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Effects of nocturnal decline in core body temperature and heart rate on sleep: a comparative study of two age groups

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Abstract

Background: Nocturnal core body temperature (CBT) decline is reflected in decreasing heart rate (HR) and is linked to sleep stage distribution, a relationship which is influenced by age and subject to temperature manipulation.
Objective: The present study investigates the relationship between CBT, HR, and sleep stage distribution by comparing two studies with healthy males: young and middle-aged men. The aim is to examine how these physiological systems might be interrelated.
Materials and methods: Data from two independent studies were compared to examine nocturnal sleep stages, CBT, and HR time courses.
Results: The findings revealed that younger men exhibited improved sleep consolidation, characterized by longer total sleep time and more slow-wave (SWS) and REM sleep, along with a sustained nocturnal decline in CBT and HR. In contrast, middle-aged men exhibited poorer sleep continuity and a less-pronounced decline in CBT, followed by an earlier rise. The CBT changes were reflected in HR.
Conclusion: These results suggest that the nocturnal profile of CBT may be linked to variations in HR and fluctuations in sleep stage distribution. Interventions that facilitate nocturnal heat loss and a reduction in CBT and HR could potentially support sleep quality across age groups, particularly regarding SWS.

Keywords
Sleep, slow-wave · Sleep, REM · Sleep stages · Body temperature regulation · Men

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The nocturnal decline in core body temperature (CBT) is a well-documented physiological process believed to support both the initiation and maintenance of sleep [1, 2]. Sleep maintenance is typically marked by fewer awakenings (W) and a reduced amount of time spent in stage N1 sleep (W + N1), often referred to as less fragmented or more consolidated sleep. The close relationship between thermoregulation and sleep is considered essential, as habitual sleep generally coincides with the circadian rhythm of CBT—tending to occur as CBT declines in the evening and ending as it begins to rise again prior to awakening [3–6]. The nocturnal heart rate (HR) is closely linked to changes in CBT and also reflects slow CBT changes during sleep [10]. Initiation of sleep is coupled with physiological changes that

facilitate this temperature drop, such as decreased heart rate (HR) and increased blood flow to distal skin regions (vasodilation), which enhances heat loss from the body's core to the periphery and then to the environment [2–6, 10]. In addition, a faster/steeper decline in CBT has been linked to a higher amount of slow-wave sleep (SWS; N3) and electroencephalography (EEG) slow-wave activity (SWA), a key component of deep restorative sleep [1, 3–5, 7, 8]. However, increased sleep pressure induced by sleep deprivation does not affect the thermoregulatory system, suggesting a unidirectional relationship between these two regulatory physiological systems [9, 10].

A significant body of research highlights that absence or impairment of this nocturnal CBT decline is frequently associated with disturbed or impaired sleep [1, 2]. For instance, individuals with certain types of insomnia, particularly sleep maintenance insomnia, have been observed to exhibit elevated CBT during the night [2]. This lack of adequate temperature downregulation at night is thought to contribute to a state of physiological hyperarousal, which is a core feature in many models of insomnia [1, 11]. Studies have shown that experimentally induced passive elevation of CBT before sleep can lead to a compensatory, more pronounced drop in CBT following sleep onset, which is accompanied by an increase in SWS [1, 12]. This underscores the importance of the CBT decline for achieving deep sleep.

The sleep stage distribution is also known to undergo changes with age, often characterized by increased awakenings, reduced sleep consolidation, and a notable reduction in SWS and SWA [4, 13]. Paralleling these sleep changes, alterations in the circadian regulation of CBT are also observed in older adults [14]. This includes a tendency toward an earlier phasing of the CBT rhythm and, importantly for sleep stage distribution, a potential reduction in the amplitude of the nocturnal CBT decline [13, 15]. Similarly, the nocturnal decline in HR was shown to be progressively attenuated by physiological ageing [29, 30].

