Vitamin B_{12} (cobalamin) plays an important role in maintaining normal cell metabolism. Its deficiency can manifest as hematological (anemia) and neurological disorders, and prolonged vitamin B12 deficiency may have serious consequences, such as increased incidence of neurological and cardiovascular diseases or increased susceptibility to SARS-CoV-2 infection. In addition to insufficient intake of vitamin B_{12}, its deficiency can be caused by malabsorption, disruption of its transport in the blood, or impairment of its cell uptake and metabolism. Despite the fact that vitamin B_{12} was isolated and characterized almost 70 years ago, the pathways transforming the ingested vitamin into a compound that actively participates in metabolism are still not fully understood.

The latest experimental research carried out by our team showed that mice with a deficiency of one of the enzymes of adenine nucleotide catabolism - ecto-5'-nucleotidase (CD73) were characterized by the presence of symptoms and disorders typical of vitamin B12 deficiency. They showed a lower intracellular concentration of vitamin B_{12} despite its normal concentration in the blood, which suggests impaired uptake of vitamin B12 into the cell. In addition, we observed a reduced activity of the processes requiring vitamin B_{12}, which was also manifested by an increase in the concentration of indicators of vitamin B_{12} deficiency, such as homocysteine and methylmalonic acid. Interestingly, CD73-deficient mice also had multiple changes in peripheral blood counts and reduced learning ability, symptoms similar to those seen with vitamin B_{12} deficiency.

Therefore, the main goal of the project is to understand why exactly CD73 is needed in the activation of vitamin B_{12}. In addition, this project aims to find new therapeutic options to restore normal CD73 function and thus maintain the proper functioning of vitamin B_{12} in the body. The assumption of the project is also to identify a specific metabolite or changes in the structure of the CD73 (NT5E) gene associated with vitamin B_{12} deficiency, and thus propose a new diagnostic test.

This project includes research on cell lines, animal research models and clinical samples. The planned experiments and analyzes will be carried out using advanced laboratory techniques such as: *in vitro* and *in vivo* fluorescence imaging, colorimetric tests, molecular biology methods (PCR and gene sequencing), enzyme immunoassays (ELISA), liquid chromatography, mass spectrometry, animal behavioral tests laboratory tests or peripheral blood analysis.

This project could, for the first time in years, identify a new protein involved in the metabolism of vitamin B_{12}. The discovery of the CD73-dependent vitamin B_{12} metabolic pathway could represent a breakthrough, helping to develop new diagnostic tools and therapeutic strategies for the treatment of vitamin B_{12} dysfunction.