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Introducing aircraft noise characteristics into models that predict sleep structure

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ABSTRACT

Most models that predict the effect of aircraft noise on sleep relate the percent awakened to the indoor noise level of the event as measured using either L_{Amax} or *SEL(A)*. However, results from laboratory and field studies indicate that nighttime noise events do not only increase the number of awakenings but also changes an individual's sleep structure. The duration of awakenings increases with noise level and there is a reduction in slow wave and rapid eye movement sleep. These changes may cause next day effects such as decreased performance and increased sleepiness as well as long-term health problems such as hypertension. Therefore, in order to predict the effect that noise-induced sleep disturbance has on health more sophisticated models of sleep disturbance may be needed. Markov and nonlinear dynamic models have been developed to predict changes in sleep structure during the night. The nonlinear dynamic models predict non-noise disturbed sleep. A discussion of whether these nonlinear models could be be used to predict sleep disturbance due to aircraft noise is provided.

INTRODUCTION

One of the effects of aircraft noise on a community is it can cause sleep disturbance. Results from laboratory and field studies indicate that nighttime aircraft noise will increase the number and duration of awakenings. It can increase the time until sleep onset and the time until slow wave sleep occurs. Nighttime aircraft noise can also decrease the amount of rapid eye movement (REM) and slow wave sleep (stages 3 and 4), (Griefahn, Robens, Bröde and Basner, 2008).

Models have been developed to predict the effect of noise on sleep. Most of these models predict the percent of the population awakened by individual events of a specific noise level (e.g. Basner *et al.* 2004; FICAN, 1997; Finegold and Elias, 2002). However, changes in the structure of sleep may also have health and welfare consequences.

Sleep disturbance may lead to short term health effects such as decreased performance and increased sleepiness which may be related to not only the number of awakenings but changes in sleep structure. Wilkinson and Campbell (1984) and Marks and Griefahn (2005) for example both found an association between the amount of slow wave sleep and next day performance. Also sleep disturbance may lead to long-term health effects such as hypertension. Studies on non-noise disturbed sleep have found an association between elevated nighttime blood pressure ("non-dipping" of blood pressure) and the number of arousals as well as the duration of slow wave sleep (Loredo, Nelesen, Ancoli-Israel and Dimsdale, 2004).

Several models have been developed to predict an individual's sleep structure. Basner (2006) developed a Markov model to predict the effect of aircraft noise on sleep. Also several nonlinear dynamic models have been developed to describe sleep regulation. One nonlinear model developed by Massaquoi and McCarley (1992) can be used to predict time spent in REM, NREM (Non-REM) and Wake. This model is being examined to determine whether it predicts behavior similar to Basner's model as well as obtained survey data. A discussion of whether this model could be altered to predict noise-induced sleep disturbance will also follow.

SLEEP STRUCTURE MODELS

Two sleep structure models, a Markov model and a nonlinear dynamic model will be described.

Markov Model

Basner (2006) has developed a Markov model to predict the effect of aircraft noise on sleep structure. The model is based on data from a laboratory study conducted at the German Aerospace Center. This Markov model can be used to predict the sleep stages an individual is in during the night. In the model the probability of transitioning from one stage to another depends on the current sleep stage an individual is in, the time since sleep onset, and whether an aircraft event is occurring.

There are four models used to calculate the transition probabilities: there is one baseline model which is used when an aircraft event is not occurring and 3 noise models. He found that aircraft noise affects the transition probabilities for 3 epochs (30 second segments) of sleep. There is one model that applies when an aircraft event is starting, another for when it is flying over, and another when the event is just ending.

Nonlinear Dynamic Models

The model developed by Basner was the only model found that predicts the effect of noise on sleep structure: however,

there have been other models developed to predict non-noise disturbed sleep patterns. These include other Markov models (e.g. Yang and Hursch, 1973; Zung, Naylor, Gianturco and Wilson, 1965) as well as nonlinear dynamic sleep models. One advantage of the nonlinear dynamic models that have been developed is that one can argue that they are more physically-based models. One of the most comprehensive nonlinear dynamic models that was found was the model developed by Massaquoi and McCarley (1992). This model is a combination of two earlier models that were developed: the extended Two-Process Model (Achermann and Borbély, 1990) and the REM Limit Cycle Reciprocal Interaction Model (McCarley and Massaquoi, 1986).

