**Summary - general public**

Food choice and eating habits have dramatically changed in Poland and other countries over the last fifty years. Nowadays our society faces not only a notorious excess of caloric intake, but also a decrease in physical activity. Consequently, the escalating global epidemic of overweight and obesity is taking over many parts of the world. Obesity leads to long-term health problems. Generally speaking, excessive accumulation of fat modifies the function of our body, promoting different diseases, such as diabetes, cardiovascular disease or cancer. Besides the impact for public health, costs for treating obesity had almost a ten-fold increase from 1998 to 2007 in Europe. Even more stunning, current research suggests that obesity lead to negative health consequences for our offspring.

When we reproduce, we pass our biological features on to our offspring through the sexual cells, also named gametes (the oocyte and the sperm). In addition to the gene code (the DNA), which contains the genetic information for all biological functions in our body, the gametes also transmit to the offspring the machinery responsible for turning genetic instructions into working proteins, as well as its programming — a phenomenon known as epigenetic. Importantly, our life experiences and environmental exposures can determine changes in the epigenetic regulation of the DNA of the gametes. Thus, in the present research proposal we will address how maternal obesity promotes epigenetic changes in the oocyte (female gamete) and on the embryo during pregnancy, affecting this way the next generation. As a result, we will be able to clearly understand: (i) the extent to which the oocyte senses maternal obesity; (ii) if putative changes established in the oocyte are preserved in the embryo and may affect embryo development during pregnancy. Different mouse models will be subjected to diet-induced obesity protocols, representative of our social dietary habits, and consequences of obesity in female mice reproductive tract will be assessed and followed up in the first offspring generation.

The present proposal exploits the synergies between two institutes, the Institute of Animal Reproduction and Food Research (IARFR), Olsztyn, Poland and the Babraham Institute (BI), Cambridge, UK. With an extensive background in reproductive medicine and endocrinology, the candidate has been producing a relevant body of preliminary data on obesity and ovarian failure. The access to adequate methodology and animal models for the study of obesity is enabled at IARFR. This permits that the animal protocol will be undertaken in IARFR and further analyses will be performed at the BI, which provides the methods and state-of-the-art facilities for epigenetic analysis. Understanding how obesity impacts the oocyte and embryo epigenetic reprogramming will allow us firstly to improve fertility in obese women and secondly to assess the potential health consequences on the offspring. Undeniably, our results will shed light on the establishment of obesity-related co-morbidities, such as cardiovascular-disease and cancer in offspring. Considering that nowadays the global number of clinically obese people is over 300 million, and is continuing to escalate, new insights on the impact of obesity on gametes and gestation and its epigenetic consequences for the offspring are extremely relevant for public health.