



Original Article

Effects of acute and chronic sleep deprivation on daytime alertness and cognitive performance of healthy snorers and non-snorers

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ABSTRACT

Background: Respiratory events during sleep usually lead to micro arousals resulting in consecutive daytime sleepiness even in healthy snorers. The present study investigated the evolution of subjective and objective daytime sleepiness and reaction time in healthy snorers submitted to acute and chronic sleep deprivation.

Methods: Objective sleepiness was measured by the MSLT, subjective sleepiness by the Karolinska Sleepiness Scale (KSS), and reaction time (RT) by the Psychomotor Vigilance Test. Mean sleep latencies, KSS scores and performance were analyzed through repeated measures ANOVAs with one between-factor (snorers and non-snorers) and two within-factors (sleep deprivation [baseline, acute, and chronic sleep deprivation] and time-of-day).

Results: The findings reveal that sleep deprivation does not enhance snoring but that, during baseline, objective daytime sleepiness is higher in snorers than in non-snorers (shorter sleep latencies) with no difference in subjective assessments. The effects of acute and chronic sleep deprivation on sleep are similar in both groups, but, after acute sleep deprivation, RT and attentional lapses (RT >500 ms) are higher in snorers. Chronic sleep deprivation produces similar results in both groups.

Conclusion: These results suggest that respiratory efforts may be involved in the increased vulnerability to sleep deprivation of healthy snorers when compared to non-snorers.

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1. Introduction

Snoring is a common complaint in the general population [1,2] and concerns about 40% of men and 20% of women, although the variability of this assessment is extremely large [3]. Since the occurrence of respiratory events usually leads to micro arousals in order to release upper airway resistance [4], the associated sleep fragmentation could result in daytime sleepiness even in healthy (non-apneic) snorers [5]. The complaint of excessive daytime sleepiness (EDS) associated with heavy nocturnal snoring usually leads to suspected and detected obstructive sleep apnea syndrome (OSAS). Clinical investigation, however, may not necessarily always demonstrate the presence of OSAS. Guilleminault et al. [6] showed that EDS could also occur in healthy snorers, with no OSAS, but with a decreased diurnal sleep latency in the mean sleep latency

test (MSLT) [7] associated with the occurrence of EEG arousals during nocturnal sleep. This suggests that isolated snoring could lead to sleep disturbances which, in turn, could produce EDS. It has also been suggested that nocturnal breathing disorders may be promoted or have their index increased over time due to sleep deprivation. According to Guilleminault and Rosekind [8], acute total sleep deprivation would worsen sleep-disordered breathing in patients with obstructive sleep apnea syndrome. Concerning healthy subjects, a study conducted by Stoohs and Dement [9] also suggests that chronic partial sleep deprivation increases the severity of upper airway resistance. In subjects with mild or moderate sleep apnea, this study shows that cumulative sleep deprivation is associated with significantly more snoring time over night and more abnormal respiratory events and significantly lower minimal oxygen saturation value than during baseline nights.

However, the impact of sleep deprivation on snoring and alertness-performance has never been tested, to our knowledge, in snorers vs. non-snorers. Moreover, the respective effects of acute sleep deprivation vs. partial chronic deprivation has not been assessed either.

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The aim of this study was, therefore, to compare subjective and objective daytime sleepiness and cognitive performance in healthy young snorers and non-snorers after total acute or partial chronic sleep deprivation.

2. Material and methods

2.1. Subjects

Sixteen healthy males participated in this experiment, with eight snorers and eight non-snorers (demographic data are summarized in Table 1). Recruitment was conducted on the basis of a two-night home assessment through an ambulatory device (Apnealink, ResMed) which allowed for the recording of snoring, oxygen saturation, respiratory airflow and apnea–hypopnea index (AHI). The ambulatory results were manually reviewed and confirmed through an adaptation night at the laboratory with full polysomnography (PSG). In the snoring group, all the subjects had more than 100 snoring events during the night (mean: 235.33 ± 69.89). In the non-snorers group, all the subjects had less than 25 events per night (14.50 ± 2.8). As shown in Table 1, whatever the group, all the subjects had an AHI strictly inferior to five. Exclusion criteria also included cardiovascular or other chronic disease, regular use of medications, history of psychiatric illness that might interfere with sleep or cognition, and usual daytime napping. All the subjects were free of any sleep or respiratory complaints (except snoring) and reported a normal level of daytime alertness at the Epworth Sleepiness Scale [10], as reported in Table 1. They were intermediate chronotypes [11]. The protocol was approved by the ethical committee of the French North-Eastern Region (CCPPRB Est IV) and subjects gave their signed informed consent.

