Article ID: WMCPLS001 ISSN 2051-0799

Narrative Topical Review of Sleep for Public Health Professionals

Corresponding Author:
Dr. Adam P Knowlden,
Assistant Professor, Department of Health Science, University of Alabama, University of Alabama P.O. Box 870311 Tuscaloosa, AL, 35487-0311 - United States of America

Submitting Author:
Mr. Adam P Knowlden,
Graduate Student, Health Promotion and Education, University of Cincinnati, University of Cincinnati 526 Teachers College P.O. Box 210068, 45221-0068 - United States of America

Submitted on:13-Apr-2013, 06:15:58 AM GMT

Accepted on:12-Sep-2013, 12:05:15 PM GMT

Published on:01-Oct-2013, 07:20:53 AM GMT

Article ID: WMCPLS001

Article Type: Original Articles

Article URL: http://webmedcentralplus.com/article_view/1

Subject Categories:PUBLIC HEALTH

Keywords: Sleep, health, epidemiology, sleep hygiene, sleep education

How to cite the article:

Knowlden AP, Sharma M.Narrative Topical Review of Sleep for Public Health Professionals. WebmedCentral *plus* PUBLIC HEALTH 2013;4(10):WMCPLS001

Source(s) of Funding:

None

Competing Interests:

None

Narrative Topical Review of Sleep for Public Health Professionals

Author(s): Knowlden AP, Sharma M

Abstract

The United States Healthy People 2020 report has outlined objectives to increase the percentage of adults who receive sufficient sleep from the 2008 baseline level of 69.6% to 70.9% by 2020. The purpose of this review was to provide public health professionals with an overview of sleep science, the impact of sleep restriction on physical and mental health, and behavioral strategies for improving sleep.Inclusion criteria for this review were (1) literature published in the English language; (2) between 1999 and 2013; (3) that focused on (a) sleep science, (b) sleep health, (c) and behavioral determinants of sleep. A total of 118 studies met the final tally of reviewed materials. Evidence suggests sleeping 7 to 8 hours is a critical element of optimum health. Despite this, sleep deprivation is commonplace in society. Epidemiological research has uncovered the adverse health ramifications of this trend, associating both insufficient and excessive sleep duration with increased rates of morbidity and mortality. Primary prevention interventions are needed to address the current sleep epidemic. Sleep hygiene and alertness management strategies can be incorporated intopublic health interventions to improve sleep outcomes and reduce physiological and psychological risks associated with sleep restriction.

Introduction

Adequate sleep is a critical component for health and well-being(1). Despite this, sleep deprivation is commonplace in society(2). Epidemiological research has uncovered the adverse health ramifications of this trend, associating both insufficient and excessive sleep duration with increased rates of morbidity and mortality(3). The Healthy People 2020 report has called for an increase in the percentage of adults who receive sufficient sleep from the 2008 baseline level of 69.6% to 70.9% by 2020 (4). The inclusion of sleep health in the United States *Healthy People 2020* objectives emphasizes a need for focused attention on sleep health from a public health perspective.

Although it is one of the primary pillars of optimal health, sleep has received minimal attention in the primary prevention, public health literature(5). A search by Ferrie et al.(6)of the epidemiological literature, identified approximately 10,000 papers on sleep in contrast to over 60,000 on obesity and more than on 50,000 smoking. Mounting evidence associatinginsufficient sleep with multiple deleterious health outcomes, including obesity, has begun to increase interest in sleep among researchers from a variety of fields(7). To the best of the authors' knowledge, there exists no comprehensive, narrative review of sleep for public health researchers and practitioners. Therefore, the purpose of this topicalreview was to provide public health professionals with anoverview of the recent literature about sleep. The specific aims of this review were to provide a topical review of (1) sleep science; (2) the impact of sleep restriction on physical and mental health; and (3) behavioral determinants of sleep relevant to the design of primary prevention public health interventions.

Methods

Inclusion criteria for this review were: (1) literature published in the English language; (2) between 1999 and 2013; (3) that focused on (a) sleep science, (b) sleep health, (c) and behavioral determinants of sleep. Database searches were conducted using EBSCOhost databases and delimited to Cumulative Index to Nursing and Allied Health Literature (CINHAL), Education Resources Information Center (ERIC), and Medline. Gray literature, including books and government reports, were also permitted in the search. Three phases of review were conducted to arrive at the final pool of sources. Phase I applied Boolean search terms of "Sleep AND Science AND Health AND behavior" to extricate the first round of articles (*n* = 155) from the CINHAL (*n* = 19), ERIC (*n* = 10), and Medline (*n* = 126) databases. Phase II encompassed preliminary distillation of the articles by removing duplicates (*n* = 3) and articles which were clearly outside the specific aims of this narrative review (*n* = 25). During phase III, the refined list of articles (*n* = 127) was reviewed by two independent researchers. Results found nine sources were outside

the scope of this review. Consequently, these articles were not considered for this review. Finally, a descendent search of all references cited from remaining phase III sources was conducted to uncover any literature that did not populate during the initial search process. No additional studies were identified through this process. Subsequently, 118total studies met the final tally of reviewed materials.

Results

History of sleep science. The first milestone in sleep science occurred in 1875 when Richard Caton, an English physician, detected electrical currents originating from the brains of animal subjects(8). In 1929, German psychiatrist Hans Berger succeeded in becoming the first to record electrical activity of the human brain(9). Berger was credited with the discovery of electroencephalography (EEG), a non-invasive technique for quantitatively measuring electrical cerebral activity (10). Berger conducted the first sleep EEG recording and observed that alpha rhythms disappeared once study participants fell asleep (11). Berger's breakthrough experiment provided an operational definition of sleep onset, establishing a fundamental paradigm for investigating sleep and sleep disorders (11).

In the mid-1930s, Loomis and colleagues recorded the first continuous overnight EEG, which led to the development of the sleep stage classification system(12). Aserinsky and Kleitmanrefined the classification in the 1950s with the discovery of rapid-eye movement sleep (REM)(13). Verification of muscle atonia during REM sleep by Jouvet, Michel, and Courjon established the modern taxonomy of human sleep stages (11, 14). Jouvet and colleagues further advanced the field by postulating that REM sleep was a third state of consciousness, characterized as an active brain within an inactive body (11, 14).

As the field progressed, concerns regarding the reproducibility and inter-rater reliability of sleep scoring began to emerge (15). In response, the Association for the Psychophysiological Study of Sleep chartered an ad hoc committee of sleep scientists in 1967 with advancing a standardized methodology for recording and scoring sleep stages (16). The effort led to the publication of *TheManual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects* in 1968 (17). Subsequent to this seminal work, sleep scientists were able to corroborate and compare research results. Sleep researchers widely accepted the manual for nearly 40 years (15). However, advances in the field called for the full-scale revision of the scoring guidebook(16). In the early 2000s, The American Academy of Sleep Medicine (AASM) commissioned the development of the updated manual, which resulted in the publication of *TheAASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology, and Technical Specifications*(18).

The first sleep disorder clinic was established at Stanford University School of Medicine in the 1970s (19). The clinic initially set out to study narcolepsy patients, but later grew to investigate an array of sleep disorders. By 2009, over 1,500 sleep centers and laboratories were accredited by the AASM (15). In conducting sleep research, researchers used polysomnography (PSG) to simultaneously monitor and record the three physiologic variables that compose the primary stages of sleep and wakefulness (20). The diagnostic tools incorporated in PSG include the electroencephalogram (EEG) to record brain activity, the electromyogram (EMG) to record muscle tone, and the electro-oculogram (EOG) to record eye movements(20).

