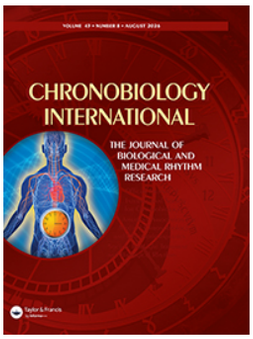

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ORIGINAL ARTICLE

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"Mental clocks" that lag behind the biological clocks: The effect of volitional delays of bedtime on sleep-wake regulation

Arcady A. Putilov[a], Evgeniy G. Verevkin[a], Dmitry S. Sveshnikov[b], Zarina V. Bakaeva[b], Elena B. Yakunina[b], Olga V. Mankaeva[b], Vladimir I. Torshin[b], Elena A. Trutneva[c], Michael M. Lapkin[c], Zhanna N. Lopatskaya[d], Roman O. Budkevich[e], Elena V. Budkevich[e], Marina P. Dyakovich[f], Olga G. Donskaya[a], Natalya V. Ligun[a], Alexandra N. Puchkova[a], and Vladimir B. Dorokhov[a]

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ABSTRACT

Bed- and risetimes of human *ad lib* sleep are determined by the endogenous mechanism of sleep-wake regulation. However, humans are capable to volitionally prolong (i.e. without any external influences) the wake phase of their sleep-wake cycle to early night hours. We hypothesized that the conscious behaviors mostly known under the terms "bedtime procrastination" and "mind-bedtime procrastination" can compete with the biological sleep-wake regulator for setting bedtimes on weekdays and/or weekends and thus this behavior can contribute to the delay of sleep relative to the bedtime signal from this regulator. University students (5375 in total) were grouped in accord with their earlier or later weekday and weekend bedtimes. To test the hypothesis, the group-averaged sleep times were calculated and simulated. Near 8% of students reported later weekday than weekend bedtimes. The simulations of their sleep times suggested that these times cannot be explained by a model of sleep-wake regulator without the additional assumption of bedtime delay caused by the volitional missing the bedtime signal from this regulator (i.e. this missing can be a result of such volitional behavior as mind-bedtime procrastination on weekdays rather than on weekends). We concluded that the concept of two conflicting – biological and "social" – "clocks" that was introduced in this journal 20 years ago can be extended to the concept of three conflicting "clocks." Bed- and risetimes on weekdays and weekends can reflect the competition between three – biological, "social," and, additionally, "mental" – "clocks." In accord with the concept of two "conflicting clocks," weekday risetimes can be mostly determined by "social clocks" competing with the biological clocks that can be solely responsible for weekend risetimes. However, weekday and/or weekend bedtimes cannot be always set by the biological clocks. Sometimes, they are determined by the additional conflict of "mental (mind) clocks" with the biological (body) clocks. This conflict can lead to weekday sleep insufficiency and can be, in particular, explained by such kind of conscious behavior as mind-bedtime procrastination.

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Sleep timing; sleep duration; two-process model; chronotype

Introduction

Under natural environmental conditions, the 24-h periodicity of the human sleep-wake cycle is established by interaction of the homeostatic drive for sleep with the circadian clocks (Borbély et al., 2016) that are entrained to the external light-dark cycle with a 24-h period (Pittendrigh and Daan 1976). Therefore, bed- and risetimes of human *ad lib* sleep are expected to be endogenously determined by two major – the circadian and the homeostatic – processes of sleep-wake regulation (Daan et al. 1984).

Under certain circumstances, these sleep times can be at odds with the times determined by these two endogenous processes. For example, the effects of challenging sleep times determined by the biological mechanisms were conceptualized as the so-called "social jetlag" (Wittmann et al. 2006) and "bedtime procrastination" (Kroese et al. 2014; Hill et al. 2024; or, in other terms, "mind-bedtime procrastination" Rubin 2020).

The term "social jetlag" was coined by Wittmann et al. (2006) in this journal paper 20 years ago. It refers to the situation of two conflicting "clocks," "social", and

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biological. Since daytime workers/learners are forced to arrive at their work/study place in the morning, they wake up on weekdays earlier instead of waking up at a later time determined by their internal (biological or body) clocks (Wittmann et al. 2006). If risetimes after weekend *ad lib* sleep are expected to be set by these biological (body) clocks, risetimes on the preceding and following weekdays seem to be set by the so-called "social clocks," i.e. by the typical demands of work-rest schedule of these daytime workers/learners. The duration of their weekday sleep is reduced after an advancing shift of weekday risetimes dictated by "social clocks" because the biological clocks fail to produce a similarly large advancing shift of weekday bedtimes (Wittmann et al. 2006).

Moreover, rise- and bedtimes can be at odds with the times determined by the endogenous processes of sleep-wake regulation due to voluntary prolongation of wakefulness on early night hours. Twelve years ago, Kroese et al. (2014) coined the term "bedtime procrastination" to introduce to new area of procrastination behaviors and habits. The behaviors and habits that are known under this term (Alshammari et al. 2023; An and Zhang 2024; Meng et al. 2024; Xu et al. 2024) have not been related so far to the concept of "social clocks" conflicting with the biological clocks (Wittmann et al. 2006). Therefore, the earlier proposed concept of two conflicting "clocks" does not take into account the possibility that such a procrastination can contribute to sleep timing and duration on weekdays and/or weekends in addition to the contribution of the biological and "social" clocks.

Since this procrastination is determined as "the volitional delay of sleep without external influences" (Hill et al. 2024), neither "social clocks" nor biological clocks can be blamed for this delay of bedtimes. Therefore, a question arises: when daytime workers/learners tend to intentionally prolong the wake phase of their sleep-wake cycle to early night hours, can this procrastination be interpreted as a conscious behavior conflicting with the endogenous mechanism of sleep-wake regulation? If the answer is "Yes, it can be," it is reasonable to suggest that the concept of just two conflicting "clocks," "social", and biological can require extension to account for an additional conflict of the biological clocks with the conscious and volitional behaviors, such as "mind-bedtime procrastination," for setting time of transition from the wake phase to the sleep phase of human sleep-wake cycle.

The rhythmic behaviors are best understood through mathematical tools and techniques (Forger 2017). Therefore, mathematical modeling enables to bridge the gaps in understanding observed responses of sleep and circadian rhythms to prolongation of wakefulness and to generate hypotheses about the mechanisms underlying these responses (Mallis et al. 2004; Postnova et al. 2012, 2014). Here, we applied this mathematical modeling methodology for testing whether sleep times reported by daytime learners point at the tendency to intentionally prolongate the wake phase of their sleep-wake cycle to early night hours by ignoring the evening bedtime signal from the endogenous mechanism of sleep-wake regulation. We enlarged the previously collected sample of university students (1582 in Putilov et al. 2025c) to demonstrate that, in order to simulate sleep times of some of these students, the accounting for the intentional prolongation of the wake phase of their sleep-wake cycle to early weekday night hours is required. This demonstration could provide support for the assumption that the bedtime set by the biological – homeostatic and circadian – processes of sleep-wake regulation was intentional missed by these students.

We hypothesized that the model-based simulations could predict the necessity of such an accounting for, at least, those students who reported later bedtime on learning (week)days than on free (weekend) days, i.e. the days of attending classes and the following days without classes, respectively. If our hypothesis is correct, a model allowing a voluntary delay of sleep could fit sleep times significantly better than a model without it, specifically in students reporting later weekday than weekend bedtimes.

Materials and Methods

Major Survey Participants and Subdivisions of the Sample

In winter season, university lecturers invited their students to respond to the questions concerning their sleep – wake habits and behavior. Their self-reported rise- and bedtimes on weekdays and weekends were collected using a Google Forms questionnaire from smartphones (Tables 1–3 and S1-S4). The specific inclusion/exclusion criteria were not applied, but health, mood, and sleep problems were addressed by administering four additional questions. The responses to these questions provided the possibility of rapid and user-friendly assessing these problems in the survey aimed on more detailed assessment of sleep – wake traits, abilities, habits, and behavior. The results of such self-assessment of health, mood, and sleep concerns are reported in Table S5.

In order to test the study hypothesis, the sample collected for simulations ($n = 4346$) was subdivided into 6 subsamples in accord with two weekday bedtimes

Table 1. Criteria for subdivision of the whole sample into 2, 3, and, finally, 6 subsamples.