2.2. Polysomnography

PSG data were recorded by a computerized system (Scan LT version 1.1, Neuroscan Medical System, USA, 2001). This included a standardized montage (F3, C3, P3, O1) referenced to the right mastoid (A2), bilateral electro-oculograms (EOG) from the outer canthus referenced to the left mastoid (A1), one submental electromyogram (EMG), and electrocardiography (ECG). Airflow was measured using a nasal canula (Apnealink, ResMed). Respiratory effort was assessed by inductance plethysmography and oxygen saturation was recorded by a transcutaneous finger pulse oximeter. Sleep apnea was defined as flow reduction comprised between 0% and 20% and hypopnea was defined as flow reduction of 50% and 3% oxygen desaturation. Flow limitation event was computed according to the definition of Hosselet et al. [12]. The sleep structure was analyzed through visual scoring, following the standard procedure defined by Rechtschaffen and Kales [13], and the criteria for EEG arousals were defined by the American Sleep Disorders Association [14]. The sleep parameters used were total sleep time (TST), sleep efficiency (SE), wake after sleep onset (WASO), latency, and percentage of the different sleep stages

(stages 1 and 2, slow wave sleep [SWS], and rapid eye movement [REM] sleep). Respiratory events were scored according to the criteria of the American Academy of Sleep Medicine [15]. We measured apnea–hypopnea index (AHI), number of snoring events, air flow limitation events, and oxygen saturation (SaO_2).

2.3. Protocol

All subjects underwent an adaptation night and an 8-h baseline night (22:30–6:30) followed by two different sleep deprivation schedules. The patients were given the recommendation to maintain regular schedules at home the week before assessment.

- The acute sleep deprivation phase (ASD) included one night of acute total sleep deprivation followed by an 8-h recovery night (22:30–6:30).
- The chronic sleep deprivation phase (CSD) included five nights of partial sleep deprivation (4h of time in bed, 2:30–6:30) followed by an 8-h recovery night (22:30–6:30).

Each sleep deprivation phase was separated by a minimum of three weeks.

Whatever the night schedule, the next day subjects performed four daytime sessions, including MSLT, the Psychomotor Vigilance Test (PVT), and subjective sleepiness assessment using the Karolinska Sleepiness Scale [16]. The sessions were designed as follows:

- 9:30: MSLT (20 min), PVT, KSS;
- 13:30: MSLT (20 min), KSS;
- 17:30: MSLT (20 min), PVT, KSS;
- 20:00: MSLT (20 min), KSS.

In the MSLT, to establish sleep onset after lights off, three epochs of stage 1 or one epoch of any other sleep stage were required. These criteria are more constraining than the actual parameters recommended by the AASM [17].

2.4. Psychomotor Vigilance Task

The PVT is a 10-min visual reaction time task [18]. Each trial generates the start of a clock represented by four zeros (seconds and milliseconds). Subjects are asked to stop the stream of the figures as quickly as possible. This task is sensitive to sleep deprivation and allows for the assessment of attentional lapses. In each session, the mean RT and %RT >500 ms (lapses) were recorded.

2.5. Statistical analyses

For MSLT, PVT, and KSS, a three-way ANOVA for repeated measures was applied with one between-subject factor (Group: snorer/non-snorers) and two within-subject factors:

- Night: N1 (baseline), N2 (acute sleep deprivation), N3 (recovery after acute sleep deprivation), N4–N8 (chronic partial sleep deprivation), and N9 (recovery after chronic partial sleep deprivation).
- Time-of-day: 9:30, 13:30, 17:30, and 20:00.

The nocturnal variables were analyzed using a two-way ANOVA for repeated measures with one between-subject factor (Group) and one within-subject factor (night). The dependent variables were the parameters used to describe sleep PSG: total sleep time (TST), sleep efficiency (SE), percentage stage 1, stage 2, SWS (stage 3), REM, latency of stage 1, stage 2, SWS (stage 3) and of REM, wake after sleep onset (WASO). The percentage of sleep stage changes is defined by the number of changes from one stage to the other per

Table 1
Demographic, subjective and respiratory data.

