Pre- and perinatal infections belong to the main causes of neonatal deaths. The high susceptibility to infection reflects the immaturity of the immune system. Premature and low birthweight babies are at particular risk due to underdevelopment of the respiratory system and other organs, impaired thermoregulation and need for invasive procedures. Newborns, especially prematurily born are not able to produce specific antibodies against pathogenic microbes, as bacteria, fungi or viruses. Thus, the very important protective role against infection is played by factors of innate immunity. Proteins belonging to the ficolin family (Ficolin-1, -2 and -3) are representatives of this branch of the immune defense. They are predominantly present in blood. The shape of their complex molecules resembles the “bunch of tulips”. They are able to recognize microorganisms and contribute to their elimination. They may directly destroy pathogens via destruction of their cell envelope. It is possible thanks to the initiation of so called complement cascade – a system of proteins present in serum, being activated in the face of infection. They also opsonize microbial cells and enhance their uptake and elimination by specialized host cells (phagocytosis, intracellular killing of pathogens). Certainly, ficolins show quite similar activity to antibodies, but in contrast to them, they are always ready (at a sufficient level) to act and their specificity (the ability to recognize microorganisms as the “foreign”) is relatively broad.

We previously analyzed selected innate immunity factors in a large cohort of Polish newborns (>1800, including 300 preterms). We postulated that one of ficolins, Ficolin-2 may play especially important role in protection against neonatal infections. Its low concentration in cord blood serum was associated not only with perinatal infections but also with increased risk of prematurity and low birthweight. Although serum level of this factor is influenced by the corresponding *FCN2* gene polymorphisms, we did not find any genetic background of higher susceptibility to infections of Ficolin-2 insufficient babies. It should be mentioned , that other researches shown the ability of Ficolin-2 to recognize numerous pathogens especially dangerous for newborns like streptococci and staphylococci, including Streptococcus agalactiae being common reason of neonatal fatal sepsis, pneumonia or meningitidis. Due to that, we are planning to search for unknown so far *FCN2* gene polymorphisms influencing concentration and activity of Ficolin-2 and determine their impact on the risk of severe infections in preterms. It should be mentioned that the protein level (Ficolin-2 including) may be also regulated by so called epigenetic mechanisms like microRNAs (miRNAs). During the development of some diseases their expression may change and lead to dysregulation of production of targets proteins. That is why we are going to determine whether profile of microRNA expression in preterms with abnormal (extremely low or extremely high) cord serum Ficolin-2 concentrations is associated with increased risk of infection. We are also planning the selection of specific miRNA influencing the synthesis of Ficolin-2 and identify polymorphims of their genes. It is worth noting that the study will be non-invasive as it requires taking several drops of cord blood only. Another goal of our investigation is to determine ability of Ficolin-2 to interact with microorganisms (bacteria, fungi) isolated from cases of severe neonatal infections. The project realization will be helpful not only for extension of the knowledge about significance of Ficolin-2 in neonatal immunity but it will also contribute to the verification of Ficolin-2 as potential biomarker of sepsis/severe infection. It might also initiate the future projects concerning the possible application of Ficolin-2 as the therapeutic agent. Elucidation of certain immune response mechanisms and search for new diagnostic/therapeutic tools is of great importance due to increasing antibiotic resistance of microbial pathogens.