The component based on the Two-Process model has two terms. One term is Process *S* which can be thought of as an individual's need for sleep which increases when an individual is awake and decreases during the night. The equation for Process *S* is,

$$\dot{S} = -gc\ SWA + rs\ (1 - S). \quad (1)$$

The second term is for slow wave activity (*SWA*), which is the power in an EEG signal between 0.5 and 4.5 Hz. Slow wave activity decreases during the night due to Process *S*. Also the level of *SWA* increases during deep sleep and decreases during REM and lighter sleep. These ultradian oscillations in slow wave activity are controlled by the REM component of the model. The equation for slow wave activity is,

$$\dot{SWA} = rc\ SWA\ (1 - SWA/SWA_{max}) + SWA\ n, \quad (2)$$

where

$$SWA_{max} = max\ (S\ (1 - .95\ min(X^4 + E/2, 1)), .05). \quad (3)$$

In Equation (2) n is uniformly distributed noise. The REM sleep model is a limit cycle model of the interaction that has been found between the firing of REM sleep promoting neurons (*X*) and REM sleep inhibiting neurons (*Y*),

$$\dot{X} = a(X)\ S_1(X)X - b(X)XY, \quad (4)$$

and

$$\dot{Y} = -cY + d_{circ}S_2(Y)(X + E)Y. \quad (5)$$

Both of these equations can also be written in the form:

$$\dot{X} + \gamma_1 X = 0, \quad (6)$$

$$\gamma_1 = b(X)Y - a(X)S_1(X), \quad (7)$$

and

$$\dot{Y} + \gamma_2 Y = 0, \quad (8)$$

$$\gamma_2 = c - d_{circ}S_2(Y)(X + E). \quad (9)$$

The solution for slow changes in γ_1 and γ_2 is approximately

$$X = e^{-\gamma_1 t} \quad (10)$$

and

$$Y = e^{-\gamma_2 t}. \quad (11)$$

In Equation (5) d_{circ} is a sinusoidal term with a period of 24 hours and the terms *a(X)*, *b(X)*, $S_1(X)$ and $S_2(Y)$ are saturation functions, as shown in Figure 1.

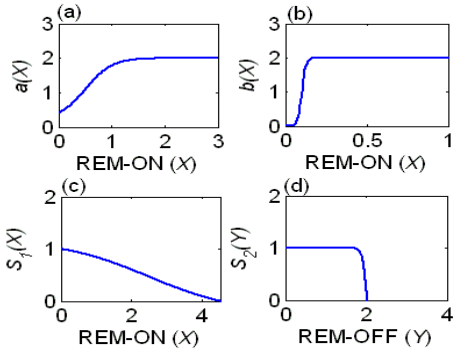

Figure 1. Saturation and coefficient functions. (a) *a(X)*, (b) *b(X)*, (c) $S_1(X)$ and (d) $S_2(Y)$.

The excitatory term *E* will lead to the prediction of awakenings when using the model. The equation for *E* is,

$$\dot{E} + kE = N. \quad (12)$$

E is a low pass filtered version of a Poisson shot noise process (*N*). The cut-off frequency of the filter is *k* and the gain is 1/*k*. Therefore the amplitude and the rate of the rise and decay of the noise are not controlled independently. The amplitude and duration of the Poisson shot noise (*N*) are uniformly distributed. The arrival time between pulses is exponentially distributed. The term *E* will decrease the level of the slow wave activity and will increase the level of *Y* (REM-OFF) activity. The excitations will, in general, lead to longer periods of NREM sleep and can lead to shortened REM periods. The baseline parameters of the model are listed in Table 1.

Table 1. Baseline model parameters (Massaquoi and McCarley, 1992) (1 unit in the model is equal to 10.7 minutes).

Model Parameters	*Baseline Values*
c	1
gc	0.05
k	10
N	Amplitude: Uniformly distributed between 1.25 and 25 Duration: Uniformly distributed between 0.25 and 0.5 Inter-arrival Time: Exponentially distributed with mean of 1.1
n	Uniformly distributed between -10 and 10
rc	3.0
rs	0.005
E_o	0.001
X_o	0.12
Y_o	0.35
S_o	2.0
SWA_o	0.1

From the outputs of the nonlinear dynamic model, the time an individual spends in three sleep states: REM, NREM and wake, can be predicted. The stage an individual is in is determined based on thresholds. When the level of *X* activity (REM-ON) is above 1.4 an individual is considered to be in REM sleep, when the level of *E* is above 0.5 and the level of *SWA* is below 0.1 an individual is considered to be awake. For all other situations an individual is considered to be in NREM sleep. An example of an output of the model with

corresponding sleep stages derived using these rules is shown in Figure 2.

