Increased Alpha (8–12 Hz) Activity during Slow Wave Sleep as a Marker for the Transition from Implicit Knowledge to Explicit Insight

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Abstract

■ The number reduction task (NRT) allows us to study the transition from implicit knowledge of hidden task regularities to explicit insight into these regularities. To identify sleep-associated neurophysiological indicators of this restructuring of knowledge representations, we measured frequency-specific power of EEG while participants slept during the night between two sessions of the NRT. Alpha (8–12 Hz) EEG power during slow wave sleep (SWS) emerged as a specific marker of the transformation of presleep implicit knowledge to postsleep explicit knowledge (ExK). Beta power during SWS was increased whenever ExK was attained after sleep, irrespective of presleep knowledge. No such EEG predictors of insight were found during Sleep Stage 2 and rapid eye movement sleep. These results support the view that it is neuronal memory reprocessing during sleep, in particular during SWS, that lays the foundations for restructuring those task-related representations in the brain that are necessary for promoting the gain of ExK. ■

INTRODUCTION

Sleep may promote insight into regularities that have remained out of awareness before sleep. Evidence for this has been provided by Wagner, Gais, Haider, Verleger, and Born (2004), using the so-called number reduction task (NRT). In each trial of this task, participants have to produce seven responses to successive pairs of digits to obtain a final response. Unknown to participants, the responses follow a hidden abstract rule (Frensch et al., 2002; Woltz, Bell, Kyllonen, & Gardner, 1996), with the last three responses mirroring the three preceding ones. Implicit knowledge (ImK) of this regularity produces speeding of the last three responses (Yordanova et al., 2008; Rose, Haider, & Büchel, 2005; Rose, Haider, Weiller, & Büchel, 2002), whereas explicit insight into the regularity leads to reducing the number of responses in each trial from seven to two by providing abstract information about the final response. Participants' discovery of this rule is easily observed as it is accompanied by an abrupt change in behavior (Haider & Rose, 2007; Lang et al., 2006). Twice as many subjects who had slept after initial learning gained insight into the regularity than subjects who had not (Wagner et al., 2004).

This postsleep increase of insight might result from the restructuring of hippocampus-dependent memories during SWS (Yordanova et al., 2008; Rasch, Büchel, Gais, & Born, 2007; Born, Rasch, & Gais, 2006; Marshall, Helgadóttir, Mölle, & Born, 2006). There is some indirect evidence from neurophysiological measurements of NRT performance in favor of such an assumption. First, implicit learning in the NRT activates the hippocampus and the medial-temporal lobe (Rose et al., 2002, 2005). Second, presleep gain of ImK about NRT structure promotes the restructuring of implicit task representations by sleep, particularly by SWS. This was evidenced by a topographical reallocation of slow EEG potentials during task performance after sleep compared with performance before sleep. Slow EEG potentials time-locked to those responses, which could be predicted by the NRT regularity and which had been speeded up already before sleep were specifically reduced at visuo-motor cortical areas contralateral to the responding hand implying a spatial reorganization of implicit representations by SWS (Yordanova, Kolev, Wagner, & Verleger, 2009). Moreover, participants who already before sleep displayed signs of implicit learning, responding faster to predictable responses, had a higher probability of gaining insight specifically after early-night sleep rich in SWS (Yordanova et al., 2008). The sleep-related mechanisms that may mediate processes of transition of ImK before sleep to explicit knowledge (ExK) after sleep remain elusive, however, as long as no direct assessment has been provided so far of the neurophysiological processes occurring during sleep.

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According to a model for a two-stage memory system, information acquired while awake is temporarily stored in a buffer system under control of the hippocampus, from where it is transferred during non-rapid eye movement (REM) sleep to long-term stores in the neocortex (Buzsáki, 1998; McClelland, McNaughton, & O'Reilly, 1995). A candidate mechanism underlying this transfer involves sharp wave–ripple activation patterns that are generated in the hippocampus (Wierzynski, Lubenov, Gu, & Siapas, 2009; Qin, McNaughton, Skaggs, & Barnes, 1997; Chrobak & Buzsáki, 1996) and are temporally associated to thalamo-cortical spindles, forming ripple–spindle events (Siapas & Wilson, 1998). This mechanism is suggested to enhance synaptic plastic changes in neocortical networks (Rosanova & Ulrich, 2005; Steriade, 2001), thus supporting the storage of information transferred from the hippocampus.

In humans, oscillatory EEG activity of non-REM sleep has been likewise implicated with neurophysiologic mechanisms of sleep-related memory consolidation (Clemens et al., 2007; Marshall & Born, 2007; Born et al., 2006; Gais & Born, 2004; Gais, Mölle, Helms, & Born, 2002). Slow (<1 Hz) cortical oscillations occurring during SWS have been demonstrated to group and synchronize both slow delta waves and frontal spindle activity from the alpha (8–12 Hz) range generated during this sleep stage (Steriade, 2003, 2006; Mölle, Marshall, Gais, & Born, 2004; Mölle, Marshall, Gais, & Born, 2002). Importantly, boosting the slow (<1 Hz) cortical oscillations during SWS by applying transcranial direct current stimulation to the frontal cortex both enhanced the power of frontal activity within 8–12 Hz (interpreted by those authors as slow spindle activity) and improved retention of declarative (explicit) memories (Marshall et al., 2006). During Sleep Stage 2 (S2) sleep, spindle EEG activity from the sigma band (~13–16 Hz; for review, see Olbrich & Achermann, 2005; De Gennaro & Ferrara, 2003; Zeitlhofer et al., 1997) has been correlated with improved motor procedural learning (Morin et al., 2008; Fogel & Smith, 2006; Milner, Fogel, & Cote, 2006) as well as visuospatial (Clemens et al., 2007), visuomotor (Tamaki, Matsuoka, Nittono, & Hori, 2008), and declarative paired word learning (Schmidt et al., 2006; Schabus et al., 2004; Gais et al., 2002). Thus, oscillatory EEG patterns of non-REM sleep, 8–12 Hz during SWS and 13–16 Hz during S2, have been discussed as neurophysiologic indicators of both explicit and implicit memory consolidation.

