Sleep deprivation: Impact on cognitive performance

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Abstract: Today, prolonged wakefulness is a widespread phenomenon. Nevertheless, in the field of sleep and wakefulness, several unanswered questions remain. Prolonged wakefulness can be due to acute total sleep deprivation (SD) or to chronic partial sleep restriction. Although the latter is more common in everyday life, the effects of total SD have been examined more thoroughly. Both total and partial SD induce adverse changes in cognitive performance. First and foremost, total SD impairs attention and working memory, but it also affects other functions, such as long-term memory and decision-making. Partial SD is found to influence attention, especially vigilance. Studies on its effects on more demanding cognitive functions are lacking. Coping with SD depends on several factors, especially aging and gender. Also interindividual differences in responses are substantial. In addition to coping with SD, recovering from it also deserves attention. Cognitive recovery processes, although insufficiently studied, seem to be more demanding in partial sleep restriction than in total SD.

Keywords: Sleep deprivation, cognitive performance, sleep restriction, recovery, aging, gender differences

Introduction

A person's quality of life can be disrupted due to many different reasons. One important yet underestimated cause for that is sleep loss (National Sleep Foundation 2007). Working hours are constantly increasing along with an emphasis on active leisure. In certain jobs, people face sleep restriction. Some professions such as health care, security and transportation require working at night. In such fields, the effect of acute total sleep deprivation (SD) on performance is crucial. Furthermore, people tend to stretch their capacity and compromise their nightly sleep, thus becoming chronically sleep deprived.

When considering the effects of sleep loss, the distinction between total and partial SD is important. Although both conditions induce several negative effects including impairments in cognitive performance, the underlying mechanisms seem to be somewhat different. Particularly, results on the recovery from SD have suggested different physiological processes. In this review, we separately consider the effects of acute total and chronic partial SD and describe the effects on cognitive performance. The emphasis on acute total SD reflects the quantity of studies carried out compared with partial SD. The effects of aging and gender, as well as interindividual differences are discussed. We concentrate on the studies that have been published since 1990.

Sleep and sleep loss

The need for sleep varies considerably between individuals (Shneerson 2000). The average sleep length is between 7 and 8.5 h per day (Kripke et al 2002; Carskadon and Dement 2005; Kronholm et al 2006). Sleep is regulated by two processes: a homeostatic process S and circadian process C (eg, Achermann 2004). The homeostatic process S

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depends on sleep and wakefulness; the need for sleep increases as wakefulness continues. The theory for circadian process C suggests a control of an endogenous circadian pacemaker, which affects thresholds for the onset and offset of a sleep episode. The interaction of these two processes determines the sleep/wake cycle and can be used to describe fluctuations in alertness and vigilance. Although revised "three-process models" (eg, Akerstedt and Folkard 1995; Van Dongen et al 2003b; Achermann 2004) have been suggested, this classical model is the principal one used for study designs in SD research.

There are many unanswered questions regarding both the functions of sleep and the effects of sleep loss. Sleep is considered to be important to body restitution, like energy conservation, thermoregulation, and tissue recovery (Maquet 2001). In addition, sleep is essential for cognitive performance, especially memory consolidation (Maquet 2001; Stickgold 2005). Sleep loss, instead, seems to activate the sympathetic nervous system, which can lead to a rise of blood pressure (Ogawa et al 2003) and an increase in cortisol secretion (Spiegel et al 1999; Lac and Chamoux 2003). Immune response may be impaired and metabolic changes such as insulin resistance may occur (for review, see Spiegel et al 2005). People who are exposed to sleep loss usually experience a decline in cognitive performance and changes in mood (for meta-analyses, see Pilcher and Huffcutt 1996; Philibert 2005).

Sleep deprivation is a study design to assess the effects of sleep loss. In acute total SD protocols, the subjects are kept awake continuously, generally for 24–72 hours. In chronic partial SD, subjects are allowed restricted sleep time during several consecutive nights. Although chronic sleep restriction is more common in the normal population and thus offers a more accurate depiction of real life conditions, total SD has been more thoroughly explored.

Cognitive performances measured in SD studies have included several domains. The most thoroughly evaluated performances include different attentional functions, working memory, and long-term memory. Visuomotor and verbal functions as well as decision-making have also been assessed. Sleep deprivation effects on cognitive performance depend on the type of task or the modality it occupies (eg, verbal, visual, or auditory). In addition, task demands and time on task may play a role. The task characteristics are discussed in more detail in following sections where the existing literature on the cognitive effects of SD is reviewed.

Mechanisms behind sleep loss effects

Some hypotheses are proposed to explain why cognitive performance is vulnerable to prolonged wakefulness. The theories can be divided roughly in two main approaches, in which SD is assumed to have (1) general effects on alertness and attention, or (2) selective effects on certain brain structures and functions. In addition, individual differences in the effects have been reported.

The general explanation relies on the two-process model of sleep regulation. Cognitive impairments would be mediated through decreased alertness and attention through lapses, slowed responses, and wake-state instability. Attentional lapses, brief moments of inattentiveness, have been considered the main reason for the decrease in cognitive performance during sleep deprivation (on lapse hypothesis, eg, Williams et al 1959, see Dorrian et al 2005; Kjellberg 1977). The lapses are caused by microsleeps characterized by very short periods of sleep-like electro-encephalography (EEG) activity (Priest et al 2001). Originally, it was thought that in between the lapses, cognitive performance almost remained intact, but the slowing of cognitive processing has also been observed independent of lapsing (Kjellberg 1977; Dorrian et al 2005). According to these hypotheses, performance during SD would most likely deteriorate in long, simple, and monotonous tasks requiring reaction speed or vigilance. In addition to the lapses and response slowing, considerable fluctuations in alertness and effort have been observed during SD. According to the wake-state instability hypothesis, those fluctuations lead to variation in performance (Doran et al 2001).

According to explanations on selective impact, SD interferes with the functioning of certain brain areas and thus impairs cognitive performance. This approach is also referred to as the 'sleep-based neuropsychological perspective' (Babkoff et al 2005). Perhaps the most famous theory in this category is the prefrontal vulnerability hypothesis, first proposed by Horne (1993). It suggests that SD especially impairs cognitive performances that depend on the prefrontal cortex. These include higher functions, such as language, executive functions, divergent thinking, and creativity. In order to show the SD effect, the tests should be complex, new, and interesting. A good performance would require cognitive flexibility and spontaneity. This theory also assumes that the deterioration of subjects' performance in simple and long tasks is merely due to boredom (Harrison and Horne 1998; Harrison and Horne 1999; Harrison and Horne 2000). The specific brain areas that are vulnerable to sleep loss have been explored using functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). Those studies, however, have mainly measured working memory or other attentional functions with the type of tasks that are

not traditionally emphasized in the prefrontal vulnerability hypothesis (for summary, see Chee et al 2006).

