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Insomnia Affected by Infection: A Case Study

Kathy Sexton-Radek*

Suburban Pulmonary and Sleep Associates/Elmhurst College, Concordia University, USA

***Corresponding author:** Kathy Sexton-Radek, PhD, C.BS, Suburban Pulmonary and Sleep Associates/Elmhurst College, Concordia University, USA, Tel: 708-442-8946; E-mail: ksrsleep@aol.com

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Abstract

An infection causes changes in immune functioning that is commonly known to impact sleep quality; patients report poor sleep when ill. A case study is described as means of reporting the association of infection influencing sleep quality. Sleep log data was collected and is reported as it reflects changes in sleep quality during the course of an infection.

Keywords: Sleep; Infection; Bacterial; Infection; Viral; Extended sleep; Sleep architecture

Insomnia Affected by Infection: A Case Study

Sleep difficulties are prominent in young adults. With social obligations and choices, the bedtimes and consequent amount of sleep becomes compromised. It is estimated that some 10% of young adults have sleep disturbances with insomnia most commonly.

The new found independence that young adults experience once at college and/or as they launch, out of the family home to work and an independent living. Multitasking of media maintenance, school and work schedules and social activities are commonly reported activities of young adults. Poor sleep in the forms of insomnia (at sleep onset or to stay asleep); fragmented sleep and resultant sleep deprivation often result in variable sleep schedules and futile attempts to recoup their sleep on the weekends [1]. It is this sleep deprivation that jeopardizes the health of the young adult. Cold and flu are reported by 7% of young adults [2]. The complexity of the immune system in its generation of cells and proteins (i.e., cytokines) to combat bacteria and viruses is compromised with poor sleep [3,4]. The suppression of the immune system from sleep loss leads to reductions in the development of antibodies, T cells, and cytokines. Infectious diseases may accompanying alterations in sleep as the immune system functioning is compromised given the fragmented sleep of insomnia (i.e., reduced opportunity for deep sleep when immune system functioning accelerates) [1,4].

Sleep is determined by sleep propensity (e.g., delta wave power) and circadian rhythm timing [5,6]. During sleep, metabolic rates are decreased with muscular inactivity. It is believed that sleep helps to conserve metabolic energy [7]. During states of high temperature, patients with an infection have a higher energy requirement. This altered arousal occurs during infection [8]. The central nervous system regulation of immune responses is driven by signaling path wags: activation of Hypothalamic Pituitary Adrenal (HPA) axis and the sympathetic nervous system.

Results from animal studies, enhanced SWS levels have been measured with microbial challenges [9].

Patient Studies. In 1930, von Economic studied lesions in the central nervous system and identified sleep as an active process [10]. More recent research has addressed this to the measurement of chronic insomnia and reduced life expectancy [10].

Initially sleep (SWS) is increased during infection as interleukin-I (IL-I) and its pyrogenic effects occur [4]. This requires metabolic energy and slow waves keep ensues [4,8,9]. After a number of hours with untreated infection, the protective effects of IL-I, release of additional interleukins (e.g., IL-M, IL-G) that require less metabolic energy and subsequently sleep becomes fragmented and fitful. Sleep is then considered an index of general well-being; less severely ill sleep better REM is consistently reduced; initial increases in SWS then a subsequent decrease in the amount of time in SWS occurs with illness [10,11]. In patient with HIV develop to AIDS, a severe reduction in SWS, as well as fragmentation in sleep. Human volunteers given rhinovirus, influenza or both slept less during incubation period and longer during the incubation period. Prion-related conditions such as Creutfeld-Jacobs and Fatal Familial Insomnia have increased SWS time and reduced wakefulness in these neurodegenerative disorders. The *T. rhodesiense* carried by the African tsetse fly induces sleepiness that results in death [11-15].

A clinic case of a 25 year old male presenting with a sleep insomnia that started six months after his recovery from head injury is now presented as it illustrates the infection and sleep association.

High fever for 24 hours
Sweats and chills for 24 hours
Nasal congestion
Sinus pain and headache
Sore throat from post nasal drip
Aches and pains
Earache (left ear)

Table 1: Patient Complaints during Infection.

Table 1 presents the infection complaints of the patients. The insomnia took the form of thirty – one hundred twenty minute sleep

onset latencies approximately four – five times a week. Additionally, he napped mid-morning for thirty to forty minutes nearly every day. A treatment course of Cognitive Behavior Therapy (CBT) was started with the patient to address his insomnia complaints. At week three the patient became ill. The patient did not begin medical care (i.e., prescription of antibiotic) right away. The anticipated changes to his sleep of greater sleep efficiency and reduction/elimination of daytime sleepiness were affected by the infection (diagnosed as sinusitis) [15]. Average sleep efficiencies at baseline was 62%, after treatment week 4 at 75% and at follow-up at 85% (i.e., Sleep efficiency= (total number of minutes sleep)/ (total number of minutes in bed) X 100).

Clinical Impressions and Conclusions

Young adult sleep quality is poor, largely due to the academic, vocational and social schedule/activities that are kept. These multitasking schedules perturb opportunities for regular bedtime and sleep durations [1]. The resultant poor sleep, so commonly experiences by young adults, occurs at time when sleep patterns are maturing; thus, the young adults poor sleep makes them susceptible to continued and worsened poor sleep. Marked changes in the patient's sleep quality occurred the presumed onset of their infection. This period is followed by an improvement in sleep, then, a worsening in sleep quality.

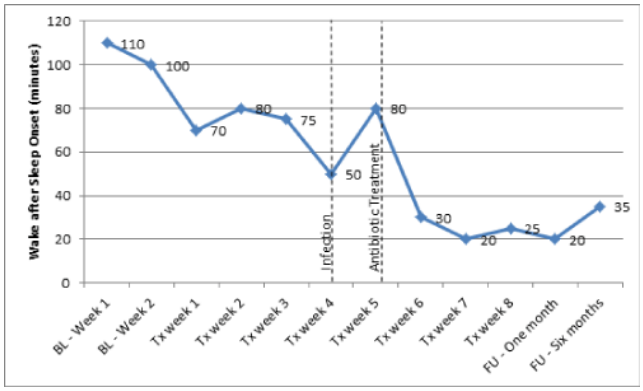

Figure 1: Wake after Sleep Onset, as measured via weekly sleep logs.

In Figure 1, wake time after sleep onset improved from 115 minutes at baseline week 1 to 70 minutes in Cognitive Behavior Therapy treatment week 1.As anticipated, after four session of CBT treatment, his reported wake after sleep onset to 50 minutes when his infection started.This time period was followed by a worsening of sleep—reportedly when his infection worsened. In Figure 1, the patient's improvement in sleep or increased consolidation in sleep interval reflects improvement from 80 to 30 minutes of wake after sleep onset. The patient's temperature data was not available; thus, the impact of CBT therapy and perhaps improvement in immune functioning to combat the infection given the antibiotic medication. The patient reported satisfaction with the CBT and stated that he felt better. With cases such as these, an extended time of measurement would have been optimal and the tracking of infection and it remittance as it corresponded to sleep behavior would allow for more exacting conclusions. In future clinical work where these elements become relevant, such measurements will be planned.

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