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A review of the environmental parameters necessary for an optimal sleep environment



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ABSTRACT

An appropriate sleep environment is critical to achieve adequate quality and quantity of sleep. General sleep hygiene recommendations suggest that individuals should maintain a cool, dark, quiet sleep environment. Our goal was to conduct a review of the evidence surrounding the optimal characteristics for the sleep environment in the categories of noise, temperature, lighting, and air quality in order to provide specific recommendations for each of these components. We found that all forms of noise in the sleep environment should be reduced to below 35 dB. The optimal ambient temperature varies based on humidity and the bedding microclimate, ranging between 17 and 28 °C at 40–60% relative humidity. Complete darkness is optimal for sleep and blue light should be avoided during the sleep opportunity. Sea level air quality, with ventilation is optimal for sleep and supplemental oxygen is a useful countermeasure for improving sleep quality at altitude. Architectural design that incorporates these elements into bedroom design may improve sleep quality among inhabitants of such environments.

1. Introduction

Achieving sleep of adequate quality and quantity is dependent on an individual having enough time available for sleep at an appropriate circadian phase, with general adherence to basic sleep hygiene recommendations, including sleeping in an environment that is conducive to sleep. Recent consensus reports on sleep need support the notion that adults require 7-9h of sleep per night [33,117], and studies of sleep and circadian rhythms support the importance of regular sleep timing in positive sleep outcomes [29,100]. Although environmental sleep disrupters are recognized as a contributor to some sleep disorders, the empirical evidence supporting the recommendations on what constitutes an appropriate sleep environment are dispersed over many studies. In addition, there are few resources available to architects and engineers that provide explicit guidance on what characteristics are necessary for designing the optimal bedroom environment. An inappropriate sleep environment can lead to disrupted sleep and reduced sleep quality even in the absence of sleep disorders and when sleep is timed to provide for an optimal sleep opportunity. The goal of this review was to identify the impact of environmental factors on sleep, including ambient noise, temperature, light, and air quality, in order to guide the design of bedroom spaces optimized for healthy sleep.

2. Methods

We conducted a literature search to identify research papers describing sleep outcomes for the environmental parameters of interest. Given the prevalence of homographs within the search terms of interest (e.g. the terms "light" and "sleep" returned numerous results on "light sleep"), we took a three-tiered approach to include the widest range of literature. We conducted a primary search in PubMed using keywords relevant to each environmental parameter (described below). We conducted a screen of the abstracts to identify papers that met our inclusion criteria. We next conducted a search of the first 200 returned results using the same keywords in Google Scholar (excluding patents and citations) to identify papers that were not returned in our initial PubMed search. Finally, we reviewed papers that were cited in manuscripts that we identified in our primary search that did not come up in our keyword searches.

2.1. Search terms

In order to identify studies describing the impact of noise on sleep, we conducted a search using the terms "environmental," "noise," "human," and "sleep." In order to identify papers related to the influence of temperature on sleep using the terms "human," "sleep,"

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https://doi.org/10.1016/j.buildenv.2018.01.020 Received 12 September 2017; Received in revised form 9 January 2018; Accepted 10 January 2018 Available online 12 January 2018 0360-1323/ © 2018 Published by Elsevier Ltd. "ambient," and "temperature." For the section on environmental lighting we searched for "human," "sleep" and "light or lighting." For the section on air quality, we conducted two searches, for the first one we searched "human," "sleep," and "air quality," and for the second search we used the terms "human," "sleep," and "air pollution."

2.2. Inclusion criteria

Our literature search was limited to original research, adult human populations, and publications in English. We eliminated papers evaluating diseased populations or those with sleep disorders, because our goal was to summarize the impact of each environmental parameter on normal sleep.

There are several methods that have been used to quantify sleep outcomes, including electroencephalography (EEG), actigraphy, and self report, with EEG being the gold standard of sleep measurement. We included manuscripts that described the impact of sleep on each of these parameters. In order to weigh the strength of the methodology used for collection of sleep outcomes, we categorized the papers that we reviewed into levels of evidence using a modification of the Silagy and Haines Levels of Evidence for the strength of a study design as described in Table 1 (2001) [1]. We excluded studies that met the Silagy and Haines Evidence Level IV, which is described as expert committee reports or opinions or clinical experiences of respected authorities.

3. Noise

Exposure to noise can disrupt sleep quality and quantity [61]. The magnitude of sleep disruption conferred by noise depends on the decibel level (dB), the frequency and pitch, duration (i.e. continuous, intermittent, or impulsive) and whether the noise is meaningful (e.g. a familiar voice). A World Health Organization working group report on noise determined that there is a causal relationship between nighttime noise exposure and self-reported sleep disturbances, use of pharmaceuticals, self-reported health problems, and insomnia-like symptoms [122]. The same group issued guidelines for noise exposure during sleep, setting the limit for average level between 30 and 40 dBA, citing that higher levels of noise in the sleep environment leads to changes in the duration of sleep stages and increase sleep fragmentation.