Individuals differ in terms of the length, timing, and structure of sleep. Therefore, it is logical to hypothesize that interindividual differences are also important in reaction to SD. Studies have consistently found that some people are more vulnerable to sleep loss than others (for review, see Van Dongen et al 2005). In reference to trait differential vulnerability to SD, Van Dongen et al (2005) have proposed the concept of the "trototype", as compared to the terms "chronotype" and "somnotype", which define interindividual differences in the timing of circadian rhythmicity and sleep duration. Since a comprehensive review of the interindividual differences in sleep and performance has been published recently (Van Dongen et al 2005), we will focus here on the studies with group comparisons and just briefly address the trait-like vulnerability.

Acute total sleep deprivation

Attention and working memory

The two most widely studied cognitive domains in SD research are attention and working memory, which in fact are interrelated. Working memory can be divided into four subsystems: phonological loop, visuospatial sketchpad, episodic buffer and central executive (Baddeley and Hitch 1974; Baddeley 2000). The phonological loop is assumed to temporarily store verbal and acoustic information (echo memory); the sketchpad, to hold visuospatial information (iconic memory), and the episodic buffer to integrate information from several different sources. The central executive controls them all. Executive processes of working memory play a role in certain attentional functions, such as sustained attention (Baddeley et al 1999), which is referred to here as vigilance. Both attention and working memory are linked to the functioning of frontal lobes (for a review, see Naghavi and Nyberg 2005). Since the frontal brain areas are vulnerable to SD (Harrison et al 2000; Thomas et al 2000), it can be hypothesized that both attention and working memory are impaired during prolonged wakefulness.

The decrease in attention and working memory due to SD is well established. Vigilance is especially impaired, but a decline is also observed in several other attentional tasks (Table 1). These include measures of auditory and visuospatial attention, serial addition and subtraction tasks, and different reaction time tasks (Table 1). The most frequently used task is the psychomotor vigilance test (PVT, lasts usually 10 min) (Dinges and Powell 1985), which is sensitive to sleep loss effects and provides information about both reaction speed and lapses. In working memory, the tests have varied from n-back style tasks with different demand levels to choice-reaction time tasks with a working memory component (Table 1). However, some studies have also failed to find any effect. After one night of SD, no difference was observed between deprived and non-deprived subjects in simple reaction time, vigilance, or selective attention tasks in one study (Forest and Godbout 2000). Performance on the Wisconsin Card Sorting Test, a measure of frontal lobe function, also remained even (Binks et al 1999; Forest and Godbout 2000). These results may be partly biased because of small sample sizes, inadequate control of the subjects' sleep history or the use of stimulants before the study.

Outcomes are inconsistent in various dual tasks used for measuring divided attention. Sleep deprivation of 24 h impaired performance in one study (Wright and Badia 1999), whereas in two others, performance was maintained after 25–35 h of SD (Drummond et al 2001; Alhola et al 2005). The divergent findings in these studies may be explained by the uneven loads between different subtests as well as by uncontrolled practice effect. Although dividing attention between different tasks puts high demands on cognitive capacity, subjects often attempt to reduce the load by automating some easier procedures of a dual or multitask. In the study by Wright and Badia (1999), the test was not described; in the study by Alhola et al (2005), subjects had to count backwards and carry out a visual search task simultaneously, and in the study by Drummond et al (2001) subjects had to memorize words and complete a serial subtraction task sequentially. In addition, differences in essential study elements, like the age and gender of participants, as well as the duration of SD, further complicate comparison of the results.

In the tasks measuring attention or working memory, two aspects of performance are important: speed and accuracy. In practice, people can switch their emphasis between the two with attentional focusing (Rinkenauer et al 2004). Oftentimes, concentrating on improving one aspect leads to the deterioration of the other. This is called the speed/accuracy trade-off phenomenon. Some SD studies have found impairment only in performance speed, whereas accuracy has remained intact (De Gennaro et al 2001; Chee and Choo 2004). In others, the results are the opposite (Kim et al 2001; Gosselin et al 2005). De Gennaro et al (2001) proposed that in self-paced tasks, there is likely to be a stronger negative impact on speed, while accuracy remains intact. In experimenter-paced tasks, the effect would be the opposite. However, many studies show detrimental effect on both speed and accuracy (eg, Smith et al 2002; Jennings et al 2003; Chee and Choo 2004;

Table 1 Cognitive tests in which deterioration of performance has been reported during acute total sleep deprivation