Division				Weekday bedtime	
Weekend bedtime	Reported bedtime		Any	Earlier	Later
			Criterion for subdivision into subsamples		
Early	Bedtime	weekday	Any	<24:30	**≥24:30**
		weekend	<24:30	<24:30	**<24:30**
Middle	Bedtime	weekday	Any	<24:30	≥24:30
		weekend	≥24:30 but <1:30	≥24:30 but <1:30	≥24:30 but <1:30
Late	Bedtime	weekday	Any	<24:30	≥24:30
		weekend	≥1:30	≥1:30	≥1:30
Any	Bedtime	weekday	Any	<24:30	≥24:30
		weekend	Any	Any	Any

Notes: 1. The major sample including 4346 participants of online survey was subdivided into two subsamples. 1. The first subdivision separated students with earlier and later self-reported weekday bedtimes (<24:30 and ≥24:30, respectively). The rounded bedtime 24:30 was chosen because 53.7% and 46.3% (i.e. two approximately similar halves of the whole sample) reported earlier and later weekday bedtime, respectively. 2. Further subdivision separated students in accord with early, middle, and late self-reported weekend bedtime. The first borderline was the same as for weekday bedtimes, <24:30, and the next borderline was set at <1:30, i.e. two h later (<24:30, ≥24:30 but <1:30, and ≥1:30, 39.0%, 13.1%, and 47.9%, respectively). 3. By combining these two criteria, the whole sample was finally subdivided into 6 subsamples characterized by the different combinations of weekday bedtime (Earlier/Later) with weekend bedtime (Early, Middle, Late): Earlier/Early, Earlier/Middle, Earlier/Late, Later/Early, Later/Middle, and Later/Late weekday/weekend bedtime. Printed in bold: As a result of such subdivision into 6 subsamples, one of these subsamples (later/early weekday/weekend bedtime) includes those students who reported that their weekday bedtime occurred later than weekend bedtime. Sleep times in 6 subdivisions were pairwise compared (Table 2) following three-way ANOVAs (Table S1).

and three weekend bedtimes (2 × 3 = 6). See the notes to Tables 1–3, and S1 for explanation of the criteria applied for the divisions into subsamples with (1) earlier and later weekday bedtimes (two primary subdivisions), (2) early, middle, and late weekend bedtimes (three following subdivisions), and (3) earlier/early, earlier/middle, earlier/late, later/early, later/middle, and later/late weekday/weekend bedtimes (2 × 3 = 6 final subdivisions).

Such subdivisions of the sample provided the possibility to examine whether sleep times reported by students from, at least, one of 6 subsamples (namely, with later/early weekday/weekend bedtimes) can be explained by prolongation of wakefulness on weekdays rather than on weekends beyond the bedtime determined by the biological sleep-wake regulating mechanism (Table 1).

Statistical Analysis

The $SPSS_{26.0}$ statistical software package (IBM, Armonk, NY, USA) was used for testing significance

Table 2. Pairwise *post hoc* comparisons of the differences in sleep times.

	Weekday bedtime			Earlier			Later	
	Weekend bedtime		Early	Middle	Late	Early	Middle	Late
Sleep	n = 4346		1349	360	535	323	479	1300
time	% = 100		31.0	8.3	12.3	7.4	11.0	29.9
Bedtime	Weekday	Weekend			Bedtime on weekends			
on	Earlier	Early	–	−1.65***	−3.11***	0.13	−1.67***	−3.65***
weekdays		Middle	−0.65***	–	−1.46***	−1.78***	−0.01	−2.00***
		Late	−0.68***	−0.04	–	−3.24***	−1.44***	−0.54***
	Later	Early	−2.85***	−2.20***	−2.17***	–	−1.80***	−3.78***
		Middle	−2.49***	−1.84***	−1.80***	0.36	–	−1.99***
		Late	−3.07***	−2.42***	−2.38***	−0.21**	−0.58***	–
Risetime					Risetime on weekends			
on	Earlier	Early	–	−0.74***	−1.66***	−0.71***	−0.97***	−1.98***
weekdays		Middle	−0.17	–	−0.92***	−0.04	−0.22	−1.24***
		Late	−0.16	0.02	–	−0.96***	−0.69***	−0.32**
	Later	Early	−0.08	0.09	0.08	–	−0.26	−1.28***
		Middle	−0.29**	−0.12	−0.14	−0.21	–	−1.01***
		Late	−0.58***	−0.41***	−0.43***	−0.50***	−0.29**	–
Time					Time in bed on weekends			
in	Earlier	Early	–	0.89***	1.42***	−0.85***	0.68***	1.65***
bed		Middle	0.46***	–	0.53***	1.74***	−0.21	0.76***
on		Late	0.51***	0.05	–	2.28***	0.75***	0.22
weekdays	Later	Early	2.75***	2.30***	2.24***	–	1.53***	2.50***
		Middle	2.18***	1.72***	1.67***	−0.58***	–	0.97***
		Late	2.46***	2.01***	1.95***	−0.29*	0.29*	–

Notes: The major sample consisting of 4346 participants of online survey was subdivided into two subsamples in accord with their weekday bedtime (<24:30 and ≥24:30) and further subdivided into three sub-samples in accord with their weekend bedtime (<24:30, ≥24:30 but <1:30, and ≥1:30). These subdivisions gave 6 subsamples in total. Significance of the differences between these 6 subsamples in sleep times, h (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$), was tested using *post-hoc* comparisons following two-way ANOVAs (Table S1, right column); *n* = and % =: Number and percentage of students included in each of 6 subsamples. See also Table 3 for the estimates of compared sleep times.

Table 3. Sleep times in the whole sample and subsamples.

Division Weekend bedtime	Reported sleep time		Any Mean	Any SEM	Weekday bedtime Earlier Mean	Earlier SEM	Later Mean	Later SEM
Early	Bedtime	weekday	23.34	0.04	22.80	0.03	**1.75**	**0.07**
		weekend	23.22	0.03	23.25	0.03	**23.11**	**0.08**
	Risetime	weekday	6.77	0.03	6.74	0.04	7.00	0.08
		weekend	9.25	0.04	9.12	0.05	9.76	0.11
	Time in bed	weekday	7.41	0.05	7.91	0.04	5.25	0.10
		weekend	10.00	0.05	9.85	0.05	10.65	0.12
Middle	Bedtime	weekday	24.14	0.06	23.45	0.05	1.32	0.07
		weekend	24.93	0.04	24.93	0.05	24.92	0.07
	Risetime	weekday	6.95	0.05	6.90	0.06	7.04	0.08
		weekend	9.94	0.06	9.84	0.08	10.12	0.10
	Time in bed	weekday	6.81	0.07	7.45	0.07	5.72	0.10
		weekend	9.01	0.07	8.91	0.08	9.19	0.11
Late	Bedtime	weekday	1.28	0.04	23.49	0.05	1.94	0.03
		weekend	2.83	0.03	2.44	0.05	2.97	0.03
	Risetime	weekday	7.21	0.03	6.90	0.06	7.32	0.04
		weekend	11.07	0.04	10.82	0.08	11.16	0.05
	Time in bed	weekday	5.93	0.05	7.40	0.07	5.39	0.04
		weekend	8.25	0.04	8.39	0.08	8.19	0.05
Any	Bedtime	weekday	24.46	0.02	23.25	0.03	1.67	0.03
		weekend	24.94	0.02	24.87	0.03	1.00	0.04
	Risetime	weekday	6.98	0.03	6.84	0.03	7.12	0.04
		weekend	10.14	0.03	9.93	0.04	10.35	0.05
	Time in bed	weekday	6.52	0.03	7.59	0.04	5.45	0.05
		weekend	9.20	0.04	9.05	0.04	9.35	0.06

Notes: Estimates obtained by ANOVAs (see Table S1). Sleep times with SEM (Standard Error of Mean) were calculated for the whole sample (any weekday bedtime and any weekend bedtime), the subsamples with two weekday bedtimes and any weekend bedtime, the subsamples with three weekend bedtimes and any weekday bedtime, and the subsamples with the combination of two weekday bedtimes (earlier and later) with three weekend bedtimes (early, middle, and late). The times for 6 subsamples (Earlier/Early, Earlier/Middle, Earlier/Late, Later/Early, Later/Middle, and Later/Late week-day/weekend bedtime) were simulated (Table 4) and illustrated in Figures 2–4. Printed in **bold**: Students included in one of 6 subsamples (later/early weekday/weekend bedtime) reported that their weekday bedtime occurred later than weekend bedtime. See also the deviations of subsample-averaged sleep times from the sample-averaged sleep times in Figure 1.

of differences between the bedtime-based subdivisions of the sample in risetimes and times in bed. Significance of the main effects of bedtime factors was tested with two-way and three-way ANOVAs. The bedtime factors were "Weekday bedtime" and "Weekend bedtime" in three-way ANOVAs and "Bedtimes" in two-way ANOVAs (earlier and later and early, middle and late, respectively, and the combination of these two weekday bedtimes with these three weekend bedtimes, i.e. earlier/early, earlier/middle, earlier/late, later/early, later/middle, and later/late weekday/weekend bedtime). The additional independent factor was "Gender" (male and female students). These ANOVAs provided the estimates of the subsample-averaged risetimes and times in bed for earlier and later weekday bedtime, for early, middle and late weekend bedtime, and, finally, for earlier/early, earlier/middle, earlier/late, later/early, later/middle, and later/late weekday/weekend bedtime (Tables 2, 3 and S1). *Post hoc* pairwise Bonferroni comparisons were used to confirm significance of differences between 6 subsamples in risetimes and times in bed (Table 2).

