

# **Modelling the Effect of Sleep Timing on Alertness Using a Simple ODE-Based Approach**

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## **Introduction**

Circadian rhythms are ~24-hour biological cycles that regulate a wide range of physiological and behavioural processes in our bodies – including sleep-wake patterns, hormone release, core body temperature and alertness. These rhythms are driven by an internal clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus which synchronises to environmental cues primarily the light-dark cycle. It is essential for the circadian clock and external time to be properly aligned in order to maintain optimal health and cognitive function. Disruptions to this alignment, such as those caused by shift work, jet lag or irregular sleep schedules, can impair alertness and increase the risk of chronic health conditions (Reid, 2019).

Alertness varies across the day, following a dynamic pattern which is influenced by both internal biological processes and behavioural factors. Understanding how these factors interact is critical because fluctuations in alertness have significant implications for cognitive performance, decision-making and safety in occupational and everyday settings (Banks and Dinges, 2007).

Mathematical models offer a valuable framework to simulate these vital processes and predict changes under different conditions, providing insights that are difficult to obtain through observation alone. The two-process model of sleep regulation, first proposed by Borbély (1982), describes the interaction between two primary biological processes which govern the regulation of sleep and wakefulness: a sleep-dependent process (Process S) and a sleep-independent circadian process (Process C).

- Process S (homeostatic sleep pressure): builds up during wakefulness periods and dissipates during sleep
- Process C (circadian rhythm): an internal ~24-hour clock that regulates sleep-wake timing and alertness independently of prior sleep duration.

Together, these systems shape the natural ebb and flow of alertness throughout the day. They explain why sleep that is mistimed relative to the circadian rhythm can result in grogginess despite sufficient sleep duration.

In this project, we have developed a simple ODE-based model that describes alertness as a function of time modulated by three components:

1. A homeostatic regulation term that drives alertness back toward a baseline value,
2. A circadian drive term that introduces oscillatory modulation and
3. A sleep suppression term that reduces alertness during sleep

This project aims to bring the model to life through an interactive interface which allows users to explore how different sleep schedules affect daily alertness. By inputting custom sleep timings, users can compare their schedule against an ideal one aligned with circadian rhythms. This simulation offers an intuitive way to understand how sleep timing and biological rhythms interact to shape alertness, highlighting the importance of when we sleep, not just how long.

## Methods

We developed an Ordinary Differential Equation (ODE) based model that simulates alertness ( $A$ ) as a function of time, modulated by three components: a homeostatic regulation term, a circadian drive term, and a sleep suppression term. This model is given by the equation:

$$\frac{dA}{dt} = -\alpha(A - A_{base}) + \beta \cdot \sin\left(\frac{2\pi t}{T_{circadian}}\right) + S(t)$$

Here,

- $A$ : Alertness level
- $A_{base}$ : Baseline alertness
- $\alpha$ : Rate of homeostatic decay/gain
- $\beta$ : Amplitude of circadian influence
- $T_{circadian}$ : Circadian period (24 hours)
- $S(t)$ : Sleep suppression term (  $-\delta$  if asleep, 0 if awake)

This formulation is inspired by simplified versions of the two-process model, in which alertness increases with circadian stimulation and decreases with prolonged wakefulness (Borbély, 1982; Achermann and Borbély, 1990).

**Table 1***Parameters and Implementation*

Parameters	Value Chosen	Reason
$A_{base}$	0.5 (on a scale of 0.0 to 1.0)	It is a mid-range baseline alertness level. It represents the neutral alertness level when neither circadian nor sleep drive dominates, acting as a midpoint, allowing the model to oscillate (Borbély, 1982).
$\alpha$	0.058	This governs how quickly sleep pressure dissipates after waking, influencing how quickly alertness recovers. Åkerstedt & Folkard (1995) suggest the half-life of sleep pressure to be between 10-15 hours, thus we chose a value that corresponds to a 12-hour half-life.
$\beta$	0.9 (on a scale of 0.1 to 1.0)	This determines the influence of the circadian rhythm on alertness. We have used circadian rhythm as a proxy for alertness as evidence shows that all components of alertness exhibit circadian variations (Valdez, 2019). We used a higher value to make the contribution more prominent.
$T_{circadian}$	24	The standard duration for the circadian rhythm.

