

Judgment of daytime sleepiness in self-reported short, long and midrange sleepers.

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Sleep-wake behavior, as well as sleepiness, is regulated by the joint action of an exponentially increasing drive for sleep -sleep homeostasis- and by variations in sleep propensity due to a biological circadian oscillator. However, large inter-individual differences remain. Short and long sleepers have been known to differ in the amount of homeostatic sleep pressure: long sleepers report higher levels of subjective sleepiness after sleep deprivation, whereas short sleepers exhibit no significant increase in sleepiness. The circadian pacemaker's program might be at the origin of the variability in habitual sleep duration in long and short sleepers. Previous studies within our group showed that the dynamics of both processes are similar in experienced sleepiness as in judged sleepiness. The aim of the present investigation was to determine whether habitual short, long and midrange sleepers exhibit similar integration patterns or whether the biological underpinnings of habitual sleep time variability emerge when judging sleepiness. Our results show an additive integration rule for homeostatic and circadian determinants of daytime sleepiness in all groups. However, rescaled functional values (RSFVs) and relative range indices (RRIs) suggest that short sleepers seem to tolerate higher levels of homeostatic sleep pressure in comparison to long sleepers and that in long sleepers, circadian variability plays a less prominent role in functional daytime sleepiness. As sleep-wake behavior is governed by both physiological mechanisms and psychological processes influenced by past experiences related to sleep and wake, these results have implications for sleep-wake-related health and safety.

* Acknowledgments: This research was supported by grant BOF1023OZR issued by the Research Council of the Vrije Universiteit Brussel. Correspondence address: Olivier Mairesse. Royal Military Academy - Research group VIPER – Vital Signs and Performance Building D (20.39) – Renaissanceaan 30 - 1040 Brussels; Tel +32 2 742 61 60; email: Olivier.Mairesse@elec.rma.ac.be

Sleepiness is a physiologically based phenomenon. Because of its potentially dangerous consequences when interfering with activities during wake, nature provided us with the ability to detect it subjectively (Horne and Burley, 2010). Sleepiness is likely to be the organism's alarm mechanism, prompting it to take appropriate actions in order to reduce potential harm (Odele-Dusseau, Bradley and Pilcher, 2010).

According to Borbély's Two-Process Model of Sleep Regulation (1982), sleep-wake behavior is regulated by the joint action of an exponentially increasing drive for sleep -sleep homeostasis or *Process S*- and by variations in sleep propensity due to a biological circadian oscillator (*Process C*). The total drive for sleep is represented by the weighted addition of both homeostatic and circadian drives, each load representing the connection strength between the respective drives and the sleep activating neuronal system in the ventrolateral preoptic area of the hypothalamus (VLPO) in the brain (Fulcher, Phillips and Robinson, 2010). Phenomenological models have further shown that subjective feelings of sleepiness are also regulated through similar processes underlying sleep-wake behavior (Åkerstedt and Folkard, 1994, 1995; Folkard, Åkerstedt, Macdonald, Tucker and Spencer, 1999; Jewett and Kronauer, 1999), and that in fact, when measured soundly, people are able to subjectively assess their levels of sleepiness (Horne and Burley, 2010). In addition, results from functional integration experiments have shown that similar processes govern judgments of sleepiness when individuals are given information of time of day and previous time in bed or total sleep time (Mairesse, Hofmans, De Valck, Cluydts and Theuns, 2007; Mairesse et al., 2010). Consequently, it is expected that the two underlying physiological mechanisms and psychological processes yield subjective appraisals of sleepiness, and that these appraisals determine how we plan our active life in function of past sleep and wake experiences.

Individuals are known to differ in the amount of sleep they require to function adequately during the day. While most adults need on average between 7 to 8 hrs sleep (Foley, Ancoli-Israel, Britz and Walsh, 2004), some people report they require less than 6 hrs (short sleepers) or more than 9 hours (long sleepers). The latter two groups have been shown to differ in the amount of homeostatic sleep pressure (Aeschbach, Cajochen, Landolt and Borbély, 1996). In particular, long sleepers report higher levels of subjective sleepiness after sleep deprivation, whereas short sleepers exhibit no significant increase in sleepiness levels (Aeschbach, Cajochen, Landolt and Borbély, 1996). Results from a study on the duration of the biological night

in long and short sleepers suggested that the circadian pacemaker's program might be at the origin of variability in habitual sleep duration. Long sleepers seem to experience a longer biological night as was observed in the temporal organization of the neuroendocrine function, temperature and subjective arousal (Aeschbach et al., 2003). However, at the operational level of the sleep homeostat no difference has been observed. Despite differences in total sleep time, (1) the amount of slow wave sleep, (2) the absolute spectral density of NREM sleep, (3) the evolution of the EEG spectra during sleep and (4) recovery sleep after sleep deprivation are comparable in short and long sleepers (Aeschbach et al., 1996). Apart from physiological differences, psychological differences between short and long sleepers have also been reported. Short sleepers seem to have a tendency for subclinical hypomania (Monk, Buysse, Welsh, Kennedy and Rose, 2001), whereas long sleepers are found to be more depressed on a daily basis (Hartmann, Baekeland and Zwilling, 1972; Webb, 1979). There also seems to be a difference in attitude towards sleep. Long sleepers valued sleep highly and recognized the importance of a good night's sleep. Short sleepers reported either a neutral or a negative attitude towards sleep, and considered it a waste of time (Hartmann, Baekeland and Zwilling, 1972). It seems important to consider these social-cognitive differences, as the decision to go to bed may influence the use of artificial light during evenings and affect the onset of the sleep promoting circadian signal (Aeschbach et al., 2003). As stated before, psychological processes also underlie decisions related to sleep-wake behavior. It is therefore of interest to know whether people with different habitual sleep durations integrate information of previous sleep and time of day in a similar way, or if the differences in the underlying physiological mechanisms translate in their daytime sleepiness judgments.

