REVIEW

Impact of lifestyle and technology developments on sleep

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Introduction

The discipline of modern sleep medicine is dated back to the early 1950s, following the discovery of rapid eye movement (REM) sleep and the identification of nightly sleep stage distribution.^{1,2} While much basic and clinical research has been done in the past six decades, advancing our understanding of the etiology and mechanisms underlying sleep disorders on the one hand, and enabling the application of clinical diagnosis and treatment practices on the other, it is important to keep in mind that sleep disorders existed and were described long before the mid-20th century.

Ancoli-Israel³ has investigated references to sleep in the *Bible* and the *Talmud*, regarding the function of sleep, the consequences of both sleep deprivation and excessive sleep, factors involved in the development of insomnia, as well as indications for its cures and treatments. Kryger^{4,5} referred to descriptions of sleep apnea in ancient Greek writings, and in the 19th century novelist Charles Dickens' *The Pickwick Papers*;

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and both Lavie^{6,7} and Guilleminault⁸ explored 19th century landmark reports of sleep apnea in the medical literature.

Clearly, sleep disorders are "nothing new under the moon",⁶ and the physiological and psychological mechanisms involved in their development remain similar throughout history. For example, Ancoli-Israel³ provides several quotes from the *Bible* regarding anxiety, stress and anguish in relation to sleeplessness; indeed, these are some of the same factors which are associated with the development of insomnia today.^{9–11}

Nevertheless, factors that potentiate the mechanisms involved in the development of sleep disorders are closely related to the "zeitgeist", ie, the sociocultural, technological and lifestyle trends which characterize an era. One salient demonstration of such factors refers to shift work, which has evolved from the 24/7 lifestyle enforced in modern societies. Shift work imposes a continuous misalignment between endogenous circadian (24-hour) rhythms and the environmental light/dark cycle. Such desynchrony in physiological and behavioral circadian rhythms has been shown to cause sleep loss and sleepiness, and to detrimentally affect mental performance, safety and health.^{12,13} Similar consequences may be found in cases of jet lag, yet another circadian rhythm disorder, which has become prevalent due to the vast expansion of transmeridian travel.^{12,14}

Other modern lifestyle factors affecting sleep are also closely linked to advances in modern technology, enabling and indeed encouraging later bedtimes and longer hours of nighttime arousal; eg, electronic media devices such as television and computers.^{15–17}Yet other factors that interfere with normal sleep/wake patterns include substances such as caffeine, nicotine, alcohol, and drugs, which are commonly consumed in attempts either to maintain alertness and arousal, or to achieve sleepiness and tranquility.^{15,18–23} Lifestyle changes in dietary and physical activity habits and the increased prevalence of overweight and obesity in modern society are also associated with sleep deprivation and disturbance.^{24–26}

The aim of this review is to highlight current lifestyle trends that have been shown in scientific investigations to be associated with sleep patterns, sleep duration and sleep quality. Available therapies used to treat some lifestyle related sleep disorders will be discussed. Future perspectives will be provided for the investigation of lifestyle factors that are associated with poor sleep, in terms of understanding the underlying mechanisms, as well as establishing appropriate therapies and public health interventions for their treatment.

Lifestyle trends associated with altered sleep patterns

The following sections will provide a review of the available investigations suggesting associations between various current lifestyle factors and sleep disturbances. Underlying mechanisms as well as some of the consequences of sleep disturbances related to lifestyle factors will also be discussed.

In an attempt to conceptualize these relationships, a hypothetical model is presented, elaborating the factors associated with chronic interference with sleep patterns in the modern world (see Figure 1). This model divides lifestyle factors into

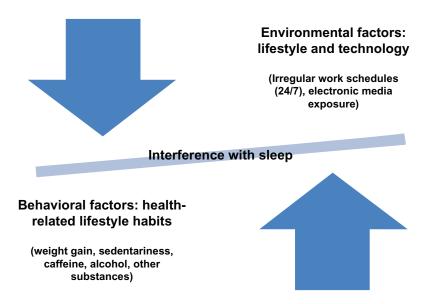


Figure I Schematic model conceptualizing the lifestyle factors that impinge on sleep, and distinguish between environmentally imposed technology-related lifestyle factors and behavioral lifestyle factors that may be considered as habits or countermeasures in response to environmental changes. Both categories ultimately create an imbalance in sleep quality, quantity and timing.

those that have emerged from environmental technological developments that disrupt normative sleep patterns, and those that may be viewed as behavioral habits, that may serve as countermeasures aimed to combat the deleterious consequences of sleep loss and sleepiness.

