

# Occupational Health and Sleep Issues in Underserved Populations



Medhat Kalliny, MD, PhD<sup>a,\*</sup>, Judith Green McKenzie, MD, MPH<sup>b</sup>

## KEYWORDS

- Sleep disorders • Sleep health disparities • Burden of occupational injury and illness
- Occupational hazards, injuries and illnesses in underserved worker populations
- Workers' compensation

## KEY POINTS

- In the United States, substantial racial, ethnic, and socioeconomic disparities exist in sleep health.
- Poor sleep is associated with a wide range of health effects, and therefore, it is important that primary care physicians working in underserved communities are aware of this disparity and target this higher-risk group for focused evaluation and intervention.
- The workplace, home, and social environment, as well as diet and genetics among other factors, work together to affect an individual's health status.
- Workplace hazards impact one's overall health status, which in turn impact one's ability to obtain, perform, and tolerate work, as well as gain satisfaction from work.
- Primary care physicians should be familiar not only with the type of work an individual does but also with workplace hazards and their effects on individual's health and how to address them.

## SLEEP DISORDERS: AN IMPORTANT PUBLIC HEALTH PROBLEM

### *Historical Background*

Sleep and dreams have been a mystery and topics of writings by philosophers, writers, religious leaders, and scientists since the inception of the recorded history.<sup>1</sup> The Greeks and Romans personified sleep through their deities: Hypnos and Somus, respectively.<sup>2</sup> Hippocrates was likely the first writer in the ancient world to mention the

---

This article originally appeared in Primary Care: Clinics in Office Practice, Volume 44, Issue 1, March 2017.

The authors have no conflicts of interest to disclose.

<sup>a</sup> Department of Family and Community Medicine, Meharry Medical College, 1005 Dr D. B. Todd Boulevard, Nashville, TN 37208, USA; <sup>b</sup> Division of Occupational Medicine, Department of Emergency Medicine, Hospital of the University of Pennsylvania, First Floor, Silverstein Pavilion, Philadelphia, PA 19104-4283, USA

\* Corresponding author.

E-mail address: [mkalliny@mmc.edu](mailto:mkalliny@mmc.edu)

Physician Assist Clin 4 (2019) 81–105

<https://doi.org/10.1016/j.cpha.2018.08.005>

2405-7991/19/© 2018 Elsevier Inc. All rights reserved.

[physicianassistant.theclinics.com](http://physicianassistant.theclinics.com)

importance of sleep in general health.<sup>3</sup> In 350 BC, the Greek philosopher, Aristotle, wrote about sleep and waking, whether they are a function of the body or the soul, and the significance of dreams.<sup>4</sup> Interestingly, in 360 BC, historical documents described obstructive sleep apnea (OSA), for the first time: Dionysius, the tyrant of Heraclea, died “choking on his own fat.”<sup>5</sup> Similar writings about sleep and health are found in Egyptian, Indian, and Chinese ancient civilizations and early modern era.<sup>6,7</sup>

In 1836, Dickens wrote about OSA in his work the *Posthumous Papers of the Pickwick Club*, wherein he described “Joe the Fat Boy” as obese and sleepy and a snorer.<sup>8</sup> Thereafter, in 1956, Burwell and colleagues<sup>9</sup> described OSA as Pickwickian syndrome. Electroencephalography changes during sleep and rapid eye movement (REM) were described for the first time by Loomis and colleagues<sup>10</sup> and Aserinsky and Kleitman,<sup>11</sup> respectively. In 1957, Dement and Kleitman<sup>12</sup> identified the stages of sleep. In 1971, Konopa and Benzer<sup>13</sup> discovered the first circadian clock gene in *Drosophila*. Later in 1972, the suprachiasmatic nucleus (SCN) was discovered as the site of the body’s internal circadian pacemaker.<sup>14</sup> Clinicians, scientists, and researchers continue to work toward a greater understanding of the cause and pathophysiology of sleep disorders. Sleep Medicine is developing into an interdisciplinary field in which integration and coordination across the traditional medical specialties, other health care providers such as dentists, and between basic and clinical science are vital.<sup>15</sup>

### **Scope of the Problem**

About 50 to 70 million Americans chronically suffer from a sleep disorder. Sleep-disordered breathing (SDB), including OSA, affects more than 15% of the population and causes excessive daytime sleepiness, injuries, hypertension, cognitive impairment, metabolic syndrome, and an increased risk of heart attack, stroke, and mortality. In children, SDB is associated with cardiovascular and metabolic risk factors, attention-related behavioral problems, and poor academic performance.<sup>16</sup>

Nationwide, 70% of adults report insufficient sleep at least once each month and 11% report such difficulties daily.<sup>17</sup> Nearly 70% of high school adolescents sleep less than the recommended 8 to 9 hours of sleep on school nights.<sup>18</sup> Short and long sleep duration are associated with up to a 2-fold increased risk of obesity, diabetes, hypertension, cardiovascular disease, stroke, depression, substance abuse, and all-cause mortality.<sup>19</sup>

Chronic insomnia is the most common sleep disorder and affects more than 20% of adults. It is a risk factor for depression, substance abuse, and impaired function.<sup>20</sup>

Chronic circadian disorders, including shift work syndrome, affect 20% of the US workforce and is associated with significant safety hazard, increased risk of cardiovascular disease, cerebrovascular disease, breast cancer, colorectal cancer, prostate cancer, obesity, diabetes, gastrointestinal disease, motor vehicle crashes, and difficulty adhering to work schedules.<sup>21,22</sup>

Restless legs syndrome affects 5% of adults and causes sleep onset and maintenance insomnia and subsequent daytime sleepiness.

Another less common disorder is narcolepsy with and without cataplexy affecting 0.05% and 3.9% of population, respectively.

In addition to its deleterious health consequences, the cumulative long-term effects of sleep disorders have a significant economic impact. Billions of dollars a year are spent on direct medical costs associated with doctor visits, hospital services, prescriptions, and over-the-counter medications.<sup>17</sup>

### ***Sleep Health in Underserved Population***

---

In the United States, substantial racial, ethnic, and socioeconomic disparities exist in sleep health. Many studies found that those with longer work hours and lower socioeconomic status report less sleep duration and/or lower sleep quality.<sup>23–25</sup> In a survey of 15,227 Hispanics of low socioeconomic status, Cespedes and his colleagues<sup>26</sup> reported that 28% had insomnia, 19% were short sleepers, and 9% were long sleepers. Grandner and colleagues<sup>27</sup> assessed sleep complaints with a telephone survey of 159,856 participants from across the United States and found that unemployment, being unmarried, lower income, and lower educational attainment were associated with more sleep complaints. Similar findings were reported by other researchers.<sup>28,29</sup>

Because poor sleep is associated with all of the untoward health effects noted above, it is important that primary care physicians (PCPs) working in underserved communities are aware of this disparity and target this higher-risk group for focused evaluation and intervention.

### **SLEEP PHYSIOLOGY. A BRIEF PRIMER FOR THE PRIMARY CARE PHYSICIAN** ***Sleep-Wake Cycle and Circadian Rhythm***

---

Sleep-wake cycle, which consists of roughly 8 hours of nocturnal sleep and 16 hours of daytime wakefulness, is controlled by 2 internal influences: sleep homeostasis and circadian rhythm. The period of circadian rhythms is about 24 hours in a normal light-dark cycle and is synchronized to the external physical environment and social/work schedules. In humans, light is the strongest synchronizing agent. Sleep-wake cycle is controlled by the suprachiasmatic nucleus (SCN) of the hypothalamus. In addition to providing synchronization in time between various rhythms, the SCN also helps promote wakefulness.<sup>30–35</sup>

It is generally agreed that sleep quality and restfulness are best when the sleep schedule is regularly synchronized to the internal circadian rhythms and the external light-dark cycle and that individuals should go to bed and wake up at around the same time each day.<sup>36–38</sup> Sleep loss results in the accumulation of a sleep debt that must eventually be repaid—by napping or sleeping longer in later cycles. Even the loss of 1 hour of sleep time that accumulates for several days can have a powerful negative effect on daytime performance, concentration, and mood.<sup>39,40</sup> One study recently quoted in the *Economist* states that sleeping 4 hours per night “has the same impact on the performance of various cognitive tasks as a blood-alcohol level of 0.1%, well over the limit for driving a car.”<sup>41</sup>

### ***Sleep Architecture***

---

Sleep architecture refers to the basic structural organization of normal sleep. Cycles of non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep are recognized by using electroencephalographic recordings. NREM sleep is divided into stages N1, N2, and N3 representing a continuum of relative depth. Each has unique characteristics, including variations in brain wave patterns, eye movements, and muscle tone.<sup>42</sup> The function of alternations between these 2 types of sleep is not yet understood, but irregular cycling and absent sleep stages are associated with sleep disorders. For example, instead of entering sleep through NREM, as is typical, individuals with narcolepsy enter sleep directly into REM sleep.<sup>43</sup>

The individual usually enters sleep through N1 within 10 to 20 minutes after lights out, progressing through stage N2, followed by stage N3, and finally, to REM. However, individuals do not remain in REM sleep the remainder of the night but, rather, cycle between stages of NREM and REM throughout the night every 90 minutes. NREM

sleep constitutes about 75% to 80% of total sleep time, and REM sleep constitutes the remaining 20% to 25%.<sup>44</sup>

Stage N1 sleep serves a transitional role in sleep-stage cycling and constitutes 2% to 5% of total sleep. Stage N2 sleep lasts approximately 10 to 25 minutes in the initial cycle and lengthens with each successive cycle, eventually constituting between 45% and 55% of the total sleep time. Stage N3 is referred to as a slow-wave sleep (SWS), most of which occurs during the first third of the night and constitutes about 15% to 20% of sleep. N3 sleep is important for feeling well rested. It is important in restorative functions, lowering inflammatory cytokines, maintaining hormone balance, and, together with REM, in restorative memory processing.

