Predictions from the Three-Process Model of Alertness

Torbjörn Åkerstedt, Simon Folkard, and Christian Portin

Åkerstedt T, Folkard S, Portin C. ***Predictions from the three-process model of alertness.*** **Aviat Space Environ Med 2004; 75(3, Suppl.):A75–83.**

This paper summarizes a computer model for predicting alertness/performance in daily life. The model uses the timing of work hours (and/or sleep hours) as input and uses both a circadian and a homeostatic component (amount of prior wake and amount of prior sleep) that are summed to yield predicted subjective alertness (on a scale between 1 and 21). The model also includes prediction of psychomotor performance on various tasks, an identification of levels at which the risk of performance/alertness impairment start, as well as a prediction of sleep latency and time of awakening from sleep. The model is currently being used to evaluate work/rest schedules for navy, airline and railway applications. It is also used for teaching sleep/wake regulation and for generating research hypotheses.

Keywords: alertness, performance, sleep, model.

SHIFT WORK AND OTHER types of non-daywork (and sleep/wake patterns) strongly affect alertness and sleep (1). Shift work also results in markedly increased sleepiness and EEG-changes characteristic of sleep (39,51,53). The increased sleepiness may be related to the higher accident risk in connection with night work (37,44,47) and to similar performance decrements (23).

Clearly, it would be of practical and theoretical interest to be able to predict the effects of altered sleep/wake patterns. Several authors have tried to do this in more or less elaborate ways. Monk, working with subjective alertness and forced desynchronization, found one circadian influence related to body temperature and another related to time since sleep (45). Minors and Waterhouse presented a model based on time of day, time since prior sleep, and subjective quality of prior sleep and nap (43). In a somewhat different approach, Spencer found that performance on a "digit symbol substitution test" could be explained mainly by a sinusoidal and a cubic component (48). However, none of these approaches were validated and they lacked inclusion of sleep.

Our own model, the Three Process Model of Alertness (TPMA), was first presented in 1987 (24,25), and subsequently expanded (to include sleep) and validated (3–6,26–28). In addition, there have been several precursory attempts to model sleep regulation (12,14,41) which, although related and inspiring, did not specifically set out to predict alertness and performance in the manner that our model does.

The purpose of the TPMA was to provide the following: 1) an integrated and quantitative description of the main factors that affect alertness and alertness-related performance; 2) a way of predicting alertness from knowledge of sleep/wake patterns or only work patterns; 3) quantitative support for evaluation of work/rest (watch) schedules in terms of alertness, fatigue, performance impairment and accident risk; 4) a quantitative tool for monitoring the state of alertness and fatigue-related error risk; 5) an educational tool for teaching sleep/wake regulation and fatigue consciousness; and 6) a tool for generating research hypotheses regarding sleep/wake regulation and its consequences.

One identified user group for the Three Process Model is schedulers and planning staff who want to evaluate the fatigue and performance effects of particular work schedules, such as those used by the road, sea, air, and railway transportation sectors. A second user group consists of occupational specialists and hygienists who work in these areas and who need information on human physiology and performance in relation to irregular work hours.

THE MODEL

Concepts and Assumptions of the Model

One main cause of shift work-induced sleepiness is the circadian pacemaker and its effect on physiology and psychology. It is clear that, in the absence of circadian regulation, irregular work hours would not constitute a problem, since the circadian low in the early morning would not reduce alertness, and since daytime sleep would not be interfered with by the increase in metabolism during the day. A number of studies involving normal night time sleep (31), sleep deprivation (34), spontaneous desynchronization (16,55), and forced desynchronization (20,30) have shown that alertness

From the IPM (T. Åkerstedt, C. Portin) and Department of Public Health Sciences, Karolinska Institutet, Stockholm, Sweden (T. Åkerstedt); and Body Rhythms and Shiftwork Centre, Department of Psychology, University of Wales Swansea, Swansea, Wales, UK (S. Folkard).

Address reprint requests to: T Åkerstedt, Ph.D., who is Professor of Behavioral Physiology, Karolinska Institutet and National Institute for Psychosocial Medicine (Deputy director), IPM/Karolinska Institutet, Box 230, 17177 Stockholm, Sweden; torbjorn.akerstedt@ipm.ki.se.

and performance display a time of day pattern with a maximum in the late afternoon and a trough in the early morning around 05:00.

A second likely cause of sleepiness in shift workers is the shortened sleep in connection with morning and night shifts (2,11,33,49,53,54). However, there are no experimental data linking this shortened sleep to reduced alertness or performance in a real life shift work setting. On the other hand, studies on sleep reduction in general indicate that a 2- to 4-h reduction of night sleep will reduce alertness, although probably not enough to make it the major cause of night shift sleepiness—the recuperative value of the last 2–3 h of an 8-h sleep seems quite low (35). Importantly, the effect of restricted sleep seems to accumulate, at least up to some upper asymptote (15,56).

A strong time of day (circadian) effect is the reason for the truncated morning sleep after night work (8). As bedtime is delayed from the night hours, sleep length will fall to about 4.5 h for morning to noon bedtimes, and then recover as the day progresses into evening. The same effect has been demonstrated under conditions of spontaneous (16) or forced (19) desynchronization. Thus, sleep is very difficult at the acrophase (maximum) of the body temperature rhythm and very easy at the nadir (minimum). One should bear in mind, though, that sleep length is also determined by prior wakefulness. Thus, a 5-h sleep at noon after a night shift would shrink to 3.5 h if a 2-h nap were permitted during the night, or to 2 h if a full night sleep (7–8 h) were permitted (9).

The reasons for the short sleep before the morning shift include the need to terminate sleep very early in the morning, and the individuals being unable to advance their bedtime to fully compensate for this change. The latter failure may be partly social but there is also a strong circadian influence on sleep latency, making early initiation of sleep very difficult (10,29). This early evening time of sleep resistance has been called the "forbidden zone" for sleep (42).