Technological developments and circadian desynchrony: alternative work schedules, jetlag, altered light exposure and electronic media

Shift work

In industrialized society, approximately 20% of the population works beyond the normative day shift, in various schedules of shift work.^{12,27–29} Ample research has been done on the effects of shift work on sleep and related health and performance outcomes.^{28,30–35}

The characteristics of sleep in shift workers have been reviewed by Åkerstedt.³³ Night shift workers exhibit shortened subsequent daytime sleep, more objective and subjective measures of sleepiness, lasting for several days following the night shift, and are prone to falling asleep during the shift, particularly towards the early morning hours. Morning shift workers experience curtailed sleep due to early rise times and subsequent daytime sleepiness. Taken together, reduced sleep and excessive sleepiness in shift workers have been implicated as mediators of impaired safety, productivity, performance and health.^{28,29,31,36,37}

To understand why shift workers experience reduced sleep time and sleepiness, it is necessary to describe the human circadian timing system. Physiological and behavioral processes, including the secretion of hormones, the sleep/wake schedule and the performance of mental activities, are known to oscillate in circadian (about 24 hours) cycles that are controlled by the biological clock, located in the suprachiasmatic nucleus of the hypothalamus. ^{12,38} Under normal conditions, circadian rhythms are entrained to the environmental light-dark cycle and are synchronized with it. Thus, time-of-day effects demonstrate increased sleep propensity and reduced alertness and performance capacity in the early morning hours, corresponding to the minimum in core body temperature, the peak of melatonin secretion and the timing of the habitual sleep phase.^{39,40} Changes in the timing of sleep and wake periods create circadian desynchrony. Shift workers on night or rotating shifts are required to remain awake during periods of increased sleep propensity; and the timing of their sleep phase is shifted to the daytime hours, when rhythms of performance, alertness and core body temperature are on the rise.^{12,39,40} Daytime sleep is shorter, ensuing in partial sleep deprivation and sleepiness. Such chronic exposure to circadian desynchrony entails serious consequences, by compromising safety, productivity and health.^{28,37,41} Some of the immediate consequences of shift work include increased risks for human error, accidents and injuries.^{31,37,42}

Shift Work Sleep Disorder (SWSD) has been defined by The International Classification of Sleep Disorders, 2nd edition43 as the presence of insomnia and/or daytime sleepiness associated with shift work. In a study on its prevalence and characteristics, the true prevalence of SWSD was 14% in night shift workers and 8% in rotating shift workers, after removing the percentage of insomnia and excessive sleepiness found in day workers.³⁶ Those with SWSD had higher rates of ulcers and depression, missed more work days and more family/social events, and had higher scores of neuroticism and more sleepiness-related accidents, than their shift work counterparts who did not have SWSD. For most of these outcomes, workers with SWSD had increased morbidity in comparison to their day work counterparts with the same symptoms (ie, insomnia and daytime sleepiness without shiftwork). These findings demonstrate the additive effects of symptoms of poor sleep (insomnia, excessive sleepiness) and work schedule (circadian desynchrony) on serious health and functional outcomes.12,36

Cross sectional and longitudinal investigations indicate that shift workers are at an increased risk for developing health problems, including gastrointestinal and cardiovascular diseases, diabetes and metabolic disorders, breast and colon cancers.^{12,34,41} Investigations on the mechanisms underlying these relationships suggest that sleep deprivation and circadian desynchrony are involved in changes in neuroendocrine function, reduced capacity of the immune system, metabolic disturbances and tumor growth.^{34,41,44,45} Exposure to light at night, leading to melatonin suppression, has also been implicated as an underlying mechanism (see below).

Flexible work systems

In addition to shift work, flexible work systems in which workers are not confined to traditional work hours have become increasingly prevalent.^{27,28} Flexibilization is a relatively new concept in organizational management, and is linked to trends in globalized economy.⁴⁶ Such nonstandard work schedules are highly variable among organizations, and may include working evenings, weekends, split shifts, on call hours or compressed work weeks.^{27,30} Though seemingly an attractive approach for allowing workers to tailor their work

schedule to their individual needs,⁴⁷ flexible work schedules may have a negative impact on workers' health.