Cognitive test	Effect	Authors
Attention		
Simple reaction time	↓	Choo et al 2005, Karakorpi et al 2006
Choice reaction time tasks	↓	Wilkinson et al 1990, Smulders et al 1997, Wright and Badia 1999, Frey et al 2004, Karakorpi et al 2006, Kendall et al 2006
Serial reaction time test	↔	Nilsson et al 2005
Vienna Test System (computerized): Vigilance, simple reaction time; Cognitrone (visual analytical ability, attention and working memory	↔	Lee et al 2003
Vigilance)	↓	Wu et al 1991, Corsi-Cabrera et al 2003, Karakorpi et al 2006, Sagaspe et al 2006, Taillard et al 2006
Flanker task (computerized: attention, vigilance?)	↓	Tsai et al 2005
Dichotic listening (vigilance)	↓	Johnsen et al 2002
Psychomotor vigilance task (PVT)	↓	Dinges et al 1994, Wright and Badia 1999, Doran et al 2001, Van Dongen et al 2003, Frey et al 2004, Graw et al 2004, Van Dongen et al 2004, Adam et al 2006, Blatter et al 2006
Serial addition and/or subtraction task	↓	Drummond et al 1999, Thomas et al 2000, Van Dongen et al 2003 and 2004, Kendall et al 2006
Two column addition	↓	Wright and Badia 1999, Frey et al 2004
Visuo-spatial attention (saccadic eye movements)	↓	Bocca and Denise 2006
Finding Embedded Figures Test	↓	Blagrove et al 1995
Auditory attention task	↓	Blagrove et al 1995, Linde et al 1999
Dual task	↓	Wright and Badia 1999, Frey et al 2004
Dual task	↔	Drummond et al 2001, Alhola et al 2005
Paced Auditory Serial Addition Test (PASAT)	↔	Binks et al 1999
Working memory		
N-back	↓	Smith et al 2002, Choo et al 2005
LTR, PLUS	↓	Chee and Choo 2004, Chee et al 2006
PLUS-L (verbal working memory)	↓	Chee et al 2006
Delayed-match-to-sample task	↓	Habeck et al 2004
Choise-reaction time task (with working memory component)	↓	Jennings et al 2003
Brown-Peterson	↓	Forest and Godbout 2000
Sternberg verbal working memory task	↓	Mu et al 2005
Working memory task	↓	Wimmer et al 1992
Digit recall	↓	Frey et al 2004
Digit span	↔	Linde and Bergström 1992 (2 studies), Quigley et al 2000
Word recall (working memory)	↔	Quigley et al 2000
Verbal working memory, visuo-spatial working memory test	↔	Nilsson et al 2005
Spatial working memory task	↔	Heuer et al 2005
Attentional power (effortful information processing)	↔	Linde and Bergström 1992 (2 studies)
Long-term memory		
Word memory test	↓	Drummond et al 2000
Temporal memory for faces (recency)	↓	Harrison and Horne 2000
Probed forced memory recall and digit recall	↓	Wright and Badia 1999
Memory search	↓	McCarthy and Waters 1997
Paired word learning (implicit memory)	↓	Forest and Godbout 2000
Episodic memory (Claeson-Dahl test)	↔	Nilsson et al 2005
Implicit memory test, prose recall, Mill Hill vocabulary test (chrystallized semantic memory), procedural memory, face memory	↔	Quigley et al 2000
Benton visual retention test	↔	Alhola et al 2005
Visuomotor performance		
Critical tracking	↓	Van Dongen et al 2004
Letter cancellation task (visual search)	↓	Casagrande et al 1997, De Gennaro et al 2001
Trail-making task	↓	Wimmer et al 1992
Maze tracing task	↓	Blatter et al 2005
Digit symbol	↓	Van Dongen et al 2003, 2004
Digit symbol, Bourdon-Wiersma, other psychomotor tests	↔	Quigley et al 2000, Alhola et al 2005
Procedural motor task	↓↔	Forest and Godbout 2000
Decision making		
Critical reasoning, Masterplanner	↓	Harrison and Horne 1999
Decision-making task	↓	Linde et al 1999, Kilgore et al 2006
Verbal functions		
Logical reasoning	↓	McCarthy and Waters 1997
Logical reasoning test (Baddeley)	↓	Blagrove et al 1995, Monk and Carrier 1997
Logical reasoning test (Baddeley)	↔	Linde and Bergström 1992 (2 studies), Quigley et al 2000, Drummond et al 2004
Word detection task, repeated acquisition of responce sequence task	↓	Van Dongen et al 2004
Vowel/consonant discrimination task, letter recognition task	↓	Wimmer et al 1992
Sentence processing, categories test, spot the word, word recognition	↔	Quigley et al 2000
Word fluency, Booklet form of the Category test	↔	Binks et al 1999
Response inhibition		
Response inhibition (the Haylings sentence completion task), verb generation to nouns	↓	Harrison and Horne 1998
Go-NoGo (response inhibition)	↓	Drummond et al 2006
Stroop (color-word, emotional, specific)	↓	Sagaspe et al 2006
Spatial Stroop (suppression of prepotent responses)	↓	Heuer et al 2005
Stroop	↔	Binks et al 1999
Other measures		
Dichotic temporal order judgment	↓	Babkoff et al 2005
Negative priming (effect vanished during SD)	↓	Harrison and Espelid 2004
Task-shifting	↓	Heuer et al 2004 (2 studies)
Simon task	↓	Heuer et al 2005
Raven's progressive matrices	↓	Linde and Bergström 1992
Figural form of the Torrance Tests of Creative Thinking	↓	Wimmer et al 1992
Modified Six Elements test (story-telling, simple arithmetic calculations and object naming)	↓	Nilsson et al 2005
Switching Task	↓	Frey et al 2004
Implicit sequence learning in the serial reaction task	↓	Heuer et al 1998, Heuer and Klein 2003
Explicit sequence learning task (serial reaction tasks)	↔	Heuer et al 1998
Luria-Nebraska Neuropsychological Battery, Calculation and digit span from WAIS	↓↔	Kim et al 2001
Number-series inductions	↔	Linde and Bergström 1992
Novel oddball task (auditory)	↓↔	Gosselin et al 2005
Random generation tasks	↓↔	Heuer et al 2005 (3 studies)
Complex navigation task	↔	Strangman et al 2005
Wisconsin Card Sorting Test (computerized), WAIS-R short form	↔	Binks et al 1999

Abbreviations: SD, sleep deprivation; WAIS, Wechsler Adult Intelligence Scale; WAIS-R, Wechsler Adult Intelligence Scale-Revised.

Habeck et al 2004; Choo et al 2005). The speed/accuracy trade-off phenomenon is moderately affected by gender, age, and individual differences in response style (Blatter et al 2006; Karakorpi et al 2006), which could be a reason for inconsistencies in the SD results. It has been argued that low signal rates increase fatigue during performance in SD studies and that subjects may even fall asleep during the test (Dorrian et al 2005). Therefore, tasks with different signal loads may produce different results in terms of performance speed and accuracy.

Long-term memory

Long-term memory can be divided between declarative and non-declarative (procedural) memory. Declarative memory is explicit and limited, whereas non-declarative memory is implicit and has a practically unlimited capacity. Declarative memory includes semantic memory, which consists of knowledge about the world, and episodic memory, which holds autobiographical information. The contents of declarative memory can be stored in visual or verbal forms and they can be voluntarily recalled. Non-declarative or procedural memory includes the information needed in everyday functioning and behavior, eg, motor and perceptual skills, conditioned functions and priming. In previous studies, long-term memory has been measured with a variety of tasks, and the results are somewhat inconsistent.

