

Age effects on spectral electroencephalogram activity prior to dream recall

SARAH L. CHELLAPPA^{1,2}, MIRJAM MÜNCH¹, VERA KNOBLAUCH¹ and CHRISTIAN CAJOCHEN¹

¹Centre for Chronobiology, Psychiatric Hospital of the University of Basel, Basel, Switzerland and ²The CAPES Foundation/Ministry of Education of Brazil, Brasilia DF, Brazil

Accepted in revised form 16 July 2011; received 4 April 2011

SUMMARY Ageing is associated with marked changes in sleep timing, structure and electroencephalographic (EEG) activity. Older people exhibit less slow-wave and spindle activity during non-rapid eye movement (NREM) sleep, together with attenuated levels of rapid eye movement (REM) sleep as compared to young individuals. However, the extent to which these age-related changes in sleep impact on dream processing remains largely unknown. Here we investigated NREM and REM sleep EEG activity prior to dream recall and no recall in 17 young (20–31 years) and 15 older volunteers (57–74 years) during a 40 h multiple nap protocol. Dream recall was assessed immediately after each nap. During NREM sleep prior to dream recall, older participants displayed higher frontal EEG delta activity (1–3 Hz) and higher centro-parietal sigma activity (12–15 Hz) than the young volunteers. Conversely, before no recall, older participants had less frontal-central delta activity and less sigma activity in frontal, central and parietal derivations than the young participants. REM sleep was associated to age-related changes, such that older participants had less frontal-central alpha (10–12 Hz) and beta (16–19 Hz) activity, irrespective of dream recall and no recall. Our data indicate that age-related differences in dream recall seem to be directly coupled to specific frequency and topography EEG patterns, particularly during NREM sleep. Thus, the spectral correlates of dreaming can help to understand the cortical pathways of dreaming.

KEYWORDS ageing, dream recall, electroencephalographic spectral analysis, non-rapid eye movement sleep, rapid eye movement sleep

INTRODUCTION

The reduction in non-rapid eye movement (NREM) slow-wave and spindle frequency activity, together with less rapid eye movement (REM) sleep and an increase in involuntary awakenings during sleep, represent the hallmarks of age-related changes in sleep (Dijk *et al.*, 1999; Münch *et al.*, 2005). Given the magnitude of age effects on sleep structure and

sleep electroencephalographic (EEG) power density, the question arises as to how it can impact on dream generation. Dreaming is a complex mental activity, driven by the interplay of the ultradian NREM–REM sleep cycle and the circadian modulation of REM sleep. REM dreaming is elicited by the selective activation of wake-activated structures in the brainstem, limbic subcortex and cortex (Braun *et al.*, 1997; Maquet *et al.*, 1996; Nofzinger, 2005). Dream-like recalls can also be obtained subsequent to NREM sleep, sleep onset or even relaxed awakening, which suggests that differences in dream recall may underlie dissimilarities in cortical activation (Antrobus, 1983; Cavallero *et al.*, 1990; Foulkes, 1993; Rosenlicht *et al.*, 1994), in sleep architecture and EEG

Correspondence: Professor Dr. Christian Cajochen, PhD, Centre for Chronobiology, Psychiatric Hospital of the University of Basel, Wilhelm Kleinstrasse 27, CH-4012 Basel, Switzerland. Tel: +416-132-55318; fax: +416-132-55556; e-mail: Christian.Cajochen@upkbs.ch

composition (Antrobus *et al.*, 1995; Casagrande *et al.*, 1996). In young participants, successful dream recall can be associated with increased REM sleep alpha and beta activity, and decreased NREM sleep delta and sigma activity prior to dream recall during a multiple-nap protocol (Chellappa *et al.*, 2011). Dream recall in young participants has also been associated to higher frontal REM sleep theta activity and lower NREM sleep alpha oscillatory activity after morning awakening (Marzano *et al.*, 2011). Dream recall declines progressively with advancing age and becomes less intense, perceptually and emotionally (Zanasi *et al.*, 2005). One candidate for this attenuated dream recall with ageing is the diminished circadian rhythm of REM sleep (Chellappa *et al.*, 2009). The idea of REM sleep as a hallmark for age differences in dreams also builds up from recent evidence, whereby patients suffering from mild degenerative dementia dream much less than healthy elderly people, possibly due to REM sleep decrease (Guérolé *et al.*, 2010). However, the extent to which age differences in NREM and REM sleep impacts on with dream recall remains largely unexplored. Thus, we investigated frequency and topographical changes in EEG activity during NREM and REM sleep prior to dream recall and no recall in young and older participants under stringent controlled laboratory conditions.

METHODS

Study participants

Study volunteers were recruited through advertisements at different Swiss universities and in newspapers. Only candidates with a Pittsburgh sleep quality index (PSQI) score ≤ 5 (Buysse *et al.*, 1989) and without extreme chronotypes ratings [between 14 and 21 points on the morning–evening questionnaire (Torsvall and Åkerstedt, 1980)] were enrolled in the study. All participants were questioned about their sleep quality, life habits and health state. Exclusion criteria were smoking, medication or drug consumption, shift work within the last 3 months and transmeridian flights during the month before the study. All volunteers underwent a physical examination, an interview, a neuropsychological assessment and a polysomnographically recorded adaptation night in order to exclude sleep disorders. Inclusion criteria were sleep efficiency $> 80\%$, periodic leg movements < 10 and apnoea–hypopnoea index < 10 . Only participants without medication (with the exception of four young women using oral contraceptives) were included in the study. Young women started the study on days 1–5 after menses onset, thus during the follicular phase of their menstrual cycle. Seventeen healthy young (nine women, eight men, age range 20–31 years; young participants correspond to those in Chellappa *et al.*, 2011) and 15 healthy older volunteers (seven women, eight men, age range 57–74 years) were enrolled in the study. All participants gave written informed consent. The study protocol, screening questionnaires and consent form were approved by the local ethics committee and conformed to the Declaration of Helsinki.

Study design

One week prior to the study (baseline week), participants were requested to abstain from excessive caffeine and alcohol (one caffeine-containing beverage per day at most and < 5 alcoholic beverages per week). They were instructed to keep a regular sleep–wake schedule during the baseline week at home (bed-times and wake times within ± 30 min of self-selected target time between 22:00 h and 02:00 h) prior to admission to the laboratory. Compliance was checked by sleep logs and ambulatory activity measurements (wrist activity monitor; Cambridge Neurotechnology Ltd, Cambridge, UK). The timing of the sleep–wake schedule during the protocol was adjusted to habitual individual sleep and wake-up times. The laboratory part of the study comprised two baseline sleep episodes, followed by a 40-h multiple nap protocol, with 10 alternating sleep–wake cycles of 75/150 min duration each and one recovery sleep episode. Polysomnography recordings and constant posture started in the afternoon after the first baseline night. Thereafter, participants remained under constant posture conditions (constant dim light levels < 8 lux during scheduled wakefulness, semi-recumbent posture in bed, food and liquid intake at regular intervals, no time cues). During scheduled sleep episodes a minor shift (45° up) in the supine posture was allowed, and lights were off (0 lux). Older participants received a daily low-dose subcutaneous heparin injection (Fragmin, 0.2 mL, 2500 IE UI $^{-1}$; Pfizer AG, Zurich, Switzerland) to prevent potential venous thrombosis.