In a study comparing traditional and nontraditional work schedules, scores of physical health, mental wellbeing and sleep quality were consistently higher for the traditional schedule workers.³⁰ Specifically, sleep quality was significantly lower in those working irregular shifts and compressed work weeks (10–12 hour 3–4 weekly shifts). Pending further investigation, the effects of flexible work systems on sleep and health may depend on workers' degree of flexibility; ie, increasing workers' control and choice of their work schedules may have beneficial effects on their sleep, health and overall wellbeing; whereas organizational enforced work schedules have a more negative impact on workers' overall health.⁴⁸

Jet lag

Jet lag is another circadian rhythm disorder which has emerged vis a vis the widespread popularity of transmeridian travel in recent decades.¹⁴ Circadian desynchrony between the sleep–wake schedule and other endogenous circadian rhythms with the environmental light–dark cycle creates symptoms of jet lag, including daytime sleepiness, difficulty falling asleep during eastward flights, early morning awakenings on westward flights, impaired alertness and performance as well as gastrointestinal problems and loss of appetite.¹²

The circadian timing system is able to readjust to new environmental time cues. On average, the adaptation rate is an hour a day, so that, depending on the number of time zones crossed and individual differences, travelers can expect their circadian system to adapt to the new time zone, given that they remain at their destination for a sufficient amount of time.¹² However, when transmeridian travel is ongoing, such as in the case of aircrew cabin workers, chronic jet lag is associated with reduced cognitive capacities and higher levels of cortisol.⁴⁹ Furthermore, chronic jet lag simulated in laboratory mice is associated with dysregulation of the immune system, that is mediated by circadian desynchrony but not by sleep loss.⁵⁰ Future investigations may further elaborate on the long term effects of jet lag on sleep, performance and health.

Both jet lag and shift work represent disorders in which a circadian system functions properly under normal conditions, but cannot adjust under voluntary or imposed phase shifts. For evaluation and management purposes, they are collectively referred to as circadian rhythm sleep disorders (CRSDs)⁵¹ (see treatment section).

Altered exposure to light

Light exposure plays a strong role in the circadian timing system. The resetting effects of light on the human circadian system have been described, demonstrating that timed exposure to bright light can create both a delay and an advance in the timing of circadian rhythms.^{52,53} Bright light during the night has also been found to have immediate effects on physiological and behavioral measures.⁵⁴ In comparison to dim light, bright light exposure decreased sleepiness, increased alertness, improved performance on behavioral tasks and attenuated the nightly drop in core body temperature. Taken together, these findings demonstrate that bright light may be used therapeutically to reset the circadian system, eg, in the case of jet lag; and to exert immediate effects of enhanced alertness in the context of nighttime shift work.

Indeed, light exposure has been shown to be a potent treatment for various circadian rhythm disorders (see section on treatment). However, accumulating lines of evidence point to the negative effects of exposure to light during the nighttime hours. Such exposure has become extremely common in various contexts in the modern world.

One line of evidence refers to nighttime light exposure as a risk factor for breast and prostate cancer.^{55–57} Epidemiological studies have shown an increased risk for breast cancer in women exposed to nighttime light in the home environment as well as in nightshift workers.^{57,58} Based on animal studies and human epidemiological studies, the light at night (LAN) theory implicates that circadian disruption and melatonin suppression by nighttime light exposure are the underlying mechanisms leading to tumor growth.^{59,60}

Another line of evidence of the harmful effects of mistimed light exposure refers to a new concept, termed asynchronization, in children and adolescents.⁶¹ This concept attempts to explain the high prevalence of insomnia and daytime sleepiness in children. Similar to the LAN theory, sleep is disrupted due to late night light exposure, causing circadian desynchrony and melatonin suppression. Also, lack of early morning light exposure prevents normal circadian entrainment to the environmental light/dark cycle. Future prospective intervention studies designed to enhance circadian entrainment in young individuals may provide support for the asynchronization model.

Finally, daylight savings time (DST), yet another relatively novel feature of modern society, has received little attention in the scientific literature. During the DST transitions in spring and autumn, social, but not environmental time cues shift abruptly, creating a one hour advance (in spring) and a

one hour delay (in autumn) of the timing of dawn relative to the shifted social norms. In a study using pooled data of several western European countries, which aimed to assess the effects of DST on physiological and behavioral entrainment to the environment, investigators have found that sleep time adjusts to the seasonal progression of dawn during standard time, but not during DST.⁶² Furthermore, when comparing morning and evening chronotypes, they found that sleep and activity rhythms fail to adjust to DST, particularly in evening chronotypes. Further investigations may assess the consequences of this failure to entrain during DST on measures of functioning and health.

