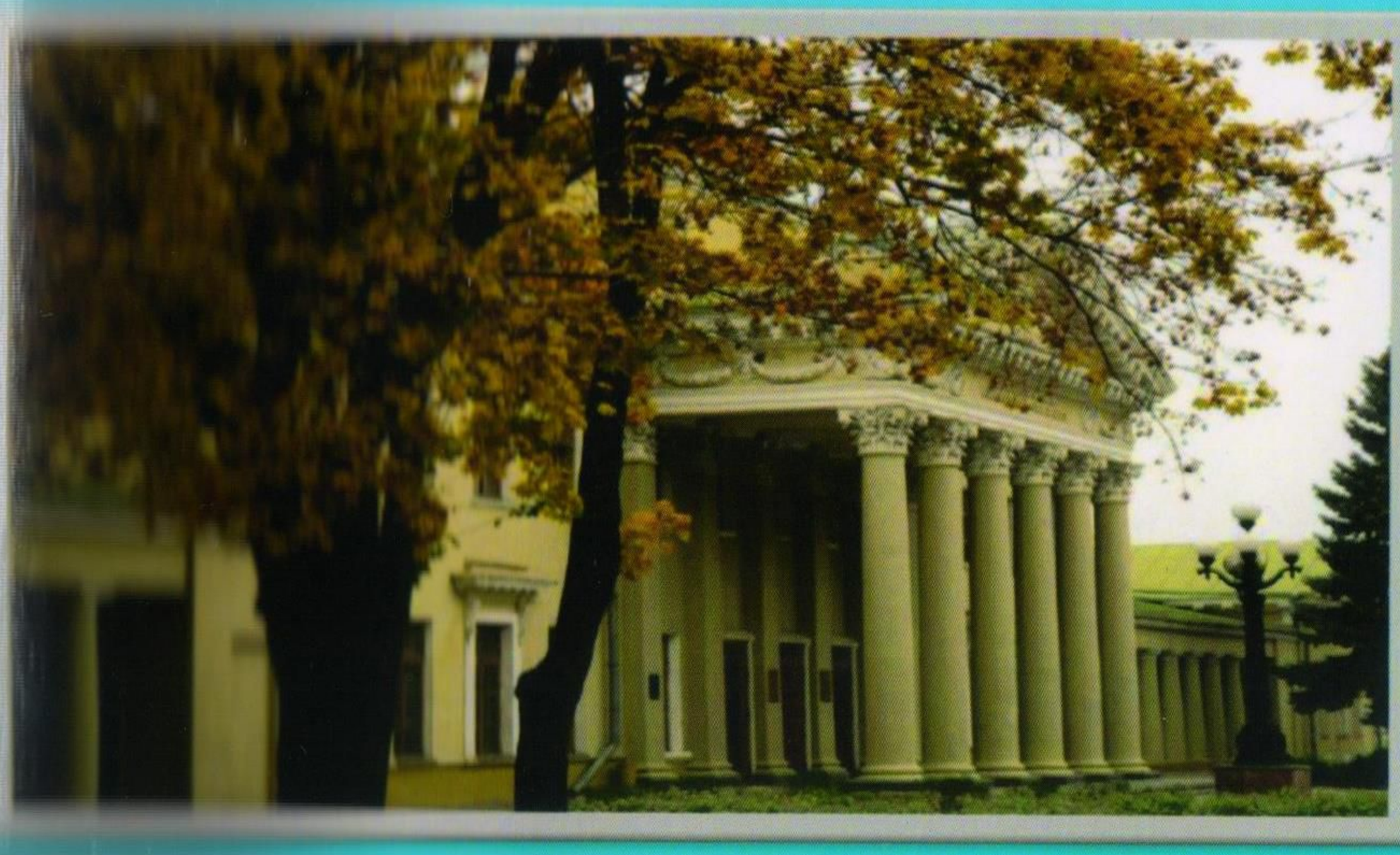

PCM (DMEM cultured with PC3 cells) (MY2 and MP2) were determined with genetic stability methods such as MNP and Spot assays. Additionally, the selected mutants and WT were separately cultivated in DMEM until logarithmic phase to gain a fermented medium (WT-DM, MP-DM and MY-DM). PC3 cells treated with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

According to the findings, the growth fitness of mutant yeasts dramatically increased in PCM, which compared to WT. Therefore, the randomly EMS-mutagenized population probably consists of the desired colonies that can normally grow in PCM. This study further displayed that WT-DM, MP-DM and MY-DM significantly decreased cell growth by inducing apoptosis in PC3 cell culture. However, MP-DM increased apoptotic index whereas it was downregulated apoptotic genes expression. Unlike WT-DM and MP-DM, MY-DM simultaneously activated many molecular pathways, for instance elevated ROS production, suppressed cell 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 growth of WT in PCM, the current study was successfully applied evolutionary engineering strategies 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 IN PATIENTS WITH POST INFARCTION CHRONIC HEART FAILURE

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Introduction: AGEs are end-products formed by oxidative and non-oxidative reactions between sugars and proteins. AGEs formcross-links with long-living tissue proteins, which cause them to accumulate in the body over 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 atherosclerotic-protective function.However, AGE-RAGE interaction can also cause inflammation and increased AGE-accumulation.AGE-accumulation in turn can cause up regulation of RAGE.Through decreased compliance of the

heart and the vasculature, AGE accumulation is considered to be related to the onset and progression of HF.

AGE levels are increased in pro-inflammatory and oxidative stress states. Either by their direct interaction with proteins, such as extracellular collagen, or by its interaction with its receptor (RAGE), AGE can lead to diastolic, systolic and vascular dysfunction. Aim of our study to determinate AGEs level changes in different age groups patients with post infarction heart failure.

Materials and methods: All individuals (25) included aged 40 to 80 years, 20 males and 5 females were diagnosed with preserved ejection fraction chronic heart failure (HFpEF), according to ESC guidelines (2012), and their functional class according to NYHA classification for HF. 20 patients of them have myocardial infarction in anamnesis. All patients got standard treatment for HFpEF according to ESC guidelines 2012. The patients with fresh acute myocardial infarction(MI), 2nd and 3rd degree heart block, diabetes mellitus(DM) glycated haemoglobin ≥7, acute, chronic renal and hepatic failure were excluded.

We divided all included patients into two main groups according to age difference (40-59 years old and ≥ 60 years old).

Results: Analyzed patients were not significantly different in blood glucose level, heart rate, WBCs, ESR and cholesterol. While more patients with triglyceridemia (25%) in patients with history of MI. Attention is drawn to the greater number of males in the group who underwent MI compared with females. In all groups, the predominant risk factor was arterial hypertension with a more severe clinical course in patients with MI in anamnesis.

84% of HFpEF pts were with 3rd functional class(Fc) while 16% were with 2nd Fc, after 2 to 4 weeks of admission all 3rd Fc converted to 2.6 and 2.5 Fc, after 6 weeks all pts was with 2nd Fc.

Median level ofAGE in observed pts was 1.59 [1.38; 1.83] mg/ml, increased level was estimated in 21 (95.5%) pts at baseline. The level ofAGE was highly correlated in age ($R = 0.68$ ($p<0.05$), MI anamnesis presence ($R = 0.71$ ($p<0.05$). > 60 years old patients with CHF characterized by significant higer AGEs level in compare with 40-59 years old pts group ($p=0.02$) at the baseline and after 2 weeks observation.

Conclusion: AGEs serum level markedly increased in old-age (≥ 60 y.o) pts with post infarction HFpEF .