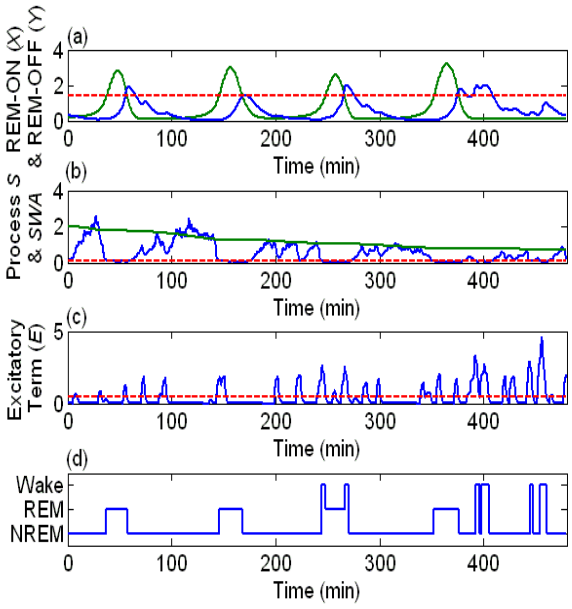

Figure 2. (a) REM-ON (green) and REM-OFF (blue) activity, (b) Process *S* (green) and slow wave activity (blue), (c) excitatory activity and (d) sleep stages. Thresholds for determining sleep stages are shown as red dashed lines.

MODEL COMPARISON

It was of interest to determine whether the output of the nonlinear dynamic model was similar to that of Basner's baseline Markov model. A comparison was made between the probabilities of being in NREM, REM and Wake through the night. To compare the two models 100 simulations using the nonlinear dynamic model were performed and the probabilities calculated from the simulated datasets. The results are shown in Figure 3.

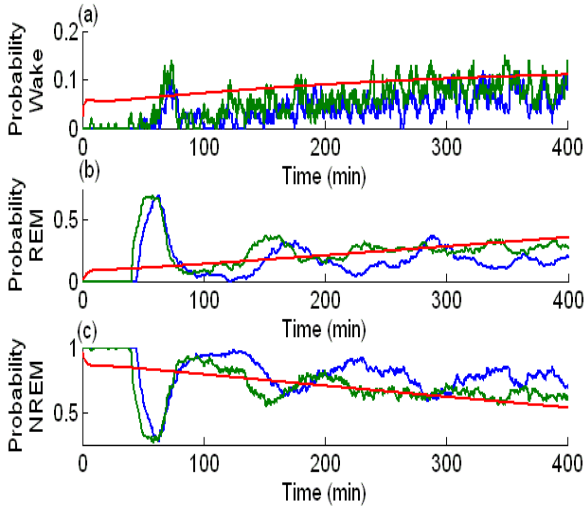

Figure 3. Probability of being in Wake, REM and NREM sleep during the night predicted by using Basner's baseline Markov model (red) and Massaquoi and McCarley's nonlinear dynamic model with the original model parameters (blue) and with a 40% increase in the value of *c* in Equation (5) and the amplitude of *N* (green).

The Massaquoi and McCarley model does predict a higher probability of being in NREM sleep and a lower probability of being in REM sleep and wake than Basner's model. To obtain a better match between models a parameter variation study was conducted to determine how altering the model parameters would change the predicted duration of REM, NREM and Wake. From completing this analysis it was found that the parameter *c* in Equation (5) had a large effect on the duration of REM sleep. An increase of *c* will lead to a faster rate of decay of *Y* (REM-OFF) activity and therefore more time will be spent in REM sleep during the night. The parameters of the Poisson noise *N* can be changed to increase the probability of awakening. Increasing the duration or amplitude or decreasing the inter-arrival time will result in an increase in awakenings. For the result shown in Figure 3 the amplitude of *N* and value of *c* was increased by 40% to obtain better agreement with Basner's model.