Electronic media exposure

Associations between sleep patterns and electronic media exposure have been reported extensively in children and adolescents^{15,17,63–68} as well as in adults.^{66,69} Overall, electronic media exposure in children and adolescents was most consistently associated with later bedtime and shorter sleep duration, and the presence of a media device in the bedroom was associated with increased exposure, later bedtime and shorter sleep.^{16,17,63} In a 1-year prospective study of young men and women, looking at the psychological effects of exposure to various types of information and communication technologies (ICT), increased internet surfing for women, and increased cell phone calls and SMS messages for men, were found to increase the risk of developing sleep disturbances.⁶⁹

Attempts to conceptualize the underpinnings of these associations remain speculative. Van den Bulck^{16,70} has referred to electronic media exposure as an unstructured and boundless leisure activity with no clear endpoints, unlike other hobbies or sports activities. Shochat and associates¹⁷ have suggested that a media device in the bedroom may indicate high availability of the device and low parental control, both leading to increased exposure. It has also been suggested that electronic media exposure may have alerting effects,^{17,70} possibly due to the suppression of melatonin by the bright light emanating from electronic screens⁷¹ as well as due to their engaging and exciting content.

Cain and Gradisar⁶⁸ have suggested a model designed to demonstrate putative factors mediating the effects of electronic media on sleep in young individuals. Based on this model, background variables such as age, socioeconomic status and parental control may impact the intensity of electronic media use, including the presence or absence of a media device in the bedroom. In turn, proposed mechanisms that lead to sleep disruption include a physiological delay in the circadian sleep/wake rhythm, as well as physiological, mental and cognitive excitement and arousal. Sleep disruption subsequently leads to impaired daytime functioning. Future lines of investigation may further validate these relationships via experimental and longitudinal designs.

Behavioral patterns and countermeasures of sleep disorders and sleepiness: weight gain, physical activity and use of substances Weight gain

Levels of obesity and overweight have dramatically risen in developed and developing societies, reaching pandemic proportions in children and adults alike.^{72,73} In the past two decades the average level of obesity in OECD countries has risen by 8 percent.⁷⁴ In the United States in 2004, 17% of the child and adolescent population was overweight and one third of the adult population was obese.⁷⁵ These rates were significantly increased in children and adolescents and in adult males (but not women) compared to rates from 1999. The public health impact of obesity has been reviewed, linking obesity with type 2 diabetes, cancers, cardiovascular diseases, musculoskeletal disorders, respiratory disorders including sleep apnea, and all-cause mortality.⁷⁶

Environmental and behavioral changes have been implicated as the major factors responsible for weight gain,⁷² particularly changes in dietary habits (ie, high fat, energy dense diets), reduced physical activities corresponding to increased sedentary activities, eg, television exposure,⁷⁷ as well as decreased duration of sleep.^{72,77–84} Studies have investigated sleep as both a cause and a consequence of weight gain.

As part of a cross-sectional survey on health and nutrition in European teens, shorter sleep was associated with higher measures of obesity, including body mass index, waist circumference and body fat percentage.⁸¹ Short sleepers reported more sedentary time, watched more television and maintained a less nutritional diet compared to adequate sleepers (≥ 8 hours of sleep per night). In a six year prospective study, a comparison of adiposity measures was made on adults who were short sleepers at baseline (≤ 6 hours per night), between those who remained short sleepers and those who increased their sleep to 7-8 hours per night. These groups were also compared to long (7-8 hours) sleepers both at baseline and at 6-year follow up. Both short sleeper groups had comparable adiposity measures at baseline, whereas at follow up, the short sleepers group increased body mass index and body mass, compared to the increased sleepers group, who showed similar measures to the long sleepers.⁸⁴

Possible mechanisms underlying the relationship between short sleep duration and obesity have been suggested. Studies have demonstrated that regulation of the hormones leptin and ghrelin, signaling satiety and appetite respectively, is disrupted due to sleep loss, resulting in increased hunger and appetite.^{83,85–87} Furthermore, it has been shown that during television viewing, metabolic rate is low⁸⁸ and metabolic risk is increased.89 It has been suggested that increased television viewing leads to reduced sleep duration, which in turn leads to increased hunger and decreased metabolic rate, ultimately resulting in overweight and obesity.78 However, in a critical review, Marshall and associates⁹⁰ have concluded that there is insufficient and conflicting evidence regarding short sleep duration as a risk factor for obesity, and warned that advocacy of such a public health message is premature. Clearly, additional experimental and longitudinal studies are needed to determine possible causal pathways between these variables.