Polysomnographic measures

Sleep was recorded polysomnographically with the Vitaport ambulatory system (Vitaport-3 digital recorder; Temec Instruments B.V., Kerkrade, the Netherlands). Twelve EEG channels, two electrooculograms, one submental electromyogram and one electrocardiogram were recorded. All signals were low-pass filtered at 30 Hz (fourth order Bessel type anti-aliasing, total 24 dB per Oct) at a time constant of 1.0 s. After online digitization by using a 12-bit AD converter (0.15 V per bit) and a sampling rate at 128 Hz for the EEG, the raw signals were stored on a Flash RAM Card (Viking, Foothill Ranch, CA, USA) and downloaded to a PC hard drive. Sleep stages were scored visually per 20-s epochs (Vitaport Paperless Sleep Scoring Software, Kerkrade, the Netherlands). EEG artefacts were detected by an automated artefact algorithm (CASA, 2000; PhyVision B.V., Gemert, the Netherlands). Spectral analysis was conducted using a Fast-Fourier transformation (FFT; 10% cosine 4-s window; approximately 5% of the sleep data were rejected epochs for an artefact contamination) which yielded a 0.25 Hz bin resolution. NREM sleep (Stages 2–4) and REM sleep were expressed as the percentage of total sleep time per nap before averaging over participants. EEG power spectra were calculated during REM sleep and NREM sleep in the frequency range 0–20 Hz. Finally, artefact-free 4-s epochs were averaged across 20-s epochs. Here, we report EEG data derived from 12 derivations (F3, F4, Fz, C3, C4, Cz, P3, P4,

Pz, O1, O2, Oz) referenced against linked mastoids (A1, A2) in the range 0.75–20 Hz.

Dream recall

Dream recall was assessed immediately at the end of each nap trial (10 naps in total) with the Sleep Mentation Questionnaire, which addresses numerous characteristics of dream recall, such as number of dreams, emotionality, vividness, pleasantness, hostility and colourfulness (Chellappa *et al.*, 2009). For the classification of dream recall, only the first question [‘How much did you dream?’ (1: greatly, 2: fairly, 3: little, 4: not at all)] was taken into account for the analysis. Participants were considered to have successful dream recall if their response to Q1 was not 4. The main advantage of a 40-h multiple nap protocol was the numerous time-points (10 in our study). Thus, dream recall could be assessed without possible effects of total sleep deprivation or selective sleep stage deprivation prior to recall. For more details concerning NREM and REM naps associated with dream recall and no recall in both young and older participants, see Chellappa *et al.*, 2009.

Classification of NREM and REM naps

A nap trial that contained only REM sleep in the last 15 min of a scheduled 75-min nap was defined as a REM nap, and a nap trial with NREM sleep in the last 15 min was defined as a NREM nap (Chellappa *et al.*, 2009). ‘Wakefulness naps’ were defined as nap trials not containing either NREM or REM sleep stages. The criterion of 15 min was based on a prior definition of NREM and REM naps, in which 20-min naps were employed (Suzuki *et al.*, 2004). Given that our study included 75-min naps (Münch *et al.*, 2005), only the last 15 min were considered for the REM sleep and NREM sleep stages, instead of 20 min, as the likelihood of having 20-min naps exclusively with REM sleep would be substantially reduced. According to these criteria, the young ($n = 17$) and older ($n = 15$) groups had a total of 170 and 150 scheduled naps, respectively. Of the 170 naps for the young, 81 (47.7%) were NREM naps (38 had dream recall and 43 had no recall), 46 (27.5%) were REM naps (35 had dream recall and 11 had no recall) and 43 (24.8%) were naps containing wakefulness. For the older group, out of 150 naps, 91 (60.7%) were NREM naps (43 had dream recall and 48 no recall), 22 (14.7%) were REM naps (12 had dream recall and 10 had no recall) and 37 (24.6%) were naps with wakefulness.

Considering that throughout the 10 scheduled naps not every nap had comparable levels of dream recall, the naps were classified into five categories (for each participant): REM naps with dream recall, REM naps with no recall, NREM naps with dream recall, NREM naps with no recall and naps with wakefulness. Thus, the classification of naps into these categories would enable the comparison of dream recall. On average, young participants had 2.05 ± 0.3 REM naps with dream recall, 0.94 ± 0.1 REM naps with no recall, 2.23 ± 0.3 NREM naps with dream recall, 2.52 ± 0.3 NREM

naps with no recall, and 2.22 ± 0.2 naps with wakefulness [mean \pm standard error of the mean (SEM)]. The older participants had 0.9 ± 0.2 REM naps with dream recall, 0.9 ± 0.1 REM naps with no recall, 2.9 ± 0.2 NREM naps with dream recall, 3.1 ± 0.2 NREM naps with no recall and 2.2 ± 0.2 naps with wakefulness (mean \pm SEM). Naps with wakefulness, which usually comprised naps 4 and 10 (‘wake-maintenance zone’), were excluded from the analysis.

Statistical analysis

For all analyses, the statistical package SAS version 9.1 (SAS Institute Inc., Cary, NC, USA) was utilized. Visually scored sleep stages were expressed as percentages of total sleep time or in minutes. The analysis of sleep-stage differences was carried out with the mixed-model analyses of variance for repeated measures (PROC Mixed) with factors ‘age’ (young \times older) and ‘time’ (10 naps; the time bins comprised 3.75 h, thus a total of 10 naps). For the accumulation curves, sleep stages [wake, NREM Stage 2 and slow wave sleep (SWS)] were collapsed into 5-min intervals of the 75 min comprising the naps. A general linear model (PROC GLM) was carried out with factors ‘age’ (young \times older), ‘time’ and ‘recall’ (dream recall \times no recall), using the Duncan’s multiple range test and corrections for multiple comparison. For comparisons during NREM and REM sleep, mixed-model analyses of variance for repeated measures (PROC Mixed) was used with factors ‘age’ (young \times older), ‘derivation’ (frontal = F3, F4 and Fz; central = C3, C4 and Cz; parietal = P3, P4 and Pz; occipital = O1, O2 and Oz) and ‘recall’ (recall \times no recall). Alpha adjustment for multiple comparisons was applied using the Tukey–Kramer test. For factor ‘derivation’, the corresponding three derivations (i.e. frontal = F3, F4 and Fz) were averaged per subject, given that there were no lateralization effects for dream recall. All *P*-values derived from r-analyses of variance (ANOVAS) were based on Huynh–Feldt’s (H-F) corrected degrees of freedom (significance level: $P < 0.05$). *Post-hoc* measurements were obtained using the Tukey–Kramer test.

