Formalisation and Implementation of the Two Process Model of Sleep Regulation

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Formalisation and Programmatic Implementation of the Two Process Model of Sleep Regulation

This report details an attempt at formalizing and implementing the Two Process Model of Sleep Regulation by Borbély (1982) which has served as a foundational theory in sleep science in the last decades. It aims to explain the control of sleep and wakefulness by postulating a sleep-dependent process S and a circadian sleep-independent process C.

For this purpose VAST (Leising et al., 2023) will be used to help formalizing the theory. The structure of this report will loosely follow the Theory Construction Methodology (TCM) by Borsboom et al. (2021). Code and figures can be found at https://github.com/advieser/FOMO-sleepreg.

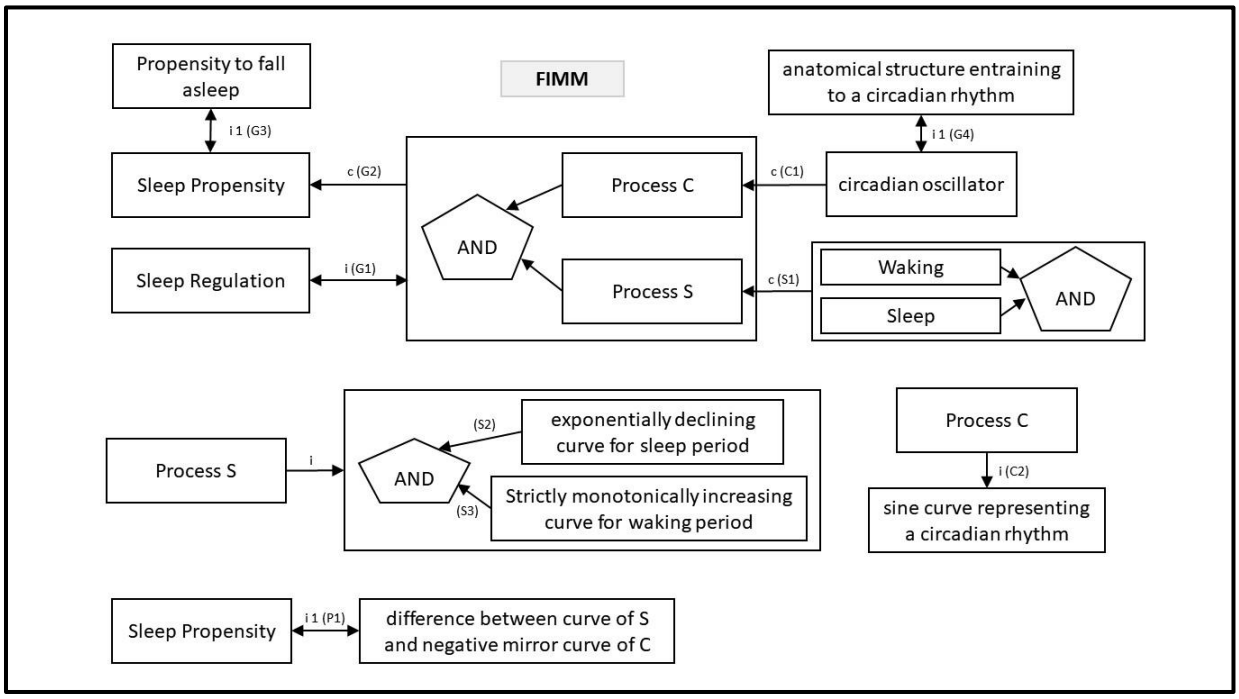
Formalisation

Focal Constructs of the Two Process Model

As its' name implies, the chosen theory mainly uses two processes to explain the regulation of sleep: *Process S* and *Process C*. Whether an individual falls asleep is determined by his or her *sleep propensity* which is directly influenced by the two processes. Figure 1 shows a VAST display formalising these constructs in the context of this project, based on key statements from the literature which can be found in Table A1 in the appendix. Note, that this is a simplified version of the model, leaving out sleep stages and EEG activity among other things, as they are not relevant to this project. Also note, that to avoid an infinite regress some concepts are not explicitly defined, such as sleep, waking, circadian rhythm and entrainment. Furthermore, definitions of sleep propensity and circadian oscillator were added for clarification purposes and were based on personal decisions, not statements from the literature. This model already contains some mathematical conceptualisations of the two processes as these are part of the original model and important for its explanative value. These may seem abstract for the moment but will be shown later (see Fig. 4).