Some research suggests that older individuals may awaken at an earlier phase of their CBT and melatonin rhythms, indicating that their sleep is more susceptible to arousal signals from the circadian pacemaker, especially during the latter part of the night [13]. The age-related decline in SWS has been specifically correlated with impairments in the nocturnal drop in CBT, suggesting that thermoregulatory changes might mediate, at least in part, the age-related changes of sleep stage distribution [2]. Studies that manipulate body temperature during sleep—such as using cooling mattresses to enhance heat loss—show promise for increasing SWS and SWA and enhancing the nocturnal drop in core body temperature regardless of age and gender [3–5, 7].

Given that middle-aged men experience less consolidated sleep than younger men and also exhibit altered thermoregulatory and cardiovascular physiology, the present comparative study between two male age groups aims to examine how these physiological systems could be interrelated.

Methods

Study design

This present study utilizes data combined from two separate studies conducted at university sleep centres in Turin, Italy (study 1; young men) and Berlin, Germany (study 2; middle-aged men). These studies investigated the impact of mattress-based temperature interventions designed to control nocturnal CBT reduction through conductive heat transfer. Both studies adhered to the Declaration of Helsinki, and all participants provided written informed consent under the approval of their respective local ethics committees. For more detailed information regarding the procedures and participant characteristics, please refer to the original publications [3, 5].

In both studies, participants were screened to exclude individuals with relevant pre-existing medical conditions and were classified as "normal sleepers." Nocturnal polysomnography was recorded concurrently with measurements of CBT and HR during a 450-min nocturnal sleep period. For a detailed description of all measured variables in both studies, see the original publications [3, 5]. Each participant slept for one night on a (cooling) high-heat-capacity mattress and one night on a (regular) low-heat-capacity mattress, with a 1-week interval between the two nights. The order of the nights was randomized and counterbalanced in a blinded manner.

Study population

Study 1 included 15 males with mean age 26.5 ± 1.4 years (range 25–30 years) and mean body mass index (BMI) 22.3 ± 1.4 kg/m^2 (range 18.9–24.6 kg/m^2), whereas study 2 included 33 males with mean age 46.2 ± 4.0 years (range 40–55 years; study 1 vs. 2: $p < 0.0001$) with BMI 25.2 ± 1.8 kg/m^2 (range 21.7–29.0 kg/m^2; study 1 vs. 2: $p < 0.0005$).

Physiological measurements

Polysomnography (PSG) was performed according to the current American Academy of Sleep Medicine (AASM) guidelines in university sleep labs with standard montages. The details are reported in the original publications. Data were blindly scored by sleep specialists according to the AASM criteria. The CBT was measured every 15 s using an ingestible temperature-sensing capsule sensor (Vitalsense Core Temperature Capsule; Hidalgo Ltd., Cambridge, UK) at a resolution of 0.01 °C. Skin temperatures were measured every minute using miniaturized thermometric buttons (ibuttons, type DS 1922L; Thermochron ibuttons, Maxim, Dallas, TX, USA). The ibuttons were attached to the mattress and subjects using adhesive surgical tape (Fixomull; Beiersdorf, Hamburg, Germany). Room temperatures were maintained at 21–23 °C constant room temperature.

Data analysis

The present analysis focuses on the differences between study 1 and 2, as no differences in mattress effects were observed in the reported data (for detailed comparisons, see Herberger, 2024 [16]). Data on individual sleep stages, CBT, and HR from both mattresses were pooled. Student's t-tests were applied for comparisons be-