A sleep shortened through truncation will also increase the time spent awake. This factor, which is also increased by delaying work hours, has not received much attention in research in shift work. Data from two early experimental sleep deprivation studies, however, clearly show a pronounced fall of alertness and performance with time awake, leveling out toward days 3–4 (controlling for circadian influence) (34,57). More recent studies have also found the same phenomenon of a gradual fall in alertness/performance in connection with forced desynchronization (20,26). The effects of prior time awake should be viewed against the fact that the night shift starts 10–16 h after rising, in contrast to the 1–2 h of the morning shift, or the 4–6 h of the afternoon shift. Thus, a night shift is usually preceded by an extended period of time awake, compared with the morning and evening work periods. Similarly, an early start to work will involve a longer period of wakefulness at any subsequent time of day than a later start and thereby increase afternoon sleepiness (40). The shorter sleep associated with early starts will also increase afternoon sleepiness.

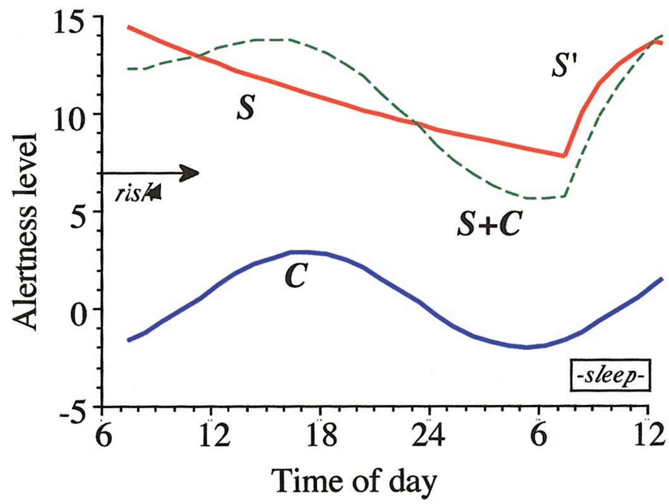

Fig. 1. The components of the Three Process Model of Alertness.

Components of the Model

Alertness and the components: Using subjective alertness data from a number of experiments of altered sleep/wake patterns we found that alertness was predictable from three parameters: S, C, and W (**Fig. 1**). Process C represents sleepiness due to circadian influences and has a sinusoidal form with an afternoon peak. Process S is an exponential function representing the time since awakening, and, therefore, is highest on awakening, with an initially steep rate of drop declining toward its lower asymptote over the course of the waking period. At sleep onset, process S is reversed and called S′ and recovery occurs in an exponential fashion that initially increases very rapidly but subsequently levels off toward an upper asymptote. Total recovery is usually accomplished in 8 h. The final component (not in Fig. 1) is the wakeup Process W, or sleep inertia. Since this variable was not part of the modeling tasks of the workshop it will not be discussed here. Since the original publication of the model we have added a process U, for "ultradian," to simulate the afternoon dip in alertness.

The parameters of the model are as follow (default values within parentheses):

$S = [Sa - Le^{(-0.0353t)} + L]$; where Sa = value of S (14) at awakening, L = lower asymptote (2.4), t = time since awakening.

$S' = U - (U - Sr)e^{-0.381t}$; where Sr = value of S at retiring (7.96); U = upper asymptote(14.3); and t = time since falling asleep.

$C = M\cos(t - p); \pi/12$; where M = amplitude (2.5), p = acrophase (in decimal hours), and t = time of day (in decimal hours).

S + C = the alertness prediction (excluding W).

The arrow at "7" indicates the level of risk (see below).

The values in parentheses are the present default values of the model.

$U = \text{mesor} - \text{Mu} * \cos\{2 * \pi/\text{period} * [t - (p + pU)]\}$

$W = \text{WInit} * e^{-\text{WDecay}} * t$; WInit = 5.7, WDecay = 0.65692, t = time since awakening.

For $S' \leq 12.5$, then: $S' = \text{prevS} + [U - (U - 12{,}5)e^{-0.381t}] - 12.5$; t = time since prevS.

The final function presented above imposes a limit on the value of S′ so that the speed of recovery cannot exceed that at a critical value on the x-axis. Otherwise it would always be possible to recover from great amounts of sleep loss within an 8-h sleep episode. The optimal level is presently being evaluated, but it seems the critical value should be set to 12.5 to yield a reasonable representation of, for example, chronic sleep loss data. Thus, at values lower than 12.5 there will be no increase in speed of recovery.

The predicted alertness is expressed as the arithmetic sum of the two (W presently excluded) functions above (S + C in Fig. 1). The scale of the model ranges normally from 1–21 (and was originally a visual analog scale), but in practice "3" corresponds to extreme sleepiness, "14" to high alertness, and "7" to a sleepiness threshold (EOG slow eye movements) (3,26). In Fig. 1 S + C shows predicted alertness when wakefulness is extended by 8 h (to 24 h), as is frequently the case, for example, with a first night shift. This particular prediction assumes that awakening occurs at 07:00 after an 8-h sleep, and thereafter no sleep occurs (due to the night shift) until 07:00 the following morning. The combined effect of S + C (long time awake and the circadian downswing) yields a fall of alertness during the night, with a trough in the early morning. After sleep is started, the steep recovery of factor S′, together with the circadian upswing causes a rapid increase in (latent) alertness during sleep.

The ultradian component: Many studies have indicated an afternoon slump in alertness regardless of whether food intake has occurred at lunch. A common assumption is that this slump is due to a 12-h component in alertness. This part of the model uses a 12-h rhythm with an amplitude of 0.5 units and an adjustable phase, set 3 h before the acrophase of C.

Performance: Performance data have been obtained from a number of mainly laboratory studies. The new parameters have been simulated using standard settings and a linear regression. Among them is the number of incidents (two wheels outside the road) or accidents (four wheels outside) in a driving simulator provided by Horne and Reyner (58). This means that we now can predict accident risk (at least in a driving simulator) of a particular work schedule. Number of hits in a performance tests have been obtained from a laboratory study (6) and mean performance on a reaction time test has been obtained from another laboratory study with 24 h of continuous wakefulness.

