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PHYSIOLOGICAL REVIEW

How much sleep do we need?

Michele Ferrara and Luigi De Gennaro

Department of Psychology, University of Rome "La Sapienza", Italy

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Summary There is increasing concern for sleeplessness-related risks in modern society. Some recent epidemiological data seem to support the view that many segments of the adult population have chronically inadequate sleep. On the other hand, some experts have claimed that our core, basic amount of sleep is around 6 h per night, and that the rest of our sleep can be easily curtailed, being unnecessary to fulfill any sleep need. However, experimental data on the effects of both acute and cumulative partial sleep deprivation (PSD) consistently point out that sleep restriction has substantial negative effects on sleepiness, motor and cognitive performance and mood, as well as on some metabolic, hormonal and immunological variables. As chronic PSD may have serious long-term adverse health effects, it should be avoided in the general population. In the short-term, the effects of sleep curtailment seem to accumulate linearly, while the effects of long-term PSD should be further investigated, as the few available studies are flawed by methodological weaknesses. On the other hand, there is evidence that extending sleep by 2–3 h beyond the norm produces only marginal benefits for an average individual. Finally, it is underlined that, as large individual differences do exist in the need for sleep, the search for *the* sleep need may be vain. A somnotypology, taking into account age, gender and the position in both the sleep–alert and the morningness–eveningness continuum, should help in the search for the actual individual sleep need.

INTRODUCTION

In the last few years there has been a growing attention to sleep and sleeplessness-related problems. This interest is mainly due to the recognition that sleepiness and fatigue are becoming endemic in the population, contributing to human error and, consequently, many (sometimes catastrophic) accidents in industrialized societies. Although recent estimates for the percentage of injuries and deaths primarily caused by sleepiness and/or fatigue are largely divergent, varying from 2% [1] to 41% [2], they involve enormous costs in terms of lives and money. There is also clear evidence that several catastrophic accidents in nuclear power plants and in transportation disasters involved human errors by personnel who had inadequate sleep and who were often working the night shift [3]. The use of artificial lighting has completely changed the traditional timing of human activities linked to the sleep–wake/dark–light cycles [4]. Social demands, an active lifestyle and the widespread network of telecommunications (internet, 24h television programmes) have also contributed to these habit changes in modern Western societies, often leading to inadequate sleep. Moreover, with the transition

Correspondence should be addressed to: Michele Ferrara, Ph.D., Dipartimento di Psicologia, Università degli Studi di Roma "La Sapienza", Via dei Marsi, 78; 00185 Rome, Italy. Fax: +39 06 44 51 667; E-mail: michele.ferrara@uniromal.it

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from an agrarian to an industrial economy, time means money. In technology-based societies characterized by global economies, shift-work is implemented to facilitate around-the-clock production, business and transportation. Shift-work (namely night-work) is often associated with severe sleepiness and performance impairment [5]. Consequently, the importance of providing education on the risks of working or driving while sleepy, in order to prevent fatigue- or sleepiness-related accidents is becoming clear [3]. Nevertheless, for a lot of people, the body's need to sleep is treated as a waste of time. In our 24-h society, we often steal night time hours for daytime activities, depriving ourselves of precious sleep.

The present review will move in the general sphere of the scientific debate regarding sleep need, formalized in 1995 by two papers published in the journal *Sleep* [6–7]. Sleep need will be referred to here as the daily amount of sleep that allows a subject to be fully awake (i.e. not sleepy) and able to sustain normal levels of performance during the daytime. Since sleep behaviour is determined to some extent by society's demands such as work, social life and family requirements, it follows that the habitual sleep schedule of an individual will not necessarily meet his/her actual sleep need. But is there a fixed sleep amount that must be obtained to avoid a sleep debt? (We will refer to a "sleep debt" to merely indicate either an acute or a chronic discrepancy between one's ideal sleep duration and the actual sleep amount obtained.) And, is our society suffering from sleep deprivation, that is, is the average 7.5 h daily sleep quota enough, or can we improve our daytime functioning by sleeping longer? The above-mentioned debate on sleep need [6, 7] was mainly centred on the latter question. On the one side, Harrison and Horne [6] stated that most people are not chronically sleep deprived. They provided some indirect evidence for their idea mainly by reporting on the poor ecological validity of sleep extension and on its limited effectiveness in improving levels of alertness and performance. On the other side, Bonnet and Arand [7] claimed that our society is chronically sleep deprived, specifically pointing to the negative effects of experimental sleep reduction on sleepiness. Although in the last 5 years no resounding advances have been made to help in further advancing this scientific debate, nevertheless in this paper we will present a comprehensive view of the available scientific evidence in support of each of the opposing positions. Moreover, we will also try to provide an answer to the question, "is there a fixed sleep need for any individual", by addressing in more detail the large and often unrecognized individual differences in the need for sleep. However, the present work is admittedly limited in its scope, as only findings from studies on normal subjects will be taken into account. Although the relationships between sleep need, sleep disturbances and psychiatric disorders are undoubtedly interesting, they are beyond the specific interests of this work, and deserve to be systematically addressed as a separate topic.

HISTORICAL CHANGES IN THE SLEEP HABITS OF THE GENERAL POPULATION

The belief that partial sleep loss is a common condition in many segments of our society has an historical basis. In the last 20 years there has been a marked decrease in leisure time experienced by American workers, and annual work hours rose from 1786 in 1969 to 1949 in 1987 [8]. It is possible that time for sleep is also included among leisure time. In fact, it seems that in the past century we have reduced our average time asleep by 20%. Webb and Agnew [9] reported a mean 1.5 h reduction in the average sleep length of children aged 8–17 years between 1910 and 1963. However, this finding has been attributed to errors of protocol and sampling procedure [6]. Actually, even the authors of the original 1913 study [10] defined their results as a "striking excess of sleep" compared with other contemporary studies. A survey of more than 5000 Japanese children during 1923 found an average of 1 h less as compared to the above-mentioned study [10]. Clearly, subjective reports of sleep habits can be influenced by long-term changes in subject compliance, experimental protocol, socio-economic status, home environment, seasonal, climatic and geographical differences [6].

Nevertheless, other support has been provided for a reduction of sleep length in the general population, independent of the age group considered. In a healthy adult group (50–65 years old) the modal number of nocturnal hours of sleep has fallen from 8–8.9 h per night, as reported by the American Cancer Society 1959 survey [11, 12], to 7–7.9 h per night in the mid-1980s [13]. In the latter study,

about 27% of the sample obtained only 6–6.9 h of sleep per night. This is a relevant finding since it was reported that men sleeping 6 h or less had 1.7 times the total age-adjusted death rate of men sleeping 7 or 8 h per night [14].

Among college students, a 30 min decrease in self-reported sleep duration was found over the 1978–1988 decade [15]; this result has been ascribed to possible uncontrolled changes in workload and time spent at college [6]. More recently, in a sample of 52 undergraduate female students monitored for 2 weeks by means of a sleep diary, it was found that the subgroup attending school from 07:00 to 12:00 delayed their sleep by 47.4 min and prolonged it by 118.2 min during weekends [16]. Moreover, the group attending school during the afternoon hours delayed their bedtime by 24 min during weekends, without changing their sleep duration. This finding strongly suggests that the sleep extension shown during the weekend by the group attending school in the morning is due to a sleep debt accumulated during workdays. Such a hypothesis is supported by the finding that the morning group took more naps than the afternoon group throughout the week, probably to compensate for sleep deprivation.

In epidemiological settings, the contribution of daytime sleep episodes (i.e. naps) to the overall daily sleep quota should be carefully taken into account, especially when middle-aged and older people are considered. In fact, the wish for more sleep can easily lead to increasing the frequency of naps in any population. As an example, in a longitudinal study of 93 adolescents examined at 2-year intervals from a mean age of 11.4 to 21.3 years, it was found that from 54.3 to 74.5% of the subjects indicated the wish for more sleep [17]. The subgroup complaining of sleep insufficiency also reported a higher frequency of daytime naps and of occasional nocturnal sleep reduction as compared to the sufficient sleep group. So, although the need for more sleep in this group of adolescents/early adults could have been a mere consequence of curtailed sleep duration or irregular sleep habits, it was accompanied by an increased napping frequency.

On the other hand, most surveys of adult, middle-aged and elderly subjects do not report any details on daytime sleep habits of the participants. In a recent epidemiological study [18] carried out on more than 400 adults between 20 and 64 years in Uppsala County, Sweden, 12% of the sample fulfilled the criteria for persistent insufficient sleep, a condition of considerable chronic sleep loss, operationally defined as a ratio below 0.80 between the amount of habitual sleep and the amount of self-estimated need for sleep more than twice a week.

Consistent results also come from a comparison of disturbed sleep and daytime fatigue reported by representative samples of comparable demographics of the upper midwestern rural adult population in the mid-1930s and the mid-1980s [19]. Contemporary men reported increased levels of fatigue and tiredness as compared to people in the post-Great Depression/pre-World War II era, although no increase in reported disturbed sleep was evident. These data again suggest that increased tiredness may be related to a more or less voluntary sleep curtailment typically seen in modern society and described in the report of the National Commission on Sleep Disorders Research [20]. More recently, in a diary study of 266 healthy subjects aged 20–50 years, Monk *et al.* [21] examined weeknight vs weekend-night differences in time-in-bed, weekday alertness and reliance on alarms as possible indicators of sleep debt. The subject selection criteria excluded potential subjects with sleep disorders, shiftworkers, parents of very young children, and people habitually sleeping less than 6 h or more than 9 h per night. The mean reported time-in-bed was 7 h 27 min during weeknights, with a mean increase of 28 min during weekends. Larger differences between weeknight and weekend time-in-bed were associated with shorter weeknight time-in-bed, with lower weekday alertness upon awakening, and with greater weekday alarm use. Although this study revealed some evidence of recovery oversleeping during the weekend, indicating a certain amount of sleep debt, for 54% of the sample this was 30 min or less. However, the strict selection criteria, and the fact that some subjects might have worked on Saturdays and/or Sundays, might have accordingly reduced the differences between weeknight and weekend sleep duration. It is relevant, however, that 7.5% of the sample spent less than 6.5 h in bed during weeknights. Since subjects reporting less than 6 h of sleep per night were excluded on pre-screening, the percentage of people sleeping far less than their need is probably underestimated. However, this study was admittedly restricted to the analysis of the morning component of the sleep diary, which required subjects to give

information only about the preceding night of sleep. Consequently, the possible confounding role of napping on the reported results cannot be completely ruled out. In fact, although 71% of the sample was asked to refrain from napping during the investigation period, nevertheless about 10% of the total subject-days involved naps. This "extra" unnoted sleep could have helped the most sleep deprived subjects to cope with daytime sleepiness, increasing their daily sleep amount.

