Timing the end of nocturnal sleep

Some people can quite accurately time the end of their night's sleep at will, without using an alarm clock[1], demonstrating that it is possible to voluntarily control a state of consciousness that is characterized by a loss of volition and attentional guidance[2]. Here we show that the expectation that sleep will come to an end at a certain time induces a marked increase in the concentration of the hormone adrenocorticotropin in the blood one hour before waking. The regulation of adrenocorticotropin release during nocturnal sleep is therefore not confined to daily rhythms; it also reflects a preparatory process in anticipation of the end of sleep.

The regulation of sleep termination has been thought to be embedded in a daily (circadian) rhythm controlling in parallel the release of pituitary and adrenal hormones[3,4]. Normally, the release of adrenocorticotropin and cortisol increases during late stages of sleeping, reaching a daily maximum at the time of spontaneous waking[5]. Adrenocorticotropin and cortisol are also released from the pituitary–adrenal system in a major adaptive response to stress, and are secreted in anticipation of stressful events[6,7]. We investigated whether the increase in the secretion of pituitary–adrenal hormones during the late stages of sleeping in part reflects anticipation of the 'stress' of the waking phase.

Fifteen healthy volunteers (mean age, 25.2 years) with regular sleep–wake rhythms were studied during three nights. We made polysomnographical recordings (electroencephalogram, electrooculogram and electromyogram) throughout the night, and took blood samples every 15 minutes to determine plasma concentrations of adrenocorticotropin and cortisol.

Lights were turned off at midnight, after subjects had been told they would be woken at either 6:00 ('short sleep', on one night) or 9:00 ('long sleep', on the other two nights). On one of the long-sleep nights they were woken at 9:00 as they expected, but on the other night they were instead woken at 6:00 ('surprise') under the pretence of a technical problem. After being woken, subjects stayed in bed for another three hours. We interviewed the volunteers at the end of the experiments, and found that all but one of the subjects (we have not used data from this subject) had expected to be woken up at the specified time. The order of the three experimental nights was balanced across subjects, with five subjects starting with short sleep, five with long sleep, and five with the surprise condition.

Plasma concentrations of adrenocorticotropin and cortisol increased during sleep, and the rate of increase did not differ among experimental conditions until about 4:30 (Fig. 1). However, when anticipating being woken up at 6:00, subjects showed a distinct increase in adrenocorticotropin levels within the last hour before waking (resulting in an average adrenocorticotropin level of 37.3 ± 3.6 pg ml^{-1}, mean ± s.e.m.), compared with the same subjects under 'surprise' conditions (25.5 ± 2.7 pg ml^{-1}, $P < 0.005$) and 'long sleep' conditions (26.5 ± 4.1 pg ml^{-1}, $P < 0.05$).

The increase in adrenocorticotropin before being woken in the 'short sleep' condition was not accompanied by a significant increase in cortisol concentration, which remained unchanged before 6:00 by the anticipation of waking. This finding cannot be explained only by cortisol release being unresponsive to adrenocorticotropin stimulation: an adrenocorticotropin-independent mechanism of adrenal regulation may also be involved[8].

In the morning, arousal from sleep induced a temporary increase in the concentrations of adrenocorticotropin and cortisol, which peaked about 30 minutes later, probably as an adaptive response to the stress of waking[5]. Compared with the level just before being woken, the increase in adrenocorticotropin levels after waking in the 'surprise' condition (an increase of 22.1 ± 3.9 pg ml^{-1}) was larger than the increase after both 'short sleep' (10.6 ± 3.2 pg ml^{-1}, $P < 0.01$) and 'long sleep' (12.2 ± 3.3 pg ml^{-1}, $P < 0.05$). A greater increase also occurred in cortisol concentrations after 'surprise' awakening (73.4 ± 6.6 ng ml^{-1}) than after awakening from 'short sleep' (40.4 ± 9.9 ng ml^{-1}, $P < 0.025$) or 'long sleep' (35.4 ± 9.8 ng ml^{-1}, $P < 0.005$).

Our polysomnographical recordings did not show any differences in the nature of the sleep between 0:00 and 6:00 that depended on the anticipation of sleep termination, in agreement with previous findings[9]. In particular, 'short sleep' was similar to sleep under 'surprise' conditions. The proportions of time spent in the different sleep stages during the 'short sleep' and 'surprise' conditions were, respectively: sleep stage 1, $8.56 \pm 0.27\%$ and $7.10 \pm 1.72\%$; sleep stage 2, $35.40 \pm 1.38\%$ and $35.72 \pm 2.20\%$; slow-wave sleep, $37.05 \pm 3.68\%$ and $37.69 \pm 3.59\%$; rapid eye movement (REM) sleep, $17.36 \pm 1.14\%$ and $18.88 \pm 1.38\%$; and wake time, $1.62 \pm 0.82\%$ and $0.59 \pm 0.04\%$. Moreover, the temporal distribution of sleep stages (including transient arousals) did not differ between the 'short sleep' and 'surprise' conditions. Final waking occurred from REM sleep in five subjects of each condition.