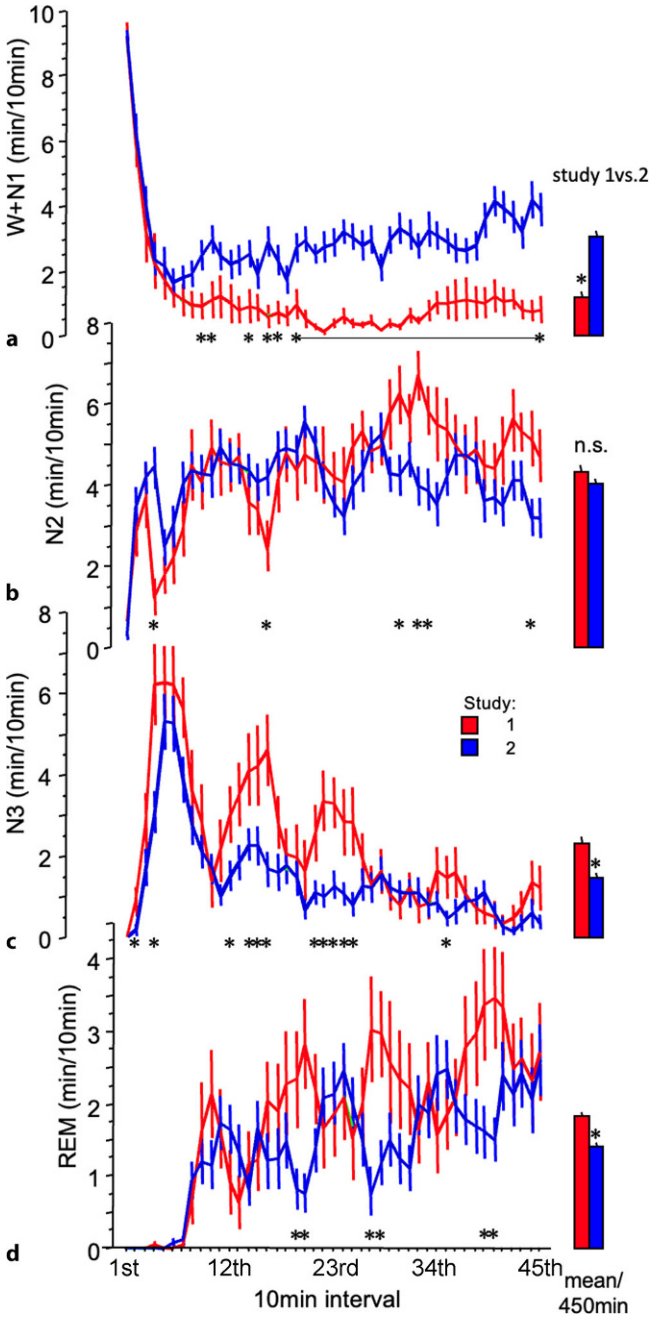

Fig. 1 ▲ Nocturnal time courses of awakening (*W*) + N1 (**a**), N2 (**b**), N3 (**c**), and rapid eye movement (*REM*; **d**) sleep, comparing the samples of study 1 (in red) and study 2 (in blue). Means ± standard error of the mean (sem) in 45 × 10-min intervals after lights out. The bar charts show nocturnal mean values ± sem (two-way ANOVA, main effect STUDY: all *p*-values at least < 0.005, except for N2 $p = 0.182$). Based on the finding that all two-way interactions (TIME × STUDY) revealed significant F values (all *p*-values at least < 0.05), false discovery rate post hoc tests were carried out. Asterisks indicate significant differences between the studies (*p*-value at least < 0.05)