Handling Sleep

As touched on above, a model intended to predict the sleepiness-inducing effects of work schedules would need to be able to model sleep. Otherwise, the model could only be used on work schedules in which sleep behavior is known. This would severely limit the usefulness of the model as knowledge of work schedule-induced sleep behavior is probably the exception rather than the rule.

Sleep latency: Sleep latency prediction was discussed by Borbély and Daan as a direction for extending the two-process model (13,17), but mainly as a theoretical exercise. We, therefore, used a series of experimental studies to model a function to transfer the alertness prediction of our alertness model into sleep latency values (5). The result was: $y = 0.56*10^{0.12x}$, where x = predicted alertness and y = sleep latency in minutes. This means that sleep latency starts around 0.5 min for the lowest level of predicted alertness (alertness score = 1). Predicted alertness rises with increasing sleep latency. At normal waking levels of predicted alertness (alertness = 10–12), sleep latency is around 10 min. At very high levels of predicted alertness (alertness = 13–17), sleep latency exceeds 20 min. These high levels of long sleep latencies occur for day time sleep episodes that have been preceded by very short periods of prior waking.

Interestingly, the relationship between sleep latency and alertness predictions from the model is curvilinear. This seems intuitively logical, considering that the mental attitude toward initiating a sleep episode may be disproportionally more negative at high levels of alertness compared with low levels—sleep attempts at high alertness may be seen as futile and aborted. In terms of predicted alertness values and their verbal interpretation (3), a sleep latency of 1 min corresponds to a predicted alertness of 3 and a subjective rating of "very sleepy, fighting sleep, an effort to keep awake." The clinical criterion of a 5-min latency corresponds to a predicted alertness of 7–8, verbally close to "sleepy but not fighting sleep." Long latencies (>20 min) correspond to predicted alertness of 13–14 and ratings of "alert" to "very alert."

Sleep termination: To model the sleep offset function we assumed that an awakening from sleep will occur whenever sufficient (latent) alertness is reached; that is, when homeostatic plus circadian alertness (S′ + C′) reaches a critical sleep offset level (C′ = C during sleep). The latter was assumed to be just below the value that S′ approaches during sleep, that is, the asymptote of S′ (4). The curve fit to sleep termination data reached a maximum of explanatory power for a level 0.1 below the asymptote. The parameters were validated against laboratory studies and a few field studies; the predicted value was close to the empirical values (4). Regrettably, there is a dearth of studies to use for validation, since practically all sleep studies have used forced awakening designs.

It should be emphasized that the findings on sleep termination confirms the findings of the two-process model of Borbély et al., but using completely different data sets, and focusing on very different types of variables (subjective alertness). In fact, the parameters of the components in our model are numerically very close to those of Borbély et al.

Sleep onset: Knowledge of bedtime and the time of rising is a required input for most models, but frequently one encounters the need to evaluate a particular work schedule without any empirical information of when sleep will be taken. Thus, there is a need for a reasonable assumption on when bedtime is likely to occur. This could be based on empirical data previously collected from similar work schedules, but that would require empirical studies of each new schedule that one wants to model/evaluate. Thus, there is a need for a more general approach. The TPMA handles this by extrapolating from field sleep/wake diary data. The model assumptions include a cognitive component that takes into account rational behavior (i.e., in several studies the normal nighttime was 23:00), a pre-morning shift bedtime based on the observation that such a bedtime is only marginally advanced compared with that of day work, and a post-night work component that predicts bedtime 1 h after the end-of-shift. The latter may, of course, be adjusted for groups that work close to the bed with sleep-at-work work facilities.

Napping behavior also needs a cognitive component. Most day workers do not nap. Individuals on night or morning shifts, however, do. The decision to take a nap is based on knowledge of whether the next work shift will interfere with normal sleep and on the perception of when sufficient sleepiness will occur. For individuals on sea watch and similar schedules, it is assumed that sleep will be attempted whenever there is a (truly) free watch that is not used for other types of work or for meals. The particular algorithm that addresses this issue is presently being validated for inclusion in the three-process model.

Special Issues

Individual differences: Individual differences occur along many dimensions. Perhaps the most obvious one is "morning/eveningness" (36), or "diurnal type" (50), which describes the displacement in time of the alertness and sleep positioning pattern. The TPMA differentiates between pronounced and moderate morning and evening types plus defines an intermediate group. Process C is phase adjusted 1 or 2 h earlier or later, respectively. This will automatically affect predicted bedtime and time of rising since both are dependent on the level of processes S + C + U. The default value is "intermediate."

The habitual need for sleep is also a well-researched topic. This issue has been dealt with in the three-process model by adjusting the time constant of the S′ function to reach full recovery in the time indicated as habitual sleep duration. The default value is 8 h.

Chronic sleep loss: Chronic partial sleep loss causes a gradual increase in sleepiness that may or may not level off (21). The latter question has not been settled but, as described above, the present model contains a "sleep break" that turns the inverted exponential function of S′ into a linear function at alertness level 12,5. This prevents the unlimited increase of the speed of recovery which is part of the function S′.

Time zone shifts: The model includes a simple phase adjustment function for time zone shifts. The adjustment occurs as a phase shift of $1 \text{ h} \cdot \text{d}^{-1}$ eastwards or westwards. The effect of light exposure on phase (or amplitude) is not included in the model, mainly because light data are extremely difficult to obtain with any reliability in most field situations.

The risk zone: The model was developed under the assumption that the predictions will be for people exposed to an average sedentary workload. Obviously, monotony as well as stress will exert masking effects. We have only limited knowledge of such effects, but one may use, for example, the effects seen in a driving simulator as an algorithm representing such soporific effects. However, since the environment changes rapidly, it would be very difficult to enter such effects into the model on a minute-by-minute basis. Rather, one should use the concept of a risk zone, indicating the highest level of alertness that would be expected after, for example, 30 min of exposure to a sleep-inducing situation.

In the same vein, one would also attempt to construct the opposite, activation-induced effects, based on field studies of groups under stress. We have obtained such parameters for aircrew during takeoff and landing maneuvers. Essentially, the activation transforms low alertness levels to high values for the duration of exposure. If the exposure is of long duration, one may assume that the activating effects will wear off. Exactly how this occurs is not known and is obviously an area that needs research.