Figure 1

VAST Display Defining Focal Constructs of the Two Process Model of Sleep Regulation



Jet Lag as a Relevant Phenomenon

Using the formalized model of the chosen theory, this report aims to investigate the occurrence of jet lag, which has previously been verbally explained using the two process model of sleep regulation (e.g. Vosko et al., 2010).

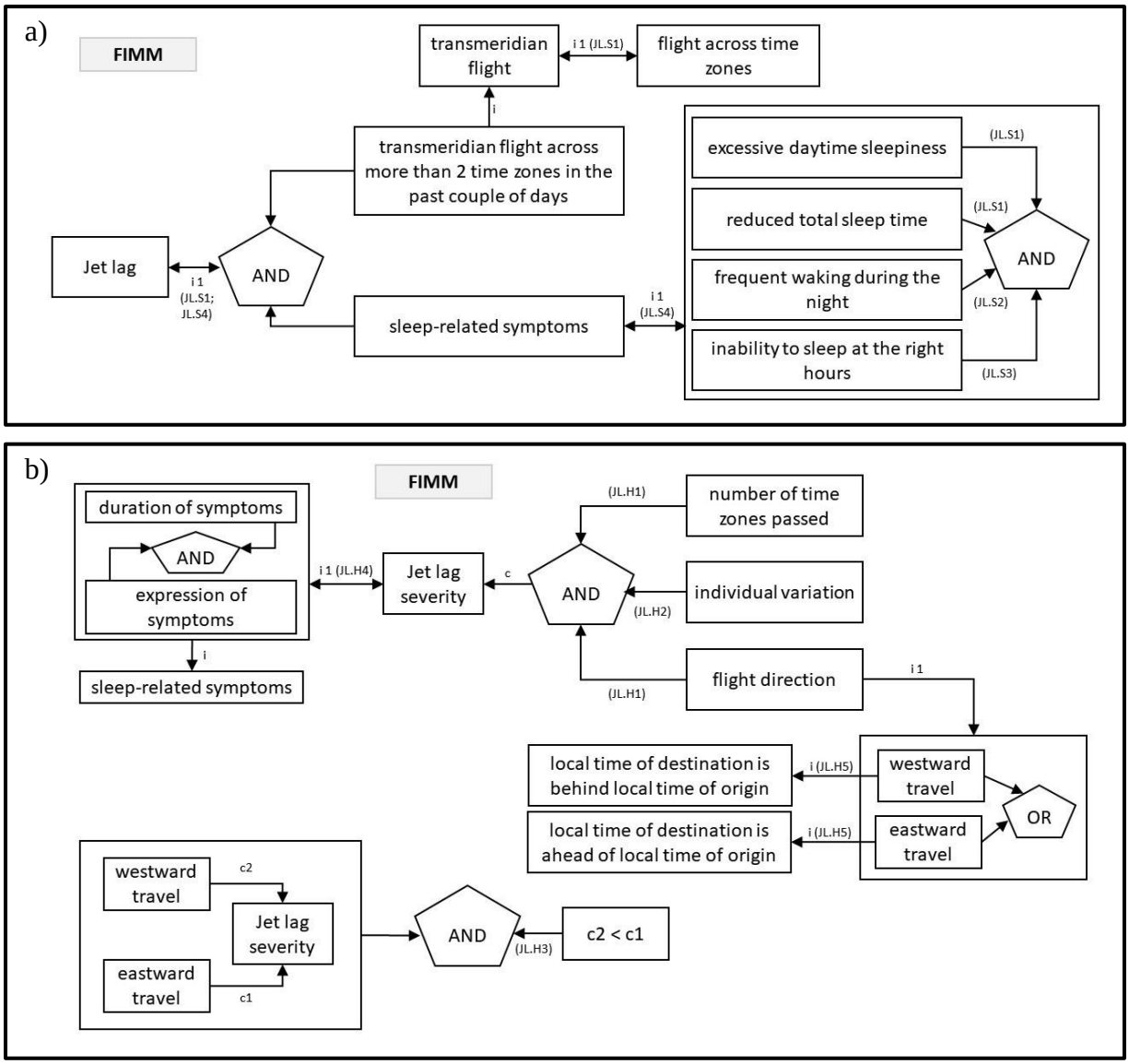
Jet lag syndrome is a circadian rhythm sleep disorder defined in the International Classification of Sleep Disorders (ICSD-3, AASM, 2014). There, jet lag is commonly understood as a syndrome of symptoms affecting sleep, cognitive, and somatic functions, following a transmeridian flight (i.e. over multiple time zones). Sleep-related symptoms mainly consist of inability to fall asleep at the right hour, frequent waking during the night, less total sleep time and excessive daytime sleepiness. Most travellers seem to tolerate westward travel better than eastward travel (AASM, 2014, as cited in Beros et al., 2021; Cingi et al., 2018; Vosko et al., 2010).