Table 2 shows the literature describing the association between noise exposure and sleep disruption. There have been several high quality randomized (category 1A) or quasi-experimental (category 2A) studies using EEG that confirm that the auditory arousal threshold that causes a transition from sleep to wake varies between individuals and sleep stage [70,87]. Awakening due to noise exposure < 50 dBA is more likely in shallow stages of sleep (i.e. stages 1 & 2), where louder noises [42,43,69,121] or noises that are in the low frequency range (~500 Hz [15], are required to cause waking from deeper stages of sleep (i.e. stages 3 & 4. It is notable that the arousal threshold in REM

Table 1

Categories of evidence.

1a. Randomized trial or meta-analysis using EEG
1b. Randomized trial or meta-analysis using actigraphy
1c. Randomized trial or meta-analysis using self-report ratings, scales, or questionnaires

2a. Controlled study or quasi-experimental study without randomization using EEG 2b. Controlled study or quasi-experimental study without randomization using actigraphy

2c. Controlled study or quasi-experimental study without randomization using self-report ratings, scales, or questionnaires

 3a. Non-experimental descriptive studies using EEG
 3b. Non-experimental descriptive studies using actigraphy
 3c. Non-experimental descriptive studies using self-report ratings, scales, or questionnaires sleep is not easily determined due to the influence of dreaming (described in Ref. [87]). Complete awakening from sleep appears to be dependent on the frequency of repetition of the noise in addition to the volume, with more frequent pulses causing more sleep disruption [70]. Similarly, the auditory arousal threshold has been shown to change with repeated exposure to noise [11] and others have shown that there are large inter-individual differences in noise sensitivity [62]. The awareness that sleeping individuals have of their surroundings can also contribute to awakenings, such as speaking a sleeper's name [78], the sound of human voices [49,50], and household activity [95].

There have been few category 1 or EEG studies that have been conducted evaluating the impact of continuous noise on sleep. The two experimental EEG studies (category 1A) that have been conducted suggest that exposure to continuous ambient noise of 39 dBA or greater is associated with increased night waking, shorter sleep duration, and poorer sleep quality, including reduced REM sleep [60,97]. In contrast, continuous noise of 62 dB containing a blend of 1-22.05 kHz has been shown to protect sleep by masking the influence of exposure to other intermittent noises [101]. In the natural environment, continuous noise pollution typically emanates from sources such as exposure to wind turbines, however, it is unclear whether exposure to wind turbine noise influences sleep. One study found a dose response relationship with poorer sleep being associated with proximity to wind turbines generating 40-52 dBA using the Pittsburgh Sleep Quality Index (PSQI) and Eppworth Sleepiness Scale (ESS; category 3C) [71]. However, another study found no association between sleep outcomes and level of wind turbine noise ranging from < 25 dBA to 46 dBA using PSQI and actigraphy (category 3B) [63]. Similarly, a study that compared EEGmeasured sleep before and after installation of wind turbines in a neighborhood found no differences in objective or subjective sleep outcomes (category 1A), however, the noise exposure in that study was the same in both conditions (37 dBA, [37]. These findings suggest that quiet environments are better for sleep, but that continuous noise may be useful in dampening intermittent noises. More research is needed to determine what level and frequency of continuous noise is detrimental to sleep outcomes.

Intermittent noise is generally perceived to be more disruptive to sleep than continuous noise. There are numerous sources of intermittent noise that can infiltrate the bedroom environment. Several randomized studies using EEG in controlled environments (category 1A) have shown that exposure to intermittent noises such as simulated door slamming [115], passing trains [94] [6], aircraft flyovers [5] [6], and traffic noise [6,83] are associated with increased night waking, increased arousals from sleep, increased slow wave sleep, increased sleep stage transitions, and reduced total sleep time. Studies in the field largely confirm these findings. Several high quality observational studies using EEG have shown that intermittent exposure to noise in the environment is associated with sleep disruption. Traffic noise has been shown to increase the frequency of awakening, increased arousals (i.e. EEG defined awakening) and sleep stage transitions, which cause increased stage 1 sleep, reduced slow wave sleep, and reduced total sleep time (category 1A and 2A) [41,107,120]. Similarly, aircraft flyovers [7] and passing trains [26] have been shown to be associated with increased awakening, increased sleep stage transitions, and reduced slow wave sleep and total sleep time (category 3A). These findings are consistent with the results of an observational EEG study (category 3A) that found that overnight bell tolling from church bells is associated with night waking in a dose response manner [14]. Several survey and quasi-experimental studies using questionnaires, self-report, or actigraphy (category 2B, 2C) confirm that exposure to intermittent noise is associated with sleep disruption. Individuals who are exposed to higher levels of traffic noise self-report more disrupted sleep [10,81,103,114] and increased use of sleeping pills [103] relative to individuals with lower nocturnal traffic noise exposure. Similar findings have been reported for actigraphically-measured sleep disruption from aircraft noise (category 2B) [96] and also questionnaire-based measures of sleep