Conversly, obesity has been associated with an increased risk for sleep disordered breathing (SDB), both in children and adults.^{91–95} Tauman and Gozal⁹¹ have reviewed the evidence that obesity is a risk factor for obstructive sleep apnea (OSA) in children, and have suggested that in obese individuals low bioavailability of leptin, an important regulatory hormone of ventilatory mechanisms, may be a possible mediator. The authors further suggest that obstructive sleep apnea may in turn contribute to the strong links between obesity and comorbidities such as cardiovascular disease and the metabolic syndrome, possibly by amplifying inflammatory processes.

Nevertheless, obesity may not unequivocally be considered as a risk factor for SDB in young individuals.^{96,97} Kohler and van den Heuvel⁹⁷ have suggested that developmental stages as well as ethnic differences and their underlying anatomic and genetic predispositions are likely to be important moderators in this relationship. It may be concluded, that while obesity and SDB have many common clinical features and likely interact reciprocally, much investigation is needed to further understand the underpinnings of their relationship.^{96,97}

Finally, it is noteworthy that studies have found an increased prevalence of insomnia in adults with SDB.^{98,99} Of a random sample of patients diagnosed with SDB, Krakow and associates⁹⁹ found that 50% also had clinically significant signs of insomnia, and that those patients with both SDB and insomnia had more medical and psychiatric disorders and consumed more sedatives and psychotropic medications compared to SDB patients without insomnia. Whereas causal

relationships between SDB and insomnia are at present speculative, it may be assumed that SDB contributes to the development of insomnia via sleep fragmentation.⁹⁸ Inasmuch as rates of SDB have increased, in part due to the increase in weight gain,^{91,94} SDB may be considered as a lifestyle-related medical condition that is associated with insomnia. Whether weight gain itself is also associated with insomnia is yet to be determined.

Physical activity

Epidemiological studies have established that levels of physical activity (PA) are low in US adolescents and adults, and that PA declines and inactivity increases between adolescence and young adulthood.^{100,101} Yet investigations focusing on the relationship between sleep and physical activity, exercise, and inactivity in the community are few. Different PA protocols and measures, as well as individual differences in age, gender, and fitness obscure the ability to support the underlying assumption that exercise promotes sleep.¹⁰² In patients with sleep apnea, a tendency towards an inverse relationship between PA and the respiratory disturbance index (RDI) was demonstrated, ie, higher PA was associated with a lower RDI.¹⁰³ Findings from the Sleep Heart Health Study (SHHS)¹⁰⁴ have shown that vigorous PA performed at least 3 hours weekly was associated with a reduced risk for SDB, particularly in obese men. Clinical prospective protocols are warranted to assess the feasibility and efficacy of applying PA in intervention programs for SDB. In older adults, PA has been found to be a protective factor for incident and persistent late life insomnia.¹⁰⁵ Yet in a study assessing sleep and PA using actigraphic (activity monitor) measures in 8-year-old children, a bidirectional inverse relationship was found, showing that increased PA during the day was associated with lower subsequent nighttime sleep quality, and improved nighttime sleep measures were related to lower levels of PA on the following day.¹⁰⁶ Clearly, further investigations must be done to identify developmental changes and to clarify the underlying mechanisms in the relationship between PA and sleep.

Caffeine, alcohol and other health risk substances

In a recent nationally representative epidemiological study in high school students in the United States, insufficient sleep (<8 hours/per night) was reported by over two thirds of the students.¹⁰⁷ Strong associations were found between insufficient sleep and several prevalent health risk behaviors, including current use of alcohol (46%), smoking cigarettes (21%) and marijuana (21%), being sexually active

(36%), being involved in a physical fight (35%), low PA (66%), increased sedentary activities (TV \ge 3 hours [36%], computers \ge 3 hours [25%], see section on electronic media above) and signs of depressed mood (28%). Earlier epidemiologic studies have also reported that sleep problems and an evening tendency in adolescents were associated with the use of alcohol, caffeine, cigarettes, and illicit drugs.^{108,109} Psychiatric problems generally modified these associations, with the exception of illicit drugs and sleep problems, which were unrelated to psychiatric problems.¹⁰⁸

Given the high prevalences of both insuffucient/disturbed sleep and the use of health-risk substances, it is imperative to understand the underlying mechanisms of these associations, in order to develop appropriate public health interventions. In this section, findings on relationships between sleep disturbances and caffeine, alcohol and cigarette smoking will be discussed, as these health risk substances have been the main focus of investigation in relation to sleep disturbances.