Another difference between Basner's Markov model and the results obtained from the nonlinear dynamic model are the oscillations in the predicted probability of being in REM and NREM sleep. Oscillations in the probabilities can be seen in Basner's original data (Basner, 2006). The values for the probability of being in REM sleep were extracted from a figure in his report and are shown in Figure 4(d). Oscillations were also apparent in the data from the laboratory study conducted by Flindell *et al.* (2000). Plotted in Figure 4 are the probabilities of being in Wake, NREM, and REM calculated for every 5 minutes using the baseline data from 9 subjects in the laboratory study. The results from Flindell *et al.*'s study follow the trends predicted by Basner's Markov model except for the probability of awakening, which is usually lower in the Flindell *et al.* study. However, this comparison was conducted using only 9 nights of data.

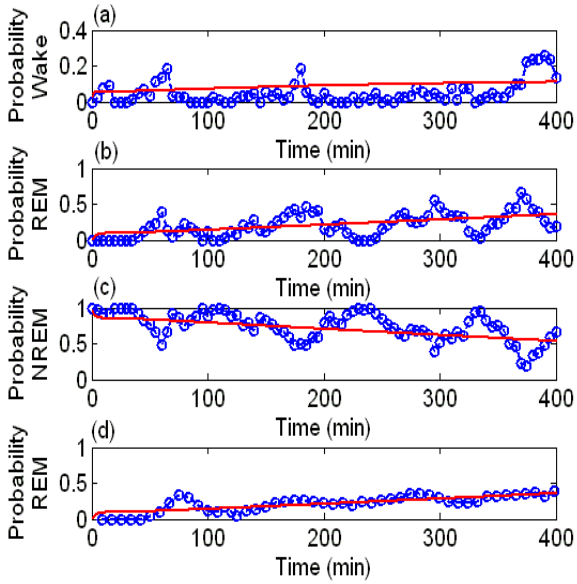

Figure 4. Probability of being in Wake, REM and NREM sleep during the night predicted using Basner's baseline Markov model (red) and (a,b,c) the probability of being in each sleep stage based on baseline data from Flindell *et al.*'s laboratory study and (d) Basner's laboratory study (extracted from Basner, 2006, Figure 9.18)

CHALLENGES WITH MODEL

While there seems to be general agreement between models and sleep data, there are several challenges in being able to use this model to predict the effect of noise on sleep. Most importantly in the Massaquoi and McCarley model, awakenings or arousals will not occur during a REM sleep period. For example, an individual could never have the following three sleep stages in consecutive epochs: REM-Wake-REM.

However, this type of behaviour can occur and has been observed in sleep data from the study conducted by Flindell *et al.* An example is shown in Figure 5 where periods of stages 1 and wake are present during the last REM cycle.

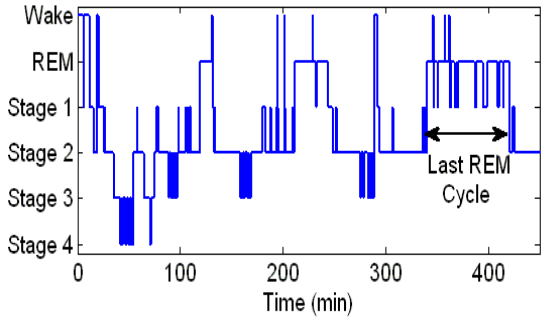

Figure 5. An example of a sleep hypnogram with brief awakenings and sleep stage changes during REM sleep.

The behaviour shown in Figure 5 cannot be predicted by Massaquoi and McCarley's model because the *E* excitatory term does not affect *X* (REM-ON) activity to any significant degree. The excitatory term will increase *Y* (REM-OFF) activity, however, during REM-sleep when the level of *X* is greater than 1.4, the value of *Y* is low and therefore it has little effect on the behavior of *X*.

From the work of Basner and Samel (2005) and Griefahn, Marks and Robens (2006) exposure to aircraft noise while asleep results in an increase in the number of awakenings as well as reduced slow wave sleep and REM sleep. Being able to control fast REM-Wake oscillations as well as fast NREM-Wake oscillations is a key component in the development of these nonlinear sleep models. While the examples in the following section are an attempt to introduce the oscillations in simulations of non-noise disturbed sleep, an understanding of how to do this will help in determining how to model the impact of noise on sleep.

POTENTIAL CHANGES TO THE MODEL

One possibility for increasing the effect of *E* on REM-sleep, is to add a term *E* into Equation (4) of the form,

$$\dot{X} = a(X)\, S_1(X)X - b(X)XY - f(X)EX, \qquad (13)$$

In Equation (13) the function *f(X)* is a saturation function which reduces the effect of *E* on the level of *X* when *X* is low, below 1.4. Therefore *E* would only have an effect on *X* when the level is above the threshold for scoring stage REM. The saturation function that was used is shown in Figure 6. If there wasn't a saturation function the level of *X* could continuously decay due to *E* and no ultradian oscillations in the obtained results would occur.