In the present study, we investigate the relation between sleep EEG features and transformation from implicit to ExK about NRT structure. We explore if spectral sleep EEG distinguishes between participants in the NRT (i) who do or do not generate insight, that is, ExK after sleep, (ii) who had or had not acquired ImK about the task regularity before sleep, and (iii) who do or do not transform such ImK to ExK after sleep. These different types of knowledge conditions were identified in the study of Yordanova et al. (2008) where early-night sleep, rich in SWS, but not late-night sleep, rich in REM sleep, supported the ImK-to-ExK transition, pointing to a critical role of SWS in the process of knowledge transformation.

To specify the relationships between sleep physiology and knowledge transformation, we analyzed the EEG recorded during sleep between two NRT sessions. Sleep EEG data were originally acquired in the context of a study where participants slept between two sessions of the NRT, either in the first or in the second half of the night. Behavioral and EEG results obtained during NRT performance before and after sleep were published elsewhere (Yordanova, Kolev, Wagner, & Verleger, 2010; Yordanova, Kolev, & Verleger, 2009; Yordanova, Kolev, Wagner, et al., 2009; Yordanova et al., 2008). Here, EEG recorded during sleep was analyzed. Registration was made from C3 and C4 electrodes only, for scoring the sleep stages according to Rechtschaffen and Kales (1968). Sleep EEG power was analyzed in the delta (1–4 Hz), theta (4–7 Hz), alpha (8–12 Hz), sigma (13–16 Hz), and beta (17–25 Hz) frequency bands.

On the basis of the evidence presented above about associations of sleep EEG with explicit and implicit memory consolidation, our main interest was on the alpha band during SWS and on the sigma band during S2 (Marshall et al., 2006; Schmidt et al., 2006; Schabus et al., 2004; Gais et al., 2002). Also, we predicted that EEG during SWS would be most sensitive to knowledge transformation because (1) SWS has been proven most relevant to the transition of implicit learning to explicit insight in the NRT (Yordanova, Kolev, Wagner, et al., 2009; Yordanova et al., 2008) and (2) SWS has been demonstrated to promote the consolidation of hippocampus-dependent memories (Diekelmann & Born, 2010; Rasch et al., 2007) that would be critically involved in the presleep implicit learning of the currently used task (Rose et al., 2002, 2005). Specifically, with respect to the above-made distinctions between participants who will or will not attain insight after sleep and who did or did not show signs of ImK before sleep, we predicted that 8–12 Hz activity during SWS would be larger in participants who will attain insight after sleep and that the level of ImK about the regularity attained already before sleep would modify the expected increase of 8–12 Hz activity during SWS.

METHODS

Participants

The same sample as reported in Yordanova et al. (2008) was used for the present spectral sleep EEG analysis. Because of artifacts in the sleep EEG data, nine participants from the original data set were not included in the present analyses. Thus, a total of 46 participants were used here for statistical comparisons, 26 (seven women) from the early-night group and 20 (nine women) from the late-night group (Table 1). They were healthy students (18–28 years old) without any history of sleep disturbances or

Table 1. Distribution of Sleep Stages in the Early- versus Late-night Group

			Early vs. Late	
	Early-night Group (n = 26)	*Late-night Group (n = 20)*	*F(1, 44)*	*p*
Wake (%)	1.51 ± 0.61	0.49 ± 0.35	1.87	*ns*
S1 (%)	6.60 ± 1.01	6.11 ± 0.71	0.14	*ns*
S2 (%)	58.89 ± 2.66	59.34 ± 2.38	0.02	*ns*
SWS (%)	27.21 ± 2.61	12.25 ± 2.10	18.53	< .001
REM (%)	5.54 ± 1.00	21.74 ± 1.43	89.96	< .001
Total sleep time (min)	192.24 ± 3.47	190.93 ± 3.26	0.07	*ns*

Means ± *SEM* are indicated. Data refer to the sleep interval between initial practice and retesting. Statistical results are from one-way ANOVA comparing early- and late-night groups.

psychiatric or neurological disorders. Before the experiment, all participants spent an adaptation night in the sleep laboratory, including placement of electrodes. They were paid for their participation and gave informed written consent before the study, which had been approved by the local ethics committee.

Stimuli and Procedure

Number Reduction Task

The task is illustrated in Figure 1A by an example trial, see Yordanova et al. (2008) for details. Briefly, on each trial, a different string of eight digits was presented, composed of the digits 1, 4, and 9. For each string, its final result had to be determined, to be highlighted by pressing the "Enter" key. This was to be achieved by sequentially processing pairs of digits from left to right according to two simple rules: (1) The result of two identical digits is the same digit (identity rule, e.g., 4 and 4 gives 4; R5 in Figure 1A). (2) The result of two non-identical digits is the remaining third digit (difference rule, e.g., 1 and 9 gives 4; R1 in Figure 1A; 4 and 1 gives 9; R2 in Figure 1A, etc.). The "1," "2," and "3" keys on the PC numeric pad were labeled accordingly as "1," "4," and "9," and served as response keys. The entered responses appeared on the screen and remained there until the end of the trial, thereby forming an expanding response string below the stimulus sequence.

