Atopic dermatitis (AD) is one of the most common chronic inflammatory skin diseases, that is characterized by intense itching and recurrent eczematous lesions. While the primary events and key drivers of the disease are topics of continuing debate, there are two major and converging pathophysiological abnormalities of epidermal structure due to decreased filaggrin expression and function and cutaneous inflammation due to inappropriate immune responses to antigens encountered in the skin. AD cannot be cured at present and the current therapy of AD consists of topical emollients for skin barrier dysfunction and topical corticosteroids or calcineurin inhibitors for skin inflammation. In severely affected cases, systemic immunosuppressants and cytostatic drugs are indicated. These conventional treatments, however, may not show uniform efficacy and can be limited by severe side effects. Therefore, searching for more efficient and safer therapeutic strategies for AD are justified. Recent studies have shown that heat shock protein 90 (Hsp90) is involved in activation of innate and adaptive cells of the immune system and the pharmacological inhibition of Hsp90 has been successfully applied in murine models of autoimmune and inflammatory diseases. The main goal of this project is to test whether application of Hsp90 inhibitor will ameliorate the severity of AD lesions in preclinical studies. In this project, we would like to estimate efficacy and safety of this experimental therapy.