REM sleep is defined by the presence of desynchronized (low-voltage, mixed-frequency) brain wave activity, muscle atonia, and bursts of REMs. During the initial cycle, the REM period may last only 1 to 5 minutes; however, it becomes progressively prolonged as the sleep episode progresses. The last REM period may last from 30 to 60 minutes. About 4 to 5 REM-NREM cycles occur during night sleep. As the night progresses, stage N3 sleep gets progressively less and the final sleep period is composed mainly of stage N2 and REM sleep. Dreaming is associated with REM sleep with approximately 80% of vivid dream recall resulting after arousal from this stage of sleep. REM sleep may also be important for memory consolidation.<sup>43–48</sup>

### ***Sleep Variants with Age***

Neither full-term nor premature neonates show clear circadian rhythm. Newborns usually sleep about 16 to 18 hours per day. Circadian rhythm begins to arise around 2 to 3 months of age, leading to sleep consolidation that manifests in greater durations of wakefulness during the day and longer periods of sleep at night. In young children, sleep amounts decrease as a child gets older. Most children discontinue napping between 3 and 5 years old. Older children are significantly more likely to experience challenges in initiating and maintaining sleep and having nightmares than younger children.<sup>49–56</sup>

Adolescents require 9 to 10 hours of sleep each night. More than a quarter of high school and college students were found to be sleep deprived. Slow-wave sleep (N3) progressively declines with advancing pubertal development; however, time spent in stage N2 increases. These changes are likely in part due to pubertal and hormonal changes that accompany the onset of puberty. With increasing age, the total sleep time and REM sleep decrease, leading to the emergence of normal sleep pattern in adults.<sup>57–59</sup>

Sleep architecture continues to change with age across adulthood. As they age, individuals tend to have earlier wake time. Older adults greater than the age of 65 typically awaken 1.33 hours earlier, and go to bed 1.07 hours earlier, than younger adults, which might be due to an advanced circadian pacemaker that accompanies age. Younger adults may experience brief awakenings, but they are usually minor and occur close to REM sleep transition; thus, sleep remains relatively consolidated. As an individual ages, slow-wave sleep (SWS) declines at a rate of about 2% per decade. Because arousal thresholds are typically highest during SWS, and because SWS declines with age, older adults experience more frequent awakenings during a sleep episode.<sup>60–62</sup>

Elderly show an increase in disturbed sleep that can create a negative impact on their quality of life, mood, and alertness. Although the ability to sleep becomes more difficult, the need to sleep does not decrease with age. Elderly usually suffer both sleep onset and maintenance insomnia, both of which are associated with depression, respiratory symptoms, and physical disability. The progressive decrease in SWS is one of the most prominent changes with aging; however, it appears to preferentially affect men. The reason for the gender difference is unclear.<sup>63–66</sup> Other

prominent factors affecting sleep in the elderly (that are beyond the scope of this text) are a decrease in melatonin levels, changes in sleep latency, nighttime awakenings, inconsistency of external cues such as light exposure, irregular mealtimes, nocturia, and decreased mobility leading to a reduction in exercise.<sup>60–66</sup>

## THE IMPACT OF SELECTED SLEEP DISORDERS—WITH SPECIFIC FOCUS ON THE UNDERSERVED

### *Sleep Deprivation*

Sleep deprivation is defined as sleeping less than the recommended 7 to 9 hours of sleep per night. The causes of sleep deprivation are multifactorial. All 4 common sleep disorders mentioned above as well as occupational and lifestyle changes can lead to sleep deprivation.<sup>67</sup> The National Health and Nutrition Examination Survey showed that about 37.1% of US adults report regularly sleeping less than 7 hours per night. Short sleep duration was more common among young adults (aged 20–39 years) and non-Hispanic blacks.<sup>68</sup> Patients with sleep deprivation experience difficulty in concentration, impaired ability to perform daily tasks, lower mental and physical well-being, worsening of chronic diseases, and increased morbidity and mortality.<sup>69,70</sup> Similar findings were reported by other studies.<sup>71,72</sup>

In a cross-sectional survey of 9714 randomly selected subjects, effect of socioeconomic factors on sleep quality was assessed. Sleep quality was strongly associated with poverty level, employment status, and education level.<sup>73</sup> It is postulated that perceived discrimination is a potential cause of sleep disturbance and its resulting health consequences. In their analysis of data of 7148 adults from Michigan and Wisconsin, Grandner and colleagues<sup>74</sup> found that perceived racial discrimination was associated with increased risks of sleep disturbance (odds ratio [OR] = 2.62,  $P < .0001$ ) and daytime fatigue (OR = 2.07,  $P < .0001$ ). Similarly, in a study of 168 Hispanic-American immigrants, perceived racism was related to increased sleep disturbance and higher levels of depressive symptoms.<sup>75</sup>

Sleep deprivation is associated with wide-ranging effects on the cardiovascular, endocrine, immune, and nervous systems, including obesity, diabetes, impaired glucose tolerance, cardiovascular disease, hypertension, depression, anxiety, fatigue, lack of concentration, increased inflammatory markers, and impairment of functional capacity.<sup>76–82</sup>

Sleep deprivation is also common among adolescents.<sup>83</sup> In a study of 242 healthy adolescents, Troxel and associates<sup>84</sup> found that adolescents from single-parent households had poorer sleep efficiency across the week and shorter sleep duration on weekends. Black adolescents from single-parent households were found to have the lowest weekend sleep efficiency. Inadequate sleep among adolescents has negative consequences for self-regulation and emotional well-being as well as poor school performance, behavioral problems, obesity, insulin resistance, and hypertension.<sup>85,86</sup>

### *Sleep-Disordered Breathing*

OSA is the most common type of sleep-disordered breathing (SDB). It is characterized by loud snoring, breathing interruptions, awakenings, gasping, and choking and usually results in excessive daytime sleepiness.<sup>87–90</sup> OSA is common in adults, with men, older individuals, and the obese being at higher risk. The prevalence of OSA ranges between 2% and 7% with higher prevalence among non-Hispanic blacks.<sup>91–93</sup> In the Sleep Heart Health Study, black men and women had significantly higher Epworth Sleepiness Scores reflecting greater daytime sleepiness.<sup>94</sup> Snoring is the most common reported symptom of OSA; however, awareness of the predominant symptom of

OSA and knowledge of its clinical significance appear to be the lowest among minorities and those with low socioeconomic status and education.<sup>28,95–100</sup>

Epidemiologic and clinical studies suggest that between 35% and 91% of patients with hypertension have OSA.<sup>101,102</sup> A strong racial disparity exists in the prevalence and treatment of hypertension and its relationship to OSA.<sup>103</sup> Jean-Louis and colleagues<sup>101</sup> reported that hypertensive blacks have a 91% prevalence of SDB. A causal association between OSA and hypertension is supported by evidence of a dose-response relationship; the higher the apnea-hypopnea index, the greater the increase in blood pressure. Treatment of OSA by continuous positive airway pressure (CPAP) therapy can reduce blood pressure levels.<sup>104–108</sup> Similarly, OSA is associated with increased risk of other cardiovascular diseases, including arrhythmias, coronary artery disease, myocardial infarction, and congestive heart failure.<sup>109–113</sup> Those with severe OSA have 3-fold higher risk of fatal cardiovascular events. CPAP therapy reduces cardiovascular risk and mortality in patients with OSA.<sup>112,114–118</sup>

OSA is associated with impaired glucose tolerance and insulin resistance especially in those with the highest apnea-hypopnea index. It has been suggested that OSA causes intermittent hypoxia and recurrent sleep arousals, which in turn stimulate the sympathetic nervous system, hypothalamic-pituitary-adrenal axis, and adipocytes with release of catecholamines, cortisol, and inflammatory cytokines and other vasoactive intermediates, which may mediate the development of glucose intolerance, insulin resistance, and, ultimately, type 2 diabetes.<sup>119–121</sup> Babu and colleagues<sup>122</sup> reported that CPAP improved glycemic control in diabetic patients with OSA.

Up to 40% of people who are morbidly obese have OSA. Obesity is a well-established risk factor for the development of OSA and could be a consequence of OSA.<sup>123</sup> OSA-associated obesity might be due to decreased physical activity secondary to excessive daytime sleepiness and/or higher levels of nonfunctional leptin.<sup>124</sup> Significant weight loss usually results in reduction of OSA severity.<sup>125</sup>

### ***Insomnia***

Insomnia is the most commonly reported sleep disorder. Insomnia could be sleep-onset insomnia; difficulty to initiate sleep or sleep maintenance insomnia; or difficulty to maintain sleep. Insomnia could result in daytime consequences, such as tiredness, lack of energy, difficulty concentrating, and/or irritability.<sup>126,127</sup> Insomnia affects between 15% and 20% of adults in the United States. However, prevalence increase was noted recently among young adults, elderly, women, whites, Hispanics, diabetics, and patients with joint pain.<sup>128,129</sup> Family history of insomnia, low socioeconomic status, stressful lifestyles, medical and psychiatric disorders, and shift work are other risk factors for insomnia.<sup>130–134</sup>

Daily experiences of discrimination, workplace harassment and incivilities, and other stressors are significant factors for poor sleep quality and insomnia.<sup>135,136</sup> In a study of 1289 pregnant Latinas, Manber and colleagues<sup>137</sup> found that depression, lack of social support, and low income were significant risk factors for insomnia; however, strong family ties, group identity, and English proficiency were protective factors.

Insomnia is conceptualized as a state of hyperarousal. The exact causes of insomnia are poorly understood. Biological, psychological, and social factors might play a role in insomnia pathogenesis. Adults with insomnia have higher levels of cortisol and adrenocorticotropic hormone, reduced cortisol awakening response, and flattened diurnal cortisol profile. Studies suggest that insomnia might be due to overactivity of multiple neural systems, particularly brainstem, hypothalamus, and basal forebrain. In addition, limbic and paralimbic structures that regulate basic emotions and instinctual behaviors, such as the amygdala, hippocampus, ventromedial

prefrontal cortex, and anterior cingulate cortex, have been shown to be abnormally active during sleep in individuals with insomnia.<sup>138–141</sup>

Pain is another major risk factor for insomnia. Bazargan and colleagues<sup>142</sup> found that patients with a level of pain of 5 or higher (on a scale of 0–10) showed a higher level of insomnia. Cognitive factors, such as worry, rumination, and fear of sleeplessness, light exposure, non-regular sleep schedule, and exposure to trauma, increase the odds of insomnia.<sup>105,143</sup> Chronic insomnia is associated with increased risk of cardiovascular diseases, such as acute myocardial infarction, hypertension, arrhythmias, cerebrovascular diseases, diabetes, psychiatric disorders, and all-cause mortality.<sup>144–147</sup>

### ***Circadian Rhythm Disorders***

Circadian rhythm sleep disorders are persistent or recurrent patterns of sleep disturbance due to misalignment of the circadian clock in relation to environmental cues and the terrestrial light-dark cycle. They usually cause insomnia, excessive sleepiness, or both and are associated with impairment of social, occupational, or other functions. Delayed sleep phase and advanced sleep phase disorders are the most common circadian rhythm disorders.<sup>148</sup>