Importantly, unmentioned to participants, all strings were generated according to the same underlying regularity, which, if discerned, allowed an early determination of the final result. Specifically, all response sequences had the form A-B-C-D-D-C-B (with A, B, C, and D representing one of the digits 1, 4, or 9), that is, the last three responses always mirrored the preceding three responses, so that the second response in each trial coincided with the final result (Figure 1A). Thus, when gaining insight into this regularity, participants abruptly cut short their sequential responding by pressing the "Enter" key already after the second response (R2), whereupon the trial was finished and the next trial started. Note that this regularity is abstract because the actual digit strings and responses changed from trial to trial. Thus, discovery of the rule cannot simply be on the basis of repetition of the same finger movements in all trials. RTs were measured separately for each response in the response string (details in Yordanova et al., 2008).

The "Split Night" Design

Participants were tested individually in a sound-attenuated room. The time schedule of the experiment is outlined in Figure 1B. There was a presleep session comprising three task blocks and a postsleep retest session of 10 task blocks at most, with 30 trials in each block. Insight was automatically identified when at least 24 correct shortcuts occurred within the same block, in which case the task was terminated. The presleep session was preceded by detailed standardized instructions given by the computer, which included a short practice block of 10 trials. To assure correct understanding of the "identity" and "difference" rules, the practice block was repeated when any mistake had occurred.

The interval between the presleep session and retest was filled with 3 hr of sleep either in the early night, rich in SWS (early-night group), or in the late night, rich in REM sleep (late-night group). Early-night participants reported to the laboratory at about 21:00 hr. After placement of electrodes (for EEG and polysomnographic recordings), they performed the first session of three blocks (including preceding computer-guided practice) at about 22:00 hr and thereafter went to bed at about 23:00 hr. After 3 hr of sleep, they were awakened to perform the 10 blocks of the retest. Late-night participants reported to the laboratory at about 22:00 hr and, after placement of electrodes, first slept for 3 hr in the early night (to "consume" SWS) before performing the first session at about 2:30 hr. Then, they slept again for another 3 hr (about 3:30–6:30 hr), followed by retesting in the morning. Participants were awakened from light Sleep Stages 1 or 2 only to avoid cognitive disturbances that may occur when