Finally, in an actigraphic investigation carried out on a large sample of San Diego adults ($n = 273$, age range: 40–64 years) it was surprisingly reported that the average sleep for men and women was only 373 min [22]. Women slept more than men (386 vs 358 min), and non-Hispanic white volunteers more than minorities (379 vs 354 min). The actual contribution of possible daytime naps to the overall daily total sleep time was not reported, even though the authors claimed that there was only little appreciable daytime sleep as measured by the actigraph. Although the actigraphic data scoring could have slightly underestimated night sleep, nevertheless these findings further suggest that the adult population sleeps less than in the past decades, also corroborating contemporary questionnaire surveys [e.g. 18, 21].

In conclusion, measuring insufficient sleep in epidemiological settings can be complex and methodologically questionable, since there is no agreed measure of sleep sufficiency [18]. As in any study based on subjective reports, a further limitation is that any concept of insufficient sleep is based on the individual's ability to correctly estimate his own sleep need and actual sleep time. Some caution in interpreting these results is required because the possibly important role of daytime naps, especially in the elderly, is often not assessed. Moreover, actigraphic recordings may lead to an underestimation of night sleep (or, conversely, to an overestimation of intrasleep wake). Both these facts (unnoted naps and the use of actigraphy) can result in spurious short sleep times. Nonetheless, the indexes used by some authors in the studies reviewed above [e.g. 18, 21] seem to be quite powerful in pointing out a real sleep debt. Taken together, the epidemiological, diary-based and actigraphic results reported here converge in indicating that insufficient sleep may be considered as a public health problem, as it persistently involves a considerable part of the population in the 17–65 years age range sleeping less than they need to on a chronic basis. Nevertheless, highly controlled studies with a sound methodology and sensitive subjective and objective measures could be useful to further clarify the important issue of insufficient sleep in the general population.

HOW MUCH SLEEP IS ENOUGH?

Although the idea that our society is largely affected by sleep restriction has become a conventional wisdom [23], sleep researchers do not seem to agree on how much we should sleep. According to the indications reported about the nature of sleep on "The National Sleep Foundation" Web page (a non-profit organization dedicated to improving the quality of life for the millions of Americans who suffer from sleep disorders and to the prevention of catastrophic accidents related to sleep deprivation or sleep disorders), the amount of sleep we need to be at our best is as individual as the amount of food we need.

However, as already mentioned, a scientifically based controversy does exist on whether the average daily sleep quota of 7.5 h is enough, and whether daytime alertness and performance can be improved by sleeping longer [6–7]. This controversy can be dated back to 1975 when Webb and Agnew, allowing young good sleepers to sleep *ad libitum*, reported an average of 2 h oversleeping [9]. Consequently, they provocatively concluded that we are chronically sleep deprived, claiming that we should sleep for 9–10 h instead of the average 7.5 h. A similar position has been recently maintained by Bonnet and Arand [7]. On the other hand, Horne [24] suggests that a "core" amount of sleep does exist, between 4.5 and 6 h per night. He claims that humans can easily adapt to a 5–6 h sleep schedule, and that the rest of our night sleep is "optional", not necessary to fulfill any sleep need nor to prevent accumulation of sleep debt [25].

Actually, the two above-mentioned positions seem to be too extreme, and do not take into account that large individual differences do exist in the optimal sleep requirement (e.g. between natural long- and short-sleepers). In the next sections we will review available scientific evidence on the effects of sleep restriction and extension, with the aim of understanding whether an optimal sleep amount can be identified for an individual.

EFFECTS OF PARTIAL SLEEP DEPRIVATION (PSD) ON SLEEP STRUCTURE

According to the two-process model of sleep regulation [26], sleep timing and characteristics are co-determined by a circadian process and a sleep-dependent homeostatic process. The latter is considered to reflect the need or pressure for sleep, which builds up during sustained wakefulness and dissipates during sleep. Slow-wave sleep (SWS) and slow-wave activity (SWA, i.e. EEG power density in the delta band) are considered to be primary markers of sleep homeostasis [e.g. 27] because they show a predictive quantitative relationship with the duration of wakefulness and sleep [28, 29]. In this theoretical frame, any increase of SWS amount or SWA during sleep can be interpreted as reflecting an increased pressure for sleep, namely an increased sleep need.

Changes in sleep structure following sleep curtailment have been documented by using a number of experimental designs, ranging from 1–2 nights of abrupt sleep reduction to weeks or months of gradual sleep curtailment [e.g. 30]. In general, a reduction of all sleep stage duration except SWS is observed when the night sleep is reduced to a minimum of 4 h [31, 32]. On the other hand, an increase in stage 4 duration has been reported in the first days of a long sleep reduction regimen [32, 33]. Similarly, a rapid increase in EEG delta and theta power density has been observed in a 4 h sleep regimen lasting 4 days, followed by a steady state in the subsequent days of sleep curtailment [34]. There is also evidence that, if the sleep curtailment regime is maintained over several days, REM sleep tends to occur earlier in the sleep period to partially compensate for REM deprivation—although it never reaches baseline levels—sleep-onset latency decreases and sleep efficiency increases [31, 32]. A REM sleep rebound has also been found in the 2 recovery nights following only 2 nights of sleep reduced to 4 h [35]. Moreover, sleep-onset REM periods have been reported when sleep restriction lasts for several days [32, 36]. Differential effects on sleep structure are observed whether sleep restriction is confined to the first or the second part of the night. Restricting sleep to the second part of the night produces higher amounts of REM and stage 4 sleep and lower stage 2, compared with restricting sleep to the first part of the night [37].

Recovery sleep after a period of sleep restriction also shows characteristic and predictable features: decreased sleep-onset latency, increased sleep efficiency and a marked REM sleep rebound [30, 34]. Slow-wave sleep rebound is generally not reported, since this part of sleep is almost completely preserved during partial sleep deprivation. However, an SWS increase has been shown after only 1 night of sleep reduced to 5 h, during which baseline SWS amount were preserved [38]. Obviously, when subjects are permitted to sleep only 3 h for 8 nights, a clear SWS rebound in the recovery night is observed [39].

In conclusion, the SWS enhancement during recovery sleep that followed acute sleep restriction to 4 or 5 h without SWS deprivation [35, 38], supports the notion that even small sleep reductions can alter sleep homeostasis, leading to an increased sleep need. The REM sleep redistribution during long-term sleep curtailment [31, 32], together with the above-mentioned evidence of SWS homeostasis after acute sleep restriction [35, 38] seem to bring contrary evidence to the idea that our core, fundamental sleep can be limited to the first three to four sleep cycles and contains only about half of our nightly REM sleep [24, 25].

EFFECTS OF ACUTE AND SHORT-TERM PSD ON SLEEPINESS AND PERFORMANCE

One problem in critically analysing the body of data on the effects of PSD is that one is faced with a variety of experimental paradigms involving completely different schedules with delayed or advanced bedtimes, ranging from 1 or 2 nights of abrupt sleep reduction to weeks or months of gradual sleep curtailment [e.g. 30]. Moreover, the allowed sleep duration may vary between 1 and 5 h. Another problem is that a wide range of performance tasks, and of subjective and objective measures of mood and sleepiness have been used. Many studies adopted only one kind of measure (objective vs subjective), making it hard to compare results: in fact, a given sleep amount may be enough to maintain acceptable levels of performance, but may not provide a subjective perception of well-being, or vice-versa. Last but not least, the subjects' basal sleep schedule, both in terms of duration and circadian

timing, can have a considerable impact on the experimental manipulations of the various studies. Optimal efficiency during wakefulness may sometimes depend more upon the maintenance of a regular sleep–wake rhythm than on obtaining a normal amount of sleep. As an example, in a group of subjects normally sleeping 8 h per night, it has been reported that a later sleep onset was associated with adverse mood changes [40], confirming previous studies which showed that delaying bedtime in normal subjects produces REM and mood changes resembling those of depression [41]. Because both PSD and sleep extension studies involve changes in the timing of sleep onset and/or offset, it can be speculated that the resulting effects are due, at least in part, to the disruption of the individuals' circadian rhythm of sleep and wakefulness. In this case, any effect attributed only to changes in sleep duration could become spurious and questionable. Similar considerations also apply to habitual sleep duration: sleep reduction will be easier for subjects not living in a chronic sleep debt condition, regardless of how long they actually sleep every night. Moreover, subjects selected because they sleep a mean of 7–8 h per night are not necessarily equivalent, as their individual sleep need may greatly differ. However, information on participants' ideal sleep duration is usually not reported. Since most studies are carried out on healthy students, it is likely that their life constraints can cause irregular sleep habits or a shortening of the mean sleep length, compared with the individual sleep need. Finally, to study the effects of sleep reduction, the recording of a "true" baseline is very important. Since different subjects may need different sleep amounts to reach an optimal level of alertness and performance, "false" baselines can contribute to yielding conflicting results.

We will refer here to acute partial sleep deprivation as a single night of sleep curtailment, while short-term PSD will include the studies where sleep restriction proceeds up to 1 week, allowing an evaluation of possible cumulative effects.