Functional Measurement (FM). In order to link models of sleep-wake regulation to more general models of cognitive function (Dijk and Larkin, 2004), we propose using Anderson's Functional Measurement (FM) methodology (1981, 1982, 2001). The core of FM lies in Information Integration Theory ([IIT]: Anderson, 1981). In IIT, the joint action of different stimuli defines thought and behavior. A set of observable physical stimuli S_1 , S_2 and S_3 engender concurrent psychological representations (s_1 , s_2 and s_3). This process is described by the valuation function and applies to physical stimuli as well as verbal or symbolic stimuli without physical metric (Anderson, 1977). These representations are combined into a single implicit response r

through the process of psychological integration. This internal process can be described by means of simple, empirically stated algebraic rules such as *addition*, *averaging* and *multiplication*. Finally, the result of the integration is translated into an observable response R by the response function (Anderson, 1982). The previously described sequence is illustrated in the FM diagram below (see Figure 1).

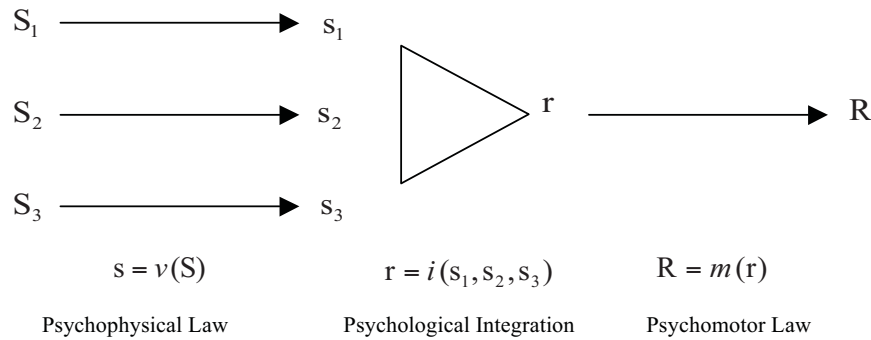


Figure 1: Functional Measurement diagram (Anderson, 1981, Weiss, 2006): S_n : observable (physical) stimuli, s_n : the subjective stimuli, r the subjective response and R the observable response, v : valuation function, i : integration function, m : response function.

The factorial design required to carry out FM experiments allows validating two premises simultaneously: (1) the algebraic rule that describes best the psychological integration that presumably took place and (2), the linearity of the response function. In a typical FM experimental set-up, stimuli are presented in all factor \times level combinations and are to be evaluated in terms of perceived intensity on a particular response scale. By means of an analysis of variance (ANOVA) the model can be checked for validity, and if this is found to be true, the data plotted in a factorial graph will reveal a particular pattern according to which algebraic rule is being used to describe the integration function.

If an additive integration rule is found, the data will show parallelism along with significant main effects and a nonsignificant interaction in the

ANOVA table. According to the parallelism theorem (Anderson, 1981) and additive integration rule implies in our case (1) that the perception of a certain level of sleepiness can be described by the addition of the magnitude of the effect of Process S and Process C, (2) that S and C do not interact in the psychological process of formulating a single response on a subjective sleepiness scale and (3) that the response measure used to translate the subjective feeling of sleepiness yields linear data.

The main difference between averaging models and additive models is that, when an averaging model applies, adding information can lower the value of the overt response instead of raising it. When testing for averaging models, stimuli from one factor are also presented without pairing them to stimuli from the other factors. For averaging models (equal weights case), parallelism should be observed in the factorial plot, except for a clear deviation from parallelism of the curve representing uncombined levels of one of the factors. In that case, in addition to significant main effects, a significant interaction should observe when performing an ANOVA including the levels of the uncombined factor.

When a multiplicative model is found, the factorial graph will exhibit a fan-like pattern when the cell means are plotted on a functional scale (i.e. spacing of the factor levels according to the marginal means). The ANOVA results will reveal a significant interaction between the two factors, with the effect located in the linear \times linear component and nonsignificant residual components (Anderson, 1981, 1982). When either one of those three integration rules is found, this implies the linearity of the response function, as monotone (ordinal) response methodology would violate the patterns of the corresponding factorial plots (for a more thorough review, see Anderson, 1977, 1981, 1982, 2001).

METHOD

Participants. Participants were recruited by means of a newsletter among students and personnel of the Vrije Universiteit Brussel. In the newsletter, they were asked to take part in an on-line survey regarding sleep quality, global sleepiness and arousal [Epworth Sleepiness Scale (ESS: Johns, 1991), Hyper Arousal Scale (HAS: Regestein, Dambrosia, Hallett, Murawski and Paine, 1993), and the Pittsburgh Sleep Quality Inventory (PSQI: Buysse, Reynolds, Monk, Berman and Kupfer, 1989)]. 159 individuals provided valid questionnaire data. Three groups were distinguished based on

their habitual sleep time (HST) reported in the PSQI: short sleepers (HST \leq 6 hrs), midrange sleepers (HST between 7 and 8 hrs) and long sleepers (HST \geq 9 hrs). The majority of the midrange sleeper group consisted of undergraduate students enrolled in the course 'Research Methods and Techniques' at the Vrije Universiteit Brussel. They were rewarded with course credits for their participation in the follow-up FM experiment. Additional long sleepers and short sleepers were recruited for the FM experiment and participation was rewarded with film tickets. From the 159 participants, 45 enrolled in the FM experiment. Thirteen individuals were considered long sleepers, three of them were males. Thirteen participants were categorized as short sleepers, two of them were males. Sixteen females and three males formed the midrange sleeper group. All participants were interviewed shortly to ensure that their adherence to a particular group was legitimate. The majority of the participants were female (82,2%) but the distribution of males and females across short, midrange and long sleepers was similar ($p = .891$, Fisher's Exact Test).

