Causes and consequences of sleep deprivation in hospitalised patients


Abstract
Sleep is a fundamental component of good health, however its promotion in acute hospital settings does not appear to be a priority. This literature review, which considered qualitative and quantitative research methodology, aimed to determine the factors that affect the quality of sleep experienced by patients in hospital, and the effects of sleep deprivation on the health and wellbeing of these individuals. Causes of sleep disruption are varied and include environmental and bio-cognitive factors, including pain, bright light, noise, anxiety and stress. The environmental and bio-cognitive consequences of sleep deprivation on the health and recovery of hospital inpatients are outlined.

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Keywords
Carbohydrate metabolism, cognitive performance, pain, patient recovery, sleep deprivation

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Literature search
A focused question was devised using the foundations of the PICO (Population (patient or...
A review of academic journals (Journal of Advanced Nursing and Sleep) and online databases (Cumulative Index to Nursing and Allied Health Literature (CINAHL), GoPubMed, Wiley Online Library and Science Direct) was undertaken to identify relevant qualitative and quantitative articles (Holloway and Wheeler 2002). In addition, articles were searched using Medical Subject Headings (MeSH) to improve the quality of returned hits. The MeSH terms included were sleep deprivation, dyssomnias, wakefulness and health (United States National Library of Medicine 2012). Inclusion and exclusion criteria were then applied to produce more relevant results (Box 1).

Critical appraisal of the literature

The articles identified by the literature search formed the basis of the main themes: the environmental and bio-cognitive causes of sleep deprivation and the bio-cognitive consequences of sleep deprivation in hospital. The articles were critically appraised to ensure they were valid, credible and reliable. The Critical Appraisal Skills Programme (CASP) tool for qualitative and quantitative data (Public Health Resource Unit 2006a, 2006b) was used to allow analysis of the strengths and limitations of the articles, while observing the appropriateness of the methodology and transferability of results (Polit and Beck 2010). Any articles that did not meet the requirements of the CASP tool were discarded and not used within this literature review.

A total of 64 articles (36 quantitative and 28 qualitative studies) were reviewed and included.

Environmental and bio-cognitive causes of sleep deprivation in hospital

Environmental causes

Environmental stimuli are a source of sleep deprivation, with critically ill patients receiving up to 60 interruptions per night (Freedman et al 2001). Therapeutic and diagnostic procedures, noise and light continually arouse patients from their sleep cycle and contribute to these interruptions (Krachman et al 1995, Gabor et al 2003, Dogan et al 2005, Friese 2008, Kamdar et al 2012, Ehlers et al 2013). However, these procedures and actions are essential owing to the physiologically unstable condition of the patient (Kamdar et al 2012).
Extraneous noise is a major source of environmental stimuli and includes staff conversations, telephones and televisions (World Health Organization (WHO) Regional Office for Europe 2009, Kamdar et al. 2012). Missildine (2008) highlighted that environmental noise in a hospital environment at night exceeded the WHO Regional Office for Europe’s (2009) recommendation of 30dB, creating an environment which is not conducive to good sleep. In addition, disturbances to sleep caused by noise creates fragmented sleep, even if the noise is registered subconsciously, with long sleep latency (length of time taken to fall asleep) (WHO Regional Office for Europe 2009). Critically ill patients are also more sensitive to environmental noise, emphasising the need for a quieter environment at night (Kamdar et al. 2012).

Freedman et al. (2001) highlighted that environmental noise was only responsible for 17% of awakenings. The literature search identified that light is another environmental factor that may be more prevalent than noise in disrupting sleep (Gabor et al. 2003, Salas and Gamaldo 2008).

The sleep-wake cycle is influenced by melatonin levels that synchronise with the circadian rhythm, and is inhibited by light exposure (Friese 2008, Salas and Gamaldo 2008, Kamdar et al. 2012). Patients treated in acute care settings are frequently exposed to bright light, affecting melatonin levels and the circadian rhythm by shifting it later, depending on the timing and intensity of light experienced (Stepanski and Wyatt 2003, Friese 2008, Kamdar et al. 2012). This desynchronisation of the circadian rhythm and sleep-wake cycle can create disrupted and fragmented sleep, thereby contributing to sleep deprivation (Krachman et al. 1995, Gabor et al. 2003, Stepanski and Wyatt 2003, Weinhouse and Schwab 2006).

