pathways could influence placebo effects, the incipient evidence being provided with regard to existence of genes that mediate placebo effects in individual patients. Placebo/nocebo genetics is an area in which research is only in a quit early phase.

Conclusions. The study results evoke the complexity of the placebo and implicitly nocebo phenomena and the difficulty of formulating a generally valid theory, which could explain in a clear and complete manner how they function. The described mechanisms have to be regarded in a complementary manner and must be analyzed and regarded as a whole. It is worth to continue the investigations of placebo/nocebo mechanisms so as to optimize the therapeutic interventions and to improve the design of clinical trials.

Key words: placebo effect, nocebo effect, response expectancy, unconditioned factors, placebo genetics

DEPARTMENT OF BIOCHEMISTRY AND CLINICAL BIOCHEMISTRY

286. IS LACTATE A USEFUL MARKER OF CARDIAC HYPOXIA?

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Introduction. According to World Health Organization report, cardiovascular diseases are the leading cause of mortality, accounting for 31% worldwide. Cardiac ischemia is the key-mechanism that underlies acute coronary syndrome and induces cellular necrosis. Prolonged ischemia and hypoxia switch the myocardial metabolism from aerobic to anaerobic glucose degradation with increased lactate formation. The accumulation of lactate in cardiac myocytes results in acidosis, altered energy metabolism and cellular membrane damage. We suppose that myocardial ischemia could cause an elevation of circulating lactate level.

Aim of the study. The research purpose was to evaluate serum lactate levels in experimental acute myocardial infarction.

Materials and methods. Forty healthy adult male rats were randomly divided into five groups: (L1) - sham; (L2) - control NaCl 0.9% solution; and with acute myocardial infarction induced by subcutaneous injection of isoproterenol hydrochloride 100 mg/ kg (one dose), and sacrificed after 6 h (L3), 24 h (L4) and 7 days (L5) respectively. Serum lactate concentration was determined using standard kit (ELITech, France). Obtained data were represented by median and interquartile range. For group comparison, the Kruskal-Wallis and Dunn nonparametric tests were performed (SPSS 23.0).

Results. The investigated groups have shown statistically insignificant difference for serum levels of lactate ($p < 0.05$). Initially in L3 was identified a slight decrease, followed by a significant increase in L4, with a repeated fall in L5 group.

Conclusions. The obtained data denote the possible release of lactate from the ischemic cardiomyocytes to the blood. Serum lactate level, following acute myocardial infarction is a useful biomarker that reflects the severity of tissue hypoxia.

Key words: acute coronary syndrome, lactate, hypoxia, myocardial metabolism