RESULTS

Sleep stages during the last 15 min of naps prior to dream recall

Total sleep time (TST) in the last 15 min (averaged across all 10 naps per subject) did not differ significantly between age groups (Table 1). Analysis of sleep stages collapsed for all 10 naps revealed that older participants had more NREM sleep than the young, with significantly more NREM sleep stage 2, less SWS (Stages 3 and 4) and less REM sleep (Mann–Whitney *U*-test, $P < 0.05$), together with a tendency for more wakefulness (Mann–Whitney *U*-test, $P = 0.06$). Analysis of the time-course of TST, NREM sleep, NREM Stage 2 and REM sleep during the last 15 min of the naps yielded more TST for older participants during naps 4 and 10 (two-way r-ANOVA, factors ‘age’ \times ‘time’; $F_{9,269} = 3.45$, $P < 0.05$). The

Table 1 Averaged sleep stages (across 10 naps per subject) for older and young subjects during the last 15 mins of naps prior to dream recall

Sleep variables	Older	Young	P
TST (min)	9.8 ± 1.3	11.3 ± 1.1	NS
Sleep efficiency (%)	66.1 ± 8.9	71.4 ± 6.4	NS
Stage 1 (%)	14.4 ± 3.8	18.4 ± 4.7	NS
Stage 2 (%)	53.4 ± 7.6	32.2 ± 6.9	<0.05
Stage 3 (%)	5.1 ± 1.1	9.8 ± 2.7	<0.05
Stage 4 (%)	2.1 ± 1.7	9.4 ± 1.5	<0.05
SWS (%)	7.2 ± 1.0	19.2 ± 2.3	<0.05
Wakefulness (%)	18.8 ± 2.9	12.6 ± 2.1	0.06
NREM sleep (%)	61.6 ± 2.5	50.4 ± 6.1	<0.05
REM sleep (%)	6.2 ± 1.3	17.6 ± 2.7	<0.05

TST, total sleep time (min; stages 1–4 + REM sleep); Sleep efficiency (% of TST); SWS, slow-wave sleep (% of TST; stage 3 + 4); Wakefulness, wake time and movement time (% TST); NREM sleep, NREM (% of TST; stage 2–4); REM sleep, REM (% of TST); *P*-values between dream recall and no recall (Mann–Whitney *U* test); NS, not significant. (Mean ± SEM).

time-course of wakefulness yielded less wakefulness (% of TST) in naps 4 and 10 and more wakefulness in naps 7 and 8 for the older participants, as compared to the young ($F_{9,257} = 3.15$, $P < 0.05$). Older participants had a tendency for more NREM sleep during nap 10 ($F_{9,227} = 1.69$, $P = 0.04$) and significantly more NREM sleep stage 2 during naps 1, 2, 7 and 10 than the young participants ($F_{9,228} = 1.79$, $P = 0.04$). REM sleep was significantly reduced in older participants during naps 7 and 8, as compared to the young ($F_{9,226} = 1.83$, $P = 0.04$) (Fig. 1). No gender differences were seen for sleep architecture nor in the remainder of this data set.

The accumulation curves of NREM naps yielded a significant interaction between factors ‘age’ × ‘time’ for wakefulness, NREM Stage 2 and SWS ($F_{4,64} = 1.7$; $P < 0.05$). Older participants had more accumulated wakefulness, more NREM Stage 2 and less SWS than young participants, particularly after 60 min of sleep onset. The interaction of factors ‘age’ × ‘recall’ elicited significant differences only for NREM sleep stage 2 ($F_{1,16} = 7.33$; $P = 0.02$). Similarly, the three-way interaction of factors ‘age’ × ‘recall’ × ‘time’ yielded significant differences only for NREM sleep stage 2 ($F_{4,64} = 2.11$; $P = 0.04$). Older participants had more accumulated NREM sleep stage 2 prior to dream recall than the young, particularly after 60 min of sleep onset, while they had less accumulated NREM Stage 2 before no recall, as compared to young participants (Fig. 2).

Dream recall differences in NREM EEG sleep spectra

A three-way *r*-ANOVA with factors ‘age’ × ‘recall’ × ‘derivation’ yielded a significant main effect for factor ‘age’ in the frequency range of 1–3 Hz (delta range) and 12–15 Hz (sigma range) ($P < 0.05$). Main effect ‘derivation’ was significant for delta (1–3 Hz) ($F_{3,149} = 42.3$, $P < 0.01$), theta (5–7.75 Hz) ($F_{3,148} = 16.4$, $P < 0.01$), sigma (12–15 Hz) ($F_{3,148} = 25.5$,

$P < 0.01$) and beta activity (16–19 Hz) ($F_{3,148} = 19.2$, $P < 0.01$). Main effect ‘recall’ was significant for delta (1–3 Hz) ($F_{1,160} = 25.4$, $P < 0.01$), sigma (12–15 Hz) ($F_{1,160} = 25.2$, $P < 0.01$) and beta activity (16–19 Hz) ($F_{1,162} = 14.2$, $P < 0.01$). A significant interaction of factors ‘age’ × ‘derivation’ × ‘recall’ was elicited for delta (1–3 Hz) ($F_{8,149} = 4.4$, $P = 0.02$) and sigma activity (12–15 Hz) ($F_{8,149} = 2.3$, $P = 0.01$). Older participants had more delta and sigma activity prior to dream recall than to no recall, while in the young, delta and sigma activity was attenuated before dream recall compared with no recall ($P < 0.05$) (Fig. 3). The topographical distribution of EEG activity was such that, before dream recall, older participants had more frontal delta activity (1–3 Hz) than the young participants, while during no recall, older volunteers had less fronto-central delta activity (Figs 4 and 5). Similarly, before dream recall, older participants had more centro-parietal sigma activity (12–15 Hz) than the young participants, while during no recall, older volunteers had less sigma activity in frontal, central and parietal derivations than the young (Figs 4 and 5).