	Snorer (n = 8)	Non-snorers (n = 8)	p
Age	25.8 ± 2.6	22.4 ± 1.65	NS
Body mass index	22.1 ± 2.0	21.3 ± 2.3	NS
Epworth Sleepiness Scale	4.2 ± 1.2	4.0 ± 1.9	NS
Snoring events	235.33 ± 69.89	14.50 ± 2.8	.00001
AHI	2.44 ± 0.65	1.67 ± 0.5	NS
Flow limitation	37.77 ± 6.26	26.22 ± 6.62	NS

hour of sleep. Arousals are defined by awakenings lasting 4–15 s and the index of arousal is the number of arousals per hour.

When statistical significance was reached at the $\alpha < .05$ level, post hoc comparisons were performed using the LSD Fisher test.

3. Results

3.1. Effects of acute and chronic sleep deprivation

3.1.1. Polysomnography

After ASD (N3 vs. N1), sleep quality is enhanced whatever the group with an increase of TST ($F[1,14] = 28.66$; $p = .0001$) and SE ($F[1,14] = 22.51$; $p = .0003$). The latencies of all the sleep stages, except REM sleep, are shortened (L1: $F[1,13] = 15.33$; $p = .002$; L2: $F[1,13] = 19.99$; $p = .001$; L3–4: $F[1,13] = 11.75$; $p = .004$). There is less stage 1 ($F[1,14] = 14.78$; $p = .001$) and WASO ($F[1,14] = 15.86$; $p = .001$) and more SWS ($F[1,14] = 20.53$; $p = .0004$). The % of sleep stage changes increases in both groups in N3 compared to baseline ($F[1,13] = 4.85$; $p = 0.04$), with no difference in the index of arousal.

After CSD, there is a progressive decrease of WASO in both groups from N4–N8 ($F[4,56] = 6.14$; $p = .0003$) concomitant with an increase in stage 2 ($F[4,56] = 3.18$, $p = .02$). CSD produces trends in stage 1 of decrease only, and in SWS (stage 3) of increase only, with no statistical difference between both groups. Sleep stage latencies are not affected by CSD in either group. The comparison between N1 (baseline) and N9 (recovery after CSD) shows no difference in TST or SE, but a shorter sleep latency ($F[1,14] = 8.48$; $p = 0.01$) and SWS latency ($F[1,14] = 5.55$; $p = 0.03$) during N9 in both groups. There is also more SWS ($F[1,14] = 13.92$; $p = 0.002$) and less stage 2 ($F[1,14] = 15.08$; $p = 0.001$) during recovery after CSD in snorers and non-snorers. Whatever the group, there is an important decrease in the index of arousal during the five nights of CSD compared to baseline and to recovery ($F[6,72] = 29.09$; $p = 0.00001$) with no difference between the two latter. The same is observed for % sleep stage changes ($F[6,72] = 39.13$; $p = 0.0001$), but with increased values during recovery compared to baseline ($p = 0.00009$).

3.1.2. Respiratory events

After ASD (N3 vs. N1), events of air flow limitation are significantly decreased in both groups ($F[1,14] = 4.77$; $p = .04$).

During CSD, there is a main effect of the night in the number of hypopneas with a decrease in N4–N6 followed by an increase in N7 and N8 in both groups ($F[4,64] = 2.51$; $p = .05$). Moreover, compared to baseline (N1), there is an increase in the number of hypopneas in both groups during recovery after CSD (N9) ($F[1,14] = 4.98$; $p = .04$).

3.1.3. MSLT

Whatever the group, ASD produces a decrease in daytime sleep latency ($F[2,30] = 35.82$; $p < .00001$) with no return to the baseline values after a single recovery night (Fig. 1).

After one night of ASD (N2), there is a main time-of-day effect ($F[3,45] = 6.77$; $p < .0007$), with increased sleep latency at 20:00 compared to 9:30 ($p < .0001$), 13:30 ($p < .0001$) and 17:30 ($p < .02$).

During CSD, the ANOVA for repeated measures reveals a significant effect of the night on sleep latency ($F[6,78] = 10.41$; $p < .0001$). Compared to N1 (baseline), the sleep latency in both groups decreases progressively from N4 to N8 and returns to the baseline values after recovery (N9). There is also a main effect of the time-of-day ($F[3,39] = 5.16$; $p < .005$), with an increased sleep latency at 20:00 compared to 9:30 ($p < .002$) or 17:30 ($p < .007$) whatever the night.