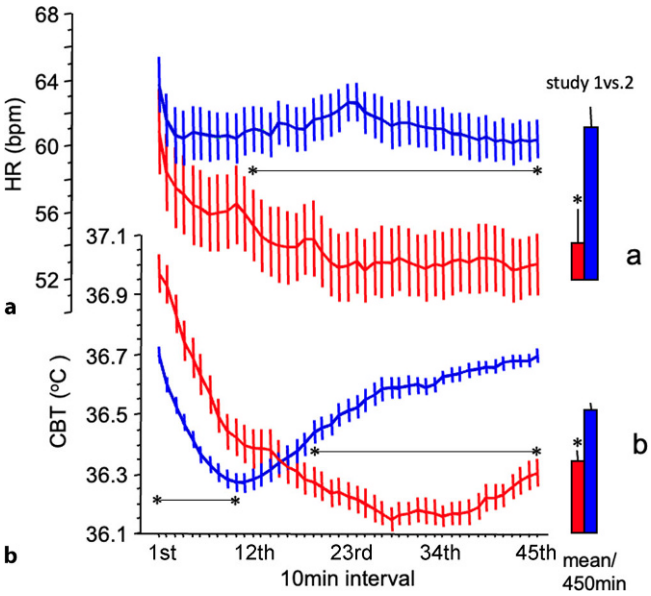

Fig. 2 ▲ Nocturnal time courses of heart rate (*HR*; **a**) and core body temperature (*CBT*; **b**), comparing the samples of study 1 (in red) vs. study 2 (in blue). Means ± standard error of the mean (sem) in 45 × 10 min intervals after lights out. The bar charts show nocturnal mean values ± sem (main effect STUDY: both *p*-values at least < 0.005). Based on the finding that both two-way interactions (TIME × STUDY) revealed significant F values (both *p*-values at least < 0.0001), false discovery rate post hoc tests were carried out. Asterisks indicate significant differences between the studies (*p*-values at least < 0.05). For detailed statistics see prior work [16]

tween mean values of study 1 and 2 (e.g., BMI).

All data were collapsed into 10-min segments (sleep stages: W, N1, N2, N3, and REM; CBT; HR) for subsequent time-course analyses. Analysis of variance (ANOVA) mixed effects models with repeated factors TIME (45 × 10 min) and MATTRESS (high- vs. low-heat-capacity mattress) and one grouping factor, STUDY (study 1 vs. 2), and SUBJECT (as a random factor) were performed using the package "nlme" in R (R Foundation for Statistical Computing, Vienna, Austria). Based on the finding that all variables revealed significant interaction terms (STUDY × TIME), post hoc comparisons between study 1 and study 2 were calculated for each of the 45 timepoints using the R package "emmeans." The analysis included false discovery rate (FDR) correction for multiple comparisons using the Benjamini–Hochberg procedure (threshold for significance: $p < 0.05$). Data processing and statistical analyses were performed using R version 4.4.1.

Results

This study investigated differences in sleep profiles, CBT, and HR in two independent study samples of young (study 1) and middle-aged men (study 2). Study 1 participants slept significantly better compared to study 2 participants: total sleep time (TST) as well as the time and relative amounts of time spent in sleep stages N3 and rapid eye movement (REM) were significantly higher, while sleep latency, time

Table 1 Nocturnal mean sleep data

	Study 1 (young)	Study 2 (middle-aged)
N1 (%)	3.9 ± 0.7	16.7 ± 1.6*
N2 (%)	50.2 ± 2.0	49.4 ± 1.4
N3 (%)	24.9 ± 1.7	16.9 ± 1.3*
REM (%)	21.1 ± 1.2	17.0 ± 1.1*
Sleep efficiency (%)	91.1 ± 1.6	81.6 ± 1.7*
Sleep onset latency (min)	10.8 ± 2.0	20.1 ± 2.0*
Total sleep time (min)	430.9 ± 7.1	391.5 ± 8.0*
Wake after sleep onset (min)	44.9 ± 7.9	68.4 ± 7.4*

Data are reported as nocturnal mean values ± standard error of the mean. For further information see the original articles [3, 5]
REM rapid eye movement
*significant differences between study 1 vs. 2; *p*-value at least < 0.05.

awake, and time in sleep stage N1 were significantly lower in study 1 (see **Fig. 1** for nocturnal time courses of sleep). Nocturnal mean sleep data are reported in **Table 1**.

Nocturnal CBT and HR showed marked differences between the two groups. The CBT in study 1 declined consistently for about 5 h after sleep onset and remained low for the remainder of the night, with a very small rise towards the end of night. Conversely, CBT in study 2 was about 0.3 °C lower at sleep onset and declined for roughly only 2 h, followed by a slow rise throughout the remaining 6 h of the night. Moreover, HR exhibited similar differences between the two groups and was strongly correlated with CBT in both studies: the average HR in study 1 followed a slow decline by up to 8 beats/min, while HR in middle-aged males showed only minor fluctuations of about 3 beats/min, which did not follow a systematic pattern throughout the night (see **Fig. 2** for nocturnal time courses of CBT and HR).