Figure 1 illustrates sleep times in the subsamples expressed as the deviation of the subsample-averaged values from the sample-averaged value.

Model of the Endogenous Sleep-Wake Regulator

To simulate sleep times, one of variants (Putilov 1995) of the two-process model of sleep-wake regulation (Daan et al. 1984) was applied. This variant describes the time course of the homeostatic process in the same mathematical terms as were used for its description in the classical variant of the two-process model (Daan et al. 1984). To maintain sleep-wake homeostasis, the process regulates daily sleep amount/depth through the increase (during waking) and decrease (during sleep) of sleep pressure. Instead of postulating a separate circadian process, as it was proposed by Daan et al. (1984), this version of the model posits that the circadian clocks simply modulate all parameters of the "classical" homeostatic process. In the present and previous simulations (e.g. Putilov 1995, 2023; Putilov and Verevkin 2024), this modulation was incorporated in the model in the form of simplest periodic (sine) function with a circadian period.

If $t1$ and $t2$ are the initial times for the buildup and decay phases of this process of sleep-wake regulation $S(t)$, e.g. for rise- and bedtimes in the present simulations, this process can be described as follows:

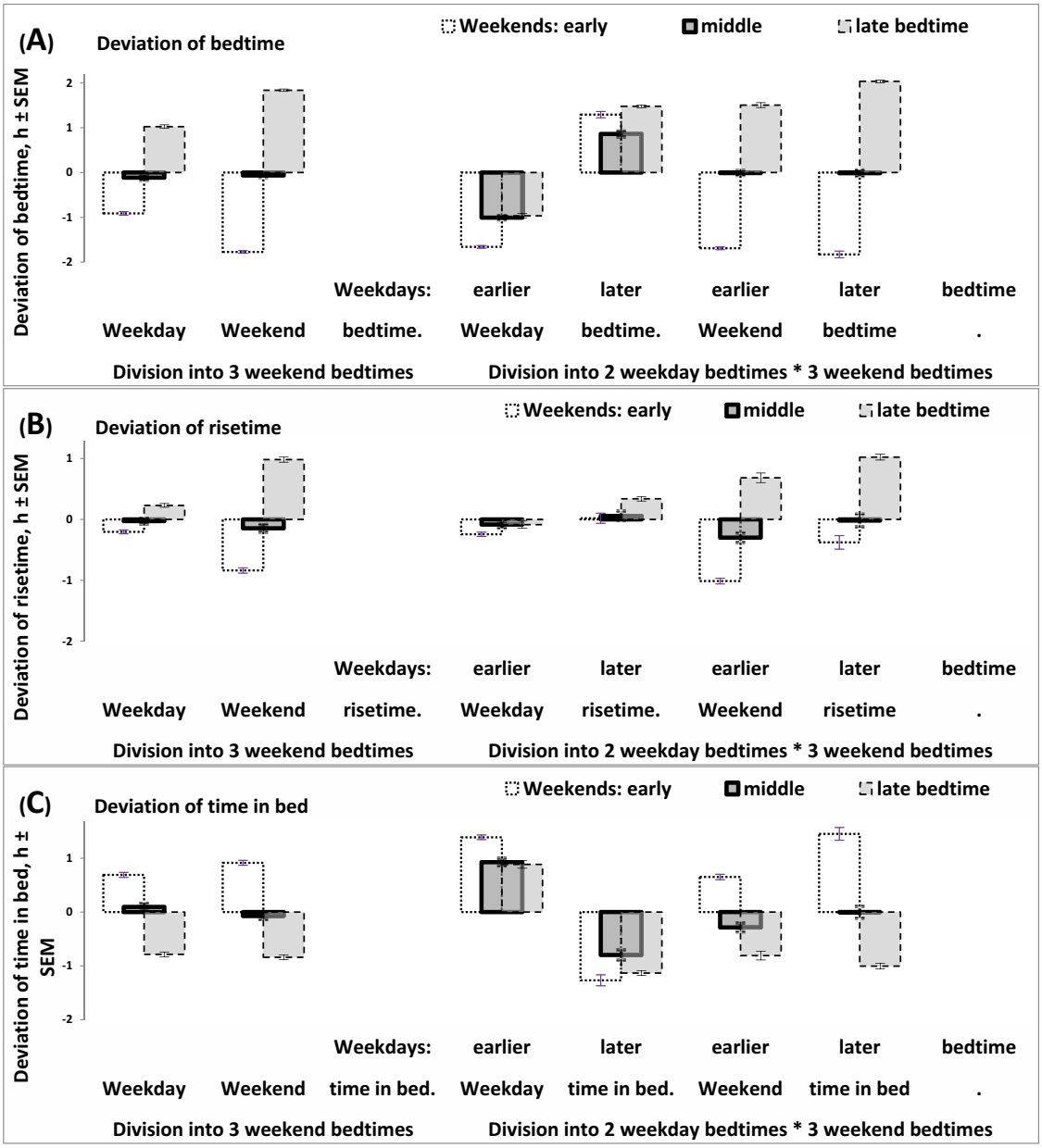

Figure 1. Sleep times on weekdays and weekends. (A) Bedtimes, (B) Risetimes, and (C) Times in bed expressed as deviations from the sample-averaged time. Left: subdivision in accord with three weekend bedtimes. Middle and right: one more subdivision – into two weekday bedtimes – gives 6 subsamples. Sleep times are reported in Table 3 and the results of statistical comparison of sleep times in 6 subsamples are reported in Tables 2 and S1.

$$S(t) = [S_u + C(t)] - \{[S_u + C(t)] - S_b\} * e^{-\frac{t-t1}{[Tb-k*C(t)]}} \quad (1a)$$

$$S(t) = [S_l + C(t)] - \{S_d - [S_l + C(t)]\} * e^{-\frac{t-t2}{[Td-k*C(t)]}} \quad (1b)$$

where

$$C(t) = A * sin(2\pi * t/\tau + \varphi_0) \quad (2)$$

is a sine function with a circadian period τ.

In other words, this function (2) describes the modulation of all parameters of the homeostatic process by the circadian clocks because it is expected that any process of the organism, including this particular homeostatic process, can be accelerated and decelerated at different times of the day due to the functioning of the circadian clocks in this organism (Putilov 1995, 2023).

