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Modelling the mammalian circadian clock: from molecular mechanism to sleep-wake cycle disorders and jet lag

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Circadian rhythms originate at the cellular level from feedback processes in genetic regulatory networks. Based on experimental observations, computational models of have been proposed for the molecular mechanism of circadian rhythms, which occur spontaneously with a period of the order of 24 h in all eukaryotic organisms, as well as in cyanobacteria. Mathematical models were initially proposed for circadian rhythms in *Drosophila* and *Neurospora*, and were later extended to the mammalian circadian clock [1]. The mammalian clock model, based on the intertwined positive and negative regulatory loops involving the *Per*, *Cry*, *Bmal1*, *Clock* and *Rev-Erb* genes, can give rise to sustained circadian oscillations of the limit cycle type. These oscillations correspond to circadian rhythms spontaneously generated by suprachiasmatic nuclei and some peripheral tissues. The results pertain not only to the molecular bases of circadian rhythms but also to physiological disorders of the sleep-wake cycle linked to perturbations of the human circadian clock [2]. Among such disorders are the familial advanced or delayed sleep phase syndromes, and the non-24 h sleep-wake syndrome associated with a loss of entrainment of the circadian clock by the periodic environment. Jet lag that follows delays or advances of the circadian clock can also be studied by means of a computational approach. The latter reveals the existence of critical conditions which might considerably slow down circadian clock recovery after jet lag [3].

[1] Leloup, J.-C. & Goldbeter, A., 2003. *Proc. Natl. Acad. Sci. USA* 100, 7051-56
[2] Leloup, J.-C. & Goldbeter, A., 2008. *BioEssays* 30, 590-600
[3] Leloup, J.-C. & Goldbeter, A. 2013. *J. Theor. Biol.* 333, 47-57

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