Countermeasures: The effects of countermeasures intrinsic to the model, such as napping, are automatically included in all predictions. Other countermeasures, such as physical activity or caffeine, are presently being implemented. It is difficult to reliably predict such parameters since the effect of different temporal patterns of caffeine intake on alertness remains unknown.

Validity and Limitations

Validity: The validity of the model was tested against laboratory and field studies of night shifts, using subjective alertness as well as EEG alpha and theta power density during waking (open eyes). Generally, the validation analyses showed that medium to high (8–14) predicted alertness seems to lack increased alpha activity, whereas there is a sharp increase, sometimes a doubling, below 7 (3,7). Clearly, high levels of predicted sleepiness in train and truck drivers co-vary with high levels of EEG alpha and theta power density, as well as with EOG slow movements. Increased alpha and theta activity may be incompatible with adequate perception of visual signals (18,38,46,52). Very similar results were observed for the EOG in the original validation studies (26). Thus, it seems that predicted values from 5 to 3 may be characterized by pronounced intrusions of sleep-related EEG and EOG changes even in the active individual. That is, there is a clear risk of behavioral errors.

Validation against performance has started recently and was investigated by trying to predict performance on a 30-min vigilance task (12 subjects, 32 signals) from a study exposing participants to 64 h of sleep deprivation (7). The results show a very close co-variation of predicted alertness with the mean ($n = 12$) number of hits on the vigilance test. One unit increase in the model corresponds to an increase of 1.9 hits (with an intercept at 3.8). Daytime performance would normally exceed 23 hits or an alertness value of 10. Extreme sleepiness in the morning after two nights of sleep loss would correspond to an alertness value of 2 and a performance value of 7 to 8 hits out of 32. Presently attempts are being made to model different types of performance

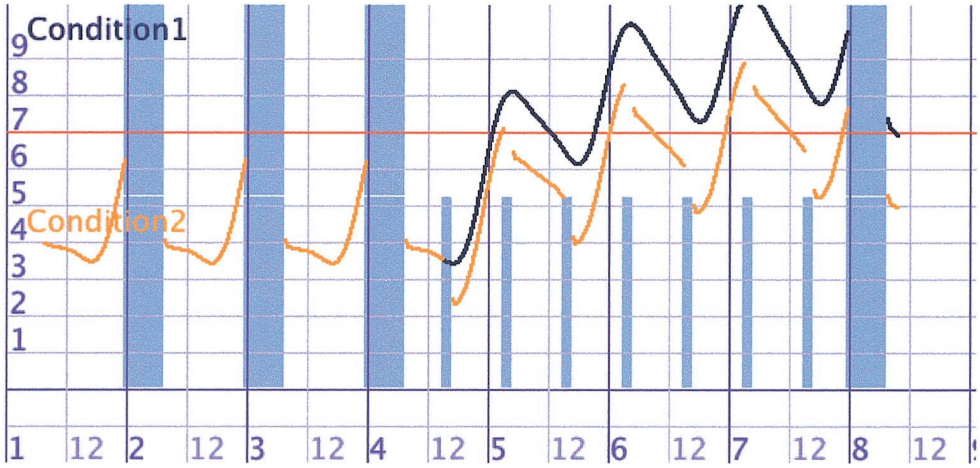

Fig. 2. Results from simulations of conditions 1 and 2 with 88 h of total sleep loss and with two 2-h naps per day. Dark bars indicate sleep.

measures, as perhaps they may exhibit slightly different functions (22,32).

Limitations: There are several limitations with regard to the TPMA and its predictions. First, it is developed on group mean data. Little experimental data on individual variations were available, except for diurnal type and sleep habits. Other limitations include unconventional decisions on when to go to bed, unforeseeable disturbances during sleep, irregular use of drugs, and many other influences. Similarly, different types of self-determined countermeasures may easily influence alertness predictions. This includes physical activity and social interaction with others. Such behavior is often seen in shift workers at night but to the best of our knowledge little systematic data are available. One must bear in mind, though, that the model is intended to be a tool for the evaluation of general effects of work schedules.

Model Interface

Input: The only required input for the software is a work schedule, that is, the start and end of each work period. The software will generate likely bedtimes and times of sleep termination automatically. Any limitations of time in bed can also be entered. If the user chooses he can also enter times of sleep. If time zone shifts are involved, the user indicates when this occurs and the number of time zones crossed. The user may also enter diurnal type (morningness/eveningness), habitual sleep length, and the presence of sleep problems.

Adjustable parameters: The adjustable parameters are as follows:

Variables of the three processes (S, C, and U);

Variables of the sleep-inertia function (W) (not used for the present modeling);

Variables of the sleep-latency function (L);

Variables of the automatic sleep-generator;

The value of the critical level (if the default value is not deemed sufficient;

Time resolution of calculation.

Output/predicted metrics: The main output is the predicted alertness curve. This may have the default form of the 1–21-point generic scale or the KSS. Performance curves include mean serial reaction time, vigilance hits, percent of time with sleep during a vigilance test, and the number of incidents or accidents in a driving simulator. In addition, the software will compute the percent of total time or work time with sleepiness levels above the critical limit. This is one of the key parameters summarizing the risk level of the particular schedule.

An equally important type of output is the estimated bedtime, the latency to sleep, and the total sleep time. In addition we also predict the number of minutes with SWS and the subjective sleep quality.

All data are stored in a tabular form for easy output. The resolution can be scaled from one minute to several hours.

SIMULATIONS

To judge the performance of the models, several standard scenarios were given (59). Below is the output of the TPMA, organized according to the scenarios. The output parameter is the Karolinska Sleepiness Scale (KSS), ranging from 1–9.

Scenario 1: 88 h of Laboratory-Based Sleep Deprivation With and Without Naps

Two conditions were simulated (**Fig. 2**). Condition 1: 88 h of continuous wakefulness starting at 07:30 after baseline sleep 3. Condition 2: 88 h of wakefulness starting at 07:30 after baseline sleep 3, except for a 2-h nap scheduled every 14:45–16:45 and 02:45–04:45 (total 7 naps).