Delayed sleep phase syndrome is characterized by sleep onset and wake times that are typically delayed 3 to 6 hours relative to conventional sleep-wake times. On the other hand, advanced sleep phase syndrome is characterized by involuntary bedtimes and awake times that are more than 3 hours earlier than societal means. In both conditions, the amount of sleep is not affected.<sup>149</sup> Circadian rhythm disorders are more prevalent in adolescents and young adults.<sup>150,151</sup> Biological, physiologic, and genetic factors play an important role in pathogenesis of circadian rhythm disorders. Nightshift workers are at higher risk for delayed sleep phase syndrome due to irregular circadian entrainment. Similarly, individuals who live in extreme latitudes and are exposed to extended periods of light may also be at increased risk.<sup>152–156</sup> Polymorphisms in circadian clock genes have been identified in familial delayed and advanced sleep phase syndromes.<sup>157–159</sup> Delayed and advanced sleep phase syndromes impair an individual's job performance and are associated with marital problems and financial difficulty. In adolescents, they are associated with increased daytime irritability, poor school performance, and psychiatric disorders.<sup>150</sup>

Treatment of delayed sleep phase syndrome requires resynchronizing to a more appropriate phase to the 24-hour light-dark cycle. In addition to a structured sleep-wake schedule and good sleep hygiene practices, potential therapies include resetting the circadian pacemaker with bright light, melatonin, or a combination of both.<sup>160</sup> Treatment options for individuals with advanced sleep phase syndrome are limited. Bright light therapy in the evening has been used successfully.<sup>161</sup> It is also hypothesized that administration of low levels of melatonin in the early morning may also be used.<sup>162</sup>

### ***Restless Leg Syndrome***

Restless leg syndrome (RLS) is one of the most common movement disorders with a prevalence of 5%. It is more common in older adults and women; however, it may be found in adolescents and teenagers. It is characterized by an irresistible urge to move the legs, which worsens during rest or inactivity, especially in the evening and at night, causing most individuals difficulty falling asleep. It also may affect the arms, trunk, or head and neck. It may also be associated with paresthesias, which individuals describe as creepy-crawly, jittery, itchy, or burning feelings. The symptoms are partially or completely relieved by movement. Individuals with RLS often experience



periodic limb movements; however, periodic limb movement disorder is not always associated RLS.<sup>163–167</sup>

RLS affects more than 20% of pregnant women secondary to transient low levels of ferritin and folate; therefore, they typically disappear within 4 weeks after delivery.<sup>168</sup> It may also be associated with attention-deficit hyperactivity disorder (ADHD). Chervin and colleagues<sup>169</sup> reported that ADHD symptoms were almost twice as likely to occur with symptoms of RLS as would be expected by chance alone.

The exact cause of RLS is not completely understood. It likely results from altered dopamine and iron metabolism. RLS commonly occurs in individuals with iron deficiency, including end-stage renal disease, iron-deficiency anemia, pregnancy, and bariatric surgery. Iron is necessary for the synthesis of dopamine and the activity of the D<sub>2</sub> dopamine receptor. In patients with RLS, reduced iron levels were noted in the substantia nigra, which is responsible for controlling voluntary movement through neurons that rely on dopamine as a neurotransmitter. The association between dopamine, iron deficiency, and RLS is further supported by observations that dopamine antagonists cause worsening of RLS symptoms, while dopamine agonists are used to treat RLS. There is strong evidence for genetic predisposition, in which susceptibility gene loci have been identified on chromosomes 12q, 14q, and 9p; however, no genetic markers are currently available.

There is early evidence that RLS is more common in underserved communities, with one recent survey noting RLS symptoms greater than 3 times per week in more than 10% of rural poor respondents.<sup>170</sup> Higher rates have also been seen in older patients, women, those with lower socioeconomic status, those with lower education, and those unemployed, retired, or disabled, and non-Hispanic whites.<sup>170,171</sup> Dopaminergic agents are the primary treatment option for individuals with RLS.<sup>169–184</sup>

In conclusion, the PCP working in underserved areas should be aware of the common sleep disturbances discussed above, their untoward health effects, and their increased incidence among underserved populations. Treatment approaches can be complex and often require multidisciplinary interventions, but the potential rewards in patient well-being, societal benefit, and physician satisfaction make addressing such issues paramount.

## **OCCUPATIONAL HEALTH IN UNDERSERVED POPULATIONS**

### ***Occupational Health Services for Underserved Populations***

#### ***Historical context***

Occupational health and safety is the field pertaining to the health and safety of the workforce and lies at the interface between work and health.<sup>185</sup> Workplace hazards impact one's health status, which in turn impacts one's ability obtain, perform, and tolerate work as well as gain satisfaction from work. Not only is it important for the PCP to know the type of work an individual does but also to be familiar with the workplace and its hazards, whether physical, chemical, biologic, mechanical, or psychosocial, will assist the astute PCP to render optimal care to their working patients. The workplace, home, and social environment, as well as diet and genetics among other factors, work together to affect an individual's health status.<sup>186</sup>

The PCP might overlook work-related causes of injury and illness if an index of suspicion does not exist.<sup>187</sup> The focus may be placed on fitting the presenting signs and symptoms into a nonoccupational cause when the diagnosis may be right at hand if occupation is queried. As such, all patients who present to their PCP should be queried as to their occupation.<sup>188</sup> This simple question is imperative, because many workers may not seek care from an occupational and environmental medicine



(OEM) physician for a work-related injury or illness for several reasons. They may not realize that their injury or illness is work related; they may not have access to an OEM physician; they may not know how to report a work related event, or they may fear reprisal.<sup>189</sup> As many as 25% of visits to PCPs are for work-related conditions.<sup>190</sup> It is in understanding a patient's work that, through educating the patient on preventive measures, future similar injuries may be prevented.<sup>191</sup> Indeed, approximately one-quarter of physicians have no work history recorded in their chart.<sup>192</sup>

### ***Workers' compensation***

Workers' compensation insurance covers workers whose injuries or illnesses arise out of work. The oldest form of social insurance in the United States and the third largest source of support for disabled workers after Social Security and Medicare, the Workers' compensation system started during the early part of the twentieth century for the purpose of providing monetary compensation for medical and rehabilitation costs and lost wages to certain workers with work-related injuries or disabilities. The Workers' compensation system can be credited for helping to create a more humane environment for covered workers. Workers' compensation statutes are based on the legal principle of "exclusive remedy" whereby an injured employee can only claim compensation within the system. Workers in effect have given up the right to sue the employer at common law. In return, the injured employee receives total reimbursement for the medical costs incurred as well as full or partial wage replacement during the period during which the employee was unable to work. The United States does not have a unified Workers' compensation law because each state, federal jurisdiction, and territory have individual systems, statutes, and regulations. As such, the PCP may need to be familiar with the law in the area in which they practice.<sup>193</sup>

Some categories of workers are excluded from Workers' compensation protection. This includes workers employed by companies with 5 or fewer employees, agricultural workers, domestic (household) workers, casual laborers, independent contractors, the self-employed, business owners and partners, and state, municipal, and nonprofit institution employees. In addition, Workers' compensation insurance is not compulsory for most private employment in some states.

### ***Health status of underserved workers***

Underserved workers have various vulnerabilities. They may not fall under the protection of Workers' compensation<sup>193</sup> and may also have no private medical insurance.<sup>194–196</sup> In the event they are insured under Workers' compensation, they may be unaware of their eligibility of the Workers' compensation laws or they may be in the country illegally and as such may not want to come forward for fear of deportation, even if the injury or illness is work related.<sup>197</sup> The PCP may also be unaware of their patient's eligibility.<sup>198</sup>

In general, underserved workers have an unequal increased burden of chronic disease.<sup>199–202</sup> Chronic disease, if unrecognized and untreated, may delay recovery from a work-related injury<sup>203</sup> or even render a worker more prone to occupational injury or illness.

Often in the Workers' compensation arena, the occupational medicine physician may be the only physician the worker sees for an extended period of time. Given that workers must often work in a job for a minimum period of time before medical insurance is available to them, there may be a financial barrier to timely referral to primary care. On the other hand, a worker may present to the PCP for treatment of an injury that is not recognized as work related. The PCP may focus on the newly

diagnosed chronic disease, such as hypertension and diabetes, without taking steps toward educating the worker on how to prevent such an injury or illness from recurring or getting worse.

### ***Burden of occupational injury and illness***

The magnitude of occupational disease and injury burden is underestimated but significant.<sup>204</sup> In 2014, private industries in the United States reported nearly 3.0 million nonfatal workplace injuries and illnesses—a rate of 3.2 cases per 100 equivalent full-time workers.<sup>205</sup> The public sector reported 5.7 cases among full-time state and local workers, such as police and firefighters, with almost half the injuries being sprains and strains. The 5 industries reporting the greatest workplace risk are the health care and social assistance sector (8.3/100 workers); transportation and warehousing (5.2/100 workers); arts, entertainment, and recreation (4.8/100 workers); agriculture, forestry, fishing, and hunting (4.8/100 workers); and manufacturing (4.4/100 workers).<sup>206</sup>

### ***Regulations and Regulatory Bodies***

#### ***The Occupational Safety and Health Administration***

The Occupational Safety and Health Act of 1970 (OSH Act) signed into law in 1970 led to the establishment of the Occupational Safety and Health Administration (OSHA) and the National Institute of Occupational Safety and Health (NIOSH). The OSH Act contains the “General Duty Clause,” which places a duty on the employer to provide a safe workplace and was created to cover all possible hazards, not just those that could be foreseen at the time. In general, the working environment has become “safer” since OSHA was created; there has been a reduction in workplace injuries and fatalities.<sup>207</sup> However, injury and illness do remain and are undercounted in underserved workers, who routinely work the more hazardous trades and who may not report or seek treatment due to fear of reprisal.<sup>189</sup>

OSHA is the agency of the Department of Labor that enforces the regulatory mandates of the OSH Act by setting standards, rules, and regulations by which covered employers are expected to conduct business. Its purpose is to prevent work-related injuries, illnesses, and occupational fatalities by issuing and enforcing standards for workplace safety and health. However, OSHA has limited resources such that it is unable to inspect every workplace that needs inspection. Priority is given to more dangerous situations or otherwise targeted inspections.<sup>208</sup>

NIOSH, a part of the Centers for Disease Control and Prevention, is the primary federal agency conducting research on the safety and health of the workplace and providing recommendations for the prevention of work-related illnesses and injuries. The National Occupational Research Agenda (NORA) is the research framework for NIOSH and the nation. Based on critical issues in workplace safety and health identified by stakeholders, goals and objectives are developed to address work-related injuries and diseases.<sup>209</sup> NORA places an explicit emphasis on worker populations that have been underserved, such as immigrant workers, health care workers, and hotel workers, among others.<sup>210</sup>