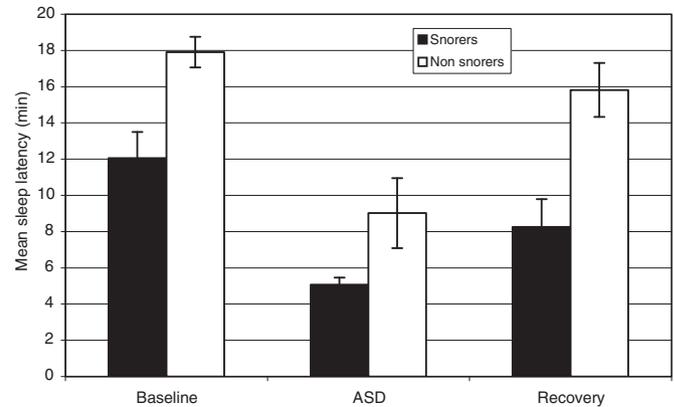


Fig. 1. Mean sleep latency (\pm SEM) in ASD after the baseline night, the acute total sleep deprivation night and the recovery night in snorers and non-snorers.

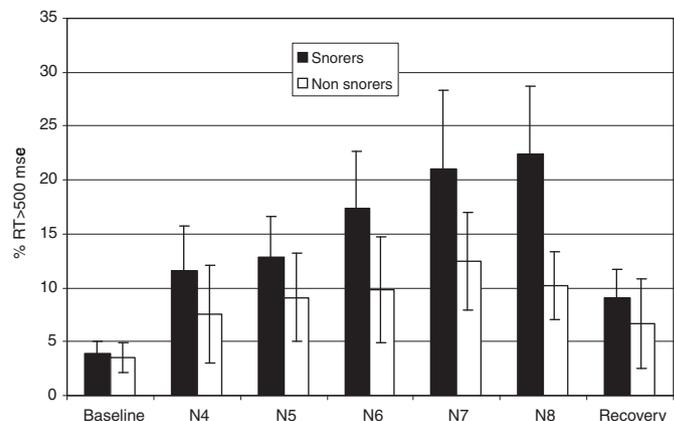


Fig. 2. Mean percentage of RT >500 ms (\pm SEM) in CSD after the baseline night, the five nights of chronic partial sleep deprivation (N4–N8) and the recovery night in snorers and non-snorers.

3.1.4. Psychomotor Vigilance Task

During ASD there is a main effect of the night ($F[2,26] = 18$; $p = .00001$), with increased RT after one night of total sleep deprivation compared to baseline ($N2 > N1$; $p = .0002$) and to recovery ($N2 > N3$; $p = .00004$). The same is observed for %RT >500 ms ($F[2,26] = 11.3$; $p = .0003$) with $N2 > N1$ ($p = .0009$) and $N2 > N3$ ($p = .002$).

During CSD, there is a general increase in RT over time ($F[6,66] = 5.56$; $p = .0001$) in both groups. After two nights of partial sleep deprivation (N5), both groups show an increase compared to baseline ($p = .02$). There is no difference between N6, N7, and N8, whatever the group. However, the post hoc comparisons show a difference in RT between N8 vs. N4 ($p = .004$) and N8 vs. N5 ($p = .02$). As shown in Fig. 2, the same profile is observed for %RT >500 ms, with an increase in both groups over the sleep deprivation period ($F[6,66] = 5.5$; $p = .0001$). However, the increase is faster than for RT since %RT >500 ms is already increased after one night of partial sleep deprivation (N4) compared to baseline ($p = .02$). It is the highest in N8 with a statistical difference compared to N4 ($p = .03$).

3.1.5. Subjective daytime sleepiness

After one night of ASD (N2), all the subjects, whatever the group, feel more sleepy ($F[2,26] = 44.16$; $p < .00001$) than after the baseline night (N1) ($p < .0001$) or the recovery night (N9) ($p < .0003$). As revealed by a significant time-of-day effect ($F[3,39] = 9.38$; $p < .00009$), sleepiness is higher at 20:00 compared

to the rest of the day ($p < .0001$), but only after the night of sleep deprivation as suggested by the night \times time-of-day interaction ($F[6,78] = 2.26$; $p < .04$).

During CSD there is a significant night effect ($F[6,78] = 15.40$; $p < .0001$), with higher scores of subjective daytime sleepiness over the whole period in both groups compared to baseline and recovery (Fig. 3).

3.2. Effects of snoring

3.2.1. Polysomnography

Table 2 summarizes the PSG data. At baseline (N1), as well as during recovery from ASD (N3 vs. N1), no statistical difference is found regarding PSG parameters between snorers and non-snorers.

During CSD (N4–N8), there is a progressive increase of TST and SE (TST: $F[4,56] = 4.82$; $p = .002$); SE: ($F[4,56] = 5.23$; $p = .001$), which is greater in snorers than in non-snorers for TST as suggested by the significant group \times night interaction ($F[4,56] = 3.05$; $p = .02$).