In the present and previous simulations, τ was assigned to 24.0 h because it is reasonable to expect that the circadian clocks of the survey participants

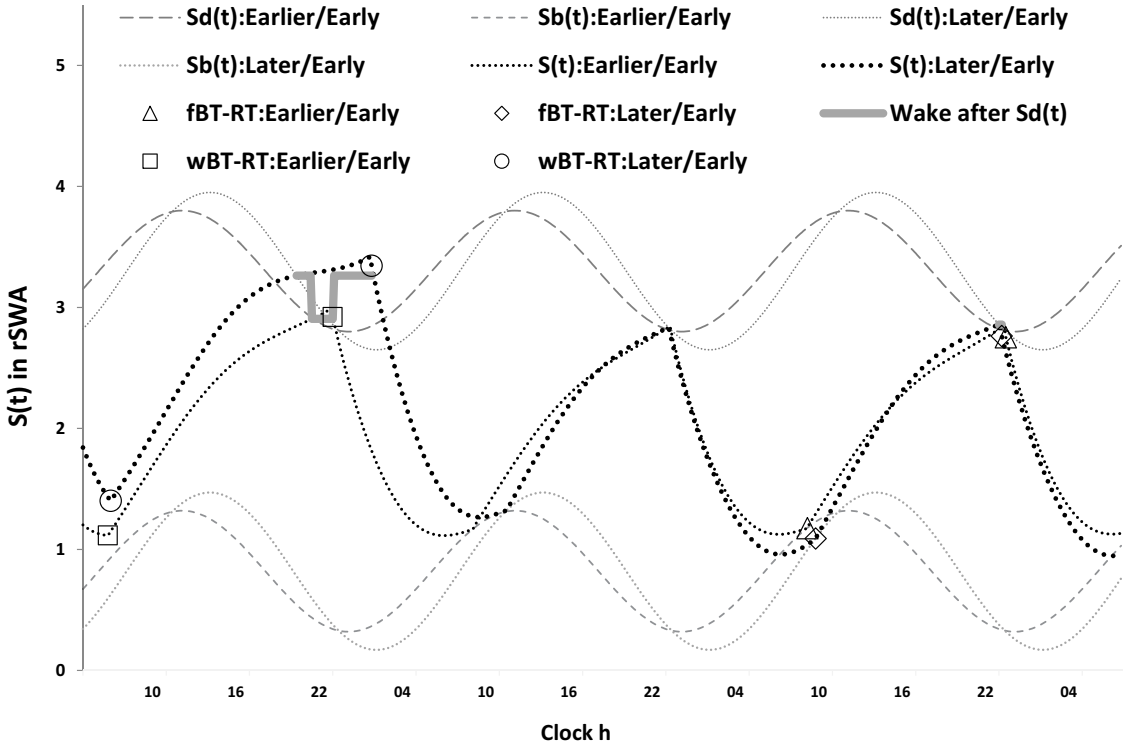

Figure 2. Simulations of three sleep-wake cycles in subsamples with early weekend bedtime. Simulations of the sleep-wake cycles are shown for the 3-day interval (between 5:00 on Friday and 5:00 of Monday).$S_d(t)$and$S_b(t)$: The highest buildup and the lowest decay of$S(t)$, i.e. bedtime and risetime, respectively, determined by the endogenous sleep-wake regulating process$S(t)$(1,2). It is simulated in rSWA (see the model parameters in Table 4 and Methods). Due to the circadian modulation$C(t)$of the parameters of$S(t)$, the endogenously determined bed- and risetimes (shown for Sunday) are expected to be restored during just one day with*ad lib*sleep (between Friday and Saturday). Wake after S_d: Delay of bedtime after reaching S_d(the bedtime set by the sleep-wake regulating mechanism); S_b: Risetime set by this mechanism on weekdays and weekends (however, risetime on weekdays occurs earlier because it is determined by "social clocks"); fBT-RT: Bed- and risetimes on weekends; wBT-RT: Bed- and risetimes on weekdays. See Table S3 for these simulated sleep times and Table 4 for the final parameters of the model.

were always controlled by (i.e. are entrained to) the external light-dark cycle with the exactly 24.0-h period.

2.4. Parameters of the Model

The parameters of this model were initially derived in Putilov (1995) from data on (1) the durations of recovery sleep after 6 gradually increasing intervals of extended wakefulness (Åkerstedt and Gillberg 1981) and (2) the levels of SWA in 10 naps (Dijk et al. 1987) and two recovery sleep episodes (Dijk et al. 1990, 1991). Notably, in the classical publication of the two-process model (Daan et al. 1984), the same data on recovery sleep (Åkerstedt and Gillberg 1981) were also simulated using this simplest (sine) function despite incorporation of a much more complex function for the circadian process in this model (Daan et al. 1984). The simulation of experimental data on SWA in Putilov (1995) provided the possibility to utilize the experimental measurements of relative slow-wave activity (rSWA) for describing $S(t)$ in simulations shown in Figures 2–4.

The initial parameters of the model (Putilov 1995) were slightly modified in the previous (Putilov and Verevkin 2018, 2024; Putilov 2023) and present simulations (Figures 2–4 and Tables 4, S2 and S3) to account for the difference between the initially simulated experimental sleep durations taken from data of the Åkerstedt and Gillberg study (Putilov 1995) and times in bed calculated from self-reported bed- and risetimes on weekdays and weekends (i.e. sleep duration is shorter than time in bed; Putilov and Verevkin 2018, 2024; Putilov 2023).

When this model was previously applied for prediction and simulation of bed- and risetimes on weekday and weekends (Putilov and Verevkin 2018, 2024; Putilov 2023), these simulations did not take into account the possible contribution of "mind-bedtime procrastination" to bedtimes. However, it was more recently found (Putilov et al. 2025c) that, in simulations of sleep times of some of university students, such kind of conscious behaviors might be necessary to account

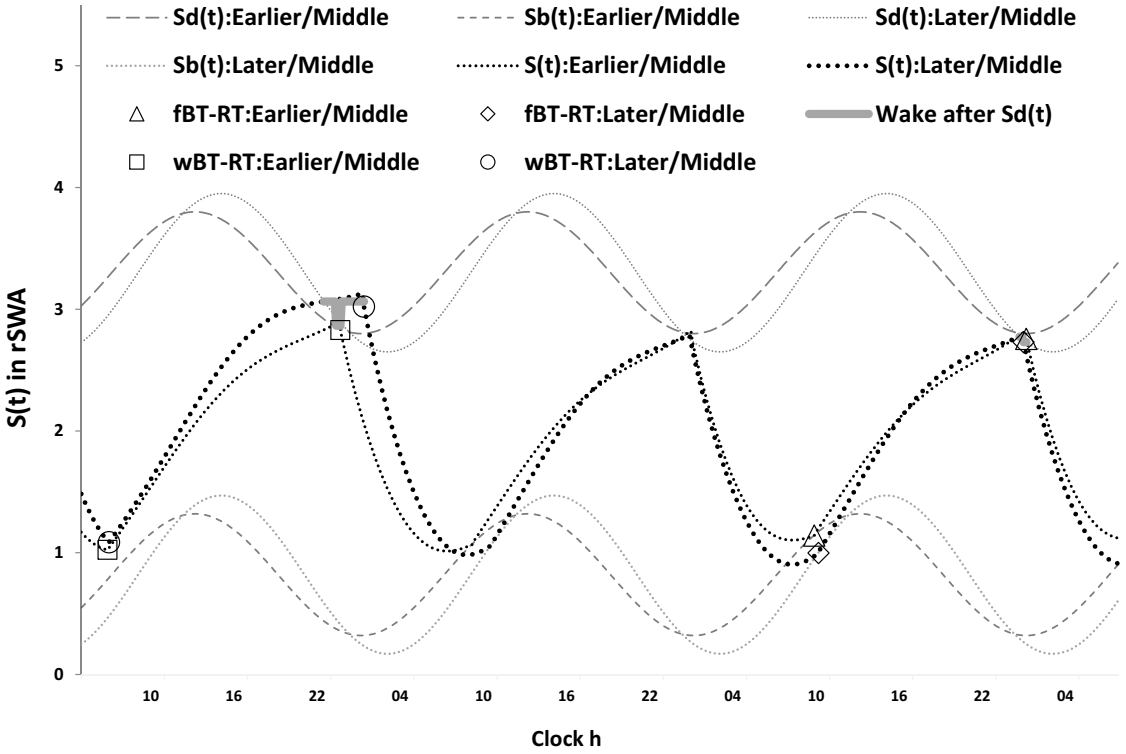

Figure 3. Simulations of three sleep-wake cycles in subsamples with middle weekend bedtime. See the legend to Figure 2.