Sleep physiology. *Sleep architecture.*Sleep is comprised of two distinct states spanning five stages of activity (21). Collectively, this construction is referred to as sleep architecture. The first state, NON-REM, is classified into four stages corresponding to EEG readings along a continuum of relative sleep depth (21). The second sleep state, REM, is characterized by episodic bursts of rapid eye movement and muscle atonia(22). NON-REM and REM sleep occur in alternating cycles lasting 90 to 120 minutes (11). A total of four to seven cycles transpire over the course of a complete sleep episode (20). The first sleep cycle consists of approximately 80 minutes of NON-REM and culminates with 10 minutes of REM(20). With each successive sleep cycle the amount of NON-REM decreases as the proportion of REM increases (11).

NON-REM stage one sleep plays a pivotal role in the initial wake-to-sleep transition, generally lasting 1 to 7 minutes (22). This stage, referred to as light sleep, is associated with a low arousal threshold (20). Duringlight sleep, the EEG waves are predominantly beta waves (14-40 cycles per second). Stage two persists approximately 10 to 25 minutes during the initial sleep cycle, and increases in length with each consecutive cycle, eventually comprising 45% to 55% of total sleep duration (11). During this stage, the EEG waves are predominantly alpha waves (8-13 cycles per second). Stage three has a brief interval and

consists ofonly 5% to 8% of total sleep(20). In this stage, EEG records theta waves (4-7 cycles per second). The final NON-REM phase, stage four, continues approximately 20 to 40 minutes during the initial sleep cycle, constituting 10% to 15% of total sleep (20). Typically, stages three and four are merged together and collectively referred to as slow-wave sleep(22). In stage four EEG records theta waves (4-7 cycles per second) and delta waves (near 1 cycle per second). REM sleep is most often associated with dreaming. During the REM state, the pons sends inhibitor input to the spinal cord to prevent the physical enactment of dreams (23). With each successive sleep cycle, the length of time spent in REM sleep increases, ultimately accounting for 20% to 25% of a sleep epoch(20).

Mechanisms of sleep-wake regulation.Sleep and wakefulness are regulated by thehomeostatic drive for sleep and the body's endogenous pacemaker, the circadian rhythm(24). Located in the preoptic area of the hypothalamus, the homeostatic system stimulates the propensity for sleep. The impetus towards sleep accumulates exponentially throughout the day until satisfied (25). As wakefulness is prolonged, the drive intensifies until the pressure to sleep becomes irresistible (11). Concomitantly, the circadian cycle regulates the diurnal sleep-wake sequence (26). Controlled by the suprachiasmatic nucleus located in the hypothalamus (11), the biological pacemaker is synchronized to the external environment (26). Coinciding with temporal cues, the circadian clock promotes wakefulness in rhythmic oscillations, with peaks in the late morning and early evening, and troughs in the mid-afternoon and middle of the night.

The two-process model of sleep regulation details the interaction between these biological forces (27). To illustrate the model, researchers define the homeostatic component as process S and the circadian system as process C (28). Process S promotes sleep and increases in propensity in a roughly linear manner during wakefulness, peaks prior to sleep onset, and dissipates precipitously as sleep ensues (29). Concurrently, process C counteracts process S by promoting wakefulness (29). Controlled by environmental cues such as light and meal timing, the endogenous circadian rhythm (process C) operates according to an intrinsic 24-hour timekeeping system which gradually wanes as the state of wake progresses (29).As the body's homeostatic drive for sleep is satisfied, the circadian waking drive increases in intensity and the cycle begins anew (27).

Sleep theory. The specific function(s) of sleep remain elusive to science (24). Studies of sleep and sleep deprivation have led to the development of three broad categories of contemporary sleep axioms: energy conservation, information processing and synaptic plasticity, and restoration of key cellular components (30).

Hypotheses which posit that sleep functions to conserve energy are rooted in the construct of natural selection (20). According to these models, animal activity and prey availability peak at specific times of the day (30). Subsequently, sleep increases the likelihood of survival by reducing energy expenditure during periods of torpor. While the energy economics theory is substantiated by numerous examples in nature, it fails to explain the selection of REM sleep, which, in most species, results in a state of increased energy expenditure (30).

Evidence suggests that sleep facilitates learning and memory through changes in brain plasticity (the capacity of the brain to change with learning) and synaptogenesis (creation of new synapses) (31). As wakefulness is associated with learning and the strengthening of synapses, Tononi and Cirelli(27)postulated that sleep regulates synaptic homeostasis. According to this theory, space and energy limitations make learning unsustainable in a state of perpetual wakefulness (27). Sleep provides a condition of external disconnect from the environment allowing for synaptic downsizing (27). This process leaves only the most robust connections intact, providing the energy and space for renewed learning (30). Tononi and Cirelli's hypothesis predicts that synaptic downsizing increases the strength of the remaining connections, thereby improving learning performance(27). Although their hypothesis accounts for numerous experimental observations, it does not take into account the role of REM sleep in learning (30).

Restoration hypotheses propose that key cellular components of macromolecule biosynthesis are expended during periods of extended wakefulness (24). Subsequently, cellular stress is amplified, and sleep is required to restore the synthesis of depleted macromolecules(24). The greatest strength of this theory is its cellular basis, which makes it applicable to all organisms and tissues (30). However, the reported changes in gene expressions are correlational and may be the result of biological processes distinct from sleep (30). In addition, similar to other sleep theories, the restoration hypothesis primarily explains NON-REM sleep and does not fully elucidate the function of REM sleep (30).

Sleep across the lifespan. Sleep requirements and architecture evolve over time, with sleep generally

becoming less efficacious with age (20). Newborns and infants require approximately 16 to 18 hours of sleep per day and spend a considerable amount of time in REM sleep (32). Infants gradually sleep more at night as circadian rhythms become entrained and responsiveness to social cues increase(33). Sleep duration decreases by 2 to 5 hours in childhood (32), possibly due to gradual decreases in daytime napping and the introduction of school routines (34). Adolescents require 9 to 10 hours of sleep, though research has found that this duration is rarely met (35). Adulthood is associated with earlier sleep and wake times (36). Elderly adults tend to experience a decrease in sleep quality (37). Although the causes for this are multifaceted, the elderly are known to experience a decrease in melatonin levels due to aging of the hypothalamic nuclei, the organ responsible for modulating circadian rhythms(38).

Sleep and health. Sleeping 7 to 8 hours on a daily basis has long been identified as a critical element of optimum health (1). Notwithstanding, research indicates that sleep deprivation is highly prevalent in society. Subsequently, the deleterious outcomes of this apparent trend have become a topic of growing concern within the scientific community (2). Epidemiological evidence has associated both insufficient (8 hours) sleep durations with increased rates of morbidity(39, 40)and mortality (3). The majority of studies report U-shaped, parabolic relationships, which indicate that sleep durations that fall outside of the 7 to 8 hour range adversely impact health (2). Injurious health consequences linked to long and short sleep duration include cardiovascular disease (41), diabetes (39), depression (42), automobile and occupational accidents (43, 44), as well as learning and memory problems(45). These findings have led many researchers to conclude that habitual sleep duration of 7 to 8 hours is essential for health maintenance (2, 46).

The causal factors explicating the association between inadequate sleep duration and an increased risk of all-cause mortality remain unclear (3, 47). As sleep is a modulator of hormone release, cardiovascular function, and glucose regulation, Bixler(2)postulated that short sleep duration contributes to an increased risk of cardiometabolic disorders including hypertension, as well as metabolic syndrome, and diabetes mellitus. Conversely, Youngstedt and Kripke(48)have proposed that mortality rates associated with excessive sleep duration are a consequence of sleep fragmentation, lethargy, and a decreased photoperiod. Furthermore, evidence indicates a bi-directional relationship between sleep and health such that inadequate sleep contributes to the progression of a number of medical and psychiatric disorders, and that these same disorders, in turn, contribute to poor sleep quality (49).

