Sleep and Aging Skin

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Linna Guan, Reena Mehra, and Elma Baron

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L. Guan (✉) • E. Baron
Department of Dermatology, Case Western Reserve University, Cleveland, OH, USA
e-mail: Linna.Guan@case.edu; Elma.Baron@UHhospitals.org

R. Mehra
Sleep Medicine Center, The Cleveland Clinic, Cleveland, OH, USA
e-mail: Mehrar@ccf.org

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Abstract

Aging is a process that is universal and commonly outwardly manifested on the skin and often most significantly noticed on the skin. The prevention of skin aging is currently a multibillion dollar industry with progressive rapid expansion and continues to expand. Intact sleep and circadian rhythm regulation is known to have protective effects against systemic inflammation, oxidative stress, hormone dysregulation, DNA damage, and other variables that contribute to aging. This chapter is focused upon the examination of factors that relate sleep to skin aging. To dissect this relationship, it is necessary to recognize the definitions of adequate sleep quality and how it is assessed. Sleep occurs in alternating cycles of NREM sleep and REM sleep and disruption can decrease sleep quality. In fact, here are over 90 sleep disorders characterized by the International Classification of Sleep Disorders. Various tools in our armamentarium can assess sleep quality and identify sleep disorders including subjective measures collected from questionnaires and objective sleep measures gleaned from polysomnography. Sleep helps regulate physiological hormone levels and metabolism. When quality sleep is disrupted, these hormone levels become abnormal and cause aberrant metabolism and increased stress on the body. Some of the major hormones regulated by sleep are cortisol, glucose, and melatonin. Furthermore, sleep deprivation enhances inflammation, increased DNA damage, and decreased DNA repair; oxidative stress and emerging data implicate sleep disruption in carcinogenic risk. These adverse pathophysiologic consequences may contribute to signs of aging such as wrinkling and alterations in pigmentation. Biologic plausibility of these underlying mechanisms and available data identifying sleep disruption as a factor compromising skin health suggest that it is important to consider methods of improving sleep quality as part of maintaining a healthy skin, in order to minimize or delay such effects.

Introduction

Every night an individual goes through a period of rest during which the body is subjected to reduced activity and decreased sensitivity to external stimuli. Such experience is loosely defined as sleep [1]. During sleep the body's temperature, blood pressure, and physiological demands drop. Sleep is a vital part of life and plays an important role in information retention and processing, hormone and metabolism regulation, cellular repair, immune response, and aging, including skin aging [2]. To better understand the effect of sleep on skin aging, we must understand sleep's complexity and its effects.

Defining Sleep

Although people experience sleep slightly differently, there are common patterns found in normal sleep. Sleep is an easily reversible process characterized by cycles of NREM (non-rapid eye movement) and REM (rapid eye movement) sleep [2]. During NREM sleep, the body experiences decreased physiological activity with decreased heart rate, decreased blood pressure, and prolonged brain waves with greater amplitudes, the latter specific to slow-wave sleep [2]. NREM sleep consists of four stages. Each progressive stage represents a deeper and more restful state. Stage 1 is defined as the transition from wakefulness to sleep characterized by slow rolling eye movements. It is often during this stage that people experience the feeling of falling followed by sudden muscle jerks (so-called myoclonic or hypnic jerks) [2]. During stage 2, the body begins to relax as brain waves become slower and the heart rate and body temperature decrease. Sporadic surges of rapid waves called sleep spindles (fast alpha waves, 12–14 Hz) are observed on encephalography reflective of thalamocortical oscillations. Stages 3 and 4 (slow-wave sleep) are defined by profound relaxation of the body. The blood pressure, breathing rate, heart rate, and body temperature further decrease while brain waves are characterized by

slow delta waves at times mixed with smaller and faster waves [2]. The body becomes immobile and it is often difficult to awake during these two stages. REM sleep differs from NREM sleep in that REM sleep is characterized by fast and chaotic brain waves with rapid and shallow breathing, increased heart rate and blood pressure, and paralyzed limbs – a tumultuous state of sleep. Dream recall often occurs during REM sleep. Healthy sleep contains alternating NREM and REM sleeps in cycles of approximately 90–110 min, repeated four to six times with progressive lengthening of REM periods such that the predominance of REM sleep occurs during the latter part of the sleep cycle [2].