Cingi et al. (2018) note that “almost all critical jet lag symptoms are caused by the disturbance to the body’s sleeping and waking schedules” (S. 61), which is the argument for why this report is going to focus on operationalizing jet lag only via its sleep-related symptoms. Figure 2a shows a VAST display defining the phenomenon of jet lag for the current report while Figure 2b shows associations pertaining to the severity of jet lag. Relationships reference statements from the literature which can be found in Table A2 in the appendix. The definition of jet lag severity in conjunction with its symptoms was based on a

personal interpretation. The choice of which symptoms mentioned in the literature to include was also a personal decision, instead of defining the complex clinical construct of insomnia.

Figure 2

VAST Display Defining Jet Lag and Severity of Jet Lag



Following TCM (Borsboom et al., 2021), once a phenomenon has been identified, its robustness should be assessed. However, no meta-analyses could be identified, that directly investigated the robustness of jet lag.

Instead, three standalone studies shall be reviewed, investigating generalizability across the four UTOS dimensions (unit, treatment, outcome, setting; Cronbach & Shapiro, 1982; Findley et al., 2021) and the found effect sizes.

As research untis, an Olympic team support staff (n = 9; Rossiter et al., 2022), a professional male football team (n=23, Fowler et al., 2017) and master triathletes (n = 12, Stevens et al., 2018) were investigated. These samples were extremely small and relatively homogeneous, consisting mainly of men and an over-proportion of professional athletes. The unit dimension cannot be considered very generalizable.

The transmeridian flights are considered as treatments in this context. The sample from Rossiter et al. (2022) underwent a 24-h eastward travel over 8 time zones (Ireland-Japan), the sample from Fowler et al. (2017) experienced a 19-h eastward flight over 11 time zones (Australia-Brazil) and the sample from Stevens et al. (2018) an northeastward journey with a mean travel time of 22.6-h of which 11.5-h were spent flying (Australia-US). These are homogeneous in the sense that no short flights were reported, and all were eastward journeys. The number of time zones passed were only slightly variable. The treatment dimension must be considered limited in its generalizability.

Overall, the three studies used self-reported data and various sleep parameters assessed via actigraphy. Comparisons were made intra-individually, no control group was used. Rossiter et al. (2022) reported a significantly decreased sleep duration and objectively measured sleep quality for 6 days and 5 days post-travel, respectively. Subjective jet lag differed significantly from pre-travel measurement for 6 days post-travel. No easily interpretable effect sizes were reported. Participants from Fowler et al. (2017) reported a significantly increased subjective experience of jet lag for 4 days post-travel ($d > 0.90$). Sleep parameters differed for only two days after travel in comparison to pre-travel measurement ($d > 0.90$). In contrast, Stevens et al. (2018) present no significant changes in sleep quality. Sleep duration was significantly increased on the first night in comparison to pre-travel measurement (chance of difference = 95-99%), but not on subsequent nights. Self-reported fatigue was likewise only increased on the first day (chance of difference = 75-95%).

This leads to a very unclear picture. Effects on sleep duration seem to differ between studies, sleep quality was affected in two studies, but for different periods of time, while one study did not find an effect on sleep quality and two studies report an increased subjective experience of jet lag. Since findings of effects were so heterogeneous, a discussion of the, few and differently measured, strengths of evidence as well as the generalizability across outcome measures doesn’t seem productive and is omitted here.

As to the settings investigated, it can be noted that all mentioned sources studied real flights instead of simulated jet lag in the lab, as may also be done. As jet lag is considered a real-world phenomenon, this is not seen as limiting the generalizability of the effect.