Dream recall differences in REM EEG sleep spectra

A three-way *r*-ANOVA with factors ‘age’ × ‘recall’ × ‘derivation’ yielded a significant effect for the factor ‘age’ in the frequency range of 10–12 Hz (alpha range) and 16–19 Hz (beta range) ($P < 0.05$). Main effect ‘derivation’ was significant for alpha (10–12 Hz) ($F_{3,167} = 10.5$, $P < 0.01$) and beta (16–19 Hz) ($F_{3,167} = 25.6$, $P < 0.01$) activity. Main effect ‘recall’ was significant for alpha (10–12 Hz) ($F_{1,155} = 5.7$, $P < 0.01$) and beta activity (16–19 Hz) ($F_{1,155} = 14.7$, $P < 0.01$). The interaction of factors ‘age’ × ‘derivation’ × ‘recall’ did not reach significance for any frequency range. Comparison of dream recall and no recall yielded no differences for alpha (10–12 Hz) and beta activity (16–19 Hz) for the older volunteers. A separate analysis for the young revealed that dream recall following REM sleep was associated with low frontal alpha activity and with high alpha and beta activity in occipital derivations (for more details, see Chellappa *et al.*, 2011). Here, we observed a clear age effect, such that older participants had less alpha and beta activity as compared to the young, irrespective of dream recall and no recall (Fig. 6). The topographic distribution of these age-related effects was such that, before dream recall and no recall, older participants had less fronto-central alpha (10–12 Hz) and beta activity (16–19 Hz) in comparison to young participants (two-way *r*-ANOVA with factors ‘age’ × ‘derivation’; $P < 0.05$) (Fig. S1).

DISCUSSION

Our data indicate clear age-related changes in specific frequency and topography NREM sleep EEG activity between dream recall and no recall. These differences are such that, in comparison to the young, older volunteers had more frontal delta and central-parietal sigma activity before dream recall,

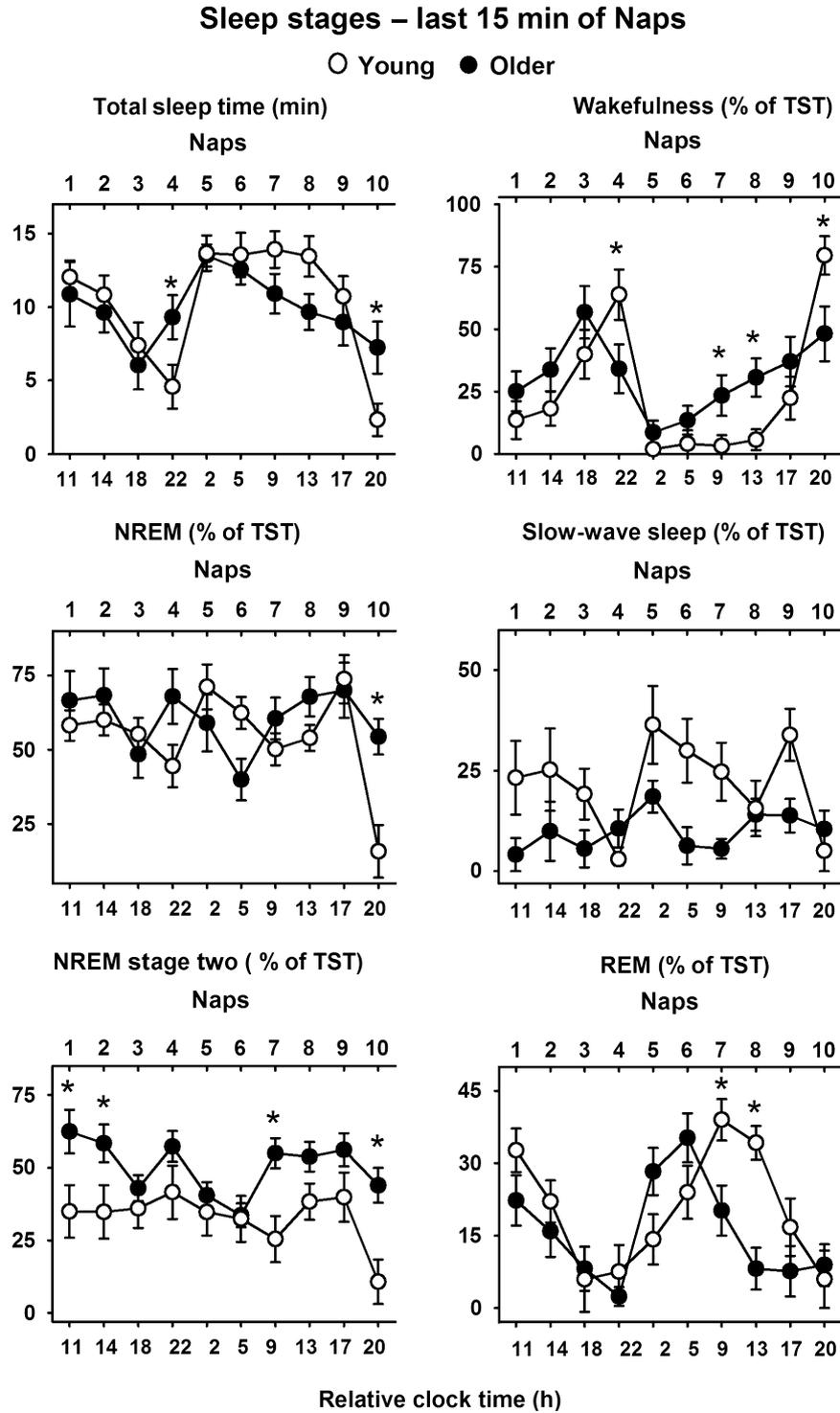


Figure 1. Time-course of sleep stages of the last 15 min of naps. White circles: young volunteers ($n = 17$), black circles: older volunteers ($n = 15$; mean \pm SEM), * $P < 0.05$.

while they had less frontal-central delta and sigma activity before no recall. Conversely, REM sleep EEG activity showed an age effect *per se*, unrelated to dream recall and no recall. These age-related changes for REM sleep were such that older volunteers had less frontal-central alpha and beta activity than the young, irrespective of recalling dreams or not.