Missildine (2008) controlled light exposure through the implementation of a night-day cycle of light, enabling patients to have a synchronised circadian rhythm, thereby promoting a better sleeping environment.

Pain
Acute and chronic pain are a source of sleep deprivation (Roehrs et al 2006, Cole and Richards 2007, Kamdar et al. 2012), with alterations in sleep architecture observed (Lautenbacher et al. 2006, Salas and Gamaldo 2008).

Moldofsky et al. (1975) made reference to a self-perpetuating cycle in which sleep deprivation and pain have an adverse effect on each other. In this study, participants underwent stage 4 sleep deprivation (Figure 1), and the results demonstrated that pain became more noticeable and intense (hyperalgesic state). This finding is cited elsewhere (Redeker 2000, Smith and Haythornthwaite 2004, Lautenbacher et al. 2006, Roehrs et al. 2006, Cole and Richards 2007).

In particular, short wave sleep deprivation is positively correlated with pain severity, therefore increasing periods of arousal and awakening during sleep (Smith and Haythornthwaite 2004, Weinhouse and Schwab 2006).

The self-perpetuating cycle of sleep deprivation and pain was evident in a study of patients with burns (Raymond et al. 2001). Poor sleep quality and increased nocturnal awakenings became a predictor for pain the following morning and pain during therapeutic procedures. In other words, poor sleep is linked to higher pain intensity during the day (Raymond et al. 2001). Palesh et al. (2007) reported that when pain becomes more severe at night, worsening problems with sleep latency and continuity may be experienced, placing individuals at risk of increased pain. Patients experiencing sleep deprivation may become hyperalgesic (extremely sensitive to pain) (Salas and Gamaldo 2008, Lee et al. 2009).

Hamilton et al. (2007) suggested that the more poor quality sleep is experienced, the more reactive an individual becomes to momentary increases in pain levels, and that sleep deprivation may be influential in the physiological adjustment to pain. Other senses may be affected by sleep deprivation, but only the hyperalgesic effect was discussed in the literature (Roehrs et al. 2006).

Empirical knowledge about how pain and sleep are related on a physiological level was not addressed in the literature. It has been suggested that sleep and pain may use common neurotransmitters, providing a potential neurobiological pathway for pain and sleep problems (Menefee et al. 2000). Moreover, it was found that poor sleep influenced the attention given to pain during the day (Smith and Haythornthwaite 2004). This enhances the self-perpetuating cycle of sleep deprivation and pain (Moldofsky et al. 1975, Raymond et al. 2001), suggesting that lack of quality sleep is linked to a psychological process that influences the perception of physical symptoms such as pain (Smith and Haythornthwaite 2004).

Anxiety and stress
Feelings of anxiety and stress increase sympathetic stimulation and consequently increase sleep arousal levels, thus disrupting sleep architecture (Kamdar et al. 2012). Sleep deprivation experienced during hospital admission has been found to be a prominent source of stress and anxiety for patients (Menefee et al. 2000, Kamdar et al. 2012). This may stem from an unfamiliar environment, worry about illness severity and low mood (Moldofsky et al. 2012).
Art & science literature review


The response and release of cortisol when stress is experienced is modulated by the hypothalamic-pituitary-adrenal axis (Hardin 2009, Benham 2010, Ganz 2012). When an individual is experiencing short wave sleep, hypothalamic-pituitary-adrenal activity is inhibited. The release of glucocorticoids in response to stress stimulates hypothalamic-pituitary-adrenal activity, affecting the ability to sleep and increasing the risk of sleep deprivation (Hardin 2009, Benham 2010). Patients in hospital are more vulnerable to the effects of stress because they are already experiencing biological processes such as pain or infection, and are therefore more likely to experience sleep deprivation (Topf 2000, Hankin and Abela 2005).