Sleep restriction. Partial sleep deprivation (PSD) is described as a nocturnal sleep state that is interrupted or reduced (50). Banks and Dinges(46)suggest that PSD can manifest in three ways. The first type prevents sleep from being physiologically consolidated, resulting in sleep fragmentation. Sleep fragmentation disrupts the normal sleep stage sequencing pattern, resulting in decreased sleep efficiency; measured by time spent in physiological sleep compared to the amount of time spent in bed (46). This type of PSD is commonly seen in certain sleep disorders such as obstructive sleep apnea (51). The second type of PSD is classified as selective sleep deprivation. This form of PSD is marked by sleep fragmentation, which results in a stage-specific deficiency. Banks and Dinges(46)classify the third category as sleep restriction and define this form of PSD as a reduction in total sleep duration. Sleep restriction is attributed to medical conditions, sleep disorders, work schedules, social and domestic responsibilities, and lifestyle choices (46, 50). Sleep debt is considered to be a consequence of cumulative sleep restriction (52).

Sleep restriction in society.Sleep loss is widespread in industrial societies (53). Several studies have noted a downward trend in the amount of sleep Americans receive. In 1960, the American Cancer Society reported modal sleep duration of 8.0–8.9 hours of sleep; by 1995 the modal category of the survey indicated that sleep duration had decreased to 7 hours(53). A study examining population-based data (n=110,441) collected between 2004 and 2007 revealed that 28.3% of U.S. adults slept six or fewer hours per night and 8.5% slept nine or more hours (54).

The inclusion of sleep health in the United States *Healthy People 2020* objectives emphasizes the magnitude of this pertinent health issue. In 2009, the Centers for Disease Control and Prevention incorporated a sleep module into the Behavioral Risk Factor Surveillance System. Analysis of the data found 35.3% of respondents (n=75,571) reported having less than 7 hours of sleep on average during a 24 hour period, 37.9% reported unintentionally falling asleep during the day at least one day in the preceding 30 days, and 4.7% reported falling asleep while driving in the preceding 30 days (55). The Healthy People report clarified the scope of the national agenda by setting an objective to increase the percentage of adults who receive sufficient sleep from

the 2008 baseline level of 69.6% to 70.9% by 2020 (4).

Short sleep duration and reduced sleep quality is not unique to American culture. A Finnish study conducted by Kronholm and colleagues (56) between 1972 and 2005 discovered an 18 minute decrease in sleep duration among the population (*n*=251,083) in addition to an increase in sleep complaints, primarily among middle-aged men. Liu et al. (57) investigated sleep loss and daytime sleepiness in the general adult population of Japan and found that 29% of those surveyed (*n*=3,030) slept less than 6 hours per night with 23% reporting insufficient sleep. In accordance with these findings, the researchers reported prevalence rates of insomnia at 21% and excessive daytime sleepiness at 15%.

Although the evidence indicates a trend towards diminishing sleep duration and quality, there are limitations with the methods used to arrive at these findings. Magee, Iverson, Huang, and Caputi(58) noted that the majority of data were collected through self-report. Several researchers have delineated the limitations of this approach and contend that objective measures of recording sleep are needed to establish the validity of epidemiological results, particularly in terms of morbidity and mortality factors as related to sleep duration (47). Furthermore, some researchers remain unconvinced that the reduction hypothesis is viable, citing a lack of comparison data from the pre-industrial era (56).

Etiology of sleep restriction.Epidemiological data has uncovered several antecedents of inadequate sleep duration includingsocioeconomic status (59), stress (56),sleep complaints(60), age-related factors (61), being single (54),working long hours (62), as well asunhealthy behaviors such as smoking, alcohol consumption, and sedentary lifestyles (2). Conversely, data has identified supportive marriages (63) and parenting as determinants that encourage healthy sleeping patterns (64).

Behavioral dynamics and health-related factors are at the core of curtailed sleep durations(65, 66). Behavioral components are interlinked to evolving societal norms and encompass lifestyle factors such as shift work, prolonged working hours, jet lag, and maintaining irregular sleep schedules(67). Consequently, sleep researchers have cited voluntary sleep restriction as a primary factor responsible for insufficient sleep in modern society(49).

Sleep disorders represent a biological or psychological inability to achieve healthy sleep(68). There are approximately 90 classified sleep disorders with the greater part being characterized by one of three symptoms: excessive daytime sleepiness, difficulty in initiating or maintaining sleep, and irregular events occurring during sleep(69). In 2003, it was reported that between 50 and 70 million Americans suffered from chronic sleep and wakefulness disorders(70). The most common sleep disorders included insomnia, narcolepsy, obstructive sleep apnea syndrome, restless leg syndrome, and shift wake-sleep disorder (68).

Neurobehavioral consequences of sleep restriction.Sleep restriction degrades behavioral alertness and cognitive performance. Moreover, these deteriorating effects are dose-dependent, resulting in exacerbated conditions of cognitive dysfunction(46, 71). To cope with sleep restriction, the body alters its normal sleep architecture by intensifying particular sleep stages while simultaneously diminishing others. Studies have uncovered that healthy adults restricted to 4 hours of sleep for multiple nights spend less time in NON-REM stage two and REM sleep, yet maintain the same amount of NON-REM slow-wave sleep relative to a typical 8-hour sleep episode (71). Although the research demonstrates that the body conserves slow-wave sleep during periods of sustained sleep restriction, cognitive deficits continue to escalate, suggesting that the brain is unable to adapt to sleep deprivation without sacrificing neurocognitive performance (46).

Psychomotor vigilance. From a neurological perspective, alertness refers to a sustained state of attention required for accurate performance of cognitive tasks (72). Alertness impacts attention processes and is highly impacted by sleep deprivation(73). Sleep deprivation has been shown to disrupt communication between the multiple brain regions responsible for managing attention (74). Studies have shown that to maintain arousal in a sleep-deprived state, the brain is forced to focus its cognitive resources on warding off the homeostatic drive for sleep, leaving a capacity deficit for tasks requiring alertness (72).

Psychomotor vigilance is particularly sensitive to sleep restriction. Research has demonstrated that sleep deprivation increases delays in alertness (75). In an experiment conducted by Van Dongen and colleagues (52), cognitive performance impairments of participants that slept 6 hours or less per night over 14 consecutive days were equivalent to cognitive performance impairments of participants subjected to two nights of total sleep deprivation. Surprisingly, the sleep deprived study participants appeared unaware of their fatigued state, as indicated by their scores on

standardized sleepiness scales.

Transportation safety issues exemplify the reality of sleep restriction's impact on psychomotor vigilance (76). Numerous investigations have implicated sleep loss as a primary cause of automobile accidents (76). Sleep-related collisions have been estimated to have an annual economic cost of $43 to $65 billion (77). Stratified by age and gender, research indicates males under 30 years are the mostly likely demographic to be involved in sleep-related crashes(78). Interestingly, sleep-related motor vehicle accidents correspond to circadian variations in sleepiness, primarily occurring between the hours of 2:00 a.m. to 8:00 a.m. and mid-afternoon(79). Multiple jobs, shift work, and sleep restriction are the salient factors contributing to sleep-related accidents (78). Sleep deprivation has also been shown to impair psychomotor performance in a manner similar to intoxication at or above the legal alcohol limit(80).

Mental health. Researchers have found that sleep deprivation generates feelings of tension, anger, and uneasiness while concomitantly diminishing drive and motivation(81). In a pioneering study, Yoo, Gujar, Hu, Jolesz, and Walker (82) demonstrated that insufficient sleep duration degrades distressful emotional coping capabilities. In their experiment, both sleep-deprived and non-sleep-deprived groups were exposed to negative visual stimuli. Incorporating brain scans, the researchers observed that in the sleep-deprived group, the amygdala—the region of the brain hypothesized to stimulate emotional responses—exhibited a 60% greater activation rate compared to the normal sleep group. In the non-sleep-deprived group, the prefrontal cortex—the logic center of the brain believed to be responsible for modulating emotional reactions—dampened the reaction of the amygdala. The variation in brain activity between the two groups suggested there was a lack of association between the prefrontal cortex and the amygdala in the sleep-deprived group, explaining the more intense emotional response. The researchers demonstrated that in a state of sleep deprivation, emotional coping is curtailed and reaction to aversive stimuli is insufficiently regulated by the brain.

