MEDICAL SCIENCES

HYPERHOMOCYSTEINEMIA IN CHILDREN LIVING NEAR THE CHERNOBYL EXCLUSION ZONE AS AN ADAPTATION TO RADIATION EXPOSURE

Yuri Bandazheuski[1]
Nataliia Dubovaya[2]

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The incorporation of ^{137}Cs into the bodies of residents of areas affected by the Chernobyl Nuclear Power Plant accident is a common occurrence [1, p. 12]. Even many years after the Chernobyl tragedy in 1986, radioactive elements have a negative impact on the bodies of children living near the Chernobyl Exclusion Zone (ChEZ).

The implementation of humanitarian projects by the European Commission and the Rhône-Alpes Regional Council (France) in Ukraine from 2013 to 2017 created conditions for in-depth clinical and laboratory examination and radiation monitoring of children in the Ivankivsky and Polessky districts of the Kyiv region living near the ChEZ.

Radiation monitoring revealed the presence of ^{137}Cs in the bodies of the children examined. The specific activity of ^{137}Cs in the bodies of the children examined ranged from 0.78 to 95.11 Bq/kg.

Incorporated ^{137}Cs radionuclides cause mitochondrial damage, leading to energy deficiency and disruption of the transmembrane calcium transport.

An inverse relationship was established between the concentration of ^{137}Cs in the children's bodies and the concentration of serum calcium [2, p. 342].

Increased serum aspartate aminotransferase (AST) activity above physiological levels wasrecorded in the blood of 37.5% of the children examined, indicating damage to the mitochondria of cardiomyocytes and, consequently, the development of metabolic stress [1, p. 28].

The involvement of radiation in the damage to the mitochondria of cardiomyocytes was confirmed by a direct correlation between the values of ^{137}Cs specific activity in the children's bodies and the AST values in their blood [1, pp. 31, 32].

[1] Ecology and Health Coordination and Analytical Centre, Ukrain
[2] Ecology and Health Coordination and Analytical Centre, Ukraine

The effects of ^{137}Cs on the cardiovascular system of children living near the ChEZ in most cases manifested themselves as various types of arrhythmias [1, p. 23] and a decrease in their adaptive capacity [3, p. 28].

Furthermore, most of the adolescents examined were found to have hyperhomocysteinemia – elevated blood levels of the sulfur-containing amino acid homocysteine (H_{cy}) due to impaired methylation [4, p. 29].

Methylation processes are closely linked to mitochondria, where tetrahydrofolate and betaine are formed. Damage to mitochondria by incorporated ^{137}Cs radionuclides, and the resulting oxidative stress, are the cause of hyperhomocysteinemia in children living near the ChEZ.

Enzymes of the folate cycle are associated with oxidative and antioxidant processes.

B_{12}-methionine synthase is an enzyme that transfers a methyl group to the H_{cy} molecule, forming internal methionine (Met).

In a subgroup of children with the GMTR:2756 genetic allele, which negatively impacts B_{12}-methionine synthase activity, H_{cy} is utilized in the transsulfuration cycle to form cysteine and glutathione, which has antioxidant activity.

In this same subgroup, a direct correlation was recorded between H_{cy} and cortisol, with blood cortisol levels higher than in the subgroup with the homozygous AMTR:2756 allele [5, pp. 197, 198, 199].

Oxidative stress caused by ^{137}Cs incorporation, in conjunction with the GMTR:2756 genetic allele, contributed to increased triiodothyronine (T_3) levels in the blood [6, p. 199].

T_3 stimulates energy metabolism in mitochondria and the formation of methyltetrahydrofolate, the active form of vitamin B_9 [7, p. 65].

The formation of methyltetrahydrofolate, in turn, stimulates the formation of the active form of vitamin B_{12}, methylcobalamin.

Thus, T_3 enhances the methylation of H_{cy} and the formation of endogenous Met.

Under conditions of calcium retention in tissues with ^{137}Cs incorporation, the adaptive response is the ability of H_{cy} to stimulate an increase in serum calcium, thereby maintaining intra- and extracellular homeostasis.

In most cases, parathyroid hormone production is maintained in the required amount, allowing phosphorus to be excreted from the body in the urine.

In children carrying the GMTR:2756 and TMTHFR:677 alleles, the relationship between parathyroid hormone and serum phosphorus is disrupted under conditions of ^{137}Cs incorporation, as evidenced by the absence of a corresponding correlation.

A decrease in parathyroid hormone production promotes phosphorus retention in the body and creates conditions for the formation of calcium-

phosphorus compounds in the blood, which subsequently leads to pathological processes in the circulatory system.

In a genetic subgroup with a homozygous variant of the GMTR:2756 allele, which suppresses B_{12}-methionine synthase activity, the inverse correlation between H_{cy} and parathyroid hormone reflects a direct negative effect of H_{cy} on the formation of this hormone [2, p. 353].

Thus, hyperhomocysteinemia, recorded in most children living near the ChEZ, with a certain state of folate cycle genes, is an adaptive response of the body aimed at reducing the negative impact of oxidative stress under conditions of exposure to incorporated ^{137}Cs radionuclides.

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