In verbal episodic memory, SD of 35 h impaired free recall, but not recognition (Drummond et al 2000). The opposite results were obtained with one night of SD (Forest and Godbout 2000). The groups in both studies were quite small (in Drummond's study, N = 13; in Forest and Godbout's study, experimental group = 9, control group = 9), which offers a possible explanation for the variation in results. In addition, Drummond et al (2000) used a within-subject design, whereas Forest and Godbout (2000) had a between-subject design. In visual memory, recognition was similar in the experimental and control groups when the measurement was taken once after 36 h SD (Harrison and Horne 2000), whereas the practice effect in visual recall was postponed by SD in a study with three measurements (baseline, 25 h SD, recovery; Alhola et al 2005). Performance was impaired in probed forced memory recall (Wright and Badia 1999), and memory search (McCarthy and Waters 1997), but no effect was found in episodic memory (Nilsson et al 2005), implicit memory, prose recall, crystallized semantic memory, procedural memory, or face memory (Quigley et al 2000). In the studies failing to find an effect, however, the subjects spent only the SD night under controlled conditions (Quigley et al 2000; Nilsson et al 2005).

Free recall and recognition are both episodic memory functions which seem to be affected differently by SD. Temporal memory for faces (recall) deteriorated during 36 h of SD, although in the same study, face recognition remained intact (Harrison and Horne 2000). In verbal memory, the same pattern was observed (Drummond et al 2000). One explanation may be different neural bases, which supports the prefrontal vulnerability hypothesis. Episodic memory is strongly associated with the functioning of the medial temporal lobes (Scoville and Milner 2000), but during free recall in a rested state, even stronger brain activation is found in the prefrontal cortex (Hwang and Golby 2006). It is unclear whether this prefrontal activation reflects episodic memory function, the organization of information in working memory, or the executive control of attention and memory. Recognition, instead, presumably relies on the thalamus in addition to medial temporal lobes (Hwang and Golby 2006). Since SD especially disturbs the functioning of frontal brain areas (Drummond et al 1999; Thomas et al 2000), it is not surprising that free recall is more affected than recognition.

Although the prefrontal cortex vulnerability hypothesis has received wide support in the field of SD research, other brain areas are also involved. For instance, the exact role of the thalamus remains unknown. Some studies measuring attention or working memory have noted an increase in thalamic activation during SD (eg, Portas et al 1998; Chee and Choo 2004; Habeck et al 2004; Choo et al 2005). This may reflect an increase in phasic arousal or an attempt to compensate attentional performance during a demanding condition of low arousal caused by SD (Coull et al 2004). In other cognitive tasks such as verbal memory (Drummond and Brown 2001) or logical reasoning (Drummond et al 2004), no increase in thalamic activation was found despite the fact that behavioral deterioration occurred. This implies that thalamic activation during SD is mainly related to some attentional function or compensation, providing further support for the hypothesis that "prefrontal dependent" recall is more affected by SD than "thalamus dependent" recognition. However, it is possible that the brain activation patterns during SD reflect something more than merely different cognitive domains. Harrison and Horne (2000) stated that their results may also reflect the difficulty of the task assigned to subjects.

Other cognitive functions

Sleep deprivation impairs visuomotor performance, which is measured with tasks of digit symbol substitution, letter cancellation, trail-making or maze tracing (Table 1). It is believed that visual tasks would be especially vulnerable to

sleep loss because iconic memory has short duration and limited capacity (Raidy and Scharff 2005). Another suggestion is that SD impedes engagement of spatial attention, which can be observed as impairments in saccadic eye movements (Bocca and Denise 2006). Decreased oculomotor functioning is associated with impaired visual performance (De Gennaro et al 2001) and sleepiness (eg, De Gennaro et al 2001; Zils et al 2005). However, further research is needed to confirm this explanation, since not all studies have found oculomotor impairment with cognitive performance decrements (Quigley et al 2000).

Reasoning ability during SD has for the most part been measured with Baddeley's logical reasoning task or its modified versions. Again the results are inconsistent (deteriorated performance was reported by Blagrove et al 1995; McCarthy and Waters 1997; Monk and Carrier 1997, and Harrison and Horne 1999; no effects were noted by Linde and Bergstrom 1992; Quigley et al 2000, or Drummond et al 2004). The studies reporting no effect have mainly used SD of ca. 24 h (Linde and Bergström 1992; Quigley et al 2000), whereas in the studies showing an adverse effect, the SD period has been longer (36 h). Thus reasoning ability seems to be maintained during short-term SD. However, choosing divergent study designs may result in different outcomes. Monk and Carrier (1997) repeated the cognitive test every 2 h and found deterioration after as little as 16 h of SD. In the studies with zero-results, cognitive tests were carried out in the morning (Linde and Bergström 1992; Quigley et al 2000) or the practice effect was not adequately controlled (Drummond et al 2004). In the studies with longer SD, the tests have been conducted either in the late afternoon (McCarthy and Waters 1997; Harrison and Horne 1999) or have been repeated several times (Blagrove et al 1995; Monk and Carrier 1997). Therefore, the different results may reflect the effect of circadian rhythm on alertness and cognitive performance. In the morning or before noon, the circadian process reaches its peak, inducing greater alertness, whereas the timing of the circadian nadir coincides with the late afternoon testing (see Achermann 2004).

In addition to the cognitive domains already introduced, total SD affects several other cognitive processes as well. It increases rigid thinking, perseveration errors, and difficulties in utilizing new information in complex tasks requiring innovative decision-making (Harrison and Horne 1999). Deterioration in decision-making also appears as more variable performance and applied strategies (Linde et al 1999), as well as more risky behavior (Killgore et al 2006). Several other tasks have been used in the sleep deprivation studies (Table 1). For example, motor function, rhythm, receptive and expressive speech, and memory measured with the Luria-Nebraska Neuropsychological Battery deteriorated after one night of SD, whereas tactile function, reading, writing, arithmetic and intellectual processes remain intact (Kim et al 2001).

The adverse effects of total SD shown in experimental designs have also been confirmed in real-life settings, mainly among health care workers, professional drivers and military personnel (Samkoff and Jacques 1991; Otmani et al 2005; Philibert 2005; Russo et al 2005). Performance of residents in routine practice and repetitive tasks requiring vigilance becomes more error-prone when wakefulness is prolonged (for a review, see Samkoff and Jacques 1991). However, in new situations or emergencies, the residents seem to be able to mobilize additional energy sources to compensate for the effects of tiredness. More recent meta-analysis shows that SD of less than 30 h causes a significant decrease in both the clinical and overall performance of both residents and non-physicians (Philibert 2005).

