Allergic contact dermatitis (ACD) is a classic example of T cell-mediated delayed type hypersensitivity. ACD develops after skin or mucosa exposure to low molecular weight substances (haptens). At the moment ACD is one of the most common occupational disease. In order to determine the mechanisms involved in ACD, experimental studies are conducted in animal models of contact hypersensitivity (CHS).

It is commonly known, that obesity is a major risk factor for many inflammatory diseases, it can also affect a disease course and severity, as well as healing process. Research conducted in the Department of Medical Biology UJ CM have shown that high fat diet -induced obesity enhances classic contact hypersensitivity reaction. At the moment, the mechanisms affecting CHS response in obesity are poorly understood. In our research, we will employ commonly used animal model of CHS (model of active sensitization), whereas obesity will be induced by special high fat diet. In order to induce CHS reaction, mice will be sensitized with hapten. Five days later, baseline ear thickness will be measured and then mice will be challenged by painting both ears with hapten. The ear swelling will be measured 24 hours later to determine the inflammatory reaction. The presence of edema can be caused by variety of factors e.g. increase in vascular permeability, which leads to the passage of fluid and leukocytes to the tissues. The results obtained in the *in vivo* tests will be confirmed by *in vitro* experiments.

High fat diet-induced obesity affects the composition of gut microbiota, and consequently influences the immune processes. This may explain the increasing prevalence of inflammatory diseases including allergy in the obese subjects. Thus, we plan to compare the bacteria that colonize the gut of mice fed either high fat diet or control diet, and to check whether oral transfer of intestinal flora from donors receiving high fat diet to naive recipients fed normal diet can modify CHS reaction in mice.

Investigating the mechanisms involved in modification of CHS and the role of natural gut flora in observed phenomenon will not only contribute to the broadening of our knowledge, but should also prove useful in the future in the context of predicting increased susceptibility of obese individuals to the diseases in which delayed hypersensitivity is the major pathophysiological response.