The sleep fragmentation indices (index of arousal, % sleep stage changes) are similar in both groups whatever the experimental condition.

3.2.2. Respiratory events

At baseline (N1), snorers do not differ from non-snorers regarding AHI (2.44 ± 0.65 vs. 1.67 ± 0.5) and the number of oxygen desaturation events (7.89 vs. 7.78). Snorers exhibit 235.33 ± 69.89 snoring events vs. 14.5 ± 2.8 for non-snorers ($F[1,14] = 12.99$; $p = .00001$).

ASD does not induce any difference between groups with regard to respiratory events.

After CSD, the comparison between baseline (N1) and recovery (N9) reveals an increase during N9 in the number of air flow limitation events in snorers only, as suggested by the group \times night interaction ($F[1,14] = 4.33$; $p = .05$). But whatever the group, sleep deprivation does not enhance snoring.

3.2.3. MSLT

MSLT applied after N1 (baseline) shows a significant main effect of the Group ($F[1,15] = 13.67$; $p < .002$) with a shorter sleep latency in snorers (12.05 ± 2.09 min) compared to non-snorers (17.89 ± 1.45 min).

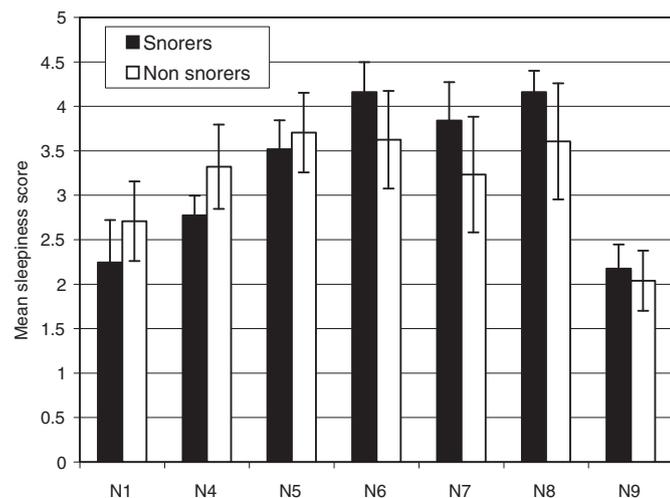


Fig. 3. Mean sleepiness score (\pm SEM) in the Karolinska Sleepiness Scale after the baseline night (N1), the five nights of chronic partial sleep deprivation (N4–N8) and the recovery night (N9) in snorers and non-snorers.

As revealed by a significant group \times night interaction ($F[6,78] = 3.09$; $p < .009$). Fig. 4 shows a stronger effect of CSD in the non-snorers group due to the fact that their sleep latency was longer at baseline and recovery compared to the snorers who showed only partial recovery.

3.2.4. Psychomotor Vigilance Task

During ASD, the ANOVA reveals a significant group \times night interaction ($F[2,28] = 6.32$; $p = .006$) with an increased RT in snorers compared to non-snorers ($p = .01$) after a total sleep deprivation night (N2). The same is observed on %RT >500 ms ($F[2,28] = 5.56$; $p = .0009$). As shown in Fig. 5, only the snorer group displays an important increase in %RT >500 ms ($p = .00008$) from N1 to N2, which decreases, however, at baseline level from N2 to N3 after recovery ($p = .0002$). Conversely, there is no significant difference between the three nights in the non-snorers group.

During CSD, there is no effect of the Group on RT or %RT >500 ms. However, there is a significant group \times time-of-day interaction ($F[1,11] = 6.6$; $p = .03$), with an increased RT at 9:30 compared to 17:30 in the snorer group ($p = .008$) whatever the night. The same is observed in the %RT >500 ms, as suggested by a significant interaction group \times time-of-day ($F[1,11] = 5$; $p = .047$) with a higher %RT >500 ms at 9:30 compared to 17:30 in snorers ($p = .02$) but not in non-snorers.

3.2.5. Subjective daytime sleepiness

The data are summarized in Table 3. During ASD, no difference is found between both groups in sleepiness assessed by the Karolinska Sleepiness Scale (KSS).

During CSD, subjective ratings in snorers and non-snorers do not differ. The ANOVA for repeated measures shows, however, a trend for a group \times night interaction ($F[4,52] = 2.20$; $p = .08$). As shown in Fig. 3, snorers tend to feel more sleepy than non-snorers during the two last nights of CSD. There is no time-of-day effect on the KSS.