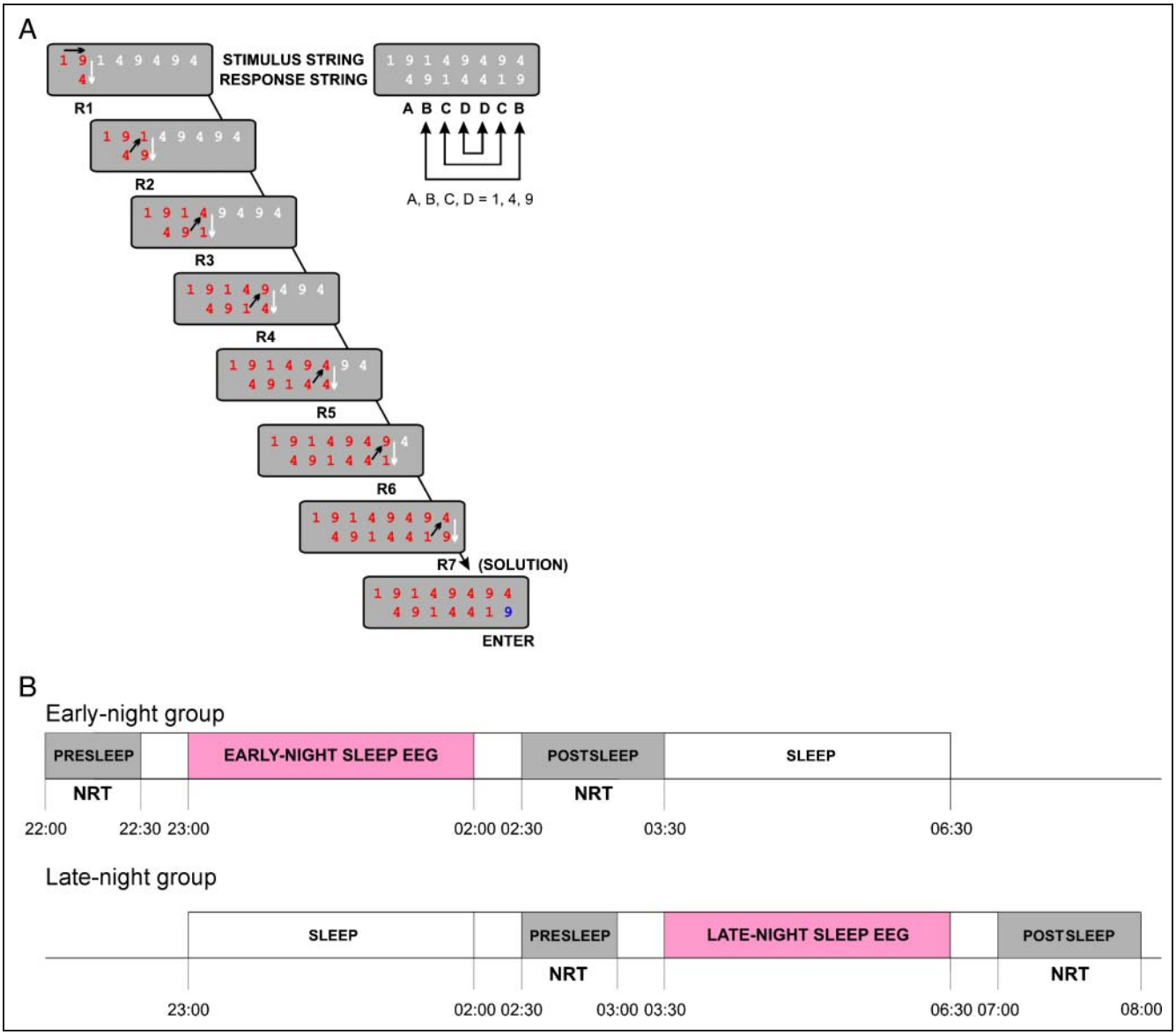

Figure 1. Task and experimental design. (A) Schematic representation of the NRT. Consecutive steps of single trial execution. Black and white arrows (not shown to participants) within the screen displays mark the consecutive steps to be performed; for example, the first two numbers 1 and 9 in the stimulus string lead to Response 4 (R1), then the same response (4) has to be compared with the next number from the stimulus string (1) leading to Response 9 (R2), and so on. The final result is the last response (R7) marked as SOLUTION, which is followed by pressing the Enter key. On the right, the structure of the response string is given by letters, with equal letters meaning equal responses, thereby forming the mirror structure B-C-D–D-C-B. (B) The experimental design. Times of sleep EEG recordings are marked in red for the two sleep groups (early- and late-night group). The time of NRT performance is also shown. Times of sleep in the two sleep groups are shown principally in the experimental design. There were minor individual deviations in total sleep time because of the constraint to wake up subjects only in a stage of light sleep. These individual deviations from the principal schedule are reflected in Table 1.

being raised out of SWS or REM sleep, which introduced a slight and nonsignificant variation in total sleep time (Table 1). As an additional control, subjective levels of sleepiness, activation, boredom, concentration, and motivation were assessed on 5-point scales immediately before and after the presleep and the postsleep sessions.

After the retesting session, participants' ExK of the hidden task structure was assessed by a behavioral test and a questionnaire. The behavioral test comprised a speeded task in which 16 different strings were presented, and participants had to indicate the final result to each string within 2 sec after string presentation. The following questionnaire began with general questions that asked for strategies used during task performance, followed by more specific questions about particular features of the sequence of stimuli and responses.

Performance Groups

Presence of ExK about the mirrored sequence of responses (x-A-B-C-C-B-A) was determined from performance and

from answers in the postexperimental questionnaire. Questionnaire data confirmed that all 13 subjects (of 46, see further below for details) whose insight into the hidden structure had been identified automatically by the task program from the shortcut in sequential responding were also able to verbalize the critical explicit rule knowledge correctly using their own words, and they were also able to give correct solutions to new digit strings within 2 sec.

ImK was assessed for correct and complete trials on the basis of faster RTs to responses that were predictable because of the mirror structure of the response strings in comparison with unpredictable responses (for details, see Yordanova et al., 2008; see also Lang et al., 2006; Rose et al., 2002, for evidence that this speeding is not just because of unspecific within-trial speeding). Briefly, for each block and participant, the mean RTs of the unpredictable responses R3 and R4 and of the predictable responses R6 and R7 were formed in each trial and were tested against each other across trials by one-way repeated measures ANOVAs. To control for multiple comparisons across blocks, effects were accepted as significant only if $p < .01$. At the presleep session, this analysis was performed for all participants, at retesting only for those who did not gain ExK (because subjects who gained insight did not perform the full amount of task blocks). Participants who satisfied neither ImK nor ExK criteria were defined as having developed no knowledge (NoK) about the hidden task regularity.

Participants who gained insight into the hidden NRT regularity before sleep had already been excluded from the present sample. Thus, according to their presleep performance, there were two groups, ImK and NoK. Either group was then subdivided into three subgroups of transition, depending on individual postsleep performance: post-ExK, subjects who gained insight after sleep; post-ImK, subjects who gained ImK after sleep; and post-NoK, subjects who had NoK after sleep. In this way, six transition subgroups were formed for both the early-night group and the late-night group, as displayed in Table 2.

Table 2. Distribution of Participants according to Their State of Knowledge before and after Sleep

	Total	*Pre-NoK*	*Pre-ImK*
Early-night group	26	13 (50.0%)	13 (50.0%)
Late-night group	20	9 (45.0%)	11 (55.0%)

(A) Pre-NoK

	Total	*Pre-NoK* ↓ *Post-NoK*	*Pre-NoK* ↓ *Post-ImK*	*Pre-NoK* ↓ *Post-ExK*
Early-night group	13	7 (53.8%)	3 (23.1%)	3 (23.1%)
Late-night group	9	3 (33.3%)	3 (33.3%)	3 (33.3%)

(B) Pre-ImK

	Total	*Pre-ImK* ↓ *Post-NoK*	*Pre-ImK* ↓ *Post-ImK*	*Pre-ImK* ↓ *Post-ExK*
Early-night group	13	2 (15.4%)	5 (38.5%)	6 (46.2%)
Late-night group	11	1 (9.1%)	9 (81.8%)	1 (9.1%)

Indicated are numbers (percentages) of participants from the early- and late-night groups, displaying different postsleep states of knowledge about the hidden task structure at retesting, depending on whether they had NoK (A) or ImK (B) already at initial practice before sleep. Abbreviations: Pre-NoK = presleep NoK; Pre-ImK = presleep ImK; Post-NoK = postsleep NoK; Post-ImK = postsleep ImK; Post-ExK = postsleep ExK; ↓ = transition of knowledge state from pre- to postsleep.

Sleep EEG: Recording and Analysis

EEG was recorded with Ag–AgCl electrodes during nighttime sleep from two scalp locations (C3 and C4) referred to the nose and was amplified with Neuroscan Synamps (frequency limits = 0.1–35 Hz, sampling rate = 200/s). Figure 1B shows the periods (marked in red) when sleep EEG was recorded for the early-night group in the first half of the night and for the late-night group in the second night-half. Additionally, submentally attached EMG and two EOG channels were recorded. Using these recordings, manual scoring of sleep stages in 30-sec periods was done by an experienced rater who was blind to the hypotheses, following the criteria of Rechtschaffen and Kales (1968). Quantitative analysis of sleep EEG frequency bands was done separately for the scored S2, SWS (S3 and S4 combined), and REM. Sleep Stage S1 (S1) was not included in the planned design and was only used for control analyses to further delineate specificity of the results obtained for S2, SWS, and REM.