Motivation

What role does motivation play in cognitive performance? Can high motivation reverse the adverse effect of SD? Does poor motivation further deteriorate performance? According to a commonly held opinion, high motivation compensates for a decrease in performance, but only a few attempts have been made to confirm this theory. Estimating the compensatory effect of motivation in performance during SD is generally difficult, because persons participating in research protocols, especially in SD studies, usually have high initial motivation. The concept of motivation is closely linked to the "attentional effort" that is considered a cognitive incentive (for a review, see Sarter et al 2006). According to Sarter et al (2006), "increases in attentional effort do not represent primarily a function of task demands but of subjects' motivation to perform." Furthermore, attentional effort is a function of explicit and implicit motivational forces and may be increased especially when the subjects are motivated or when they detect signals of performance decrements (Sarter et al 2006).

Harrison and Horne (1998, 1999) suggest that the deterioration of cognitive performance during SD could be due to boredom and lack of motivation caused by repeated tasks, especially if the tests are simple and monotonous. They used short, novel, and interesting tasks to abolish this motivational gap, yet still noted that SD impaired performance. In contrast, other researchers suggest that sleep-deprived subjects could maintain performance in short tasks by being able to temporarily increase their attentional effort. When a task is longer,

performance deteriorates as a function of time. A meta-analysis by Pilcher and Huffcutt (1996) provides support for that: total SD of less than 45 h deteriorated performance more severely in complex tasks with a long duration than in simple and short tasks. Based on this, it is probably necessary to make a distinction between mere attentional effort and more general motivation. Although attentional effort reflects motivational aspects in performance, motivation in a broader sense can be considered a long-term process such as achieving a previously set goal, eg, completing a study protocol. If one has already invested a great deal of time and effort in the participation, motivation to follow through may be increased.

Different aspects of motivation were investigated in a study with 72 h SD, where the subjects evaluated both motivation to perform the tasks and motivation to carry out leisure activities (Mikulincer et al 1989). Cognitive tasks were repeated every two hours. Performance motivation decreased only during the second night of SD, whereas leisure motivation decreased from the second day until the end of the study on the third day. The authors concluded that the subjects were more motivated to complete experimental testing than to enjoy leisure activities because by performing the tasks, they could advance the completion of the study. The researchers suggested that the increased motivation towards the tasks on the third day reflected the "end spurt effect" caused by the anticipation of sleep.

Providing the subjects with feedback on their performance or rewarding them for effort or good performance is shown to help maintain performance both in normal, non-deprived conditions (Tomporowski and Tinsley 1996) and during SD (Horne and Pettitt 1985; Steyvers 1987; Steyvers and Gaillard 1993). In a large study with 61 subjects (experimental group = 29), with SD of 34–36 h, and with a comprehensive test battery, the subjects were continuously encouraged and provided with 2–3 minute breaks between the tests (Binks et al 1999). Furthermore, they were told they would receive a monetary award for completing all tests with "honest effort". As result, no deteriorating effect on cognitive performance was found. Unfortunately, a non-motivated control group was not included and thus the effect of motivation remained uncertain. In general, since this issue has not been addressed sufficiently, it is difficult to specify the role of motivation in performance. It seems that motivation affects performance, but it also appears that SD can lead to a loss of motivation.

Self-evaluation of cognitive performance

It has been suggested that the self-evaluation of cognitive performance is impaired by SD. During 36 h SD, the subjects became more confident that their answers were correct as the wakefulness continued (Harrison and Horne 2000). Confidence was even stronger when the answer was actually wrong. In another study, performance was similar between sleep-deprived and control groups in several attentional assessments, but the deprived subjects evaluated their performance as moderately impaired (Binks et al 1999). The controls considered that their performance was high.

The ability to evaluate one's own cognitive performance depends on age and on the study design. Young people seem to underestimate the effect of SD, whereas older people seem to overestimate it. In a simple reaction time task, both young (aged 20–25 years) and aging (aged 52–63 years) subjects considered that their performance had deteriorated after 24 h SD, although performance was actually impaired only in young subjects (Philip et al 2004). When it comes to the study design and methodology, the way in which the self-evaluation is done may affect the outcome. The answers possibly reflect presuppositions of the subjects or their desire to please the researcher. The repetition of tasks is also essential. Evaluation ability is poor in studies with one measurement only (Binks et al 1999; Harrison and Horne 2000; Philip et al 2004), whereas in repeated measures, the subjects are shown to be able to assess their performance quite reliably during 60–64 h SD and recovery (Baranski et al 1994; Baranski and Pigeau 1997). Thus, self-evaluation is likely to be more accurate when subjects can compare their performance with baseline.

Chronic partial sleep restriction

Although chronic partial sleep restriction is common in everyday life and even more prevalent than total SD, surprisingly few studies have evaluated its effects on cognitive performance. Even fewer studies have compared the effects of acute total sleep deprivation and chronic partial sleep restriction. Belenky and co-workers (2003) evaluated the effect of partial sleep restriction in a laboratory setting in groups which were allowed to spend 3, 5 or 7 h in bed daily for seven consecutive days. The control group spent 9 h in bed. In the 3 h group, both speed and accuracy in the PVT deteriorated almost linearly as the sleep restriction continued. In this group, performance was clearly the worst. In the 5- and 7 h groups, performance speed deteriorated after the first two restriction nights, but then remained stable (though impaired) during the rest of the sleep restriction from the third night onwards. Impairment was greater in the 5- than 7 h group. Accuracy followed the same pattern in the 7 h group, but further declined in the 5 h group as the study went on.

The control group's performance did not change during the study. Intriguingly, a highly similar pattern was observed in another study with the same task when sleep was restricted by 33% of the subject's habitual nightly sleep, which resulted in 5 h of sleep per night on average (Dinges et al 1997). Both speed and accuracy were impaired at the beginning of the sleep restriction period followed by a plateau and finally, another drop after the seventh night of deprivation. However, no change was found in probed recall memory or serial addition tests, probably because of the practice effect and short duration of the tests (serial addition test: 1 min).

It is difficult to compare the effects of total and partial SD based on existing literature due to large variation in methodologies, including the length of SD or the type of cognitive measures. The only study that has compared total and partial SD found that after controlling learning effects, cognitive performance declined almost linearly in the course of the study in all four experimental groups (Van Dongen et al 2003a): one group was exposed to 3 nights total SD, and in other experimental groups, time in bed was restricted to 4 or 6 h for 14 consecutive days. The control group was allowed 8 h in bed for 14 days. Impairment in psychomotor vigilance test and digit symbol substitution task for the 4 h group after 14 days was equal to that of the total SD group after 2 nights. Deterioration in the serial addition/subtraction task for the 4 h group was similar to that of the total SD group after 1 night. The effect of 6 h restricted sleep corresponded to 1 night of total SD in psychomotor vigilance and digit symbol. Performance remained unaffected in the control group.