| Noise Source | Type of Disruption | Sleep Measure | Noise Level | Type of Noise | Source |
|---|---|---------------------------------|---|---------------|---------------------------------|
| Church bells | Dose response between awakening and noise level | EEG questionnaire | 30-70 dB | Intermittent | [14] |
| Rail noise | Higher noise level: increased arousals, awakening, S1, SWS, sleep stage transitions, reduced TST | EEG | 30-65 dBA | Intermittent | [6,26,94] |
| Aircraft noise | Higher modeled aircraft noise = no difference in sleep insufficiency compared to lower modeled noise | Questionnaire | Modeled noise of < 60 , 65 , $> 65 dBA$ | Intermittent | [34] |
| Aircraft noise | Reduced sleep quality | Actigraphy self-report | Quiet control, 60 dBA | Intermittent | [96] |
| Aircraft noise | Higher noise level: increased awakening, arousals sleep-stage transitions, reduced SWS, TST | EEG | 30-65 dBA | Intermittent | [5-7] |
| Traffic noise | Increased latency, awakening, consumption of sleeping pills, reduced sleep quality | Questionnaire self-report | > 45-75 dBA | Not reported | [103,114] |
| Traffic noise | Reduced TST, increased sleep latency, awakening | Actigraphy sleep logs | 40-45 dB (inside bedroom) | Not reported | [81] |
| Traffic noise | Higher noise level: increased awakening, S1, arousals sleep-stage transitions, reduced SWS, TST, WASO | EEG | 30-60 dBA | Intermittent | [41,83,107,120] |
| Traffic and rail noise | Individuals living in a dwelling with a quiet side reported reduced sleep quality | Questionnaire | Modeled noise of $< 40,40-60$, and $> 60 \text{ dBA}$ | Intermittent | [10] |
| Aircraft, traffic, and rail noise | Increased sleep latency, WASO, S1, reduced TST, sleep efficiency, SWS, REM | EEG | 32 dB vs. 39, 44, 50 dBA | Continuous | [60] |
| Wind turbine noise | Dose response relationship with poorer sleep associated with nearness to PSQI, ESS wind turbines | PSQI, ESS | 40-52 dBA | Continuous | [71] |
| Wind turbine (pre/post installation) | No differences in EEG sleep, reduced self-reported sleep quality | EEG self-report | 37 dBA (pre), 37 dBA (post) | Continuous | [37] |
| Wind turbine noise | No association between sleep outcomes and level of noise | Actigraphy, PSQI self-report | < 25, 25-30, 30-35, 35-40, 40-46 dBA Continuous | Continuous | [63] |
| General noise in the sleep environment | Fragmentation of sleep and impact on duration of various sleep stages | N/A | 35 dBL _{Amax,} inside | Not specified | World Health Organization Limit |

- dBA = A-weighted decibels (where low frequencies are reduced); dBL_{Amax, inside} = maximum levels per event inside a bedroom; dB = decibels; EEG = electroencephalogram; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale.

disruption from rail noise (category 3C) [10]. In contrast, a questionnaire-based study (category 3C) comparing modeled aircraft noise on sleep found no changes in sleep insufficiency among those living under higher modeled level noises [34], however, that study used the exposure category of < 60 dBA as a comparison group, which would include noises loud enough to be disruptive to sleep. Together, these findings largely confirm that intermittent noise is disruptive to sleep outcomes.

In summary, we reviewed numerous high quality studies that support the notion that noise pollution causes sleep disruption. Our findings suggest that exposure to intermittent noises above 35 dB are associated with reduced sleep quality and quantity. There were few studies describing the influence of continuous noise exposure on sleep outcomes warranting further research in this area. In general, our findings support the importance of locating bedrooms away from common spaces in order to reduce the impact of household and familiar noise on sleep. In buildings where sleep and common spaces must be colocated, such as in hospitals, hotels, and dormitories, measures to reduce noise emanating from other rooms (such as sound attenuating doors) should confer a positive impact on sleep quality and quality for residents. Similarly, bedrooms should be insulated against exposure to noise pollution from the outside environment, particularly when buildings are situated near highways, railways, and airports. In cases where noise pollution cannot be eliminated through insulation or other sound attenuating measures, the use continuous white noise may be useful in minimizing sleep disruption.

4. Ambient temperature

The relationship between endogenous core body temperature, skin temperature, ambient temperature, airflow and humidity, clothing, and insulation of bedding must all be taken into account when evaluating the impact of temperature on sleep (Fig. 1). Under normal conditions, the circadian rhythm of core body temperature declines just prior to the time of optimal sleep onset and continues to decline throughout the sleep episode, reaching a nadir at approximately 6 h after sleep onset [25,48]. During a circadian entrained sleep episode, the morning waking occurs on the rising phase of core body temperature rhythm. This change in core temperature arises from a drop in heat production due to a reduction in the metabolic rate [99] and heat loss due to inactivity [48]. In contrast, proximal and distal skin temperatures rise during sleep, relative to active waking [113]. In situations where individuals have control of their sleep environment, optimal temperature conditions are typically self-selected through the use of bedding and clothing to create a microclimate within the ambient environment.

