Background: Cerebral palsy (CP) is one of the most severe consequences of prenatal hypoxia-ischemia (H-I). We have a unique rabbit model of CP that develops after acute placental insufficiency at preterm gestation, based on the clinical paradigm of abruptio placentae. This model is the first to reliably lead to a CP phenotype. It allows us to rigorously test not only putative therapies but also mechanistic pathways of CP in animals. A non-invasive MRI biomarker, using apparent diffusion coefficient (ADC), allows us to predict the phenotype (postnatal hypertonic or non-hypertonic) immediately after H-I and thus identify susceptible animals in the same litter.

Hypothesis: The individual susceptibility of rabbit fetuses to HI-induced motor deficits of cerebral palsy is underlined by differential expression of key susceptibility genes

Method: Brain samples were collected from hypertonic (sick) and non-hypertonic (healthy) animal based on MRI facilitated predictions and subsequently RNA was extracted. Gene expression levels were compared between the two groups using differential display (DD) assay due to the lack of full sequencing information of rabbit genome. The resulting DD bands from PCR reaction were purified and sequenced. Then the full-length gene sequences were obtained by using Rapid Amplification of cDNA Ends (RACE) PCR. Homologous genes in human, mouse and rat were identified with NCBI BLAST.

Results: A brand new gene (Lei-2) and several other new genes that has homologous with human's were identified has significant diffident expression levels between sick and healthy animal brain. Their sequences were submitted to GenBank. The exact functions of those genes in brain were currently under investigation.