According to the well-controlled studies (Dinges et al 1997; Belenky et al 2003; Van Dongen et al 2003a), the less sleep obtained due to sleep restriction, the more cognitive performance is impaired. Otherwise, it is difficult to draw conclusions about the effects of chronic sleep restriction because of methodological problems in the previous studies. Blagrove et al (1995) compared subjects that slept at home either 5 h or 8 h per night for 4 weeks and found no effect in a short task of logical reasoning (duration 5 min). The statistical analyses were compromised by the small sample size (6 subjects in the experimental group and only 4 subjects in the control group). In another protocol, they also carried out auditory vigilance test, two column addition, finding embedded figures, and logical reasoning (10 min) tasks, and again no effect was observed with groups of 6–8 subjects having 4, 5 or 8 h sleep per night for 7, 19 or 40 weeks respectively (Blagrove et al 1995). Casement et al (2006) reported no change in working memory and motor speed in the group whose sleep was restricted to 4 h per night for 9 nights. In the control group, performance improved. The study was carried out in a controlled clinical environment, but only one short test session per day was included, which means that subjects may have been able to temporarily increase their effort and thus maintain their performance. Furthermore, the results were confounded by the practice effect. In other sleep restriction studies, SD cannot be considered chronic, since the length of the restriction has been 1–3 nights (Stenuit and Kerkhofs 2005; Swann et al 2006; Versace et al 2006).

Since chronic partial SD mimics every day life situations more than acute total SD, additional studies on how it affects cognitive performance are warranted. In addition, the tasks used in previous studies have been quite short and simple, and trials with more demanding cognitive tasks are required. The effects of sleep restriction have also been addressed by drive simulation studies, which are interesting and practical designs. Just one night of restricted sleep (4 h) increased right edge-line crossings in a motorway drive simulation of 90 minutes (Otmani et al 2005). However, neither the drivers' position in the lane nor the amplitude and frequency of steering wheel movements were affected. One sleep-restricted night did not increase the probability of a crash, but after five nights of partial SD, the quantity of accidents increased (Thorne et al 1999).

Cognitive recovering from sleep deprivation

The recovery processes of cognitive performance after sleep loss are still obscure. In many SD studies, the recovery period has either not been included in the protocol or was not reported. Recovery sleep is distinct from normal sleep. Sleep latency is shorter, sleep efficiency is higher, the amounts of SWS and REM-sleep are increased and percentages of stage 1 sleep and awake are decreased (Armitage et al 2001; Kilduff et al 2005). The characteristics of recovery sleep may also depend on circumstances and some differences seem to come with eg, aging (Kalleinen et al 2006). Evidence suggests that one sleep period (at least eight hours) can reverse the adverse effects of total SD on cognition (Brendel et al 1990; Corsi-Cabrera et al 2003; Adam et al 2006; Drummond et al 2006; Kendall et al 2006). The tasks have been mainly simple attentional tasks; for example, the PVT used by Adam et al (2006) has been proven to have practically no learning curve and little if any correlation with aptitude (Durmer and Dinges 2005). Thus, it is likely that the improvement was mostly caused by the recovery process and not just the practice effect.

After chronic partial sleep restriction, the recovery process of cognitive functioning seems to take longer than after

acute total SD. Performance in the PVT was not restored after one 10 h recovery night, but approached the baseline level after two 10 h nights in a study with seven consecutive sleep restriction nights with 5 h sleep/night (Dinges et al 1997). Using the same test, three 8 h recovery nights were not enough to restore performance after one week of sleep restriction even in the group that spent 7 h time in bed (the study is explained in greater detail in paragraph 1 of "Partial sleep restriction", Belenky et al 2003). The group that spent 3 h in bed showed the greatest decline as well as the greatest recovery, although it did not reach baseline level again. In the 5 h group, a similar deterioration-recovery curve was observed, although it was not as steep. Those authors concluded that during mild and moderate chronic partial SD, the brain adapted to a stressful condition to maintain performance, yet at a reduced level. This adaptation process was obviously so demanding that it postponed the restoration of normal functioning. According to their results, it could be further interpreted that when sleep restriction was severe, no such adaptation occurred, which in turn allowed for greater recovery. However, these results may be biased because of poor statistical sensitivity in multiple comparisons. They have also been criticized by eg, Van Dongen et al (2004), who pointed out that another confounding factor may have been considerable interindividual differences in recovery rates. Since interindividual differences have been observed in response to SD, it is likely – although not yet adequately verified – that those individual traits also affect the recuperation.

Sleep deprivation in different populations

Aging

Sleep structure changes with aging. Slow wave sleep and sleep efficiency decrease, and alterations in the circadian rhythm occur (for reviews, see Dzaja et al 2005; Gaudreau et al 2005). Sleep complaints also become more frequent (Leger et al 2000). Yet, during prolonged wakefulness, cognitive performance seems to be maintained better in aging people than in younger ones (Bonnet and Rosa 1987; Smulders et al 1997; Philip et al 2004; Stenuit and Kerkhofs 2005). Total SD of 24 h deteriorated vigilance in young subjects (20–25 years), whereas performance in aging subjects (52–63 years) remained unaffected (Philip et al 2004). Similarly, during three consecutive nights of partial SD (4 h in bed) performance in psychomotor vigilance task declined more in young subjects (20–30 years) than in aging ones (55–65 years, Stenuit and Kerkhofs 2005). In visual episodic memory, visuomotor performance and divided attention, aging subjects (58–72 years) were able to maintain their performance after 25 h of SD and showed improvement only after a recovery night (Alhola et al 2005). However, no comparison with young subjects was made in that study.

Sleep deprivation deteriorates accuracy of performance, especially in young subjects (Brendel et al 1990; Smulders et al 1997; Adam et al 2006; Karakorpi et al 2006). Regarding performance speed, however, results have been inconsistent and the performance of aging subjects has declined more, less, or equally compared to that of younger people. In simple and two-choice reaction time tasks as well as in a vigilance task, reaction speed was impaired in aging subjects (59–72 years) during 40 h SD, whereas young subjects (20–26 years) kept up their speed (Karakorpi et al 2006). These results followed the speed/accuracy trade-off phenomenon so that aging subjects maintained accuracy at the expense of speed and the younger ones did the opposite. In contrast, two other studies found that young subjects were slower than aging subjects (Brendel et al 1990; Adam et al 2006). During 24 h wakefulness, performance speed in a vigilance task was impaired in both 20- and 80-year-olds, but more so in the young subjects (Brendel et al 1990). This was confirmed in another study with 40 h SD (Adam et al 2006). When measuring reaction speed in three different choice-reaction time tasks, performance deteriorated similarly in young (18–24 years) and aging (62–73 years) subjects after 28 h total SD (Smulders et al 1997).

