Sleep Medicine 37 (2017) 88–97

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Review Article

Exploring the nap paradox: are mid-day sleep bouts a friend or foe?

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ARTICLE INFO

Article history:
Received 10 November 2016
Received in revised form
11 January 2017
Accepted 18 January 2017
Available online 6 March 2017

Keywords:
Nap
Cognition
Learning
Memory
Emotion
Inflammation

ABSTRACT

The mid-day nap, sometimes called a siesta, is a ubiquitous occurrence across the lifespan. It is well established that in addition to reducing sleepiness, mid-day naps offer a variety of benefits: memory consolidation, preparation for subsequent learning, executive functioning enhancement, and a boost in emotional stability. These benefits are present even if a sufficient amount of sleep is obtained during the night prior. However, we present a paradox: in spite of these reported benefits of naps, frequent napping has also been associated with numerous negative outcomes (eg, cognitive decline, hypertension, diabetes), particularly in older populations. This association exists even when statistically controlling for relevant health- and sleep-affecting determinants. An emerging hypothesis suggests inflammation is a mediator between mid-day naps and poor health outcomes, yet further research is necessary. Given this, it may be premature to 'prescribe' naps as a health enhancer. Herein, we aggregate findings from several branches of sleep research (eg, developmental neuroscience, cognitive neuroscience, sleep medicine) to critically examine the paradoxical role of naps in cognitive and somatic health. This review uncovers gaps in the literature to guide research opportunities in the field.

1. Introduction

The mid-day nap, sometimes called a siesta, is ubiquitous. Naps are most frequent in infancy and into toddlerhood [1]. Young adult naps are less frequent, depending on cultural expectations, geographic location [2], and employment status [3]. In late life, especially after retirement, napping again becomes more prevalent [4], either because of age-related changes in sleep and circadian rhythmicity or because of psychosocial or psychological changes (eg, more free time, higher incidence of depression) [5].

The cognitive benefits of a mid-day nap have become more apparent in recent years. Naps facilitate executive functioning [6,22,23], memory formation [9–17] subsequent learning [18,19] and emotional processing [20–24]. Yet, paradoxically, there are also a multitude of studies linking frequent napping with negative outcomes, especially in older populations [5,25–27].

Here we review recent research that has unveiled the unique properties of naps and their functional contribution to cognitive and emotional processing. We first characterize the physiological architecture of naps. Next, we turn to behavioral studies that provide evidence of the beneficial functions naps serve. We then review evidence that naps may be detrimental to health, including evidence that inflammation may be related to naps and health outcomes. Finally, we discuss the implications of napping and examine whether napping should be prescribed to enhance health.

1.1. Physiology of naps

1.1.1. Nap architecture

Sleep is not homogenous, but is rather composed of multiple physiologically unique stages. Non-rapid eye movement (NREM) stages, which are further divided into stage 1 (N1), stage 2 (N2), and stage 3 (N3 or slow wave sleep (SWS)), is associated with low energy expenditure and high neuronal synchronization [28]. Conversely, rapid eye movement (REM) sleep is associated with high brain activity and energy expenditure comparable to wake.

Only recently has the physiology of naps in healthy individuals been considered. In infants, naps are indistinguishable from nocturnal sleep, as both are REM-rich [1] (Fig. 1). Later during early childhood, naps are predominantly composed of NREM sleep with very little REM [29]. Young adult naps, if of substantial length, will contain both NREM and REM bouts [30]. Naps of older adults are

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http://dx.doi.org/10.1016/j.sleep.2017.01.019
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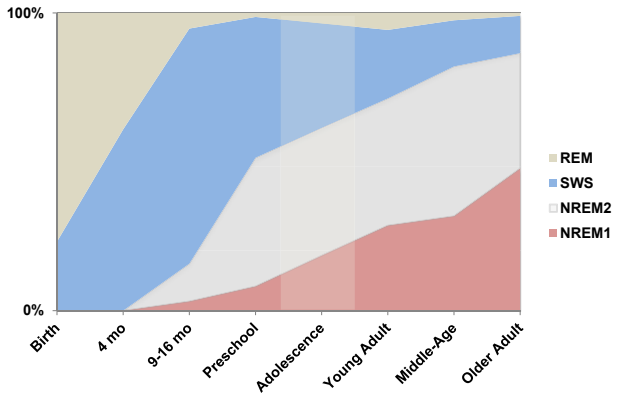

Fig. 1. Nap architecture from infancy to older adulthood. Dimmed region represents extrapolated data [1,9,29,117–119].

dominated by lighter NREM stages, a short bout of SWS, and less often, REM sleep [9].