The increase in adrenocorticotropin release before the expected time of waking indicates that anticipation, which is generally considered to be a unique characteristic of the regulation of conscious action, pervades sleep. The anticipatory adrenocorticotropin increase may also facilitate spontaneous waking[10]. The regulation of adrenocorticotropin release points to a mechanism that quickly adjusts endocrine activity to sharp changes in the duration of sleep.

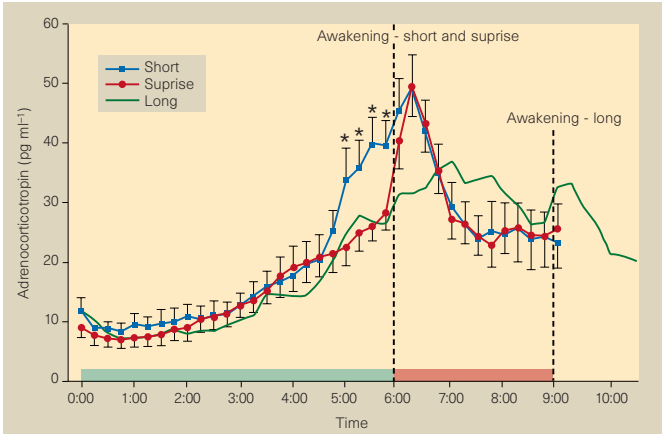

Figure 1 Mean plasma concentrations of adrenocorticotropin for subjects under 'short sleep', 'surprise' and 'long sleep' conditions (see text). Standard error of the mean is shown for 'short sleep' and 'surprise' conditions. Broken vertical lines indicate waking. Subjects stayed in bed for three hours after waking. Asterisks indicate statistically significant differences between 'short sleep' and 'surprise' conditions. The increase in adrenocorticotropin before the end of 'short sleep' was also significant compared with levels during 'long sleep' in the hour before 6:00. The low adrenocorticotropin before 'surprise' waking, compared with levels before waking from 'short sleep', was fully compensated by increased hormone release after waking, so adrenocorticotropin levels in these conditions were identical 30 minutes after waking.

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Air traffic may increase cirrus cloudiness

High-level cirrus clouds can evolve[1,2] from the condensation trails of aircraft, which form as the mixture of warm, humid exhaust gases and colder, drier air exceeds water saturation[3]. In addition, the particles in exhaust plumes from aircraft may allow ice nucleation at lower supersaturations than those required under natural conditions[4]. This mechanism is sensitive to environmental conditions, but may occur downstream of the exhaust aerosol source regions. Here I show that cirrus clouds increased in occurrence and coverage in the main air-traffic flight corridors between 1982 and 1991.

I used synoptic cloud reports from land and ship stations for 1982 to 1991 (ref. 5) to establish the relationship between variations in the occurrence of cirrus cloud and the spatial distribution of aviation fuel consumption[6,7]. During this period there was a large increase in total fuel consumption by air traffic, with an average annual growth rate of 3.2% (ref. 8). I computed the cirrus occurrence frequency at the 3°×3° resolution from cloud reports (see Supplementary Information). The change in cirrus occurrence frequency is then computed as the difference in frequency between 1987–1991 and 1982–1986, whereas its trend is estimated as the absolute percentage increase along a best-fit line over the 10 years of data.

The average change in cirrus occurrence over land and ocean is plotted against fuel consumption in Fig. 1a, b. Global annual cirrus occurrence increases on average by 1.1% and 3.5% for land and ocean, respectively, with regional average increases of 2.9% to 4.6% over the principal flight corridors (defined as the twenty 3°×3° grid-boxes with most air traffic). The largest changes correspond to positive trends in cirrus occurrence of 8.5% and 7.0% (Table 1). Over North America, the trend in cirrus occurrence varies between 2.4% and 9.9% per decade, depending on the season. It is largest in December–May, when there is the highest frequency of persistent condensation trails[9]. Between 39° and 42° N, close to the Great Lakes, the trend in annual mean cirrus occurrence is 13.3%, more than twice that for the rest of North America. Over ocean, the trend is also very large in the North Atlantic flight corridor, hence the positive slope in Fig. 1c. Changes in cirrus occurrence over the North Atlantic flight corridor are significantly larger than over the rest of the North Atlantic (Student's *t*-test, $P = 0.03$). Table 1 indicates that, with only one exception, the trend in cirrus increase is larger averaged over the 20 grid-boxes in each region with most air traffic for Eurasia, North America and the North Atlantic Ocean in all seasons.