Even though there is some evidence that older subjects tolerate SD better than young subjects, it is difficult to determine the age effect during SD with precision. However, because of age-related changes in many aspects of sleep and wakefulness, it is plausible that aging influences reactions to SD. As suggested previously, the weaker SD effect in aging may be due to attenuation of the circadian amplitude, which is reflected in the performance curve in vigilance tasks (Blatter et al 2006). Also, changes in the homeostatic process may play a role. During wakefulness, the accumulation of sleep pressure seems to be reduced in aging (Murillo-Rodriguez et al 2004), which could leave older subjects more alert. There is also evidence that aging subjects recover faster from SD than young subjects in terms of physiological sleep (Bonnet and Rosa 1987; Brendel et al 1990). This faster recovery in sleep state may also mean better restoration of cognitive performance (Bonnet and Rosa 1987; Brendel et al 1990). However, more research is necessary to confirm these hypotheses.

The age effect found in previous studies could also be explained by methodological factors, such as inadequate control of the baseline conditions. Younger subjects are usually more chronically sleep deprived (National Sleep Foundation 2002) due to several reasons, such as studying, career building or raising children. Chronic sleep restriction may cause long-term changes in brain functions that are not reversible during short adaptation and baseline periods in sleep laboratory studies. Even though subjects of certain studies were instructed to maintain a regular 8 h sleep schedule for 3–5 days, this may not be enough to erase the previous "sleep debt" (Brendel et al 1990; Philip et al 2004; Adam et al 2006). Furthermore, in the long run, people tend to get used to experiencing sleepiness (Van Dongen et al 2003a) and thus may not even recognize being chronically sleep deprived. Perhaps aging people also have more experience that helps them to cope with the challenges posed by SD. Nevertheless, based on the available studies, it is impossible to distinguish the factors behind the age effect.

Gender

There are dissimilarities between genders in sleep structure measured with polysomnography (for a review, see Manber and Armitage 1999). Furthermore, women of all ages report more sleeping problems than men (Leger et al 2000). Sex hormones affect sleep through several mechanisms, both genomic and nongenomic, including neurochemical and vascular mechanisms (for a review, see Dzaja et al 2005). This ensures instant and short-term effects as well as long-term ones.

It is possible that physiological responses to SD are not equal among men and women. During SD of 38 h, EEG showed more sleep activity in men than in women during waking rest and cognitive performance (Corsi-Cabrera et al 2003). Presumably, therefore, one recovery night of nine hours would be enough to restore waking EEG activity in men, but not in women. Only a few studies have examined gender differences in cognitive performance during SD. In a vigilance task, performance was more impaired in men but returned to the baseline level in both men and women after recovery sleep (Corsi-Cabrera et al 2003). In another study, women performed better than men in verbal and in visuo-constructive tasks during 35 h SD (Binks et al 1999). No gender differences were observed in word fluency, maintenance or suppression of attention, auditory attention or cognitive flexibility. In that study, however, only one point of measurement was included, and so the difference in performance could be caused by SD or initial distinctions between the gender groups.

Few attempts have been made to evaluate the effect of sex hormones on coping with SD. It has been suggested that hormone therapy, which is widely used for women during their menopausal transition to help alleviate climacteric symptoms, attenuates physiological stress response (Lindheim et al 1992). However, after 25 h of total SD, no difference was observed between hormone therapy users and nonusers in visual episodic memory, visuomotor performance, verbal attention and shared attention (Alhola et al 2005). In addition, during 40 h of SD, hormone therapy did not produce any advantage in reaction time or vigilance tasks (Karakorpi et al 2006).

The previous studies suggest that women cope with continuous wakefulness better than men. According to evolution, the demands of child nurturing and rearing in women would support this hypothesis (Corsi-Cabrera et al 2003), but that certainly does not constitute a comprehensive explanation today. Gender differences during SD could be due to either physiological or social factors. There are differences in the brain structure and functioning of men and women (Ragland et al 2000; Cowell et al 2007). These can be seen in cognitive performance in normal, non-deprived conditions: men typically have better spatial abilities and mental rotation, and higher visuo-constructive performance, whereas women perform better in visuomotor speed and some verbal functions, especially verbal fluency (for a review, see Kimura 1996). Men and women also exhibit behavioral and lifestyle differences, which are mainly due to socialization and gender roles (Eagly and Wood 1999). Current literature, however, provides only minimal evidence of differential effects during SD, and does not resolve the issue of sexual dimorphism in coping with SD.

Interindividual differences

Several studies provide evidence that during total SD, performance becomes more variable as assessed from the within-subject point of view (eg, Smith et al 2002; Habeck et al 2004; Choo et al 2005). This is considered to reflect the wake-state instability caused by prolonged wakefulness. However, Doran et al (2001) were probably the first to also examine between-subjects variability, which they found to increase in PVT as wakefulness was extended to 88 hours. They suggested that some people are more vulnerable to the effects of sleep loss than others, which could probably explain the lack of significant results in some group comparisons. These differences between subjects could have arguably been caused by differences in sleep history, but the sleep patterns for the preceding week were controlled with sleep diaries, actigraph, and calls to the time-stamped voice recorder.

The interindividual variability has been further examined with a thorough protocol where a three night study (baseline, 36 h SD and recovery) was carried out three times (Van Dongen et al 2004). Sleep history was manipulated by instructing subjects to stay in bed for either 6 or 12 h per night for one week before the study. The 12 h procedure was repeated and the order of the conditions was counterbalanced. The cognitive test selection included serial addition/subtraction task, digit symbol, critical tracking, word detection, repeated acquisition of response sequences, and PVT. The authors concluded that interindividual differences were systematic and independent from sleep history. The trait-like differential vulnerability to sleep loss has received support from an fMRI study attempting to reveal the neural basis for the interindividual differences (Chuah et al 2006). They used a go/no-go task to measure response inhibition after 24 h of sleep deprivation. The results indicated that the subjects less vulnerable to SD had lower prefrontal cortex activation at the rested wakefulness than the more vulnerable subjects. During SD, activation increased temporarily in the prefrontal cortex and in some other areas only in the less vulnerable subjects. Since interindividual differences have also been found in other sleep-related variables, such as duration, timing, and quality of sleep, sleepiness, and circadian phase (Van Dongen 1998; Van Dongen et al 2005), it is plausible that the tolerance to SD may also vary. Nevertheless, more studies are needed for further support.

