

Abstract

The suprachiasmatic nuclei (SCN) play a key role in the adult organism as a major circadian oscillator whose rhythm is stable and temperature-compensated. Temperature resistance in the SCN has not been well understood during fetal development. The aim of this study was to experimentally test whether changes in body temperature could be a potential non-light maternal signal and synchronize fetal circadian rhythms. The research uses organotypic SCN explants taken from the *mPer2^{Luc}* transgenic mouse model, which allows monitoring of the circadian activity of the PER2 protein by real-time bioluminescence. The results showed that fetal SCN is sensitive to temperature changes. Repeated temperature cycles had a significant synchronizing effect on fetal SCN and restart the dampened oscillations. Pharmacological inhibition of the HSF1 protein by quercetin partially inhibited the response of the fetal clock to temperature cycles. The values of the temperature coefficient Q_{10} suggest a lower degree of temperature compensation in the fetal SCN compared to adults. This work compares the differences in circadian clock properties in fetal and fully developed SCN, contributes to the understanding of the development of circadian oscillators and opens the way for further research on maternal synchronization.

Keywords: circadian clock, temperature synchronization, ontogenesis, suprachiasmatic nuclei