Fig. 2 shows that sleepiness (KSS) is normal during the first 3 d and then starts to rise and crosses the critical line during the first night awake. After the second day awake sleepiness is critical, not only during the night, but also during the entire day. This is further exacerbated after the third night awake.

Under Condition 2, with 2-h naps twice a day, the sleepiness increase is strongly counteracted although the day values during the third day of continuous wakefulness stay above the critical line. One night of recovery sleep is not sufficient for full recovery, particularly after total sleep deprivation.

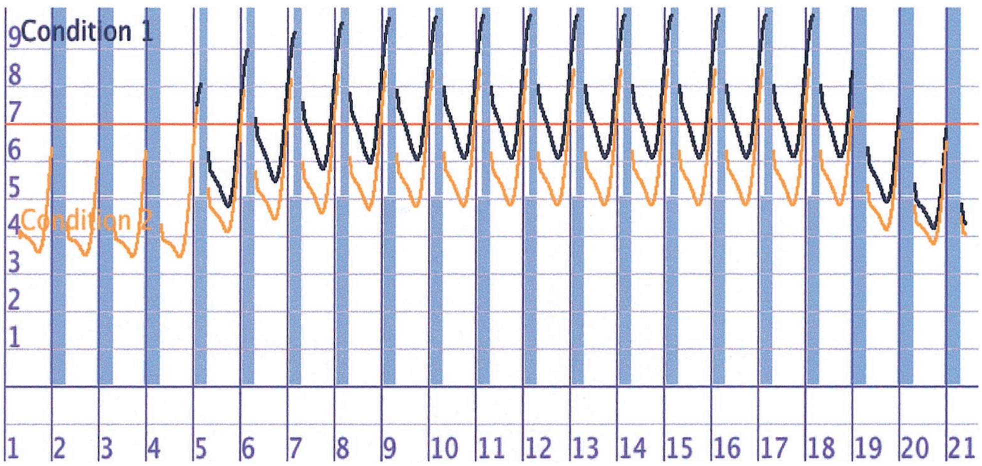

Fig. 3. Results from simulations of conditions 1 and 2, with 4 and 8 h of sleep per day, respectively. Dark bars indicate sleep.

Scenario 2: 14 d of Partial Sleep Deprivation in a Laboratory

Two conditions were simulated**.** Condition 1: 14 d of 20 h of scheduled wakefulness scheduled 07:30–03:30 followed by sleep with 4 h of time in bed scheduled 03:30–07:30. Condition 2: 14 d of 18 h of scheduled wakefulness scheduled 07:30–01:30 followed by sleep with 6 h of time in bed scheduled 01:30–07:30

Condition 1 shows an increase of sleepiness across the first 3 d of reduced sleep (**Fig. 3**). Most of the days still only indicate intermediate sleepiness even if evening values reach far above critical levels. Recovery sleep does not provide full adjustment despite three successive sleep periods.

Condition 2 with 6 h of sleep also shows a gradual increase of sleepiness across the first days but levels out on a very moderate level.

Scenario 3: Field Data Collected from Experienced Freight Locomotive Engineers on the Extra Board

Scenario 3 involves very irregular work patterns for 10 train drivers. Since this is too much to present in curves we only present the percentage of time at risk (7–8 = orange, >8 = red) for each work period (**Fig. 4**). The results indicate that most drivers will drive at risk except drivers 4 and 6. High risk is seen for drivers 2, 7, and 9. In most cases, the drives at risk occur at night with reductions of prior sleep.

Scenario 4: Theoretical Schedule for Ultra-Long-Range (ULR) Flight Operations Involving Four Crewmembers

Fig. 5a is derived from reported rest durations. The simulations suggest that pilots 1–3 will be at moderate risk during the outbound flight, mainly because prior sleep is insufficient and the flight is undertaken

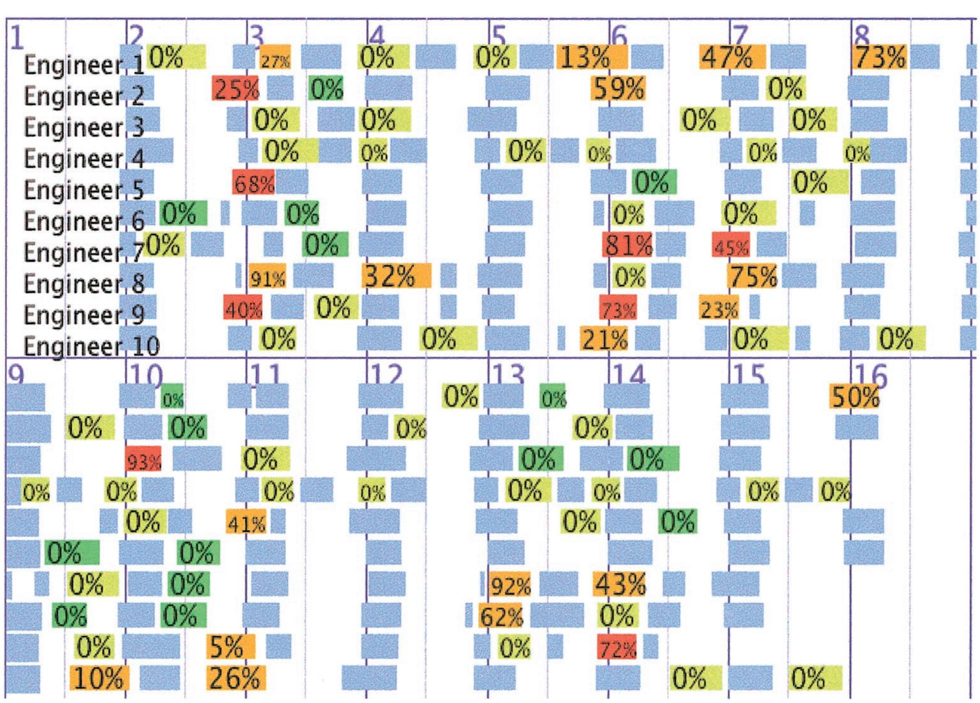

Fig. 4. Simulations of alertness for 10 locomotive engineers across 14 d of irregularly scheduled work. Yellow = work at acceptable alertness, orange = work at sleepiness/risk, red = work at very high sleepiness/risk, blue = rest/sleep.