Acute sleep restriction

Many studies have examined the effects of a single night of PSD by means of several performance and sleepiness measures. In a classic experiment, Wilkinson *et al.* [42] compared the effects on performance of one and two nights of sleep of different lengths (7.5, 5, 3, 2, 1, 0 h of sleep allowed). After 1 night of PSD, calculation (addition) was impaired with less than 3 h of sleep, while vigilance was impaired with less than 5 h. Moreover, cumulative effects of PSD were found, since performance on both tasks was worse on the second day of sleep reduction. Taub and Berger [43] evaluated 10 subjects accurately screened to have an habitual retiring time at 00:00–00:30, 7–8 h of uninterrupted nocturnal sleep and no evidence of daytime naps. Following 1 night of sleep reduced to 5 h (from 03:00 to 08:00), they reported that accuracy and speed of response on a vigilance task were significantly poorer, and negative mood significantly greater, compared with the habitual 8 h sleep condition. Sleep reduction had no effect on performance on an addition task. Carskadon *et al.* have performed sleep restriction studies in different age groups. One acute study, carried out in a group of 12-year-old subjects [44], found increased daytime sleepiness as measured by Multiple Sleep Latency test (MSLT) after 1 night of sleep reduced to 4 h, although inconsistent performance deficits were shown.

A linear decrease in sleep latency to MSLT has also been reported [45] in response to parametric reductions of time in bed (8, 6, 4, 0 h) in a group of 32 subjects. It must be underlined that a sleep curtailment by as little as 2 h was followed by a significant increase in sleepiness levels, as indicated by a MSLT latency shortening between 2.2 and 7 min. Similarly, a linear increase in the propensity to sleep (MSLT latency) and of subjective sleepiness as a function of the increase in sleep restrictions (8, 5, 4, 3, 2, 1 h of sleep) has been found in six subjects normally sleeping 7.5–8.5 h per night [46]. In this case, however, the linear increase of objective sleepiness seems to be a statistical artifact, since the inspection of the means clearly shows a sharp and sudden drop of MSLT sleep latencies from the 8 h to the 5 h condition, with the mean MSLT latency in the five different reduction conditions ranging from 5 min 2 s to 2 min 31 s. Vigilance performance was only marginally affected. In a further sleep restriction study, Gillberg and Akerstedt [47] found that a 4 h sleep (between 03:00 and 07:00) negatively affected the subjective feeling of being well-rested as compared to a baseline 8 h condition; furthermore, the mean sleep latency to MSLT was significantly shorter and reaction time performance was worse after the PSD night. In partial disagreement with the studies previously described, it

has been recently reported [48] that, in a sleep restriction paradigm allowing 8, 4, 2 or 0 h of sleep, the relationship between the previous total sleep time and the subsequent sleep onset latency measured during the Maintenance of Wakefulness Test (MWT) follows an exponentially decaying function. The authors stressed that there was no significant difference in the mean MWT latency between the 8 h and the 4 h conditions, with a reduction of only 1 min. Consequently, they claimed that "since the ability to maintain wakefulness . . . may be of special relevance for the estimation of the importance of 'sufficient' sleep length, it could still be argued that a small, transient reduction in nocturnal sleep length may not be as significant as often suggested" [48, pp. 172–173]. On the other hand, these data should also be interpreted as an effect of the scarce sensitivity of the MWT to moderate and acute sleep loss [e.g. 49, 50], as well as (at least partly) due to a "ceiling effect", since at one of the two times-of-day considered (11:00) MWT latencies after the 8 h sleep always reached 20 min (the maximum test duration).

Short-term sleep restriction

As already reported, when PSD lasts beyond one day, cumulative effects are typically found. Wilkinson *et al.* [42] showed that performance on both a vigilance and a calculation task was worse on the second day of sleep reduction. Carskadon and Dement [51], having reduced the sleep allotment to 4 h for 2 consecutive nights in 12 subjects, showed a progressive and significant increase of sleepiness, as indicated by reduced sleep latencies to the MSLT. In this case the MSLT scores regained basal values after 1 night of recovery sleep. Similarly, 2 nights of sleep restricted to 4 h in the first or second half of the night [37] cumulatively impaired performance on a simple reaction time task in a group of eight healthy young women who habitually slept for 8–8.5 h a night. However, since no difference in performance deficits was found between the two PSD conditions (one leaving the time of sleep onset unchanged with respect to baseline sleep and the other delaying it by 4 h), it was concluded that sleep loss *per se*, more than any change in sleep architecture, was responsible for the negative effects on performance. The lack of significant differences between the effects of the two PSD conditions seems to indicate that the negative influence of sleep reduction on mood, vigilance and performance reported by others after PSD obtained by postponing the sleep episode [e.g. 43, 47] cannot be simply attributed to the delayed bedtime [40, 41]. Nevertheless, it is not possible to rule out the specific effects of the disruption of the individual sleep–wake rhythm. In an extension of the Wilkinson *et al.* study [42], Hamilton *et al.* [52] evaluated performance following 7.5, 6 and 4 h of sleep per night for 4 days. They reported cumulative negative effects of PSD on vigilance and addition performance, although performance on a digit-span task was surprisingly above baseline values in the 4 h condition.

As far as longer sleep reduction regimens are concerned, Webb and Agnew [39] found no uniform or consistent performance decrements in eight young subjects allowed only 3 h of sleep for 8 consecutive days. Performance on a paced addition test, on a vigilance task and on a reaction time task began deteriorating after 7–8 days of PSD. However, these results are largely affected by learning effects that obscured earlier performance decrements by counteracting the effects of sleepiness. Carskadon and Dement [53] assessed, in a group of 10 young subjects, the effects of 7 days of sleep restricted to 5 h on subjective (Stanford Sleepiness Scale, SSS; Analog Sleepiness Scale) and objective (MSLT) sleepiness. The participants had regular sleep habits and reported habitual sleep times of 9 h nightly. An immediate increase in daytime sleepiness assessed by means of SSS was found, while sleepiness measured by the analog scale and the MSLT increased after the second day of PSD. Moreover, both subjective measures levelled off after the fourth day of sleep restriction, while MSLT showed a progressive increase of sleepiness throughout the 7 PSD days. This result is particularly interesting since it shows a clear dissociation between different measures of sleepiness; the possible low sensitivity of subjective measures of sleepiness in detecting, in some situations, the cumulative effects of several days of sleep curtailment should be kept in mind when interpreting the results of studies based only on subjective reports. It must be noted, however, that the selection of subjects with habitual sleep times close to the limit of the definition of long-sleepers (>9 h) may have contributed to the results. In other words, it is conceivable that the effects of sleep reduction will be more dramatic for habitual long-

than short- or normal-sleepers. This hypothesis is supported by the finding that short sleepers tolerate total sleep loss better than long sleepers [54].

Cumulative effects on subjective sleepiness and performance have been reported also after 5 nights of sleep reduced to a mean of 4.6 h per night [55, 56] in a group of seven young subjects with habitual sleep duration between 7 and 8.5 h per night. In this study, however, testing sessions were scheduled only after the 1st and the 5th day of PSD, not allowing a step by step monitoring of the PSD effects. The practice effects that emerged and the lack of any control on the participants' sleep (they slept at home unmonitored throughout the experiment) are two major faults of this study. On the other hand, in another study [57], six subjects with regular nocturnal sleep habits lasting 7.5–8.5 h, slept 4.3 h for 6 nights without reporting any deficit in several performance and vigilance tasks except for a significant reduction in total number of correct additions performed after 3 nights of PSD. These results, however, contrast with the subjective reports of the sleep-reduced subjects as regards a decreased ability to concentrate and having headaches, and with the inability of the majority of them to continue beyond the 6th day on the sleep-curtailment regimen.

Finally, in a group of 16 young adults with regular sleep habits (habitual mean sleep duration not reported), Dinges *et al.* [58] showed significant cumulative effects of 1 week of sleep reduced to 5 h per night on subjective (SSS) and objective (MSLT) sleepiness, mood disturbances and psychomotor performance. As already reported by others [53], subjective sleepiness and fatigue showed an immediate increase in response to PSD, while number of lapses on the vigilance task were significantly increased only after the 2nd day of sleep restriction. MSLT mean latency, measured only on the 5th PSD day, was dramatically decreased. The effects of sleep restriction seemed to level off between the 2nd and 5th day of PSD for subjective variables, and between the 2nd and 6th day for psychomotor variables, suggesting that some kind of adaptation to sleep restriction may occur following an initial clear drop in vigilance and alertness. However, the authors underlined that "virtually every performance and mood variable . . . displayed continued growth of deficit in the final day or two of PSD" [58, p. 274]. This interesting finding could be explained, as suggested by the authors, by a step-like function in sleepiness growth, at least for this size of sleep curtailment. The increase of subjective sleepiness and decrease of performance levels in the last days of PSD was confirmed to further continue on the 8th day in a subgroup of eight subjects tested after an additional night of sleep restriction. The psychomotor performance changes (number of lapses) across the 7 days of PSD reported in this study were remarkably similar to the MSLT results reported in an almost identical experimental paradigm [53], the correlation between the two data sets being -0.95. Since the best fitting polynomial trend in both these functions was linear, according to Dinges *et al.* it can be concluded that the effects on sleepiness and performance of 7 days of sleep reduced to 5 h "are cumulative and show little evidence of having reached an asymptote" [58, p. 275].

EFFECTS OF LONG-TERM AND GRADUAL SLEEP REDUCTION

Only a few studies have examined the effects of a chronic, long-term (from 2 weeks up to 2 months) reduction of sleep length. Webb and Agnew [33] curtailed the daily sleep time of 15 young subjects from their average sleep length of 7.5–8.0 h to 5.5 h per night, for 60 consecutive nights. Sleep and performance were monitored in the laboratory only once every 7 days. Polygraphic recordings of sleep were carried out between 01:00 and 06:30, but the mean bedtime and awakening time during the reduction period at home were not reported. As the experiment progressed, a slight but steady decline in correct detections in an auditory vigilance task was reported. At the very beginning of the sleep reduction regimen, a significant drop in the number of false positives in the same vigilance task occurred; this decrease continued throughout the experiment. The authors attributed the latter result to a decline in the subjects' willingness to perform the task rather than to the lowered vigilance levels due to sleep loss. No significant changes on an addition task and on a word memory test were found. As far as subjective measures are concerned, participants reported an immediate and significant increase in the difficulty in getting up in the morning; after the first week, this negative feeling stabilized although it always remained significantly different from baseline. Subjective daytime drowsiness also followed a similar pattern of variations during the experiment.