For analysis of frequency bands, after artifact rejection procedures (automatic threshold of ±200 μV in EOG records and manual inspection of all channels), the artifact-free records for each sleep stage (S2, SWS, and REM sleep) were divided into equal-sized epochs of 5.12-sec duration each. The mean number (±*SD*) of epochs in the early-night group was 1193 (±362) for S2, 553 (±281) for SWS, and 137 (±30) for REM sleep and in the late-night group 1290 (±282) for S2, 251 (±93) for SWS, and 455 (±152) for REM sleep, reflecting the distribution of different sleep stages in early- and late-night sleep. After applying a 20% Hanning window, these epochs were fast Fourier transformed with a frequency resolution of 0.195 Hz and averaged separately for each sleep stage. The averaged power spectra were digitally smoothed by a 3-point moving average. For analysis, several neighboring bins were averaged to reduce the frequency resolution to ~1 Hz and

provide measures for each ~1 Hz frequency bin from 1 to 25 Hz.

Spectral Sleep EEG Analysis

The hypotheses of the present study were tested by analysis of spectral power of sleep EEG in specific frequency bands: delta (1–4 Hz), theta (4–7 Hz), alpha (8–12 Hz), sigma (13–16 Hz), and beta (17–25 Hz). Band-specific average spectral measures were subjected to repeated measures ANOVAs as described below. We also used MANOVAs, in which 1-Hz power bins were analyzed separately for each frequency band, for each sleep stage, and for each hemisphere, with the single 1-Hz spectral values within each frequency band being treated as multivariate data. These single-bin MANOVAs could control for the frequency specificity of results and accounted for the gross interindividual variability (De Gennaro & Ferrara, 2003). Thus, there were two statistical designs: (1) repeated measures ANOVAs for average power values computed for five frequency bands and (2) frequency bin-specific MANOVAs for each band. Before statistical analyses, power values were log10-transformed to normalize distributions.

For both the ANOVAs and MANOVAs, there were two between-subject factors: Sleep Group (early night vs. late night) and Knowledge Group. The factor Knowledge Group was formed in three ways to enable assessing the relationship of spectral sleep EEG to (1) ExK generation after sleep, (2) ImK acquisition before sleep, and (3) transition of ImK to ExK during sleep. Accordingly, there were three classifications of participants (cf. Table 2): (1) ExK group reflecting whether ExK was or was not attained in the session after sleep (solvers [post-ExK] vs. nonsolvers [post-NoK and post-ImK]). After sleep, there were 9 early-night solvers, 17 early-night nonsolvers, 4 late-night solvers, and 16 late-night nonsolvers. (2) ImK group reflecting whether ImK was or was not gained before sleep (pre-ImK vs. pre-NoK, A vs. B in Table 2). As denoted in Table 2, the four subgroups, pre-ImK versus pre-NoK in the early- and late-night group, had 13, 9, 13, and 11 subjects, respectively. (3) Knowledge transformation group reflecting whether ImK before sleep remained implicit after sleep (nontransformers [pre-ImK to post-ImK]) or became explicit after sleep (transformers [pre-ImK to post-ExK]). As evident from Table 2, the four subgroups had 5, 9, 6, and 1 subjects, respectively, which was not enough for proper statistical analyses. Therefore, participants were pooled across early- and late-night groups, yielding 14 nontransformers and 7 transformers altogether.

For each of the three definitions of Knowledge Group, ANOVAs were performed separately for each frequency band (delta, theta, alpha, sigma, beta) and for each sleep stage (SWS, S2, REM). The between-subject factors were Sleep Group and Knowledge Group, and the within-subject factor was Hemisphere (C3 vs. C4). Because the present study tested specific hypotheses related to frequency band and sleep stage, no correction for multiple tests was required for multiple ANOVAs.

MANOVA applications to defined frequency bands, however, required a correction for multiple tests depending on the number of frequency bins and the number of leads, which, according to the full Bonferroni adjustment (Bland & Altman, 1995; simple interactive statistical analysis on www.quantitativeskills.com/sisa/), reduced the alpha level p to .006 for delta, theta, and sigma (4 frequency bins × 2 leads) to .005 for alpha (5 frequency bins × 2 leads) and to <.003 for the beta band (9 frequency bins × 2 leads). However, cross correlations within separate frequency bands revealed means for the Pearson correlation coefficients of ~1 for the beta band, between ~.9 and ~1 for the delta, theta, and sigma bands, and .82 for the alpha band. A full Bonferroni adjustment is largely unrecommended for highly correlated multivariate-dependent variables when they are not produced by repeated measures observations (i.e., when the sphericity assumption is not violated), as in the present case (Perneger, 1998). This means that a full Bonferroni adjustment from alpha level $p = .05$ to $p = .006$, .005, or .003, is justified with fully uncorrelated ($r = 0$) dependent variables, whereas no adjustment at all is applied to fully correlated data ($r = 1$). For other values of the correlations, a corrected alpha should be between the two extremes, depending on the strength of correlation (Perneger, 1998; Sankoh, Huque, & Dubey, 1997). With this regard, no corrections of p were adopted for any of the frequency bands, with exception of the alpha band (corrected p criterion was .04).