### ***Selected Underserved Worker Populations: Hazards, Injuries, and Illnesses***

#### ***Health care workers***

Health care workers (HCWs) are within the sector with the highest rate of occupational injury (health care and social assistance sector), yet 11% are underserved in they have no health insurance.<sup>211,212</sup> The uninsured HCW is more likely to be young, unmarried, African American or Hispanic, to have lower income, and to work part time, and is less

likely to have a college degree. Within this group, home health aides have the highest rate of uninsurance at 23.8%; licensed practical nurses are next at 14.5%, and then registered nurses at 5%. Nursing home workers were more likely to be uninsured than hospital-based workers with a rate of 20% compared with 8.2%.<sup>213</sup>

Despite being among the least insured in this sector, workers at nursing and residential care facilities experience the highest rate of workplace injuries and illnesses, with 8.3 incidents per 100 employees, whereas hospital-based health care workers saw an incidence rate of 7.0 per 100 employees. Ambulatory health care services and social assistance were more in line with the cross-industry average, with incidence rates of 2.8 and 3.5 per 100 employees, respectively.<sup>206</sup>

Injuries in this industry are mostly due to heavy lifting when handling patients<sup>214</sup> and exposure to blood-borne pathogens.<sup>215</sup> Home HCWs (home health aides, personal/home care aides, companions, nursing assistants, or home health nurses), among the least insured and most underserved in this sector, provide hands-on long-term care and personal assistance to clients with disabilities or other chronic conditions in patients' homes and in community-based services such as group homes. They have little control over their work environment, where in addition to encountering hazards, such as blood-borne pathogens, other biological hazards, and ergonomic hazards, may also encounter violence, latex, hostile animals, animal waste, tripping hazards, hazards on the road as they drive from home to home, overexertion, stress, guns and other weapons, illegal drugs, verbal abuse, temperature extremes, unhygienic conditions, lack of water, and otherwise dangerous conditions.<sup>214</sup>

With lack of insurance come delays in seeking care, fewer prevention visits, and poorer health status.<sup>211</sup> As such, if these underserved workers present, the PCP may be able to use this as an opportunity to try to address other issues such as chronic disease<sup>216</sup> and psychological strain,<sup>217</sup> if present. Even with insurance, these HCWs may be less likely to file a Workers' compensation claim.<sup>189</sup> The astute PCP will query appropriately regarding the work relatedness of the injury or illness, thus optimizing the visit and not only treating the injury or illness but also counseling on prevention of further similar work-related injury or illness. The Bureau of Labor Statistics (BLS) projects home health care employment as the fastest growing occupation for this decade.<sup>218</sup> The PCP, being aware of their working conditions, will be better able to serve them.

### **Hotel workers**

The hotel employee is another demographic that is underinsured. Many room attendants are immigrant or minority women, with most being Asian, Latin American, or African American.<sup>219</sup> Migrant and many immigrant workers are not covered by labor legislation and fear losing their jobs and hence livelihood.<sup>189</sup> Immigrants comprise 39.7% of maids and housekeeping cleaners<sup>220</sup> and are less likely to report an injury or illness that occur and also less likely to file a Workers' compensation claim.<sup>206</sup>

Despite this, hotel workers have higher rates of occupational injury and sustain more severe injuries than those in the service sector as a whole<sup>221</sup> with an overall injury rate in housekeepers at ~5.2 injuries per 100 worker-years. The highest rate was found for Hispanic housekeepers (10.6/100) with acute trauma rates highest in kitchen workers (4.0/100). Independently associated risk factors for injury in these workers are older age, female gender, and being Hispanic. In general, Hispanic workers have the highest rate of fatal and nonfatal OSHA-reported injuries in the United States, followed by black non-Hispanic workers.

The workload of hotel housekeepers that results in high injury and illness rates surpassing the national average involves physical hazards such as constant

repositioning, changing body postures, including bending, kneeling, lifting, stooping, squatting, twisting, and pushing<sup>221</sup> during room cleaning work, which is physically strenuous. They are also exposed to chemical hazards, such as cleaning products, and biological hazards, such as blood and waste.<sup>222</sup> They are also at risk for psychological distress as they experience conflicts within the workplace.<sup>223</sup> In addition, they often work isolated with little interaction with other housekeepers while on the job,<sup>224</sup> which can contribute to psychosocial stress.

The astute PCP will give optimal care within this context with an eye to prevention, in terms of both chronic disease and work-related injury and illness prevention as well as psychosocial support. The worker may not attribute their symptoms to work for fear of reprisal, in the case of immigration issues, the fear of deportation, for example, and this may affect the amount of information the worker shares with the PCP.<sup>225–227</sup>

### ***International Occupational Health***

---

#### ***Burden of occupational injury and illness worldwide***

Exposure to occupational hazards results in a significant proportion of the burden of disease and injury worldwide.<sup>228</sup> Much could be prevented using prevention strategies.<sup>229</sup> According to the International Labor Organization (ILO), 6300 people die every day as a result of occupational accidents or work-related diseases: more than 2.3 million deaths per year. There are 317 million accidents on the job annually. Many of these incidents lead to extended absences from work. There is a vast human cost of this daily adversity. Indeed, the economic burden of poor occupational safety and health (OSH) practices is estimated at 4% of global gross domestic product each year.<sup>230</sup> Employers face issues such as costly early retirement, absenteeism, loss of skilled staff, and high insurance premiums, due to work-related accidents and diseases. According to data from the World Health Organization, occupational risk factors are responsible for 8.8% of the global burden of mortality due to unintentional injuries. This is thought to be an underestimate due to underreporting. These global data are inadequate as they do not include intentional injury at work or commuting injury. Known prevention strategies implemented widely would diminish the avoidable burden of injuries in the workplace.<sup>231</sup>

#### ***Regulation and enforcement***

In 2003, the ILO adopted a Global strategy to improve occupational safety and health (OSH), which included the introduction of a preventive safety and health culture, the promotion and development of relevant instruments, and technical assistance. The ILO Constitution set forth the principle that workers should be protected from sickness, disease, and injury arising from their employment. ILO standards on OSH provide essential tools for governments, employers, and workers to establish such practices and to provide for maximum safety at work because many of these tragedies can be prevented through implementing sound prevention, reporting, and inspection practices. ILO Codes of Practice provide practical guidelines for public authorities, employers, workers, enterprises, and specialized OSH protection bodies. These instruments are not legally binding and are not intended to replace the laws, regulations, or standards in various countries. They provide guidance on safety and health at work.<sup>232</sup> More recently, ILO devised a Plan of Action toward a significant reduction in the unacceptable human suffering and economic losses that are still caused by work-related accidents and illnesses worldwide. ILO affirms the right to “decent, safe and healthy working conditions and environment.”<sup>233</sup>

An example of parallel legislation to protect workers in 3 systems are the OSH Act in the United States,<sup>234</sup> legislation in Canada through the Canadian Centre for

Occupational Health and Safety (CCOHS, 2015<sup>235</sup> and the Health and Safety at Work Act of Britain<sup>236</sup>). Other countries may base their legislation on these. The OSH Act passed with the goal to “to assure so far as possible every working man and woman in the Nation safe and healthful working conditions” places the responsibility for eliminating or minimizing hazardous conditions on the employer.<sup>234</sup> The CCOHS stipulates that an employer is to “exercise due diligence to implement a plan to identify possible workplace hazards and carry out corrective action to prevent accidents or injuries arising from these hazards,”<sup>235</sup> whereas The Health and Safety at Work Act of Britain<sup>236</sup> stipulates that “employers are to ensure as far as reasonably practicable, the health, safety and welfare at work of all his employees.”

## SUMMARY

As employers, governments, and national and international organizations work toward improving the health and safety of all workers, the road toward improved worker health will continue to be made clearer. There are inequities in that some workers are afforded care, whereas others are afforded suboptimal care or no care at all. Poverty and marginalized status in societies play a role. Indeed, in the United States, health insurance and Workers' compensation insurance are usually tethered to full-time employment so this safety net is only available to covered lives. Even so, workers with a real or perceived barrier to seeking care for a related injury or illness, covered or not, will not receive this benefit. Prevention is the optimal way to improve worker health and safety. As the science improves and occupational health and safety becomes more mainstream, PCPs will be increasingly equipped to help workers and worker populations attain high-quality, longer lives free of preventable disease, disability, injury, and premature death.<sup>237</sup>

## REFERENCES

1. Deak M, Epstein LJ. History of polysomnography. *Sleep Med Clin* 2009;4(3): 313–21.
2. Dement WC. History of sleep medicine. *Sleep Med Clin* 2008;3(2):147–56.
3. Adams CD. The Genuine Works of Hippocrates. 1868. Available at: [https://archive.org/stream/genuineworksofhi01hippuoft/genuineworksofhi01hippuoft\\_djvu.txt](https://archive.org/stream/genuineworksofhi01hippuoft/genuineworksofhi01hippuoft_djvu.txt). Accessed May 25, 2016.
4. Aristotle. On Sleep and Sleepiness. 350 B.C. Available at: <http://classics.mit.edu/Aristotle/sleep.html>. Accessed May 25, 2016.
5. Kryger MH. Sleep apnea. From the needles of Dionysius to continuous positive airway pressure. *Arch Intern Med* 1983;143(12):2301–3.
6. Barbara O'Neill. Sleep and the sleeping in ancient Egypt. 2012. Available at: <http://www.egyptological.com/2012/04/sleep-and-the-sleeping-in-ancientegypt-8146>. Accessed May 25, 2016.
7. Shakespeare W. “Prince of Denmark.” *The complete works of William Shakespeare*. London: Collins; 1960. p. 1141. Act II, Scene ii.
8. Dickens C. The Posthumous Papers of the Pickwick Club. 1836. Available at: <http://charlesdickenspage.com/pickwick.html>. Accessed May 25, 2016.
9. Burwell CD, Robin ED, Whaley RD, et al. Extreme obesity associated with alveolar hypoventilation: a Pickwickian syndrome. *Am J Med* 1956;2:811–8.
10. Loomis AL, Harvey EN, Hobart GA. Cerebral states during sleep as studied by human brain potentials. *J Exp Psychol* 1937;21:127–44.
11. Aserinsky E, Kleitman N. Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science* 1953;118(3062):273–4.

