Iron deficiency is a common disorder in patients with end stage renal failure and on renal replacement therapy. In most cases intravenous infusion of iron compounds is done to correct deficit of that ion. Such treatment has properties of the "double-edged sword". It helps to correct the anemia, but at the same time appearance of a large dose of iron results in increased generation of free radicals, what may result in damage to the surrounding tissues. In patients treated with chronic peritoneal dialysis part of the iron load administered intravenously can diffuse into the peritoneal cavity filled with the dialysis fluid, which does not contain iron binding proteins. In our previous study we found that iron sucrose given intravenously in peritoneal dialysis patients induced inflammation in the peritoneal cavity and was toxic towards the peritoneal mesothelial cells. Furthermore we found that iron from the dialysate accumulated in the mesothelial cells. In the preliminary experiments we found that after intravenous infusion of Monofer® - Iron Isomaltoside 1000 in the end stage renal failure patients treated with chronic peritoneal dialysis, inflammation was induced within the peritoneal cavity and the dialysate was cytotoxic to peritoneal mesothelial cells in *in vitro* culture. Glutathione precursor – N-Acetylcysteine reduced the cytotoxicity of the dialysate towards the mesothelial cells.

We plan to evaluate what is the peritoneal reaction to intravenous treatment with three different iron compound used in Poland in end stage renal failure patients: Iron Sucrose, Iron Isomaltoside and Iron Carboxymaltose in a patients treated with chronic peritoneal dialysis. Acute and chronic cytotoxic effect of these drugs and dialysate containing these substances on function of the mesothelial cells will be studied, to verify possible proangiogenic and/or profibrotic effect of such treatment in the peritoneum. Such changes may lead to ultrafiltration failure and termination of peritoneal dialysis. Iron induces the oxidative stress which can activate various intracellular pathways leading to pathological changes. We plan to study the role of NF-κB pathway in these processes. Effect of the studied iron compounds and dialysates containing these drugs on the mesothelial cells will be studied in a separate group of experiments in presence of NF-κB inhibitor: dehydroxymethylepoxyquinomicin (DHMEQ). Previously we found that DHMEQ reduces the negative effect of the dialysate on the mesothelial cells in *in vitro* culture. Based on our preliminary data, we want also to evaluate if N-Acetylcysteine, used as additive to the dialysate or given intravenously, is effective in reducing the acute and chronic cytotoxicity of iron towards the peritoneal mesothelium.

Proposed study will provide us with knowledge to what extent intravenous treatment with iron compounds affects the intraperitoneal homeostasis and may predispose to long term deterioration of the peritoneal function as the dialysis membrane. The potential mechanism of such effect will be proposed. Additionally we may be able to elaborate an effective approach to protect the peritoneum in such conditions.