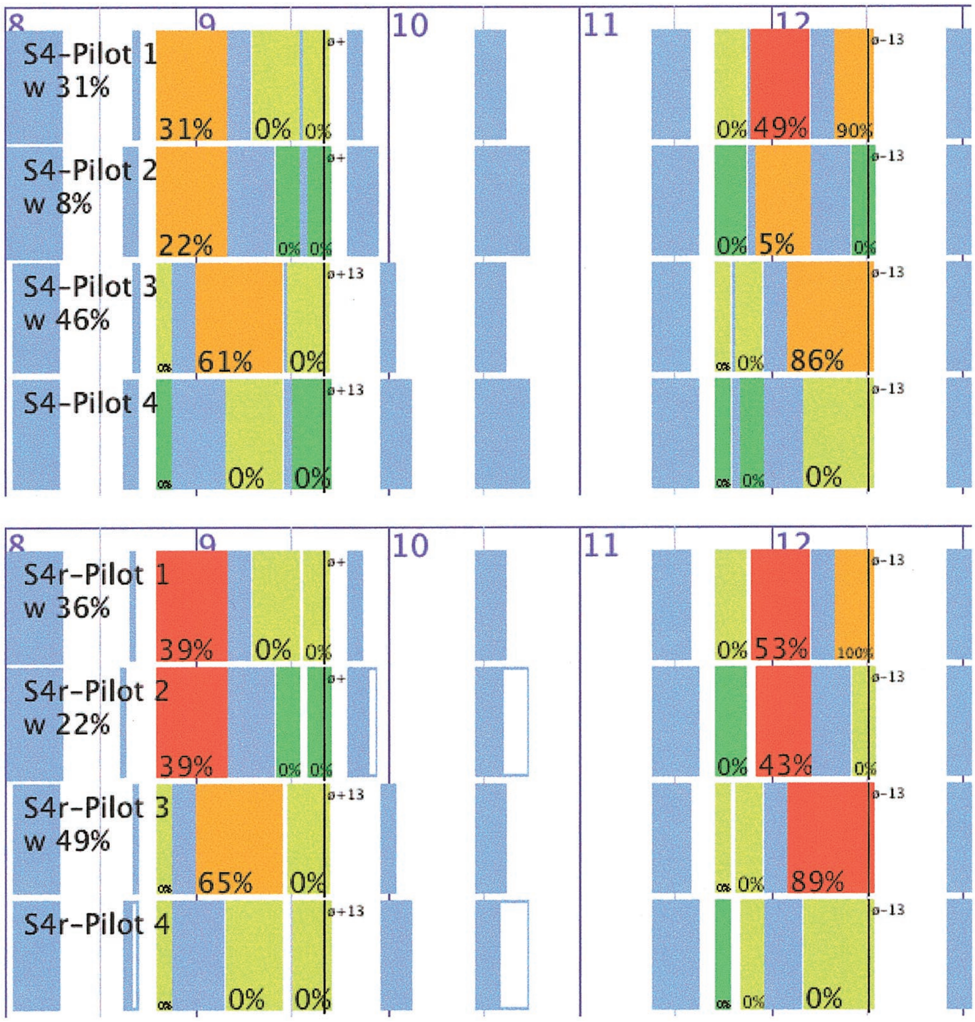

Fig. 5. a (upper) and b (lower). Simulated sleepiness in ultra-long-range pilots using reported sleep length (a) and modeled sleep length (b). Red = high risk, orange = moderate risk, yellow = low risk, green = no risk.

at night. Pilot 4 takes a proper nap before the flight and goes on duty rather late. On the return flight pilot 1 flies at risk due to short prior sleep and flying at night. Pilot 2 obtained more prior night sleep and has a lower risk. Pilot 3 works a long time at moderate risk, but avoids the highest risk level because of an in-flight nap and later duty. Pilot 4 avoids risk because he again sleeps well prior to the flight and gets an in-flight nap, together with a later, more day-oriented duty.

Fig. 5b shows increased time at risk for all pilots when the simulations produce estimates of sleep based on the model parameters. Since we predict that less sleep is obtained than what is available during the rest periods, the time at risk is increased for all pilots.

Scenario 5: 7 d of Sleep Restriction Followed by 3 d of Recovery Sleep in a Laboratory

Condition 1 involved 6 h of sleep per day, and condition 2 involved 3 h. Three 8-h periods of recovery sleep followed.

The simulation (**Fig. 6**) shows a moderate increase in sleepiness for the 6-h condition and a steep increase for the 3-h condition. Rate of increase levels out for both conditions on the fourth day. The sleep debt has yet to be completely recovered after the third recovery sleep.

CONCLUSIONS

The predictive capacity of the TPMA, given the amount of variance it explains in empirically collected data, is fairly respectable. It should be emphasized that the present model is a general approach in which sleep duration, speed of recovery during sleep, time awake, circadian phase, and performance are thoroughly integrated. A nap will affect and be affected by subsequent sleep (latency, depth, duration). Sleep loss and time awake will affect subsequent sleep (latency, depth, duration), including naps. Furthermore, the resulting sleep will be directly related to alertness and performance. This interactive approach is unique to this model. Another important approach is the prediction of sleep from work schedules, a necessary component in a model that evaluates schedules. Still, there is much work to be done. One of the most urgent tasks is to evaluate the effect of time on task. Despite the widespread notion that the duration of a duty period is important to safety, there is not a single study that has evaluated its influence while controlling for time of day, prior sleep, and prior time awake.