Stimuli and design. All participants were given individualized versions of the FM experiment according to their habitual sleep time (HST). The stimuli were presented in random order according to a 4×6 full-factorial design with three replications. On-screen descriptions of the time of day (Process C), HST, a reduction of HST by half or a total absence of sleep (Process S) were used as stimulus material. Additionally, levels of Process C were presented without any indication of sleep time as a qualitative test for averaging type of integration. An overview of the stimuli is displayed in Table 1.

Procedure. Ratings were obtained by means of the Karolinska Sleepiness Scale ([KSS], Åkerstedt and Gillberg, 1990), a 9-point category scale with labels ranging from "extremely alert" to "extremely sleepy, great effort to stay awake". In order to control for floor and ceiling effects, the participants went through a procedure to anchor the response scale. In this anchoring procedure, each respondent had to indicate which unique combination of stimulus levels corresponded best to their maximum level of sleepiness, and which unique combination that corresponded to their maximum level of alertness. All participants were tested separately in PC- equipped sound-proof rooms. For each stimulus combination, they were instructed to indicate which statement of the KSS described best their level of functional sleepiness in the given situation (see Table 1), considering waking up at 8 AM. KSS statements were presented on a 1024×768 pixel PC screen. Re-

sponses were made by selecting the radio buttons next to the relevant KSS-item. The experiment was designed using the FM BUILDER suite, JAVA-based software developed to conduct full-factorial FM experiments using text or pictorial stimuli (Mairesse, Hofmans and Theuns, 2008). In order to avoid non-compliance to the experiment by continuously clicking through the trials, a 1-second delay before the appearance of the next-button was included.

Table 1. Stimuli for a midrange sleeper with reported habitual sleep time of 8 hrs.

Factor	Level	Stimulus
Process S	no sleep	<i>"You did not sleep"</i>
	HST/2	<i>"You slept 4 hours"</i>
	HST	<i>"You slept 8 hours"</i>
	uncombined	no text
Process C	10 AM	<i>"It is now 10 AM"</i>
	12 AM	<i>"It is now 12 AM"</i>
	2 PM	<i>"It is now 2 PM"</i>
	4 PM	<i>"It is now 4 PM"</i>
	6 PM	<i>"It is now 6 PM"</i>
	8 PM	<i>"It is now 8 PM"</i>

RESULTS

Descriptives. Differences in age, habitual sleep time, sleep quality and global sleepiness across short, midrange and long sleepers are displayed in Table 2. Age, habitual sleep time and total PSQI scores differ significantly between groups. Post-hoc analyses[†] show that only the midrange sleeper

[†] For all post-hoc tests a Tukey HSD correction is applied

group and the long sleeper group differ significantly in age (mean difference = 9.83 yrs, $p < .005$). Midrange sleepers report significantly longer HSTs than short sleepers (mean difference = 1.50 hrs, $p < .001$) and shorter HSTs than long sleepers (mean difference = 2.21 hrs, $p < .001$). HST in short sleepers is also significantly shorter than in long sleepers (mean difference = 3.71, $p < .001$). Total PSQI scores are significantly larger in short sleepers than in midrange sleepers (mean difference = 1.96, $p < .05$) and than in long sleepers (mean difference = 3.54, $p < .001$). These results, however, do not imply that sleep quality in short sleepers is lower than in midrange and long sleepers. After controlling for age and habitual sleep time (component 3), differences in total PSQI scores do not reach statistical significance [$F(2,40) = 1.052, p = .359$].

Cognitive algebra. Visual inspection of the data averaged over participants reveals parallelism in all three groups (see Figure 2). According to Andersons' parallelism theorem (1981, 1982), these results suggest an additive integration of time of day and previous sleep, and at the same time imply linearity of the response scale. Statistically, this is reflected in the presence of significant main effects and in the absence of a significant interaction. The results of the omnibus ANOVA are displayed in Table 3.

Model selection. Our results provide visual support for a general adding-type integration rule over an averaging rule. Statistically, ANOVAs including the uncombined factor revealed no significant interaction of Process S and Process C [$F(15,630) = 1.757, p = .104$, partial $\eta^2 = .04$] and no between-group interaction of both processes [$F(30,630) = .535, p = .899$, partial $\eta^2 = .03$].

Table 2. Age, habitual sleep time (HST) and global measures of arousal/sleepiness and sleep quality: Hyper Arousal Scale (HAS), Epworth Sleepiness Scale (ESS) and Pittsburgh Sleep Quality Index (PSQI). Age is expressed in years, HST in hours. Values are significant at $p < .05^*$, $p < .005^{**}$ and $p < .001^{***}$. Dependent variables displayed in bold are controlled for age differences by means of (multivariate) analyses of covariance.