Subjects reported a decreased sleep onset latency from the first week, even though this report was substantiated by EEG findings only during the first and the last experimental weeks. Finally, no significant mood changes were found. Although the authors claimed that their subjects were able to maintain a 5.5 h sleep regimen for 8 consecutive weeks with limited behavioural consequences, it has to be underlined that the very few training sessions provided on the tasks may have caused practice effects in the course of the experiment. Moreover, uncontrolled napping or other lacks of compliance to the experimental protocol could have helped the subjects to sustain the long sleep reduction without dramatic negative effects.

More recently, Blagrove *et al.* [57] submitted a group of eight subjects, regularly sleeping 7.5–8.5 h per night, to a 4-week sleep reduction to 5.2 h per night and, in a second experiment, eight subjects with the same above-mentioned sleep habits to 18 nights of sleep reduced to a mean of 5.3 h per night. All subjects slept at home for the whole study and were continuously monitored by means of actimeters. In the first study, subjects showed no speed or accuracy deficits on a logical reasoning task, as compared to a control group. It is noteworthy, however, that two out of eight subjects did not meet the sleep reduction conditions, being excluded from the analyses. Similarly, in the second experiment, the main effect of sleep reduction was an increase in subjective fatigue and sleepiness. However, performance on several cognitive tasks did not substantiate these findings. In fact, there was only a nearly significant ($P = 0.06$) decrease in the total number of correct detections in the auditory vigilance task following 3 nights of sleep reduction, as compared to the control group. The authors concluded that the subjective discomfort readily caused by sleep reduction, and the reluctance of most of the subjects to continue on the PSD regimen, were not matched by decreases in performance on tasks sensitive to sleep deprivation. As already noted for many other PSD studies involving performance assessments over several days [e.g. 33, 39], robust practice effects were also evident in this case for all the reported tasks. Such learning curves compromise the validity of any conclusion about the lack of PSD effects on performance. As already stressed by Dinges *et al.* [58], the presence of a practice control group does not necessarily avoid this confounding, since the interaction between practice and sleep loss effects could be nonlinear over time. The possibility that PSD could have had no effect on the shape and/or the slope of the learning curves, giving way to quite similar trends in the experimental and control groups, should also be considered.

Finally, Dinges *et al.* [59] performed a dose-response experiment of chronic sleep reduction by allowing either 4 ($n = 13$), 6 ($n = 13$) or 8 ($n = 9$) hours in bed for 14 consecutive laboratory nights, and assessing performance and sleepiness every 2 h. The participants were screened to have stable sleep–wake cycles and 6.6–8.6 h of sleep per night. Compared with the 8 h control condition, both the 4 and 6 h sleep restrictions resulted in linear decrements across the 14 days for almost all performance measures (frequency and duration of lapses in a vigilance task; number of correct substitutions in the Digit Symbol Substitution test; overall performance in a serial addition–subtraction test). Performance deteriorated most rapidly and dramatically in the 4 h condition, reaching levels by the 13th day similar to those found after 2 nights of total sleep deprivation. Although less severely, in the 6 h condition performance also deteriorated significantly, being equivalent by the 5th day to performance after 1 night without sleep. Uncontrolled dramatic sleep attacks were reported in one-fourth of the 6 h condition subjects, and in one half of the 4 h condition subjects after the 6th day of sleep restriction. The incidence of such attacks peaked during the last 2 experimental days. In sharp contrast with performance results, most of the subjective mood and sleepiness ratings did not show differences as a function of sleep reduction. On the whole, these data seem to clearly point out that, when properly and frequently assessed, cognitive performance during chronic sleep restriction is strongly degraded. This impairment is dependent on the magnitude of sleep reduction. It is noteworthy, however, that even a sleep length of 6 h, well within the estimated amount of "core sleep" [24, 25], does not prevent cumulative performance deficits that, by the 5th day of PSD, become equivalent to those seen after 1 night of total sleep loss. The large discrepancy between objective and subjective data [see also 53] points out again that the negative effects of PSD may be unappreciated by the individual, even though his/her functioning falls below safety limits within the first week of sleep restriction. It is not clear as to what extent the large age range

of the selected subjects (21–38 years, mean = 28 years) and the inclusion of people habitually sleeping only 6.6 h per night (this was the lower limit of the habitual sleep duration range reported by the authors), may have influenced the results in an uncontrolled manner. Young adults habitually sleeping less than 7 h per night could live in a condition of chronic sleep restriction: a further reduction of sleep allotment can have stronger negative effects in such a population than in a group of subjects habitually sleeping as long as they need. On the other hand, if they were natural short sleepers, they should have been less sensitive to sleep reductions (see below the section on "Individual differences in sleep need" and ref. 54).

The long-term PSD studies described above all involved abrupt changes of sleep habits (i.e. by the first experimental day sleep was curtailed by a certain amount). However, in order to understand whether sleep length could be shortened for very long periods of time without negative effects on individual well-being and whether the new sleep habits can be fully adapted to and maintained even after the experimental imposition is removed, the best way seems to be to implement a gradual sleep reduction paradigm. The first study of this type was carried out by Johnson and MacLeod [60] on three young adults who reduced their sleep by 30 min every 2 weeks from a 7.5 h baseline to 4 h. The final 4 h regimen was kept for 3 weeks, but one of the subjects quit the study during the 4.5 h regimen. Negative mood, fatigue, concentration problems and performance deficits (memory, additions and reading comprehension) appeared between the 5.5 h and the 4 h regimens, although they were not marked or consistent. An 8-month follow-up indicated that both subjects adopted sleep regimens 1–1.5 h shorter than their previous baseline. It should also be recalled that the subjects reported difficulties in keeping the schedule in the course of the study, as indicated by unintentional napping and oversleeping episodes. In a replication of this study [32, 61], three couples habitually sleeping about 8 h per night and a fourth couple with a sleep pattern of 6.5 h per night reduced their sleep by 30 min every 2 weeks until they reached 6.5 h; then sleep was reduced by 30 min every 3 weeks until 5 h. Finally, sleep was shortened by a further 30 min every 4 weeks. Sleep was recorded at home 3 nights a week. Performance on a word memory test, an addition test, auditory vigilance, digit span and mood were tested at intervals during the entire study. In contrast to the Johnson and MacLeod study [60], none of the subjects achieved the 4 h schedule. As regards the six subjects with an 8-h baseline sleep, two reduced their sleep to 5.5 h, two to 5 h and two reached 4.5 h. The 6.5 h baseline couple reached the 5 h regimen. All subjects reported increasing levels of fatigue and sleepiness as sleep time decreased. They began to verbally complain of discomfort between 6.5 and 6 h of sleep and decided to stop the gradual sleep reduction because they were overwhelmed by fatigue, falling asleep in class and not being very vigilant while driving. On the other hand, none of the performance measures declined during the study, probably due to strong practice effects. In a 1-year follow-up the three couples with an 8-h sleep habit were still sleeping 1–2.5 h less than their previous baseline, while the other couple had returned to 6.5 h of sleep. Finally, Horne and Wilkinson [62] studied six subjects, habitually sleeping 7.75–8.25 h per night, in a gradual sleep restriction paradigm: a 1-week 1-h reduction, 3 weeks with a 1.5 h reduction, and 2 other weeks with a 2 h reduction. A control group maintaining baseline sleep was run simultaneously. Subjects reported once a week to the laboratory and were tested on an auditory vigilance test, an EEG assessment of sleepiness (that involved measuring the latency to the first 3 min of stage 2 recorded with subjects in bed instructed to stay awake), and a subjective measure of sleepiness. With regard to vigilance performance, similar practice effects were found in both experimental and control groups. Overall daytime sleepiness did not increase in the reduction group; only transient increases in subjective sleepiness were found at the highest reduction levels. Experimental subjects experienced no major difficulties except for some tiredness upon awakening and near lunchtime. Also in this case, most volunteers reported having maintained a 1-h sleep reduction in a 3-month follow-up.

In conclusion, there are some major problems with most of the chronic long-term and the gradual sleep reduction studies. Most of them are flawed by clear practice effects, so that interpretation of the results (or of the lack of any significant result) becomes very difficult. In these cases, however, a striking divergence between subjective and objective outcomes becomes apparent. In fact, while performance levels tend to be stable or to show only a slight decline during the sleep reduction period,

as practice effects counteract those of PSD, the subjective measures show significantly increased levels of fatigue and sleepiness [33, 57, 60, 62]. As already mentioned before [see also ref. 58, p. 268], the use of a control group does not necessarily solve this problem. We also agree with Carskadon and Roth [30] who pointed out that, considering the very small sample sizes of these studies, subjects' self-selection may have been a confounding variable. Moreover, as underlined even by Horne and Wilkinson [62], the outcomes of all the long-term experiments (except Dinges *et al.* [59], in which subjects were monitored in the laboratory throughout the study) "depended heavily on the reliability and truthfulness of the subjects to keep the schedules and to report any deviations from these". Also taking into account that a significant, uncomfortable increase in negative feelings and sleepiness has always been reported in all the long-term sleep reduction studies, and that in the only well-controlled PSD study using a fair sample size and very frequent performance assessment [59], strong neurobehavioural deficits have been shown, it may be concluded that the claims of little, if any, effect on daytime sleepiness and performance as a consequence of a chronic 1.5–2 h sleep restriction do not seem to be substantiated by robust and reliable data.

EFFECTS OF PSD ON OTHER PHYSIOLOGICAL PARAMETERS

There seems to be a general consensus that the main function of sleep is the restoration of brain energy metabolism, and that sleep loss has little or no effect on peripheral function [24, 25, 63, 64]. However, some recent studies that investigated metabolic, hormonal and immunological variables in people restricting their sleep, have dramatically shown that this is not the case.