4. Discussion

Our study reveals that even in normal sleep conditions (i.e., without sleep deprivation), objective daytime sleepiness measured by the MSLT is higher in snorers than in non-snorers. It has been shown a number of times that a decrease in diurnal mean sleep latency reflects either acute or chronic sleep debt [19]. Therefore, our results during the baseline suggest that snorers, despite regular sleep schedules, may suffer from chronic sleep debt. Several studies have previously reported the occurrence of daytime sleepiness in healthy snorers [20,21]. It was initially suggested that the mechanism responsible for this relationship could be similar to that of apnea/hypopnea and rely upon sleep fragmentation due to enhanced resistance of the upper airway [6,22]. However, according to Gottlieb et al. [23,24], the relationship between snoring and daytime sleepiness is probably independent of apnea–hypopnea frequency, suggesting that it does not simply reflect a sleep fragmentation effect. Daytime sleepiness in healthy snorers could reflect increased respiratory efforts [25,26], producing less restorative sleep. Since we found no specific increase in the sleep fragmentation indices in snorers, the second hypothesis could be more relevant with regards to our findings. But more accurate measures of nocturnal respiratory efforts must be undertaken in order to conclude.

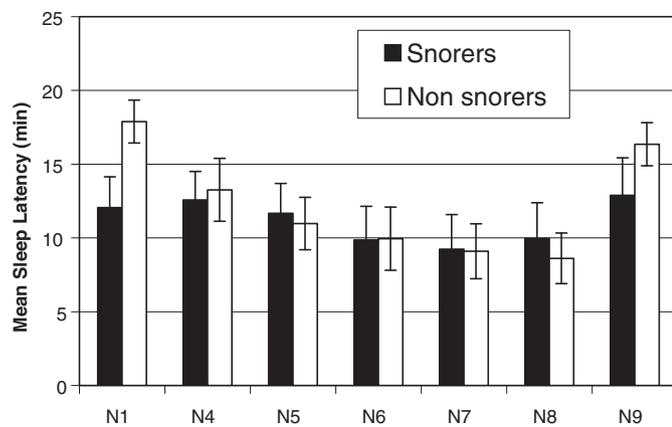
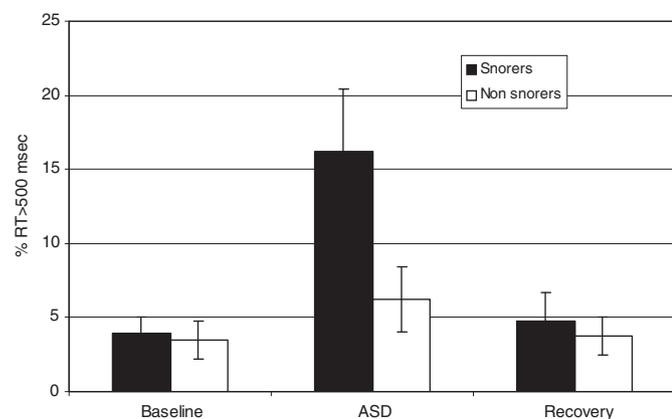
Elevated sleepiness at baseline in snorers could also suggest that a sleep debt in snorers may have been more pronounced than in non-snorers due to previous irregular sleep–wake cycles. We acknowledge as a limitation of our study that we have no proof that snorers were sleeping as much as non-snorers the week

Table 2Polysomnographic data for snorers and non-snorers during baseline, acute and chronic sleep deprivation, and recovery (mean \pm SD).