Test	Short Sleepers		Midrange Sleepers		Long Sleepers		<i>F</i> -value
	mean	SD	mean	SD	mean	SD	
Age	30.8 5	7.13	33.5 3	9.12	23.6 9	5.98	6.31**
HAS	43.3 8	8.62	41.3 7	8.58	44.6 9	7.92	.63
HST	5.77	.39	7.26	.42	9.48	.50	206.51** *
ESS	8.54	3.18	8.79	3.70	7.69	5.22	.29
PSQI							
Total	7.38	2.72	5.42	2.01	3.85	1.46	3.81*
Comp. 1	1.62	.51	1.21	.79	1.00	.58	2.03
Comp. 2	1.77	1.09	1.05	1.03	1.00	.91	1.81
Comp. 3	1.15	.38	.68	.48	.00	.00	20.87***
Comp. 4	.54	.97	.16	.37	.08	.28	2.70
Comp. 5	1.23	.60	1.11	.46	1.00	.41	.83
Comp. 6	.00	.00	.21	.71	.00	.00	1.79
Comp. 7	1.08	.64	1.21	.63	.77	.60	1.31

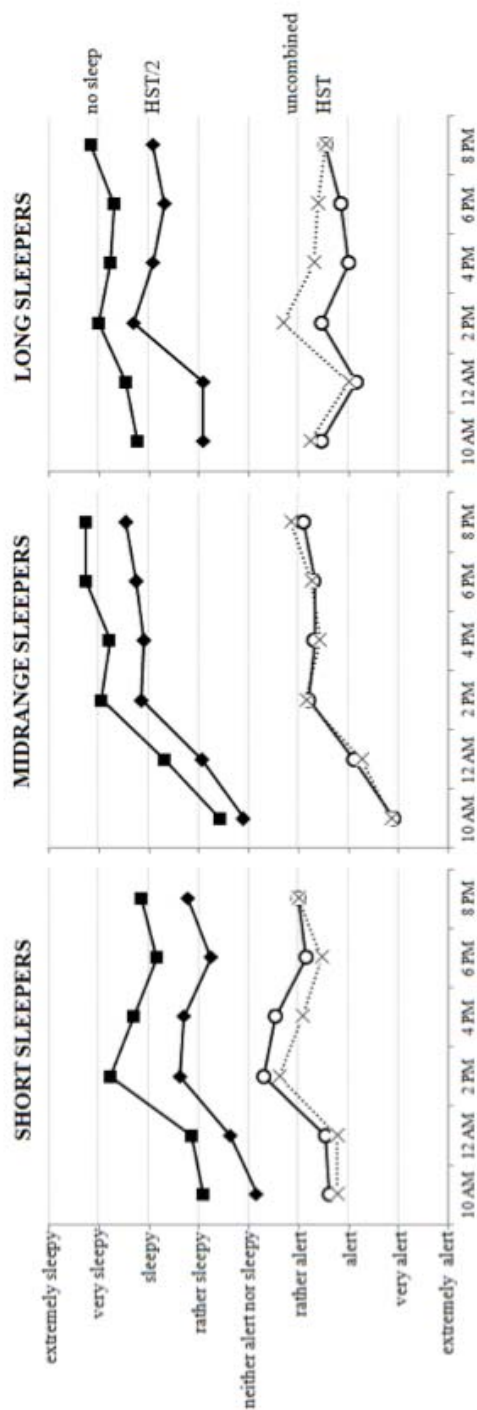


Figure 2. Median KSS scores averaged over participants plotted against time of day (Process C): 10 AM, 12 AM, 2 PM, 4 PM, 6 PM and 8 PM. Separate lines represent levels of Process S: no sleep (■), HST/2 (◆) and HST (○). The dashed line shows the results for the uncombined Process C (×).

Table 3. Omnibus ANOVA with exclusion of the uncombined levels of Process C. A Greenhouse-Geisser correction on p -values is used for violations of sphericity.

Factor	df	Error df	F -value	p -value	Partial η^2
S	2	84	279.893	.000	.870
$S \times group$	4	84	5.444	.003	.206
C	5	210	11.355	.000	.213
$C \times group$	10	210	1.653	.161	.073
$S \times C$	10	420	1.951	.084	.044
$S \times C \times group$	20	420	.603	.816	.028
$group$	2	42	.625	.540	.029

Single subject analyses. Whereas group analyses are often useful to uncover a general integration model, in some cases however, the co-occurrence of different patterns in the data (i.e. reflective patterns, see Mairesse et al., 2007) balances out the group means causing a failure to detect specific effect in the group ANOVA. Single subject analyses provide a protection against such ecological fallacy and a firmer validation of the underlying integration model. Adherence to a particular integration rule was defined based on visual inspection of individual factorial plots and individual ANOVAs. Parallelism was observed in 9 out of 13 short sleepers; two individuals showed no integration of levels of Process C, one participant showed almost no effect of Process S, and one pattern remained indeterminate. In the midrange sleeper group, 10 participants used an additive integration rule and five participants a multiplicative one. One participant reported not to react on differences in Process C, and three participants followed undistinguishable integration rules. Seven out of 13 long sleepers followed an additive integration rule and one subject followed a multiplicative rule. There was no effect of Process C in one participant and four patterns were

not identifiable. In summary, the majority of the participants seem to follow an adding-type integration rule, whereas a smaller amount of participants integrates both homeostatic and circadian processes according to a multiplicative rule. Examples of the different integration patterns are displayed in Figure 3.

Group differences. As apparent from Figure 2, the spacing between the curves representing Process S levels differs between groups. Statistically, this difference is supported by a significant $S \times \text{group}$ interaction. *A posteriori* tests showed statistically significant differences between mean levels of judged sleepiness when information on sleep restriction is presented (HST/2 level) between short and midrange sleepers, [$t(42) = -.855, p < .01^\dagger$, $R^2 = .16$] and between short and long sleepers [$t(42) = -.794, p < .05, R^2 = .21$]. With respect to information on sleep deprivation (no sleep level), we found only a difference between short and long sleepers [$t(42) = -.857, p < .05, R^2 = .18$]. We found no statistically significant differences between groups with respect to time of day variations of sleepiness judgments. However, visual inspection of the data suggests a difference in curvature of the data between groups. Statistically, this difference is only reflected by a trend in the quadratic group \times Process C interaction term [$F(2,42) = 2.523, p = .092$, partial $\eta^2 = .11$].