Discussion

This study investigated the interplay between nocturnal CBT, HR, and sleep architecture by statistically comparing two cohorts of healthy adult males of differing ages: a younger group (mean age 26.5 years; study 1) and a middle-aged group (mean age 46.2 years; study 2). Our findings confirm and extend previous research, highlighting distinct age-related patterns in nocturnal CBT and sleep stage distribution.

The primary observation was a significant difference in sleep consolidation and nocturnal CBT profiles between the two age groups. Study 1 exhibited objectively more sleep (increased sleep efficiency), characterized by TST, a higher proportion of slow-wave (N3) and REM sleep, and shorter sleep onset latency (SOL) and wake after sleep onset (WASO) compared to study 2. Sleep stage 2 (N2) did not differ between the groups. This aligns with established literature indicating a general decline in sleep consolidation and alterations in sleep architecture with advancing age, particularly a reduction in N3 sleep [13, 17, 18].

The nocturnal CBT trajectories also differed markedly. In study 1, CBT showed a sustained decline for approximately 5 h after sleep onset, remaining low thereafter. Conversely, in study 2, CBT was lower at sleep onset and declined over the initial 2 h, but it then began a gradual ascent for the remainder of the night. This pattern in the middle-aged group—an earlier nadir and a subsequent rise—is consistent with reports of age-related changes in circadian rhythmicity, including a tendency towards phase advancement and a potential reduction in the amplitude of the CBT rhythm [13, 15]. It has been specifically noted that while baseline CBT might not differ significantly in healthy aging, the dynamics of its nocturnal fall and rise can be altered [15]. Already nearly 100 years ago, Kleitman and Doctorsky (1933) stated that going to bed at night when the muscle tonus is low leads to a further decrease in tonus, as indicated by the low CBT [19]. It is therefore quite possible that the decline in CBT after lying down and falling asleep [20] differs between the two age groups, likely due to the well-documented aging process of the cardiovascular system [21].

The early upswing in CBT and HR after the initial drop after lights out in older adults may reflect age-related changes in the autonomic, vascular, and thermoregulatory systems that reduce the efficiency of the physiological "shutdown" that aids sleep consolidation. In younger adults, parasympathetic activation lowers HR and CBT after lights out, while in older adults, sympathetic dominance and weaker parasympathetic activation prevent a persistent drop, limiting the cardiovascular shift needed for restful sleep. Additionally, the decline in CBT is hindered by less efficient heat transfer from the core to the environment due to impaired blood flow redistribution.

Interplay between age, core body temperature, heart rate, and sleep stage distribution

A central question arising from these observations concerns the nature of the relationship between CBT, HR, and sleep stage distribution, particularly in the context of aging. Is the observed difference in sleep primarily a consequence of age-related changes in the circadian pacemaker, or does CBT itself directly modulate sleep structure—independently of or in addition to circadian influences? Our comparative study cannot provide conclusive evidence in this regard; however, it can at least highlight plausible associations in line with the existing literature that could be examined in prospective studies. Historically, research has pointed towards a change in the circadian pacemaker system with age, including a diminished circadian amplitude, e.g., as measured by CBT rhythm [17]. This could inherently lead to a shallower nocturnal CBT dip and poorer sleep consolidation. However, it is also noted that older individuals typically exhibit an earlier-timed circadian phase [13]. Thus, simply attributing the observed sleep differences solely to age as a global factor affecting circadian output is an oversimplification. Our findings, when viewed alongside evidence from temperature intervention studies [3–5, 7, 8], suggest a more direct role of CBT in modulating sleep. The original studies from which our current data are derived demonstrated that using a high-heat-capacity mattress to enhance conductive heat loss and lower CBT led to significant increases in N3 sleep and a reduction in HR in both young and middle-aged men [3, 5]. Quantitative sleep measures