12. Dement W, Kleitman N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroencephalogr Clin Neurophysiol* 1957;9(4):673–90.
13. Konopka RJ, Benzer S. Clock mutants of *drosophila melanogaster*. *Proc Natl Acad Sci U S A* 1971;68:2112–6.
14. Moore RY, Eichler VB. Loss of a circadian adrenal corticosterone rhythm following suprachiasmatic lesions in the rat. *Brain Res* 1972;42(1):201–6.
15. Shepard JJW, Buysse DJ, Chesson JAL, et al. History of the development of sleep medicine in the United States. *J Clin Sleep Med* 2005;1(1):61–82.
16. NHLBI (National Heart, Lung, and Blood Institute). National sleep disorders research plan, 2011. Bethesda (MD): National Institutes of Health; 2011.
17. Centers for Disease Control and Prevention (CDC). Perceived insufficient rest or sleep among adults—United States, 2008. *MMWR Morb Mortal Wkly Rep* 2009; 58:1179.
18. Centers for Disease Control and Prevention (CDC). Youth risk behavior surveillance—United States, 2009. *MMWR Morb Mortal Wkly Rep* 2010;59:1.
19. National Sleep Foundation. Sleep in America Poll. 2008. Washington, DC. Available at: <http://www.sleepfoundation.org/article/sleep-america-polls/2008-sleep-performanceand-the-workplace>. Accessed May 25, 2016.
20. Roth T, Ancoli-Israel S. Daytime consequences and correlates of insomnia in the United States: results of the 1991 National Sleep Foundation survey. II. *Sleep* 1999;22(suppl 2):S354–8.
21. Healthy people 2020. Department of Health and Human Services; 2010. Office of Disease Prevention and Health Promotion Publication No. B0132.
22. Klauer SG, Dingus TA, Neale VL, et al. The impact of driver inattention on near-crash/crash risk: an analysis using the 100-car naturalistic driving study data. Washington, DC: National Highway Traffic Safety Administration; 2006. p. 1–192. HS810594.
23. Gellis LA, Lichstein KL, Scarinci IC, et al. Socioeconomic status and insomnia. *J Abnorm Psychol* 2005;114(1):111–8.
24. Moore PJ, Adler NE, Williams DR, et al. Socioeconomic status and health: the role of sleep. *Psychosom Med* 2002;64(2):337–44.
25. Krueger PM, Friedman EM. Sleep duration in the United States: a cross-sectional population-based study. *Am J Epidemiol* 2009;169(9):1052–63.
26. Cespedes EM, Dudley KA, Sotres-Alvarez D, et al. Joint associations of insomnia and sleep duration with prevalent diabetes: the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). *J Diabetes* 2016;8(3):387–97.
27. Grandner MA, Patel NP, Gehrman PR, et al. Who gets the best sleep? Ethnic and socioeconomic factors related to sleep complaints. *Sleep Med* 2010;11:470–8.
28. Chen X, Wang R, Zee P, et al. Racial/ethnic differences in sleep disturbances: the Multi-Ethnic Study of Atherosclerosis (MESA). *Sleep* 2015;38(6):877–88.
29. Hale L, Troxel WM, Kravitz HM, et al. Acculturation and sleep among a multi-ethnic sample of women: the Study of Women's Health across the Nation (SWAN). *Sleep* 2014;37(2):309–17.
30. Ancoli-Israel S, Ayalon L, Salzman C. Sleep in the elderly: normal variations and common sleep disorders. *Harv Rev Psychiatry* 2008;16:279–86.
31. Clayton J, Kyriacou C, Reppert S. Keeping time with the human genome. *Nature* 2001;409:829–31.
32. Shanahan T, Czeisler C. Physiological effects of light on the human circadian pacemaker. *Semin Perinatol* 2000;24:299–320.



33. Mistlberger R, Skene D. Social influences on mammalian circadian rhythms: animal and human studies. *Biol Rev Camb Philos Soc* 2004;79:533–56.
34. Van Someren E. More than a marker: interaction between the circadian regulation of temperature and sleep, age-related changes, and treatment possibilities. *Chronobiol Int* 2000;17:313–54.
35. Beersma D, Gordij M. Circadian control of the sleep-wake cycle. *Physiol Behav* 2007;90:190–5.
36. Åkerstedt T. Altered sleep/wake patterns and mental performance. *Physiol Behav* 2007;90:209–18.
37. Moore RY. Circadian rhythms: basic neurobiology and clinical applications. *Annu Rev Med* 1998;48:253–66.
38. Basheer R, Strecker RE, Thakkar MM, et al. Adenosine and sleep-wake regulation. *Prog Neurobiol* 2004;73:379–96.
39. Blagrove M, Alexander C, Horne JA. The effects of chronic sleep reduction on the performance of cognitive tasks sensitive to sleep deprivation. *Appl Cogn Psychol* 1995;9:21–40.
40. Bonnet MH, Rosa RR. Sleep and performance in young adults and older normals and insomniacs during acute sleep loss and recovery. *Biol Psychol* 1987;25:153–72.
41. The Economist. Life in the Fast Lane. Business people are racing to learn from Formula One drivers. Available at: <http://www.economist.com/news/business/21699456-business-people-are-racing-learn-formula-one-drivers-life-fast-lane>. Accessed June 6, 2016.
42. Zepelin H, Siegel JM, Tobler I. Mammalian sleep. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 91–100.
43. Carskadon M, Dement W. Normal human sleep: an overview. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 13–23.
44. Westerman D. *The concise sleep medicine handbook*. 2nd edition. Atlanta (GA): CreateSpace Independent Publishing Platform; 2013. p. 1–10.
45. Gais S, Molle M, Helms K, et al. Learning-dependent increases in sleep spindle density. *J Neurosci* 2002;22(15):6830–4.
46. Bader G, Gillberg C, Johnson M, et al. Activity and sleep in children with ADHD. *Sleep* 2003;26:A136.
47. Crick F, Mitchison G. The function of dream sleep. *Nature* 1983;304(5922):111–4.
48. Smith C, Lapp L. Increases in number of REMS and REM density in humans following an intensive learning period. *Sleep* 1991;14(4):325–30.
49. Mirmiran M, Maas Y, Ariagno R. Development of fetal and neonatal sleep and circadian rhythms. *Sleep Med Rev* 2003;7:321–34.
50. Goessel-Symank R, Grimmer I, Korte J, et al. Actigraphic monitoring of the activity-rest behavior of preterm and full-term infants at 20 months of age. *Chronobiol Int* 2004;21:661–71.
51. Adair RH, Bauchner H. Sleep problems in childhood. *Curr Probl Pediatr* 1993;23(4):142, 147–70.
52. Biagioni E, Boldrini A, Giganti F, et al. Distribution of sleep and wakefulness EEG patterns in 24-h recordings of preterm and full-term newborns. *Early Hum Dev* 2005;81:333–9.
53. Rivkees S. A developing circadian rhythmicity in infants. *Pediatrics* 2003;112:373–81.



54. Jenni OG, Carskadon MA. Sleep research society. SRS basics of sleep guide. Westchester (IL): Sleep Research Society; 2000. p. 11–9. Normal human sleep at different ages: infants to adolescents.
55. Roffward HP, Muzio JN, Dement WC. Ontogenetic development of the human sleep-dream cycle. *Science* 1966;152(3722):604–19.
56. Beltramini AU, Hertzog ME. Sleep and bedtime behavior in preschool-aged children. *Pediatrics* 1983;71(2):153–8.
57. Mercer PW, Merritt SL, Cowell JM. Differences in reported sleep need among adolescents. *J Adolesc Health* 1998;23(5):259–63.
58. Figueiro M, Rea M. Evening daylight may cause adolescents to sleep less in spring than in winter. *Chronobiol Int* 2010;27:1242–58.
59. Karacan I, Anch M, Thornby JI, et al. Longitudinal sleep patterns during pubertal growth: four-year follow up. *Pediatr Res* 1975;9(11):842–6.
60. Dijk DJ, 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.
61. Duffy JF, Dijk DJ, Klerman EB, et al. Later endogenous circadian temperature nadir relative to an earlier wake time in older people. *Am J Physiol* 1998;275(5 Pt 2):R1478–87.
62. Astrom C, Trojaborg W. Relationship of age to power spectrum analysis of EEG during sleep. *J Clin Neurophysiol* 1992;9(3):424–30.
63. Ancoli-Israel S, Sleep Research Society. SRS basics of sleep guide. Westchester (IL): Sleep Research Society; 2005. p. 21–6. Normal human sleep at different ages: sleep in older adults.
64. Reynolds CF III, Kupfer DJ, Taska LS, et al. Sleep of healthy seniors: a revisit. *Sleep* 1985;8(1):20–9.
65. Redline S, Kirchner HL, Quan SF, et al. The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Arch Intern Med* 2004;164(4):406–18.
66. Monk TH, Buysse DJ, Reynolds CF III, et al. Circadian temperature rhythms of older people. *Exp Gerontol* 1995;30(5):455–74.
67. Dinges D, Rogers N, Baynard MD. Chronic sleep deprivation. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 67–76.
68. Centers for Disease Control and Prevention (CDC). Effect of short sleep duration on daily activities—United States, 2005–2008. *MMWR Morb Mortal Wkly Rep* 2011;60(8):239–42.
69. Nunes J, Jean-Louis G, Zizi F, et al. Sleep duration among black and white Americans: results of the National Health Interview Survey. *J Natl Med Assoc* 2008;100(3):317–22.
70. Hale L, Do DP. Racial differences in self-reports of sleep duration in a population-based study. *Sleep* 2007;30(9):1096–103.
71. Jackson CL, Redline S, Kawachi I, et al. Racial disparities in short sleep duration by occupation and industry. *Am J Epidemiol* 2013;178(9):1442–51.
72. Hale L, Rivero-Fuentes E. Negative acculturation in sleep duration among Mexican immigrants and Mexican Americans. *J Immigr Minor Health* 2011;13(2):402–7.
73. Patel NP, Grandner MA, Xie D, et al. “Sleep disparity” in the population: poor sleep quality is strongly associated with poverty and ethnicity. *BMC Public Health* 2010;10:475.