*Memory and learning. S*leep restriction has been shown to obstruct higher cognitive functions including attention, memory, abstract thinking, creativity, and problem solving(83). Human memory is categorized into short-term memory, also called working memory, and three systems of long-term memory (LTM) (84). LTM is divided into two branches: declarative and non-declarative (85). Declarative memory refers to consciously accessible memories, and is classified as either episodic or semantic (86). Episodic memories include autobiographical recollections of one's life such as birthdays or other memorable events(87). Semantic memories include facts, information, and general knowledge that had been acquired, devoid of the temporal context in which the information was obtained (87). Non-declarative memories are acquired and recalled without conscious thought, and are related to procedural tasks such as performing a skill or problem solving (85). Memory classifications provide a convenient methodology for experimentation; however, memory systems rarely operate in isolation. For example, higher learning requires the conjoint effort of memory types (85).

The introduction of the declarative/non-declarative dichotomy gave rise to a renewed interest in the relationship between sleep and synaptic plasticity (86). Research has strengthened the relationship between sleep and the memory-learning processes, yet the extent of sleep's role in this process is the subject of much debate (88). Memory formations are thought to occur in distinct stages over time, beginning with memory encoding(85). Encoding includes the processing of a physical sensory input into the memory. Researchers hypothesize that once a memory is encoded it undergoes memory consolidation, a process suspected of making a labile memory increasingly stable(85). Sleep is considered a mediator of memory consolidation, yet it remains to be determined which aspects of memory are affected by sleep(53). Nevertheless, researchers have concluded that both REM and NON-REM are necessary for learning and that sleep loss impairs the consolidation of declarative and procedural memories (53).

Physiological consequences of sleep restriction. Epidemiological evidence has associated short (8 hours) sleep durations with an increased risk of all-cause mortality (2). In addition, numerous physiological indices are altered by sleep restriction including a reduction in T cells (89), a reduction in glucose(90), a reduction in leptin levels (91), an increase in blood pressure (92), and elevated levels of C-reactive protein (93). Researchers speculate that these short-term alterations serve as a catalyst for the negative health implications revealed in the epidemiological research (46).

Cardiovascular morbidity has been associated with sleep restriction in numerous epidemiological studies (46). The causal sequence linking chronic sleep restriction with increased risk of cardiovascular disease has yet to be identified. Researchers have speculated that elevated levels of C-reactive protein,

which rise in response to inflammation in the body and have been demonstrated to increase during sleep restriction, contribute to the cardiovascular morbidity rates reported in epidemiological investigations(46, 93).

Chronic sleep restriction has been shown to negatively affect endocrine function and carbohydrate metabolism, leading many researchers to speculate a causal relationship between sleep restriction and obesity (91). Several studies have examined the parallel between the current obesity epidemic and the growing societal trend of chronic sleep curtailment(58, 94). Potential causal pathways connecting insufficient sleep and obesity include impaired glucose metabolism, an increase in caloric intake caused by hyperphagia, feelings of fatigue contributing to lower physical activity levels, and a decline in core body temperature affecting energy expenditure via thermoregulation(95, 96). Obesity is also a primary risk factor for obstructive sleep apnea, a type of sleep-disordered breathing linked to hypertension. Despite the heightened media attention regarding the possible link between sleep and obesity, Marshall et al. (94)has cautioned that the evidence that sleep duration is causally linked to obesity remains inconclusive and currently advises against the promotion of sleep behavior modification as an intervention for obesity.

Sleep disorders.There are approximately 90 classified sleep disorders identified by the American Sleep DisordersAssociation's *International Classification of Sleep Disorders*(97). The American Psychiatric Association's (APA) *Diagnostic and Statistical Manual of Mental Disorders*(98) organizes sleep disorders into four comprehensive sections.

*Primary sleep disorders.*Primary sleep disorders are speculated todevelop from abnormalities in the physiological functions that regulate sleep (98). This category is divided into two general types: dyssomnias and parasomnias. Dyssomniasare characterized by an inability to fall asleep or stay asleep. This spectrum of disorders includes circadian rhythm disorder, narcolepsy, and breathing-related disorders such as obstructive sleep apnea syndrome, primary insomnia, and primary hypersomnia. Parasomnias are the second type of primary sleep disorders. Parasomnias disrupt the sleep-wake cycle by activating physiological systems at inappropriate times. Sleepwalking, nightmare, and sleep terror disorders are examples of parasomnias.

Circadian rhythm somnipathies encompass the family of sleep disorders affecting the timing of sleep (98). This category includes a number of disorder types relevant to behavioral components of sleep restriction including delayed sleep phase, jet lag, and shift work. Of particular interest to this research report is the delayed sleep phase type characterized by a delay of the sleep-wake cycle relative to societal demands. Essentially, this disorder represents a circadian mismatch. Individuals with this condition initiate sleep, maintain sleep, and awaken consistently when left to their own sleep schedule; yet, they exhibit difficulty modifying their sleep pattern to coincide with traditionally-timed social and occupational obligations. As a result, these individuals are frequently chronically sleep deprived. Delayed sleep phase type is distinguished from volitional patterns of delayed sleep hours by the ability of the individual to fall asleep at earlier times after a period of recovery sleep.

Sleep disorders related to another mental disorder. Sleep disturbances are frequent features of psychiatric disorders (98). The primary characteristic of this category is the presence of insomnia or hypersomnia, temporally or causally, related to another mental disorder. Hypersomnia is diagnosed as excessive nighttime sleep or repeated daytime sleep episodes lasting for at least one month. Alternatively, insomnia is categorized as the inability to initiate sleep, maintain sleep, or receive restorative sleep. Insomnia is associated with a mental disorder in more than one-third of cases (99), with the most common disorder being depression(100).

Insomnia is the most widely reported sleep problem, afflicting an estimated 10% of the general adult population in the United States (100). To treat insomnia, clinicians rely upon a variety of treatment modalities including pharmacotherapy, psychological therapy, light therapy, and exercise (101). Pharmacotherapy is the most frequent method used for treating acute insomnia. This approach incorporates the prescription of sedative-hypnotics to induce or maintain sleep. Psychological therapy, primarily in the form of cognitive behavioral therapy, is considered the ideal modality for treating chronic insomnia (102). Cognitive behavioral therapy consists of a range of psychotherapeutic methods including stimulus control treatment, sleep restriction therapy, cognitive control, thought suppression, imagery and relaxation, cognitive restricting, and paradoxical intention (101). Light therapy and exercise are based on the physiology of human circadian rhythms. Internal biorhythms are particularly sensitive to environmental cues, and selective light exposure is presumed to aid in resetting circadian rhythms (103). Physically active individuals have been found to have higher sleep quality as well (47). An explanation for this

phenomenon has not been resolved, but it is often attributed to behavioral patterning as opposed to aerobic fitness (101).

Sleep disorders due to a general medical condition are typically assumed to be a direct result of a general medical condition affecting physiological functioning (98). *Substance-induced sleep disorder*s relate to sleep disturbances resulting from the use of a substance, including medications (98).

Sleep hygiene and alertness management. Sleep hygiene and alertness management are effective methodologies for improving sleep quality (104) and enhancing alertness (67). As stand-alones, these techniques are not used for the treatment of sleep disorders (101), but are considered beneficial to the general population to assist in optimizing sleep and coping with the unwanted effects of unavoidable fatigue.