However, it can be noted that all participants were in some way part of a high stakes competition and within training camps during the study period, which is very limiting to the generalizability.

In conclusion, based on the review of three studies, jet lag cannot be considered a robust phenomenon. However, it seems reasonable to assume that jet lag is vastly more robust, and that this contradiction might mainly arise from the small number of studies reviewed here.

Formal Model

In this section, results from previous sections are integrated to create a VAST analysis displaying a formal model that aims to explain jet lag by applying the Two Process Model of Sleep Regulation. While some sources cite this model as an explanation for jet lag (e.g. Vosko et al., 2010), the explanations were insufficiently explicated, especially in regard to stating the respective roles of the two processes. Thus, additional assumptions and interpretation had to be made.

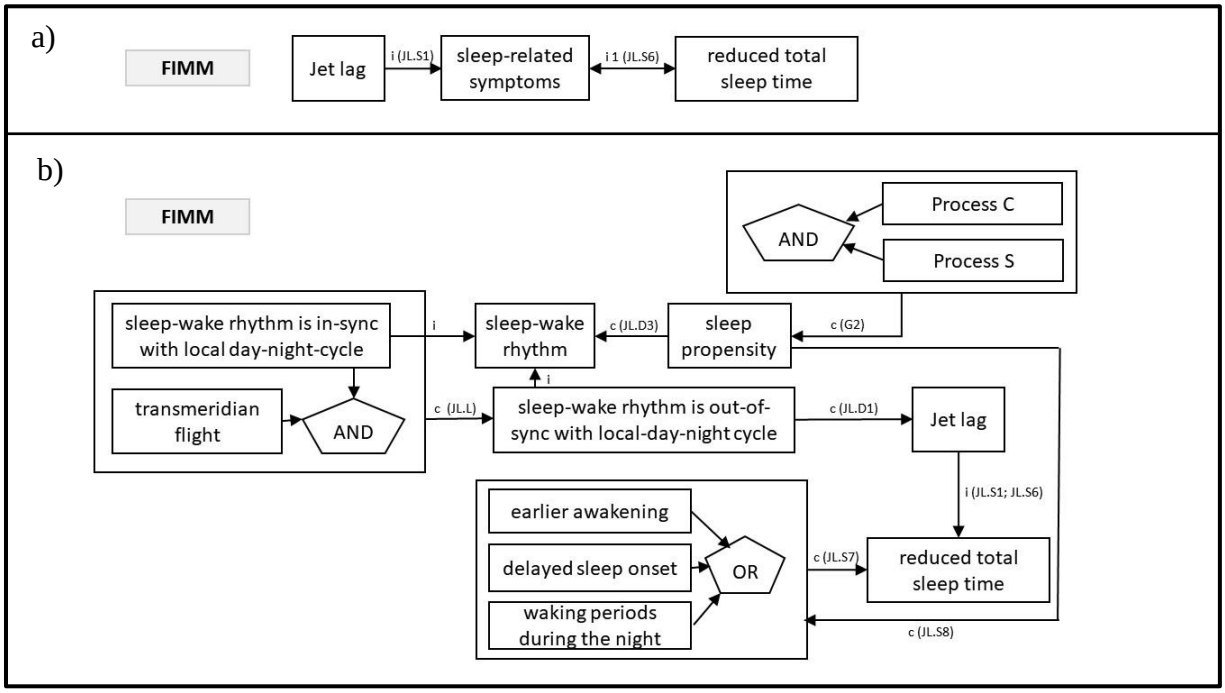
To simplify this process, some definitions are now modified or newly introduced: Sleep-related symptoms now only contain reduced total sleep time (Tab. A2/JL.S6, see Fig. 3a) as this seem easiest to explain and additionally is one of the two symptoms explicitly mentioned by the ICSD-3 (AASM, 2014, as cited in Beros et al., 2021). The causes for reduction in total sleep time and that they are influenced by sleep propensity are based on personal deduction (Tab. A2/JL.S7), not on statements from the literature. What in the literature has been referred to as “circadian rhythm” in specifically this context has been termed “sleep-wake cycle”, as is done in German (Tab. A2/JL.D2). In the chosen instantiation of the model, sleep propensity is the only cause of an individual’s sleep-wake-cycle (Tab. A2/JL.D3). In reality, this is obviously dependent on more variables, most of all the rhythm of an individual’s social environment (work, hobbies, etc.).

