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## Ambient temperature and human sleep

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Ambient temperature is a common factor of the environment, but some of its effects on human biology are still unknown. In laboratory situation, ambient temperature levels are often mentionned but rarely controlled. Moreover accurate variations within a narrow range are difficult to obtain and specially to maintain. Mostly, if mentionned, environmental condition is defined by the air temperature inside the experimental room. However, all the thermal characteristics of the environment should be taken into account and importance of wall temperatures, air humidity and air velocity on a defined climate should also be emphasized.

The effects of ambient temperature on human sleep have been increasingly studied in the last decade, the main reason being that in animals thermoregulatory processes have been found present in SWS and absent in REM sleep<sup>9,35</sup>.

In laboratory situations, the general interest was first focused on low and mainly high ambient temperatures 14,18,39 and very little attention has been paid to the influence of ambient temperatures fluctuating around thermoneutrality. It appears, however, that even slight changes of the ambient temperature within the thermoneutrality zone can induce modifications of sleep structure.

Apart from studies performed under laboratory conditions, real life studies in extreme ambient conditions have also been carried out<sup>2,23</sup>. They have revealed the important adaptative ability of man to live in harsh environments where tough ambient temperature is only one aspect of these difficult living conditions.

Effect of the ambient temperature on the EEG stages of sleep

Most sleep laboratory studies on the effect of temperature on sleep have included all-night electroencephalogram monitoring which provides objective and traditional measures of sleep quality.

Within a certain range of ambient temperature that should be referred to as 'thermal comfort zone', the quantitative measures of sleep such as sleep stage latencies, time spent in each sleep stage, number and duration of nocturnal awakenings, and occurrence of phasic events such as activation phases, are only slightly modified8,29. Affecting this thermal comfort zone are clothing and bed covering of the sleeping subjects. In a study where clothing and covering consisted of pyjamas, two cotton sheets and one wool blanket, Candas et al.5 found that the microclimate temperature established inside the bed varied from 28.6°C to 30.9°C, while the ambient temperature (air and wall temperatures being equal) varied from 16°C to 25°C. In this experiment the microclimate temperature measured inside the bed was found to be constant at 29.6°C for both ambient temperatures of 19°C and 22°C. These results suggest that thermoneutrality inside the bed lies around 30°C, a value in agreement with results of McPherson<sup>27</sup>. The preferred room temperature during sleep was found to be around 19°C and subjects' reports showed that subjective discomfort increased as room temperature deviated from this condition<sup>5</sup>.

As ambient temperature increases or decreases from the above mentioned thermoneutral range, the structure of sleep is modified. At both high and low temperatures there is a marked increase in the number and duration of the periods of wakefulness<sup>17,18,29</sup>. Kendel et al. 18 pointed out that unclothed and uncovered subjects awoke from cold at 26 °C and below. This ambient temperature is very close to the value found in another study where 26.1 °C recorded inside the bed corresponded to an ambient temperature of 13 °C and where an increase in nocturnal awakenings was clearly observed<sup>29</sup>.

Fever has also been found to be associated with a greater number of awakenings, increased total waking time, and reduced amounts of REM sleep and slow wave sleep<sup>15</sup>. The elevated ambient temperature induces very similar effects on the structure of these two stages of sleep<sup>16,34</sup> while stage 2 was found to be remarkably constant<sup>18</sup>. Haskell et al.<sup>11</sup> noted that although REM sleep latency was increased at high and low temperature, REM sleep was depressed to a greater extent by lower than by higher temperatures whereas the reverse was observed for SWS. The duration of the REM phases is shortened in artificially-induced fever<sup>15</sup> as well as at high ambient temperature<sup>41</sup>.

In a recent study we found that the average REM cycle length significantly decreased when the ambient temperature was elevated31. Thus, although the other REM sleep characteristics such as total duration of REM sleep, average REM period, and REM sleep latency did not significantly differ with one ambient temperature condition and another, the average REM cycle length varied from 108.6 min at an ambient temperature of 13°C to 85.2 min in the 25°C condition. Naitoh et al.<sup>32</sup> noted a shortening of the REM cycle when covered patients were sleeping at 26 °C room temperature compared to patients sleeping at 18 °C. The modification of the interval between successive REM phases without modification of the average duration of the REM periods, would then suggest that the mechanisms underlying REM rhythmicity are different from those responsible for REM maintenance. Several studies have shown the relationship between REM production and autonomic variables such as body temperature.