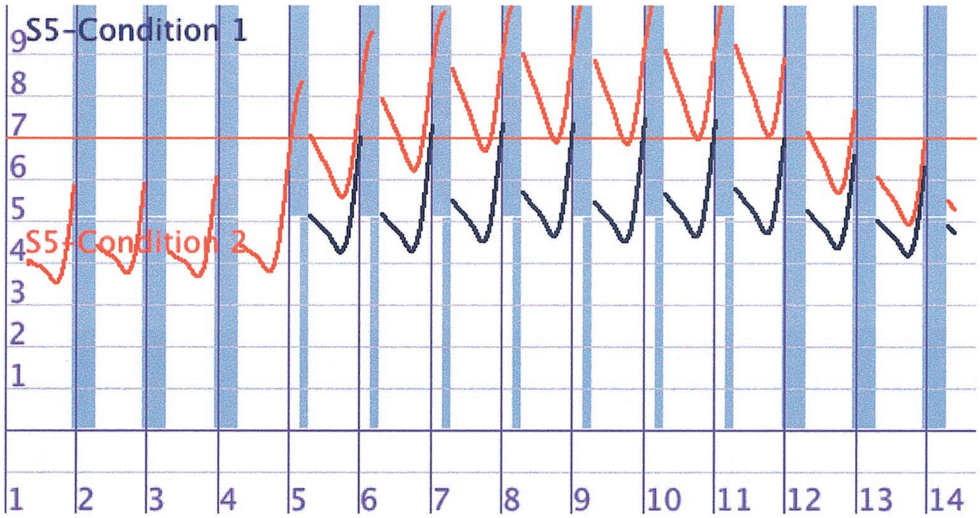

Fig. 6. Simulation of alertness for one condition of 6 h sleep per day and another with 3 h sleep.

ACKNOWLEDGMENTS

This work was supported by a grant from the Swedish Defence Research Institute, Stockholm, Sweden.

REFERENCES

1. Åkerstedt T. Work hours, sleepiness and the underlying mechanism. J Sleep Res 1995; 4(Suppl 2):15–22.
2. Åkerstedt T, Arnetz BB, Anderzén I. Physicians during and following night call duty - 41 hour ambulatory recording of sleep. Electroencephalogr Clin Neurophysiol 1990; 76:193–6.
3. Åkerstedt T, Folkard S. Validation of the S and C components of the three-process model of alertness regulation. Sleep 1995; 18:1–6.
4. Åkerstedt T, Folkard S. Predicting duration of sleep from the three-process model of alertness regulation. Occup Environ Med 1996; 53:136–41.
5. Åkerstedt T, Folkard S. Predicting sleep latency from the three-process model of alertness regulation. Psychophysiology 1996; 33:385–39.
6. Åkerstedt T, Folkard S. The three-process model of alertness and its extension to performance, sleep latency, and sleep length. Chronobiol Int 1997; 14:115–23.
7. Åkerstedt T, Folkard S, Kecklund G. The prediction of intentional and unintentional sleep onset. In: Harsh J, Ogilvie R, eds. Sleep onset. Washington, DC: American Psychological Association 1993.
8. Åkerstedt T, Gillberg M. The circadian variation of experimentally displaced sleep. Sleep 1981; 4:159–69.
9. Åkerstedt T, Gillberg M. A dose-response study of sleep loss and spontaneous sleep termination. Psychophysiology 1986; 23: 293–7.
10. Åkerstedt T, Hume KI, Minors DS, et al. Sleep on a shortening day/night schedule. Electroencephalogr Clinical Neurophysiol 1992; 82:102–11.
11. Åkerstedt T, Kecklund G, Knutsson A. Spectral analysis of sleep electroencephalography in rotating three-shift work. Scand J Work Environ Health 1991; 17:330–6.
12. Borbély A. Sleep regulation: Circadian rhythm and homeostasis. In: Ganten D, Pfaff D, eds. Sleep. Clinical and experimental aspects. Berlin: Springer Verlag; 1982:83–104.
13. Borbély A, Achermann P, Trachsel L, Tobler I. Sleep initiation and initial sleep intensity: interactions of homeostatic and circadian mechanisms. J Biol Rhythms 1989; 4:149–60.
14. Borbély AA. A two-process model of sleep regulation. Hum Neurobiol 1982; 1:195–204.
15. Carskadon MA, Dement WC. Cumulative effects of sleep restriction on daytime sleepiness. Psychophysiology 1981; 18:107–13.
16. Czeisler CA, Weitzman ED, Moore-Ede MC, et al. Human sleep: its duration and organization depend on its circadian phase. Science 1980; 210:1264–7.
17. Daan S, Beersma DGM, Borbély AA. Timing of human sleep: recovery process gated by a circadian pacemaker. Am J Physiol 1984; 246:R161–78.
18. Daniel RS. Alpha and theta EEG in vigilance. Percept Mot Skills 1967; 25:697–703.
19. Dijk DJ, Czeisler CA. Paradoxical timing of the circadian rhythm of sleep propensity serves to consolidate sleep and wakefulness in humans. Neurosci Lett 1994; 166:63–8.
20. Dijk DJ, Duffy JF, Czeisler CA. Circadian and sleep-wake dependent aspects of subjective alertness and cognitive performance. J Sleep Res 1992; 1:112–7.
21. Dinges DF, Pack F, Williams K, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. Sleep 1997; 20:267–77.
22. Folkard S. Circadian and homeostatic regulation of alertness and performance. In: Åkerstedt T, Kecklund G, eds. Work hours, sleepiness and accidents. Vol. 248. Stockholm: IPM and Karolinska Institute; 1994:42–3.
23. Folkard S. Effects on performance efficiency. In: Colquhoun P, Costa G, Folkard S, Knauth P, eds. Shiftwork. Problems and solutions. Frankfurt am Main: Peter Lang GmbH; 1996:65–87.
24. Folkard S, Åkerstedt T. Towards a model for the prediction of alertness and/or fatigue on different sleep/wake schedules. In: Oginski A, Pokorski J, Rutenfranz J, eds. Contemporary advances in shiftwork research. Krakow: Medical Academy; 1987:231–40.
25. Folkard S, Åkerstedt T. Towards, the prediction of alertness on abnormal sleep/wake schedules. In: Coblentz A, ed. Vigilance and performance in automatized systems. Dordrecht: Kluwer; 1989:287–96.
26. Folkard S, Åkerstedt T. A three process model of the regulation of alertness and sleepiness. In: Ogilvie R, Broughton R, eds. Sleep, arousal and performance: problems and promises. Boston: Birkhäuser; 1991:11–26.
27. Folkard S, Åkerstedt T, Macdonald I, et al. Beyond the three-process model of alertness: estimating phase, time on shift, and successive night effects. J Biol Rhythms 1999; 14:577–87.
28. Folkard S, Åkerstedt T, Macdonald L, et al. Refinement of the three-process model of alertness to account for trends in accident risk. In: Knauth SHP, Folkard GCS, eds. Shiftwork in the 21st Century. Vol. 17. Frankfurt am Main: Peter Lang GmbH; 2000:49–54.
29. Folkard S, Barton J. Does the "forbidden zone" for sleep onset influence morning shift sleep duration? Ergonomics 1993; 36: 85–91.
30. Folkard S, Hume KI, Minors DS, Waterhouse JM, Watson FL. Independence of the circadian rhythm in alertness from the sleep/wake cycle. Nature 1985; 313:678–9.
31. Folkard S, Monk TH. Circadian performance rhythms. In: Folkard S, Monk TH, eds. Hours of work—temporal factors in work scheduling. New York: John Wiley; 1985:37–52.
32. Folkard S, Totterdell P. Dissecting circadian performance

