Iron is a necessary microelement for almost all living organisms. Interestingly, the only known organism that successfully can survive without iron ions are Lactobacillus bacteria. Iron participates in many biochemical processes, among which erythropoiesis, the process of the red blood cells formation is the most important. Thus, iron deficiency in the body that is characterized by a disturbance in the production of hemoglobin of the red blood cells is called anemia. On the other hand, excessive accumulation of iron in the body can result in toxicity, so the systemic bioavailability of iron must be tightly controlled to prevent iron deficiency disorders and iron overloads, which according to WHO is one of the most prevalent disorder in the world. In this project, we focus on iron deficiency anemia that affects up to 2 billion people worldwide and mostly affects neonates and young children. Children from 0 to 5 years are the most affected because of the high demand for iron during rapid growth and differentiation. Lack of iron in the early stages of life can cause changes in cognitive functions (learning) and the development of the nervous system later in life. Importantly, the majority of fetal iron stores are the result of iron transfer from the mother in the third trimester, so the shortening of pregnancy, i.e. premature delivery, causes premature to have insufficient iron in the body. Due to the increased risk of developing anemia in premature babies, iron supplementation is recommended in some situations. Treatment of iron deficiency is mainly based on the administration of iron supplements, ultimately even blood transfusions. Due to the potential toxicity of oral iron supplementation, special care should be taken in the treatment of premature babies and newborns. Therefore, the **first** goal of the project is to create an animal model of prematurity reflecting anemia in humans. The results of many studies, including our own, indicate that newborn piglets are a suitable model for studying iron metabolism in newborns. First, pig anemia is the most common deficiency disorder in the early postpartum period in pigs and develops into a critical illness. The piglet anemia model appears to accurately reflect this defect observed in premature human babies because the iron content in their liver is very low. Secondly, the pig is increasingly used in biomedical research for human genetic and nutritional diseases. Thus, to develop an animal model of premature babies we will use sows pregnancy in which the cesarean section will take place on the 107th day of pregnancy, which corresponds to the last trimester of human pregnancy. Due to large litters in sows, there are no plans in the project for pharmacological induction of premature delivery. The **second** goal of the proposed project will be the validation of the animal model in terms of its usefulness in iron metabolism research, i.e. comparison of premature piglets with those born on time (tissue and blood biochemical analysis, including heme, non-heme, total iron content). A **third** objective will be to use the resulting animal model to test the usefulness of the innovative, highly bioavailable Sucrosomial® Iron (SI) in the treatment/prevention of anemia in piglets. SI represents an innovative, commercially available oral iron-containing carrier in which iron is protected by a lipid bilayer membrane and sucrose matrix ensuring its stability, safety and bioavailability. SI has never been used in prematurity and this project is a first attempt. In this part of the project our **hypothesis** assumes that **SI given orally can bypass the biggest problem of prematurity, which is the immaturity of the duodenum in iron protein transporters and in consequence restore iron stores during prematurity**. In this purpose, a comparison between control unsupplemented premature piglets and SI-supplemented premature piglets will be made using molecular and biochemical analyzes in the liver, spleen and intestine. The use of modern analyzes will allow the identification and determination of the level of genes potentially involved in the occurrence of anemia and iron trafficking. The experiment will be performed on Polish Landrace 45 piglets from 6 litters. Preterm piglets will be obtained by caesarean section on day107 of gestation. Term piglets will be obtained after natural delivery at about 115 day of gestation. All piglets will be fed until day 10 after natural birth, when untreated piglets show evidence of "physiological" anemia (i.e. preterm piglets for 18 days and term piglets for 10 days). Experimental groups: N1D1 – term controls and P1D1 – preterm controls, sacrificed on day 1 after birth; NC and NS – term, controls and SI supplemented *per os* from day 2 after birth with 2 mg Fe per piglet, respectively; PC and PS – preterm, controls and SI supplemented *per os* from day 10 after birth with 2 mg of Fe per piglet, respectively; PF – premature piglets receiving daily *per os* from day 2 after birth with 2 mg of Fe per piglet in the form of $FeSO_4$; On day 10 tissue samples will be collected postmortem. In carrying out this project, we hope to create a new, reliable animal model that most closely reflects the anemia of premature human babies. Moreover, based on it, we would like to demonstrate the usefulness (or lack thereof) of Sucrosomial® Iron in the treatment/prevention of anemia in piglets, and in the consequence of infants.