$\delta$	0.4	Alertness is expected to decrease during a period of sleep (Thomas <i>et al.</i> , 2000). This value captures the balance between the physiological state of sleep and the underlying circadian influences. While asleep, $S(t)$ introduces a negative value to account for the reduced responsiveness of the brain during sleep periods.
$A_0$	$A_{base}$	The initial value for alertness is chosen as the base value, so that the model can evolve based on the inputs, avoiding bias.
$max\_penalty$	0.2 to 0.3	Research has shown a 20-30% decrease in cognitive performance because of misaligned schedules, which are representative of the suboptimal sleep time (user's input) (Wright <i>et al.</i> , 2013). We make an array of values from 0.2-0.3, from which the model randomly chooses one to implement in a run.

Alertness was initialised at a baseline ( $A_0 = A_{base}$ ) and simulated over a 24-hour period. We implemented the model in Python using the *scipy.integrate.odeint* function.

In this model, we can compare the user's sleep schedule to our main aligned sleep schedule.

- Aligned sleep (fixed sleep): 10:30 PM to 06:30 AM (8-hour duration of sleep) (Clinic, 2024)

- Misaligned sleep (custom sleep): User Input value

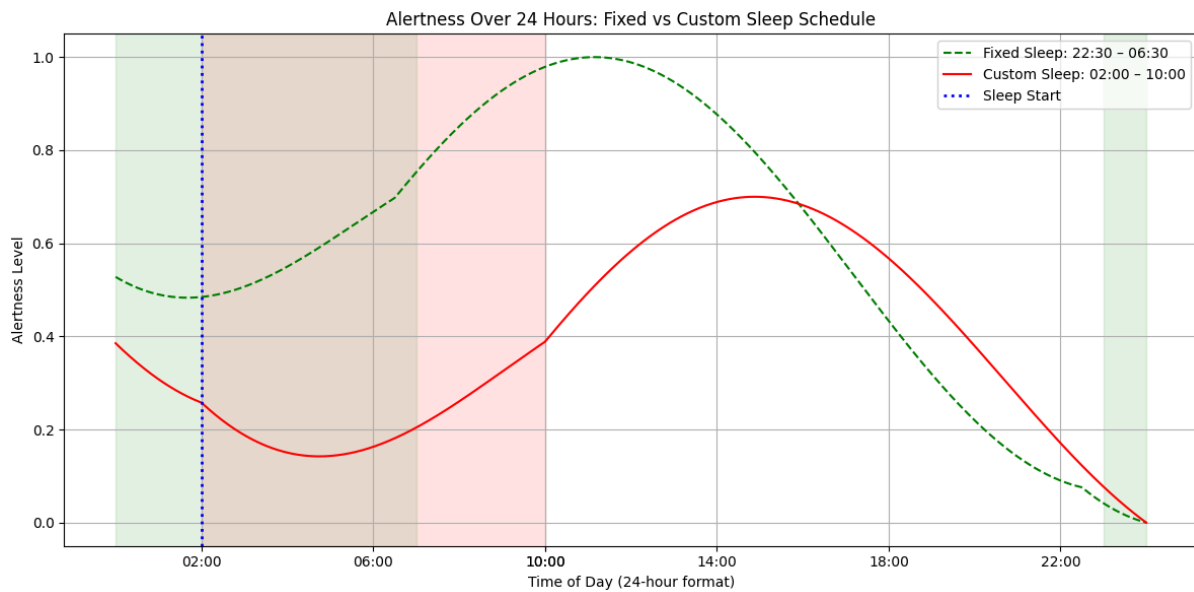
A function defined whether the person was asleep at each time point, applying the  $S(t)$  suppression accordingly.

