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The impact of B vitamins on the functioning of methylation cycle in the liver and the kidneys of hyper- and hypothyroid rats.

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Hyperhomocysteinemia is a risk factor for endothelial dysfunction and, consequently, for cardiovascular disease and multiple other conditions. Impairment of homocysteine metabolism is known to occur in thyroid dysfunction.
In particular, patients with hypothyroidism have significantly higher homocysteine levels than healthy people. Metabolism of homocysteine occurs in methylation cycle (whose normal functioning is dependent on tissue pools of vitamins B9, B12 and betaine), and also in reactions of trans-sulfonation, where pyridoxal phosphate (a pyridoxine derivative) acts as a coenzyme.
AIM: The aim of this study was to perform an experimental feasibility assessment of using pyridoxine, betaine, folic acid and cyanocobalamin to correct the methionine and homocysteine metabolism impaired by hyper- and hypothyroidism.
MATERIALS AND METHODS: Prolonged hyperthyroidism and hypothyroidism were modeled in experimental rats by dosing the animals with L-thyroxine and thiamazole, respectively, for 21 days.
RESULTS: Prolonged hyper- and hypothyroidism was found to cause oppositely directional changes in homocysteine metabolism. Hyperthyroidism was causing a significant increase in activity of S-adenosyl-methionine synthase, betaine-homocysteine methyltransferase and S-adenosylhomocysteine hydrolase in the liver and kidneys compared to control group of animals. Such directionality of changes in activities of above mentioned enzymes has led to a reduction in serum homocysteine levels. Hypothyroidism inhibited the activity of S-adenosyl-methionine synthase, betainehomocysteine methyltransferase and S-adenosylhomocysteine hydrolase in the liver and in the kidneys of rats compared to controls. Betaine partially prevented impaired betaine-homocysteine methyltransferase activity in hyper- and hypothyroidism. Folic acid, cyanocobalamin and pyridoxine significantly reduced homocysteine levels in the

blood of animals with hypothyroidism.
CONCLUSIONS: A conclusion was made that the above agents could be effective
factors to prevent endothelial dysfunction in hypothyroidism.

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