In an overall control analysis performed to assess general differences in sleep spectra between the early-night and the late-night groups, power values of the five frequency bands were entered into a common ANOVA, separately for each sleep stage (S2, SWS, REM) with a between-subject factor sleep group and within-subject factors hemisphere (C3 vs. C4) and band (delta, theta, alpha, sigma, beta). For repeated measures factors with more than two levels, degrees of freedom were corrected using the Greenhouse–Geisser procedure. A variety of control analyses also were conducted using the same statistical methods (see specific details in Results).

Relations to Sleep Spindle Activity

Main analyses (see Results) revealed specific associations of spectral alpha (8–12 Hz) and sigma (13–16 Hz) bands with sleep-related knowledge transformation and generation. Frequencies of these bands have been traditionally related to spindle activity of the sleep EEG (De Gennaro & Ferrara, 2003). One criterion for identifying spindle activity during sleep is the topography of spindles. Fast spindles (>13 Hz) from the sigma band during S2 have a typical parietal distribution, whereas slow spindles during S2 (~11–13 Hz) and slow spindles during SWS (8–12 Hz) appear typically with frontal distribution (Marshall et al., 2006; Anderer et al., 2001). Because here we only

have two recordings at central (C3 and C4) electrodes, we cannot use these topographic criteria (in addition to frequency-based criteria) to identify spindles and infer relations between spectral EEG and spindles. Yet, to provide some link to spindle sleep EEG activity and a reference to existing data, we used other established criteria for spindle identification related to the power and temporal duration of spindles. Thus, spindles were detected by using an algorithm adopted from previous studies (Marshall et al., 2006; Gais et al., 2002): The EEG signal was filtered in the alpha (8–12 Hz for SWS) and sigma bands (13–16 Hz for each sleep stage) using infinite impulse response digital filters (24 dB/octave, BrainVision Analyzer 2.0.1 User Manual, 2009). The root mean square (RMS) of each 100-msec interval was calculated. Spindles were defined as consecutive intervals of at least 0.5 sec and at most 3.5 sec for which the RMS signal exceeded a threshold (adapted to visually identified templates on the basis of individual RMS means). If the interval is longer than 3.5 sec, the entire period was excluded and search started again from the next below-threshold point. Spindle density was defined as the number of such spindle events in 30-sec epochs. Spindle densities of alpha band during SWS individually averaged across all 30-sec epochs were compared between "transformers" and "nontransformers" in an ANOVA with the factors knowledge transformation group and hemisphere. Also, spindle densities of sigma band during all sleep stages were compared between subjects who had or had not acquired ImK before sleep.

EEG Recording and Analysis during NRT

To assess specific associations of the results to sleep, the EEG recorded during NRT performance also was analyzed. This EEG was recorded continuously from 28 scalp electrodes along with EOG, referenced to linked mastoids, amplified by using a Neuroscan Synamps (Brain Products GmbH, Gilching, version 1.05) and further processed. EEG and EOG were filtered within the pass-band 0.03–70 Hz and sampled with a frequency of 250 Hz. For evaluation of EEG changes during task execution, 30 artifact-free and EOG-corrected (Gratton, Coles, & Donchin, 1983) EEG segments were collected for two conditions of NRT performance (end of presleep practice and start of post-sleep performance). Segments with a length of 800 msec were collected before each of the seven responses, tapered by Hanning windows with a length of 20% from the epoch boundaries, and analyzed using a fast Fourier transform to obtain the power spectrum with a frequency resolution of 0.977 Hz (for details, see Yordanova, Kolev, & Verleger, 2009; Yordanova, Kolev, Wagner, et al., 2009). Single-trial spectra were averaged, and mean values were calculated for each single-frequency bin from 2 to 18 Hz and for the frequency bands alpha (8–12 Hz), sigma (13–16 Hz), and beta (17–25 Hz). Statistical analyses (MANOVAs) were performed for C3 and C4 electrodes and for each frequency bin and band after a log10 transformation, as described for sleep EEG.

RESULTS

Sleep EEG and Gain of Insight after Sleep

The first analysis used the ExK factor and compared participants who did ("solvers," post-ExK, $n = 13$) or did not ("nonsolvers," post-NoK and post-ImK, $n = 33$) gain insight after sleep (Table 2). As shown in Figure 2, ExK after sleep was associated with higher beta power in SWS ($F(1/42) = 6.84$, $p = .012$ in the ANOVA; $F(1/44) = 7.8–11.3$, $.009 \geq p \geq .001$ for each frequency in the MANOVA of