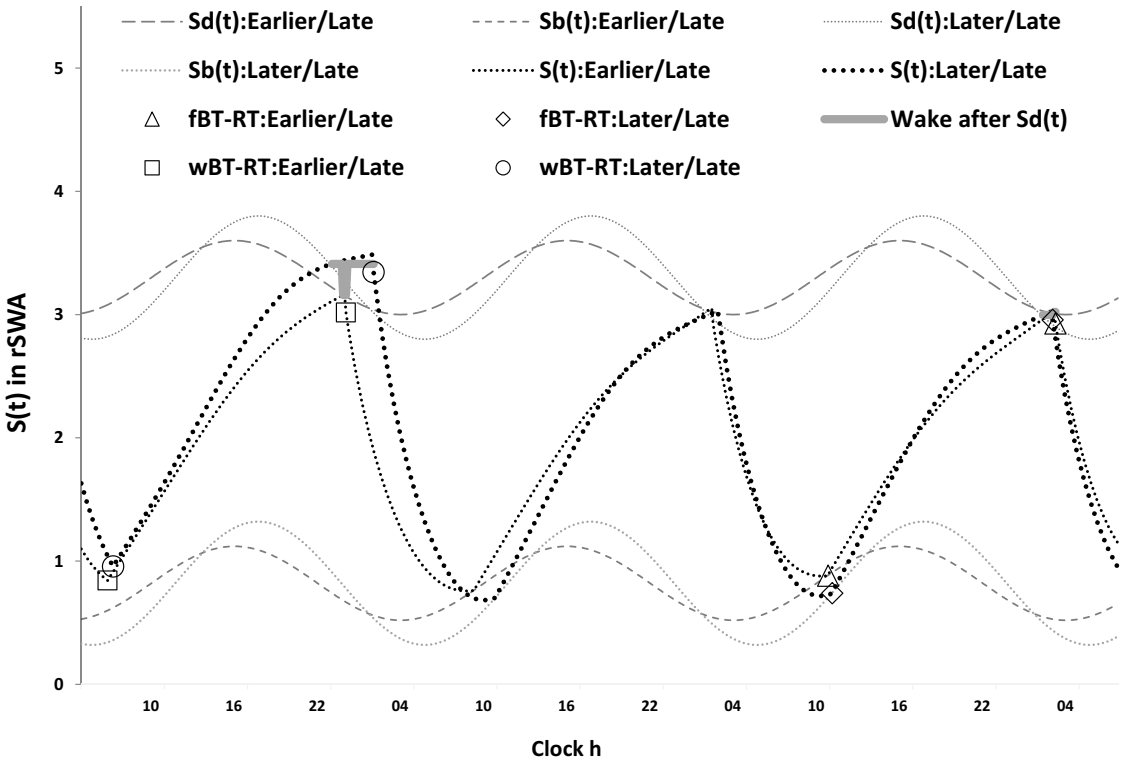

Figure 4. Simulations of three sleep-wake cycles in subsamples with late weekend bedtime. See the legend to Figure 2.

for. These were students reporting the disagreed trait and state assessments of morningness-eveningness, i.e. lark and owl types with late and early weekend sleep timing, respectively (141 of 1582 students of the whole sample). For example, up to one third of all lark type students can be practicing late weekend sleep timing, possibly, due to the attempts to remain in synch with their peers who, being mostly owl types, prefer to go to sleep later (Putilov et al. 2025c).

Preliminary and Final Simulations

The present simulations of sleep times (Tables 4, S2, and S3) were performed using the least-squares method. We compared 9 pairs of empirical and simulated sleep times (bed- and risetimes and times in bed of weekdays and weekends) and weekday-weekend gaps in these sleep times (in bed- and risetimes and in times in bed) to determine the maximal of 9 deviations of a simulated value from an empirical value (see the bottom line and notes to Tables 4 and S2,). The obtained fit was considered as being good when this maximal of 9 deviations was reduced during fitting sleep times to 0.1-h or shorter time interval (i.e. <6 min). This 6 min is the length of time interval between two adjacent points of the simulated curves of $S(t)$ shown in Figures 2–4. The deviation larger than 0.3 h (>18 min) was interpreted as a failure to simulate empirically obtained sleep times.

The comparison of the final and preliminary simulations is provided in Table 4 and Table S2, respectively. These preliminary and final simulations were obtained with and without inclusion in the model of an assumption that bedtime delays beyond the bedtime set by the biological clocks (either Table S2 or Table 4, respectively). Namely, the comparison of results of preliminary and final simulations was aimed at demonstrating that, when this assumption was not included (Table S2), sleep times calculated for, at least, one of 6 subdivisions of the sample (later/early subsample) cannot be explained because the sleep times reported by students from this subsample cannot be well-fitted with the model (e.g. the deviation is larger than 0.3 h; see the bottom line of Table S2).

Table 4. Final parameters of the model of sleep-wake regulation.

Weekend bedtime		Early		Middle		Late	
Weekday bedtime		Earlier	Later	Earlier	Later	Earlier	Later
Inverse exponential buildup (1a) and exponential decay phases (1b) of the sleep-wake regulating process *S(t)*	S_b (lowest decay), rSWA	0.82	0.82	0.82	0.82	0.82	0.82
	S_d (highest buildup), rSWA	3.30	3.30	3.30	3.30	3.30	3.30
	S_l (lower asymptote), rSWA	0.70	0.70	0.70	0.70	0.70	0.70
	S_u (upper asymptote), rSWA	6.25	6.25	6.25	6.25	6.25	6.25
	T_d (time constant for decay), h	30.29	27.26	32.66	29.78	29.07	26.09
	T_b (time constant for buildup), h	4.35	4.39	3.92	3.67	2.97	2.56
24-h sine wave-form modulation (2) of parameters of the buildup (1a) and decay phases (1b) of *S(t)*	φ_{max} (circadian peak), clock h	12.14	14.15	13.19	15.10	16.06	17.78
	A (circadian amplitude), rSWA	0.50	0.65	0.50	0.65	0.30	0.50
	τ (circadian period), h	24.0	24.0	24.0	24.0	24.0	24.0
	k (circadian impact: twofold)	2.00	2.00	2.00	2.00	2.00	2.00
Initial times for the buildup (1a) and decay phases (1b) of *S(t)*	*t1*, clock h, weekend risetime	9.1	9.8	8.9	9.2	10.8	11.1
	t2, clock h, weekend bedtime	23.3	23.2	24.9	24.9	2.4	2.9
Prolongation of the buildup	+ h on weekday	1.30	5.30	0.40	2.70	0.00	3.00
phase beyond *t2*	+ h on weekend	0.30	0.00	0.20	0.20	0.20	0.70
Maximal of deviations of simulated time from reported time, h		0.06	0.09	0.02	0.04	0.02	0.03

Note: Parameters of the model-based simulations of time course of the process of sleep-wake regulation $S(t)$. In the absence of the circadian clocks, this process can be simply described as daily alternations of an inverse exponential buildup (1a) and an exponential decay (1b) during the wake and sleep phases of the sleep-wake cycle, respectively. The circadian influence was proposed ($A > 0$) in the form of sine function with 24-h period, $C(t)$ (2), because the circadian clocks modulate all parameters of $S(t)$ (1a and 1b). $S_d(t)$ and $S_d(t)$ in (1b) and (1a), respectively: the highest allowed buildup and the lowest allowed decay of $S(t)$, i.e. bed- and risetimes, respectively, determined by this endogenous sleep-wake regulator (1,2). The initial parameters of the model of sleep-wake regulating process $S(t)$ were derived in Putilov (1995) by simulating data on sleep durations after several later bedtimes and relative Slow Wave Activity (rSWA) in naps and two extended sleep episodes (mean rSWA = 1 in a baseline night sleep episode). These initial parameters of the model were slightly modified to account for the difference between the initially simulated experimental sleep durations and time in bed reported in questionnaire studies. The present final simulations differ from the previous and preliminary simulations of sleep times by accounting for a prolongation of the buildup phase behind *t*2 (+h). In these final simulations, variation in several parameters was not allowed to clarify the differences between subsamples in the circadian phase, φ_{max}, and +h. Maximal of deviations of simulated time from reported time, h: The largest of 9 differences between simulated and reported times (time in bed and bed- and risetimes on weekdays and weekends, and weekend-weekday gaps in time in bed and bed- and risetimes). Clock time is given in decimal hours.

Since a good agreement between empirical and simulated sleep times was obtained after the accounting for this assumption in the model applied for the final simulations (less than 0.1-h or <6 min), one more assumption was added. We proposed that some of the parameters can be viewed as being identical for all 6 subdivisions of the sample (Table 4). These parameters included S_d, S_b, S_u, and S_l that were previously derived from data on SWA and, therefore, they cannot be directly calculated in the present study. Such an assumption of fixed parameters provided the possibility to clarify the contribution of just few remaining parameters to the difference between subsamples. These remaining parameters include the circadian phase φ that also cannot be directly calculated from the present empirical data on sleep times.