Measuring Sleep Quality

It is important clinically to be able to define sleep quality because of the immense role sleep plays in various aspects of aging. There are tools that have been used in sleep-related research and/or clinical care to evaluate sleep quality, which can also be used to determine the effect of sleep on skin aging.

Sleep Quality Questionnaires

The evaluation of sleep quality accounts for not only sleep duration but also other factors. One tool for measuring sleep quality is the Pittsburgh Sleep Quality Index (PSQI). The PSQI utilizes several parameters including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction over the last month [3]. The PSQI, developed in 1989, is a survey taken by an individual or clinical study subject that yields a number of points that then translate into a quantitative score. The maximum number of points on the survey is 21 and the minimum number of points is 0. The higher the number of points, the poorer the sleep. A score greater than 5 is associated with poor sleep quality [3]. It is one of the simple but effective tools for determining patient sleep quality and often used for research studies with large sample sizes. Other sleep-focused questionnaires that may assist with understanding subjective sleep quality include the Sleep Quality Questionnaire, Epworth Sleepiness Scale (assesses dozing propensity), Functional Outcomes of Sleep Questionnaire, and questionnaires focused on screening for sleep-disordered breathing such as the Berlin and STOP-BANG questionnaires. Furthermore, questionnaires targeted toward other sleep disorders such as insomnia and restless legs syndrome are also available.

Polysomnography

The gold standard tool for evaluating sleep disorders such as sleep-disordered breathing and disruption of sleep architecture is polysomnography. It requires the patient to sleep overnight in a sleep laboratory while his brain activity and vitals are monitored using sensors attached to the scalp and the body [4]. Some of the commonly monitored physiologic parameters measured include brain electrical activity using an electroencephalogram (EEG), eye movements using an electrooculogram (EOG), and heart rate using an electrocardiogram (EKG). Other vitals such as respirations (airflow and nasal transducer), thoracoabdominal effort (typically via inductance plethysmography), and oxygen saturation are also often monitored. Polysomnography is a clinical diagnostic tool that can be used to detect sleep disorders such as narcolepsy, idiopathic hypersomnia, parasomnias, and sleep apnea. The main drawback of polysomnography, particularly in research, is that it is expensive and time consuming to perform in large scale.

Actigraphy

An increasingly common tool for assessing outpatient sleep quality is wrist actigraphy [5]. The actigraph estimates a patient's activity using the frequency of movement in the patient's arm. Usually the actigraph is a small, watch-like device that the patient wears on the nondominant wrist. Since

muscle movements are more limited during sleep, the actigraph can measure sleep-wake cycles by monitoring the patient's movements. Actigraphy is an excellent tool for sleep studies that involve longer periods of time and unlike polysomnography can characterize sleep quality measures and patterns over multiple days/nights. Usually the actigraph is used to monitor patients for 1–2 weeks at a time [5]. It is relatively inexpensive. However, it lacks some details of sleep quality that the sleep questionnaires and polysomnography can provide.

The Effect of Sleep on Hormone Regulation and Skin Aging

Skin aging is classified into two types, intrinsic and extrinsic. Extrinsic skin aging is attributed to external factors such as UV radiation. Intrinsic aging of the skin is defined by structural changes due to genetics and normal physiology [6]. Unlike extrinsic aging that is accounted for by UV and toxin exposure, hormone changes and dysregulation are responsible for the majority of intrinsic aging of the skin [6]. Hormone dysregulation not only leads to aberrant metabolism which is ultimately responsible for the generation of reactive oxygen species (ROS) but also prevents the production of important antioxidants responsible for counteracting ROS and the damage they cause [7]. Sleep is one of the main regulators of hormone balance and rate of metabolism.