(■ **Table 1**) further support this, showing higher percentages of N3 on the cooling mattress in both study 1 and study 2 [3, 5] and demonstrating a higher percentage of the night spent in deep sleep (N3%). This suggests that actively lowering CBT can improve deep sleep irrespective of the baseline age-related circadian characteristics. This aligns with work proposing that a failure of CBT downregulation at sleep onset could lead to SWS deficiency in insomnia [1, 12] and with studies discussing hyperarousal and elevated CBT [2, 11] and HR [22] in insomnia. The capacity of a cooling mattress to facilitate a more pronounced drop in CBT, as seen in the original intervention studies, appears to directly translate into enhanced N3 sleep—a stage crucial for restorative processes [3–5]. Therefore, while age-related changes in circadian timing and amplitude undoubtedly contribute to the differing sleep and CBT profiles observed in our cohorts, the direct physiological link between a lower CBT and improved N3 sleep and calmer HR seems to be an independent and significant factor. The mechanisms likely involve the intricate connections between thermoregulatory centres in the hypothalamus and sleep-regulating nuclei, where a cooler core temperature may facilitate the neuronal processes underlying N3 sleep generation and reduce sympathetic tone [6, 23–25].

Limitations

This study combines data from two independent cohorts, which, while matched for health status, inherently introduces variability. The age groups are distinct, but a continuous age spectrum was not assessed. Our analysis and focus on the age of the two groups is rather observational and limited to hypothesis generation; we cannot prove that age is the sole responsible variable, even though prior literature supports the idea. Studies were conducted in two different university sleep laboratories (Turin, Italy, and Berlin, Germany). Although identical protocols and lab conditions were used—including standardized temperature measurements—regional factors may still have introduced variability. These could include environmental or climatic differences or variations in inter-rater reliability of sleep scoring. The interpretation of causality between CBT and sleep, while supported by intervention literature, remains complex in an observational comparison between age groups where multiple physiological systems are changing concurrently. Various physiological changes associated with the onset of sleep appear to complement one another. For instance, simply lying down—even without falling asleep—can lead to decreases in CBT and HR, effects similar to those observed during the transition into sleep following lights out [20, 26]. However, to conclusively disentangle and clarify these effects—particularly in relation to the aging process—longitudinal intra-individual studies are required. Additionally, the relationship between sleep latency and thermoregulatory changes cannot be conclusively examined across the studies, as the necessary values prior to lights out are not available. In the present study, only men were examined (whereas a third study included only women [4]). To make well-founded and generalizable statements, studies involving mixed-gender groups with different ages are needed.

Implications and future directions

An interesting observation was the difference in non-REM (NREM) and REM sleep cycles. In our samples, younger men showed distinct REM and NREM sleep cycles, while middle-aged men also had less defined peaks. This difference, while purely observational in the current comparative analysis, might hint at subtle age-related alterations in the timing mechanisms governing NREM–REM cycling and its interaction with the circadian rhythm, which could be an avenue for more focused investigation in the future. Within this context, a previous study has shown that so-called quality sleep—comprising N3 and REM sleep—is most strongly associated with the age-related increase in overnight metabolic rate [27], which aligns well with our results.

The confirmation of distinct nocturnal CBT and HR profiles associated with differing sleep stage distribution across age groups reinforces the importance of thermoregulation in sleep health. However, unlike in smaller animals such as mice [6, 24], there is no clear temporal association between the NREM–REM sleep cycle and CBT in humans [16]. Our findings instead support the speculation that younger men, compared to their middle-aged counterparts, experience an enhanced nocturnal N3 sleep stage, potentially through increased sleep cycle amplitude. This appears to be accompanied—if not partly caused—by slower but more pronounced nocturnal declines in HR and CBT, as observed in study 1. Notably, evidence that direct modulation of CBT, e.g., via cooling mattresses, can enhance N3 sleep and lower HR is particularly relevant. This was demonstrated in our studies using a cooling high-heat-capacity mattress [3–5] and is further supported by findings from other investigations using different cooling methods [7, 8]. The potential effect of cooling mattresses on blood pressure is currently under investigation.