Van Onselen et al (2010) categorised patients according to their anxiety level during hospital admission and found that those with lower levels reported the least sleep deprivation. In another study, patients with a long-term condition who experienced anxiety during hospitalisation found it more difficult to fall asleep and had less stage 3 and stage 4 sleep (Edéll-Gustafsson 2002) (Figure 1). The consequences of stress and anxiety are evident in what has been termed the ‘first night’ effect, whereby emotions caused by hospitalisation and being in a strange environment expose an individual to sleep deprivation (Missildine 2008). Furthermore, Missildine (2008) identified that sleep efficiency did not improve after the first night, suggesting that many patients are continually sleep deprived throughout their stay in hospital.

Patients who remain awake or experience difficulty sleeping are exposed to stressful input as a result of perceiving or presenting their problems as considerably worse than they are (Meerlo et al 2008, Ehlers et al 2013). This increases hypothalamic-pituitary-adrenal activity to beyond the level of wakefulness, creating a more hostile internal environment for sleep (Meerlo et al 2008, WHO Regional Office for Europe 2009). Moreover, patients who are cared for in a critical care environment can experience higher stress and anxiety levels as they usually do not have time to prepare for their admission and find themselves heavily sedated, unable to move, and with unfamiliar people carrying out intimate and invasive procedures (Çelik et al 2005).

Bio-cognitive consequences of sleep deprivation in hospital

Sleep is identified as an essential biological function that supports homeostatic systems. Therefore, sleep deprivation has bio-cognitive consequences (Walker 2008, Mullington et al 2009, WHO Regional Office for Europe 2009).

Immune function

Sleep deprivation can result in altered immune function, thereby increasing an individual's susceptibility to illness (Benca and Quintas 1997, Weinhouse and Schwab 2006, WHO Regional Office for Europe 2009). Short wave sleep deprivation has been positively correlated to slower recovery from illness (WHO Regional Office for Europe 2009). This is highlighted by findings from a study by Cohen et al (2009), who identified an increased risk of contracting the common cold after receiving less than seven hours of sleep per night. However, the literature review surrounding the topic of sleep deprivation and susceptibility to illness identified a paucity of studies that were relevant and reliable, and met the inclusion criteria.

Inflammation

Sleep deprivation places the body in a perceived state of inflammation by increasing the secretion of inflammatory markers, thereby heightening the immune response and stimulating the stress response (Irwin et al 2006, Friese 2008, Cohen et al 2009, WHO Regional Office for Europe 2009, Kamdar et al 2012). This was demonstrated by McEwen (2006) who observed an increase in pro-inflammatory cytokine levels after sleep was restricted to four hours. These cytokines were released in response to activated immune cells involved in the amplification of the inflammatory process. Benca and Quintas (1997) identified that sleep deprivation has an indirect effect on immune defence. They stated that sleep deprivation causes significant impairment to the central nervous system and that this triggers a cascade of effects on physiological processes, affecting the regulation of the quality and quantity of immune responses.

In addition, glucocorticoids are activated by the release of pro-inflammatory cytokines, causing a physiological response to stress while being a component in circadian rhythm regulation (McEwen 2006, Meerlo et al 2008, WHO Regional Office for Europe 2009). Therefore, elevated levels of glucocorticoids caused by sleep deprivation can affect the patient’s circadian rhythm and intensify the stress response (Mullington et al 2009, WHO Regional Office for Europe 2009, Kamdar et al 2012). Meerlo et al (2008) reported that this stress system activation affects brain functioning and metabolic processes, therefore negatively affecting health.

Elevated glucocorticoid levels caused by sleep deprivation appear small, but when sleep
deprivation is continual, the small but recurrent elevations may combine to become a significant load, ultimately shifting the brain from adaptation to disease (Meerlo et al 2008). It is also noted that curtailing sleep may have cardiovascular consequences; for example, women sleeping less than five hours a night have a 39% increased risk of developing coronary heart disease (Ayas et al 2003).

**Parasympathetic and sympathetic equilibrium**

The literature review identified that one of the significant physiological systems affected by sleep deprivation is parasympathetic and sympathetic equilibrium. It has been shown that when non-REM sleep is fragmented or not achieved, the sympathetic system remains stimulated, maintaining blood pressure, heart rate and systemic vascular resistance at a higher level (Mullington et al 2009, WHO Regional Office for Europe 2009, Kamdar et al 2012). This increased sympathetic system stimulation is also seen during other times of stress, and the increase in blood pressure may lead to endothelial shear, adding to the inflammatory markers produced by cells (Banks and Dinges 2007, Mullington et al 2009). Furthermore, sleep deprivation affects several metabolic and endocrine functions, with negative alterations mimicking vital factors of ageing and possibly increasing the severity of age-related chronic disorders (Spiegel et al 1999).