Additional Survey of Perceived Times of Readiness to Fall Asleep

The final simulations suggested, in particular, that 6 subsamples differ in delays of bedtime beyond the bedtime set by the biological clocks (Table 4). To empirically confirm the existence of such differences, an additional sample was collected in February 2026. It was analyzed to explore the capability to percept such differences in delays by comparing answers to two additional questions about clock times when a survey participant starts to feel on weekdays and weekends that his/her body is already ready to sleep. These additional clock times were subtracted from reported bedtime to provide the estimate of perceived prolongation of wakefulness beyond the time for its desired termination determined by the biological mechanism of sleep-wake regulation (Table S4). Using this additional sample of 1029 university students, we tested whether the differences between 6 subsamples of the major sample in simulated delays (Table 4) resemble the differences between 6 new similar subsamples in the perceived delay of reported bedtime relative to the time of readiness of the body to fall asleep (Table S4).

Results

Sleep Times in One of 6 Subsamples Pointed at a Tendency to Self-Deprive Sleep

Mean ages ± standard deviation of 1271 male and 3075 female students of the major survey were 19.2 ± 2.0 years and 19.4 ± 1.7 years, respectively. In the additional survey, mean ages ± standard deviation of 280 male and 749 female students were 20.2 ± 2.1 years and 20.4 ± 3.0 years, respectively.

Rather unusual behavior was expected for one of these 6 subdivisions based on reported weekday and weekend bedtimes: with later/early weekday/weekend bedtime. It included 323 students (7.4% of the whole sample) reported delayed bedtime on weekdays rather than on weekends. As a result, the weekday bedtime in this smallest in size subsample delayed relative to weekend bedtime (Tables 1–3 and S3). Despite this delay, the students from this subdivision resembled students from 5 other subdivisions by reporting, as expected, advanced risetimes on weekdays relative to risetimes on weekends (Tables 3 and S3).

The most plausible explanation of such a large delay of weekday bedtime reported by these students seems to be the tendency to self-deprive sleep on weekdays rather than on weekends by voluntary prolongation of weekday wakefulness beyond the bedtime determined by the biological mechanism of sleep-wake regulation. At least partly, this unusual pattern of weekday and weekend sleep timing can be explained by practicing behaviors known under the terms “mind-bedtime procrastination” or, in short, “bedtime procrastination.”

Effect of a Tendency to Self-Deprive Sleep on Other Sleep Times

As shown in Table 2, *post hoc* pairwise comparisons suggested the significant difference in weekend bedtimes between the subsamples with earlier/early and later/late weekday/weekend bedtimes. It was equal to −3.65 h (Table 2, upper part), while, compared to this difference, the difference between these subsamples in weekend risetimes was much smaller (−1.98 h; Table 2, middle part).

Such a large difference in bedtime relative to difference in risetime can be also explained by delaying bedtime relative to the bedtime set by the biological mechanism of sleep-wake regulation.

The remaining difference, that is the difference between bedtimes minus the difference between risetimes (e.g. 3.65 h-1.98 h; Table 2, upper and middle part), is fully explained by the difference in weekend time in bed (e.g. 3.65 h-1.98 h ~1.65 h; Table 2, lower part). The tendency to regularly self-deprive sleep during weekdays’ evening and early night hours (Table 2, lower part) led to the dramatic reduction of time in bed (<6 h, see Table 3).

Accounting for the Effect of Bedtime Delays in Simulations

Simulations of sleep times (Tables 4, S2, and S3) were performed to determine whether a model of the

processes of sleep-wake regulation requires an additional term that can provide an explanation of the volitional delays of bedtime on weekdays and/or weekends (i.e. the delays that can occur without any external influences, e.g. "social clocks," by simply ignoring the autonomous influences from the biological clocks). Preliminary simulations were performed with an assumption that the time of transition between wakefulness and sleep is determined exclusively by the two (homeostatic and circadian) processes of sleep-wake regulation. In mathematical terms, these preliminary simulations postulated that bedtime can occur exactly at $t2$ that is the time of reaching the highest buildup, S_d, of the sleep-wake regulating process $S(t)$. No wakefulness after this time can be predicted in these preliminary simulations. As expected, the model, in general, was capable of accurately simulating sleep times in 5 of 6 subdivisions of the whole sample. The maximal deviation of simulated values from empirical values was not larger than 0.3 h. As expected, the exception was the simulation of sleep times of students from the subsample with later/early weekday/weekend bedtime (e.g. when bedtimes were reported to occur later on weekdays than weekends). Table S2 (the last line) shows that the reported sleep times were not accurately fitted in this subdivision characterized by later/early weekday/weekend bedtime. The deviation of simulated values from empirical values was larger than one hour that we interpreted as a failure to simulate these empirical times. Indeed, the model predicts that, inevitably, weekday risetimes set by "social clocks" always occur earlier than *ad lib* weekend risetimes set by the biological clocks and, in turn, the earlier weekday than weekend risetime leads to an earlier weekday than weekend bedtime. Consequently, when weekend bedtimes are exclusively set by the internal biological mechanism of sleep-wake regulation, they cannot be advanced relative weekday bedtimes. When students from the later/early weekday/weekend bedtime subdivision reported later bedtimes on weekdays than weekends, they revealed their proneness to self-deprive sleep on weekdays, particularly, due to practicing such conscious behaviors as "mind-bedtime procrastination."

Bedtime Delays Can Lead to the Circadian Phase Delays

Unlike the preliminary simulations, the final simulations were performed by allowing a non-zero prolongation of wakefulness on weekdays and weekends in, at least, some of 6 subsamples. In other words, it was postulated that bedtime can occur not exactly at $t2$ that is the time of the highest buildup, S_d, of the sleep-wake regulating process, S, but it can delay on +h (i.e. >0.0 h). Particularly, this delay measured as +h was found to be relatively large in the subsamples with later weekday bedtimes (i.e. later/early, later/middle and later/late weekday/weekend bedtime).

The simulations also suggested that a long prolongation of wakefulness can lead to a delay of the circadian phase. Such a delay can be provoked by the additional exposure to artificial lighting sources in early night hours. In the case of subsamples with later weekday bedtime, the difference for subsamples with earlier weekday bedtime in the circadian phase (φ_{max}) did not exceed two h, i.e. it was smaller than the difference between them in the extension of wakefulness, h (Table 4).

Bedtime Delays Can Lead to the Weekday Sleep Insufficiency

Consequently, time in bed on weekdays was considerably reduced in subsamples with later weekday bedtime compared to the subsamples with earlier weekday bedtime (Table 2).

The model predicted and simulations of sleep times confirmed that time in bed on weekends was shorter after initiation and termination of sleep at a later circadian phase. In particular, weekend times in bed in the later/late subsample were shorter than in the earlier/late and any of 4 other subsamples (Table 4 and Figures 1–4).

Since weekday risetime is controlled by "social clocks," students from the later/late subsample were unable to accordingly shift their weekday risetime on later hours. As a result, the duration of weekday sleep after late weekend bedtimes was short and such sleep reduction was not fully compensated by a bit earlier bedtime on weekdays in this subsample (Tables 3 and S3, and Figures 1–5).

Ad Lib Sleep Timing and Duration in Later and Earlier Weekend Sleepers

The model-based simulations confirmed that weekend sleep reported by the vast majority of survey participants can be viewed as *ad lib* sleep. Consequently, such a sleep in the simulations was almost always terminated at the clock time set by the internal sleep-wake regulating mechanism (Figures 2 and 3). Therefore, although the model predicted a somewhat shorter duration of weekend *ad lib* sleep after later bedtimes compared to that after earlier bedtimes, this reduction cannot be interpreted as indicating sleep insufficiency (Figure 5

