Zika virus (ZIKV) was the cause of recent epidemic in South America and the reach of it's carrier – Aedes mosquito – in the warm season stretches far into the North America. Symptoms of the infection in humans are mild and similar to common flu. However, infection during first or early second trimester of pregnancy might result in microcephaly – a birth defect characterized by small head and various levels of mental handicap. It is known that increased level of apoptosis (cellular self-destruction) in the developing brain cells contributes to microcephaly and indeed preliminary experiments show that Zika infection does induce apoptosis in neural cells. The chain of events starting from Zika infection and ending in apoptosis is unknown. Gaining knowledge of this mechanism would be the first step towards stopping or preventing it by developing effective treatment strategy.

We plan to investigate whether the increased levels of apoptosis, caused by the viral infection, is a result of disruption in the cellular structure called nucleolus. The nucleolus is responsible for production of ribosomes – molecular factories supplying the cell with proteins. In the developing cells, disrupted ribosome production leads to the halt of their growth and apoptosis. Data obtained from the infected cells confirm that at least one Zika protein migrates to the nucleolus and that nucleolar disruption is induced by the virus. We also know that a group of proteins related to various genetic syndromes manifesting in microcephaly resides in nucleolus.

Using the mass spectrometry, we intend to investigate the interaction of two Zika proteins (protein C and NS5) with host cells and proteins. We intend to focus on proteins directly involved in ribosome production. Our research will be performed in human neuroprogenitor cells and the results obtained from mass spectrometry will be confirmed with microscopy and immunological methods in this and other cellular models.