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The role of sleep in the regulation of body weight

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ABSTRACT

Sleep participates in the regulation of body weight. The amount of sleep and synchronization of the biological clock are both necessary to achieve the energy balance and the secretion of hormones that contribute to weight regulation. In this review, we first reconsider what normal physiological sleep is and what the normative values of sleep are in the general population. Second, we explain how the biological clock regulates the hormones that may be involved in weight control. Third, we provide some recent data on how sleep may be disturbed by sleep disorders or reduced by sleep debt with consequences on weight. Finally, we explore the relationships between sleep debt and obesity.

1. Introduction

Having a regular good night of sleep is recommended as one of the major requirements for good health in children, but also in adults (WHO, 2005; NIH, 2015). Sleep has a crucial role in many somatic, cognitive, and psychological processes and sleeping well appears to be a health imperative, essential for survival (Siegel, 2009; Diekelmann and Born, 2010; Dolgin, 2013; Luister et al., 2012; Benedict et al., 2014). Although scientists are still studying the concepts of basal sleep needs, increasing evidence shows that sleeping too little may impact severely on metabolism with a higher risk of diabetes, overweight or obesity (Stranges et al., 2008; Buxton and Marcelli, 2010; Cappuccio et al., 2010a; Cohen-Mansfield and Perach, 2012; Knutson, 2010; Cappuccio et al., 2011; Chao et al., 2011; Theorell-Haglöw et al., 2012; Faraut et al., 2013; Guo et al., 2013; Kronholm et al., 2013; Nagai et al., 2013; Ramos et al., 2013a, 2013b) and even mortality risks (Kripke et al., 2002; Stranges et al., 2008; Gallichio et al., 2009; Cappuccio et al., 2010b; Grandner et al., 2010; Kronholm et al., 2013; Kurina et al., 2013; Maia et al., 2013) across populations. Sleep research has already shown that sleeping too little can affect memory and immunity, and can jeopardize safety (Buxton et al., 2010; Cappuccio et al., 2010a; Cohen-Mansfield and Perach, 2012; Knutson, 2010; Cappuccio et al., 2011; Chao et al., 2011; Theorell-Haglöw et al., 2012; Faraut et al., 2013; Guo et al., 2013; Kronholm et al., 2013; Nagai et al., 2013; Ramos et al., 2013a, 2013b) and even survival (Kripke et al., 2002; Stranges et al., 2008; Gallichio et al., 2009; Cappuccio et al., 2010b; Grandner et al., 2010; Kronholm et al., 2013; Kurina et al., 2013; Maia et al., 2013). In recent years, chronic short sleep duration (<6 h) has also been associated with an increased risk of overweight, obesity, diabetes, hypertension and cardiovascular diseases (Buxton et al., 2010; Cappuccio et al., 2010a; Cohen-Mansfield and Perach, 2012; Knutson, 2010; Cappuccio et al., 2011; Chao et al., 2011; Theorell-Haglöw et al., 2012; Faraut et al., 2013; Guo et al., 2013; Kronholm et al., 2013; Nagai et al., 2013; Ramos et al., 2013a).

However, it is not clearly understood whether extending the duration of sleep may have a protective metabolic impact in short duration sleepers, as has been suggested by some preliminary studies (Gumenyuk et al., 2013; Markwald et al., 2013).

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Several meta analyses and reviews have been recently devoted to the association between sleep loss and obesity or diabetes (Bayon et al., 2014; Capuccio et al., 2010; Galicchio et al., 2009; Knutson and Van Cauter, 2008; Kurina et al., 2013; Magee and Hale, 2012; Schmid et al., 2015). All underlined the significant association between sleep deficit and the regulation of weight. In this review, we want however to describe more precisely the basis of sleep physiology and the biological clock in order to help readers to understand how sleep is useful for weight regulation.

The goal of this review is to better understand the role of sleep in the regulation of body weight. It would be then first necessary to describe what normal physiological sleep is, and what the normative values of sleep are in the general population.

In the second part we would give details on how the biological clock regulates hormones that may contribute to weight control.

Thirdly we will try to better understand, on the basis of recent epidemiological data, how sleep may be disturbed by sleep disorders or reduced by sleep debt with consequences on weight.

Finally we will try to carefully explore the pathways explaining the relationships between sleep debt and obesity.

2. Part 1, normal physiological sleep

2.1. Sleep physiology

It is still mysterious to observe that most of the species on the planet are regulated by a permanent alternation of sleep and wakefulness around a 24-h period. Consciousness and activity appear every morning and our days are spent performing occupational and social activities (including meals and physical activity, which directly affect body weight). Then, every evening, we retire to lie down and sleep for several hours in an unconscious state, except for dreaming. This period, which has always been considered as non-active, and unproductive by some, is probably one of the most important times of the 24-h day for conservation of energy, modulation of brain activity and clarification and organization of mood and cognition. Sleep is frequently viewed as a part of the 24-h cycle and a response to the previous period of wakefulness. It is less often described in its own merit. Physiologists have, however, described sleep in humans, which is now recorded and analyzed according to international consensus rules (Littner et al., 2003).