During wake and sleep, the core and skin temperature interact to maintain a balance between heat loss and heat production. Ambient thermoneutrality, the point at which ambient air temperature allows for optimal maintenance of core (36-38 °C, 96.8-100.4 °F) and skin temperature (32 °C, 89.6 °F), in the absence of clothing and bedding, ranges from 27.9 to 28.5 °C (~82-83 °F; [44]. Ambient temperatures above this range induce sweating, while temperatures below this range induce shivering. Table 3 summarizes the ambient temperatures required to maintain thermoneutrality with varying insulation. There are several high quality studies (category 1A) that support the notion that ambient temperature and insulating material must be sufficient to maintain thermoneutrality. When individuals sleep with minimal clothing and no blankets, ambient temperatures above and below the thermoneutral zone increase night waking, while temperatures above the thermoneutral zone both increase night waking and alter sleep architecture [3,17,32,109]. In addition, one study demonstrated that there are sex differences in tolerance to heat and cold, with men experiencing better sleep quality at lower temperatures compared to women [79]. When insulation, such as clothing and a blanket are used, hotter ambient temperatures are more disruptive to sleep compared to colder temperatures, likely due to the fact that access to insulating material allows

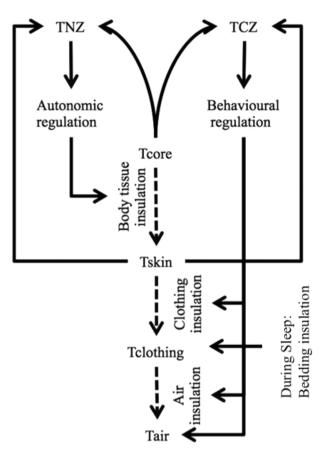


Fig. 1. Schematic overview of autonomic and behavioral control of thermal insulation. Solid arrows denote relation and/or control and dashed arrows denote heat flow. Adapted from Ref. [44]. TNZ = thermal neutral zone, TCZ = thermal comfort zone, Tcore = core body temperature, Tskin = skin temperature, Tclothing = clothing insulation, Tair = ambient temperature and airflow.

for individuals to modify the sleep environment during a sleep episode (i.e. through the addition or removal of blankets) [51,56,75]. This finding has also been demonstrated in individuals sleeping with an electric blanket, where warmer ambient temperatures combined with the heat from an electric blanket are more disruptive compared to sleep without an electric blanket (category 1A) [28,40]. In contrast, EEG-derived sleep outcomes have been shown to be improved when individuals sleep with an electric blanket under a very cold ambient temperature of 3 $^{\circ}$ C (category 1A) [77], supporting the need to maintain balance between ambient temperature and the bedding microclimate to support sleep.

Changes in temperature during a sleep episode can also influence sleep quality according to several category 1A studies. When ambient temperature is changed from cooler temperatures (25 °C) early in the night to hotter temperatures (28 °C) later in the night, sleep latency is increased relative to a night under constant temperature (26 °C) [52]. In contrast, when ambient temperature is hotter (29.5 °C) at the beginning of the night and slowly adjusted to a cooler temperature (27.5 °C), slow wave sleep has been shown to be enhanced [109]. Similarly, skin warming through use of a thermosuit (warmed to \sim 34 °C with no other insulation) has been shown to reduce sleep latency, enhance slow wave sleep and reduce night waking relative to skin cooling (category 1A) [85,86]. It is important to note that despite these observed improvements in sleep quality associated with warmer temperatures, individuals self report thermal comfort as worse when temperatures are elevated prior to attempting to sleep [52]. These findings suggest that manipulations in ambient or microclimate temperature within the thermoneutral zone can enhance or disrupt sleep depending on the type of manipulation used.

Table 3

Summary of studies describing the influence on temperature on sleep.