Figure 3b shows the final formalized model. The two processes influence sleep propensity which influences the sleep-wake rhythm before as well as after the transmeridian flight. This flight leads to a desynchronization between the sleep-wake cycle that is still based on the starting location and the environmental day-night-cycle at the destination. Desynchonisation leads to jet-leg which implies a reduced total sleep time. At this stage, an explicit explanation for how the different causes of the reduction in total sleep time are dependent on the the two processes of sleep regulation, is sadly missing, though they are

influenced by sleep propensity. This model leaves out the process of synchronising to the new environment which would ultimately be necessary for reducing jet lag symptoms.

Figure 3

VAST Display of a Formal Model Explaining Jet Lag Through The Two Process Theory of Sleep Regulation



Implementation

Originally, an implementation of the presented model in R was intended to be described here. However, this was not successful. Reasons for this will be discussed in the next section. This section will instead include the considerations that were made while attempting the implementation and how the current implementation in R deviates from these.

The goal was to have a model that for a given agent could compute their sleep and wake times for a period from the day of travel to 3 days post-travel, so that total sleep time could be computed. This could then have been compared with the total sleep time of agents with the same properties that had not travelled.

The model by Borbély (1982) offers some information about the mathematical properties of the two processes and and how sleep propensity follows from these. The two process functions are modelled to resemble Figure 5 in Borbély (1982) and thus leading to a sleep period from 23:00 to 06:00 under normal conditions. Sleep propensity is defined as the summation of Process S and Process C (Tab. A1/P1), meaning that Process C is understood to

inhibit sleep. For a more intuitive presentation, $\bar{C} \coloneqq -C$ was defined as the negative mirror of Process C (Tab. A1/C3). Awakening is defined as happening when the curves of $\bar{C}$ and S intersect (Tab. A1/P2, P3) , so when sleep propensity is 0.

The original authors make no explicit assumption about when sleep is initiated. For this reason, an additional assumption had to be made, that an agent would have a set (local) time when they would go to sleep (bedtime), but they would sleep till natural awakening. By doing so, the day-night cycle could be taken into consideration.

Agents can then be characterised by their current state (asleep vs. awake), and their current sleep propensity, depending on the time. Furthermore, they should be associated with a list of sleep and wake times, cumulating in their total sleep time per night. Each agent would have a fixed bedtime that stays constant when changing time zones (i.e. 23:00 in both time zones).

The following tables give an overview over how central variables (Tab. 1), and their functional relationships (Tab. 2) could be conceptualised. Figure 4 then shows the functions from Table 2 as plots.

Table 1

Definitions for Variables and Agent Parameters

Construct	**Scale level**	**Range**	**Anchors/Notes/Definitions**
time	continous	$[0, 72[$	- Overall, 3 days, measured in hours since start of study; day 1 is travel day; day 2 and 3 are post-travel; - 0: 00:00 on day 1 in local time of origin - 24: 00:00 on day 2 in local time of origin - …
Level of Process C	continuous	$[-1; 0]$	- -1: Minimum of sleep propensity due to Process C (thus "inhibiting" sleep) - 0: Maximum of sleep propensity due to Process C - Chosen, so that $-C$ is always positive like in the original figure.
Level of Process S	continuous	$[0; +\infty[$	- 0: minimum of sleep propensity due to Process S - theoretically no upper limit, however sleep should begin earlier
Sleep propensity	continuous	$[0, +\infty[$	- 0: no propensity to sleep, leading to awakening when asleep before - theoretically no upper limit due to Process S.
State	binary	$\{0,1\}$	0 = asleep and 1 = awake
Bedtime	continuous	$[0, 24[$	Fixed time of day an agent goes to sleep.