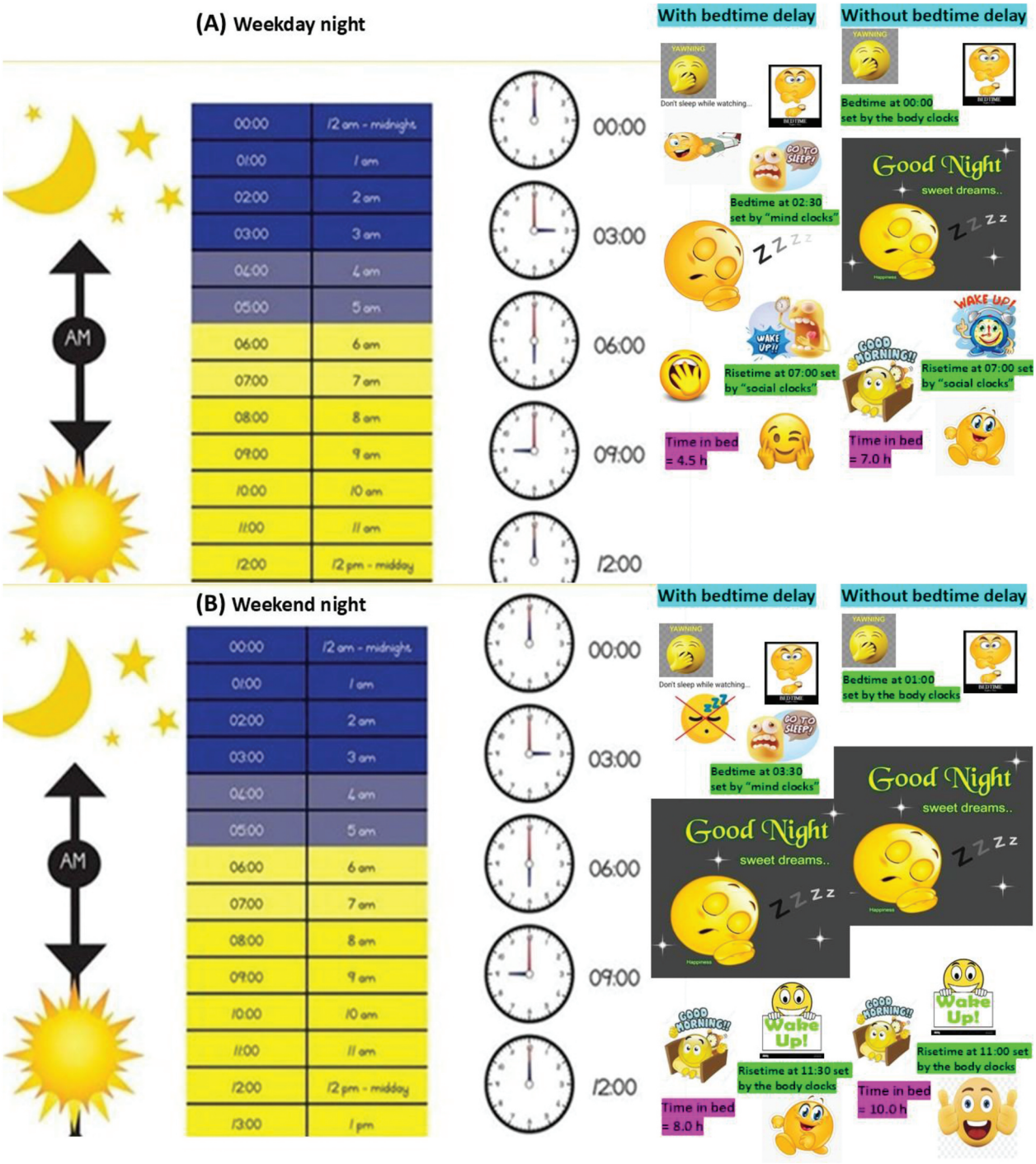

Figure 5. Hypothetic examples of the effects of bedtime delay on weekday and weekend sleep. Sleep times of two hypothetical individuals with and without delay of bedtime. (A) and (B) Delay is practicing on weekdays and weekends, respectively. (A) Delay of bedtime practicing by one of these two individuals leads to a prolongation of the light phase of the 24-h light-dark cycle on night hours. This prolongation causes a delay of the circadian phase that, in turn, causes a delay of bedtime signal from the body (biological) clocks. A habit known under the terms "bedtime procrastination" and "mind-bedtime procrastination" might be one of most plausible causes of the conflict of "mind (mental) clocks" with the body clocks. It leads to the additional delay of bedtime relative to the bedtime set by the body clocks. As a result, one of the individuals is suffering from weekday sleep insufficiency (time in bed < 6.0 h). (B) Unlike weekday risetime that is set by "social clocks," weekend risetime after*ad libs*leep during night between Friday and Saturday is set by the body clocks. Therefore, risetime occurs later on weekends (B) than on the previous weekdays (A). As a consequence of this later risetime, weekend bedtime also occurs later than weekday bedtime. Sleep initiated at a later phase (during early night hours) is shorter than sleep initiated at an earlier phase due to the modulating influence of the body clocks on the sleep homeostatic process. Despite a profound difference in weekend time in bed between these two individuals, none of them suffers from sleep insufficiency on weekends (time in bed > 6.0 h in both).

and see Supplementary Comments for more details on sleep times illustrated in Figure 5).

Surprisingly, the simulations suggested a larger delaying shift (+h) on weekdays in subdivisions with early weekend bedtimes as compared to the subdivisions with late weekend bedtimes (i.e. earlier/early and later/early subsamples vs. earlier/late and later/late subsamples). It can be speculated that some of these "early birds" can go to sleep relatively late because, for instance, they can try to remain in synch with their peers who mostly prefer late sleep timing.

As for the difference between subsamples in delaying shift (+h) on weekends, they supported a common opinion that, since evening types preferred to go to bed later, they more often than morning types prolongated weekend wakefulness to early night during free days. As a result, they seemed to more often than morning types ignored the bedtime signal sent by their biological mechanism of sleep-wake regulation on these days. This behavior led to further increase in the difference in bedtime between earlier and later weekend sleepers. However, sleep duration was shorter in later sleepers because, as predicted by the model-based simulations, such sleep reduction is a consequence of initiation of sleep at a later circadian phase (Table 4 and Figures 2–5).

Comparison of Perceived Times of Readiness to Fall Asleep

The final simulations suggested the following differences between subsamples in bedtime delays beyond the bedtime set by the biological clocks (Table 4). 1. These delays were not much smaller for weekdays than for weekends. 2. The delays were larger for three subsamples with later weekday bedtimes than for three subsamples with earlier weekday bedtimes. 3. The subsample combining later weekday bedtimes with early weekend bedtimes showed the largest delay. Table S4 includes the results of the additional survey that confirmed such differences as indicated by the estimates of gaps between reported bedtime and time when survey participants start to feel like their bodies are ready to sleep. Namely, these gaps in bedtime were found to be (1) very similar for weekdays and weekends, (2) larger in three subsamples with later weekday bedtimes than in three subsamples with earlier weekday bedtimes, and (3) the subsample with later weekday bedtimes and early weekend bedtimes showed the largest gap (Table S4). Again, this was the smallest of 6 subsamples (8.5%; see the last line of Table S4).

Association of Perceived Times of Readiness to Fall Asleep with Time in Bed and Health

Moreover, larger weekday and weekend gaps were significantly associated with a larger reduction of weekday sleep. For instance, the gaps negatively correlated with weekday time in bed (Spearman's rho attained the values of −0.323 and −0.295, respectively, $p < 0.001$ for both). Consequently, longer gaps were associated with health, mood, and sleep problems (Tables S6 and S7).

Discussion

The Hypothetical Additional "Clocks" That Contribute to Bedtime Delays

The present results suggested it is possible to extend the concept of conflicting biological and "social clocks" (Wittmann et al. 2006) by linking it to the concept of "mind-bedtime procrastination" (Rubin 2020). It seems that, sometimes, the biological clocks cannot solely determine bedtimes on weekdays and/or weekends. Instead, "mind-bedtime procrastination" and other similar conscious behaviors can be involved in another conflict with the biological clocks. Such behaviors can lead to missing the bedtime signal from the biological clocks. Particularly, analysis of sleep times in two large samples of university students demonstrated that bedtimes reported by 7.4% and 8.5% of survey participants (from 4346 and 1029, respectively) cannot be simulated without the additional assumption that weekday bedtime can delay relative to the bedtime set by the biological sleep-wake regulator. When this assumption was accounting for in the final simulations, the results supported the interpretation of such delays as reflecting an additional conflict between "clocks" competing for setting bedtimes on weekdays and/or weekends. Therefore, the concept of conflicting "mental" (or "mind") clocks might be added to the concept of conflicting "social clocks." Both "social clocks" and "mental clocks" can disobey the biological (or body) clocks but the former conflict leads to earlier weekday wakeups while the latter conflict leads to later weekday and/or weekend bedtimes.

An Explanation of Reduction of Weekend Sleep Duration After Delays of Bedtime

For the last 4 decades, the two-process model of sleep-wake regulation (Daan et al. 1984) remains the major source of our understanding of the basic mechanisms generating the sleep-wake cycles (Borbély et al., 2016). The model (Daan et al. 1984) predicts two opposite effects of the homeostatic and circadian processes on duration of sleep initiated after its partial deprivation in

the early night hours. The rule of homeostatic regulation suggests that the longer the duration of previous wakefulness, the longer the duration of the following sleep. Therefore, the homeostatic process regulates *ad lib* sleep in such a way that a lengthening of preceding wakefulness (i.e. partial sleep deprivation) leads to a proportional lengthening of sleep duration. When, in light of knowledge on this effect, people tend to think about duration of their sleep after extension of wakefulness on early night hours, they often come to the conclusion that they can sleep more after such an extension, especially on weekends, and thus recover/compensate/pay back/catch up on lost sleep.