| Condition | Temperature °C (\pm SD) | Relative Humidity | Sleep Outcome | Impact on Sleep | Reference |
|------------------------------------|--|--|--------------------|--|--------------|
| Clothed (awake) Nude (awake) | 19.4 °C (± 5.0) 28.2 °C (± 0.3) | not reported not reported | N/A | None, thermoneutrality for waking measures | [44] |
| Clothed | 17, 20, 23 °C | 50% | EEG self-report | Men reported better sleep quality, shorter sleep latency at 17 °C, women reported better sleep quality, shorter latency at 23 °C, SWS reduced at 17 °C for both sexes | [79] |
| Clothed | 20, 35 °C | ~20% | EEG | Increased S4, reduced S2 in the 20 °C condition | [3] |
| Semi-nude | 21, 24, 29, 34, 37 °C (\pm 0.5) | 20-60% | EEG | More wakefulness, lower TST, reduced REM in 21 °C and 37 °C relative to 24 °C, 29 °C, 34 °C | [32] |
| Clothed | 23, 26, 30 °C | 10-95% | EEG self-report | Reduced sleep quality, shorter sleep latency, reduced SWS in 23 $^\circ C$ and 30 $^\circ C$ sleep conditions | [51] |
| Clothed | 26, 25–28 °C | | EEG self-report | Increased sleep latency in 25 °C condition, reduced comfort in 28 °C condition | [52] |
| Clothed | 26, 32 °C | 50% | EEG | Increased WASO, awakening, reduced REM, efficiency in 32 °C | [75] |
| Semi-nude | 27.5, 29.5 °C | 60% | EEG | Increased SWS when temperature slowly reduced from 29.5 to 27.5 during the sleep episode | [109] |
| Semi-nude | 28.5, 32, 39 °C | ~28% | EEG | 30% of SWS episodes disrupted by heat (39 °C), 60% disrupted by cold (28.5 °C), 67% of REM episodes disrupted by heat, 78% disrupted by cold | [17] |
| Clothed | 32 °C (± 0.5) | 80% (+airflow) 80% (-airflow) | EEG | No difference in sleep compared to 26 °C 50% humidity condition with airflow Reduced sleep duration, increased waking duration, reduced sleep efficiency | [76,77,112] |
| Clothed | 32 °C (± 0.5) | 80% | EEG | Increased waking, reduced S2 and S4 compared to 26 °C 50% humidity | [111] |
| Semi-nude | 35 °C | 75% | EEG | Reduced sleep efficiency, reduced SWS, REM compared to 29 °C 50/75% humidity | [73] |
| Clothed | 20, 35 °C | ~28% | EEG | Increased S1, sleep stage transitions, WASO, reduced REM duration | [56] |
| Cutaneous warming thermosuit | ~ 34 °C (suit) | Not reported | EEG | Faster sleep latency with skin warming compared to core and distal warming | [85] |
| Cutaneous warming thermosuit | \sim 31.7 °C (\pm 0.1), 34.6 °C (\pm 0.1) (suit) | Not reported | EEG | Warmer temperature reduced night waking, increased SWS | [86] |
| Electric blanket vs. control | 3 °C (ambient) | 50-80% | EEG | Reduction in S1 and REM with electric blanket | [76,77] |
| Electric blanket vs. control | 36 °C (core) | Not reported | EEG | Reduction in TST, increased night waking, reduced S1, REM, SWS | [40] |
| Electric blanket Cooling pillow | Not reported 26 °C 32 °C | Not reported 50% 80% | EEG EEG | Increased wake, sleep stage transitions with blanket vs. control Increased waking in 32 °C, 80% humidity compared to 26 °C, 50% humidity, but no difference in sleep with cooling pillow in 32 °C, 80% | [28] [74] |

EEG = electroencephalography; SWS = slow wave sleep; S1 = stage 1 sleep; S2 = stage 2 sleep; REM = rapid eye movement sleep; TST denotes total sleep time; WASO = wake after sleep onset.

Ambient temperature and humidity are intimately linked. The optimal humidity for human comfort ranges between 40 and 60%. As humidity levels rise above this range, the ambient temperature feels hotter than the measured dry bulb temperature and as they fall below this range, the ambient temperature feels colder [102]. Exposure to very low humidity levels, such as during a cold winter, leads to increased prevalence of nosebleeds [90], itching, and dry eyes [105]. Conversely, exposure to high humidity is associated with poorer air quality and increased air concentration of mites, airborne fungal spores, bacteria and viruses and airborne contaminants [8]. When humidity levels are outside the 40-60% range, sleep is also negatively affected [38,73,74,76,111,112]. Although, the introduction of a fan in hot humid environments appears to improve EEG sleep quality (category 1A) [112]. Together, these findings highlight the importance of maintaining humidity levels in the sleep environment to levels consistent with daytime comfort levels.

In summary, our review of the impact of temperature on sleep quality revealed a large number of high quality studies that used goldstandard measures to characterize sleep disruption arising from controlled exposures to varying temperatures. These findings support the recommendation that the optimal design for the bedroom environment should include climate control capable of maintaining ambient temperatures between 17 and 28 °C with relative humidity between 40 and 60%, assuming that sufficient insulation is available to allow individuals a bedding microclimate that is maintained within the thermoneutral zone. Warmer temperatures within the thermoneutral zone that allow for warmer skin temperatures appear to facilitate sleep onset, while cooler temperatures within the thermoneutral zone appear to enhance slow wave sleep, while temperatures above and below the thermoneutral zone are associated with poorer sleep quality. In buildings where individuals may not have control of their ambient temperature, such as in hospitals, prisons, apartments, and nursing homes, maintaining ambient temperatures in the lower range may be desirable to prevent overheating (unless such individuals do not have access to sufficient bedding). In buildings where it is not possible to install climate-control systems, such as may be the case for temporary shelters, design features that maintain thermoneutrality and minimize heat retention and cold are desirable.

