Three distinct mathematical formulations of different extent of complexity were postulated by the elaborated version of the two-process model (Achermann & Borbély, 1990; Achermann et al., 1993) for numeral simulations of the three - ultradian, homeostatic, and circadian – processes of sleep-wake regulation.

Data on the time courses of an EEG index (i.e., slow-wave activity) obtained in, at least, two - baseline and recovery - sleep episodes are necessary for simulation of the homeostatic process. The present simulation study was aimed on testing whether this model-based approach to description of diurnal and ultradian oscillations of sleep-wake states and sub-states can be simplified (conceptually, mathematically, and methodologically).

CONCLUSIONS

- In theoretical terms, the suggested approach to modeling of the ultradian sleep homeostasis might help in uncovering most fundamental (i.e., homeostatic) principles of sleep regulation, and,
- In practical terms, it might facilitate application of the methods of model-based analysis of sleep-wake regulating mechanisms to polysomnographic routine sleep recordings (including numerous recordings of night sleep collected in clinical sleep laboratories around the world).

A prediction based on the two-process model (left) and a simulation based on the proposed model – X(t) (right)

A simulation based on the two-process model (left) and a prediction based on the new model – X(t) (right)

A prediction based on the two-process model (left) and a simulation (sleep) and a prediction (wakefulness) based on the proposed model – X(t) and Xu(t) (right)

The same (i.e., homeostatic) regulatory principle was suggested to underlie both circadian oscillations of sleep and wake states and ultradian oscillations of sleep sub-states.

The equations (1) suggest that a primary regulating process underlies ultradian oscillations of sleep deepening-lightening during sleep episode. Each ultradian cycle consists of the ultradian buildup and decay around the set point, X(t).

An additional regulation was suggested to underlie the circadian sleep-wake cycle. After termination of the primary oscillations (sleep deepening-lightening) by wakefulness, the level of X(t) is decaying below its normal range observed during sleep episode. This decay requires corrections of the point that was suggested to be proportional to the changes in an upper asymptote of X(t) (1), Xu(t) (2).

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Major publications: Putilov A.A. Simulation of an ultradian sleep homeostasis through fitting time courses of its EEG indicators obtained during baseline recordings of night sleep. Biol. Rhythm Res. (online: http://www.t便捷online.com/biobio/10.1002/07401812.000005216, 12-06-10055-a, and 12-06-18001-e). I have benefited greatly from help of Olga Donskaya, Dr. Vladislav Pachlokhov, Dr. Konstantin Danilenko, and Dmitriy Zolotarev (Helfie) in the EEG data collection and analyses, and from help of Evgeny Verkudin in simulation of these data.

A simplified approach to description of diurnal and ultradian oscillations of sleep homeostasis might help in uncovering most fundamental (i.e., homeostatic) principles of sleep regulation, and,

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A simulation based on the two-process model (left) and a prediction based on the new model – X(t) (right)

Results of simulations: First, the suggested similar formulas (1, 2) were applied for description of both circadian (2) and ultradian facets (1) of the homeostatic process regulating a normal all-night sleep episode. The parameters were derived by fitting a group-average time course of the EEG indexes (either score on the 1st principal component of the EEG spectrum or slow-wave activity) obtained for only baseline sleep of 14 women of different age. A correlation coefficient between this empirical time course and its simulation attained value 0.89.

Second, the derived parameters of the proposed model were used for prediction of a group-average curve representing 24-h sequences of very short (up to 20 min) naps obtained for 9 sleep deprived and 6 sleep restricted young men. A coefficient between the predicted and empirical curves (0.88) was only slightly lower than a coefficient between the same empirical curve and its direct simulation with the two-process model (0.82).

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