Atopic dermatitis (AD) is the most common skin disease affecting children and is prevalent in 30% of infants, however up to 85% of AD cases in childhood develop up to 5 years of age. This disease is characterized by wide spectrum of clinical symptoms and complicated pathophysiology. Despite age of patients, AD is predominantly Th2 associated disease, however recent studies indicate that different disease endotypes are present in certain age groups and specific immune mechanisms affecting children and adult AD may be different. Atopic dermatitis is often associated with other allergic diseases. Food allergy is connected with AD and eczema is the strongest risk factor for food allergy development in infants. Understanding of immunologic pathways leading to development and progression of AD and its connection with other allergic diseases is restricted, mainly due to invasiveness of methods of obtaining biologic material which is classical skin biopsy. The aim of this research is assessment of local, skin pathologic immune mechanisms involved in pathogenesis of AD in children aged 0-2 years of age. We will assess expression of proinflammatory and structural proteins of epidermis using minimally invasive method of skin tape stripping. Furthermore local skin biomarkers will be compared to systemic, well known AD biomarkers.

Children with AD in active phase of the disease, aged 0-2 years of age will be included into the study. Control group will consist of healthy, age and gender matched children. Patients will be recruited during routine allergy diagnostic visit in the Department of Pediatric Respiratory Diseases and Allergy (T0 – inclusion into the study). During medical interview exact medical history concerning disease course, onset, socio-economic factors will be collected. Subsequently clinical examination and disease severity assessment will be conducted using SCORAD index (SCORing Atopic Dermatitis), EASI (Eczema Area and Severity Index), BSA (Body Surface Area) and POEM (Patient Oriented Eczema Measure). Quality of life of patients will be assessed using IDQOL (Infants' Dermatitis Quality of Life Index) questionnaire. Skin barrier function expressed as epidermal hydration will be examined using non-invasive method of corneometry (Conrneometer CM 825). Blood samples will be taken for routine allergy diagnostics (blood morphology, total and specific IgE of the most common food and air borne allergens concentration) and for CCL17 chemokine concentration assessment using ELISA method. Furthermore, epidermal specimens will be taken using minimally invasive method of skin tape-stripping. As a next step expression of proinflammatory cytokines (IL-13, IL-19, IL-21, IL-26, IL-34) and structural protein (filaggrin 1) expression will evaluated using quantitative RT-PCR method. Moreover analysis of connection of local, skin biomarkers with disease severity and allergic sensitization to most common allergens will be conducted. New, potential biomarkers will be compared to well established disease biomarkers which are tIgE, CCL17 and eosinophil count. After one year (T1) and two years (T2) children will be examined or contacted to assess presence and trajectory of AD and development of other allergic diseases.

This research will lead to better understanding of AD pathogenesis in children at the age of 0-2 years. Furthermore data obtained during this research may lead to discovery of potential biomarkers which connected with AD severity, progression and connection of other allergic diseases.