5. Light

Light leads to sleep disruption due to two factors; first, light resets the circadian pacemaker, leading to a shift in the timing of circadian phase relative to the scheduled sleep episode. Second, light is an environmental stimulus that can cause sleep disruption and night waking. The circadian rhythm is reset by exposure to light through the eyes Summary of studies describing the influence of lighting conditions on sleep.

| Lighting Condition | Change in Sleep | Sleep Measure | Source |
|--|---|-----------------------------------|---------|
| Pre-bedtime exposure to an e-reader compared to reading a book | Increased sleep latency, reduced REM | EEG | [18] |
| Pre-bedtime exposure to 685 nm light for 30 min compared to placebo | Improved PSQI score, increased sleep quality, increased sleep duration, shorter sleep latency | PSQI | [125] |
| 6.5 h of pre-bedtime light exposure of 6700 k compared to 3000 k | Reduced stage 4 sleep | EEG | [47] |
| 2 h of pre-bedtime exposure to 460 nm vs. 550 nm or darkness | Reduced SWS, lower delta power in the first sleep cycle, higher delta power in the third sleep cycle, reduced REM | EEG | [67] |
| Exposure to elevated levels of light at night ~289 radiance | Increased risk of sleep dissatisfaction, reduced sleep quality, increased confusional arousals, short sleep duration, delayed bed/wake time | Questionnaire | [72] |
| Bedside light of ~40 lux on at night compared to darkness | Increased stage 1, reduced SWS, increased awakenings, reduced theta, delta, and spindles | EEG | [20] |
| 2 ms flashes of light every 30 s for 1 h 2–3 h after bedtime vs. darkness | No changes | EEG | [124] |
| Exposure to 5 or 10 lux during sleep compared to darkness | Increased waso, stage 1, reduced stage 2, increased awakening, increased REM | EEG | [19] |
| Later morning timing of first exposure to > 10 lux of light | Increase waking, increased self-reported sleep disturbance, shorter REM latency | EEG, actigraphy, questionnaire | [116] |
| Morning light of 1000 lux for 1 h | Reduced awakenings, reduced daytime napping | Actigraphy, EEG, questionnaire | [45,46] |
| Five mornings of 8000 lux for 1 h | Reduced awakenings | Actigraphy | [93] |
| Morning exposure to dawn simulator of 100–300 lux | Improved sleep quality | GSQS | [55] |
| Dawn simulation 30 min prior to waking vs. control | Improved sleep quality | Self report | [108] |
| Dawn simulation of 10,000 lux of blue/green light prior to waking | No change in sleep between conditions | Actigraphy, PSQI, self-report | [31] |

SWS = slow wave sleep, EEG = electroencephalography, k = kelvin; nm = nanometers, PSQI = Pittsburgh Sleep Quality Index, Groningen Sleep Quality Scale.

[22,23,123]. Inappropriately timed light exposure is capable of inhibiting sleep onset and leading to shifts in circadian phase that can impact sleep on subsequent nights. Evening light exposure as low as 65 lux is capable of shifting circadian phase of melatonin by 1 h compared to evening light exposure of 3 lux [16].

The potency of the light stimulus on human circadian physiology depends on the wavelength of light in addition to the intensity of the light. The human circadian pacemaker is most sensitive to short-wavelength light in the 460–480 nm range [13,58,84]. This response follows a dose response with higher irradiances of blue light eliciting greater melatonin suppression and phase shifting effects [119]. In contrast, exposure to low intensity red light does not suppress melatonin [12].

The direct effects of evening or morning light exposure on sleep are difficult to disentangle from secondary effects on sleep related to circadian phase shifts, however, there have been some studies that suggest that light has an effect on sleep quality and architecture. Table 4 summarizes our findings on the influence of lighting on sleep. Exposure to light immediately before sleep alters sleep timing and architecture, but these changes seems to be dependent on the wavelength of light. High quality studies using EEG (category 1A) suggest that exposure to blue light of a high color temperature (6700 k) or narrow bandwidth (460 nm) before bed alters the distribution of slow wave [47] and REM sleep [67] at the beginning of the sleep episode relative to pre-bedtime exposure to darkness or lower color temperatures and wavelengths of light. Similarly, pre-bedtime exposure to light-emitting e-readers, with color temperatures that include a high proportion of blue light are associated with increased sleep latency and reduced REM sleep compared to pre-bedtime reading of a paper book with a bedside lamp (category 1A) [18]. In contrast, a randomized trial using the PSQI as a sleep outcome (category 1C) demonstrated that exposure to red light in the 30 min before bed is associated with improved self-reported sleep quality, shorter sleep latency, and increased sleep duration [125].

Exposure to light in the morning also appears to affect sleep outcomes. Later first exposure to morning light is associated with increased waking, shorter REM latency, and reduced self-reported sleep quality compared to earlier exposure to morning light (category 2A) [116]. Similarly, actigraphy and EEG studies (category 1A, 1B) have demonstrated that high intensity morning light exposure of > 1000 lux after waking reduces night waking and increases sleep quality during subsequent sleep episodes [45,93], which is supported by self-reported data as well [46] (category 2C). However, findings from studies using dawn simulators, which introduce a light stimulus during sleep have shown improvements in self-reported sleep quality (category 1C, 2C) [55,108], but no change in objectively measured sleep outcomes (category 1B) [31].

