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Skin temperature and sleep-onset latency: Changes with age and insomnia

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Abstract

Throughout the 24-hour day, the occurrence of sleep and wakefulness is closely related to changes in body temperatures. Changes in skin temperature may causally affect the ability to initiate and maintain sleep. First, we briefly summarize a previously proposed neurobiological mechanism that couples skin temperature to sleep propensity. Next we review previous findings on the relation between skin temperature and sleep-onset latency, indicating that sleep propensity can be enhanced by warming the skin to the level that normally occurs prior to – and during – sleep. Finally, we present new data indicating age- and insomnia-related changes in the sleep-onset latency response to foot warming, and evaluate whether different methods of foot warming could provide an applicable strategy to address sleep complaints. Foot temperature manipulations included footbaths before sleep onset (*1*), and heatable bed socks applied either before (*2*) or after lights-off (*3*). In adults, sleep-onset was accelerated by warm and neutral bed socks after lights-off and correlated to the increase in foot temperature. This increase was attenuated in elderly subjects. In elderly subjects without sleep difficulties, sleep onset could be accelerated with neutral bed socks after lights-off and a warm footbath prior to lights-off. In elderly insomniacs, none of the treatments accelerated sleep onset. We illustrate that elderly subjects show an attenuated increase in foot temperature after lights-off and lose the relationship between pre-sleep heat-loss activation and sleep latency. The sensitivity of sleep propensity to foot warming changes with age and is attenuated in age-related insomnia.

Keywords: Sleep electroencephalogram; Core body temperature; Skin temperature; Thermoregulation; Sleep-onset latency; Ageing; Insomnia; Foot warming

1. Introduction

Both sleep initiation and termination are temporally related to the circadian rhythm of core body temperature (CBT) and skin temperature. The habitual sleep period coincides with the diurnal phase of lowered CBT and the rise of CBT heralds the end of the sleep period. Habitual sleep onset coincides with the maximal rate of decline in CBT [1,2]. This decline is to a large extent caused by increased skin blood flow, and consequently skin warming and heat loss. Moreover, the habitual sleep period coincides with the diurnal phase of increased skin temperature. A functional link between skin temperature and sleep has been suggested by Kräuchi and colleagues [3,4]. In a series of controlled laboratory studies, they showed that the gradient between the skin temperature of the hands and feet and the proximal skin temperature was highly correlated with subsequent sleep-onset latency. A key question is whether this correlation merely results from a single underlying sleep propensity increase that first shows in autonomous measures like skin vasodilation and only later in the central nervous system, as measured by the sleep-electroencephalogram (EEG). An alternative hypothesis [5] proposed that changes in skin temperature causally affect the ability to initiate and maintain sleep. The neurobiological mechanism proposed to underlie this causal relation is as follows.

1.1. Neurobiology and behavior

It has been shown that a subpopulation of warm-sensitive neurons (WSNs) in the preoptic area and anterior hypothalamus (POAH) spontaneously increases its firing rate at sleep onset. Experimental local warming of the POAH induces a similar increase in firing rate and facilitates sleep [6–8]. Consequently, it has been proposed that sleep would be facilitated when brain temperature exceeds a threshold level [6]. However, this

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proposition is in opposition to the chronobiological perspective – namely, that sleep propensity is actually minimal during the phase of high CBT. We proposed the warm-sensitive neurons involved in sleep regulation to be sensitive to skin temperature as well. The circadian phase of elevated skin temperature coincides with the period of maximal sleep propensity, and animal studies show that the activity of a high percentage of locally warm-sensitive neurons is strongly modulated by thermoafferent projections to the POAH originating in the skin [9]. Afferents conveying information about skin temperature modulate the firing rate of thermosensitive neurons in the POAH at least as strong as does local brain temperature. In case of simultaneous differential local brain temperature and skin temperature manipulations, the latter dominate the POAH response [9,10]. In addition, a recent *human* neuroimaging study demonstrated hypothalamic activation with warming of the skin [11]. Thus, the changes induced by direct local CBT warming and leading to sleep-related alterations in firing rate – changes that can be observed in experimental conditions – may well be induced by warming of the skin under more natural conditions.