Sleep deprivation can be especially dangerous in critically ill patients, as abrupt biological fluctuations can affect homeostasis, thus altering the haemodynamics and immune mechanisms in already physiologically comprised individuals (Kamdar et al 2012).

**Carbohydrate metabolism**

The literature suggests that recurrent sleep deprivation may be a potential contributor to the development of insulin resistance, with detrimental effects on carbohydrate metabolism. The study by Spiegel et al (1999) is a significant point of reference with regard to the relationship between sleep deprivation and carbohydrate metabolism. This study measured carbohydrate metabolism after recurrent sleep restriction and discovered that, when participants had only four hours sleep a night, there was clear impairment of carbohydrate tolerance. Glucose clearance was 40% slower in participants than in non-sleep deprived individuals, which is equivalent to that in a person with type 2 diabetes (Spiegel et al 1999).

It is suggested that the decrease in insulin response to glucose clearance could be related to sympathetic and parasympathetic control of the pancreas, since parasympathetic stimulation releases insulin (Spiegel et al 2005). Parasympathetic balance is disrupted during sleep deprivation and less insulin is likely to be released (Spiegel et al 2005). Decreased carbohydrate tolerance and increased sympathetic tone are identified as significant risk factors for the development of insulin resistance, obesity and hypertension, all of which can lead to the development of type 2 diabetes (Spiegel et al 1999, Banks and Dinges 2007, Van Cauter et al 2008, WHO Regional Office for Europe 2009).

**Cognitive performance**

It has been suggested that sleep deprivation influences aspects of cognitive performance, thus affecting patients’ quality of life (Friesen 2008). Behavioural alertness and tasks requiring vigilance are adversely affected by sleep loss (Pilcher and Huffcutt 1996, Belenky et al 2003, Van Dongen et al 2003, Banks and Dinges 2007, WHO Regional Office for Europe 2009). Furthermore, sleep loss leads to a significant reduction in energy levels, impairing physical recovery from illness and potentially affecting mobilisation and rehabilitation (WHO Regional Office for Europe 2009, Kamdar et al 2012).

During periods of sleep deprivation, cognitive alertness declines and individuals appear markedly less alert (Banks and Dinges 2007), with performance levels stabilising at a reduced level (Belenky et al 2003). Spiegel et al (1999) reported that sleep duration restricted to four hours per night reflects a decrease in brain activity through a reduction of cerebral use of glucose, contributing to a decline in alertness and performance. Therefore, because neurobiological processes are affected, the ability to make decisions and carry out usual behaviour becomes more difficult (Banks and Dinges 2007, Stone et al 2008a, WHO Regional Office for Europe 2009, Lim and Dinges 2010).

Sleep disruption is implicated in the incidence of falls as a result of imperfect balance or reduction in reaction time (Stone 2008a, WHO Regional Office for Europe 2009) caused by impaired cognitive performance (Department of Health (DH) 2001, Minkel et al 2012). This poses a major health risk for older patients, and in particular those who have slept for less than five hours (Stone et al 2008a, 2008b). Additional time and assistance may be required during physiotherapy and occupational therapy as a result of reduced cognitive ability, therefore hindering timely discharge (DH 2004, Salas and Gamaldo 2008, Walker 2008).
Discussion
From the breadth and depth of the reviewed literature, and based on the application of inclusion and exclusion criteria, articles that were relevant and appropriate to the subject were selected. The quality of journal articles returned by the search varied, as the quality and quantity of results differed between studies reviewed.

Many of the cohort studies had small sample sizes, therefore affecting the transferability and reliability of the findings (Newell and Burnard 2011). However, carrying out cohort studies that use the gold standard of polysomnography (Bourne et al 2007) to measure quantitatively the amount of sleep received by participants would be expensive and time consuming on a large scale. The mix of qualitative and quantitative studies reviewed produced numerous findings and individual viewpoints, adding depth and understanding to the literature review.