Cortisol

It has long been known that cortisol levels cycle in a circadian rhythm [8]. Cortisol is released in a pulsatile manner following adrenocorticotropic hormone (ACTH) release. The level of cortisol typically falls during the night, shortly after falling asleep, and reaches its lowest around midnight [9]. It then slowly reaches its peak in the morning around 3–9 a.m. [10]. Many diseases are associated with the dysfunction of this cyclical process including Cushing's syndrome, Alzheimer's disease, metabolic syndrome, and various mood disorders [11].

Studies have shown that short-term sleep deprivation of only one night corresponded with a 45 % elevation of cortisol levels the following night [9]. Long-term sleep deprivation showed similar results with elevated levels of glucocorticoids [12]. In the skin, elevated levels of glucocorticoids translate to lower permeability barrier homeostasis, less stratum corneum cohesion, decreased wound healing, and depressed innate immunity in the epidermis [13], all of which in turn could lead to intrinsic aging of the skin. Furthermore, sleep deprivation can cause physiological stress which induces excess glucocorticoid secretion and thus enhance skin aging. Insomniac psychologic stress, also known as IPS, is known to decrease epidermal cell proliferation, impair epidermal differentiation, and decrease the density and size of corneodesmosomes which hold keratinocytes together [14]. This can disrupt the skin's barrier against exogenous insults. The excess glucocorticoids also inhibit the synthesis of lipids, causing lower production and secretion of lamellar bodies which can lead to further damage to the epidermal barrier [15].

Glucose

During restful sleep, metabolic rate is reduced by approximately 15 % [16]. During the day when metabolic demand and glucose utilization are high, there is an increase in oxidative stress leading to increase in the production of radical oxygen species. During parts of non-REM sleep, there is a decrease in metabolic rate and brain temperature. This decrease helps the repair of damage from metabolically active periods.

When sleep deprivation occurs, glucose clearance is reduced by 40 % and the response of insulin to glucose is also reduced, leading to an elevated level of serum glucose [17]. The reduction of glucose tolerance after sleep deprivation is partially attributed to the increased levels of glucocorticoids. However, there also seem to be other mechanisms involved [7]. Studies have shown that slow-wave sleep (SWS) plays an important

role in regulating insulin and maintaining glucose homeostasis [8].

The elevation of glucose concentration negatively impacts the proliferation of keratinocytes as well as fibroblasts of the skin [18, 19]. Impairment of proliferation of skin fibroblasts leads to delayed wound healing [19], similar to the delay in wound healing observed in the skin of the elderly [20]. There is also evidence that an increase in glucose concentration leads to the terminal differentiation and change in morphology of these keratinocytes [18]. These terminally differentiated cells lose their ability to generate new cells and are arrested in a terminal state through the upregulation of cyclin-dependent kinase inhibitors and downregulation of positive mediators of the cell cycle [21]. This parallels the phenomenon of cellular senescence in that these keratinocytes treated with high-glucose levels show characteristics similar to senescent cells [21]. At high concentrations of glucose, the keratinocytes are larger and flatter and did not exhibit orientation toward each other which can negatively impact the function and integrity of the keratinocytes [18].

Melatonin

Melatonin is another hormone that has been extensively studied in relationship to sleep. Its release from the pineal gland via stimulation from the suprachiasmatic nuclei (SCN) of the anterobasal hypothalamus displays circadian rhythm [8]. Its levels are higher in the biological night and lower in the biological day [8]. Melatonin can also be produced in extrapineal organs such as cutaneous cells, but the level of secretion is variable and usually displays no circadian rhythm [22]. The release of melatonin can help reduce sleep latency, increase total sleep time, and improve sleep maintenance [8]. Furthermore, the therapeutic effects of melatonin on the skin are extensive. Skin cells express both membrane-bound and nuclear melatonin receptors which allow melatonin to play a role in multiple vital functions such as hair growth cycling, hair pigmentation, melanoma control, antioxidant activity, and suppression of ultraviolet-induced damage to skin cells [23]. Melatonin has been shown to decrease aging-related skin changes from oxidative stress and prevent extrinsic aging of the skin through protection against UV-induced skin aging [24, 25], both of which will be discussed later in more detail.