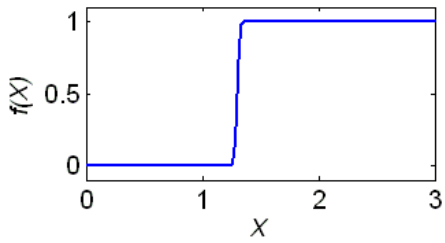

Figure 6. Saturation function *f(x)* used in Equation (13).

An example of the results typically obtained from the model when this excitation term is added is shown in Figure 7. In Figure 7(a) the oscillations at points A and B are an outcome of this change in the equations. These oscillations in *X* result in the prediction of changes in sleep stage during a REM sleep cycle which is shown in Figure 7(d).

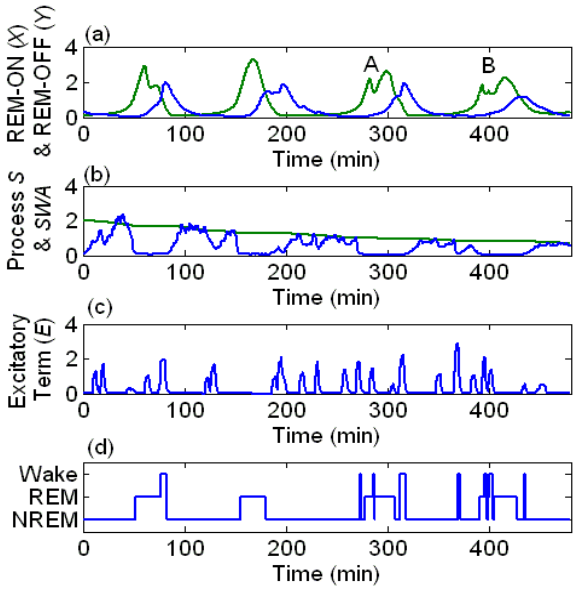

Figure 7. (a) REM-ON (green) and REM-OFF (blue) activity, (b) Process *S* (green) and slow wave activity (blue), (c) excitatory activity and (d) sleep stages when an excitation term is added to the equation for *X*.

The addition of *E* to Equation (4) however, still does not cause the changes that are desired on a short enough time scale. To have activity on a shorter time scale the equation for *E* would need to be changed. First the value of k would need to be increased as the cut-off frequency is currently set to 10 which is approximately 1.6 cycles per unit time or per 10.7 minutes. Also the higher the cut-off frequency of the low pass filter the lower the amplitude of the excitation, because the gain in Equation (12) is *1/k*. The equation would need to be changed so that the amplitude and cut-off frequency are independent. One simple change that could be made is to alter Equation (12) so it is of the form;

$$\dot{E} + kE = kAN, \qquad (14)$$

where A is an arbitrary amplitude.

The other difficulty in obtaining faster transient activity in the model is the decay and rise of terms when a noise event occurs which are controlled by γ_1 and γ_2. The increase in level of *Y* is dependent on the level of the excitation. The greater the level of *E* the faster the rise of *Y*. However, the decay of *Y* is not largely dependent on the excitation. To increase the rate of the decay the value of *c* would need to be increased. This, however, would shift when REM sleep and NREM sleep occur. An example of the results for two values of *c*, 1 and 1.4, is shown in Figure 8. To clearly demonstrate the effect of *c* on the results each excitation was set at the same height and duration and with the events evenly spaced through time. The higher the value of *c*, in general the more REM cycles in the model, for this example there are four complete REM cycles when *c* is equal to the original value of 1 and there are 5 complete cycles when *c* is increased by 40% to 1.4 .

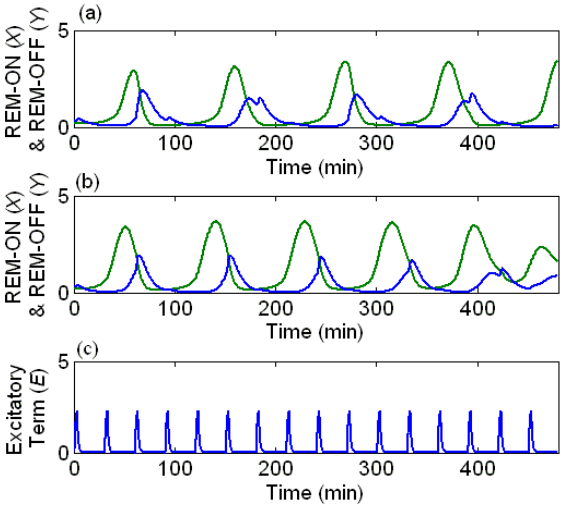

Figure 8. REM-ON (green) and REM-OFF (blue) activity, (a) c is equal to 1, (b) c increased by 40% and equal to 1.4 and (c) the excitatory term used for both simulations.