Sleep hygiene. Sleep hygiene operates under the assumption that behavioral and environmental factors can be modified to enhance sleep quality (104). Sleep hygieneconsists of a series of practices designed to optimize the biological processes regulating human sleep. From the time of its conception in 1977 by Peter Hauri, the opinions of what constitute ideal sleep hygienehave continued to evolve (104). Hauri(105)has suggested that many of the sleep hygieneprinciples are relative to each individual and should be subjected to trial-and-error and analyzed over time through the maintenance of a sleep log. Hauri has noted that in practice most patients are only capable of adopting three or four sleep hygieneprinciples at a time as the implementation of the rules often requires significant behavioral modification.

Habitual sleep/wake time. Adhering to a consistent wake/sleep time promotes optimal sleep propensity and consolidation (104). Hauri(105) has advised establishing a regular wake time and incorporating a sleep log to determine optimal sleep duration. It is commonly accepted that the circadian and homeostatic processes will eventually synchronize to the fixed wake time, and sleep time would regulate itself automatically. Hauri has asserted that the innate circadian rhythm is longer than 24 hours in adolescents and young adults. Maintaining a regular arousal time is alleged to aid in consolidating this extended duration into a 24-hour time frame.

Napping. Daytime naps have been shown to have an adverse effect on nocturnal sleep quality by diminishing the homeostatic pressure for sleep (104). Notwithstanding, short naps of 15 minutes can improve performance, while longer late-afternoon naps have the potential to disrupt nocturnal sleep quality (106). Hauri(105) has recommended that naps be explored as an option and, if found to be beneficial, tailored according to individual need.

*Caffeine.*Caffeine is the most popular psychostimulant for thwarting sleepiness (107). The neuromodulator adenosine is believed to be an endogenous sleep-promoting substance that increases in tandem with the sleep homeostat(108). Caffeine is believed to block adenosine receptors in the central nervous system thereby staving off the homeostatic drive for sleep. Caffeine has a half-life of 6 hours;therefore, it is recommended to not consume caffeine within 4 to 6 hours of scheduled sleep time. Restricting caffeine consumption prior to sleep time is one of the few consistent recommendations proposed by sleep hygieneexperts (104).

*Alcohol and tobacco.*Alcohol has several inimical effects on sleep hygiene including suppression of REM sleep, sleep fragmentation, and delay of sleep latency (104). Low doses of alcohol can assist in promoting sleep onset; however, alcohol consumed up to 6 hours prior to sleep time has been empirically verified to result in sleep fragmentation (109). Consequently, it is generally recommended that alcohol be avoided prior to sleep and not be employed as a hypnotic (105). Similar to caffeine, alcohol avoidance is one of the few SH practices that are recommended across the research (104). As well, nicotine is considered a mild stimulant and thus should be avoided prior to sleep (105). However, the full effect of nicotine's interaction on sleep remains unclear (104).

*Body temperature and passive heating.*The circadian rhythm is assessed by measuring core temperature. Hauri(105) has contended that sleep quality is tied to a natural trough in the daily temperature curve. Intense exercise is known to cause an increase in core temperature resulting in a compensatory decrease in core temperature 4 to 6 hours post-workout (Horne & Staff, 1983). The subsequent decrease in core temperature leads to a cooling effect that is believed to assist in promoting sleep. Exercise performed later in the day is also thought to increase sleep depth (47). While biologically plausible, the evidence is mixed on the sleep-promoting efficacy of exercise (110). Passive heating, such as remaining in a hot bath for 30 minutes, has been advocated as a mechanism to prompt core temperature reaction (111). Liao (112) reported that passive heating increased the depth of sleep in elderly individuals with insomnia. The cooling aftereffect was observed peaking approximately 2 to 4 hours post-passive heating, and could be incorporated

as an alternative to intense physical activity(105).

Environment. In addition to the homeostatic and circadian processes, the autonomic nervous system assists in regulating sleep. Sleep onset requires a decrease in sympathetic activity and an increase in parasympathetic equilibrium (11). Stimuli which amplify sympathetic activity disrupt sleep, irrespective of whether the origin of the stimuli is endogenous or exogenous (11). This premise explains why caffeine consumption (exogenous) or anxiety-focused rumination (endogenous) promotes wakefulness when entertained near sleep onset (11). Researchers speculate that this mechanism exists for survival purposes, such as when hazardous conditions require the body to maintain wakefulness. However, this function is also suspected to hinder sleep and promote insomnia. Environmental sleep hygienepractices are believed to promote nocturnal tranquility in a manner similar to classical conditioning (11).

A number of environmental factors have been proposed to foster SH such as eliminating light and noise from the bedroom, regulating bedroom temperature, eliminating light-emitting bedroom clocks, ensuring the bed is comfortable and suitable for sleep, and using the bedroom exclusively for sleep(63, 105). Nightly rituals are believed to serve as triggers to prepare the mind for sleep (63). Mild routines have been recommended and are encouraged to be used in conjunction with other SH practices to reinforce sleep latency. The creation of a “worry list” can cultivate an environment conducive to sleep by assisting in allaying mental stress (63). Integrating this technique requires the individual to develop a list of tasks and goals that have to be completed in the near future and mentally detaching from the list to clear the mind of anxiety-producing thoughts.

Inadequate sleep hygiene. The AASM(97) has established a set of criteria for diagnosing inadequate sleep hygiene. Inadequate sleep hygieneis characterized by practices that produce increased arousal and practices that contradict the known principles of sleep organization. The central tenet of inadequate sleep hygieneis that the practices are under the behavioral control of the individual and are not the result of an underlying physiological or psychological disorder.

Studies show that arousal can be augmented by substances such as caffeine, tobacco, and alcohol (97). Stress and excitement also contribute and include stimulating activities occurring within close proximity to sleep time such as intense physical activity, involved mental tasks, or social events. Environmental arousers—for example, allowing light to seep into the bedroom—are also labeled as sleep disruptions. The biological processes that regulate sleep are also susceptible to inadequate sleep hygiene. Spending too much time in bed, excessive variation in sleep/wake time, and taking naps throughout the day are considered indicators of poor sleep hygiene.

Alertness management.The context of modern society has led many researchers to conclude that stints of voluntary sleep restriction are inescapable. Alertness management incorporates a blend of SH techniques and fatigue management strategies to mitigate the effects of unavoidable sleep loss (67). Fatigue is an enervator of several psychological functions including accuracy and focus, multi-tasking, social interaction, emotional stability, and rational thought.

*Optimizing sleep.*In general, this tactic coincides with sleep hygienepractices with an additional recommendation to use hypnotics to cope with situations that make sleep onset difficult such as shift lag, poor sleep environments, or unconventional schedules (67). A variety of hypnotics are available, and consideration of the ideal soporific is dependent upon a number of factors. Temazepam has a half-life of 8 to 10 hours and is considered useful for increasing sleep duration. Extended-release zolpidem improves sleep maintenance without the extended half-life of temazepam. Zolpidem or zaleplon are ideal for short-sleep periods and have a lower post-sleep sedation side effect.

Dietary supplementsmay alsooffer potential for optimizing sleep. One such supplement is melatonin; a popular, over-the-counter, sleep aid. Melatonin is a hormone produced at night by the pineal gland and is responsible for regulating the sleep-wake cycle. As a dietary supplement, melatonin isconsumedto adjust the body’s internal clock, primarily due to jet lag or shift-work disorder. It is also used to promote sleep onset in cases of insomnia. It should be noted that little scientific research exists to substantiate the efficacy of non-hypnotic sleep aids such as melatonin (113). Thus, caution is warranted in promoting their use until more research is conducted.