Relative range indices. Weight parameters provide an indication of the importance of a particular factor. However, in adding-type models such as found in this study, weights cannot be determined due to a lack of uniqueness in the parameter estimations (Anderson, 1982). Instead, an index of importance can be calculated using the relative range of the factors (*RRI*). Let R_X be the range of variable X and R_i be the range of any variable, then the *RRI* can be written as Eq. (1)

$$\text{Eq. (1)} \quad RRI_X = 100(R_X / \sum R_i)$$

† one-tailed significance

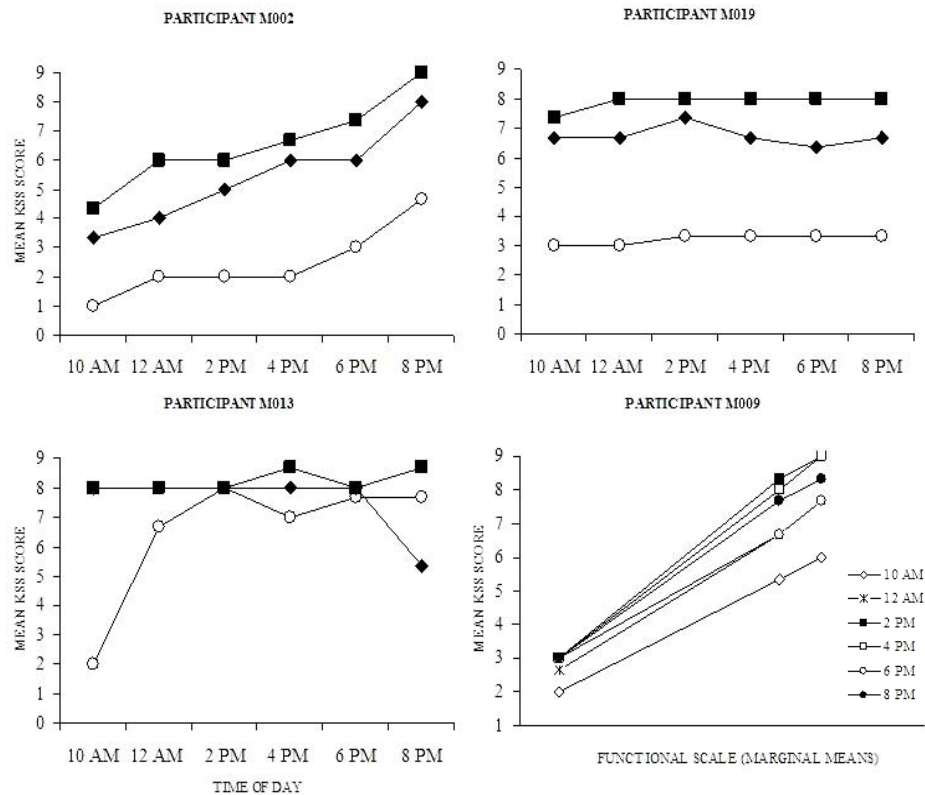


Figure 3. Individual factorial plots of four midrange participants. The upper left panel shows the factorial plot of participant M002 following an additive integration rule. In the upper right panel, the factorial plot of participant M0019 shows no clear integration of Process C. In the lower left panel, the factorial plots shows no identifiable integration pattern (participant M013). In all three aforementioned graphs the mean KSS scores plotted against time of day (Process C): 10 AM, 12 AM, 2 PM, 4 PM, 6 PM and 8 PM. Separate lines represent levels of Process S: no sleep (■), HST/2 (◆) and HST (○). In the lower right panel the cell means of all levels of Process C are plotted against the marginal means (functional scale) to reveal the linear fan pattern proper to a multiplication integration rule (participant M009).

In order to allow computation of the *RRIs*, (1) the selection of stimuli should correspond to the maximal range or to an ecologically valid range of stimuli, (2) a linear model must hold and (3) the response scale should be linear (Anderson, 1982). At first sight, defining an index of importance other than the partial η^2 seems of little use. However, partial η^2 is not only dependent on the three previously stated requirements, but also on the spacing of the stimulus levels within the range. The *RRI*, on the other hand, has the advantage of being defined in terms of the response range, without depending on stimulus scaling (Anderson, 1982). The *RRIs* for all groups are displayed in Table 4. Contrast tests showed statistically significant differences between *RRIs* of short and long sleepers, [$t(42) = -1.805, p < .05, R^2 = .09$]. None of the remaining between-group comparisons were statistically significant [short sleepers vs. midrange sleepers: $t(42) = -1.470, p = .075, R^2 = .05$; midrange sleepers vs. long sleepers: $t(42) = -.497, p = .311, R^2 = .01$].

Table 4. Mean (SD) Relative Range Indices for short, midrange and long sleepers. *RRIs* were calculated for each participant separately and then averaged within groups.