NREM dream recall

Neuroimaging studies have revealed that NREM sleep is associated with a deactivation of ascending arousal systems, including the pons and midbrain, which may render into a reduced level of global forebrain activation (Braun *et al.*, 1997;

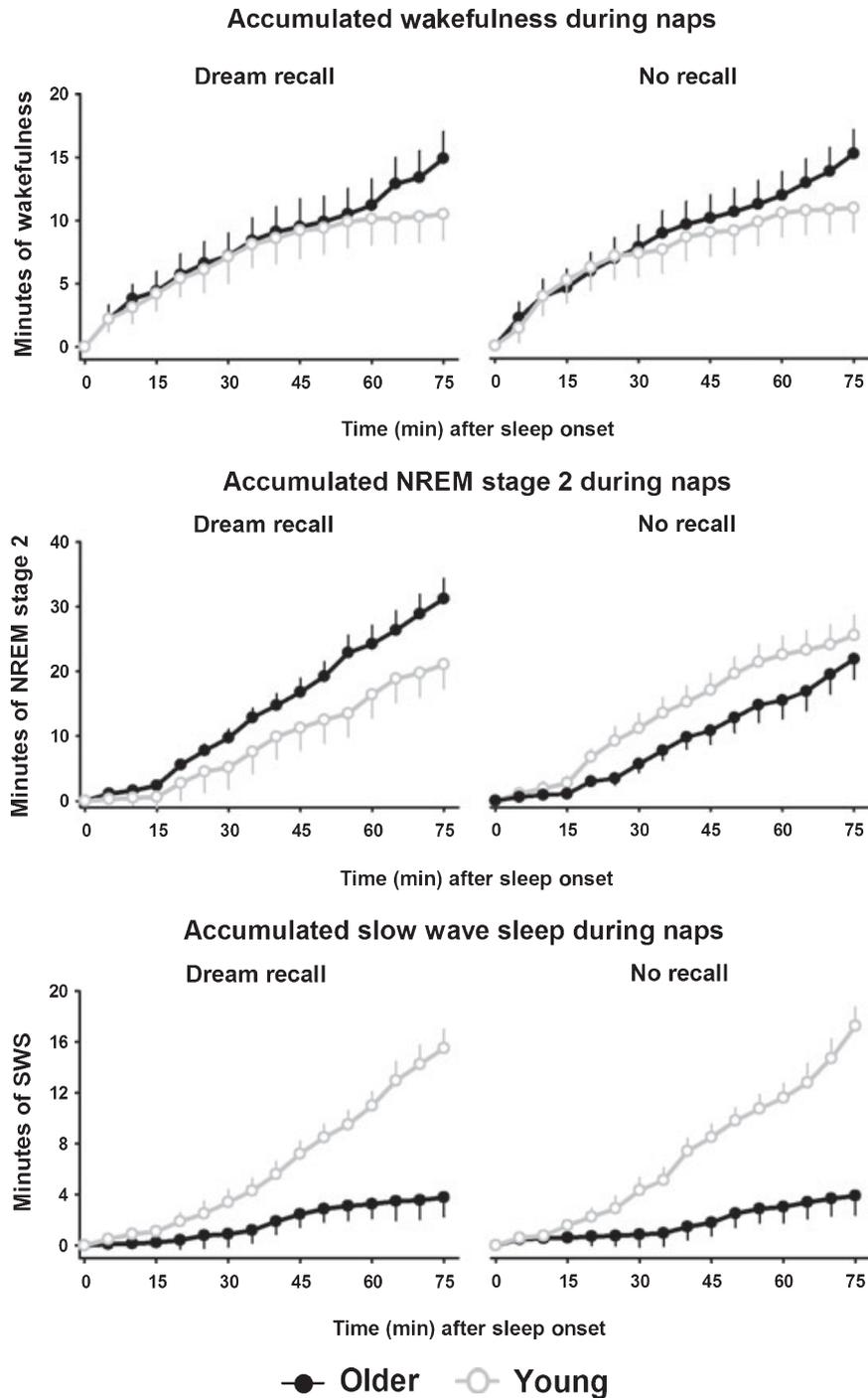


Figure 2. Accumulation curves for wakefulness (upper panel), non-rapid eye movement (NREM) sleep stage 2 (middle panel) and NREM slow wave sleep (SWS) (bottom panel) after sleep onset during the naps (10 naps in total) in older (black lines) and young (grey lines) participants. Data are plot relative to elapsed time (min) after sleep onset. Mean and SEM values are shown for each 5-min bin.

Hofle *et al.*, 1997). Furthermore, the regional deactivation during NREM sleep of multi-modal association cortices in prefrontal and parietal areas, compared to wakefulness (Braun *et al.*, 1997), may selectively constrain higher-level cognitive activity in NREM sleep (Hobson and Pace-Schott, 2002). On an electrophysiological level, NREM sleep is associated with a wide range of intrinsic slow thalamocortical oscillatory

rhythms—sleep spindles and slow-waves – which lead to impaired synaptic responsiveness (Timofeev *et al.*, 2001). These specific NREM-related EEG and neuroimaging milieu help to explain why NREM consciousness is curtailed. Within this context, it can be assumed that more NREM sleep spindles and slow-waves may result in a disfacilitation for ongoing cognitive processing. This seems to hold true for the young

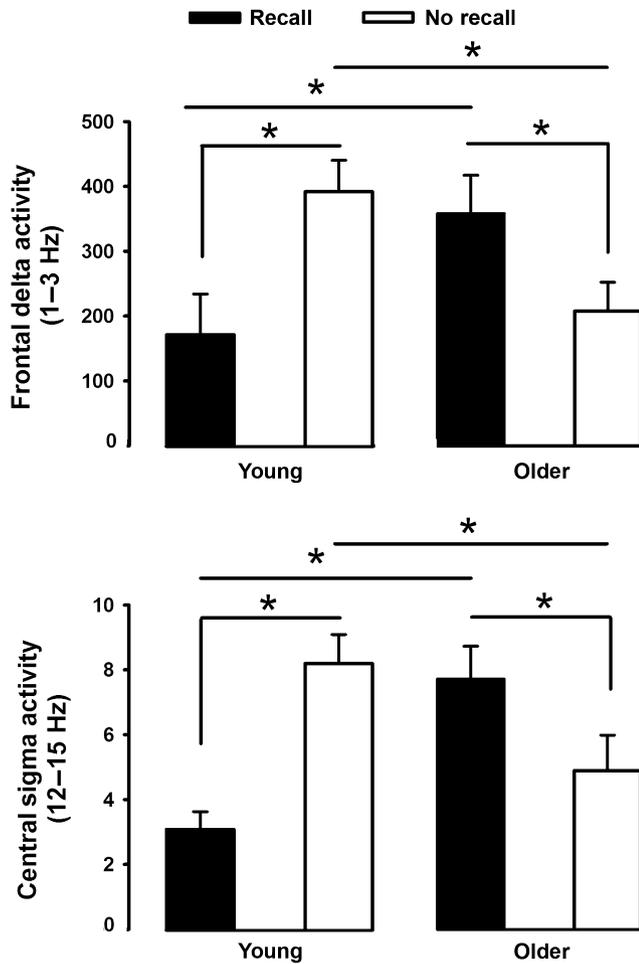


Figure 3. Non-rapid eye movement (NREM) sleep between older and young participants prior to dream recall and no recall. Upper panel illustrates frontal delta activity (1-3 Hz) and bottom panel depicts central sigma activity (12-15 Hz) for young and older participants before dream recall (black bars) and no recall (white bars) (mean \pm SEM; * $P < 0.05$).

under a multiple-nap protocol (Chellappa *et al.*, 2011), although no changes are observed for these frequency ranges if dream recall is assessed only following an entire night episode (Marzano *et al.*, 2011). However, a dissimilar scenario happened for the older participants, with higher levels of delta and sigma activity prior to dream recall.

