POSSIBLE CARDIOPROTECTIVE EFFECTS OF VITAMINS B6 AND B9: LESSONS FROM CARDIOMETABOLIC MODELS

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In order to see if there is a relationship between homocysteine and vitamins B6 and B9 (folic acid, folate), as possible targets in cardioprotection, we established a few models of cardiometabolic diseases in rats (intraperitoneally induced hypermethioninemia and hyperhomocysteinemia, streptozotocin-induced diabetes mellitus, monocrotaline-induced right heart failure, and isoprenaline-induced myocardial infarction) during last time. These cardiometabolic models include complex processes like oxidative stress, inflammation, endothelial dysfunction, gasotransmitters, myocardial injury and cardiovascular remodeling. It was found that plasma levels of homocysteine were significantly increased in experimentally induced hyperhomocysteinemia and isoprenaline-induced myocardial infarction. Although vitamins B6 and/or B9 deficiency can cause hyperhomocysteinemia, and hyperhomocysteinemia is associated with cardiovascular risk or injury, the application of these vitamins, individually or together, confirmed to affect oxidative stress, inflammation, and gasotransmitters. In addition, observational clinical study was also done with an aim to determine the relationship between biomarkers of homocysteine metabolism, inflammation, endothelial dysfunction and severity of coronary artery disease (SYNTAX score) in patients with stable angina pectoris. The increased plasma level of homocysteine, interleukin 6, hs CRP, fibrinogen, and erythrocyte sedimentation rate were detected in patients with high clinical SYNTAX score (>33). Although there is a confirmed role of homocysteine in CVD, the obtained results do not confirm appropriate therapeutic effects of vitamins B6 and B9 in monitored pathophysiological processes.