Although it has been long recognized that total sleep deprivation has immunosuppressive effects in humans [e.g. 65], it has been recently shown, in a group of 42 healthy subjects, that even a modest early night PSD (between 22:00 and 03:00) reduces natural killer activity and cellular immune responses [66]. After 1 night of recovery sleep, natural killer activity regained baseline levels, while interleukin-2 production remained suppressed. Similar negative effects on natural killer cell activity have been reported after 1 night of late-night PSD (between 03:00 and 07:00) in a group of 24 healthy subjects [67]. Partial sleep deprivation has been shown to induce some alterations also in the hypotalamo-pituitary-adrenal (HPA) axis function [68]. One night of sleep reduced to 4 h (between 04:00 and 08:00) was followed by a delay in the return to quiescence of cortisol secretion, resulting in an increase of evening cortisol levels. According to the authors, these findings suggest that the mechanism of HPA recovery from stimulation are altered by acute partial sleep loss. Under chronic PSD conditions, these effects may "facilitate the development of central as well as peripheral disturbances associated with glucocorticoid excess, such as memory deficits due to impaired hippocampal function and insulin resistance" [68, p. 869]. These findings have been confirmed and extended in a recent study [69] involving 6 nights of sleep curtailment (sleep allowed between 01:00 and 05:00). Results showed that, in the sleep-debt condition, glucose tolerance was significantly lower than in the normally rested condition (a 3-day baseline with 8 h of sleep). This finding is consistent with a clinically significant impairment of carbohydrate tolerance. Decreased thyrotropin concentration, increased evening cortisol concentration and increased activity of the sympathetic nervous system were also found. As decreased carbohydrate tolerance and increased sympathetic activity are well-recognized risk factors for the development of insulin resistance, obesity and hypertension, these results strongly suggest that chronic sleep loss can be pathogenetic and also increase the severity of pathologies such as diabetes and hypertension.

Taken together, the studies briefly summarized in this section show that, in addition to the negative effects on sleepiness, performance and mood, PSD, especially on a chronic basis, may have serious long-term adverse health effects.

CONCLUSIONS: WHAT DO PSD STUDIES TELL US?

The studies of acute partial sleep loss, as well as those of cumulative sleep restrictions of different lengths and durations, are quite consistent in indicating that PSD negatively affects sleepiness and performance, as well as some metabolic, hormonal and immunological variables. There is evidence that the short-term cumulative effects of PSD on different measures of sleepiness and performance may

be linear, not having reached an asymptote in a week of sleep reduced to 5 h per night [53, 58]. A step-like function in sleepiness growth has also been proposed [58] suggesting that, following an initial drop in alertness, some kind of adaptation to sleep restriction may occur followed by a further sleepiness increase.

We do believe that the negative findings (i.e. no effect of PSD on different performance measures) reported by some long-term and gradual sleep reduction experiments do not weaken our conclusions, since those studies are flawed by several methodological problems: strong learning effects that obscure or counteract the effects of increasing sleepiness on performance; very small sample sizes; possible self-selection of subjects; lack of laboratory control of subject compliance with the PSD protocol; evaluation of performance at too large intervals and for limited periods of time. So, the real possibility to easily shift from an 8-h sleep to a 5–6 h schedule in a stable manner and without cognitive and/or behavioural consequences needs the support of stronger and more consistent data. The "subjective" conclusions of this narrative review on the effects of PSD are strengthened by the more "objective" ones of a recent meta-analysis on the effects of total and partial sleep deprivation [70]. Meta-analysis is an alternative approach to summarizing research in a given field, based on a quantitative rather than qualitative combination of studies. This method can provide interesting details on the strength and consistency of an experimental effect, as well as on its moderating factors. The authors used a sleep duration of 5 h among the inclusion criteria for PSD studies. Surprisingly, PSD appeared to have considerably greater overall negative effects than either short-term (45 h) or long-term (>45 h) total sleep deprivation. On average, PSD subjects performed at a level 2 standard deviations (SD) below that of non-sleep-deprived subjects, while total-deprived subjects showed a 1 SD difference. Partial sleep deprivation appeared to have the least effect on motor performance tasks (about 1 SD below baseline), while both cognitive performance (3 SD below baseline) and mood (4 SD below baseline) were largely more decreased with PSD than with total sleep deprivation. Supplemental analyses revealed that PSD subjects showed a worse cognitive performance on simple tasks than on complex tasks, and on long tasks (10 min) more than on short ones (6 min). As far as motor performance is concerned, performance was worse on long tasks (8 min) than on short tasks (3 min). Although the large effect of PSD on mood can be partly due to a subjective overestimation of the adverse effects of sleep curtailment, the unexpected dramatic results on cognitive performance are noteworthy. It must be recognized that four out of the six PSD studies considered in this meta-analysis (total sample size = 450) were carried out on medical residents, and three of them used medical-related tasks as dependent measures. In our view, these sampling and task details, rather than weaken the results, may somewhat increase their interest, because of their inherent larger ecological validity. On the other hand, the greater negative effects of PSD compared to total sleep deprivation reported in this meta-analysis are quite surprising and in open contradiction with linear or exponential increases of sleepiness and performance deficits found in some studies in which total sleep time was varied, including a zero-sleep time condition [42, 45, 46, 48, 71]. Although it is not easy to find a clear and satisfying explanation for this finding, a possibility is that the particular nature of both the subjects and the tasks adopted in most of the PSD studies considered in the meta-analysis significantly contributed to this unexpected result.

It is concluded that PSD has a substantial effect on sleepiness, motor and cognitive performance and mood. In some cases, the adverse effects of PSD can be greater even than those of total sleep deprivation; they are evident even after a few days of mild (e.g. 2 h) sleep reduction. Since they have been largely underestimated even by experts in the field, the effects of partial sleep reduction should be more extensively investigated.

Nevertheless, a few methodological "caveat" must be pointed out here. First, it must be recognized that some of the studies here reviewed are hardly comparable, since they used many different PSD paradigms (i.e. advanced bedtime vs delayed waketime vs *ad libitum* sleep with free bedtime and waketime), subjects with different sleep habits, and several subjective and objective outcome measures. Furthermore, studies adopting only one type of outcome measure are not comparable at all [e.g. 39, 42, 45]. In fact, a given sleep amount might allow normal levels of performance during the subsequent wakefulness,

but might be not enough to provide a subjective perception of alertness and well-being, or vice-versa. Whenever results are not obscured by learning effects, this differential influence of a given sleep quantity on subjective and objective variables can explain the sharp discrepancies between measures often reported in PSD studies [e.g. 53, 59]. These discrepancies point out that, in some cases, subjects under a chronic sleep debt condition may dangerously underestimate their sleepiness level and its consequence on cognitive functioning. In other cases, however, the subjects undergoing sleep curtailment seem to be quite able to subjectively detect increasing levels of fatigue and sleepiness [33, 57, 60, 62].

As already mentioned before, strictly speaking the results of most PSD studies should be attributed to an interaction between the effects of sleep curtailment and those of the delay of habitual sleep onset. Such a delay has been reported to negatively affect at least mood [40, 41]. However, it has to be stressed that, in an experimental comparison of the effects of two different PSD paradigms (one obtained by postponing bedtime by 4 h, the other by awakening subjects after the first 4 h of sleep initiated at habitual bedtime), no significant differences in performance deficits were found [37]. Consequently, the adverse effects on both objective and subjective measures reported in the sleep-delayed PSD paradigms [e.g. 43, 47] cannot be simply due to the postponed sleep onset. Nevertheless, in any PSD study the possibility that the disruption of the individual sleep–wake rhythm may co-determine the outcomes does definitely exist, and does not seem to be avoidable.

Moreover, although the reviewed PSD studies often selected young normal subjects habitually sleeping 7.0–8.5 h per night, with only two exceptions [53, 59], it has to be recognized that people showing the same habitual sleep schedule (e.g. 7.5 h of sleep per night, between 23:00 and 07:00) may greatly differ as far as the fulfillment of their individual sleep need is concerned. Because, in the studies reviewed above, the possible discrepancy between habitual sleep schedule and individual sleep need has never been assessed (or reported), it is not possible to ascertain whether or not the evaluated subjects were in a chronic sleep debt condition. Most of these methodological considerations also apply to sleep extension studies.

THE EFFECTS OF SLEEP EXTENSION ON SUBSEQUENT ALERTNESS AND PERFORMANCE: ARE THERE ANY BENEFITS IN SLEEPING LONGER?

Another possible approach to shed more light on the sleep need issue is provided by studies in which nocturnal sleep is extended beyond its habitual duration. There is experimental evidence that subjects normally sleeping 7–8 h per night are able to obtain up to 3.5 h of sleep more than their usual quota [e.g. 72]. However, as already pointed out in the PSD section, individual basal sleep schedule, habitual sleep length and timing are basic sleep characteristics to be carefully considered during the screening and selection of experimental subjects, since they can considerably influence the outcomes. This is particularly true in sleep extension studies, since anecdotal and objective evidence shows that the ability to sleep 10 h or more is a distinctive feature of only some individuals [e.g. 73]. As an example, Webb [74] recorded nocturnal *ad libitum* sleep of 14 pairs of fraternal twins (mean age: 19.6 years) and 14 pairs of identical twins (mean age: 18.6 years), reporting that only eight out of 56 subjects were able to sleep more than 10 h, and only four completed 11 h of sleep. In another study on the EEG features of extended sleep, two out of eight subjects habitually sleeping 7–8 h, but reporting the ability to voluntarily stay asleep much longer [75], were unable to extend sleep and were thus excluded from the analysis.

If subjects who report being able to sleep more than 10 h do not necessarily succeed, it becomes clear how difficult sleep extension can be for people not used to it. Differences in subject sampling criteria and methodological choices for the timing of the extended sleep may in part explain some of the contrasting results in this field. As an example, detrimental effects of 2 nights of extended sleep have been reported on both a vigilance and a complex motor performance task [76]. An increase in the number of misses and in reaction times in a vigilance task has also been shown after 1 night of sleep allowed between 21:00 and 08:00 [43], as compared to an 8-h control condition. These effects were not seen in the morning session, but became evident in the afternoon. Negative affects were also greater after sleep extension. It must be stressed

that, when at home, the 10 subjects selected for this study habitually retired at about midnight and had 7–8 h of sleep per night. In the sleep extension condition, they managed to sleep 9.3 h (vs 7.1 h during baseline), but their mean sleep onset latency rose to 55 min (vs 13.6 in baseline) because of the advanced bedtime. Similar results have been reported, in a sample of 16 subjects with the same sleep habits as in the previous study, 90 min after awakening from an *ad libitum* sleep extension [77]: performance on a four-choice reaction task, an auditory vigilance task and a short-term memory task was worse, and subjective sleepiness was higher, compared with a control condition. In this case, however, in the *ad-lib* sleep extension condition, subjects were free to go to bed and to rise at their own inclination. As a consequence, retiring time greatly varied among subjects (between 00:08 and 04:30), as did rising time (09:30–14:30). Although these subjects slept 2.1 h more than in the baseline condition, the interaction between changes in individual sleep habits (the participants habitually slept 7–8 h with a mean bedtime around midnight), delays in bedtime and rising time and the sleep extension itself, makes any interpretation of the results difficult.