## Results

**Figure 1. User Input Values for Sleep Schedule and Alertness Reduction**

```
Alertness reduced by 22.0 %
Enter your sleep time and wake time in 24-hour format (e.g., 04:00 for 4 AM):
Enter your sleep time (HH:MM): 02:00
Enter your wake time (HH:MM): 10:00
```

**Figure 2. Alertness Over 24 Hours: Fixed vs Custom Sleep Schedule**



*Note: The figure illustrates the rise and fall in alertness over 24 hours, for a fixed and custom sleep schedule. The X-axis denotes the Time of Day (24-hour format) and the Y-axis denotes the*

*Alertness level (on a scale of 0.0 to 1.0). The shaded regions (blue and red) indicate the duration of sleep for both sleep schedules.*

The simulated alertness profiles, charted over a 24-hour period for both sleep schedules, are shown in *Figure 1*. The model demonstrates a substantial difference in daytime alertness depending upon sleep timing. The Fixed Sleep (aligned sleep) results in much higher alertness during morning and afternoon hours, with a gradual rise post-wake and a peak before the afternoon, followed by a steady decline as the day progresses, due to the regular functioning of the circadian rhythm. The alertness hits a peak value of 1.0 (very high) around 3 hours after wake (around 10 AM). For the purpose of executing the model, Custom Sleep values were chosen as follows: 2 AM (sleep time) to 10 AM (waketime). These result in markedly lower alertness during the earlier part of the day and a delayed peak in alertness during the afternoon. The peak reaches a value of 0.8, much less than that of the Fixed Sleep. Alertness is reduced by 22.0% with the Custom Sleep schedule, indicating that the timing of sleep affects next-day alertness.

These patterns reflect the alignment (or misalignment) of sleep timing with the endogenous circadian peak (Reid, 2019). Even though both sleep windows have the same duration, the misaligned sleep schedule overlaps less optimally with the circadian alerting signal, resulting in diminished alertness during typical daytime hours. This result is consistent with empirical findings showing that individuals with delayed sleep schedules exhibit poorer daytime performance and increased sleep inertia (Phillips *et al.*, 2019; Wright *et al.*, 2006). The model also highlights the critical window in the early morning when alertness is lowest for late sleepers



- a period often associated with low or diminished cognitive function and increased accident risk (Leso *et al.*, 2021).

## **Discussion**

Our results illustrate how simple models can effectively capture important physiological dynamics. The key insight from this study is that timing of sleep, not just duration, critically determines alertness outcomes (Genzel *et al.*, 2013). By simulating two equally long sleep periods with different onsets, we show that alignment with the circadian phase significantly affects alertness throughout the day.

The inclusion of a circadian oscillation is grounded in the model of Process C , which describes oscillatory modulation of alertness (Daan *et al.*, 1984). Our model's homeostatic component represents the natural recovery of alertness during sleep and its decline during wake, consistent with Process S in Borbely's framework. Although we use a simplified sinusoidal circadian component to model sleep, this approach provides a simplistic framework for simulating the effects of different sleep strategies.

The model captures several key components observed in empirical data like the modulation of alertness across the day by circadian phase, and the dampening of morning alertness following delayed sleep. The model reproduces the phenomenon where a misaligned sleep schedule

produces an alertness profile that is not only shifted later, but also diminished in amplitude, consistent with reduced daytime performance and cognition.

There are several important limitations to our model. The circadian rhythm is modeled as a fixed sine wave, independent of sleep-wake state or light exposure. In reality, the circadian rhythm is sensitive to environmental light cues and can shift dynamically (Jewett *et al.*, 1999). The model also assumes abrupt sleep transitions and does not account for sleep inertia, omitting the cumulative effects of chronic circadian misalignment that could arise in shift workers or during travel (Leso *et al.*, 2021).

Despite these simplifications, the model demonstrates the value of sleep timing in shaping the next-day alertness, and provides a useful educational and exploratory tool. Extensions of this model could incorporate phase-response curves, light sensitivity, or adaptation over multiple days to better match observed physiology and behavior.

**Table 2***Individual Contribution*

<b>Name</b>	<b>Aadya Sood</b>	<b>Tamanna Balachandran</b>	<b>Yashasvee Singh</b>
<b>Conceptualisation</b>	Yes	Yes	Yes
<b>Coding/Calculations</b>	Yes	Yes	Yes
<b>Analysis/Interpretation of model outcome</b>	Yes	Yes	Yes
<b>Writing - Original Draft</b>	Yes	Yes	Yes
<b>Writing - Review &amp; Editing</b>	Yes	Yes	Yes
<b>Visualisation</b>	Yes	Yes	Yes
<b>Project Management</b>	Yes	Yes	Yes
<b>Other (Specify)</b>	-	-	-

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