Effect of the ambient temperature on the autonomic and motor variables

The effect of ambient temperature during sleep can also be seen on the autonomic variables and especially body temperature. Thermosensitive areas in the hypothalamus have long been known to participate in the regulation of body temperature. Hammel<sup>10</sup> has presented a theoretical model of thermoregulation which postulates the existence of a hypothalamic controller with an adjustable set point temperature. In man during normal daily living, the internal temperature follows a circadian rhythm closely related to the sleep-wakefulness cycle<sup>20</sup>. Under normal conditions, the rectal temperature tends to decrease during the night until it reaches a minimum in the early morning hours. In subjects living isolated from external time cues, Zulley et al.42,43 found that REM sleep is negatively correlated with body temperature and also that body temperature influences sleep duration. The higher the body temperature at sleep onset, the longer the duration of sleep. Similarly, Czeisler et al.7 showed that the timing of REM sleep is controlled, at least in part, by an endogenous circadian oscillator coupled to another oscillator which is responsible for the body temperature cycle. They also presented the hypothesis that the relationship between REM sleep propensity and the body temperature cycle represents either the close coupling of two potentially independent circadian oscillators or a direct effect of body temperature on REM sleep timing. These authors also hypothesized that the REM sleep propensity rhythm was more closely tied to the body temperature rhythm than to the timing of the sleep-wakefulness cycle itself.

It has been shown (fig. 1) that the rectal temperature during sleep depends on the ambient temperature not only for the level but also for the shape of the curve<sup>1,5</sup>. Under cold conditions the body temperature decreases early and is sustained longer at low levels than under warm conditions<sup>40</sup>. In cold conditions, the major factors in maintaining body heat balance are heat production<sup>21,22</sup> shivering and vasomotor activity<sup>38</sup>.

In a recent study carried out in 5 levels of ambient temperature, Libert et al.<sup>25</sup> compared body heat storage calculated from rectal and mean skin temperature variations using the weighting factors determined by Burton<sup>4</sup>. It was found that during slow wave episodes body heat storage decreased when air temperature increased. During REM sleep however a positive linear relationship was described between body heat storage and air temperature.

In a study done in the Arctic during the winter, at ambient temperatures ranging from  $-29\,^{\circ}\text{C}$  to  $-35\,^{\circ}\text{C}$  (subjects were sleeping in sleeping bags), Buguet et al.<sup>3</sup> found that mean skin temperature variations in REM sleep were dependent on core temperature. In the neutral environment (baseline nights) where rectal temperature was always above  $36\,^{\circ}\text{C}$ , mean skin temperature increased in REM sleep. In cold conditions, mean skin temperature measured in REM sleep still increased when rectal temperature was high but decreased when rectal temperature was lower than  $36\,^{\circ}\text{C}$ .

At elevated ambient temperatures, the nocturnal drop in body temperature does not occur and sometimes greater variation of the temperature level has been observed <sup>14,33</sup>. However Haskell et al. <sup>12</sup> did not observe the marked increase in rectal and mean skin temperatures during REM sleep at air temperatures of 34°C and 37°C as mentioned by Hénane et al. <sup>14</sup>.

Rodbard<sup>37</sup> found that in birds and mammals a rise in body temperature was associated with a rise in blood pressure and heart rate, whereas a lowering of body temperature elicited the opposite effects<sup>37</sup>. The increase in heart rate in hot conditions compared to neutral conditions has also been described during sleep in

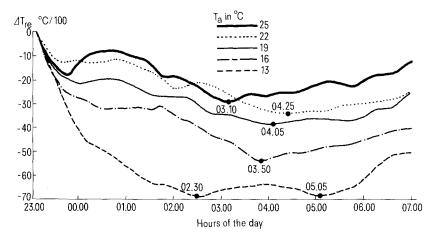


Figure 1. Mean rectal temperature variation ( $\triangle$  T<sub>re</sub>) calculated for 5 subjects sleeping under each of 5 air temperature (T<sub>a</sub>) conditions. Time of the minimum levels of the rectal temperature are indicated on the curves. (Reprinted from Candas et al.<sup>5</sup>. By permission of P.O. Fanger, ed.)

man<sup>16,30,34</sup>. This increase in heart rate level is often coupled with a greater variation which is due to an increase in the number of body movements. Such increase in body movements or in postural changes observed at elevated ambient temperature has already been described in several studies<sup>18,28,29,34</sup>. Moreover, increases in body motility are associated with a more fragmented sleep since body movements are frequently accompanied by awakenings.