The induction of melatonin expression from the SCN is dependent primarily on the light/dark cycling within the day [26]. Because light is the most effective suppressor, the introduction of artificial light in modern-day life alters the induction of melatonin secretion through the SCN [26]. Studies have shown that the intensity of light used directly correlates to the amount of suppression of melatonin levels [27]. The common practice of using artificial light at night to increase productivity has a negative impact on the circadian rhythm of melatonin. Dysregulation of melatonin ultimately has an adverse effect on the integrity of the skin and prevents melatonin's natural antiaging benefits on the skin. This leads to both increases in extrinsic aging of the skin from UV damage as well as intrinsic aging of the skin from oxidative stress.

The interplay among various hormones involved in the maintenance of skin health and integrity is extensive and complex. While significant research efforts are still ongoing on how sleep can impact these hormones, it is agreed that sleep deprivation negatively impacts changes in these hormone levels that ultimately result in damage and accelerated aging of the skin.

Cellular Repair and Sleep

Sleep is a process indispensable for cellular repair. During sleep, the body undergoes cellular repair and renewal and protects against other harmful exposures. This process is vital to the maintenance of the integrity and structure of the skin and recovery of the skin from UV damage [28]. Dysregulation of sleep is linked with an increase in inflammatory activity [29], increase in cellular injury [30], increase in DNA damage [31], increase in radical oxygen species production [32], and change in transcription levels of various genes [33], all of which may contribute to both intrinsic and extrinsic aging of the skin.

Inflammation

Inflammation is a process mediated by various cytokines, notably IL-6 and TNF-α [34]. IL-6 has many functions including elevating body temperature through activation of C-reactive protein, activating other inflammatory cytokine pathways, stimulating the production of neutrophils in the bone marrow, and attracting neutrophils to the site of inflammation [35]. TNF-α also plays many roles in the body from coordination of organ development to mediation of acute adaptive immune response and apoptosis [36]. Elevated levels of both IL-6 and TNF-α are observed in modest sleep restriction [29]. These cytokines have been linked to the loss of facial subcutaneous fat through the inhibition of preadipocyte differentiation [37]. Adipocytes are the fat-storing cells of the dermis and loss of adipocyte differentiation can ultimately cause increased wrinkling and sagging, resulting in the appearance of an aged face [38].

DNA Damage and Repair

A vital process for the retardation of aging is DNA repair. Repairing DNA helps maintain cellular function and regulation and prevents apoptosis of the cell. Sleep deprivation not only causes cell damage but also predisposes the cells to replication errors. Significantly increased DNA damage has been found in murine models that suffer total sleep deprivation over the control [30]. Diseases that disrupt sleep and cause poor sleep quality in humans such as sleep apnea can cause increased stress on the DNA. Sleep apnea patients are found to have a significant increase in susceptibility to DNA damage and reduction in DNA repair [39]. Although total sleep deprivation studies on humans cannot be fully replicated due to ethical concerns, it can be inferred that similar results can be obtained.

Furthermore, DNA repair has been shown to be regulated by factors that display a circadian pattern [31]. Numerous proteins, including several that regulate DNA repair, are controlled by the biological clock. Timeless, Tipin, and human CLK-2 are all factors that regulate the biological clock that also have been shown to directly affect ATR/Chk1-controlled DNA damage checkpoints during S-phase progression [31]. ATR and its downstream effects, Chk1, are responsible for the regulation of replication checkpoints where ssDNA have stalled which can result in deleterious conformations or collapsed replication forks [40].