Caffeine

Caffeine is a widely consumed psychoactive substance. As an adenosine antagonist, caffeine has been shown to attenuate electroencephalographic markers associated with the decrease of homeostatic sleep pressure.¹¹⁰ Caffeine has been shown to combat sleepiness and to restore alertness and performance in experimental designs; however, habitual daily caffeine consumption has been related to sleep disruption and sleepiness.¹¹¹ Evening consumption of caffeine has been shown to increase sleep latency and decrease sleep duration, sleep efficiency and stage 2 sleep in young and middle-aged adults.¹¹²

Caffeinated beverages have become increasingly popular among young individuals. Based on an epidemiologic study of over 7000 Icelandic 9th and 10th graders, 76% reported daily consumption,¹⁹ as was found in the United States poll of the National Sleep Foundation.¹¹³ Such a high prevalence greatly exceeds the lifetime prevalence of alcohol (56%) or nicotine use (28%) in the adolescent population.¹⁹ Though seemingly benign, caffeine consumption was independantly associated with increased daytime sleepiness and decreased academic performance, and substantially contributed to the negative relationships between nicotine and alcohol use and academic performance.¹⁹

Increased nightly caffeine consumption has also been associated with increased multitasking of electronic media devices prior to bedtime in middle and high school students.¹⁵ Students who both multitasked and consumed caffeine were 70% more likely to fall asleep at school, and 20% more likely to report difficulty falling asleep on school nights.

In a survey exploring patterns as well as reasons and expectations of caffeine consumption among adolescents, 95% of a high school sample consumed caffeine within the 2 weeks of the survey.¹⁸ Students reported drinking mostly caffeinated sodas, followed by coffee, with highest consumption rates in the evening hours. High caffeine consumers who drank both sodas and coffee reported more dependence issues, had higher expectations regarding caffeine as an energy enhancer and a substance that gets them through the day, and reported more daytime sleepiness compared to the lower caffeine consumers. Nevertheless, adolescents in this sample generally showed little concern with any of the effects of caffeine use, possibly indicating that they are simply unaware of these effects.

While most studies have focused on the effects of moderate levels of caffeine consumed in coffee and/or caffeinated soft drinks, few have focused on energy drinks, which are a fast growing segment of the beverage market and are particularly popular among youth.¹¹⁴ The content of caffeine in these drinks ranges between 50–500 mg per can or bottle. Findings from the Icelandic study showed that 38% of the daily caffeine consumers drank energy drinks, following 66% who were cola drinkers.¹⁹

The acute and long term effects of energy drinks on health and performance are largely unknown;¹¹⁴ however, reports of caffeine intoxication suggest that these drinks may increase problems of caffeine dependance and withdrawal as well as use of other drug substances. Longitudinal studies assessing the effects of energy drinks on sleep are warranted, taking into consideration not only differences in caffeine content and additional ingredients, but also looking at differences in drinking and sleeping patterns on weekdays versus weekends, as well as possible demographic mediating factors such as age, gender, socioeconomic status and culture.

Alcohol

The use of alcohol in young individuals has been shown to increase over time. In a recent study from the Netherlands comprised of two cohorts (1993, 2005–2008) and of 3 age groups (13–15, 16–17 and 18–21), the prevalence of alcohol initiation increased, particularly in the youngest age group (from 63% in 1993 to 74% in 2005–2008). Quantities of alcohol consumption also increased significantly between cohorts.¹¹⁵

The effects of alcohol on sleep are well documented. Based on PSG recordings in young adults, evening

consumption of a high alcohol dose (breath alcohol concentration [BrAC] 0.10) reduced sleep latency and efficiency and increased wake time throughout the night.¹¹⁶ Sleep consolidation was high during the first half of the night, and decreased during the second half. Subjective sleepiness increased, and sleep quality ratings decreased. These effects were more pronounced for women. Beyond its effects on sleep, alcohol created hangover and reduced performance on tasks requiring sustained attention and speed on the following morning.¹¹⁷

