It is well known that thinking is a process that consumes a lot of energy, and the energy needs of our brain are met in a priority way over others in the body. The cells that use the most energy in the brain are neurons that communicate with each other through synaptic connections, specialized contact points called synapses. Mitochondria are responsible for energy production in the cell, and synaptic activity is associated with increased demand for mitochondrial ATP production. ATP molecules, the fuel necessary to drive all life processes, are very quickly used by the cell and must be produced practically at the place where they are consumed. That is why synapses that need a lot of energy also need well-functioning mitochondria. In our recent studies, we have shown that many mitochondria-building proteins are produced in synapses, in a process called local synaptic translation. That may indicate that synapses produce proteins necessary for building mitochondria on-site to secure their “powerhouses” and ensure their proper functioning. Here one can ask what will happen if the balance is disturbed and the mitochondria supplying energy to synapses will work incorrectly? Based on our preliminary results, we can say that when the local protein synthesis in the synapse is disturbed, such as in the mouse model of fragile X syndrome, we observe altered respiration and morphology of synaptic mitochondria. In the proposed project, we are planning studies that will allow us to understand the role of mitochondria and their dysfunction in synapses of two mouse models of genetically determined neurodevelopmental disorders associated with autism. We expect that the knowledge gained during the implementation of the project will allow us to propose the new therapeutic targets for the treatment of neurodevelopmental diseases co-occurring with autism spectrum disorders.