	Short sleepers		Midrange sleepers		Long sleepers	
Factor	Mean	SD	Mean	SD	Mean	SD
<i>Process S</i>	47.59	27.28	58.47	17.04	62.15	17.29
<i>Process C</i>	52.41		41.53		37.85	

Functional scale values. When an additive model holds, marginal means provide an estimation of the gross stimulus values (s_i 's in the FM diagram in Figure 1) and are justifiable for functional scaling purposes (Anderson, 1982). Functional scale values are standardized in order to allow illustrative between-group comparisons. The functional scale values and rescaled functional scale values of both factors across all three groups are displayed in Table 5. Functional and rescaled functional values for Process S and C are displayed in Figure 4 and Figure 5 respectively to reveal the shape of the homeostat and the circadian oscillator in sleepiness judgments.

Table 5. Gross functional scale values (marginal means) and rescaled functional scale values (RFSVs) of homeostatic and circadian factors derived for short, midrange and long sleepers.

Factor	Level	Short sleepers		Midrange sleepers		Long sleepers	
		FSV	RFSV	FSV	RFSV	FSV	RFSV
<i>Process S</i>	HST	3.97	0	3.34	0	3.26	0
	HST/2	5.82	64	6.68	81.72	6.62	75.29
	no sleep	6.86	100	7.42	100	7.72	100
	10 AM	4.72	0	4.26	0	5.56	17.65
	12 AM	5.00	18.03	5.18	18.03	5.41	0
<i>Process C</i>	2 PM	6.28	100	6.30	100	6.28	100
	4 PM	6.03	83.61	6.19	83.61	5.90	55.88
	6 PM	5.49	49.18	6.40	49.18	5.85	50
	8 PM	5.79	68.85	6.54	68.85	6.18	88.24

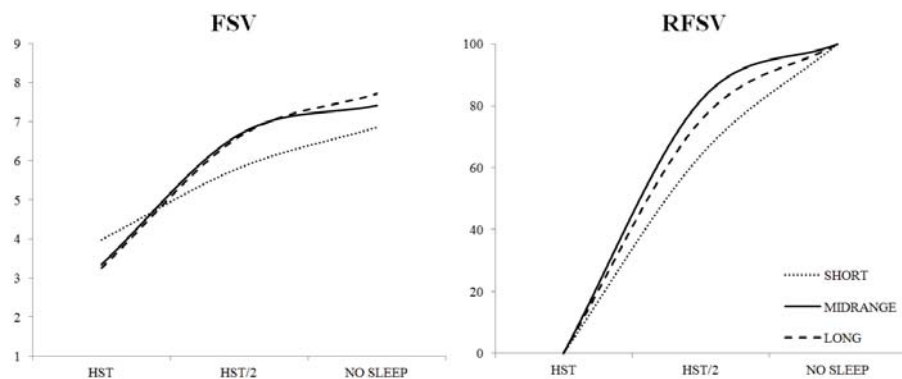


Figure 4. Functional and rescaled functional scale values of Process S derived for short, midrange and long sleepers. For the homeostatic factor, HST was rescaled as 0 (minimum sleepiness) and the no sleep-level as 100 (maximum sleepiness). Values are plotted along levels of Process S to reveal the shape of the sleep homeostat.

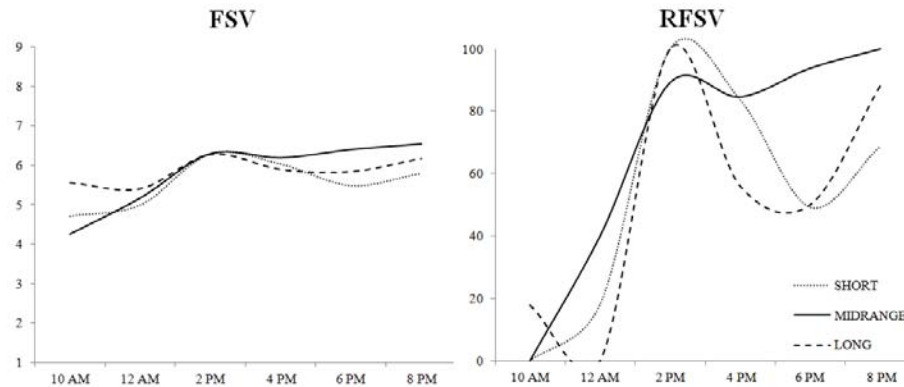


Figure 5. Functional and rescaled functional scale values of Process C derived for short, midrange and long sleepers. Within each group, the level of Process C corresponding with the maximum scale value was rescaled as 100; the level of Process C corresponding with the minimum scale value was rescaled as 0. Values are plotted along the biological day to reveal ultradian/circadian rhythmicity.

DISCUSSION

In general, our results show that the functional integration of homeostatic and circadian processes for judgments of sleepiness is best described by an additive rule. The observed parallelism in the group factorial plots simultaneously supports the additivity of previous sleep and time of day, as well as the linearity of the response scale (KSS). Group findings are also supported at the individual level and they are in line with what was previously reported in two similar integration tasks using ratings of experienced and judged sleepiness by means of the KSS and the VAS (Mairesse et al., 2007) and in performance measures (Dijk and Larkin, 2004).