	Snorers				Non-snorers			
	N1	N3	N4–N8	N9	N1	N3	N4–N8	N9
TST	264.75 \pm 42.7	339.62 \pm 66.9	191.77 \pm 24.03	361.75 \pm 51	276 \pm 94.4	391.87 \pm 23.53	196.79 \pm 21.5	386.42 \pm 40.39
SE	61.32 \pm 8.77	73.95 \pm 14.6	86.01 \pm 7.91	80.72 \pm 9.3	63.06 \pm 19.84	83.51 \pm 5.2	87.09 \pm 8.25	83.05 \pm 8.1
L1	22.75 \pm 8.81	11 \pm 4.7	12.97 \pm 10.08	20.5 \pm 10.04	36.75 \pm 28.55	7.71 \pm 2.28	15.94 \pm 23.8	21.25 \pm 26.55
L2	24.37 \pm 8.75	21.64 \pm 20.4	19.75 \pm 31.9	21.06 \pm 9.96	38.27 \pm 28.61	9.68 \pm 4.14	12.06 \pm 10.83	13.06 \pm 7.44
LSWS	30.93 \pm 9.54	17.66 \pm 7.6	21.33 \pm 12.05	29.25 \pm 11.1	45.27 \pm 27.57	17.5 \pm 10.54	23.03 \pm 29.5	20.56 \pm 8.89
LREM	255.31 \pm 95	179.07 \pm 77.7	119.28 \pm 46.58	149 \pm 63.55	181.24 \pm 29.14	190.5 \pm 54.65	95.55 \pm 35.54	81.68 \pm 48.21
% 1	22.13 \pm 6.23	14.5 \pm 5.98	7.97 \pm 5.07	10.57 \pm 2.84	11.93 \pm 5.69	9.81 \pm 4.14	7.84 \pm 5.14	11.81 \pm 4.96
% 2	33. \pm 5.36	27.17 \pm 5.52	29.75 \pm 8	37.15 \pm 5.66	33.48 \pm 11.79	30.7 \pm 7.32	25.65 \pm 6.57	29.96 \pm 8.67
% SWS	28.71 \pm 7.41	39.32 \pm 5.8	46.56 \pm 8.43	33.35 \pm 10	35.68 \pm 16.31	39.21 \pm 5.77	44.22 \pm 7.57	30.91 \pm 6.05
% REM	16.12 \pm 10.6	22.57 \pm 6.11	16.6 \pm 8.87	18.92 \pm 10.2	21.55 \pm 6.23	20.26 \pm 5.38	23.05 \pm 9.44	27.32 \pm 9.15
WASO	41.51 \pm 8.89	27.33 \pm 15.2	16.05 \pm 11.23	20.2 \pm 10.79	37.28 \pm 19.68	16.67 \pm 5.2	13.89 \pm 8.2	17.2 \pm 8.23
Nr SSC	12.5 \pm 3.74	10.53 \pm 2.28	9.93 \pm 2.69	10.06 \pm 2.06	12.37 \pm 2.6	9.66 \pm 2.29	9.83 \pm 2.42	10.6 \pm 2.92
Arousal Index	14.5 \pm 4.17	13.25 \pm 2.18	6.15 \pm 2.62	13.25 \pm 2.94	13. \pm 4.63	13.25 \pm 3.19	5.44 \pm 1.62	14 \pm 5.63

N1, baseline, N3, recovery after acute sleep deprivation; N4–N8, chronic sleep deprivation; N9, recovery after chronic sleep deprivation.

TST, total sleep time; SE, sleep efficiency; L1–L2–LSWS–LREM, latency stage 1–2–SWS–REM; WASO, wake after sleep onset, Nr SSC, number of sleep stage changes, Arousal Index, arousal/hour.

**Fig. 4.** Mean sleep latency (\pm SEM) in CSD after the baseline night (N1), the five nights of chronic partial sleep deprivation (N4–N8) and the recovery night (N9) in snorers and non-snorers.**Fig. 5.** Mean percentage of RT >500 ms (\pm SEM) in ASD after the baseline night, the acute total sleep deprivation night and the recovery night in snorers and non-snorers.

before. Such a possibility is, however, unlikely for two reasons: A all of the subjects were given the recommendation to maintain regular sleep schedules the week before the study, and, in case snorers would suffer acute sleep deprivation due to irregular sleep

wake cycles the days before, one would expect a visible effect on the PSG data during the baseline night, but this was not the case. No difference was found in baseline sleep parameters between snorers and non-snorers. Therefore, if chronic sleep debt exists due to respiratory efforts linked with snoring, this effect could be quite subtle and also explain why, conversely, in our survey, subjective sleepiness assessed with the Karolinska Sleepiness Scale was not different between snorers and non-snorers. This discrepancy between subjective and objective sleepiness in snorers compared to non-snorers suggests that snorers may underestimate their daytime sleepiness, perhaps because they assimilate this state with normality. In other words, sleepiness in snorers may drive them to underestimate their own alertness deficit. A similar result was also found in patients with obstructive sleep apnea by Greneche et al. [27]. These authors found no relationship between KSS scores and delta–theta power (i.e., subjective and objective measures) during a 24-h period of sustained wakefulness.