*Fatigue countermeasures.*Caldwell and colleagues (67)recommend five strategies for temporarily preserving performance during a state of fatigue. The authors recommend that these strategies be utilized intermittently as needed and that regular sleep be resumed as soon as conditions permit.

Limiting time on task and rest breaks. Restraining the total duration of time available to work promotes

healthier sleep habits and offsets the effects of time-on-task fatigue (114). Rest periods taken during a work shift assist in counteracting the negative neurocognitive side effects of sleep loss. However, the benefits of a rest period during a sleep-deprived state are minimal for long-term performance (115).

*Napping and posture.*A nap taken during periods of prolonged wakefulness is the most effective non-pharmaceutical method for bolstering alertness. Bonnet (116) found that a nap taken prior to a 52-hour block of continuous work sustained performance for 24 hours when compared to a non-nap group; however, the benefits dissipated by the second night of sleep loss. Nap timing and length are important aspects to consider. Nap duration is directly proportional to performance benefit (67). Daytime naps taken in a non-sleep-deprived state have been recommended for improving night-work performance. For planning a nap, experts contend that an important consideration is the timing of the nap in relation to the circadian phase. The circadian rhythm regulates core body temperature. Sleep propensity is the greatest when the core body temperature troughs and lowest when the core body temperature peaks. Generally, core temperature peaks in the early evening and troughs during the pre-dawn and early morning hours. Nap inducement can be difficult to initiate during temperature peaks, yet naps during this phase tend to produce the least amount of post-sleep lethargy. Naps initiated during the circadian trough offer the best performance benefits, but are accompanied by a higher likelihood of sleep inertia (117).

Certain postures aid in inhibiting fatigue. Upright positions reduce sleepiness compared to prone positions (118). During periods when fatigue is high, working in a standing position can preserve alertness (67).

*Psychoactive stimulants.*Caffeine is a short-acting stimulant clinically demonstrated to reduce performance deficits associated with sleep loss (67). Controlled experiments have found that caffeine improves performance at doses of 75 to 100 mg following acute restriction of sleep and at doses of 200 to 600 mg after a night of total sleep loss (116). However, frequent use of caffeine results in tolerance and produces withdrawal side-effects(116).

Discussion

The twentieth century witnessed monumental leaps in the scientific understanding of human sleep(18). Sleep research was developed into a testable science with the discovery of electroencephalography and later refined with the introduction of polysomnography(20). These advancements provided a deeper understanding of sleep physiology and the impact of sleep on human health. Although a putative theory of sleep currently eludes scientists, the interaction between the homeostatic mechanism and circadian rhythm in the modulation of sleep and wakefulness has been successfully described by the two-process model of sleep regulation(27).

Sleeping 7 to 8 hours on a daily basis has long been identified as a critical element of optimum health(1). Despite this, a mounting body of evidence suggests that sleep restriction is on the rise in the society. Epidemiological research has uncovered the health ramifications of this trend, associating both insufficient and excessive sleep durations with increased rates of morbidity and mortality(2). Sleep restriction has also been shown to have neurobehavioral consequences. Salient symptoms of sleep deprivation include impairment of neurocognitive and psychomotor performance, emotional imbalance, and overall lower life satisfaction. Researchers speculate that short-term physiological alterations due to sleep restriction could serve as a catalyst for the long-term negative health implications revealed in the epidemiological research.

Sleep is one of the core components of optimal health, yet has received minimal attention in the health promotion and education literature. Sleep hygiene and alertness management can improve behavioral and environmental conditions responsible for inducing the physiological and environmental conditions necessary for quality sleep (67, 104). Sleep hygiene and alertness management principles should be promoted to the general public through public health interventions.

References

1. Belloc NB, Breslow L. Relationship of physical health status and health practices. Prev Med. 1972;1(3):409-21.
2. Bixler E. Sleep and society: an epidemiological perspective. Sleep Med. 2009;10:S3-S6.
3. Hublin C, Partinen M, Koskenvuo M, Kaprio J. Sleep and mortality: a population-based 22-year follow-up study. Sleep. 2007;30(10):1245.
4. U.S. Department of Health and Human Services.Healthy People 2020: the road ahead. 2010. Available from: healthypeople.gov/2020/

5. Hillman DR, Murphy AS, Antic R, Pezzullo L. The economic cost of sleep disorders. Sleep. 2006;29(3):299.
6. Ferrie JE, Kumari M, Salo P, Singh-Manoux A, Kivimäki M. Sleep epidemiology—a rapidly growing field. Int J Epidemiol. 2011;40(6):1431-7.
7. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta?analysis. Obesity. 2008;16(2):265-74.
8. Caton R. The electric currents of the brain. Br Med J. 1875;2:278.
9. Berger H. Über das Elektroenkephalogramm des Menschen. Achte Mitteilung. Arch Psychiatr Nervenkr. 1933;87:452-69.
10. Tudor M, Tudor L, Tudor K. Hans Berger (1873-1941)--the history of electroencephalography. Acta medica Croatica. 2005;59(4):307.
11. Hirshkowitz M. Normal human sleep: an overview. Med Clin North Am. 2004;88(3).
12. Loomis AL, Harvey EN, Hobart G. Cerebral states during sleep, as studied by human brain potentials. J Exp Psychol. 1937;21(2):127.
13. Aserinsky E, Kleitman N. Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. Science. 1953;118(3062):273-4.
14. Jouvet M, Michel F, Courjon J. Sur un stade d'activité électrique cérébrale rapide au cours du sommeil physiologique. CR Soc Biol(Paris). 1959;153:1024-8.
15. Deak M, Epstein LJ. The history of polysomnography. Sleep Med Clin. 2009;4(3):313-21.
16. Hori T, Sugita Y, Koga E, Shirakawa S, Inoue K, Uchida S, et al. Proposed supplements and amendments to 'a manual of standardized terminology, techniques and scoring system for sleep stages of human subjects', the Rechtschaffen & Kales (1968) standard. Psychiatry Clin Neurosci. 2001;55(3):305-10.
17. Rechtschaffen A, Kales A. A manual of standardized, techniques and scoring system for sleep stages in human subjects. Washington DC, US Government Printing Office, 1968.NIH Publication No. 204.
18. Iber C, Ancoli-Israel S, Chesson A: Quan SF for the American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. 1st ed. Westchester, Ill, American Academy of Sleep Medicine, 2007.
19. Kilduff TS, Lein ES, de la Iglesia H, Sakurai T, Fu Y-h, Shaw P. New developments in sleep research: molecular genetics, gene expression, and systems neurobiology. Journal Neurosci. 2008;28(46):11814-8.
20. Markov D, Goldman M. Normal sleep and circadian rhythms: neurobiologic mechanisms underlying sleep and wakefulness. PsychiatrClin North Am. 2006;29(4):841-53.
21. Sinton CM, McCarley RW, editors. Neurophysiological mechanisms of sleep and wakefulness: a question of balance. Semin Neurol; 2004; 24(3):211-13.
22. Fuller PM, Gooley JJ, Saper CB. Neurobiology of the sleep-wake cycle: sleep architecture, circadian regulation, and regulatory feedback. J Biol Rhythms. 2006;21(6):482-93.
23. Attarian H, Schenck C, Mahowald M. Presumed REM sleep behavior disorder arising from cataplexy and wakeful dreaming. Sleep Med. 2000;1(2):131-3.
24. Mackiewicz M, Shockley KR, Romer MA, Galante RJ, Zimmerman JE, Naidoo N, et al. Macromolecule biosynthesis: a key function of sleep. Physiol Genomics. 2007;31(3):441-57.
25. Benington JH. Sleep homeostasis and the function of sleep. Sleep. 2000;23(7):959.
26. Reid KJ, Burgess HJ. Circadian rhythm sleep disorders. Prim Care. 2005;32(2):449.
27. Tononi G, Cirelli C. Sleep function and synaptic homeostasis. Sleep Med Rev. 2006;10(1):49-62.
28. Borbély AA. A two process model of sleep regulation. Hum Neurobiol. 1982.
29. Borb AA, Achermann P. Sleep homeostasis and models of sleep regulation. J Biol Rhythms. 1999;14(6):559-70.
30. Mignot E. Why we sleep: the temporal organization of recovery. PLoS Biol. 2008;6(4):e106.
31. Vyazovskiy VV, Cirelli C, Pfister-Genskow M, Faraguna U, Tononi G. Molecular and electrophysiological evidence for net synaptic potentiation in wake and depression in sleep. Nat Neurosci. 2008;11(2):200-8.
32. Roffwarg HP, Muzio JN, Dement WC. Ontogenetic development of the human sleep-dream cycle. Science. 1966;152(3722):604-19.
33. Mirmiran M, Maas YG, Ariagno RL. Development of fetal and neonatal sleep and circadian rhythms. Sleep Med Rev. 2003;7(4):321-34.
34. Jenni OG, O'Connor BB. Children's sleep: an interplay between culture and biology. Pediatrics. 2005;115(Supplement 1):204-16.
35. Mercer PW, Merritt SL, Cowell JM. Differences in reported sleep need among adolescents. J Adolesc Health. 1998;23(5):259-63.
36. Dijk D-J, Duffy JF, Czeisler CA. Contribution of circadian physiology and sleep homeostasis to age-related changes in human sleep. Chronobiol Int. 2000;17(3):285-311.
37. Van Cauter E, Leproult R, Plat L. Age-related changes in slow wave sleep and REM sleep and relationship with growth hormone and cortisol levels in