Sleep homeostat. In line with what was previously described in studies using electrophysiological, temperature and subjective measurements of sleepiness in constant routine protocols (Aeschbach et al., 1996, 2001), our results provide support for the fact that short sleepers experience a higher homeostatic sleep pressure. An explanation for these findings in the context of subjective sleepiness judgments is that past experience of sleepiness in various conditions results in subjective appraisals of sleepiness based on

time of day and previous sleep paralleling the physiological components of sleepiness. No between-group statistical differences in functional sleepiness have been observed at habitual levels of previous sleep (HST). On average, neither short nor long sleepers judge daytime sleepiness to be different from midrange sleepers when considering their habitual sleep time. Moreover, in all groups ESS scores are similar, suggesting that actual global daytime sleepiness is equivalent in short; midrange and long sleepers despite large differences in habitual sleep time. However, when prompted to judge daytime sleepiness as if they were restricted or deprived from sleep, short sleepers report statistically lower levels of functional sleepiness compared to midrange sleepers (HST/2) and long sleepers (HST/2 and no sleep). Short sleepers judge their tolerance to sleep restriction and deprivation to be higher in comparison to long sleepers, which is consistent with what was described early on by Hartmann et al. (1972) and confirmed by Aeschbach et al. (1996, 2001). Tentative plots of smoothed *RFSV*s curves illustrate that the saturating exponential increase in functional sleepiness is related to a decrease in prior sleep time and that this phenomenon is dissimilar across groups. The reduced curvature in the smoothed *RFSV* curve for short sleepers indicates the higher judged tolerance for sleep restriction as compared to midrange and long sleepers. This greater tolerance is also translated in the significant difference in relative importance of Process S in functional sleepiness ratings. In long sleepers, more than 60% of the response range is accounted for by manipulations of Process S, whereas in short sleepers Process S covers less than 50% of the stimulus range. As short sleepers have the experience of being less vulnerable to sleep deprivation, their judgments are likely to be less affected by manipulations of previous sleep in comparison to long sleepers.

Circadian/ultradian variability. Our results show that, on average, judgments of sleepiness over the day do not differ between short, midrange and long sleepers. As visible from the functional scale value plot (left panel of Figure 5), roughly similar profiles emerge for short, midrange and long sleepers, with a turning point located around 2PM, most likely corresponding to the post-lunch dip in actual alertness and performance previously described in Monk, Buysse, Reynolds and Kupfer (1996) and in Horne and Baulk (2004). Statistically, this difference is supported by a marginal significant trend in the group \times Process C quadratic interaction term. Rescaled functional scale values (right panel of Figure 5) show the dissimilarities in the curvature of the data between groups by emphasizing differences be-

tween maximal and minimal levels of judged sleepiness more clearly. Sleepiness during the day is judged to be minimal at 10 AM in short and midrange sleepers, while for long sleepers minimal sleepiness occurs around midday. Short and long sleepers judge sleepiness to be maximal around the post-lunch dip (2PM), whereas for midrange sleepers maximal sleepiness occurs at 8 PM. Furthermore, long sleepers report higher levels of judged sleepiness during morning hours (10 AM). In both short and long sleepers, levels of judged sleepiness slightly decrease in the early evening. This period coincides with the *sleep forbidden zone* (Lavie, 1986), a time stretch of increased wakefulness occurring a few hours before habitual sleep onset supposedly caused by the evening surge of thyrotropin (Pereira and Alves, 2011).

Taken together, our results suggest a longer biological night in long sleepers, which is in line with previous findings by Hartmann et al. (1972) and Aeschbach et al. (2003). Indeed, a delayed onset of the circadian wake promoting regulator implies higher levels of sleepiness during morning hours that coincide with the habitual time in bed. The presence of high plasma melatonin during this period increases sleep propensity (Hughes and Badia, 1997) and for long sleepers, the experience of being awake at that moment most likely translates in their sleepiness judgments. It remains unclear why midrange sleepers do not exhibit a similar judgment pattern. A possible explanation is that, due to being aware of their "unusual" habitual sleep duration (see Hartmann et al., 1972), short and long sleepers are somewhat better judges of sleepiness as compared to midrange sleepers, and are therefore better able to distinguish variations in sleepiness during the day.

CONCLUSION

The aim of the present investigation was to determine whether habitual short, long and midrange sleepers exhibit similar integration patterns or whether the biological underpinnings of habitual sleep time causes differences in the how these groups estimate their sleepiness. Our results show an additive integration rule for homeostatic and circadian determinants of daytime sleepiness in short, long and midrange sleepers. However, relative range indices of sleepiness judgments suggest that short sleepers tolerate higher levels of sleep pressure in comparison to long sleepers. In turn, cir-

cadian variability play a less prominent role in judgments of daytime sleepiness in long sleepers. Reasonably, sleep-wake behavior is not only under the influence of physiological processes, but also depend on subjective appraisals of sleepiness reflected by sleepiness judgments. So, physiological and psychological processes both affect for instance the decision to go to bed at a specific time of day, influencing the onset of the sleep promoting processes (Aeschbach et al., 2003), resulting in a perpetuation of diverging sleeping habits in long and short sleepers. This has important implications considering that shorter and longer sleep durations are associated with higher all-cause mortality. However, it remains unclear if self-reports are sufficient to differentiate between natural, and thus possibly healthy short or long sleepers or sleep restrictors or over-extendors potentially damaging their health (Grandner and Patel, 2009). Therefore, further investigation relating sleepiness judgments and actual sleepiness, based on carefully selected and electrophysiologically validated groups, is needed.

REFERENCES

- Aeschbach, D., Cajochen, C., Landolt, H. and Borbély, A.A. (1996). Homeostatic sleep regulation in habitual short sleepers and long sleepers. *American Journal of Physiology*, 270(1Pt2), R41-53.
- Aeschbach, D., Postolache, T.T., Sher, L., Matthews, J.R., Jackson, M.A. and Wehr, T.A. (2001). Evidence from the waking electroencephalogram that short sleepers live under higher homeostatic sleep pressure than long sleepers. *Neuroscience*, 102(3), 493-502.
- Aeschbach, D., Sher, L., Postolache, T.T., Matthews, J.R., Jackson, M.A. and Wehr, T.A. (2003). A longer biological night in long sleepers than in short sleepers. *The Journal of Clinical Endocrinology and Metabolism*, 88(1), 26-30.
- Åkerstedt, T. and Folkard, S. (1994). Prediction of intentional and unintentional sleep onset. In R. Ogilvie and J.R. Harsch (Eds.), *Sleep onset: normal and abnormal processes* (pp. 73-87). Washington DC: American Psychological Association.
- Åkerstedt, T. and Folkard, S. (1995). Validation of the S and C components of the three-process model of alertness regulation. *Sleep*, 18(1), 1-6.
- Åkerstedt, T. and Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience*, 52, 29-37.
- Anderson, N. H. (2001). *Empirical direction in design and analysis*. New Jersey: Lawrence Erlbaum Associates.
- Anderson, N. H., and Shanteau, J. (1977). Weak inference with linear models. *Psychological Bulletin*, 84, 1155-1170.