NPAS2, neuronal PAS domain protein 2, is a core circadian gene and a transcriptional regulator, which has been linked to DNA repair [41]. NPAS2 protein forms a heterodimer with BMAL1 protein as a part of the positive circadian feedback loop to activate circadian genes and regulate circadian rhythm [41]. Silencing the NPAS2 gene has been shown to decrease cell viability, increase susceptibility to DNA damage, and decrease DNA damage repair capacity [41]. Dysregulation of sleep and circadian rhythm can impair the DNA repair mechanisms. Skin aging and carcinogenesis are closely linked with DNA damage and inhibition of DNA repair [42]. Poor sleep quality can result in elevated levels of DNA damage and reduced levels of DNA repair.

Oxidative Stress

Along with increased DNA damage, sleep deprivation and sleep-disordered breathing also have been associated with elevated levels of reactive oxygen species (ROS) [30]. ROS refers to free radicals such as hydroxyl (OH•), superoxide ($O2^-$), nitric oxide (NO•), thyl (RS•), and peroxyl (RO_2•) and non-free radicals such as peroxynitrite ($ONOO^-$), hypochlorous acid (HOCl), hydrogen peroxide (H_2O_2), singlet oxygen ($^1O_2$), and ozone (O_3) [43]. Even normal metabolism naturally generates ROS. The electron transport system in the mitochondria of many cells uses electron carriers such as nicotinamide dinucleotide (NAD+) and flavin adenine dinucleotide (FAD) which are later reoxidized in the mitochondria to generate ATP [43]. During this process of ATP production, significant ROS by-products are generated. It is estimated that approximately 1–2 % of all

consumed oxygen is used for superoxide production [44]. In most cells, ROS is generated mainly in the mitochondria, although it is important to note that cells like hepatocytes can produce more ROS in the peroxisomes and endoplasmic reticulum than in the mitochondria [43].

ROS is also produced by various other pathways such as inflammation and UV damage. ROS plays important functions in normal physiology from destroying microbes in macrophages to regulating apoptosis [43]. However, superfluous levels of ROS can be produced during sleep deprivation. Being awake requires high levels of neuronal metabolism to retain electric potentials of the neurons [32]. As mentioned previously, sleep decreases levels of metabolism which can reduce ROS generation and provide for a state of increased antioxidant action to protect against free radicals [32]. Increased oxidative stress from sleep deprivation causes unwarranted apoptosis and cellular dysfunction through DNA damage and modification of proteins and lipids [43].

Oxidative damage is the major cause of skin aging. In the dermis, ROS imbalance leads to the destruction of collagen or impairment of collagen synthesis [44]. Breakdown of the dermal matrix causes wrinkling, a major sign of skin aging. ROS also increases destruction or impairment of differentiation of melanocytes, leaving the skin hypopigmented and vitiligo-like, which is another manifestation of skin aging [45].

Role of Antioxidants

Antioxidants can prevent and even reverse damage done by ROS. There are both enzymatic antioxidants and nonenzymatic antioxidants. Nonenzymatic antioxidants include compounds such as vitamin C, vitamin E, beta-carotene, and CoQ10 [45]. Most of these compounds such as vitamin C and vitamin E exert their antioxidant effects through the donation of an electron to a dangerous free radical. After donation of an electron, both vitamin C and vitamin E form radicals. However, they are relatively unreactive compared with the ROS and may be reduced back to its original state or donate its other electron [45]. Beta-carotene takes the free radical from the ROS to form an epoxide that can be later degraded while CoQ10 blocks lipids from forming peroxides. Enzymatic antioxidants function to "dismutate" superoxides to hydrogen peroxide through various mechanisms [45]. Some of these enzymes include superoxide dismutases, catalases, glutathione peroxidases, ferritin, and peroxiredoxins [45].

Melatonin functions as both a nonenzymatic antioxidant and an activator of enzymatic antioxidants. It scavenges ROS through single electron transfer, hydrogen transfer, and radical adduct formation [46]. It also activates antioxidant enzymes, inhibits prooxidant enzymes, and improves mitochondrial function to reduce radical formation [46]. Skin aging is marked by decrease in skin thickness, flattening of the dermoepidermal junction, and decrease in hair follicles and dermal papillae [24]. Interestingly, melatonin has also been proved to be able to reverse the process of skin aging through increasing the thickness of the epidermis and dermis, hair follicles, and papillae in murine models [24]. This further lends support to the role of melatonin as an important antioxidant that can relieve oxidative stress to hinder the skin aging process.