74. Grandner MA, Hale L, Jackson N, et al. Perceived racial discrimination as an independent predictor of sleep disturbance and daytime fatigue. *Behav Sleep Med* 2012;10(4):235–49.
75. Steffen PR, Bowden M. Sleep disturbance mediates the relationship between perceived racism and depressive symptoms. *Ethn Dis* 2006;16(1):16–21.
76. Brimah P, Oulds F, Olafiranye O, et al. Sleep duration and reported functional capacity among black and white US adults. *J Clin Sleep Med* 2013;9(6):605–9.
77. Beihl DA, Liese AD, Haffner SM. Sleep duration as a risk factor for incident type 2 diabetes in a multiethnic cohort. *Ann Epidemiol* 2009;19(5):351–7.
78. Bidulescu A, Din-Dzietham R, Coverson DL, et al. Interaction of sleep quality and psychosocial stress on obesity in African Americans: the Cardiovascular Health Epidemiology Study (CHES). *BMC Public Health* 2010;10:581.
79. Owens JF, Buysse DJ, Hall M, et al. Napping, nighttime sleep, and cardiovascular risk factors in mid-life adults. *J Clin Sleep Med* 2010;6(4):330–5.
80. Spaeth AM, Dinges DF, Goel N. Sex and race differences in caloric intake during sleep restriction in healthy adults. *Am J Clin Nutr* 2014;100(2):559–66.
81. Hairston KG, Bryer-Ash M, Norris JM, et al. Sleep duration and five-year abdominal fat accumulation in a minority cohort: the IRAS family study. *Sleep* 2010;33(3):289–95.
82. Simpson NS, Banks S, Arroyo S, et al. Effects of sleep restriction on adiponectin levels in healthy men and women. *Physiol Behav* 2010;101(5):693–8.
83. Matthews K, Hall M, Dahl R. Sleep in healthy black and white adolescents. *Pediatrics* 2014;133(5):e1189–96.
84. Troxel WM, Lee L, Hall M, et al. Single-parent family structure and sleep problems in black and white adolescents. *Sleep Med* 2014;15(2):255–61.
85. Combs D, Goodwin JL, Quan SF, et al. Longitudinal differences in sleep duration in Hispanic and Caucasian children. *Sleep Med* 2016;18:61–6.
86. Martinez SM, Tschann JM, Greenspan LC, et al. Is it time for bed? Short sleep duration increases risk of obesity in Mexican American children. *Sleep Med* 2014;15(12):1484–9.
87. Phillipson EA. Sleep apnea—a major public health problem. *N Engl J Med* 1993;328:1271–3.
88. Rosen RC, Zozula R, Jahn EG, et al. Low rates of recognition of sleep disorders in primary care: comparison of a community-based versus clinical academic setting. *Sleep Med* 2001;2:47–55.
89. White DP. Central sleep apnea. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 969–82.
90. Thorpy MJ. Classification of sleep disorders. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 615–25.
91. Redline S, Tishler PV, Hans MG, et al. Racial differences in sleep-disordered breathing in African-Americans and Caucasians. *Am J Respir Crit Care Med* 1997;155:186–92.
92. Ancoli-Israel S, Klauber MR, Stepnowsky C, et al. Sleep-disordered breathing in African-American elderly. *Am J Respir Crit Care Med* 1995;152:1946–9.
93. Kripke DF, Ancoli-Israel S, Klauber MR, et al. Prevalence of sleep disordered breathing in ages 40–64 years: a population-based survey. *Sleep* 1997;20:65–76.

94. O'Connor GT, Lind BK, Lee ET, et al. Sleep Heart Health Study Investigators. Variation in symptoms of sleep-disordered breathing with race and ethnicity: the Sleep Heart Health Study. *Sleep* 2003;26(1):74–9.
95. Friedman M, Bliznikas D, Klein M, et al. Comparison of the incidences of obstructive sleep apnea-hypopnea syndrome in African-Americans versus Caucasian-Americans. *Otolaryngol Head Neck Surg* 2006;134(4):545–50.
96. Ram S, Seirawan H, Kumar SK, et al. Prevalence and impact of sleep disorders and sleep habits in the United States. *Sleep Breath* 2010;14(1):63–70.
97. Schmidt-Nowara WW, Coultas DB, Wiggins C, et al. Snoring in a Hispanic-American population. Risk factors and association with hypertension and other morbidity. *Arch Intern Med* 1990;150:597–601.
98. Goldstein NA, Abramowitz T, Weedon J, et al. Racial/ethnic differences in the prevalence of snoring and sleep disordered breathing in young children. *J Clin Sleep Med* 2011;7(2):163–71.
99. Bouscoulet LT, Vazquez-Garcia JC, Muino A, et al. Prevalence of sleep related symptoms in four Latin American cities. *J Clin Sleep Med* 2008;4:579–85.
100. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;328:1230–5.
101. Jean-Louis G, Zizi F, Casimir G, et al. Sleep-disordered breathing and hypertension among African Americans. *J Hum Hypertens* 2005;19(6):485–90.
102. Sjöström C, Lindberg E, Elmasry A, et al. Prevalence of sleep apnoea and snoring in hypertensive men: a population based study. *Thorax* 2002;57(7):602–7.
103. Lackland DT, Lin Y, Tilley BC, et al. An assessment of racial differences in clinical practices for hypertension at primary care sites for medically underserved patients. *J Clin Hypertens (Greenwich)* 2004;6:26–31.
104. Meetze K, Gillespie MB, Lee F. Obstructive sleep apnea: a comparison of black and white subjects. *Laryngoscope* 2002;112:1271–4.
105. Partinen M, Hublin C. Epidemiology of sleep disorders. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 626–47.
106. Faulx MD, Larkin EK, Hoit BD, et al. Sex influences endothelial function in sleep-disordered breathing. *Sleep* 2004;27(6):1113–20.
107. Nieto FJ, Herrington DM, Redline S, et al. Sleep apnea and markers of vascular endothelial function in a large community sample of older adults. *Am J Respir Crit Care Med* 2004;169(3):354–60.
108. Robinson GV, Stradling JR, Davies RJ. Sleep 6: Obstructive sleep apnoea/hypopnoea syndrome and hypertension. *Thorax* 2004b;59(12):1089–94.
109. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med* 2002;165(9):1217–39.
110. Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. *Am J Respir Crit Care Med* 2001;163(1):19–25.
111. Jennum P, Hein HO, Suadicani P, et al. Risk of ischemic heart disease in self-reported snorers. A prospective study of 2,937 men aged 54 to 74 years: the Copenhagen Male Study. *Chest* 1995;108(1):138–42.
112. Yaggi HK, Concato J, Kernan WN, et al. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med* 2005;353(19):2034–41.
113. Bradley TD, Logan AG, Kimoff RJ, et al. Continuous positive airway pressure for central sleep apnea and heart failure. *N Engl J Med* 2005;353(19):2025–33.

114. Marin JM, Carrizo SJ, Vicente E, et al. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005; 365(9464):1046–53.
115. Doherty LS, Kiely JL, Swan V, et al. Long-term effects of nasal continuous positive airway pressure therapy on cardiovascular outcomes in sleep apnea syndrome. *Chest* 2005;127(6):2076–84.
116. He J, Kryger MH, Zorick FJ, et al. Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. *Chest* 1988;94(1):9–14.
117. Ancoli-Israel S, Kripke DF, Klauber MR, et al. Morbidity, mortality and sleep-disordered breathing in community dwelling elderly. *Sleep* 1996;19(4):277–82.
118. Lindberg E, Janson C, Svardsudd K, et al. Increased mortality among sleepy snorers: a prospective population-based study. *Thorax* 1998;53(8):631–7.
119. Martin BC, Warram JH, Krolewski AS, et al. Role of glucose and insulin resistance in development of type 2 diabetes mellitus: results of a 25-year follow-up study. *Lancet* 1992;340(8825):925–9.
120. Punjabi NM, Beamer BA. Sleep apnea and metabolic dysfunction. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 1034–42.
121. Al-Delaimy WK, Manson JE, Willett WC, et al. Snoring as a risk factor for type II diabetes mellitus: a prospective study. *Am J Epidemiol* 2002;155(5):387–93.
122. Babu AR, Herdegen J, Fogelfeld L, et al. Type 2 diabetes, glycemic control, and continuous positive airway pressure in obstructive sleep apnea. *Arch Intern Med* 2005;165(4):447–52.
123. Phillips BG, Hisel TM, Kato M, et al. Recent weight gain in patients with newly diagnosed obstructive sleep apnea. *J Hypertens* 1999;17(9):1297–300.
124. Phillips BG, Kato M, Narkiewicz K, et al. Increases in leptin levels, sympathetic drive, and weight gain in obstructive sleep apnea. *Am J Physiol Heart Circ Physiol* 2000;279(1):H234–7.
125. Kalra M, Inge T, Garcia V, et al. Obstructive sleep apnea in extremely overweight adolescents undergoing bariatric surgery. *Obes Res* 2005;13(7):1175–9.
126. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev* 2002;6(2):97–111.
127. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-IV..* 4th edition. Washington, DC: American Psychiatric Association; 2000.
128. Ford ES, Cunningham TJ, Giles WH, et al. Trends in insomnia and excessive daytime sleepiness among U.S. adults from 2002 to 2012. *Sleep Med* 2015; 16(3):372–8.
129. Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. *Am J Psychiatry* 1997;154(10):1417–23.
130. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *J Am Med Assoc* 1989;262(11): 1479–84.
131. Edinger JD, Means MK. Overview of insomnia: definitions, epidemiology, differential diagnosis, and assessment. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 702–13.
132. Petrov ME, Lichstein KL, Baldwin CM. Prevalence of sleep disorders by sex and ethnicity among older adolescents and emerging adults: relations to daytime functioning, working memory and mental health. *J Adolesc* 2014;37(5):587–97.