healthy men. JAMA. 2000;284(7):861-8.
38. Garfinkel D, Laudon M, Nof D, Zisapel N. Improvement of sleep quality in elderly people by controlled-release melatonin. Lancet. 1995;346(8974):541-3.
39. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Sleep duration as a risk factor for diabetes incidence in a large US sample. Sleep. 2007;30(12):1667.
40. Hall MH, Muldoon MF, Jennings JR, Buysse DJ, Flory JD, Manuck SB. Self-reported sleep duration is associated with the metabolic syndrome in midlife adults. Sleep. 2008;31(5):635.
41. Ayas NT, White DP, Manson JE, Stampfer MJ, Speizer FE, Malhotra A, et al. A prospective study of sleep duration and coronary heart disease in women. Arch Intern Med. 2003;163(2):205.
42. Strine TW, Chapman DP. Associations of frequent sleep insufficiency with health-related quality of life and health behaviors. Sleep Med. 2005;6(1):23-7.
43. Teran-Santos J, Jimenez-Gomez A, Cordero-Guevara J. The association between sleep apnea and the risk of traffic accidents. N Engl J Med. 1999;340(11):847-51.
44. Lockley SW, Cronin JW, Evans EE, Cade BE, Lee CJ, Landrigan CP, et al. Effect of reducing interns' weekly work hours on sleep and attentional failures. N Engl J Med. 2004;351(18):1829-37.
45. Stickgold R, Hobson JA, Fosse R, Fosse M. Sleep, learning, and dreams: off-line memory reprocessing. Science. 2001;294(5544):1052-7.
46. Banks S, Dinges DF. Behavioral and physiological consequences of sleep restriction. J Clin Sleep Med. 2007;3(5):519.
47. Youngstedt SD. Effects of exercise on sleep. Clin Sports Med. 2005;24(2).
48. Youngstedt SD, Kripke DF. Long sleep and mortality: rationale for sleep restriction. Sleep Med Rev. 2004;8(3):159.
49. Zee PC, Turek FW. Sleep and health: everywhere and in both directions. Arch Intern Med. 2006;166(16):1686.
50. Weinger MB, Ancoli-Israel S. Sleep deprivation and clinical performance. JAMA. 2002;287(8):955-7.
51. Bennett LS, Langford BA, Stradling JR, Davies RJ. Sleep fragmentation indices as predictors of daytime sleepiness and nCPAP response in obstructive sleep apnea. Am J Respir Crit Care Med. 1998;158(3):778-86.
52. Van Dongen H, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. Sleep. 2003;26(2):117-26.
53. Curcio G, Ferrara M, De Gennaro L. Sleep loss, learning capacity and academic performance. Sleep Med Rev. 2006;10(5):323-38.
54. Krueger PM, Friedman EM. Sleep duration in the United States: a cross-sectional population-based study. Am J Epidemiol. 2009;169(9):1052-63.
55. McKnight-Eily L, Liu Y, Wheaton A, Croft J, Perry G, Okoro C, et al. Unhealthy sleep-related behaviors---12 States, 2009. MMWR Morb Mortal Wkly Rep. 2011;60:233-8.
56. Kronholm E, Partonen T, Laatikainen T, Peltonen M, Härmä M, Hublin C, et al. Trends in self?reported sleep duration and insomnia?related symptoms in Finland from 1972 to 2005: a comparative review and re?analysis of Finnish population samples. J Sleep Res. 2008;17(1):54-62.
57. Liu X, Uchiyama M, Kim K, Okawa M, Shibui K, Kudo Y, et al. Sleep loss and daytime sleepiness in the general adult population of Japan. Psychiatry Res. 2000;93(1):1.
58. Magee CA, Iverson D, Huang X-F, Caputi P. A link between chronic sleep restriction and obesity: methodological considerations. Public Health. 2008;122(12):1373-81.
59. Patel SR. Social and demographic factors related to sleep duration. Sleep. 2007;30(9):1077.
60. Grandner MA, Kripke DF. Self-reported sleep complaints with long and short sleep: a nationally representative sample. Psychosom Med. 2004;66(2):239-41.
61. Ohayon M, Carskadon M, Guilleminault C, Vitiello M. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep. 2004;27(7):1255.
62. Scott LD, Hwang W-T, Rogers AE, Nysse T, Dean GE, Dinges DF. The relationship between nurse work schedules, sleep duration, and drowsy driving. Sleep. 2007;30(12):1801.
63. Friedman L, Benson K, Noda A, Zarcone V, Wicks DA, O'Connell K, et al. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol. 2000;13(1):17-27.
64. Umberson D. Family status and health behaviors: Social control as a dimension of social integration. J Health Soc Behav. 1987:306-19.
65. Monk TH, Buysse DJ, Rose LR, Hall JaA, Kupfer DJ. The sleep of healthy people-a diary study. Chronobiol Int. 2000;17(1):49-60.
66. Tamakoshi A, Ohno Y. Self-reported sleep duration as a predictor of all-cause mortality: results from the JACC study, Japan. Sleep. 2004;27(1):51.
67. Caldwell J, Caldwell J, Schmidt R. Alertness