Effects of ambient temperature on the endocrine system

Few studies have mentioned the effects of ambient temperature during sleep on the endocrine system. In this type of experimental approach, the exposure to low or high ambient temperature is used as a stress factor. In a study done in 1973, Kendel et al. 18 found that the morning concentration of plasma corticoids was lower after a night at 37°C than after a night at 27°C or 31°C. Following the warmer nights, urinary excretion of total corticoids and 17 corticosteroids was also lowered. In the morning urine samples, adrenaline excretion was greater at the higher room temperature, whereas the noradrenaline excretion was higher at the lower temperature. In another study, Beck et al. showed that low ambient temperature was associated with significantly increased plasma cortisol levels throughout the night. In addition, low ambient temperature activated the release of thyroid-stimulating-hormone (TSH) in the 2nd part of the night. The modifications were observed in the absence of any distinguishable changes in EEG sleep parameters.

## Thermoregulation during sleep

Being a homeotherm, man must regulate his temperature during sleep. This regulation depends on thermal sensitivity and thermoregulatory responses. As it appears that thermoregulatory responses are sleep stage-dependent, it is important to determine whether or not thermal sensitivity is sleep stage dependent.

In a recent study, Candas et al.6 recorded 5 male subjects sleeping uncovered and semi-nude on a hammock in a climatic chamber at an operative temperature of 32°C. In each experimental night, first during SWS and then during REM sleep, operative temperature of the chamber was changed either from 32°C to 39°C or from 32°C to 28.5°C. When the variation of the temperature did not provoke a sleep stage change, the operative temperature was maintained for 10 min before being slowly and linearly reset to 32°C, with an inverted slope of temperature variation. With such an experimental design, the whole body surface was stimulated by the ambient temperature variations and therefore, the mean skin temperature was considered representative of the thermal information which was operating on the thermosensitive system. Results showed that thermal sensitivity was maintained in REM sleep and that REM sleep was even more disturbed by thermal transients than SWS. Moreover, and similarly to what has been already described during wakefulness<sup>19</sup>, during sleep sensitivity to cooling appeared to be greater than sensitivity to warming.

Libert et al. 26 investigated the thermoregulatory responses induced by ambient temperature variations. To do so these authors used a sweat collecting capsule using a dew-point hygrometer technique<sup>24</sup> that was stuck on the subjects' chest. This device allowed them to record immediate variations of the local sweating rate in response to ambient temperature changes. In figure 2, mean local sweating rates and body temperature changes are plotted against time from the start of thermal transient applied in well-defined SWS and REM sleep. Results showed that during SWS as well as during REM sleep, local sweat rate varies as a function of esophageal temperature variations. However, the major difference in the sweating response between SWS and REM sleep was the time lag of sweat onset which was shorter in the former than in the latter stage. This study indicates that the thermoregulatory response to heat is not abolished during sleep in man. As shown in figure 2, in comparison with SWS, during REM sleep the sweating response is obtained at a cost of high mean skin and esophageal temperatures. This indicates a lower sensitivity of the thermoregulatory system in REM sleep than in SWS.

This confirms the results of Haskell et. al.13 showing that the increase in oxygen consumption in REM sleep in a low temperature condition suggests that REM sleep is certainly not as thermally disruptive in man as in other mammals. On the other hand, this finding is not in agreement with those of Shapiro et al.39 and Hénane et al.<sup>14</sup>. However, it must be pointed out that the results of these authors were mainly based on whole-body sweat rate when ambient temperature was maintained at a constant level throughout the night. In the study of Libert et al.26 the ambient temperature was intermittently changed in clearly-defined sleep stages and the sweating rate was measured on only a part of the body. Such contrasts in the methodology used in these studies could explain the differences observed by the different authors.

