The fast pace of our daily life challenges the organism's adaptive processes. Irregular sleep-wake rhythm, for example caused by shift work and sedentary lifestyle, together with the availability of high caloric food lead to imbalance between energy consumption and expenditure. Motor activity, food intake and exposure to light used to be highly synchronized in the past. Nowadays this synchronization is disrupted or lacking, which can increase vulnerability to metabolic disorders and lead to obesity.

Obesity is affecting approximately 600 million people worldwide. In 2016 the number of obese in Poland reached 18% and 21% of the male and female population, respectively. Obesity increases the risk of type 2 diabetes, cardiovascular disease, hyperlipidemia, and some cancers. Such diseases often start with a disruption of behavioral and physiological circadian rhythms. Our most important biological clock is located in the suprachiasmatic nucleus (SCN) of the hypothalamus. To function properly this clock must be sensitive to changes in environmental factors, most importantly the daily variation in light intensity, but also non-photic cues.

The main receiver of non-photic signals is the intergeniculate leaflet of the lateral geniculate body (IGL). IGL integrates photic and non-photic cues to further relay them to the SCN. The timing and regularity of food intake is thought to be a relevant entraining non-photic stimulus for our biological clock. An important satiety signal is mediated by the glucagon family peptides, such as glucagon-like peptide-1 (GLP-1) and glucagon-like peptide-2 (GLP-2) as well as oxyntomodulin. These peptides are produced by a small population of neurons in the brainstem and influence dorsomedial hypothalamus (DMH). The DMH connects to elements of the biological clock, and importantly to systems monitoring food availability and centers regulating body weight and our energy balance.

Currently we lack detailed understanding of the action of glucagon-family peptides on the activity of IGL-SCN-DMH axis. Therefore, we propose a project aimed at filling this knowledge gap. Our research will also focus on circadian/periprandial rhythmicity of the expression of glucagon family peptides. Experiments will compare 2 groups of animals, one fed with balanced (control) and the other with high-fat diet. Feeding the animals the high fat diet (containing 60% of energy from fat) is a commonly used model of diet-induced obesity.

The project is a pioneering attempt to bridge research on the biological clock with research on systems involved in the control of food intake. Additional knowledge about how these two seemingly distant systems interact in their control of the physiology of nutrition may give a better understanding of the causes of obesity in adults and children.