In the North Pacific Ocean there is the largest increase in cirrus occurrence between 40° and 60° N. There is little air traffic here, but the region lies downwind of the high-traffic area along the east coast of Asia. It is plausible that cirrus occurrence in the North Pacific is favoured by the presence of soot aerosols emitted by aircraft and transported eastwards to areas where conditions for cirrus formation are often met; a large amount of natural cirrus cloud is found over this region. Along the east coast of Asia there has been a decrease in cirrus occurrence, which might be explained by a decrease in the mean flight level (computed from refs 6,7), which lowers the potential for condensation-trail formation.

A search for other possible causes of increased cirrus, such as the effects of the El Chichón and Mount Pinatubo volcanic aerosols[10], or long-term changes in relative

Table 1 **Trends in cirrus cloud occurrence, cirrus amount when present and cirrus amount**

	Occurrence					AWP	Amount
	DJF	MAM	JJA	SON	Annual	Annual	Annual
Land	2.5/6.4	1.8/9.7	2.4/6.8	0.6/2.2	1.7/8.5	−1.9/−1.9	0.0/2.9
Eurasia	1.2/2.6	−0.8/1.1	1.7/4.4	−0.8/−1.4	0.6/1.6		
N. America	7.0/7.8	9.9/16.5	3.2/9.5	2.4/7.3	5.6/13.3		
Ocean	6.0/8.3	5.5/6.3	6.4/8.7	7.7/6.0	6.2/7.1	0.4/−4.2	2.3/1.0
N. Atlantic	4.5/8.4	5.1/6.3	3.3/8.7	3.9/6.0	4.1/7.1		
N. Pacific	8.7/5.8	8.4/1.9	5.8/2.5	10.4/4.8	8.4/4.1		

In each column, the first figure reports the trend over the whole region, and the second figure shows the trend over the 20 grid-boxes with most air traffic in 1992. For North America, the second figure is for the 10 grid-boxes with most air traffic. Months (December to November) are denoted by their initial letter; AWP, amount when present. Trends are given as per cent per decade.

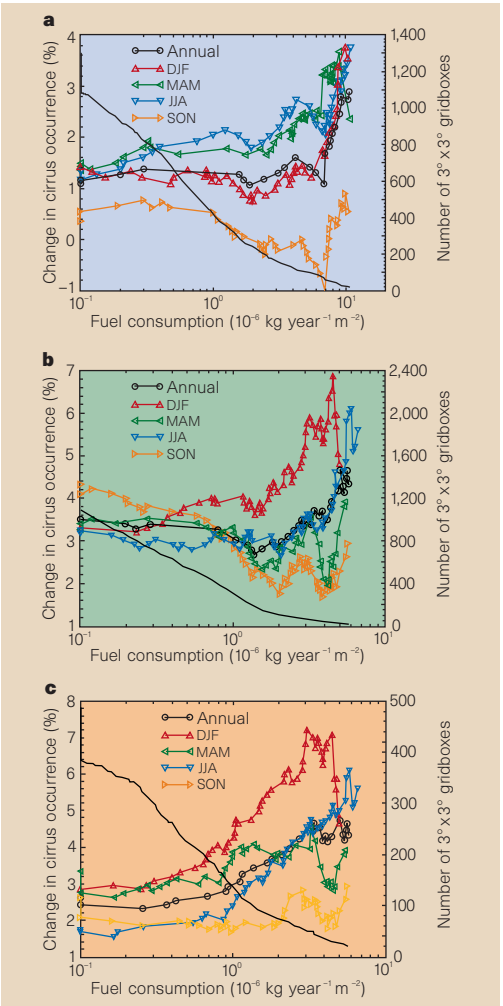

Figure 1 Difference in cirrus occurrence frequency between 1987–1991 and 1982–1986. **a,** Land; **b,** ocean; **c,** North Atlantic Ocean. Results are averaged over grid-boxes with the 1992 fuel consumption at altitudes more than 8 km greater than the values displayed on the *x*-axis, and show annual means (circles) and seasonal means (triangles). The frequency of cirrus occurrence is defined as the ratio of the number of high-cloud observations to the number of potential high-level cloud observations (situations where low- and middle-level clouds do not completely obscure the higher level of the atmosphere). Only observations meeting the illuminance criterion defined by Hahn *et al.*[15] are included, introducing a bias towards daytime. The bold line represents the number of 3°×3° grid-boxes from which the annual mean is calculated. The curves are truncated when the number of grid-boxes used to calculate the mean falls below 20.