Clearly, at first sight, these outcomes could raise the question of whether an extra sleep quota is really effective in ameliorating daytime sleepiness and performance. On the other hand, however, some other studies found an improvement, at least in daytime sleepiness (measured by MSLT) and in a reaction time task, following extended sleep. Nine young subjects (mean age = 19), habitually sleeping 7.5–8.5 h, extended sleep by advancing lights-out time (time in bed between 22:00 and 08:00) for 4 consecutive nights [51]. Gradually increasing MSLT scores on the days following sleep extension were found, although they were significant only after the 2nd extension night. The authors claimed that this finding "supports the suggestion that an 8-h bed time represents a 'chronic sleep deprivation' condition in young adults", even though sleep extension resulted in a decrease of sleep efficiency compared with baseline (91% vs 85%). Unfortunately, no performance measures or mood evaluations were collected. In another study [78], 12 "sleepy" subjects (with a basal average MSLT latency of 6 min) and 12 "alert" subjects (with a basal average MSLT latency of 16 min) extended their time in bed to 10 h for 6 consecutive nights by advancing bedtime (time in bed: 21:30–07:30). At home, the sleepy group (mean age: 25.2 years) habitually slept 7 h (between 00:06 and 07:30), while the alert group (mean age: 23.8) reported a 7.55 h total sleep time (between 00:10 and 08:00). Data on sleep structure during the extension nights were unfortunately not provided, although a decrease of sleep efficiency was found only in the sleepy group (a mean of 89.1% vs 95.4% in baseline). Nevertheless, the sleepy subjects showed an immediate increase in MSLT latency at 1000 h, reaching a plateau after the 3rd extension night; the alert group, after a slight shortening of MSLT latency after the first extension night, showed a small (<2 min) but significant improvement only after the 6th night. Small but significant improvements were also found in reaction times (approx. 10% reduction), but no effect on a tracking task. The authors admitted that practice effects could have been present. In a replication of this study, an extension to 10 h for 14 consecutive nights (time in bed: 21:30–07:30) was effective in increasing to about 10 min the mean daily MSLT latency in a group of "sleepy" subjects [79]. However, some sleepy subjects did not benefit at all from the extension paradigm, their mean MSLT latency remaining at 8 min or less. In a more recent study, 11 subjects (habitually sleeping 7–8 h per night, between 00:00 and 07:30) extended their sleep up to 10 h for 14 consecutive nights, monitored by actigraphs, by advancing bedtime to 22:00 [80]. Interestingly, two subjects found it very hard to adapt to extended sleep, and one of them withdrew after the 4th extension night, complaining of unacceptable levels of sleep disturbance (frequent awakenings, difficulty with reinitiating sleep). An EEG was recorded on the 7th and 14th extension nights, showing that total sleep time increased by about 1 h for both nights as compared to baseline, as well as sleep onset latency and intra-sleep wake. A clear decrease in delta activity, particularly during the first cycle, was also shown by spectral analysis. Multiple Sleep Latency test scores showed a small (1 min) but significant improvement during sleep extension, but only at 1600 h. On the other hand, no improvements in self-rated mood or sleepiness were found. A small (12%) but significant reduction in reaction times recorded in an auditory vigilance task were also reported, while the ability to detect target tones did not change significantly. As observed by Harrison and Horne [6, p. 902], "performance improvements of this magnitude have little established ecological validity". In a classic study of sleep extension [81], Gagnon *et al.* selected 10 young

subjects, usually sleeping 7–9 h but occasionally able to sleep for 12 h, to evaluate the effects of different sleep extension paradigms on sleep structure. In a first experiment, the above-mentioned subjects were asked to sleep 15 h consecutively (lights off at midnight), while in a second experiment bedtime was delayed to 04:00 and the same subjects were asked to try to sleep until 19:00. In a third experiment, 10 different subjects, with the same sleep habits as before, after 3 nights during which a 7–8 h sleep was scheduled between 04:00 and 23:00–00:00, were again asked to sleep 15 h, between 04:00 and 19:00. In the first two experiments, eight out of 10 subjects were able to sleep between 12 and 15 h, while in the third only six out of 10 were successful. Although sleep-onset latencies during the extension nights were not reported, the hypnograms clearly showed that they never exceeded 30 min; wakefulness ranged between 102 and 137 min in the three conditions. Subjective evaluations of sleep quality were significantly lower following the prolonged nights than following the baseline sleep, irrespective of the experiment. On the other hand, sleep extension did not negatively affect mood. Finally, Wehr *et al.* [82] submitted, for 4 weeks, 16 healthy young subjects to a short-day regime with 10 h of light (activity permitted) and 14 h of dark during which participants were instructed to remain at bed rest and to sleep whenever possible. Subjects' habitual sleep schedules were not reported; one of them became severely depressed during the long-nights period and was removed from the experiment. Subjects slept an average of 10.6 h in the long nights, compared with 7.6 h in the short (8 h) "control" nights. After transfer from short nights to long nights, the individuals reported being less fatigued, happier and more energetic. The increased total sleep time and the improved self-reported vigor and mood were interpreted by the authors as evidence of a pre-existing sleep debt. However, in the 4th week of long nights, compared with a week of short nights, there were significant increases of sleep onset latency, wake-after-sleep onset and advances of the awakening time. Moreover, during the long-nights period there was a gradual decrement of mean total sleep time, which was of about 8 h during the last week of the study. Consequently, after the recovery of a possible pre-existing sleep debt obtained in the first 10–14 days of sleep extension, an additional 6-h opportunity for sleep led only to an extra 30–60 min of sleep. Furthermore, the mood improvements attributed to the sleep extension could also be due to the advance of sleep timing with respect to habitual sleep times [40]. Finally, as noted by the authors, the possible confounding effects of darkness *per se*, enforced bed rest and confinement to a boring environment remain to be determined. On the whole, the reviewed data do not seem to support the idea that extending sleep by 2 or 3 h per night may provide clear advantages to the following daytime functioning of an average healthy subject. Only some individuals seem to be able to extend their sleep satisfactorily [e.g. 73], while others may find it so hard and uncomfortable that, in some cases, they withdraw from the study [e.g. 80, 82].

The only well-established effect of sleep extension is a slight increase in the average daytime MSLT latency, comprised between 1 and 5 min, while the effects on performance are at least contradictory. However, the increased MSLT scores in the so-called sleepy subjects do not prove the need for extra sleep in the average individual as these subjects, with baseline MSLT scores of about 6 min, can be considered relatively unusual [80]. On the other hand, the so-called alert subjects do not seem to represent such an extreme level of alertness. With baseline MSLT scores of about 16 min, they should be considered as normal individuals [6], although there is still confusion about the cut-off point to differentiate normal from abnormal daytime sleepiness, and the sensitivity and accuracy of MSLT has been questioned [e.g. 83]. Nevertheless, these subjects are poorly sensitive to the alerting effects of sleep extension, indicating that subjects showing normal levels of daytime sleepiness may get minimal benefits by sleeping more than their norm.

In any case, as already pointed out, the subjects' habitual sleep length and timing, as well as their peculiar self-reported ability to sleep for long periods of time (>10 h), may have greatly influenced the outcomes of the sleep extension studies. Some of the negative effects of sleep extension might have been related to the considerably delayed awakening time typically seen in the *ad libitum* sleep conditions [e.g. 77, 81]. A circadian dysrhythmia [78] resulting from the change in sleep schedule is a possible explanation for these results. On the other hand, extending sleep by advancing bedtime seems to cause only slight positive effects on objective sleepiness and performance [51, 78–80] and no negative effects on self-rated mood and sleepiness [80]. However, in these cases, a clear increase of sleep onset latency is typically observed [43, 80], as

individuals who habitually go to bed around midnight are forced to suddenly advance their bedtime. In conclusion, we agree with other authors [6, 25, 80] that the available evidence suggest that extending the 7–8-h normal sleep by 2 or more hours produces only marginal advantages for the sleepiness and performance of an average individual. As a result, it may be claimed that a mean 7–8 h daily sleep quota is enough for most people. Only subjects habitually curtailing their sleep compared to their actual need, such as young students [e.g. 51], can significantly take advantage of sleeping longer. The slight improvement of MSLT scores and reaction times reported in the so-called sleepy subjects [78, 79] after several days of sleep extension do not prove, in our view, that those subjects live under a condition of chronic sleep debt. In fact, the only distinctive feature of sleepy subjects is a short (6 min) average daily sleep latency on the MSLT, notwithstanding an average 7–8 h of sleep nightly. However, they do not complain of daytime sleepiness; nor do they claim to sleep less than they need. As showed by Harrison and Horne [80], even subjects with high mean baseline MSLT scores (16 min) are able to extend their habitual sleep by 1 h. Consequently, this ability *per se* cannot be invoked in support of a chronic insufficient sleep. Finally, it must be emphasized that the ecological validity of a 10–12% shortening of reaction times and a 1–2 min lengthening of MSLT sleep latencies is, at least, doubtful [80]. Sleep extension does not seem to be a viable solution to increase the alertness of the average individual also because, in real life, the disruption of habitual work and social schedules and the loss of 2 h of productive wakefulness would not be outweighed by the (few) accrued benefits.

INDIVIDUAL DIFFERENCES IN SLEEP NEED: TOWARDS A SOMNOTYPOLOGY?

When debating whether we should sleep more or less than our average, it should be borne in mind that, although most people sleep an average of about 7–8 h per night, sleep need, as many other variables, shows a continuous (presumably gaussian) distribution. Consequently, regardless of the different schools of thought about the actual sleep requirement, it should be recalled that each individual has his/her own sleep need. Unfortunately, individual differences have scarcely been taken into account in the scientific controversy on the possibility that our society is sleep deprived [6–7].