133. Roberts RE, Roberts CR, Chan W. Ethnic differences in symptoms of insomnia among adolescents. *Sleep* 2006;29:359–65.
134. Shafazand S, Wallace DM, Vargas SS, et al. Sleep disordered breathing, insomnia symptoms, and sleep quality in a clinical cohort of US Hispanics in South Florida. *J Clin Sleep Med* 2012;8(5):507–14.
135. Slopen N, Williams DR. Discrimination, other psychosocial stressors, and self-reported sleep duration and difficulties. *Sleep* 2014;37(1):147–56.
136. Lewis TT, Troxel WM, Kravitz HM, et al. Chronic exposure to everyday discrimination and sleep in a multiethnic sample of middle-aged women. *Health Psychol* 2013;32(7):810–9.
137. Manber R, Steidtmann D, Chambers AS, et al. Factors associated with clinically significant insomnia among pregnant low-income Latinas. *J Womens Health (Larchmt)* 2013;22(8):694–701.
138. Perlis ML, Smith MT, Pigeon WR. Etiology and pathophysiology of insomnia. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 714–25.
139. Vgontzas AN, Bixler EO, Lin HM, et al. Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications. *J Clin Endocrinol Metab* 2001;86(8):3787–94.
140. Hansen AM, Thomsen JF, Kaergaard A, et al. Salivary cortisol and sleep problems among civil servants. *Psychoneuroendocrinology* 2012;37(7):1086–95.
141. Nofzinger EA, Buysse DJ, Germain A, et al. Alterations in regional cerebral glucose metabolism across waking and non-rapid eye movement sleep in depression. *Arch Gen Psychiatry* 2005;62(4):387–96.
142. Bazargan M, Yazdanshenas H, Gordon D, et al. Pain in community-dwelling elderly African Americans. *J Aging Health* 2016;28(3):403–25.
143. Hall Brown TS, Akeeb A, Mellman TA. The role of trauma type in the risk for insomnia. *J Clin Sleep Med* 2015;11(7):735–9.
144. Laugsand LE, Vatten LJ, Platou C, et al. Insomnia and the risk of acute myocardial infarction: a population study. *Circulation* 2011;124(19):2073–81.
145. Spiegelhalder K, Scholtes C, Riemann D. The association between insomnia and cardiovascular diseases. *Nat Sci Sleep* 2010;2:71–8.
146. Fernandez-Mendoza J, Vgontzas AN, Liao D, et al. Insomnia with objective short sleep duration and incident hypertension: the Penn State Cohort. *Hypertension* 2012;60(4):929–35.
147. Charniack EP, Ceron-Fuentes J, Florez H, et al. Influence of race and ethnicity on alternative medicine as a self-treatment preference for common medical conditions in a population of multi-ethnic urban elderly. *Complement Ther Clin Pract* 2008;14:116–23.
148. Reid KJ, Zee PC. Circadian disorders of the sleep-wake cycle. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier/Saunders; 2005. p. 691–701.
149. Weitzman ED, Czeisler CA, Coleman RM, et al. Delayed sleep phase syndrome. A chronobiological disorder with sleep-onset insomnia. *Arch Gen Psychiatry* 1981;38(7):737–46.
150. Regestein QR, Monk TH. Delayed sleep phase syndrome: a review of its clinical aspects. *Am J Psychiatry* 1995;152(4):602–8.
151. Ando K, Kripke DF, Ancoli-Israel S. Estimated prevalence of delayed and advanced sleep phase syndromes. *J Sleep Res* 1995;24:509.
152. Santhi N, Duffy JF, Horowitz TS, et al. Scheduling of sleep/darkness affects the circadian phase of night shift workers. *Neurosci Lett* 2005;384(3):316–20.

153. Pereira DS, Tufik S, Louzada FM, et al. Association of the length polymorphism in the human Per3 gene with the delayed sleep-phase syndrome: does latitude have an influence upon it? *Sleep* 2005;28(1):29–32.
154. Gronfier C, Wright KP Jr, Kronauer RE, et al. Efficacy of a single sequence of intermittent bright light pulses for delaying circadian phase in humans. *Am J Physiol Endocrinol Metab* 2004;287(1):174–81.
155. Czeisler CA, Richardson GS, Zimmerman JC, et al. Entrainment of human circadian rhythms by light-dark cycles: a reassessment. *Photochem Photobiol* 1981; 34(2):239–47.
156. Archer SN, Robilliard DL, Skene DJ, et al. A length polymorphism in the circadian clock gene Per3 is linked to delayed sleep phase syndrome and extreme diurnal preference. *Sleep* 2003;26(4):413–5.
157. Satoh K, Mishima K, Inoue Y, et al. Two pedigrees of familial advanced sleep phase syndrome in Japan. *Sleep* 2003;26(4):416–7.
158. Shiino Y, Nakajima S, Ozeki Y, et al. Mutation screening of the human period 2 gene in bipolar disorder. *Neurosci Lett* 2003;338(1):82–4.
159. Xu Y, Padiath QS, Shapiro RE, et al. Functional consequences of a CKIdelta mutation causing familial advanced sleep phase syndrome. *Nature* 2005; 434(7033):640–4.
160. Weyerbrock A, Timmer J, Hohagen F, et al. Effects of light and chronotherapy on human circadian rhythms in delayed sleep phase syndrome: cytokines, cortisol, growth hormone, and the sleep-wake cycle. *Biol Psychiatry* 1996;40(8):794–7.
161. Palmer CR, Kripke DF, Savage HC Jr, et al. Efficacy of enhanced evening light for advanced sleep phase syndrome. *Behav Sleep Med* 2003;1(4):213–26.
162. Lewy AJ, Ahmed S, Sack RL. Phase shifting the human circadian clock using melatonin. *Behav Brain Res* 1996;73(1–2):131–4.
163. Michaud M, Chabli A, Lavigne G, et al. Arm restlessness in patients with restless legs syndrome. *Mov Disord* 2000;15(2):289–93.
164. Montplaisir J, Boucher S, Poirier G, et al. Clinical, polysomnographic, and genetic characteristics of restless legs syndrome: a study of 133 patients diagnosed with new standard criteria. *Mov Disord* 1997;12(1):61–5.
165. Phillips B, Hening W, Britz P, et al. Prevalence and correlates of restless legs syndrome: 2 results from the 2005 National Sleep Foundation poll. *Chest* 2006;129(1):76–80.
166. Kryger MH, Otake K, Foerster J. Low body stores of iron and restless legs syndrome: a correctable cause of insomnia in adolescents and teenagers. *Sleep Med* 2002;3(2):127–32.
167. Nichols DA, Allen RP, Grauke JH, et al. Restless legs syndrome symptoms in primary care: a prevalence study. *Arch Intern Med* 2002;163(18):2323–9.
168. Lee KA, Zaffke ME, Baratte-Beebe K. Restless legs syndrome and sleep disturbance during pregnancy: the role of folate and iron. *J Womens Health Gend Based Med* 2001;10(4):335–41.
169. Chervin RD, Hedger AK, Dillon JE, et al. Associations between symptoms of inattention, hyperactivity, restless legs, and periodic leg movements. *Sleep* 2002;25(2):213–8.
170. Innes KE, Flack KL, Selfe TK, et al. Restless legs syndrome in an Appalachian primary care population: prevalence, demographic and lifestyle correlates, and burden. *J Clin Sleep Med* 2013;9:1065–75.
171. Szentkiralyi A, Fendrich K, Hoffmann W, et al. Socio-economic risk factors for incident restless legs syndrome in the general population. *J Sleep Res* 2012; 21:561–8.



172. Allen RP, Picchietti D, Hening WA, et al. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology. A report from the Restless Legs Syndrome Diagnosis and Epidemiology Workshop at the National Institutes of Health. *Sleep Med* 2003;4(2):101–19.
173. Winkelmann J, Muller-Myhsok B, Wittchen HU, et al. Complex segregation analysis of restless legs syndrome provides evidence for an autosomal dominant mode of inheritance in early age at onset families. *Ann Neurol* 2002;52(3):297–302.
174. Desautels A, Turecki G, Montplaisir J, et al. Identification of a major susceptibility locus for restless legs syndrome on chromosome 12q. *Am J Hum Genet* 2001;69(6):1266–70.
175. Bonati MT, Ferini-Strambi L, Aridon P, et al. Autosomal dominant restless legs syndrome maps on chromosome 14q. *Brain* 2003;126(6):1485–92.
176. Chen S, Ondo WG, Rao S, et al. Genomewide linkage scan identifies a novel susceptibility locus for restless legs syndrome on chromosome 9p. *Am J Hum Genet* 2004;74(5):876–85.
177. Silber MH, Richardson JW. Multiple blood donations associated with iron deficiency in patients with restless legs syndrome. *Mayo Clin Proc* 2003;78(1):52–4.
178. Connor JR, Boyer PJ, Menzies SL, et al. Neuropathological examination suggests impaired brain iron acquisition in restless legs syndrome. *Neurology* 2003;61(3):304–9.
179. Turjanski N, Lees AJ, Brooks DJ. Striatal dopaminergic function in restless legs syndrome: 18F-dopa and 11C-raclopride PET studies. *Neurology* 1999;52(5):932–7.
180. Winkelmann J, Schadrack J, Wetter TC, et al. Opioid and dopamine antagonist drug challenges in untreated restless legs syndrome patients. *Sleep Med* 2001;2(1):57–61.
181. Stiasny K, Wetter TC, Winkelmann J, et al. Long-term effects of pergolide in the treatment of restless legs syndrome. *Neurology* 2001;56(10):1399–402.
182. Winkelmann JW, Chertow GM, Lazarus JM. Restless legs syndrome in end-stage renal disease. *Am J Kidney Dis* 1996;28(3):372–8.
183. Unruh ML, Levey AS, D'Ambrosio C, et al. Restless legs symptoms among incident dialysis patients: association with lower quality of life and shorter survival. *Am J Kidney Dis* 2004;43(5):900–9.
184. Hening WA, Allen RP, Earley CJ, et al. Restless legs syndrome task force of the standards of practice committee of the American Academy of Sleep Medicine. An update on the dopaminergic treatment of restless legs syndrome and periodic limb movement disorder. *Sleep* 2004;27(3):560–83.
185. Gochfeld M. Occupational medicine practice in the United States since the industrial revolution. *J Occup Environ Med* 2005;47(2):115–31.
186. Healthy People 2020. Social Determinants of Health. Available at: <https://www.healthypeople.gov/2020/topics-objectives/topic/social-determinants-of-healthbook>. Accessed April 1, 2016.
187. Breyre A, Green-McKenzie J. A case of acute lead toxicity associated with Ayurvedic supplements. *British Medical Journal Case Reports*, in press. Available at: <http://casereports.bmj.com>. Accessed May 25, 2016.
188. Jones C, Shofer F, Duran M, et al. Assessment of medical student exposure to occupational and environmental medicine in medical school curriculum. Presented at: American Occupational Health Conference. Chicago (IL), April 11, 2016.