The review highlighted that sleep deprivation can be caused and influenced by many factors in the hospital environment. Pain, the environment, stress and anxiety were important factors in sleep deprivation experienced by patients. Noise and light were identified as the most disruptive causes of sleep deprivation (Freedman et al 2001, Gabor et al 2003, Doğan et al 2005, Friese 2008, Kamdar et al 2012). In addition, patients experiencing pain tend to have stage 4 sleep deprivation, which contributes to a hyperalgesic state, creating a self-perpetuating cycle in which sleep deprivation and pain have a negative effect on each other (Moldofsky et al 1975, Redeker 2000, Smith and Haythornthwaite 2004, Lautenbacher et al 2006, Roehrs et al 2006). Stress and anxiety have a negative effect on sleep patterns, with increased sleep latency and a reduction in stage 3 and stage 4 sleep. Moreover, when patients cannot sleep they expose themselves to stressful thoughts, increasing their arousal state to that above a wakeful level, further exacerbating negative mood (Menefee et al 2000, Meerlo et al 2008, Sharra and Allen 2009, Kamdar et al 2012).

Bio-cognitive systems that are disrupted and altered by sleep deprivation have a negative effect on health (Walker 2008, Mullington et al 2009, WHO Regional Office for Europe 2009). The literature established that sleep deprivation affects several metabolic and endocrine functions, the most notable being amplification of the immune system, and disruption of the sympathetic and parasympathetic equilibrium (Benca and Quintas 1997, McEwen 2006, Weinhouse and Schwab 2006, Kamdar et al 2012). Sleep deprivation also creates disturbance in the metabolic and endocrine systems and, when combined with sympathetic stimulation, is linked to a change in carbohydrate metabolism and insulin resistance, indicating a possible link with the development of type 2 diabetes (Spiegel et al 1999, McEwen 2006, Suarez 2008, Van Cauter et al 2008, Mullington et al 2009). Moreover, sleep deprivation can alter cognitive function, which has a negative effect on the rehabilitation of patients (Banks and Dinges 2007). The ability to make decisions and carry out daily activities is also affected, leading to an increase in falls and accidents, especially among older patients (Banks and Dinges 2007, Stone et al 2008a, WHO Regional Office for Europe 2009).

This literature review highlighted a lack of information on the sleep hygiene habits of patients during hospitalisation. Therefore, a possible area for further research could include the benefits of a sleep hygiene routine and the effect this has on sleep deprivation experienced by patients in hospital.

Recommendations
Sleep deprivation should be a major cause for concern for healthcare professionals, and change needs to be implemented at ward level. The literature review demonstrated that patients during both short and long-term hospital stays are at risk of sleep deprivation and associated negative health outcomes.

Patients require good quality sleep to protect their health and wellbeing. Reducing the effect of environmental factors, in particular focusing on behaviour modification to reduce excess noise and light, may be the most effective strategy for the majority of patients. This is because many of the contributors to sleep deprivation present in the environment are amenable to change (Kahn et al 1998). Reducing the effect of environmental factors may be achieved by ensuring the main lights are switched off at an acceptable and regular time every night, with dimmer bedside lights being used if required during the night. Monsén and Edéll-Gustafsson (2005) suggested that environmental noise be kept to a minimum by reducing the level of staff conversations and the volume of the telephone ring sound, and limiting unnecessary nursing interventions to enable patients to have fewer disruptions at night.

Conclusion
The literature review aimed to determine the factors that affect the level of sleep experienced by patients in hospital and the effects of sleep deprivation on the health and wellbeing of these
individuals. Causes of sleep disruption included environmental and physiological factors. For sleep-deprived patients, there were bio-physiological consequences for their health and recovery, with particular effects on immune functioning, inflammation, parasympathetic equilibrium, carbohydrate metabolism and cognitive performance. Nurses should endeavour to ensure that patients in their care receive adequate pain management and reassurance to reduce anxiety and stress. They should also aim to reduce environmental factors such as noise and light, thereby helping to minimise the negative effects of sleep deprivation.

References


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