As alcohol consumption often begins in early adolescence, and as sleep disturbances are highly prevalent in this age group, Pieters and associates¹¹⁸ sought to investigate possible mediators of the relationship between the two. They found that puberty was related to alcohol use directly, but also indirectly, via sleep problems and delayed phase preference that typically begins in early adolescence. It was concluded that puberty-related changes in sleep regulation may underlie the vulnerability of young adolescents to alcohol consumption. In a prospective study of children of alcoholics, sleep problems in early childhood predicted substance use in adolescence, particularly in boys.¹¹⁹ Whether these finding generalize to children of parents free of substance abuse may be determined in future prospective investigations.

Cigarette smoking

Cigarette smoking has been recognized as a behavior that interferes with sleep. Laboratory and survey studies have reported that adult smokers experience more difficulty falling asleep and more sleep fragmentation than non smokers, probably due to the stimulant effects of nicotine.^{120–122} In a study assessing smoking patterns and sleep quality, night smokers had a higher prevalence of poor sleep and reported more sleep disturbances than non-night smokers.¹²³ Sleep duration was considerably shorter in night smokers who had poor sleep, compared to poor sleepers who did not smoke at night. Furthermore, night smoking and sleep disturbances both increased the risk of smoking cessation failure.

In smokers compared to nonsmokers, a higher prevalence of sleep bruxism has been reported.¹²⁴ However, no differences have been found for either restless legs syndrome (RLS) and periodic limb movements in sleep (PLMS),¹²⁴ or respiratory disturbances in healthy individuals.¹²⁵

Although general trends across cohorts demonstrate a substantial reduction in the prevalence of cigarette smoking and increased smoking cessation, adolescents and young adults constitute the most vulnerable age range for smoking initiation.^{126,127} In adolescents, cigarette smoking has been associated with an evening preference and with early puberty.^{109,128} In a survey on perceptions of sleep in high school adolescents, 6% of the entire sample reported that they smoke as a means to help them fall asleep.¹²⁹ In a prospective 2 wave study of insomnia and risk taking behaviors, the presence of insomnia predicted smoking within each wave, but not longitudinally.¹³⁰ Clearly, more prospective studies are needed to understand the direction of this relationship and how it may change developmentally.

Treatment of lifestyle and technology related sleep disturbances

The development of treatment strategies designed for specific lifestyle related sleep disturbances have largely focused on the treatment of circadian rhythm sleep disorders, ie, shift work and jet lag. Few interventions have been developed for other environmental and behavioral lifestyle factors affecting sleep, such as electronic media exposure, physical activity, weight gain and substance use. For standard guidelines on the diagnosis and treatment of sleep disorders, readers are referred to the International Classification of Sleep Disorders, 2nd edition.⁴³

Treatment of circadian rhythm sleep disorders (CRSD) and altered light exposure

Shift work and jet lag are treated based on guidelines for the treatment of CRSD,⁵¹ which include prescribed sleep scheduling, phase shifting with light exposure and/or melatonin (and its agonist) administration, and symptomatic treatments to combat insomnia and excessive sleepiness.

Prescribed sleep scheduling refers to changes in sleep and wake timing that are designed to optimize sleep quality during the sleep period and alertness during the wake period. "Chronotherapy" refers to treatment that is optimized by application based on oscillations of the circadian timing system. It was initially introduced to treat individuals with delayed sleep phase by gradual delay of sleep episodes until achieving a desired sleep phase.¹³¹ Chronotherapy and chronopharmacy have been under investigation for application in the treatment of cancers¹³² as well as hypertension and asthma.^{133,134} In shift workers, investigations assessing optimal shift schedules based on chronobiological principles have suggested that clockwise rotation is more beneficial to workers than counterclockwise rotation, in terms of adaptation, health and wellbeing.^{41,135}

Another sleep scheduling strategy is planned napping either prior to or during the work shift, aimed to offset the immediate effects of sleepiness on performance during the nightshift.^{136,137} These and other studies have demonstrated that short naps of 20–40 minutes were sufficient to improve alertness and performance measures during the night.