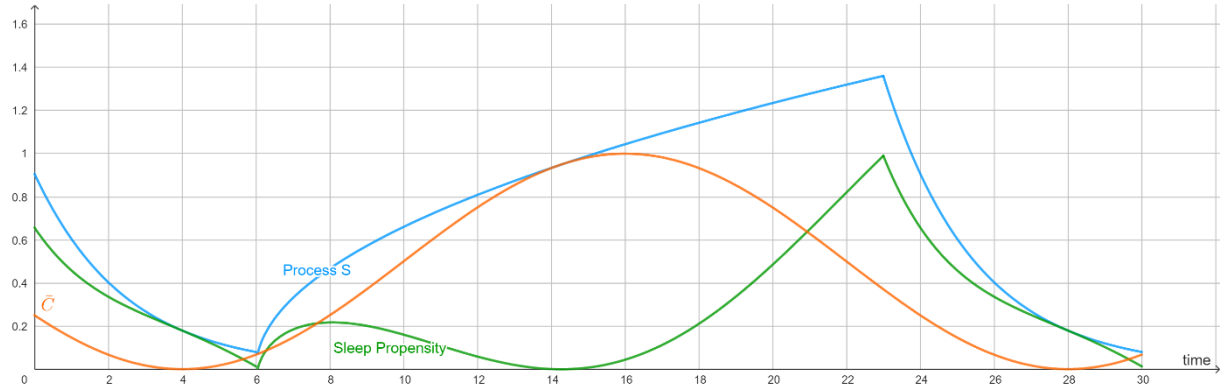
Table 2

Functional Relationships

ID	Consideration	Function
F1	**Construct**: Process C = f(time)	
	- sine curve with a period of 24-h. - minimum at 16:00 and maximum at 04:00 (Tab. A1/C2) - should always be negative, so that $\bar{C}$ is only positive as in original figure. - amplitude = 1 for simplicity (Tab. A1/C6)	$C(t) = -0.5\left(\sin\left((t+2)\cdot\frac{\pi}{12}\right)-1\right)$
F2	**Construct**: Process S = f(time, last_awakening, last_sleep_onset)	
	- strictly monotonically increasing when awake (Tab. A1/S2) - exponentially decreasing when asleep (Tab. A1/S3) - for wake period 06:00-23:00: tangential to $-C$ at 14:00, maximum at 23:00, intersect with $-C$ at 06:00 (from original figure)	$S(t, la, lso) = \begin{cases} 0.33\cdot\sqrt{t-la} & \text{if awake} \\ 1.36\cdot 1.5^{-t+lso} & \text{if asleep} \end{cases}$ where la is last awakening and lso is last sleep onset.
F3	**Construct**: Sleep Propensity = f(time, Process_C, Process_S)	
	- as defined in the original paper (Tab. A1/P1)	$P(t) = C(t) + S(t)$

Figure 4

Plots of Functional Relationships



Note: Plot showing Process S (blue), the negative mirror of Process C ($\bar{C}$; orange) and the sleep propensity (green), under normal (no jet lag) conditions. Sleep happens from 23:00 to 06:00 each day.

The current implementation in R uses these considerations to calculate sleep propensity for discrete time steps, noting when the agent should go to sleep based on their fixed bedtime, or when they should wake based on their sleep propensity, for a single agent It does not calculate total sleep time nor are transmeridian flights considered at all. Sadly, this implementation also contains a bug, in that sleep propensity is consistently too high, leading to eternal sleep for the agent once they fall asleep.

Concrete Problems with Implementation

The implementation of the formal model derived in Figure 3 proved difficult mostly due to two separate problems: (1) the model was underspecified, and (2) the interaction between processes and agent states was more complex than anticipated. I will discuss both points separately.

The formal model essentially mirrored the explanation given by Vosko et al. (2010). As became apparent while formulating this model, the verbal explanation did not offer the explanatory value I had ascribed to it before, especially not explicitly describing the role of process S in the etiology of jet lag. I made additional assumptions and simplifications in an attempt to concretise the relationship between constructs. While some deficiencies were still apparent, I decided to continue with the implementation. However, during this process, the problems with the model became more apparent, as it wasn't helpful enough in guiding my decisions in implementation. At this point, I would tend to take a step back to improve the formal model first, but this was not possible in the time I had remaining. Ultimately, I come to the conclusion that the underspecification was in part due to me underestimating the complexity of the original model and overestimating its explicitness due to its already mathematical nature. For example, the y-axis of the Process C was never explicated. It could also not have been the same as used for Process S (EEG activity), so that the mathematical relationship between the two process was left unclear. Both influence sleep propensity, the scale of which was also never specified.