However, the model (Daan et al. 1984; Putilov 1995) does not predict that sleep episode initiated in early night hours instead of evening hours can be elongated. Instead, it predicts that sleep duration can be reduced due to the influence of the circadian (or biological or body) clocks on the homeostatic sleep process. Empirical data and their model-based simulations suggested (Daan et al. 1984; Putilov 1995) that, most likely, sleep duration can be shortened due to such an influence of the circadian clocks on the duration of sleep episode initiated in early night hours. Such a shortening is expected because the delayed sleep is initiated at the falling phase of the circadian influence (e.g. before 3 a.m.) to be terminated at its rising phase (e.g. after 3 a.m.; Table 4 and Figures 2–4). Therefore, the longer the delay of habitual bedtimes during early night hours, the shorter the following sleep episode. Importantly, when the duration of sleep is reduced after such extension of wakefulness, this extension cannot cause the following recovering/compensation/paying back/catching up on lost sleep (Putilov et al. 2025b).

As expected, the reduction of time on bed was clearly observed in the results of comparison of students with earlier and later bedtimes. In accord with the prediction of the model, weekend times in bed in the subsamples with late weekend bedtimes were shorter than these times in the subsamples with early weekend bedtimes. Particularly, weekend time in bed was shorter in the later/late subsample than in the earlier/late subsample.

Possible Health Impacts of the Competition Between "Mental" And Biological Clocks

Weekday sleep insufficiency appears to be the most harmful consequence of the delay of weekday bedtime beyond the bedtime set by the biological clocks. Numerous adverse health effects of sleep insufficiency were reported in the literature on sleep medicine (e.g. Strine and Chapman 2005; Gangwisch et al. 2006; Steptoe et al. 2006; Banks Dinges 2007; Roane and Taylor 2008; Cohen et al. 2009; Buxton and Marcelli 2010; Cappuccio et al. 2010; Grandner et al. 2010; Morselli et al. 2010; Thompson et al. 2011; von Ruesten et al. 2012; Soucise et al. 2017; Russell et al. 2023). Therefore, the present results have practical implications for the prediction and prevention of health problems associated with late bedtimes in vulnerable individuals.

For instance, the model-based prediction of weekday sleep loss due to late weekend bedtimes can be taken into account for navigating among the potential intervention strategies aimed at mitigating the adverse health impacts of weekday sleep insufficiency and weekday-weekend sleep displacement. Irrespective of how long was the duration of *ad lib* sleep on weekends, it is necessary to apply the interventions aimed at reducing the weekend delay of bedtime as a possible cause of sleep insufficiency and displacement in the following weekdays (see Supplementary Materials for such strategies and discussion of effects of modern electrical lighting environment and chronotype)

We found that, on weekdays and weekends, the gaps between bedtimes and times of readiness to sleep were associated with insufficient weekday sleep, and, therefore, also with health, mood and sleep concerns. Recently, Kok et al. (2026) published similar results showing that, when students delayed their bedtime by going to bed later than intended, they more frequently had poorer self-rated sleep, higher pre-sleep arousal, and higher depression scores. Such a later than intended bedtime was associated with shorter sleep by 0.91 h on study nights and 0.86 h on non-study nights because there was no compensatory delay in risetime. Moreover, next-morning mood and daytime sleepiness were worse compared with nights with no bedtime delay (Kok et al. 2026).

Hypothetical Mechanisms Underlying "Mental Clocks" And Their Effect on Health

Previously, we already discussed in this journal a hypothetic mechanism of negative health impact from the extension of the wake phase of the sleep-wake cycle (Putilov et al. 2025a). This discussion is also included in Supplementary Materials to explain the possibility of compromising health via such kind of circadian misalignment as the misalignment of central and peripheral rhythms.

Future research is required for the elaboration of potential brain mechanisms responsible for the tendency to voluntary overcome the biological need for falling asleep. We can only speculate that, unlike the

circadian and sleep-wake homeostatic mechanisms located in the oldest brain regions, this behavior can be linked to high-level processes produced by the cerebral cortex (e.g. decision making, problem-solving, thinking, motivation, awareness, attention, learning, etc.).

Study Limitations

Several limitations of this study require acknowledgement. The profound change in sleep-wake characteristics across ages does not allow the generalization of the results to the whole lifespan. The application of cross-sectional and non-repeated measures design in the survey of weekday and weekend sleep times was also among its limitations.

The delays of bedtimes we linked to "mind-bedtime procrastination," we did not assess this behavior in our study. The reason was that its assessments are not designed to measure the delay of reported bedtime relative to the bedtime set by the internal sleep-wake regulating mechanism. We tried to assess the perception of such a delay in the additional survey. Empirical evidence for the ability of survey participants to perceive this delay was provided by analysis of answers to questions about time when body is ready for falling sleep on the weekdays and weekends. However, such reports remain to be validated because it is unknown whether the answers to the questions about perceived delay can accurately inform about the bedtime determined by the internal sleep-wake regulating mechanism.

Moreover, we did not ask the participants about naps, and, therefore, our simulations did not account for their possible influence on bedtimes. We also did not ask about possible weekend obligations that can force some of survey participants to wake up earlier even on weekends.

Finally, our sample cannot inform about the phase positions of objective markers of the circadian clocks. Therefore, data collected in the present study cannot allow the direct calculation of phase angle between the circadian phase and sleep timing. However, the lack of objective measurements cannot challenge the major conclusions of the present study about the necessity to account for the conflict of the biological clocks with "mental clocks." Such a conflict was revealed, irrespective of the particular circadian parameters of the model, and it was most probably linked to the tendency to volitionally prolongate the wake phase of the sleep-wake cycle despite the bedtime signals from the biological clocks.

Conclusions

The results of the present study supported the hypothesis that a model of sleep-wake regulator allowing a voluntary delay of sleep onset on night hours could fit sleep times significantly better than a model without it, specifically in people reporting later weekday than weekend bedtimes. The result suggested that the conflict between three rather than two kinds of "clocks," biological, "social," and, additionally, "mental" can explain bed- and risetime on weekdays and weekends. The latter clocks can be linked to conscious behaviors mostly conceptualized in the previously published studies under such terms as "bedtime procrastination" and "mind-bedtime procrastination." It seems that, if weekday risetimes can be mostly determined by "social clocks" and if weekend risetimes can be mostly determined by the biological clocks, bedtimes on weekdays and weekends cannot be always solely determined by the biological clocks. They can reflect the additional conflict of the biological clocks with "mental clocks" disobeying the bedtime signal from the biological clocks. Weekday prolongation of the wake phase of the sleep-wake cycle beyond the biologically determined bedtime (or, in other terms, self-deprivation of sleep during early night hours) can lead to such negative health consequences such as weekday sleep insufficiency.

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Author Contributions

CRediT: **Arcady A. Putilov:** Conceptualization, Data curation, Formal analysis, Methodology, Software, Supervision, Visualization, Writing – original draft, Writing – review & editing; **Evgeniy G. Verevkin:** Methodology, Software, Writing – original draft; **Dmitry S. Sveshnikov:** Data curation, Funding acquisition, Investigation, Methodology, Software, Supervision, Writing – original draft; **Zarina V. Bakaeva:** Investigation, Writing – original draft; **Elena B. Yakunina:** Investigation, Writing – original draft; **Olga V. Mankaeva:** Investigation, Writing – original draft; **Vladimir I. Torshin:** Investigation, Writing – original draft; **Elena A. Trutneva:** Investigation, Writing – original draft; **Michael M. Lapkin:** Investigation, Writing – original draft; **Zhanna N.**

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Data Availability Statement

Data of the surveys and the results of simulations are available from the first author upon reasonable request.

Informed Consent Statement

The study participants were informed in detail about the survey procedure, and informed consent was obtained from each participant in the form of the answer "Agree" to the following statement: "I give informed consent to anonymously and voluntarily participate in this online survey of sleep-wake behavior and habits."

Institutional Review Board Statement

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of the Institute of Higher Nervous Activity and Neurophysiology in June 2019 (Approval#12402–02-7112).

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