Abstract

The circadian clock generates circadian rhythms, which participate on regulation of a number of signalling pathways. Disruption of the circadian regulatory mechanism is linked to a development and a progression of certain types of cancer including colorectal tumorigenesis. Progression of tumorigenesis depends on the cell cycle machinery related to cell proliferation and apoptosis. MiRNAs play a role in initiation and progression of tumorigenesis because they interfere in regulatory pathways associated with tumorigenesis.

The aim of the thesis was to determinate existence of circadian rhytms in clock controlled genes (*Tef*, *Dbp*), miRNAs (miR-1-3p, miR-16-5p, miR-34a-5p, miR-155-5p, miR-192-3p) and genes of the cell cycle machinery (*Ccnd1*, *Ccne1*, *Ccna1*, *Ccnb1*) and apoptosis (*Casp3*, *Bcl2*, *Bad*). Further, to compare detected circadian rhythms during aging and neoplastic transformation of colon by quantitative RT-PCR.

We have observed circadian expression of *Tef*, *Dbp*, *Ccne1*, *Ccna1*, *Ccnb1*, *Casp3* and *Bcl2* in young mice colon, *Tef*, *Dbp*, miR-1-3p, *Ccne1*, *Ccna1* in old mice colon and *Tef* and *Dbp* in colorectal tumors.

In summary, circadian expression of clock controlled genes varied but was maintained in mice colorectal tumors. In aging we demonstrated weakening of circadian rhythms of the genes of the cell cycle machinery and apoptosis. The larger effect on circadian expression of the cell cycle and apoptosis genes has been observed in aging compared with neoplastic transformation.