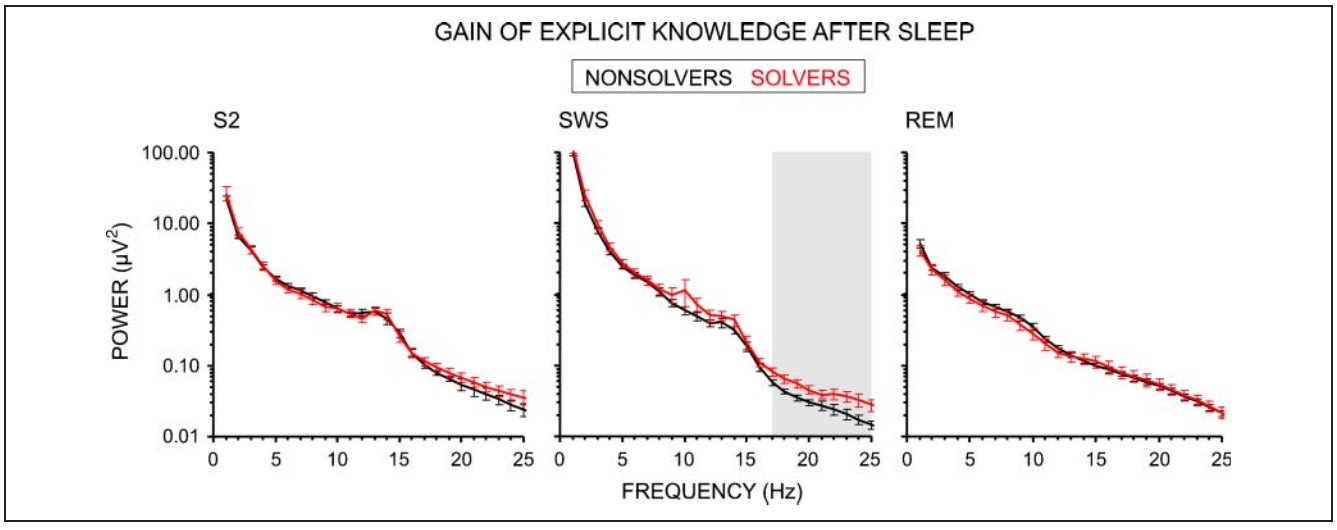

Figure 2. Power spectrum of sleep EEG for solvers and nonsolvers. Solvers are the 13 "post-ExK" participants (compiled in the rightmost column of Table 2), nonsolvers are all other 33 participants (both "post-NoK" and "post-ImK" in Table 2). Depicted is the grand average power spectrum (across electrodes C3 and C4) for three sleep stages: S2, SWS and REM. The shaded area in SWS indicates the frequency range of significant differences between nonsolvers and solvers. Standard error bars are presented for each frequency bin.

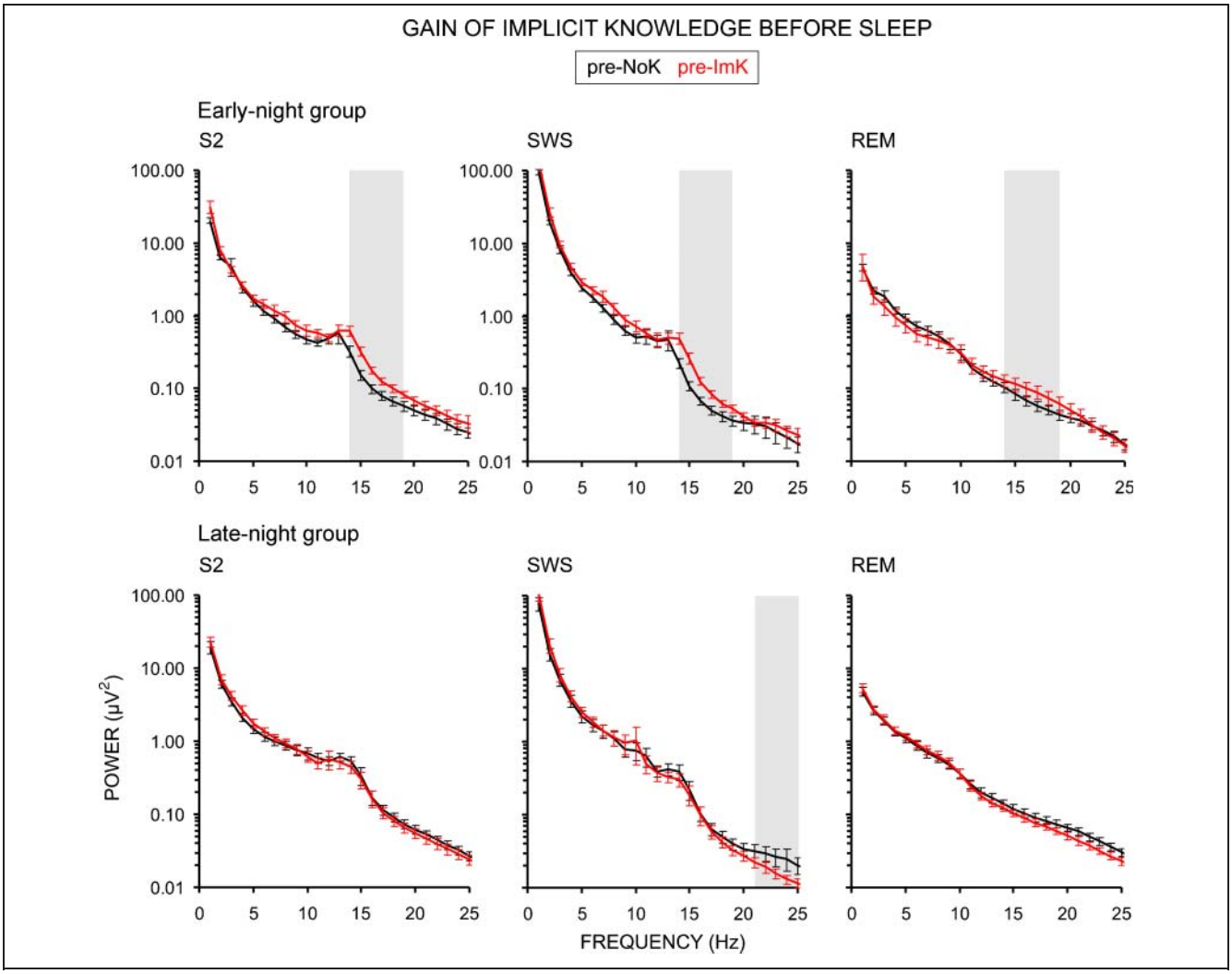

Figure 3. Power spectrum of sleep EEG for participants with and without ImK before sleep. Depicted is the grand average power spectrum (10–25 Hz) across electrodes C3 and C4 for three sleep stages: S2, SWS, and REM. Shaded areas indicate frequency ranges of significant differences between subjects with no ImK about the hidden regularity of the task before sleep ("pre-NoK," i.e., all participants compiled in Table 2A) and subjects with such ImK ("pre-ImK," i.e., all participants compiled in Table 2B). Standard error bars are presented for each frequency bin.

the beta band). This effect did not depend on the night group (Sleep Group × ExK, $p > .4$). Other frequency bands of SWS EEG or spectral features of S2 EEG and REM EEG did not distinguish solvers from nonsolvers (Figure 2). Because myographic activity may specifically contaminate spectra of higher-frequency EEG, the effect of ExK was tested for the EMG signal within 17–25 Hz. No such effects were obtained ($F(1/42) = 0.68$, $p = .4$ in the ANOVA; $F(1/44) = 0.009$–1.2, $.9 \geq p \geq .2$ in the MANOVAs), thus excluding EMG contributions to the SWS beta increase in solvers.