Carcinogenesis

As the skin ages, it becomes more susceptible to carcinogenesis. The effects of UV light accumulate over time and contribute to photoaging of the skin. Some of the mechanisms involved in the increase in susceptibility to carcinogenesis include increase in ROS production leading to damage of DNA, reduction in DNA repair ability, and decline in immune surveillance. These are all processes that may be associated with sleep deprivation. Sleep deprivation also causes increase in ROS production, decrease in RNA repair ability, and attenuation in immune function [43]. Various cohort studies have shown that "long sleepers" of greater than 9 h have been shown to have decreased risk of breast cancer compared with those who sleep less [47]. Sleep is a time when

the body can undergo repair and renewal, which likely explains the association between sleep deprivation, skin aging, and cancer development.

Sleep and Skin Aging

Sleep Quality and Skin Aging

The effectiveness of sleep is dependent on more than just the duration of sleep. Other factors such as sleep latency and sleep disturbances are also critical measurements of sleep quality. Sleep quality has a significant impact on the aging of the skin. It has been reported that chronic poor sleep quality, evaluated using the PSQI score, is associated with lower skin function, integrity, and perception [28]. Those with scores less than 5 on the PSQI and an average sleep duration of 7–9 h are considered good sleeper, while those with scores greater than 5 on the PSQI and an average sleep duration of less than 5 h were considered poor sleepers. The good sleepers were shown to have significantly lower intrinsic aging than the poor sleepers, determined using SCINEXA [28].

SCINEXA is an index used to evaluate intrinsic versus extrinsic skin aging based on 5 noninvasive parameters of intrinsic aging and 18 noninvasive parameters of extrinsic aging [48]. Major intrinsic aging parameters are reduced fat tissue and uneven pigmentation. Major extrinsic aging parameters include carcinomas and permanent erythema [48].

Good sleepers have better recovery from erythema 24 h after UV radiation compared with poor sleepers. Good sleepers also have better skin barrier recovery [28]. To test skin barrier recovery, both good sleepers and poor sleepers were tape stripped to the same transepidermal water loss (TEWL). After 3 days, good sleepers showed markedly greater barrier recovery (30 %) than poor sleepers [28]. The good sleepers also had better satisfaction with their overall appearance than the poor sleepers did [28].

Chronic poor sleep quality affects intrinsic aging of the skin, recovery from UV erythema, and skin barrier recovery. Poor sleep quality not only affects the integrity and function of the skin but also negatively impacts self-perception.

Sleep and Facial Appearance

In a separate study, subjects' facial appearances were evaluated post good sleep and post sleep deprivation [49]. The study used pictures of subjects following 8 h of sleep and subjects following 5 h of sleep and then 31 h of sleep deprivation. Random evaluators were asked to rate the pictures on various signs of fatigue. The factors include perceived fatigue, hanging eyelids, swollen eyes, glazed eyes, dark circles under eyes, pale skin, wrinkles/lines around the eyes, rash/eczema, sadness, tense lips, and droopy corners of the mouth. It was noted that those with 5 h of sleep followed by 31 h of sleep deprivation had more hanging eyelids, redder eyes, more swollen eyes, darker circles under the eyes, paler skin, more wrinkles and fine lines around the eyes, and droopier corners of the mouth [49]. They were also noted to look more fatigued and sad [49]. Items that did not seem to differ between the two groups were level of glazed eyes, rash/eczema, and tense lips [49].

The idea that sleep has a significant impact on facial appearance is not a new one. However, with consumers spending billions of dollars on cosmetic products to enhance facial appearance, it is important to remember that just having a good sleep can significantly improve facial appearance.