Methodological issues and common biases

Although the adverse effects of SD on cognitive performance are quite well established, some studies have failed to detect any deterioration. Inadequate descriptions of study protocols or subject characteristics in some studies make it difficult to interpret the neutral results. However, it is likely that such results are due to methodological shortcomings, such as insensitive cognitive measures, failure to control the practice effect or other confounding factors, like individual sleep history or napping during the study. Also, if the task is carried out only once during the SD period, the results may be influenced by circadian rhythm.

Sleep deprivation studies are laborious and expensive to carry out, which may lead to compromises in the study design: for example, a small sample size can reduce the statistical power of the study, but a larger population may come at the expense of other methodological issues, such as a reduction in the cognitive test selection or in the number of nights spent in the sleep laboratory. Comparison of the results is also complicated because the length of sleep restriction varies and the studies are designed either within- or between-subjects.

Sleeping in unfamiliar surroundings may impair sleep quality. An adaptation night at the sleep laboratory is used to minimize this first night effect. However, in several studies, this has been neglected and the SD period has been preceded by a "normal" night at home (eg, Harrison and Horne 2000; Jennings et al 2003; Choo et al 2005). Although sleeping at home certainly reflects a subject's reality more accurately, it does not allow for precise control and information of sleeping conditions. Adding a portable recording, such as an actigraph, provides objective information about eg, bedtime and resting periods. In some studies, the first night in the sleep laboratory has been the baseline (eg, Drummond et al 2000; Forest and Godbout 2000; De Gennaro et al 2001; Drummond et al 2001), whereas others have included one adaptation night (eg, Casagrande et al 1997; Alhola et al 2005). Yet, it may be questionable to use data from the second night as the baseline because sleep quality can be better than normal due to the rebound from the first night. Accordingly, only data from the third night should be accepted, which has been the case in a few studies (Thomas et al 2000; Van Dongen et al 2003a). This, however, makes the procedure very hard. Furthermore, study protocols can be improved by adding an ambulatory EEG recording to confirm the wakefulness of the subjects during the study.

In sleep studies, a common pitfall is recruitment methods**.** Enrolment via advertisements or from sleep clinics favors the selection of subjects with sleeping problems or concerns about their cognitive performance. Thus, strict exclusion criteria regarding physical or mental diseases or sleeping problems are essential. Further, sleeping habits should be controlled to make sure that the subjects are not initially sleep deprived. For this, use of a sleep diary for eg, 1–3 weeks before the experiment (eg, De Gennaro et al 2001; Habeck et al 2004; Alhola et al 2005) or an actigraph is applicable (Harrison and Horne 1999; Thomas et al 2000).

The use of medication or stimulants, such as caffeine, alcohol or tobacco, is often prohibited before the experiment (eg, Thomas et al 2000; Van Dongen et al 2003a; Habeck et al 2004; Alhola et al 2005; Choo et al 2005). In some studies, the subjects have been required to refrain from these substances only 24 h before the study (Habeck et al 2004; Choo et al 2005), which may increase withdrawal symptoms and dropping out of the study. Thus a longer abstinence, eg, 1–2 weeks, is more appropriate (Van Dongen et al 2003a; Alhola et al 2005).

A variety of cognitive tests, from simple reaction time measures to complex decision-making tasks requiring creativity and reasoning, have been used to evaluate the effect of SD on cognition. The greatest problem in repeated cognitive testing is the practice effect, which easily conceals any adverse effects of SD. Therefore, careful control over learning is essential. Cognitive processes are also intertwined in several ways, which makes it difficult to specify exactly which cognitive functions are utilized in certain performances. Because attention is involved in performing any cognitive task, a decrease in other cognitive domains during SD may be mediated through impaired attention. In complex tasks, however, applying previous knowledge and use of strategies or creativity may be more essential. Some studies have concentrated on neural correlates of cognitive functioning during continuous wakefulness. Both fMRI (Portas et al 1998; Drummond et al 2000; Drummond et al 2001; Chee and Choo 2004; Habeck et al 2004; Choo et al 2005) and PET have been used (Thomas et al 2000). Although these trials yield interesting information about brain functioning, the use of imaging techniques limits the selection of cognitive tests that could be carried out at the same time.

Dorrian et al (2005) have compiled a list of criteria for neurocognitive tests that would be suitable for investigating sleep deprivation effects. The criteria include psychometric quality, ie, reliability and validity, but the tests should also reflect a fundamental aspect of waking neurocognitive functions and it should be possible to interpret them in a meaningful way. The tasks should be repeatable, independent of aptitude, and they should be short with a high signal load. These criteria are not met in some studies. Dorrian et al (2005) also argued that vigilance is the underlying factor through which the sleep deprivation effects are mediated in all other tasks. However, although attention is needed to perform any task to some extent, the hypothesis that sleep deprivation can have an independent effect on other cognitive functions such as memory cannot be ruled out. Nevertheless, when measuring other cognitive functions, the characteristics of the task should be considered carefully and, eg, for repeated measures of memory, parallel test versions should be used.

Conclusion

The negative effect of both acute total and chronic partial SD on attention and working memory is supported by existing literature. Total SD impairs a range of other cognitive functions as well. In partial SD, a more thorough evaluation of higher cognitive functions is needed. Furthermore, the effects of SD have not been thoroughly compared among some essential subpopulations.

Aging influences a person's ability to cope with SD. Although in general the cognitive performance of aging people is often poorer than that of younger individuals, during SD performance in older subjects seems to deteriorate less. Based on the scarce evidence, it seems that in terms of cognitive performance, women may endure prolonged wakefulness better than men, whereas physiologically they recover slower. Tolerating SD can also depend on individual traits. However, mechanisms inducing differences between the young and aging and between men and women or different individuals are mostly unclear. Several reasons such as physiological mechanisms as well as social or environmental factors may be involved. In conclusion, there is great variation in SD studies in terms of both subject selections and methods, and this makes it difficult to compare the different studies. In the future, methodological issues should be considered more thoroughly.

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