NREM delta activity

Ageing is associated unambiguously with a reduction of slow-wave sleep and NREM delta activity, which can be explained by reduced homeostatic sleep drive, reduced circadian drive promoting sleep in the second half of the night, or an altered interaction of the circadian and homeostatic process (Dijk and Czeisler, 1995; Dijk *et al.*, 1999). Thus, it is not surprising that before no recall, older participants had less delta activity as compared to the young. However, the higher delta activity before dream recall in older volunteers is counterintuitive. In our study, the interaction of factors 'age', 'recall' and 'time' was significant only for NREM sleep stage 2, such that the older participants had more accumulated NREM stage 2 prior to dream recall, while they had less accumulated before no recall, in comparison to young participants. This sleep stage comprises the NREM stage most connected to dream recall, as compared to NREM sleep stages 3 and 4 (Takeuchi *et al.*, 2003). Our results indicate a significant effect for this sleep stage, but not for SWS and wakefulness. Thus, we speculate that more NREM sleep stage 2 prior to dream recall may have resulted in more delta and sigma activity in our older participants. Nevertheless, this should be viewed with caution, as more NREM sleep stage 2 may not necessarily imply more delta and spindle activity.

NREM awakenings

In our study, older participants had more awakenings during NREM naps than the young. Sleep of older people is

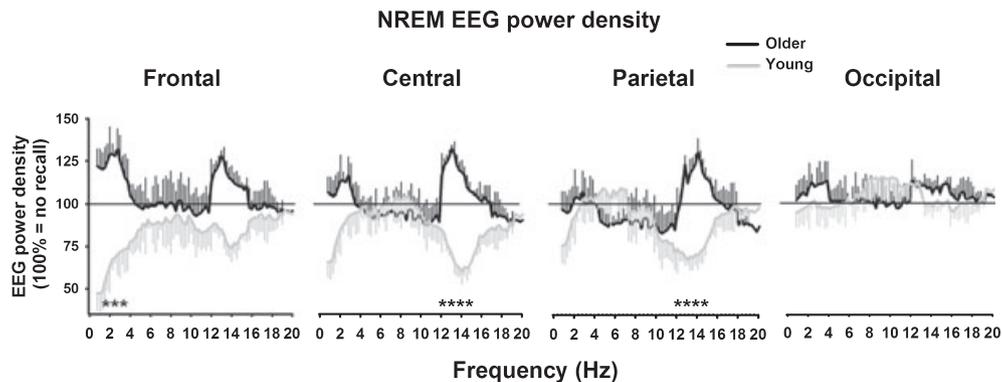


Figure 4. Non-rapid eye movement (NREM) sleep electroencephalogram (EEG) power density between older (black lines) and young participants (grey lines) before dream recall for frontal, central, parietal and occipital derivations. EEG power density values per 0.25 Hz bin during NREM sleep before dream recall are expressed as percentage of the corresponding average values for NREM EEG power density for no recall. Horizontal line represents 100% of EEG activity prior to no recall. Mean values are shown for each 0.25 Hz frequency bin in the range of 0.75-20 Hz. Horizontal stars near the abscissa at the bottom indicate frequency bins with a significant interaction 'age' \times 'derivation' \times 'recall' ($P < 0.05$).

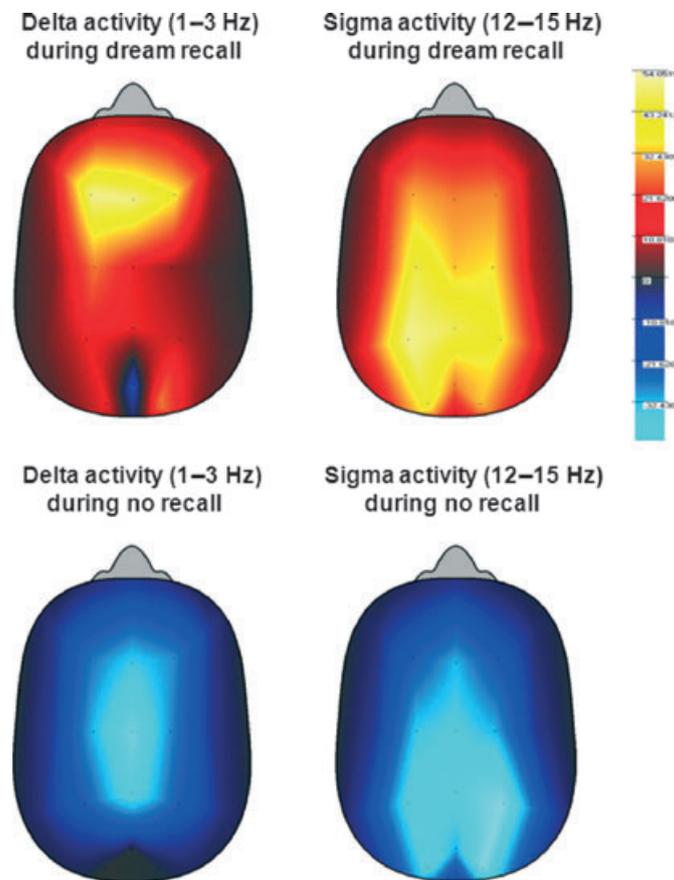


Figure 5. Top panel: left and right panels illustrate, respectively, the topographical distribution of non-rapid eye movement (NREM) delta (1–3 Hz) and sigma (12–15 Hz) activity before dream recall in older and young participants [NREM electroencephalogram (EEG) spectra, indexed as a ratio of older/young]. Bottom panel: left and right panels illustrate, respectively, the topographical distribution of NREM delta (1–3 Hz) and sigma (12–15 Hz) activity during no recall in older and young participants (NREM EEG spectra, indexed as a ratio of older/young). Scales: warmer colours (particularly yellow) indicate maximum difference between older and young EEG activity (EEG activity of older > EEG activity of young), while colder colours (particularly light blue) indicate minimum difference between older and young EEG activity (EEG activity of older < EEG activity of young).

interrupted frequently by awakenings, and this deterioration of sleep continuity may be due to an attenuated consolidation of NREM sleep (Dijk *et al.*, 2001). With respect to dreaming, in a previous study the neural networks underlying NREM dreaming could be related to intra-NREM sleep arousal processes (Takeuchi *et al.*, 2003). Thus, NREM dreaming could be an effect of the recall of perceptual experiences incorporated into the memory during awakenings from NREM sleep (Takeuchi *et al.*, 2003). In this context, more NREM awakenings in the older participants might have facilitated their recollection of dreams. However, the interaction of ‘age’ × ‘recall’ × ‘time’ was not significant for accumulated wakefulness, but only for NREM sleep stage 2. Thus, it is more likely that the differences in accumulated NREM sleep stage 2, rather than the differences in accumulated wakefulness, underlie the age effects on NREM dream recall.