1.1.2. Homeostatic pressure and circadian rhythmicity govern daytime sleep characteristics

Sleep is hypothesized to be regulated by two processes: Process S, which reflects homeostatic 'sleep pressure,' and Process C, which constitutes circadian (ie, endogenous) rhythmicity [31]. On a simplified level, Process S has been hypothesized to be the result of extracellular adenosine accumulation [32], which intensifies with the amount of time spent awake. Process C, on the other hand, has been hypothesized to be the result of genetically-driven changes in alertness via the suprachiasmatic nucleus of the hypothalamus [31], in addition to other factors (eg, the REM-on and REM-off switch posited to be in the tegmentum of the brainstem [33,34]). Process C cycles non-linearly, and troughs of alertness typically occur during the night and postprandially (ie, after lunch). Sleep pressure accumulated during a normal day, via Process S, is believed to initiate the onset of NREM sleep upon sleep onset [16]. Process C modulates REM sleep. Circadian influences, which affect core temperature and hormonal fluctuations, modulate REM onset, both during the night and the day.

The effects of Processes S and C on sleep are important to understanding nap sleep architecture (ie, sleep staging). Given the influence of sleep pressure on NREM, naps taken following sleep deprivation or those taken later in the day comprise mostly NREM sleep [35]. On the other hand, circadian rhythmicity resulting from Process C induces REM-rich naps early in the day. The post-prandial nap, which occurs during a circadian alertness dip but also after many hours spent awake, tends to contain both NREM and REM, although this may vary with age [9].

2. Naps benefit cognitive functions

2.1. Sleepiness and cognition

Following sleep deprivation, sleep restriction, or even a normal night of sleep, sleepiness increases with time spent awake, while cognitive abilities, such as working memory, decrease. However, a mid-day nap has been shown to effectively assist with 'recovery' of these faculties by minimizing homeostatic sleep pressure.

2.1.1. Homeostatic sleep pressure

The search for which "sleep factor" contributes to the rise and dissipation of homeostatic sleep pressure has been lengthy. Much evidence points to adenosine, a byproduct of cellular energy and metabolism (ie, hydrolysis of adenosine tri-phosphate [ATP]), and a neuromodulator that orchestrates the release of post-synaptic neurotransmitters [36], as being a critical sleep factor [32]. In theory, when cerebral energy (ie, glycogen) is required, glycogenolysis takes place, leaving an adenosine byproduct. During subsequent NREM sleep, the activity of neurotransmitters that heavily utilize glycogen during wake is reduced, and synthesis of new glycogen stores can begin. Accumulated adenosine dissipates to provide energy for glycogen replenishment. Thus, after a sufficient amount of NREM, homeostatic sleep pressure is reduced, and the process may begin anew (Fig. 2).

However, adenosine does not affect the brain uniformly, as there are several adenosine receptor types (ie, A1, A2a, A2b, and A3) with differing downstream effects [37]. Sleep-impacting effects of adenosine seem to mainly involve A1 and A2a receptors. For example, blocking A1 receptors decreases sleep [38], whereas infusing adenosine to A1 receptors promotes sleep [39]. Further, SWS is induced when A2a receptors in the subarachnoid space below the basal forebrain are promoted [40]. Additional evidence for both receptor types playing a role in sleep regulation comes from studies showing caffeine promotes wakefulness by blocking adenosine's activation of both A1 and A2a receptors [37]. A2b and A3 receptors have a relatively low affinity for adenosine and their sleep-promoting effects, if any, are poorly understood.

Although adenosine has been the focus of many recent studies, it is not the only identified sleep factor. Several other chemicals,

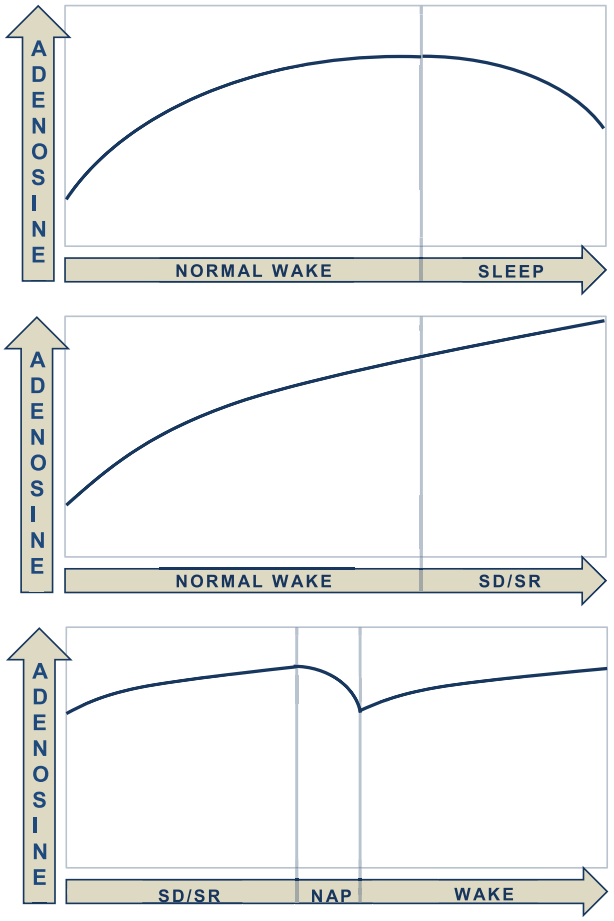

Fig. 2. Adenosine, one of the so called "sleep factors," is thought to accumulate and dissipate depending on the state [32]. SD = sleep deprivation; SR = sleep restriction.