Light pollution, such as that infiltrating a room from outside street lamps or from low levels of light emitted from sources inside the sleep environment can also disrupt sleep. In a large-scale ecological study (category 2C), Ohayon and Milesi found that individuals living in areas with higher light radiance levels during the night were more likely to report poor sleep outcomes and dissatisfaction with sleep [72]. These findings have been confirmed in controlled EEG studies (category 1A) that have shown that exposure to as little as 5 lux of light, such as that emanating from a dim nightlight or table lamp, during the sleep episode results in changes in sleep architecture compared to sleep in darkness [19,20]. However, millisecond flashes of light during the sleep episode do not appear to alter EEG-measured sleep outcomes relative to placebo (category 1A) [124].

In summary, the current evidence surrounding the role of light exposure immediately before, during, and after the sleep episode suggests that light influences sleep outcomes. More research is needed to understand how the wavelength, intensity, and timing of light before and after sleep should be optimized to improve sleep outcomes. Current evidence supports the notion that pre-bedtime exposure to higher color temperature lighting containing more blue light is associated with poorer sleep outcomes, whereas warmer color temperatures may facilitate or improve sleep. More research is needed to understand how dawn simulators and morning light exposure influence sleep, particularly due to seasonal influences on the natural timing of dawn. There appears to be a direct negative impact of continuous light exposure during sleep, which supports the notion that complete darkness is

optimal for sleep. There are numerous steps that could be taken when designing buildings to eliminate or minimize light pollution in the sleep environment. Bedroom windows should be equipped with blackout capabilities, particularly in environments such as hospitals and hotels, where individuals have no ability to install external shades. In cases where this is not possible, providing individuals with eye masks may be useful. Dynamic lighting systems that modulate the spectral frequency in overhead lights to low color temperature lighting during the night, beginning in the hours prior to bedtime and shifting to higher color temperatures in the morning may minimize sleep disruption and inadvertent circadian phase shifts. In situations where such dynamic lighting systems are not feasible to install, it would be beneficial for nightlights and indicator lights illuminated during the sleep episode to utilize long wavelength red light.

6. Air quality

Poor air quality or gaseous air mixtures that deviate from typical Earth-based sea level air mixtures are capable of causing sleep disruption and impaired breathing during sleep. Exposure to reduced levels of O_2 and elevated levels of CO_2 can lead to sleep disruption. Ventilatory responses to hypercapnia have been shown to be lower during sleep than during wake [57,91] and a mean value of 3.8% (range 2.3–6.5%) end-tidal CO_2 partial pressure has been shown to cause awakening from sleep [30]. Similarly, there is a decreased respiratory response to CO_2 exposure during sleep compared to waking [88].

Table 5 summarizes the studies that have been conducted evaluating the impact of changes in air quality on sleep outcomes. There have been few studies examining air quality or airflow and ventilation in the sea-level bedroom environment and few of these studies have employed gold-standard measures to collect sleep outcomes. Students randomized to sleep in a dormitory with no airflow, where CO₂ levels averaged 2585 ppm, experienced reduced sleep quality and more disrupted sleep compared to a night of sleep with airflow (category 1B) [104]. This finding is important, given that one study examining 500 bedrooms in Denmark found that only 32% had nightly CO2 concentrations that averaged less than 1000 ppm [9]. Similarly, poor air quality, cigarette smoke, and room scents have been reported to be disruptive to sleep quality (category 3C) [2], as have exposure to cooking fumes [118]. Interestingly, exposure to pleasant scents such as lavender oil and unpleasant scents, such as ammonium sulfide have both been shown to increase delta power and spindle activity in EEG measured sleep (category 1A) [80]. Concerns associated with the presence of stagnant air, or intrusive smells in the sleep environment may be remedied by the introduction of a fan or open window; however, a study examining a personalized ventilation system (a fan that delivered fresh air to the face at 0.15 m/s) found no differences in actigraphyderived sleep quality or duration compared to no supplemental ventilation (category 1B) [53]. Although there were no observed differences

Table 5

Summary of studies describing air quality factors and their influence on sleep.

in sleep outcomes in that study, study participants reported that they preferred sleeping with the airflow. Similarly, another study found that individuals prefer constant airflow of 0.6 m/second over intermittent airflow (category 2B) [126].

Although few studies have been conducted on air quality outcomes in the typical bedroom environment, a great deal of research has examined sleep at altitude, which affects the gas mixtures that individuals breathe during sleep. The lower partial pressure of oxygen that individuals experience when sleeping at altitude can cause periodic breathing at 3000 m [64], and hypoxemia can occur during sleep at 4000 m [66]. Numerous category 1A and category 2A EEG studies have shown that this disturbed breathing may account for reductions in slow wave sleep [24,27,35,39,54,65,89,92,98,106] and increases in sleep fragmentation (category 1A, 2A, and 2C) [21,36,82,89,110,127] that have been reported among individuals sleeping at altitudes over 2000 m. Individuals who reside at altitude appear to be able to acclimatize to their environment and do not appear to experience sleep disruption associated with altitude [128]. Oxygen enrichment has been shown to increase slow wave sleep and reduce apneas among individuals sleeping at altitude, making it a potential countermeasure for individuals who are at altitude for short durations (category 1A, 1C) [4,59,68].