Concerning the effects of both sleep deprivation paradigms, the additive sleep deprivation, either acute or chronic, did not produce differential effects on objective daytime alertness in snorers vs. non-snorers. ASD produced an important decrease in mean sleep latency in all subjects whatever their respiratory status, with no return to their baseline level after the recovery night. During CSD, subjects exhibited a progressive decrease in mean sleep latency in response to cumulative sleep restriction, but recovery was optimal only for non-snorers, while snorers showed some residual effects of sleep deprivation. This discrepancy cannot be explained by an aggravation of snoring. The respiratory status was even slightly improved in snorers, with a decrease in the number of hypopnea during the first three nights of sleep restriction. However, a sharp increase was observed in the last period of chronic sleep deprivation, as well as during the recovery night. It seems, therefore, that, at least in a first step, ventilatory disturbances take advantage from sleep deepening due to previous sleep deprivation. But as cumulative sleep debt increases, breathing disorders reappear, and in a more pronounced manner. Hence, these results only partially agree with that of Stooth and Dement [9], who showed that partial chronic sleep deprivation with four hours of sleep during six consecutive nights produced a decrement in upper airway resistance with a decrease of O₂ saturation in moderate apneic patients and an increase of snoring even in non-snoring subjects. All together, our results suggest that snoring produces permanent reduction in daytime alertness even though subjects are not aware of their state. Chronic (but not acute) sleep deprivation could aggravate breathing disorders.

Table 3Subjective and cognitive data in snorers and non-snorers during baseline, acute and chronic sleep deprivation, and recovery (mean \pm SD).

	Snorers					Non-snorers				
	N1	N2	N3	N4–N8	N9	N1	N2	N3	N4–N8	N9
KSS score	2.24 \pm 1.6	4.6 \pm 2.18	1.97 \pm 1.43	3.69 \pm 1.72	2.17 \pm 1.26	2.57 \pm 1.63	4.51 \pm 2.02	2.29 \pm 1.8	3.30 \pm 1.87	1.94 \pm 1.13
PVT										
RT	332.5 \pm 30.65	429.63 \pm 90.5	329.12 \pm 40.4	414.16 \pm 109	377.72 \pm 111	317.73 \pm 45.8	333.23 \pm 68	303.4 \pm 47	359.05 \pm 72	328.35 \pm 69.8
%RT >500 ms	4.02 \pm 3.93	16.18 \pm 13.8	4.75 \pm 5.49	17.05 \pm 17	9.05 \pm 11.23	3.5 \pm 3.92	5.68 \pm 8.02	3.72 \pm 4	9.53 \pm 12.4	6.65 \pm 11.5

KSS: Karolinska Sleepiness Scale; PVT: Psychomotor Vigilance Task.

Concerning cognitive performance, the effects of sleep deprivation have been widely documented on a number of tasks, including attention tasks, either in partial chronic [28–30] or in acute total sleep deprivation [31,32]. In the PVT, no specific effect was observed during baseline between snorers and non-snorers. By contrast, ASD was more deleterious for snorers than for non-snorers with an important increase in RT and RT >500 ms (reflecting attentional lapses) after one night of total sleep deprivation in the snorer group only. Recently, Miccoli et al. [33] compared performance after partial and total sleep deprivation. They reported that attentional lapses (omissions) were observed only after total sleep deprivation because the conditions related to this paradigm are the most difficult. It could therefore be suggested that the impaired performance exhibited by snorers after ASD reflect exacerbated effects of a long term sleep debt. In this respect, CSD was less harmful to snorers showing similar deficits in both groups, except for a time-of-day effect in the snorer group with increased RT and RT >500 ms in the morning. This is in agreement with Verstraeten et al. [34], who reported a deficit in focused attention in healthy snorers even in the absence of sleep deprivation with decreased performance during the morning compared to the evening. One explanation refers to a possible sleep inertia effect known to increase considerably with prior sleep deprivation [35]. If so, this would reinforce the assumption of an underlying cumulative sleep debt with which snorers are used to cope. All together, in the present experiment, performance decrement was more pronounced in snorers, suggesting a higher sensitivity in this group to total and, to a lesser degree, to partial sleep loss.

Therefore, we speculate that long term experience of snoring could result in permanent chronic sleep debt, but also that snorers could have more difficulty in overcoming supplementary sleep deprivation, especially when it is acute and total. In turn, this could lead them to develop compensatory mechanisms likely to counteract the adverse effects of snoring, resulting in an overestimation of the level of alertness. But it could also allow snorers to maintain satisfactory respiratory effort capacities during the night, e.g., via a sensitisation process of chemoreceptors producing a cascade of chemical or physiological events likely to decrease the upper airway resistance. This has, however, to be confirmed with middle aged or elderly subjects who have experienced snoring for a very long time.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: doi:10.1016/j.sleep.2011.06.017.

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