The behaviors that occur while preparing for sleep strongly favor an increase in skin temperature. The postural change from upright or sitting to a supine position [12,13], the use of bedding to create a microclimate of 34 °C to 36 °C [14–16], and the relaxation associated with the preparedness to sleep that is signaled by lights-off [17] – all promote an increase in skin temperature. Since warming of the skin due to these changes occurs already before sleep onset, it could affect the process of falling asleep.

1.2. More evidence for a modulatory role of circadian changes in skin temperature

Several studies have shown that temperatures of the skin and, more specifically, temperatures of the skin of the extremities (i.e. hands and feet) increase prior to sleep onset. The potential role of skin temperature in sleep onset, was already recognized by Magnussen in 1939 [18]. He reported that peripheral vasodilation and hence an increase in peripheral skin temperature indicated "Schlafbereitschaft" or "sleep preparedness". Also, Kleitman reported on an increase in toe temperature before sleep onset [19]. Brown confirmed the elevation of toe temperature around sleep onset, and suggested that it was related to the onset of the first period of slow-wave sleep rather than to sleep onset [20]. Van den Heuvel and colleagues also reported on increased peripheral temperatures in the hand and foot prior to and after habitual sleep onset [21]. Kräuchi and colleagues showed that the degree of heat loss at the skin of the hands and feet relative to the proximal part of the body (distal to proximal gradient, or DPG) was the best physiological predictor of a fast sleep onset under strictly controlled experimental conditions [3,4]. Fronczek et al. [22] demonstrated that the DPG was increased in relation to the very short sleep-onset latencies of narcoleptic subjects, and that the association between skin temperature and sleep-onset latency was even stronger for proximal and distal skin temperature per se than for their difference. Lack and Gradisar focused on finger temperature on a finer timescale and showed that a rapid increase prior to the onset of sleep [23]. In another study, Gradisar and Lack concluded that the rise in finger temperature before sleep onset drives the decline in core body temperature, which in turn is related to sleep onset [24]. Recently, we showed that in a natural setting both distal and proximal skin temperature strongly increase around habitual bed times [25].

1.3. Thermal manipulations

In addition to the observational, correlational studies on diurnal changes in skin temperatures in relation to sleep onset, several studies have investigated the effect of manipulating body temperature on sleep-onset latency, by applying warm baths, warm blankets or water-perfused suits. Horne and colleagues showed in young adults that whole-body warming in the early afternoon induced sleepiness both during and following the warm baths, and decreased sleep-onset latency [26,27]. Other studies of bathing have demonstrated shorter sleep-onset latencies following passive body heating in the evening, but not after heating in the morning, and it has been suggested that the drop in core body temperature following heating of the body underlies these findings [28,29]. Sung and Tochihara showed that immersion of the body or the feet and lower legs only in a hot water bath before bedtime affected core temperature only marginally, but did result in an elevated skin temperature during the first part of the night and improved sleep-onset latency [30].

Other studies have applied passive body heating in elderly subjects. Kanda and colleagues [31] reported an increase in ease of falling asleep for both young and elderly subjects after taking a hot bath in the evening. Dorsey and colleagues [32–34] showed that taking a hot bath 1.5 to 2 h before bedtime resulted in a significant increase in SWS, but did not report on sleep onset.

It has been suggested that the mechanism by means of which passive heating of the body affects sleep is that warming promotes a subsequent steep fall in core body temperature, mimicking the decrease in CBT seen in the hours preceding habitual bedtime [26–28,30–32,34,35]. We have subsequently proposed that it is not so much the steep decrease in core body temperature but rather the underlying heat-loss activation that increases skin blood flow, and thereby skin temperature and heat loss, that is causally involved in the increase in sleep propensity. Of the aforementioned studies, only the study of Sung and Tochihara included both polysomnography and skin temperature measurements [30]. Of note, in this study, the sleep-promoting effects subsided as soon as a pre-sleep hot footbath-induced increase in skin temperature had normalized after 2 h of sleep.

Two studies explored the effects of sleeping with an electric blanket. Fletcher and colleagues found no effects on core body temperature in the first 3 h of sleep, but did not report on sleep onset or skin temperature [36]. Okamoto-Mizuno and co-workers showed an elevated foot temperature and bed microclimate temperature when using an electric blanket, but did not find an effect on sleep onset [37].

Using a thermo-suit for more controlled skin temperature manipulations, we showed reduced sleep-onset latencies with subtle warming of the proximal skin in the comfortable