management strategies for operational contexts. Sleep Med Rev. 2008;12(4):257.
68. Szentkirályi A, Madarász CZ, Novák M. Sleep disorders: impact on daytime functioning and quality of life. Expert Rev Pharmacoecon Outcomes Res. 2009;9(1):49-64.
69. Panossian LA, Avidan AY. Review of sleep disorders. Med Clin North Am. 2009;93(2):407-25.
70. U.S. Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute, National Center on Sleep Disorders Research, Trans-NIH Sleep Research Coordinating Committee. National sleep disorders research plan. 2003 July. NIH Publication No. 03-5209.
71. Belenky G, Wesensten NJ, Thorne DR, Thomas ML, Sing HC, Redmond DP, et al. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: A sleep dose?response study. J Sleep Res. 2003;12(1):1-12.
72. Tomasi D, Wang R, Telang F, Boronikolas V, Jayne M, Wang G-J, et al. Impairment of attentional networks after 1 night of sleep deprivation. Cereb Cortex. 2009;19(1):233-40.
73. Kendall AP, Kautz MA, Russo MB, Killgore WD. Effects of sleep deprivation on lateral visual attention. Int J Neurosci. 2006;116(10):1125-38.
74. Flombaum JI, Posner MI. The activation of attentional networks. Neuroimage. 2005;26:471-9.
75. Balkin TJ, Bliese PD, Belenky G, Sing H, Thorne DR, Thomas M, et al. Comparative utility of instruments for monitoring sleepiness?related performance decrements in the operational environment. J Sleep Res. 2004;13(3):219-27.
76. Carskadon MA. Sleep deprivation: health consequences and societal impact. Med Clin North Am. 2004;88(3):767-76.
77. Leger D. The cost of sleep-related accidents: a report for the National Commission on Sleep Disorders Research. Sleep. 1994;17(1):84.
78. National Institutes of Health, National Heart, Lung, and Blood Institute, National Center on Sleep Disorders Research, National Highway Traffic Safety Administration. Drowsy driving and automobile crashes: report and recommendation. 1998 Apr. Report No.: DOT HS 808 707
79. Pack AI, Pack AM, Rodgman E, Cucchiara A, Dinges DF, Schwab CW. Characteristics of crashes attributed to the driver having fallen asleep. Accid Anal Prev. 1995;27(6):769-75.
80. Dawson D, Reid K. Fatigue, alcohol and performance impairment. Nature. 1997;388(6639):235-37.
81. Kim D-J, Lee H-P, Kim MS, Park Y-J, Go H-J, Kim K-S, et al. The effect of total sleep deprivation on cognitive functions in normal adult male subjects. Int J Neurosci. 2001;109(1-2):127-37.
82. Yoo S-S, Gujar N, Hu P, Jolesz FA, Walker MP. The human emotional brain without sleep—a prefrontal amygdala disconnect. Curr Biol. 2007;17(20):R877-R8.
83. Harrison Y, Horne JA. The impact of sleep deprivation of decision making: A review. J Exp Psychol Appl. 2000;6(3):236-49.
84. Rauchs G, Desgranges B, Foret J, Eustache F. The relationships between memory systems and sleep stages. J Sleep Res. 2005;14(2):123-40.
85. Walker MP, Stickgold R. Sleep, memory, and plasticity. Annu Rev Psychol. 2006;57:139-66.
86. Gais S, Born J. Declarative memory consolidation: mechanisms acting during human sleep. Learn Mem. 2004;11(6):679-85.
87. Tulving E, Markowitsch HJ. Episodic and declarative memory: role of the hippocampus. Hippocampus. 1998;8(3):198-204.
88. Siegel JM. The REM sleep-memory consolidation hypothesis. Science. 2001;294(5544):1058-63.
89. Irwin M, McClintick J, Costlow C, Fortner M, White J, Gillin J. Partial night sleep deprivation reduces natural killer and cellular immune responses in humans. FASEB J. 1996;10(5):643-53.
90. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. The Lancet. 1999;354(9188):1435-9.
91. Spiegel K, Leproult R, L'Hermite-Balériaux M, Copinschi G, Penev PD, Van Cauter E. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. J Clin Endocrinol Metab. 2004;89(11):5762-71.
92. Tochikubo O, Ikeda A, Miyajima E, Ishii M. Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. Hypertension. 1996;27(6):1318-24.
93. Meier-Ewert HK, Ridker PM, Rifai N, Regan MM, Price NJ, Dinges DF, et al. Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. J Am Coll Cardiol. 2004;43(4):678-83.
94. Marshall NS, Glozier N, Grunstein RR. Is sleep duration related to obesity? A critical review of the epidemiological evidence. Sleep Med Rev. 2008;12(4):289-98.
95. Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. Sleep Med Rev. 2007;11(3):163.
96. Patel S, Ayas N, Malhotra M, White D, Schernhammer E, Speizer F, et al. A prospective study of sleep duration and mortality risk in women.

Sleep. 2004;27(3):440.
97. American Academy of Sleep Medicine. International Classification of Sleep Disorders: Diagnostic and Coding Manual, Revised. Rochester, Minn: American Sleep Academy of Sleep Medicine; 2001.
98. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR. American Psychiatric Publishing, Inc.; 2000.
99. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev. 2002;6(2):97-111.
100. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry. 1996;39(6):411-8.
101. Espie CA, Kyle SD. Primary insomnia: an overview of practical management using cognitive behavioral techniques. Sleep Med Clin. 2009;4(4):559-69.
102. Jacobs GD, Pace-Schott EF, Stickgold R, Otto MW. Cognitive behavior therapy and pharmacotherapy for insomnia: a randomized controlled trial and direct comparison. Arch Intern Med. 2004;164(17):1888.
103. Bjorvatn B, Pallesen S. A practical approach to circadian rhythm sleep disorders. Sleep Med Rev. 2009;13(1):47.
104. Stepanski EJ, Wyatt JK. Use of sleep hygiene in the treatment of insomnia. Sleep Med Rev. 2003;7(3):215-25.
105. Hauri P. Sleep hygiene, relaxation therapy, and cognitive interventions. In: Hauri P, editor. Case studies in insomnia. New York: Plenum Medical Book Company, 1991:65-84.
106. Sousa I, Araújo J, Azevedo C. The effect of a sleep hygiene education program on the sleep–wake cycle of Brazilian adolescent students. Sleep Biol Rhythms. 2007;5(4):251-8.
107. Nehlig A. Are we dependent upon coffee and caffeine? A review on human and animal data. Neurosci Biobehav Rev. 1999;23(4):563-76.
108. Porkka-Heiskanen T, Alanko L, Kalinchuk A, Stenberg D. Adenosine and sleep. Sleep Med Rev. 2002;6(4):321-32.
109. Landolt H-P, Roth C, Dijk D-J, Borbely AA. Late-afternoon ethanol intake affects nocturnal sleep and the sleep EEG in middle-aged men. J Clin Psychopharmacol. 1996;16(6):428-36.
110. Driver HS, Taylor SR. Exercise and sleep. Sleep Med Rev. 2000;4(4):387-402.
111. Horne J, Reid A. Night-time sleep EEG changes following body heating in a warm bath. Electroencephalogr Clin Neurophysiol. 1985;60(2):154-7.
112. Liao W-C. Effects of passive body heating on body temperature and sleep regulation in the elderly: a systematic review. Int J Nurs Stud. 2002;39(8):803-10.
113. Rosa RR, Bonnet MH. Performance and alertness on 8 h and 12 h rotating shifts at a natural gas utility. Ergonomics. 1993;36(10):1177-93.
114. Cornu C, Remontet L, Noel-Baron F, Nicolas A, Feugier-Favier N, Roy P, et al. A dietary supplement to improve the quality of sleep: a randomized placebo controlled trial. BMC Complement Altern Med. 2010;10(1):29.
115. Pigeau R, Naitoh P, Buguet A, McCann C, Baranski J, Taylor M, et al. Modafinil, d-amphetamine and placebo during 64 hours of sustained mental work. I. Effects on mood, fatigue, cognitive performance and body temperature. J Sleep Res. 1995;4(4):212-28.
116. Bonnet M, Balkin T, Dinges D, Roehrs T, Rogers N, Wesensten N, et al. The use of stimulants to modify performance during sleep loss: a review by the sleep deprivation and Stimulant Task Force of the American Academy of Sleep Medicine. Sleep. 2005;28(9):1163.
117. Bonnet M. The effect of varying prophylactic naps on performance, alertness and mood throughout a 52-hour continuous operation. Sleep. 1991;14(4):307-15.
118. Dinges DF, Orne MT, Orne EC. Assessing performance upon abrupt awakening from naps during quasi-continuous operations. Behavior Research Methods, Instruments, & Computers. 1985;17:37-45.