Although different from those seen in SWS, thermoregulatory responses to heat persist in REM sleep. This raises the question of sensitivity difference of the thermoregulatory system in these two stages of sleep. One possible explanation could be based on the assumption that thermoregulation during sleep depends on variations of the hypothalamic command for thermoregulation as demonstrated in animals by Parmeggiani et al.<sup>36</sup> and Glotzbach et al.<sup>9</sup>. Another explanation could be based on a change in the excitability and/or the activity of extrahypothalamic structures such as the spinal cord and the sweat gland itself. Indeed the depression of sweating in REM sleep might be quite consistent with a modification of the sympathetic command.

In conclusion, we can consider that ambient temperature might play an important role in the organization of human sleep. There can be an antagonistic interaction between sleep and thermoregulatory processes. In REM sleep the thermoregulatory system is less active and the sweating response is less precisely regulated than during SWS. Therefore the maintenance of homeothermy becomes more difficult. This may lead to a conflict between the necessity of temperature regulation and, at the same time, of maintainance of a sleep state less effi-

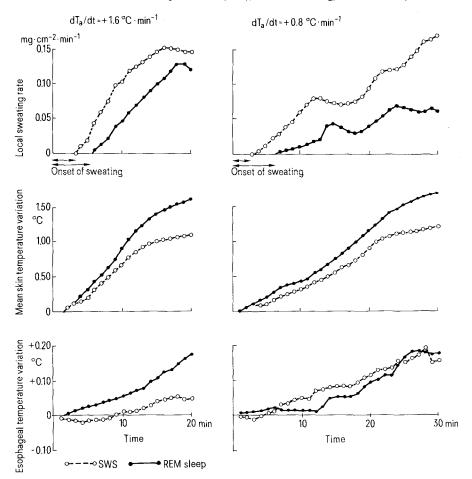


Figure 2. Mean local sweating rates and body temperatures for SWS (open circles) and REM sleep (full circles) in response to thermal transients  $(dT_a/dt = +1.6\,^{\circ}\text{C}\cdot\text{min}^{-1}$  for 10 min and  $dT_a/dt = +0.8\,^{\circ}\text{C}\cdot\text{min}^{-1}$  for 20 min).

cient for thermoregulation than other sleep stages. Slight changes in temperature, within the thermal comfort zone, although not accompanied by dramatic EEG sleep modifications, may affect body heat exchanges and therefore the body temperature. Data suggesting a close coupling between body temperature cycle and the programming of certain stages of sleep, such as REM sleep, should incite sleep researchers to pay more atten-

tion to the level of ambient temperature during sleep. Finally, the study of the mechanisms of thermoregulation would benefit from recordings made during sleep. The unusual functioning of the thermoregulatory system to heat or cold in REM sleep when compared to that observed in waking or even in non-REM sleep, might be used to study more deeply the thermoregulatory mechanisms in humans.

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## Sleep disturbance due to transportation noise: ear plugs vs oral drugs\*

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## Introduction

From 1970 onwards, an increasing proportion of the research on noise has been oriented toward assessing its influence on sleep. Notwithstanding some interesting results which will be summarized in this paper, a major problem arises from the fact that such research is aimed at studying the impact of a physical phenomenon—noise—on a physiological phenomenon—sleep—while the exact definition and nature of both phenomena are still controversial. For the vast majority of human beings sleep quality is reflected only by subjective feelings upon awakening and, in so far as they later may lead to a complaint, they must be considered as being impor-

tant. Despite this and with the exception of a few caricatural situations, there is though little correlation between these subjective reports and 'measurements'. Instead of reviewing all the data on the relationships between noise(s) and sleep perturbations as presented in several symposia and published in reviews<sup>15,38,61</sup>, we will attempt here to approach this problem from a more general point of view, and pose 4 questions:

The first question we shall address will be epidemiological: are there any differences in the patterns of drug consumption between populations living in areas with different noise levels?

In view of the fact that some adaptation may occur in the course of long periods will then consider the effects