Before starting this project, I only knew this theory as an apparently simple explanation of sleeping and waking behaviour through only two constructs. As I started going deeper into the theory, I was pleasantly surprised how mathematically formalised the original model already was. Even more so, as it was subject to formalisation and simulation in the past (Achermann et al., 1993). When starting implementation, it became apparent that a functional representation was not as easy as anticipated, as the functions describing the processes were constantly changing as consequence of their own return values. While I believe that this can

be solved, given enough time, I had to choose at one point to abandon the approach, and cut my losses.

Aside from these points, some time loss must be attributed to my first approach which relied on using datetimes for the implementation, which soon proved to be too confusing, although I had prior experience dealing with them in R.

General Discussion

In this section, I will briefly reflect on things I noted while following the methodology we learned about in the seminar and which I employed for this project.

For one, I noticed that the definition of the two processes was difficult using VAST, as their effects were mostly explicated using plots and not verbalisation. At this point, later problems in formalising the relationships between constructs already became evident.

Furthermore, the review of the phenomenon proved difficult. Although, jet lag is a very well-known effect that almost all people seem to experience, the scientific literature does not reflect this. Studies directly investigating the effect were extremely sparse and samples extremely small, as is sadly common in sleep science.

While integrating the definitions of focal constructs and jet lag into the formal model, I noticed how it became more and more complex and more difficult to understand the association as I went on. I think I should have taken a step back at this point and simplified the constructs I chose earlier.

Even though the implementation failed, some insight could still be gained about the original model. While it was mathematical in nature, through the process of formalisation, underspecifications still became apparent, as was described in the last section.

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Appendix

Table A1

Atomised Statements pertaining to the Two Process Model of Sleep Regulation

ID	Quote	Reference	Category/Comment
G1	"[…] two separate processes underlie **sleep regulation**."	Borbély (1982), p. 199	General
G2	"Sleep propensity is assumed to be determined by the combined action of the two processes."	Borbély (1982), p. 199	General, Combination for sleep propensity
G3	Defining Sleep propensity as "propensity to fall asleep" for clarification.	Personal Decision	General, Sleep Propensity
G4	Defining a "circadian oscillator" as a "anatomical structure entraining to a circadian rhythm". (Today understood to be the Suprachiasmatic Nucleus)	Personal Decision	General
S1	"Process S is regarded as the major **sleep-dependent** component of total sleep propensity."	Borbély (1982), p. 199	Process S, total sleep propensity
S2	About Figure 4: "The upper part of the figure shows the **exponential decline** of slow wave activity during a **sleep period** following upon a **regular waking period** (continuous line) […]"	Borbély (1982), p. 199	Process S, Mathmatical Association
S3	About Figure 4: "The thin interrupted lines interpolate the values **between two consecutive sleep periods** and represent the **rising** level of SWS propensity with the **progression of waking**"	Borbély (1982), p. 199	Process S, Mathmatical Association
S4	I decide to leave out Slow Wave Activity (SWA) for the model. Note however, that it is a biological covariate of Process S, as described in the article, e.g.: "The lower section of the figure shows the timecourse of	Personal Decision; Borbély (1982), p. 199	

	Process S which is derived from the measured values of slow wave activity during sleep and from the interpolated slow wave propensity during waking."		
C1	"It [Process C] is assumed to be controlled by a **circadian oscillator** which is **unaffected** by the occurrence of waking and sleep."	Borbély (1982), p. 199	Process C
C2	"The **minimum** of the **sine** function representing Process C was set for 1600 hours, the **maximum** for 0400 hours."	Borbély (1982), p. 199	Process C, Mathmatical Association
C3	"[…] negative function (mirror image) of Process C (designated $\bar{C}$)."	Borbély (1982), p. 199	Curve $\bar{C}$
C4	"Curve $\bar{C}$ maybe considered as reflecting the circadian variation of the "sleep threshold" which is highest when circadian sleep propensity is lowest (i.e. in the afternoon)."	Borbély (1982), p. 199	Curve $\bar{C}$
C5	The latter [total sleep propensity] is assumed to be the combined result of Process S and of a **circadian sleep process** (Process C)	Borbély (1982), p. 199	Sleep Propensity, Process C
C6	"The amplitude of the circadian rhythm (curve $\bar{C}$) has been defined arbitrarily."	Borbély (1982), p. 200	Curve $\bar{C}$
P1	"Total sleep propensity, which corresponds to the summation of S and C, is therefore represented by the difference between S and $\bar{C}$"	Borbély (1982), p. 199	Total Sleep Propensity
P2	"After sleep onset at 2300 hours the difference between the curves (hatched area) decreases until it reaches Zero at the time of awakening."	Borbély (1982), p. 199	Total Sleep Propensity, Sleep onset, Awakening