Diurnal Cycle of Skin Wrinkling

One of the major signs of skin aging that can be visualized is wrinkling. A study published in 2004 found that wrinkle formation on the face differed in the morning versus the afternoon [50]. It was revealed that measurements of skin wrinkling were significantly higher in the afternoon versus in the morning on the forehead, corner of the eye, and nasolabial groove. Skin thickness and skin elasticity were also measured and were both found to be significantly less in the afternoon than in the morning. It is speculated that this phenomenon is caused by a shift of fluid from

the skin of the face into the limbs from the morning to the afternoon [50]. Sleep would have a significant impact on this diurnal cycle since the horizontal position of sleeping would bring the shift in fluid back to the face. Therefore, it can be inferred that sleep deprivation would prevent the shift of fluid back to the face and promote wrinkle formation.

Sleep Lines and Facial Wrinkles

Sleep position can also affect the formation of facial wrinkles. Wrinkles or sleep lines are caused by continuous and repetitive pressure placed on the face from sleeping in the same position [51]. This constant force can eventually lead to elongation of the facial muscles and compression of wrinkles that persists even throughout the day. Since on average a person sleeps for one-third of his life, sleeping causes deepening of the wrinkles that worsens with aging [51]. Sleep lines are generally found as 2–3 parallel lines within a general area of the face [52]. Common areas of these lines are found in the lateral orbital, temporal, frontal, and buccal regions. People who sleep in prone positions are more likely to develop sleep lines than those who sleep in supine positions [52]. Furthermore, those who sleep on their left side are more likely to develop sleep lines than those who sleep on their right side [53]. Certain pillows can relieve the pressure on certain areas of the face and redistribute the gravitational force [51], but ultimately the only way to eliminate any pressure on the face is to sleep supine.

Independent of sleep position, individuals who have a mutation in the melanocortin-1 receptor (MC1R) gene also demonstrate an increased likelihood to form sleep lines [54]. The MC1R protein binds to melanocortin hormones such as ACTH (adrenocorticotropic hormone) and MSH (melanocyte-stimulating hormone) to regulate skin and hair color. It was also shown to have a role in photoaging of the skin. Certain mutations of this gene have been linked to significantly increased risk for photoaging when compared with wild type [54]. The same mutations of MC1R that are linked with photoaging have been implicated in increase in development of sleep lines.

The Role of Gender

Gender differences affect many aspects of the brain, most notably cognition and sleep. Some cognitive differences between the genders include that women perform better on verbal and memory tasks, while men performed better in spatial tasks [55]. Studies have also shown that women display less age-associated cognitive decline than men and that men show age-related neurocognitive decline at a younger age than women [55–57].

Differences in the sleep quality between the two genders have been well documented in previous literature [58]. Sleep differences that have been reported include: females experience longer sleep latency than males, women younger than 55 years of age report more sleepiness than men, older women sleep on average 20 min less than men, and women have more SWS and less NREM stage 1 than men [59]. As women age, they are 40 % more likely to develop insomnia and two times more likely to develop restless legs syndrome than men [60]. Mouse models have shown that some of these differences are due to the different sex hormones' involvement with each gender [59]. Murine models have shown that gonadectomy in female and male rates eliminated gender differences in the sleep-wake cycle, but hormone replacement of physiological levels restored these differences [61]. Furthermore, ovarian steroids were shown to inhibit NREM and REM sleep with greater suppression in female rats than in male rats [61].

On a different note, a study found that aging affected gender categorization of male and female adult faces differently. Compared to images of younger females, aging female faces took progressively longer amount of time to categorize. On the other hand, images of aging male faces took progressively shorter time to categorize [62]. The study suggests that female faces are progressively more difficult to categorize as female, while male faces are more easily identifiable as male as they age.

These findings suggest that although women display less cognitive decline as they age, their sleep quality is lower when compared to men. This puts the females at elevated risk for skin aging compared to the males. Furthermore, a large part of societal beauty standards comes from the distinction between the two genders. Since female faces seem to lose its "feminine" characteristics as they age, the desire to prevent facial aging is even more pronounced in aging females.