rhythms: implications for shiftwork. Ergonomics 1993; 36:283–8.
33. Foret J, Benoit O. Shiftwork. The level of adjustment to schedule reversal assessed by a sleep study. Waking Sleeping 1978; 2:107–12.
34. Fröberg JE, Karlsson CG, Levi L, Lidberg L. Psychobiological circadian rhythms during a 72-hour vigil. Försvarsmedicin 1975; II:192–201.
35. Gillberg M. Sleepiness and its relation to the length, content, and continuity of sleep. J Sleep Res 1995; 4(suppl 2):37–40.
36. Horne JA, Östberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol 1976; 4:77–110.
37. Horne JA, Reyner LA. Sleep related vehicle accidents. Br J Med 1995; 310:565–7.
38. Horvath M, Frantik E, Kopriva K, Meissner J. EEG theta activity increase coinciding with performance decrement in a monotonous task. Act Nerv Sup 1976; 18:207–10.
39. Kecklund G, Åkerstedt T. Sleepiness in long distance truck driving: an ambulatory EEG study of night driving. Ergonomics 1993; 36:1007–17.
40. Kecklund G, Åkerstedt T, Lowden A, von Heidenberg C. Sleep and early morning work. J Sleep Res 1994; 3(Suppl 1):124.
41. Kronauer RE, Czeisler CA, Pilato SF, Moore-Ede MC, Weitzman ED. Mathematical model of the human circadian system with two interacting oscillators. Am J Physiol 1982; 242.
42. Lavie P. Ultrashort sleep-waking schedule. III. "Gates" and "forbidden zones" for sleep. Electroencephalog Clin Neurophysiol 1986; 63:414–25.
43. Minors DS, Waterhouse JM. The role of naps in alleviating sleepiness during an irregular sleep-wake schedule. Ergonomics 1987; 30:1261–73.
44. Mitler MM, Carskadon MA, Czeisler CA, Dement WC, Dinges DF, Graeber RC. Catastrophes, sleep and public policy: concensus report. Sleep 1988; 11:100–9.
45. Monk M. Subjective ratings of sleepiness—the underlying circadian mechanisms. Sleep 1987; 10:343–53.
46. O'Hanlon JF, Beatty J. Concurrence of electroencephalographic and performance changes during a simulated radar watch and some implications for the arousal theory of vigilance. In: Mackie RR, ed. Vigilance. New York: Plenum Press, 1977; 189–202.
47. Smith L, Folkard S, Poole CJM. Increased injuries on night shift. Lancet 1994; 344:1137–9.
48. Spencer MB. The influence of irregularity of rest and activity on performance: a model based on time since sleep and time of day. Ergonomics 1987; 30:1275–6.
49. Tilley AJ, Wilkinson RT, Drud M. Night and day shifts compared in terms of the quality and quantity of sleep recorded in the home and performance measured at work: a pilot study. In: Reinberg A, Vieux N, Andlauer P, eds. Night and shift work. Biological and social aspects. Vol. 30. Oxford: Pergamon Press, 1981; 187–96.
50. Torsvall L, Åkerstedt T. A diurnal type scale. Scand J Work Environ Health 1980; 6:283–90.
51. Torsvall L, Åkerstedt T. Sleepiness on the job: continuously measured EEG changes in train drivers. Electroencephalogr Clin Neurophysiol 1987; 66:502–11.
52. Torsvall L, Åkerstedt T. Extreme sleepiness: quantification of EOG and spectral EEG parameters. Int J Neurosci 1988; 38:435–41.
53. Torsvall L, Åkerstedt T, Gillander K, Knutsson A. Sleep on the night shift: 24-hour EEG monitoring of spontaneous sleep/wake behavior. Psychophysiology 1989; 26:352–8.
54. Torsvall L, Åkerstedt T, Gillberg M. Age, sleep and irregular work hours: a field study with EEG recording, catecholamine excretion, and self-ratings. Scand J Work Environ Health 1981; 7:196–203.
55. Wever RA. Influence of physical workload on free-running circadian rhythms in man. Pflügers Arch 1979; 381:119–26.
56. Wilkinson RT, Edwards RS, Haines E. Performance following a night of reduced sleep. Psychonomic Science 1966; 5:471–2.
57. Williams HL, Lubin A, Goodnow JJ. Impaired performance with acute sleep loss. Psychol Monogr 1959; 73:1–26.
58. Reyner LA, Horne JA. Falling asleep whilst driving: are drivers aware of prior sleepiness? Int J Legal Med 1998; 111:120–33.
59. Van Dongen HPA. Comparison of mathematical model predictions to experimental data of fatigue and performance. Aviat Space Environ Med 2004; 75(3, Suppl):A15–36.