NREM sigma activity

The increased sigma activity prior to dream recall in older participants is likely to be driven by differences in the

circadian modulation of spindle frequency. This assumption seems to hold true since, as indicated in Fig. S2, older participants had more delta and spindle activity during dream recall, while they had less high spindle activity before no recall, in comparison to the young, with clear differences between the biological day and night. Spindles are under circadian control such that the circadian pacemaker actively promotes spindles during the night, possibly to mediate sleep consolidation (Dijk and Czeisler, 1995). Young people have a clear-cut circadian modulation of higher EEG spindle frequency, phase-locked with the circadian rhythm of melatonin (Knoblauch *et al.*, 2005). This circadian modulation is such that they show less fast spindle frequencies during the night and more during the day, while older people have less distinctive day–night differences (Knoblauch *et al.*, 2005). Considering that diurnal fast spindle frequencies could represent a circadian waking signal, less well-defined day–night differences in spindle frequency may represent a reduction of this signal with more ‘sleep intrusions’. This, in turn, might have resulted in more sigma activity prior to dream recall in older volunteers.

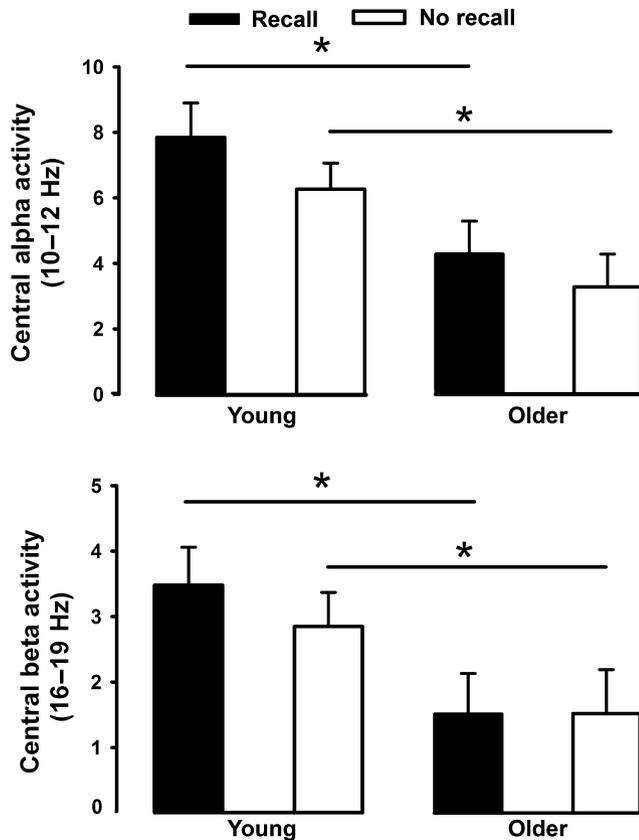


Figure 6. Rapid eye movement (REM) sleep dream between older and young participants prior to dream recall and no recall. Upper panel illustrates central alpha activity (10–12 Hz) and bottom panel depicts central beta activity (16–19 Hz) for young and older participants before dream recall (black bars) and no recall (white bars) (mean \pm SEM; * $P < 0.05$).

REM dream recall

REM sleep has an indisputable role in dreaming, and is associated with increased alpha activity (Esposito *et al.*, 2004), which may reflect cognitive elaboration prior to awakening. Recently, successful dream recall was associated with higher frontal REM sleep 5–7 Hz (theta) activity, a finding that may reflect the increase in frontal theta activity during successful encoding of episodic memories in wakefulness (Marzano *et al.*, 2011). In our study, older volunteers had decreased alpha and beta activity irrespective of dream recall and no recall. The percentage of REM sleep undergoes a decline during middle-aged adulthood, and remains stable in people above 60 years (Ohayon *et al.*, 2004). Shorter REM sleep duration does not imply less EEG activity; however, the reduction of REM sleep in older participants may have resulted in an overall decrease of EEG activity, irrespective of recalling dreams or not.

Limitations

Considering that during the 10 scheduled naps, not every nap had comparable levels of dream recall, the naps were classified into REM naps with and without dream recall and NREM

naps with and without dream recall. This classification of naps enabled us to perform a comparison for NREM naps with and without dream recall and no recall, and also for REM naps with no recall, between young and older participants. However, this categorization implies that our analyses were based on values collapsed as a function of a variable range of raw observations, such that some values were missing and others were based on mean values. This situation occurred for REM naps with dream recall, such that the older volunteers had much fewer REM naps with dream recall compared to the young. This may be one of the factors accounting for no REM recall differences between the two groups.

CONCLUSIONS

We previously showed that older people have attenuated dream recall, which may be related to an overall decrease in REM sleep (Chellappa *et al.*, 2009). Here (see Fig. S1) these age-related effects were such that specific topography and spectral correlates of REM dream recall were reduced (frontal-central alpha and beta activity). Conversely, more delta and sigma activity in the last part of NREM naps favoured successful dream recall in the older participants, which was not the case in the young. This probably happened due to more NREM sleep stage 2 in older participants, which was higher only prior to dream recall. The underlying mechanisms for these differences during NREM sleep remain elusive, and certainly warrant future interest. Taken together, age differences in dream recall seem to be directly coupled to specific frequency and topography-EEG activity patterns, particularly during NREM sleep. The understanding of these spectral correlates of dreaming may shed light onto the cortical pathways of dream generation.

ACKNOWLEDGEMENTS

We thank Claudia Renz, Marie-France Dattler and Giovanni Balestrieri for their help in data acquisition, the volunteers for participating and Marcel Hofstetter for programming the visual contour plots. This research was supported by Swiss National Science Foundation Grants (3100-055385.98 and 3130-054991.98), Velux Foundation (Switzerland) and Bühlmann Laboratories (Switzerland).