189. Hidden Tragedy: Underreporting of Workplace Injuries and Illnesses. US House of Representatives. Available at: <http://www.bls.gov/iif/laborcommreport061908.pdf>. Accessed May 15, 2016.
190. Won J, Dembe A. Services provided by family physicians for patients with occupational injuries and illnesses. *Ann Fam Med* 2006;4(2):138–47.
191. American College of Occupational and Environmental Medicine. The personal physician's role in helping patients with medical conditions stay at work or return to work. Available at: [http://www.acoem.org/PhysiciansRole\\_ReturntoWork.aspx](http://www.acoem.org/PhysiciansRole_ReturntoWork.aspx). Accessed November 1, 2012.
192. Politi BJ, Arena VC, Schwerha J, et al. Occupational medical history taking: how are today's physicians doing? A cross-sectional investigation of the frequency of occupational history taking by physicians in a major US teaching center. *J Occup Environ Med* 2004;46(6):550–5.
193. Kiselica D, Sibson B, Green-McKenzie J. Workers compensation: a historical review and description of a legal and social insurance system. *Clin Occup Environ Med* 2004;4(2):237–48.
194. Shinkman R. Getting underserved groups to seek health insurance. *Med Educ Online* 2015. <https://doi.org/10.3402/meo.v20.27535>.
195. VanderWielen L, Vanderbilt A, Crossman S. Health disparities and underserved populations: a potential solution, medical school partnerships with free clinics to improve curriculum. *Med Educ Online* 2015;20:27535.
196. Anthony D, El Rayess F, Esquibel AY, et al. Building a workforce of physicians to care for underserved patients. *R I Med J* (2013) 2014;97(9):31–5.
197. Vanichkachorn G, Roy B, Lopez R, et al. Evaluation and treatment of the acutely injured worker. *Am Fam Physician* 2014;89(1):17–24.
198. Vanichkachorn G, Green-McKenzie J, Emmett E. Occupational health care. *Family medicine: principles & practice*. 7th edition. New York: Springer; 2015.
199. Nadkarni M. The double whammy of chronic illness in underserved populations: can we afford not to care? New York: DHHS; 2004. Available at: <http://www.medscape.com/viewarticle/472495>. Accessed April 30, 2016.
200. US Department of Health and Human Services. Trends in the Health of Americans. Hyattsville (MD): US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics Office of Information Services; 2003.
201. Smedley B, Stith A, Nelson A. Unequal treatment. Confronting racial and ethnic disparities in healthcare. Washington, DC: The National Academies Press; 2003.
202. Preventing chronic disease: eliminating the leading preventable causes of premature death and disability in the United States. Available at: <http://www.cdc.gov/chronicdisease/pdf/preventing-chronic-disease-508.pdf>. Accessed May 25, 2016.
203. Nielson M, Olayiwola J, Grundy P, et al. The Patient-Centered Medical Home's Impact on cost & quality: an annual update of the evidence. Patient Centered Primary Care Collaborative 2012-2013. Available at: <http://www.milbank.org/uploads/documents/reports>. Accessed May 25, 2016.
204. Schulte P. Characterizing the burden of occupational injury and disease. *J Occup Environ Med* 2005;47(6):607–22.
205. Numbers: workplace injuries. U.S. Bureau of Labor Statistics. 2013. Available at: <http://www.ilo.org/global/standards/subjects-covered-by-international-labour-standards/occupational-safety-and-health/lang-en/index.htm>. Accessed March 1, 2016.

206. Corbin K. The 5 Most Injury-Prone Industries. Available at: <http://jobs.aol.com/articles/2011/12/08/the-5-most-injury-prone-industries/Schools.com>. Accessed May 25, 2016.
207. Walter L. OSHA's role in reducing occupational injuries, fatalities. Available at: <http://ehstoday.com/osha/osha-s-role-reducing-occupational-injuries-fatalities-infographic>. Accessed May 15, 2016.
208. OSHA fact Sheet. Available at: [https://www.osha.gov/OshDoc/data\\_General\\_Facts/factsheet-inspections.pdf](https://www.osha.gov/OshDoc/data_General_Facts/factsheet-inspections.pdf). Accessed May 15, 2016.
209. National Occupational Safety and Health Administration. About NORA... Partnerships, Research and Practice. Available at: <http://www.cdc.gov/niosh/nora/about.html>. Accessed May 15, 2016.
210. Centers for Disease Control. National Occupational Safety and Health Administration. NORA Sector Agendas. Available at: <http://www.cdc.gov/niosh/nora/comment/agendas/>. Accessed May 15, 2016.
211. Chou C-F, Johnson PJ, Ward A, et al. Health care coverage and the health care industry. *Am J Public Health* 2009;99(12):2282–8.
212. Ward BW, Clarke TC, Freeman G. et al. National Health Interview Survey. Division of Health Interview Statistics, National Center for Health Statistics. Available at: <http://www.cdc.gov/nchs/data/nhis/earlyrelease/earlyrelease201506.pdf>. Accessed March 30, 2016.
213. Johnson PJ, Blewett LA, Ruggles S, et al. Four decades of population health data: the integrated health interview series. *Epidemiology* 2008;19:872–5.
214. Occupational Hazards in Home health care. NIOSH Health and Safety Topics. Available at: [https://www.osha.gov/SLTC/home\\_healthcare/#](https://www.osha.gov/SLTC/home_healthcare/#). Accessed March 1, 2016.
215. Green-McKenzie J, McCarthy R, Shofer F. Characterisation of occupational blood and body fluid exposures beyond the Needlestick Safety and Prevention Act. *J Infect Prev* 2016;17(5):226–32. Available at: <http://bjj.sagepub.com/content/early/2016/04/29/1757177416645339.abstract>. Accessed May 20, 2016.
216. McLellan R, Sherman B, Loeppke R, et al. Optimizing health care delivery by integrating workplaces, homes and communities: how occupational and environmental medicine can serve as a vital connecting link between accountable care organizations and the patient-centered medical home. *J Occup Environ Med* 2012;54(4):504–12.
217. Ruotsalainen JH, Verbeek JH, Mariné A, et al. Preventing occupational stress in healthcare workers. *Cochrane Database Syst Rev* 2015;(4). CD002892. Available at: [http://www.cochrane.org/CD002892/OCCHEALTH\\_preventing-occupational-stress-in-healthcare-workers](http://www.cochrane.org/CD002892/OCCHEALTH_preventing-occupational-stress-in-healthcare-workers).
218. Occupational Hazards in Home health care. NIOSH health and safety topics. Available at: <http://www.cdc.gov/niosh/docs/2010-125/pdfs/2010-125.pdf>; [https://www.osha.gov/SLTC/home\\_healthcare/#](https://www.osha.gov/SLTC/home_healthcare/#). Accessed March 1, 2016.
219. Buchanan S, Vossenas P, Krause N, et al. Occupational injury disparities in the US hotel industry. *Am J Ind Med* 2010;53:116–25.
220. Singer A. Immigrant workers in the U.S. Labor Force. Washington, DC: Brookings; 2012. Available at: [http://www.brookings.edu/~media/research/files/papers/2012/3/15%20immigrant%20workers%20singer/0315\\_immigrant\\_workers\\_singer.pdf](http://www.brookings.edu/~media/research/files/papers/2012/3/15%20immigrant%20workers%20singer/0315_immigrant_workers_singer.pdf).
221. Chou C-F, Johnson PJ, Ward A, et al. Occupational outlook handbook, maids and housekeeping cleaners: pay. Washington, DC: Bureau of Labor Statistics; 2013. Available at: <http://www.bls.gov/ooh/building-and-grounds-cleaning/maids-and-housekeeping-cleaners.htm#tab-5>.

222. Sanon MA. Agency-hired hotel housekeepers: an at-risk group for adverse health outcomes. *Workplace Health Saf* 2014;62(2):86.
223. Knox A. Lost in translation: an analysis of temporary work agency employment in hotels. *Work Employ Soc* 2010;24:449–67.
224. Wells MJ. Unionization and immigrant incorporation in San Francisco hotels. *Soc Probl* 2000;47:241–65.
225. Underserved Workers Gain Access to UCSF OEM Specialists. Available at: <http://coeh.berkeley.edu/bridges/Summer2013/UnderservedWorkers.html>. Accessed March 1, 2016.
226. Legal Consciousness of Undocumented Latinos: Fear and Stigma as Barriers to Claims-Making for First- and 1.5-Generation Immigrants. Law and Society Review. Available at: <http://plone3.sscnet.ucla.edu:8080/chavez/chavez/people-faculty-and-staff/core-faculty-1/faculty-files/LegalConsciousnessofUndocumentedLatinos.pdf>. Accessed May 29, 2016.
227. American College of Occupational and Environmental Medicine Whitepaper. The Health of Immigrant Workers in the US. Available at: [https://www.acoem.org/uploadedFiles/About\\_ACOEM/Components\\_And\\_Sections/Section\\_Home\\_Pages/White%20Paper%20-%20THE%20HEALTH%20STATUS%20OF%20IMMI GRANT%20WORKERS%20IN%20THE%20US%205-1-2012.pdf](https://www.acoem.org/uploadedFiles/About_ACOEM/Components_And_Sections/Section_Home_Pages/White%20Paper%20-%20THE%20HEALTH%20STATUS%20OF%20IMMI GRANT%20WORKERS%20IN%20THE%20US%205-1-2012.pdf). Accessed May 1, 2012.
228. Leigh J, Macaskill P, Kuosma E, et al. Global burden of disease and injury due to occupational factors. *Epidemiology* 1999;10(5):626–31.
229. Nelson D, Concha-Barrientos M, Driscoll T, et al. The global burden of selected occupational disease and injury risks: methodology and summary. *Am J Ind Med* 2005;48:400–18.
230. International Labour Standards on Occupational Safety and Health. Available at: <http://www.ilo.org/global/standards/subjects-covered-by-international-labour-standards/occupational-safety-and-health/lang-en/index.htm>. Accessed March 30, 2016.
231. Concha-Barrientos M, Nelson D, Fingerhut M, et al. Global burden due to occupational injury. *Am J Ind Med* 2005;48(6):470–81.
232. Global Strategy on Occupational Safety and Health. Conclusions adopted by the International Labor Conference at its 91st Session. International Labour Organization 2994. 2003. Available at: [http://www.ilo.org/safework/info/policy-documents/WCMS\\_107535/lang-en/index.htm](http://www.ilo.org/safework/info/policy-documents/WCMS_107535/lang-en/index.htm). Accessed May 23, 2016.
233. Plan of action (2010-2016) to achieve widespread ratification and effective implementation of the occupational safety and health instruments. Available at: [http://www.ilo.org/global/standards/WCMS\\_125616/lang-en/index.htm](http://www.ilo.org/global/standards/WCMS_125616/lang-en/index.htm). Accessed June 1, 2016.
234. Occupational Safety and Health Administration. 1970. Available at: [http://www.osha.gov/pls/oshaweb/owadisp.show\\_document?p\\_table=OSHACT&p\\_id=2743](http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=OSHACT&p_id=2743). Accessed May 1, 2016.
235. Canadian Center for Occupational Safety and Health Legislation: Due Diligence. 2015. Available at: <http://www.ccohs.ca/topics/legislation/duediligence>. Accessed May 1, 2016.
236. UK Legislation, 1974 UK Legislation (1974) Health and Safety at Work etc. Act. c. 37 Part I General duties Section 2. Available at: <http://www.legislation.gov.uk/ukpga/1974/37/section/2>. Accessed May 1, 2016.
237. Office of Disease Prevention and Health Promotion. Healthy People 2020. Available at: <https://www.healthypeople.gov>. Accessed April 1, 2016.