Another method for introducing noise into the model is to include a Gaussian distributed white noise term (n) in the equation for X,

$$\dot{X} = a(X)\, S_1(X)X - b(X)XY + nX, \qquad (15)$$

The noise is multiplied by X, without this multiplication the level of X can become negative. The results for four different amplitudes of noise, Gaussian noise with a standard deviation of 5, 10, 20 and 40 are shown in Figure 9. When the standard deviation of the noise is increased, larger oscillations did occur. However, adding Gaussian distributed white noise does not provide a lot of control over the frequency and depth of oscillations.

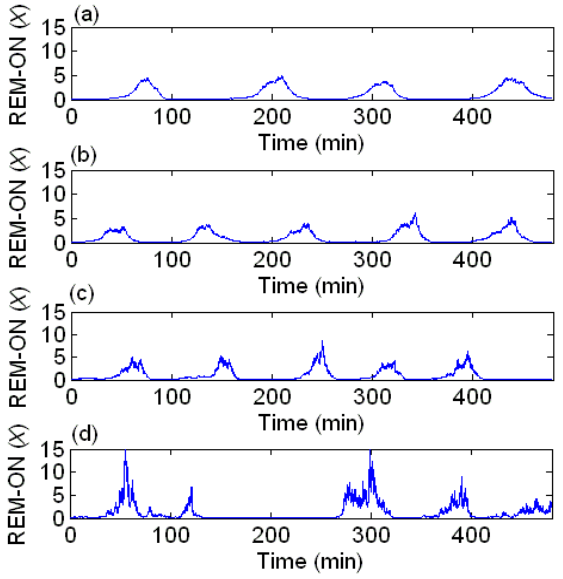

Figure 9. Results of REM-ON (X) for different amounts of noise, (a) σ = 5, (b) σ=10, (c) σ=20 and (d) σ=40.

Another possibility would be to add band-passed noise or a sinusoidal term to the model instead of Gaussian distributed white noise. This would also be added as n in Equation (15). An example of results obtained using both methods are shown in Figure 10. This does introduce oscillations on the time scale that we are interested in but this requires much further exploration.

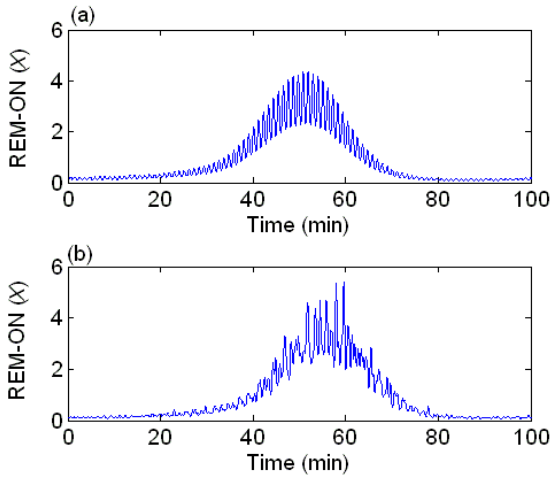

Figure 10. Results of REM-ON (X) with (a) added sinusoidal noise term with frequency of 4 oscillations per minute and amplitude of 40. (b) added noise which was band-passed between frequencies of 1 and 4 oscillations per minute with an amplitude of 50.

CONCLUDING COMMENTS

The baseline model developed by Massaquoi and McCarley predicts similar results in terms of the probability of being in NREM, REM and Wake stages as Basner's baseline model. However, the limitation of the model for predicting noise-induced sleep disturbance is that it is difficult for faster changes in the terms to occur. For the model to predict short arousals in sleep additional noise or excitations will have to be added. Results of initial investigations show that further work is needed to tailor the form those excitations should take. It would also be desirable to have physical rationale supporting the introduction of such terms which would also require additions to the model to produce predictions that have the required "noise" characteristics.

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