REFERENCES

- Antrobus, J. REM and NREM sleep reports: comparison of word frequencies by cognitive classes. *Psychophysiology*, 1983, 20: 562–568.
- Antrobus, J., Kondo, T., Reinsel, R. and Fein, G. Dreaming in the late morning: summation of REM and diurnal cortical activation. *Conscious. Cogn.*, 1995, 4: 275–299.
- Braun, A. R., Balkin, T. J., Wesensten, N. J. *et al.* Regional cerebral blood flow throughout the sleep–wake cycle An $H_2^{15}O$ PET study. *Brain*, 1997, 120: 1173–1197.
- Buysse, D. J., Reynolds, C. F., Monk, T., Berman, S. R. and Kupfer, D. J. The Pittsburgh sleep quality index: a new instrument for

- psychiatric practice and research. *Psychiatry Res.*, 1989, 28: 193–213.
- Casagrande, M., Violani, C., Lucidi, F., Buttinelli, E. and Bertini, M. Variations in sleep mentation as a function of time of night. *Int. J. Neurosci.*, 1996, 85: 19–30.
- Cavallero, C., Foulkes, D., Hollifield, M. and Terry, R. Memory sources of REM and NREM dreams. *Sleep*, 1990, 13: 449–455.
- Chellappa, S. L., Münch, M., Blatter, K., Knoblauch, V. and Cajochen, C. Does the circadian modulation modify with age? *Sleep*, 2009, 32: 1201–1209.
- Chellappa, S. L., Frey, S., Knoblauch, V. and Cajochen, C. Cortical activation patterns herald successful dream recall after NREM and REM sleep. *Biol. Psychol.*, 2011, 87: 251–256.
- Dijk, D. J. and Czeisler, C. A. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *J. Neurosci.*, 1995, 15: 3526–3538.
- Dijk, D. J., Duffy, J. F., Riel, E., Shanahan, T. L. and Czeisler, C. A. Ageing and the circadian and homeostatic regulation of human sleep during forced desynchrony of rest, melatonin and temperature rhythms. *J. Physiol.*, 1999, 516: 611–627.
- Dijk, D. J., Duffy, J. F. and Czeisler, C. A. Age-related increase in awakenings: impaired consolidation of nonREM sleep at all circadian phases. *Sleep*, 2001, 24: 565–577.
- Esposito, M. J., Nielsen, T. A. and Paquette, T. Reduced Alpha power associated with the recall of mentation from Stage 2 and Stage REM sleep. *Psychophysiology*, 2004, 41: 288–297.
- Foulkes, D. Normal and abnormal REM sleep regulation: dreaming and REM sleep. *J. Sleep Res.*, 1993, 2: 199–202.
- Guérolé, F., Marcaggi, G., Baleyte, J. M. and Garma, L. Dreams in normal and pathological aging. *Psychol. Neuropsychiatr. Vieil.*, 2010, 8: 87–96.
- Hobson, J. A. and Pace-Schott, E. F. The cognitive neuroscience of sleep: neuronal systems, consciousness and learning. *Nat. Rev. Neurosci.*, 2002, 3: 679–693.
- Hofle, N., Paus, T., Reutens, D., et al. Regional cerebral blood flow changes as a function of delta and spindle activity during slow wave sleep in humans. *J. Neurosci.*, 1997, 17: 4800–4808.
- Knoblauch, V., Münch, M., Blatter, K. et al. Age-related changes in the circadian modulation of sleep-spindle frequency during nap sleep. *Sleep*, 2005, 28: 1093–1101.
- Maquet, P., Peters, J., Aerts, J. et al. Functional neuroanatomy of human rapid-eye movement sleep and dreaming. *Nature*, 1996, 383: 163–166.
- Marzano, C., Ferrara, M., Mauro, F. et al. Recalling and forgetting dreams: theta and alpha oscillations during sleep predict subsequent dream recall. *J. Neurosci.*, 2011, 31: 6674–6683.
- Münch, M., Knoblauch, V., Blatter, K. et al. Age-related attenuation of the evening circadian arousal signal in humans. *Neurobiol. Aging*, 2005, 26: 1307–1319.
- Nofzinger, E. A. Neuroimaging and sleep medicine. *Sleep Med. Rev.*, 2005, 9: 157–172.
- Ohayon, M. M., Carskadon, M., Guilleminault, C. and Vitiello, M. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep*, 2004, 27: 1255–1273.
- Rosenlicht, N., Maloney, T. and Feinberg, I. Dream report length is more dependent on arousal level than prior REM duration. *Brain Res. Bull.*, 1994, 34: 99–101.
- Suzuki, H., Uchiyama, M., Tagaya, H. et al. Dreaming during non-rapid eye movement sleep in the absence of prior rapid eye movement sleep. *Sleep*, 2004, 27: 1486–1490.
- Takeuchi, T., Ogilvie, R. D., Murphy, T. I. and Ferrelli, A. V. EEG activities during elicited sleep onset REM and NREM periods reflect different mechanisms of dream generation. *Clin. Neurophysiol.*, 2003, 114: 210–220.
- Timofeev, I., Grenier, F. and Steriade, M. Disfacilitation and active inhibition in the neocortex during the natural sleep–wake cycle: an intracellular study. *Proc. Natl Acad. Sci. USA*, 2001, 98: 1924–1929.
- Torsvall, L. and Åkerstedt, T. A diurnal type scale Construction, consistency and validation in shift work. *Scand. J. Work Environ. Health*, 1980, 6: 283–290.
- Zanasi, M., De Persis, S., Caporali, M. and Siracusano, A. Dreams and age. *Percept. Mot. Skills*, 2005, 100: 925–938.

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Figure S1. Rapid eye movement (REM) sleep electroencephalogram (EEG) power density before dream recall (black lines) and no recall (grey lines) for frontal, central, parietal and occipital derivations. EEG power density values per 0.25 Hz bin during REM sleep in older participants are expressed as percentage of the corresponding average values in the young. Horizontal line represents 100% of EEG activity in young participants. Mean values are shown for each 0.25 Hz frequency bin in the range of 0.75–20 Hz.

Figure S2. Non-rapid eye movement (NREM) sleep electroencephalogram (EEG) power density prior to dream recall and no recall during the biological day and night expressed as a relative ratio of day/night condition (where 0 = corresponding EEG power density for biological night). Data are presented for frontal, central, parietal and occipital derivations. EEG power density values per 0.25 Hz bin during NREM sleep are depicted for the older participants (black lines) and young participants (grey lines). Mean values are shown for each 0.25 Hz frequency bin in the range of 0.75–20 Hz. Horizontal stars near the abscissa at the bottom indicate frequency bins with a significant interaction ‘age’ × ‘derivation’ × ‘recall’ × ‘biological day/night’ ($P < 0.05$).

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.