In summary, our review of air quality effects on the sleep environment suggest that the design elements necessary to ensure adequate air quality and ventilation are important for ensuring adequate sleep quality. There is a large amount of high quality evidence from studies of individuals at elevation to support the importance of maintaining sealevel gas mixtures for normal respiratory function and preservation of slow wave sleep. In hotels and other temporary residences at altitudes of 2000 m or greater, air systems that provide supplemental oxygen may be useful in mitigating the negative effects of sleeping at altitude among individuals who are not acclimatized to such air mixtures. There are a limited number of studies examining the influence of odors and ventilation on sleep, therefore more research is needed in this area. It appears that even at sea level, ventilation, through the ability to open windows or a fan, is helpful to reduce CO₂ stagnation and other intrusive scents. It is possible that exposure to some scents may enhance deep sleep, however, more research is needed in this area.

7. Conclusions and research gaps

Sleep is critical to health and daytime functioning. In order for individuals to achieve optimal sleep, they must have access to a sleep environment that allows them to achieve quality sleep, free of external disruption. We found that the optimal sleep environment should be insulated to attenuate intermittent noise, in particular noises above 35 dB. Some evidence suggests that continuous noise may be useful in situations where intermittent noise cannot be eliminated or reduced. More research is needed to understand what level and frequency of continuous noise is beneficial or detrimental to sleep outcomes. The

| Air Quality Condition | Change in Sleep | Sleep Measure | Source |
|--|---|--------------------------|-------------------------------------|
| Altitude of \geq 2000 m (effective O2 of \leq 16%) | Reduced SWS | EEG | [24,27,35,39,54,64,65,89,92,98,106] |
| Altitude of \geq 2000 m (effective O ₂ of \leq 16%) | Increased awakening | EEG | [21,36,82,89,110,127] |
| Altitude of 3800 m with O ₂ enrichment (O ₂ of 24%) | Increased SWS | EEG | [4,59] |
| Exposure to lavender oil, vetiver oil, vanillin, or ammonium sulfide | Increased delta power, increased spindle activity | EEG | [80] |
| Exposure to cooking fumes | Long sleep latency, reduced sleep quality | PSQI | [118] |
| Dormitory with no ventilation (CO $_2$ levels ~ 2395 ppm) compared to ventilated room | Reduced sleep efficiency | Actigraphy questionnaire | [104] |
| Poor bedroom air quality, exposure to cigarette smoke or room scents | Self-rated poor sleep | Questionnaire | [2] |
| Personalized ventilation device | No change in sleep quality | Actigraphy questionnaire | [53] |

SWS = slow wave sleep, EEG = electroencephalography, m = meters, PSQI = Pittsburgh Sleep Quality Index; O₂ = oxygen; CO₂ = carbon dioxide.

sleep environment should be capable of being cooled or heated to ranging between 17 and 28 °C with 40-60% humidity and should provide the inhabitant with some level of temperature control, either through providing the ability to change the thermostat or through the provision of insulating material. It appears that individuals can achieve normal sleep at very low ambient temperatures when sufficient insulation is provided, however, there are limited studies on this topic, warranting additional research in this area. Light should be eliminated from the sleep environment and mitigations such as black out capabilities for windows, should be provided for individuals who must sleep during the day. Safety lighting that must be illuminated during the sleep episode should be shifted towards long-wavelength light in order to minimize circadian disruption, but more research is needed in this area to determine how different wavelengths and intensities of light, such as those emanating from indicator lights, may influence sleep. We found several studies that support the notion that exposure to light before bed has a direct effect on sleep outcomes, but it is difficult to disentangle the circadian phase shifting properties of light from such influences on sleep. Additional research is needed in this area to separate these effects. The optimal air quality for sleep should mimic the sea-level atmosphere. Oxygen enrichment may be useful for promoting sleep at altitude for unacclimated individuals. More research is needed on how scents and airflow influence sleep as we identified few studies examining these factors. However, the available evidence suggests that it is helpful to have airflow in the sleep environment in order to reduce intrusive scents and CO2. In conclusion, our review summarizes findings on what specific factors constitute an optimal sleep environment. These recommendations should help reduce confusion surrounding the ideal environmental conditions for sleep and provide building and habitat designers with guidance on what parameters are required in the bedroom environment to facilitate optimal sleep.

8. Limitations

Due to the breadth of research on sleep related outcomes, interactions between sleep quality and various special populations were not considered in this review. It is possible that individuals with sleep disorders, for example, may benefit from different environmental conditions than the ones described in the current review. Additionally, although a systematic effort was undertaken to include all relevant articles pertaining to this review, it is possible that some articles were missed in this process. This, in part, could be due to search terms that have various meanings, applications, and populations of study (e.g., animal models). As with any literature review, the quality of the evidence is dependent upon the present state of the literature. We believe that our categorizations of the levels of evidence throughout this paper help identify gaps in the literature and areas for future study.

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