Recommendations for Good Sleep

Knowing the benefits of sleep and the detrimental effects of sleep deprivation on the skin, it is natural to want to know what it takes to achieve good quality sleep. There are different approaches to improving sleep quality. Two approaches are discussed here, sleep hygiene and lavender.

Sleep Hygiene

Sleep hygiene is a term coined by Dr. Peter Hauri to define various ways for patients to deal with their insomnia [63]. It recommends incorporation of various behaviors and environmental conditions that are conducive to good quality sleep and avoidance of behaviors and environmental conditions that result in poor quality sleep [64]. It is often used to treat or prevent sleep disorders and is a major component of cognitive behavioral therapy or multimodal therapy for treating insomnia [65]. Although there has been mixed findings on the effectiveness of incorporating sleep hygiene to improve sleep quality in the experimental setting, the components of sleep hygiene can still provide useful information to aid individuals with finding good quality sleep solutions.

Up to date, there has not been a consensus on what elements to include in a sleep hygiene treatment. However, some of the factors often associated with sleep hygiene include consistency of sleep and wake times, avoidance of daytime naps, avoidance of alcohol particularly close to bedtime, and exercise (preferably in the morning) before bedtime [64]. Having a regular bedtime helps align the circadian rhythm for the promotion of sleep [64]. It has been demonstrated that sleeping at approximately the same time every night and aligning light exposure around the same time increase the propensity for sleep. Daytime naps reduce nocturnal sleep pressure and are associated with decrease in depth of nighttime sleep and increased latency before sleep onset. Avoidance of daytime naps is associated with good sleep hygiene. Alcohol is well documented as a suppressant of nocturnal REM sleep and reduces upper airway muscle tone, hence resulting in worsening of sleep-disordered breathing [64]. It interferes with good quality sleep and should be avoided before bedtime. Studies have shown that exercise has a mixed effect on sleep. Exercising later in the day can help promote sleep depth. However, exercising prior to bedtime can delay sleep onset [64]. Increasing body temperature before bedtime has been shown to increase the depth of sleep. Activities such as taking a hot bath before bedtime have been proven conducive to good quality rest. It is suggested that a rapid decline in core body temperature before sleep can decrease sleep latency [64]. It is also recommended that the average adult obtain 7–8 h of sleep as in particular reduced sleep duration has been associated with long-term adverse health consequences such as weight gain and increased cardiovascular risk as well as compromise of alertness and vigilance.

Improving sleep hygiene can positively impact sleep quality. However, each individual is different and may be affected by different factors. An effective way to find out what constitutes good sleep hygiene is to experiment with different routines and environments.

Lavender and Sleep

Various studies have noted that there is a positive effect of lavender on sleep quality. A study on healthy Japanese students reported that nighttime exposure to lavender helps prevent sleepiness upon awakening [66]. In postpartum women, a similar effect was observed. Postpartum women

using lavender aromatherapy at night were shown to have a significant increase in sleep quality at 8 weeks follow-up evaluated using PSQI [67]. Lavender has also been indicated in attenuation of depression [68].

For those with mild sleep disturbances and those who would like to improve sleep quality, lavender aromatherapy at night can be suggested. There are no negative side effects of this inhaled essential oil when used appropriately [69].

Conclusion

One of the obvious signs of aging occurs where everyone can see it – on the skin. The market's demand to prevent and slow skin aging drives a multibillion dollar industry today. However, sound approaches to improve skin function and protect skin against aging do not come from expensive creams or gels. One of the effective ways to protect the skin is through adequate quantity and quality of sleep. Good quality sleep can attenuate sleep aging through many mechanisms highlighted in this chapter.

On the other hand, skin plays an integral role in regulating sleep from thermoregulation during sleep-to-sleep onset and sleep latency. All of which can in return augment the quality of sleep. Since the discovery of the relationship between sleep and skin aging, there is a stronger focus on improvement of sleep quality from a dermatological standpoint. We expect more studies in the future that are aimed at understanding this dynamic field.

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