Table A2

Atomised Statements pertaining to Jet Lag

ID	Quote	Reference	Category/Comment
JL.S1	"The criteria for diagnosing jet lag are set out in the third edition of the [ICSD-3], as follows: 1. There is complaint of insomnia or **excessive daytime sleepiness**, accompanied by a **reduction of total sleep time**, associated with transmeridian (crossing of time zones) jet travel across at least two time zones; 2. There is an associated impairment of daytime function, general malaise, or somatic symptoms[…] within one to two days after travel; and 3. [exclusion criteria]"	AASM (2014), as cited in Beros et al. (2021), p. 2	Sleep (and other) symptoms, transmeridian flight
JL.S2	"Signs of jet lag can vary between debilitated awareness, **insomnia**, **feeling tired during the day** and **frequent waking during the night**."	Cingi et al. (2018), p. 59	Sleep (and other) symptoms
JL.S3	"The symptoms of jet lag can include: sleep disturbance: The most common sign of jet lag reported is the disruption of daily sleep schedulep. People may often discover that they are **unable to sleep at the right hourp.** They may experience **insomnia at night** while **feeling sleepy throughout the day**. […]"	Cingi et al. (2018), p. 62	Symptoms
JL.S4	As insomnia is a very wide term for sleeping disorders, I choose to limit myself to the symptoms explicitly mention in the literature.	Personal Decision	Sleep symptoms, definition

	I summarise these (excessive daytime sleepiness, frequent waking during the night, inability to fall asleep at the right hour and less total sleep time) as "sleep-related symptoms".		
JL.S5	"Almost all critical jet lag symptoms are caused by the disturbance to the body's sleeping and waking schedules."	Cingi et al. (2018), p. 61	Sleep symptoms are most important, since they lead to the otherp.
JL.S6	The definition of sleep-related symptoms is updated for the formal model: They now only encompass reduced total sleep time.	Personal Decision	Sleep symptoms, definition
JL.S7	Reduced total sleep time is likely to be caused by (a) earlier awakening, (b) delayed sleep onset or (c) waking periods during the night (inclusive or).	Personal Decision	
JL.S8	The causes listed in JL.S7 are dependent on the sleep propensity, as they depend on then the individual falls asleep.	Personal Decision	
JL.H1	"It is important to note that the **number of time zones surpassed** and **in which direction these zones were** contribute to the harshness of jet lag."	Cingi et al. (2018), p. 59	Influences on severity
JL.H2	"Not all travelers crossing time zones suffer from jet lag to the same degree, and these differences probably result from individual variation."	Vosko et al. (2010), p. 192	Influences on severity: Individual variation
JL.H3	"In general, because of endogenous circadian rhythmicity longer than a 24-h period, westward travel is tolerated better than eastward travel; a phase delay (staying up later than usual) is easier than the eastward phase advance."	Weingarten & Collop (2013), P.1397	Influences on severity: direction

JL.H4	I interpret increased jet lag severity as either the symptoms being expressed more strongly (e.g. longer amount of missed sleep) or as lasting longer (duration).	Personal Decision	Severity Definition
JL.H5	Westward travel implies that local time of destination is behind local time of origin. Eastward travel implies that local time of destination is ahead of local time of origin.	Personal Addition	Direction
JL.D1	"The reason for jet lag is due to the disharmonizing of the body's circadian structure and the day to night cycle of the destination the passenger has travelled to."	Cingi et al. (2018), p. 59	desynchronisation
JL.L	Logical conclusion: If circadian rhythm is in-sync with local day-night-cycle at the origin, and a person takes a transmeridian flight, thus passing multiple time zones, the circadian rhythm of that person is not in-sync with the (changed) local time at the destination.	Personal Decision	desynchronisation
JL.D2	Decision to call "circadian rhythm" "sleep-wake-rhythm" as is done in German, to avoid confusion with other circadian rhythms (e.g. body temperature).	Personal Decision	Definition
JL.D3	Sleep propensity influences the sleep-wake-rhythm.	Personal deduction	Deduction