The circadian resetting and immediate alerting effects of light exposure have been described (see section on altered exposure to light). Though well-established, the applicability of achieving the effects of light to counter nighttime sleepiness in shift workers has been hampered by investigations demonstrating that nighttime light exposure and its suppressant effect on melatonin production may be the cause of an increased risk of tumor growth in animal studies.¹³⁸ However, investigational efforts to overcome this untoward effect, eg, by filtering low wavelength light, which suppresses melatonin, without compromising the positive effects of light on alertness and performance,139 may lead to revised guidelines for safe and efficient exposure to bright light during the night shift. Avoidance of early morning bright light on the ride home from work has also been found to be beneficial to allow a smoother transition to the morning sleep period.¹⁴⁰ For jet lag, appropriately timed light exposure at the destination of travel has also been found to be beneficial.¹⁴¹ Westward travelers may benefit from evening light exposure and avoiding morning light exposure at their destination; while eastward travelers should avoid evening light and be exposed to morning light, in order to achieve an advanced sleep phase.

To improve sleep in CRSD, melatonin and its agonist as well as sleep medications are recommended prior to the desired sleep time. However, sedating medications should be used with caution when used during the day, as they compromise alertness, safety and performance. Conversely, wake-promoting agents, modafinil and armodafinil, have proven effective for increasing alertness and performance during shift work.¹⁴¹

Finally, maintenance of sleep hygiene practices, such as achieving an adequate amount of sleep, sleeping in a dark and quiet environment and controlling caffeine and alcohol consumption, are recommended for the treatment of insomnia related to CRSD.^{12,51}

Behavioral lifestyle factors

While intervention studies aimed to modify health related lifestyle behaviors (eg, weight gain, sedentary activity and substance use) have shown some positive outcomes, few have reported sleep as a major outcome.

Randomized clinical trials have demonstrated that weight reduction has a significant impact on OSA.^{142,143}

In overweight individuals with mild OSA, a lifestyle intervention with a very low calorie diet significantly reduced body weight and apnea/hypopnea index (AHI), and improved related symptoms and measures of quality of life.¹⁴³ Similar findings were found for overweight individuals with moderate to severe OSA.¹⁴² Improvements were maintained at the 1 year follow-up in both studies.

Exercise interventions have shown some benefits in sleep quality for clinical populations^{144,145} and postmenopausal women.¹⁴⁶ Tai Chi, a traditional Chinese exercise of low to moderate intensity has shown positive benefits on sleep quality and daytime sleepiness in older adults.^{147,148} Aerobic exercise has been shown to decrease symptoms of sleep disordered breathing in overweight children.¹⁴⁹

In a public health intervention aimed at enhancing multiple health behaviors in high school adolescents,¹⁵⁰ positive outcomes included reductions in alcohol use and increases in fruit and vegetable consumption and participation in relaxation activities. Sleep measures, exercise, cigarette and marijuana use remained unchanged. Health education interventions specifically targeting adolescent sleep problems have shown that despite improvements in sleep knowledge and motivation, no behavioral changes in sleep practices were achieved.^{151,152} However, in a recent cognitive behavioral therapy (CBT) intervention on school-aged children, significant improvements were found in sleep measures such as sleep latency, sleep efficiency and wake time after sleep onset, and were maintained at follow-up.¹⁵³

In summary, interventions aimed at improving sleep disturbances that are related to specific behavioral lifestyle habits are only beginning to emerge. It is interesting to note that conversely, sleep extension has been recommended as an intervention to avoid weight gain and allow weight loss, particularly in young individuals;^{83,154,155} however, to date, outcome studies have yet to be reported.

Future perspectives

Despite ample evidence of the associations between lifestyle health behaviors and sleep quality, a comprehensive understanding of the causal relationships may be difficult to achieve. Lifestyle, technology and health behaviors including sleep are all intertwined and strongly embedded in the cultural and social environment. Prospective and longitudinal investigations following sleep patterns and related lifestyle behaviors are needed, to attempt to tease apart some of these variables and establish the temporal order of events. Such studies may also incorporate demographic, psychosocial and biological inter-individual differences,

to develop mediating models and to establish possible underlying mechanisms.

The development of public health interventions targeting specific lifestyle behaviors associated with poor sleep, tailored for different age groups, are warranted. Electronic media exposure, eating, and physical activity habits in children; risk behaviors and appropriately timed light exposure in adolescents and young adults are some examples.

Disclosure

The author reports no conflicts of interest in this work.

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