Gender

It has been consistently reported that women have a greater need for sleep than men [e.g. 18]; longer sleep durations among adult women have been documented by both subjective and objective reports [e.g. 22, 84]. A significantly longer self-reported total sleep time and a higher sleep need among females has been found [85] also in a questionnaire-based study of 529 young subjects (mean age 32.4 years). In this case, even the difference between the two above-mentioned variables was significantly higher among women, indicating a higher level of sleep debt. Home-sleep monitoring by logs and wrist actimetry in a sample of 400 adults confirmed that sleep period time was markedly longer for women [86]. Moreover, women retired to bed and fell asleep earlier than men and reported more awakenings, more total time spent awake during the night and poorer sleep quality. More recently, the sleep polygraphy of 52 healthy subjects was recorded at night in their homes, while they were following their normal daily activities and habitual sleep schedules [87]. Men between 35 and 70 years showed more stage 1 sleep than females. On the other hand, females had more SWS than males, particularly in the oldest subjects (50–70 years). Although an increase of SWS can be considered as a marker of increased sleep need [e.g. 27], the lack of significant gender difference in total sleep time is in disagreement with longer sleep durations among women previously reported by means of questionnaires and actigraphic recordings [22, 85–86].

Age

As far as the effects of aging on sleep need are concerned, it is well known from questionnaire surveys [e.g. 18], actigraphy monitoring [e.g. 86] and polysomnographic recordings [e.g. 88] that the adult life-span is characterized by a general decline of sleep duration as a function of age. The changes in sleep duration in infancy have been largely documented [e.g. 89]: from an average total sleep time of about 16 h in the first days of life, sleep duration falls to about 14 h by the end of the first month, and then declines more slowly to about 12 h by the

sixth month. There is a slow decline of about 30 min per year from age 1 through 5. Beyond age 5, total sleep duration becomes an indirect measure of sleep need, due to the intervention of external factors, as school [89]. There is a further slow decline of sleep time from age 5 to age 10, and then from adolescence levels of about 9–10 h to adult levels of about 7–8 h [90].

Increasing age is also related to earlier bedtime and waketime, decreased time in bed and total sleep time, as well as to higher morningness scores [86, 88]. A recent meta-analysis on age-related changes in sleep [91] confirmed that aging negatively affects sleep maintenance and sleep length. On the other hand, a small correlation between age and sleep latency was found. Moreover, the sleep of older people is generally less consolidated and SWS, SWA and spindle activity are attenuated [e.g. 92]. It is currently unclear whether these age-related changes in non-REM sleep homeostasis merely reflect changes in the EEG generating mechanisms, or a reduced efficiency of the sleep process, or whether they indicate an age-related attenuation of the build-up of sleep propensity during wakefulness (sleep need). However, it must be stressed that the homeostatic processes are still functioning in older subjects, since they respond to sleep loss with an increase in both SWS and SWA [e.g. 93].

In a series of studies over the last few years, Dijk *et al.* have further investigated the specific contributions of circadian and homeostatic components to age-related changes in sleep [for a review, see 93]. In a constant routine protocol it has been confirmed that the phase of the human circadian pacemaker is advanced in older people, while its intrinsic period does not show an age-related reduction [94]. Recently, in a study of the circadian variation of sleep propensity during forced desynchrony, a marked reduction in total sleep time and of sleep consolidation was observed in older people in all circadian phases [95]. However, the shortest sleep latencies, although normally located around the temperature nadir, were longer in older people, suggesting an attenuation of the circadian drive for sleep limited to that circadian phase (i.e. early morning hours). According to the authors, age-related changes in sleep duration and consolidation appear to be due to an interaction between a reduction of the homeostatic drive for sleep and a reduced amplitude of the circadian signal promoting sleep in the early morning hours [95].

Morningness–eveningness

The morningness–eveningness dimension can be considered an important predictor of the individual's habitual sleep patterns. As an example, in the Carrier *et al.* study [88], carried out on 110 adults aged between 20 and 59 years, after controlling for the effect of age, morningness was associated with earlier bedtime and waketime, less time in sleep and less REM sleep, and better alertness upon awakening. A very recent survey on 617 subjects between 17 and 80 years [96] confirmed and extended the Carrier *et al.* findings [88] by showing that evening-types (E-types) express a greater sleep need, but they tend to show a reduced weekday sleep more than morning-types (M-types). In fact, 11.4% of this group slept only 75% of their ideal sleep duration in the course of the week. Moreover, during weekends E-types delayed their sleep–wake schedules and slept longer than the M-types. Notwithstanding the cumulative partial sleep loss and the irregular sleep–wake schedules, the E-types did not rate themselves to be sleepier than the M-types. As far as the laboratory findings are concerned, M-types tend to show higher nocturnal sleep efficiency than the E-types, while the latter group sleep longer during the morning after sleep deprivation [97]. Sleep tendency (measured by MSLT) in the E-types is significantly higher in the morning hours, even after an 8-h sleep [98]. Morning and evening persons have an early and late "sleep gate" respectively, evidenced in an ultrashort 7-min sleep/13-min wake cycle [97]; this phase difference of 2 h in the circadian sleepiness/alertness rhythm [99] seems to be a stable individual psychophysiological trait with high intra-subject consistency. It has been recently shown that M-types (or larks) have advanced melatonin and core body temperature rhythms as compared to E-types (or owls); however, the interval between the awakening time and the temperature nadir and melatonin crest is longer in M-types, that is, larks wake up at an earlier clock time but at a later circadian phase [99, 100]. The latter finding can explain the higher levels of alertness upon awakening reported by M-types [88], and may be related to a shorter intrinsic period of the circadian pacemaker [101]. The circadian phase under entrained conditions has been recently associated to human *CLOCK* gene polymorphisms, that is, genetically determined

changes in the period of the circadian oscillator could eventually result in different eveningness–morningness preferences [102].

On the whole, these results show that the E-types may express a greater need for sleep, accompanied by a greater plasticity of the sleep–wake cycle leading to less sensitivity to sleep restriction. This could explain why they adapt better than other chronotypes to shift work [e.g. 103]. Conversely, the earlier endogenous circadian phase of M-types [97, 100] can give a biological basis of why they are scarcely suited to adapting to night work [99]. A genetic component for the owl-lark behaviour has also been suggested [102].

Long and short sleepers

It has been recognized for some time that individuals do exist who naturally sleep far more or less than the average. These subjects, placed at two ends of the presumably continuous sleep-need distribution, are referred to as long and short sleepers [e.g. 104]. The psychological characterization of these subjects is beyond the interests of the present paper [e.g. 105]. However, it seems noteworthy that a reduced sleep need in the short-sleepers has been postulated, having ruled out the possibility that they are chronically sleep deprived [106]. Hartmann *et al.* [106] proposed that long sleepers should actually require more REM sleep than short sleepers, this differential REM-sleep-need being possibly related to personality and psychological state.

The ability to extend sleep far beyond the norm seems to be an individual psychophysiological trait. Subjects habitually sleeping 7–9 h per night but able to extend their sleep beyond 10–11 h under normal circumstances (i.e. when not sleep deprived) and at their will have been called "sleep extensors" [73, 107]. A comparison between sleep extensors and a group of subjects who had comparable sleep habits but claimed to be unable to sleep more than 9 h, showed that the circadian typology is the major trait discriminating the two groups, the "extensors" being more E-type [73]. Moreover, they indicated a greater sleep need than the "normal sleepers" to feel fully refreshed (9.0 h vs 7.7 h). Recently, the question has been addressed of whether short sleepers have a lower sleep need than long sleepers, or whether they simply have a higher tolerance to sleep pressure [54]. Sleep structure and SWA (as a marker of the homeostatic sleep process corresponding to non-REM sleep pressure) of nine carefully screened habitual short sleepers (<6 h) and seven habitual long sleepers (>9 h) have been studied during 1 night of baseline sleep, as well as during 2 nights of recovery sleep following 1 night of sleep deprivation. Subjects filled in a sleep log during the 2 weeks before the experiment: long sleepers went to bed $\sim$1.5 h earlier and woke up $\sim$2.5 h later than the short sleepers. Both groups delayed their sleep period during the weekend but, interestingly, only short sleepers slept longer during weekends (6.8 h vs 5.5 h during weekdays). The baseline night of the long sleepers was characterized by a significantly longer sleep onset latency and a lower sleep efficiency compared to the short sleepers. During each of the three laboratory nights, long sleepers showed a much longer total sleep time (>3 h) than short sleepers, but the SWS amount did not significantly differ between the two groups in any of the nights. However, a further analysis of the longest common sleep interval (first 293 min) showed that the short sleepers had more SWS and less stage 2 than the long sleepers. These findings, together with the shorter sleep onset latency, support the hypothesis that short sleepers live under a higher non-REM sleep pressure than long sleepers. This interpretation is further supported by the faster rise in sleep spindle activity (power density in the 12.25–15 Hz range) and its earlier peak in the short sleepers, since similar changes are typically seen after sleep deprivation. Moreover, the higher levels of SWS and SWA in the final sleep cycle point out that short sleepers are still under higher non-REM pressure than long sleepers at the end of their sleep period. As far as the response to sleep deprivation is concerned, larger relative decreases of sleep latency and increases of sleep efficiency were observed in the long sleepers, while a reduction of REM sleep duration during the first recovery night was present only in the short sleepers. In addition, EEG power spectra in the low-frequency range were more enhanced and the sleep spindle activity was more reduced in the long sleepers. The estimated time constants, that is the decay rate of SWA in the two groups, did not significantly differ. This implies that long and short sleepers have identical homeostatic sleep regulatory mechanisms. Short sleepers, however, "live on a higher level of

(Process) S, i.e. they initiate sleep on a higher level of S than short sleepers, and they also terminate sleep on a higher level of S" (Process S defined as the sleep-dependent homeostatic process) [54]. Nevertheless, they are able to tolerate a higher non-REM sleep pressure, showing a lower responsiveness to sleep loss as indicated by both sleep parameters (sleep latency, sleep efficiency, SWA and REM density) and subjective ratings of energy and fatigue.