2.1.1. Three states of vigilance during the night (wakefulness, NREM and REM sleep)

To observe the physiological states of sleep, it is necessary to perform polysomnography (PSG): a simultaneous recording of electroencephalogram (EEG), electromyogram (EMG) and electro-oculogram (EOG). Other recordings are also sometimes, but not always, performed: respiration, electrocardiogram (ECG) and internal temperature (Littner et al., 2003).

When we are awake, the EEG is characterized by alfa waves (8–12 Hz oscillations), EMG shows high muscle tone and the EOG shows fast saccadic movements. When we lie down and fall asleep, oscillations appear on the EEG, including alpha and theta waves (4.5–8 Hz), K-complexes (isolated sharp negative deflections) and spindles (12–15 Hz 0.5–2 s long periods), which are characteristics of stage 2. At the same time, muscle tone decreases on the EMG and the EOG shows slow rolling eye movements. After a sufficient period of sleep, slow-wave (0–4 Hz) large amplitude oscillations appear that progressively dominate the EEG, with no eye movements on the EOG. This period is called SWS or non-rapid eye movement (NREM) sleep (Iber et al., 2004; Silber et al., 2007).

Rapid eye movement (REM) sleep is a totally different state of consciousness that usually appears abruptly following a first period of 60–90 min of NREM. It is characterized by an association of low amplitude theta-like oscillations, muscle tone is drastically decreased on EMG and the EOG shows rapid and symmetric eye movements (Iber et al., 2004; Silber et al., 2007).

In normal good sleepers, NREM and REM sleep alternate throughout the night with a periodicity of 60–90 min called the sleep cycle. A sleep cycle begins by a transition from wake or from first stages of SWS (Stage 1 and 2) to deeper and continuous Stage 3 SWS and then followed by an episode of REM. A typical 7–8 h night of sleep includes four to five sleep cycles. The duration of each cycle is about the same throughout the night, but the durations of the NREM and REM components are not. At the beginning of the night, REM episodes are short and NREM long; at the end, REM periods are longer and NREM shorter (Iber et al., 2004; Silber et al., 2007).

Good quality sleep is defined by several criteria: the sleep onset latency (SOL), the total sleep time (TST), wake after sleep onset (WASO) and the percentages of NREM and REM sleep. SOL is defined as the time period between switching off the light and the first period of sleep. It usually takes less than 30 min to fall asleep for adults. Most good sleepers report an SOL of 10–15 min. WASO is defined as the time spent awake during sleep (less than 30 min a night for good sleepers). The TST is the time between the first period of sleep and the final awakening (with WASO deducted). The TST varies from childhood (10–12 h) to the elderly (6–7 h) and is usually around 7 h in the general population on weekdays (Ohayon et al., 2004).

2.2. Normative data on sleep

Exactly how many hours of sleep per day are needed in adults according to their age and environmental and socio-cultural issues is unknown. Some authors recommend 8 h of sleep, whereas others believe 7 h may be sufficient for adults, with a shorter duration being necessary in the elderly (Ohayon et al., 2004; Horne, 2011; Leger et al., 2011, 2012; National sleep foundation, 2013; Leger et al., 2014; Grandner et al., 2014).

Most experts agree that sleep has to compete with multiple tasks in today's 24-h society, which may have resulted in a marked reduction in sleep duration around the planet, especially for adolescents and young adults (Leger et al., 2012; Bin et al., 2013). However, a recent survey in an impressive sample of adults (328 018 subjects) from 10 countries, showed that sleep times had not reduced in several countries, except in the most active and young subjects (Bin et al., 2012, 2013).

3. Part 2: the biological clock and its circadian regulation of hormones contributing to body weight

3.1. The biological clock

The sleep–wake cycle in humans is driven by two inter-related processes: the homeostatic process (which describes the progressive impact of time spent awake on the need for sleep) and the circadian process, which is dependent on the biological clock (Borbély, 1998). This clock is situated in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus, at the top of the optic chiasm and next to the third ventricle, and has been shown to synchronize many physiological and behavioral variables on a 24-h rest and activity pattern. Studies conducted in the 1970s on subjects isolated from all environmental factors provided evidence of an endogenous circadian rhythm of the SCN (Moore-Ede, 1983), which can be considered as a central circadian clock, but with other, peripheral clocks located in most of the peripheral tissues and organs, allowing independent expression of some local physiological cell groups (Pilorz et al., 2014; Summa and Tureck, 2014).