Implications of climate for sleep consolidation

A brief but pertinent consideration is the context of global climate change. Rising ambient temperatures and more frequent heatwaves are increasingly recognized as being detrimental to sleep [28]. Understanding the physiological impact of thermal stress on sleep and the potential for interventions that promote nocturnal heat loss, such as cooling mattresses or other environmental modifications, is becoming increasingly crucial. Future research could explore the efficacy of such interventions in mitigating the negative effects of heat exposure on sleep, especially in vulnerable populations such as the elderly. Further research should also continue to disentangle the direct effects of CBT on sleep from the broader age-related changes in circadian regulation. Longitudinal studies tracking individuals over decades would be invaluable in this regard. Additionally, exploring the nuanced differences in NREM–REM sleep periodicity and their physiological underpinnings could offer new insights into age-related sleep changes. Future studies must also show that not only the ambient air temperature is important, but

also the dissipation of body heat via conduction, e.g., into the mattress.

Conclusion

This study highlights significant age-related differences in nocturnal CBT, HR, and sleep architecture between healthy young and middle-aged men—the younger demonstrated a more sustained nocturnal decline in CBT and HR as well as superior consolidated sleep. While age-related alterations in the cardiovascular and circadian systems likely contribute to these differences, evidence from cooling mattress intervention studies suggests that CBT itself is a critical modulator of sleep stage distribution, particularly of N3 sleep and HR. These findings emphasize the importance of the sleep thermal environment and suggest that strategies aimed at facilitating nocturnal conductive heat loss may hold promise for improving sleep across different age groups—a consideration of growing importance in a warming global climate.

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Data Availability. The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest. S. Herberger, K. Lederer, A. Cicolin, M. Mason, M. Glos, C. Aurnhammer, M. Neumann, T. Penzel, I. Fietze, and K. Kräuchi declare that they have no competing interests.

This article does not contain any studies with human participants or animals performed by any of the authors.

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Zusammenfassung

Auswirkungen des nächtlichen Abfalls der Körperkerntemperatur und Herzfrequenz auf den Schlaf: Vergleichsstudie zweier Altersgruppen

Hintergrund: Der nächtliche Abfall der Körperkerntemperatur (KKT) spiegelt sich in einer Abnahme der Herzfrequenz (HF) wider und steht in Zusammenhang mit der Verteilung der Schlafstadien. Diese Beziehung wird vom Alter beeinflusst und kann durch Temperaturmanipulation verändert werden.
Ziele: In der vorliegenden Studie wurde der Zusammenhang zwischen KKT, HF und der Verteilung der Schlafstadien anhand des Vergleichs zweier Studien mit gesunden Männern, einmal jungen und einmal mittleren Alters, untersucht. Ziel war es, mögliche Wechselwirkungen dieser physiologischen Systeme zu analysieren.
Material und Methoden: Es wurden Daten aus 2 unabhängigen Studien verglichen, um nächtliche Schlafstadien sowie die Verläufe von KKT und HF zu untersuchen.
Ergebnisse: Die Befunde zeigten, dass jüngere Männer eine bessere Schlafkonsolidierung aufwiesen, gekennzeichnet durch eine längere Gesamtschlafdauer sowie mehr Tiefschlaf (SWS) und REM-Schlaf, begleitet von einem anhaltenden nächtlichen Abfall von KKT und HF. Im Gegensatz dazu zeigten mittelalte Männer eine geringere Schlafkontinuität sowie einen weniger ausgeprägten KKT-Abfall, gefolgt von einem früheren Anstieg. Veränderungen der KKT spiegelten sich in der HF wider.
Schlussfolgerungen: Die vorliegenden Ergebnisse deuten darauf hin, dass das nächtliche KKT-Profil mit Schwankungen der HF und Veränderungen in der Verteilung der Schlafstadien verbunden sein könnte. Interventionen, die den nächtlichen konduktiven Wärmeverlust sowie eine Abnahme von KKT und HF fördern, könnten potenziell den Schlaf altersübergreifend verbessern, insbesondere den Tiefschlaf (SWS).

Schlüsselwörter
Slow-Wave-Schlaf · REM-Schlaf · Schlafstadien · Körpertemperaturregulierung · Männer

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