- Anderson, N.H. (1981). *Foundations of information integration theory*. New York: Academic Press.
- Anderson, N.H. (1982). *Methods of information integration theory*. New York: Academic Press.
- Borbély, A. A. (1982). A two-process model of sleep regulation. *Human Neurobiology*, 1, 195-204.
- Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R. and Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Journal of Psychiatric Research*, 28(2), 193-213.
- Dijk, D. J., and Larkin, W. (2004). Fatigue and performance models: general background and commentary on the circadian alertness simulator for fatigue risk assessment in transportation. *Aviation, Space, and Environmental Medicine*, 75, A119-A121.
- Foley, D., Ancoli-Israel, S., Britz, P. and Walsh, J. (2004). Sleep disturbances and chronic disease in older adults: Results of the 2003 National Sleep Foundation Sleep in America Survey. *Journal of Psychosomatic Research*, 56(5), 497-502.
- Folkard S., Åkerstedt, T., Macdonald, I., Tucker, P. and Spencer, M. (1999). Beyond the three-process model of alertness: Estimating phase, time of shift and successive night effects. *Journal of Biological Rhythms*, 14, 577-587.
- Fulcher, B.D., Phillips, A.J.K., Robinson, P.A. (2010). Quantitative physiologically based modeling of subjective fatigue during sleep deprivation. *Journal of Theoretical Biology*, 264(2), 407-419.
- Grandner, M. A. and Patel, N. P. (2009). From Sleep Duration to Mortality: Implications of Meta-Analysis and Future Directions. *Journal of Sleep Research*, 18, 145-147.
- Hartmann, E., Baekeland, F. and Zwillig, G.R. (1972). Psychological differences between long and short sleepers. *Archives of General Psychiatry*, 26, 463-468.
- Horne, J.A. and Baulk, S.D. (2004). Awareness of sleepiness while driving. *Psychophysiology*, 41, 161-165.
- Horne, J.A. and Burley, C.V. (2010). We know when we are sleepy: Subjective versus objective measurements of moderate sleepiness in healthy adults. *Biological Psychology*, 83(3), 266-268.
- Hughes, R.J. and Badia, P. (1997). Sleep-promoting and hypothermic effects of daytime melatonin administration in humans. *Sleep*, 20, 124-131.
- Jewett, M. E., and Kronauer, R. E. (1999). Interactive Mathematical Models of Subjective Alertness and Cognitive Throughput in Humans. *Journal of Biological Rhythms*, 14, 588-597.
- Johns, M.W. (1991). A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*, 14, 540-545.
- Lavie P. (1986). Ultrashort sleep-waking schedule. III. 'Gates' and 'Forbidden Zones' for sleep. *Electroencephalography and Clinical Neurophysiology*. 63(5), 414-25.
- Mairesse, O., Hofmans, J. and Theuns, P. (2008). The Functional Measurement Experiment Builder suite: two JAVA-based programs to generate and run Functional Measurement

- experiments. *Behavioral Research Methods*, 40(2), 408-412.
- Mairesse, O., Hofmans, J., De Valck, E., Cluydts, R. and Theuns, P. (2007). On the *linearity* of subjective sleepiness measures. *Teorie and Modelli*, XII, 289-298.
- Mairesse, O., Hofmans, J., Neu, D., de Oliveira, A., Cluydts, R. and Theuns, P. (2010). The algebra of sleepiness: investigating the interaction of homeostatic (S) and circadian (C) processes in sleepiness using linear metrics. *Psicologica*, 31, 541-559.
- Monk, T.H., Buysse, D.J., Reynolds C.F. III and Kupfer, D.J. (1996). Circadian determinants of the postlunch dip in performance. *Chronobiology International*, 13, 123-133.
- Monk, T.H., Buysse, D.J., Welsch, D.K., Kennedy, K.S. and Rose, L.R. (2001). A sleep diary and questionnaire study of naturally short sleepers. *Journal of Sleep Research*, 10, 173-179.
- Odle-Dusseau, H.N., Bradley, J.L. and Pilcher, J.J. (2010). Subjective perceptions of the effects of sustained performance under sleep-deprivation conditions. *Chronobiology International*, 27(2), 318-333.
- Pereira, J.C. and Alves R.C. (2011). The “Forbidden Zone for Sleep” might be caused by the evening thyrotropin surge and its biological purpose is to enhance survival: a hypothesis. *Sleep Science*, 4(3), 105-109.
- Regestein, Q.R., Dambrosia, J., Hallett, M., Murawski, B. and Paine, M. (1993). Daytime alertness in patients with primary insomnia. *American Journal of Psychiatry*, 150, 1529-1534.
- Webb, W.B. (1979). Are short and long sleepers different? *Psychological reports*, 44, 259-264.
- Weiss, D.J. (2006). *Analysis of variance and functional measurement: A practical guide*. New York: Oxford University Press.