

Abstract

Circadian clocks form an endogenous time-keeping system present in most organisms, synchronizing physiological and behavioural processes with periodically changing environmental conditions. The system comprises of the master clock in the suprachiasmatic nuclei of the hypothalamus and numerous subsidiary clocks in peripheral tissues. Its molecular design is constituted by the clock genes, which are rhythmically expressed, form a series of transcriptional/translational feedback loops and influence the expression of various other genes involved in metabolic pathways. The peripheral clocks are dependent on the master clock, although they can be entrained with external cues like food intake timing and diet composition. This desynchronization leads to the disruption of clock gene oscillation, which can potentially have serious impact on metabolic processes and increase the risk of metabolic disorders.

The aim of this thesis is to summarize current knowledge on the relationship of molecular chronobiology and nutrition with a focus on the molecular mechanisms through which can food, especially its intake timing and composition, influence the crosstalk between clock gene expression and cellular metabolism. The thesis also emphasises the potential effect of circadian clock disruption on the risk of metabolic disease development.