Sleepy–alert subjects

Lavie and Zvuluni [108] found that two main aspects of the sleep propensity function have highly significant within-subject stability: the timing of the nocturnal "sleep gate" (i.e. the beginning of maximal sleep propensity), and the overall level of sleepiness. The former variable is strongly related to the morningness–eveningness dimension, with M-types showing an earlier sleep-gate timing. According to the latter variable, individuals can be categorized along the sleepy–alert dimension. Sleepy subjects [see also 78, 79] have shorter sleep latencies and higher sleep efficiency during the night, and sleep more during the day in an ultrashort sleep–wake paradigm. Moreover, these subjects have a very short daytime sleep latency (6 min) even after 8 h of nocturnal sleep; however, they do not complain of excessive daytime sleepiness or report napping during the day [78, 79]. Are sleepy subjects experiencing a chronic insufficient amount of sleep relative to the individual's biological sleep need [e.g. 79], or do they simply show a peculiar ability to sleep as they wish? The so-called sleepy subjects seem to be comparable to those with "high sleepability without sleepiness" recently described by Harrison and Horne [109]. These subjects show low MSLT scores even after *ad libitum* nocturnal sleep, no evidence of daytime napping or complaints of daytime sleepiness and normal levels of performance to a vigilance task. The low MSLT scores persist even after they are given *ad libitum* sleep at night [109]. Taken together, these findings point to the existence of an individual peculiar ability to fall asleep quickly, related to the stable individual trait in sleep propensity shown by Lavie and Zvuluni [108]. This ability to switch off quickly is not necessarily related to a previous sleep debt, as the sleepy subjects do not complain about sleeping less than their need.

In conclusion, large and often unexplored individual differences do exist among individuals in their need for sleep. The proposal by Lavie and Zvuluni [108] of a somnotypology seems, indeed, to be quite interesting. Age, and, to a lesser extent gender, should be considered as important modulating factors of the individual need for sleep. The position along the short/long-sleeper dimension should also be taken into account in an individual multitrait somnotypology. Finally, the phase of the circadian sleepiness–alertness rhythm and the period of the circadian pacemaker, which has been suggested to be genetically determined resulting in different morningness–eveningness preferences, are to be considered as major determinants of the individual need for sleep, providing the biological substratum of such a somnotypology.

CONCLUSIONS

The search for the minimal sleep amount necessary to maintain high levels of task performance and efficient functioning for long periods of time [110], somewhat resembles the search for the philosopher's stone. The concept that an optimal value for sleep obtained exists is not universally accepted, nor is it clear what "optimal" means [30].

The increasing concern for the risks associated with self or society-imposed restrictions of sleep [e.g. 3] stems from the idea that we are chronically sleep deprived [7, 9]. In fact, in western societies the lifestyles and social or economic constraints easily lead to shorten our sleep time during the weekdays. Many people have very early work times: to obtain a 7–8 h sleep period, it would be necessary to go to sleep in the early evening, restricting social and family life. Moreover, the widespread implementation of shift-work to facilitate around-the-clock production is contributing to challenge the traditional human sleep–wake activity. In all these cases, a chronic curtailment of the sleep period is hardly avoidable, giving way to a gap between individual sleep need and habitual sleep schedule.

On the other hand, from a diametrically opposite position, some sleep researchers suggest that the optimal sleep amount may be 6 h per night or less, and that the rest of our sleep is optional [24, 25].

Thus, from the latter point of view, our society is not at all chronically sleep-deprived and, consequently, we do not need to have more sleep [6]. As suggested by Bonnet and Arand, "perhaps a more profitable way of viewing sleep is as a logarithmic process where large benefits accrue in initial hours but increasingly small benefits continue to accrue as the length of the sleep period increases" [7, p. 910]. This view is confirmed by a recent cross-study comparison [71] reporting that performance shows an exponentially saturating recovery during sleep. Such an asymptotic recovery can explain why performance and sleepiness are often near baseline levels after a single night of sleep curtailment [e.g. 42, 48, 51]; it also explains why sleep extension does not induce substantial benefits [e.g. 78]. As a result, it can be claimed that a mean 7–8 h daily sleep quota is enough for most people. Only subjects habitually curtailing their sleep compared to their actual need, such as young students [e.g. 51], can significantly gain some benefit from sleeping longer.

The experimental evidence reviewed here suggests that the truth about sleep need lies between the two above-mentioned extreme positions [30]. The average 7–8 h daily sleep quota is probably enough for most people. However, a large and robust body of data indicates unequivocally that even a relatively modest sleep curtailment of that average sleep amount (e.g. 2 h) leads to impaired levels of alertness and performance [42, 45]. There is also clear evidence that, at least in the short term (up to 1 week), cumulative effects of sleep restriction are seen on several measures of performance and vigilance [37, 42, 51–53, 55, 56]. Detrimental effects of PSD seem to accumulate linearly, although a step-like function in sleepiness and performance deficit growth has also been suggested [58]: in this case, after an initial drop in alertness and performance, a kind of adaptation to sleep restriction should occur, then followed by a further sleepiness and performance worsening.

As already said more extensively before, the (sometimes contradictory) outcomes of the long-term sleep restriction studies are to be considered with caution, given their several methodological weaknesses. Although more careful experiments should be performed to better clarify the long-term effects of partial sleep deprivation, it must be recalled that a recent, well-controlled PSD study reported strong cumulative neurobehavioural deficits after as little as 5 days of sleep reduced to 6 h per night [59]. If confirmed, these findings should bring strong contrary evidence to the idea of a "core sleep" of about 5–6 h per night. Moreover, it has been recently shown that PSD negatively affects several metabolic, endocrinological and immunological parameters [66–69], pointing out that chronic sleep restriction may also have serious long-term adverse health effects. Given these premises, the recent epidemiological actigraphic-based data [22] reporting that the average sleep duration for 273 adults living in San Diego is only 6 h 13 min deserve much attention.

On the other hand, the demonstration of several negative effects of PSD should not automatically lead to posit the need for extending sleep beyond the norm. This logical conclusion is strengthened by results coming from the studies of sleep extension. In fact, several negative effects of sleep extension on subjective sleepiness, mood, vigilance and performance have been reported [43, 76, 77], but slight but statistically significant improvements of daytime sleepiness and reaction times have also been found [51, 78–80]. However, the practical implications and the ecological validity of such small improvements still remain to be established. It can be concluded that, on a cost-benefit analysis, extending sleep beyond the individual's norm will produce only minimal benefits which are "out of proportion with the actual costs to the individuals in terms of reduced active wakefulness and deterioration in sleep efficiency" [6, p. 906]. Only those habitually curtailing their sleep compared to their actual need (e.g. very young students, shift-workers), and, obviously, subjects biologically suited to extend sleep (i.e. long sleepers, sleep extensors) can significantly benefit from sleeping longer.

Finally, the two opposite positions in the "sleep-need debate" do not properly take into account that large interindividual differences do exist in the need for sleep. If the 7–8 h average sleep is enough for most people, some recent epidemiological findings suggest that a substantial part of the adult population have a sleep quota below the average. We should also bear in mind that smaller parts of the population may need to sleep longer than the average. Apart from the long recognized natural long and short sleepers, other psychophysiological and chronobiological dimensions (with a biological and genetic substratum) should be considered as determinants of the individual sleep need, such as both the sleepy–alert and the morningness–eveningness continuum.

Thus, it may be somewhat confusing to search for *the* sleep need. The best way to determine an *individual* sleep need is still to go to bed when tired and sleepy, and to get up in the morning when feeling refreshed, without any alarm [7]. Any sleep length less than this empirically defined sleep need will presumably lead to a sleep debt and to its consequences in terms of sleepiness and reduced performance capability, especially when this debt becomes a chronic condition.

Research Agenda

1. Dose-response studies to elucidate the relations between sleep duration and level of sleepiness and performance using a multidimensional approach (i.e. taking into account several different subjective and objective measures) are needed.
2. The cumulative effects of different schedules of gradual sleep restriction on a wide array of tasks should be extensively assessed in strictly controlled experimental settings.
3. Learning effects should be carefully avoided by extensively training subjects before the study, or, alternatively, by choosing tasks not affected by learning.
4. The shape of the functions (linear, step-like, exponential) of the cumulative effects of sleep restrictions of different lengths and durations should be investigated, in order to predict them in applicational settings.
5. Studies of sleep extension on subjects showing the largest difference between weeknight and weekend sleep duration should be carried out, to evaluate the real effectiveness of sleeping longer in people habitually curtailing their sleep.
6. An individual multi-trait somnotypology should be outlined, taking into account the following variables: age; gender; morningness–eveningness; position along the sleepy–alert and short/long-sleeper dimensions.

Practice Points

1. A scientifically based controversy does exist on whether the average daily sleep quota of 7.5 h is enough, and whether daytime alertness and performance can be improved by sleeping longer.
2. There is increasing concern for sleeplessness-related risks in modern society, as the accidents primarily caused by sleepiness and/or fatigue involve enormous costs in terms of lives and money.
3. Epidemiological, diary-based and actigraphic results indicate that insufficient sleep may be considered as a public health problem, as it persistently involves a considerable part of the population in the 17–65 years age range sleeping less than their need on a chronic basis.
4. Experimental data on the effects of both acute and cumulative partial sleep deprivation consistently point out that sleep restriction has substantial negative effects on sleepiness, motor and cognitive performance and mood, as well as on some metabolic, hormonal and immunological variables.
5. The effects of long-term partial sleep deprivation should be further investigated, as the few available studies are flawed by methodological weaknesses.
6. On the other hand, extending sleep by 2–3 h beyond the norm produces only marginal benefits for an average individual.
7. Large individual differences do exist in the need for sleep, although they have scarcely been taken into account.
8. A somnotypology, considering age, gender and the position in both the sleepy-alert and the morningness–eveningness continuum, should help in the search for the actual individual sleep need.
9. As a chronic curtailment of the empirically defined individual sleep need will lead to negative consequences in terms of sleepiness and reduced performance capability, and may have serious long-term adverse health effects, it should be avoided in the general population.

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This work is dedicated to the memory of Vincenzo Ferrara.

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