Type 1 diabetes (T1D) is an organ-specific Th1-mediated autoimmune disease with a globally increasing incidence. A significant rise in new cases has also been observed in the Czech Republic, particularly in the youngest age group (0–4 years). T lymphocytes and the cytokines they produce are believed to play a central role in the development of T1D. At present, we are unable to detect early signs of cellular autoreactivity leading to the destruction of pancreatic beta cells, and therefore, this disease cannot yet be successfully cured or prevented.

The naive immune system of a newborn, not yet exposed to external environmental influences, may serve as an important model for studying the pathogenesis of T1D. We analyzed cord blood from 22 newborns with a first-degree relative affected by T1D (T1DR) and 15 newborns with no family history of the disease. Using a protein microarray, we assessed the production of 23 cytokines—both before and after stimulation with diabetogenic autoantigens.

In the T1DR group, we observed low baseline secretion levels of all detected cytokines: GM-CSF ($p=0.025$), GRO ($p=0.002$), GRO-alpha ($p=0.027$), IL-1 alpha ($p=0.051$), IL-3 ($p=0.008$), IL-7 ($p=0.027$), IL-8 ($p=0.042$), MCP-3 ($p=0.022$), MIG ($p=0.034$), and RANTES ($p=0.004$).

In comparison, the control group showed significantly reduced levels after stimulation only for G-CSF ($p=0.030$) and GRO-alpha ($p=0.041$). Additionally, a significant post-stimulation decrease in G-CSF ($p=0.030$) and MCP-2 ($p=0.009$) was observed in the control group when compared to the T1DR group.

These findings suggest that the immune system of T1DR newborns is less mature and more sensitive than that of newborns from healthy mothers. Notably, the influence of a high-risk genotype on protein microarray results was also significant. T1DR infants with a high-risk genotype had elevated baseline levels of G-CSF ($p=0.038$), GM-CSF ($p=0.020$), and GRO-alpha ($p=0.033$). After stimulation, they also showed a tendency toward a Th1-type response, with increased levels of IL-2 ($p=0.020$) and IFN-gamma ($p=0.001$), compared to T1DR infants at "low risk for T1D development."