To control how specific this increase of beta was to off-line processing during sleep and to exclude accidental trait-like differences between groups, a supplementary MANOVA tested the effects of Sleep Group and ExK on beta EEG activity measured during NRT performance at C3 and C4 before and after sleep. The main and interactive effects of Sleep Group ($F(1/44) = 0.001$–1.52, $.9 \geq p \geq .2$) and ExK ($F(1/44) = 0.004$–1.6, $.9 \geq p \geq .2$) were not significant ($.9 \geq p \geq .2$). To further control for the specific associations of beta increase with SWS, a MANOVA was performed for spectral beta EEG activity also during light sleep (S1) in the beginning of the sleep period, that is, during the first sleep cycle. No significant main or interactive effects of these factors were found ($F(1/44) = 0.001$–0.6, $.97 \geq p \geq .4$).

Sleep EEG and Gain of ImK before Sleep

Furthermore, sleep EEG was analyzed according to whether ImK was apparent before sleep ("pre-ImK," $n = 24$; Table 2B) or not ("pre-NoK," $n = 22$; Table 2A). For these analyses, the ImK factor was used. As Figure 3 shows, gain of ImK before sleep was associated with sigma and beta activity in each sleep stage (SWS, S2, and REM), but these associations were different for the early- and the

late-night group (Sleep Group × ImK: $F(1/42) = 4.4–6.6$, $.05 \geq p \geq .01$ in the ANOVAs for sigma activity; $F(1/42) = 3.9–8.9$, $.05 \geq p \geq .005$ in the ANOVAs for beta activity). Sigma and beta MANOVAs for SWS, S2, and REM specified the frequency characteristics of these effects by yielding significant Sleep Group × ImK interactions for each frequency bin from 14 to 19 Hz for all sleep stages ($F(1/44) = 4.5–12.0$, $.05 \geq p \geq .001$ for each frequency bin). For SWS there were also interactive effects for 21–25 Hz frequency bins ($F(1/44) = 4.07–7.3$, $.05 \geq p \geq .007$).

Thus, in the early-night group, ImK before sleep produced larger power for 14–19 Hz frequency bins indicating a major effect on sleep sigma EEG activity slightly shifted to higher frequencies. The increase was observed equally for all sleep stages (SWS: $F(1/25) = 4.3–12.1$, $.05 \geq p \geq .002$; S2: $F(1/25) = 4.5–12.8$, $.05 \geq p \geq .002$; REM: $F(1/25) = 5.1–15.4$, $.03 \geq p \geq .001$). No such increase was obtained in the late-night group. Rather, in this group, beta EEG activity of SWS was smaller in participants with than without ImK, $F(1/19) = 4.3–6.6$, $.03 \geq p \geq .019$ for 21–25 Hz frequency bins (Figure 3).

Again, it was controlled whether the increase of sigma activity in the early-night group was specific to sleep, by analyzing the predefined 13–16 Hz sigma band as well as the presently significant 14–19 Hz activities during NRT performance before and after sleep. No effect involving pre-ImK became significant ($p > .2$, for all relevant comparisons). Analysis of spindle density in the 13–16 Hz band demonstrated that spectral power effects were not accompanied by a related increase in the number of spindles (Sleep Group × ImK: $F(1/42) = 0.30–0.85$, $p \geq .3$ in the ANOVAs for each sleep stage), with similar results found for the 14–19 Hz activity. None of the group factors became significant.

Sleep EEG and Transition of Implicit to ExK

Finally, sleep EEG frequency bands were investigated in their relation to transformation of ImK before sleep to ExK after sleep. One analysis was restricted to those participants who had acquired ImK before sleep, contrasting "transformers," that is, those who became solvers after sleep (pre-ImK to post-ExK, $n = 7$; Table 2B) to "nontransformers" who stayed with this ImK after sleep (pre-ImK to post-ImK, $n = 14$; Table 2B), pooled across early- and late-night groups because else, the number of subjects would be too low. As illustrated in Figure 4, transformation of presleep ImK to postsleep ExK was specifically associated with SWS, in manifesting main effects of knowledge transformation in ANOVAs on the average alpha 8–12 Hz ($F(1/19) = 4.4$, $p = .05$) and beta bands ($F(1/19) = 6.1$, $p = .02$). For the alpha band, there was a trend for a stronger effect on the right (Knowledge Transformation × Hemisphere interaction, $F(1/19) = 3.6$, $p = .07$). Larger spectral power of transformers was confirmed and specified with respect to frequency by MANOVAs yielding significant effects of Knowledge Transformation for 10–11 Hz frequency bins in the alpha MANOVA ($F(1/19) = 5.1–5.3$, $p \leq .04$) and 17–25 Hz frequency bins in the beta MANOVA ($F(1/19) = 5.9–8.0$, $.05 \geq p \geq .01$). No effects of Knowledge Transformation group on any of the frequency bands were found for S2 and REM sleep stages.

Very similar results were obtained when the seven transformers (pre-ImK to post-ExK) were compared with the

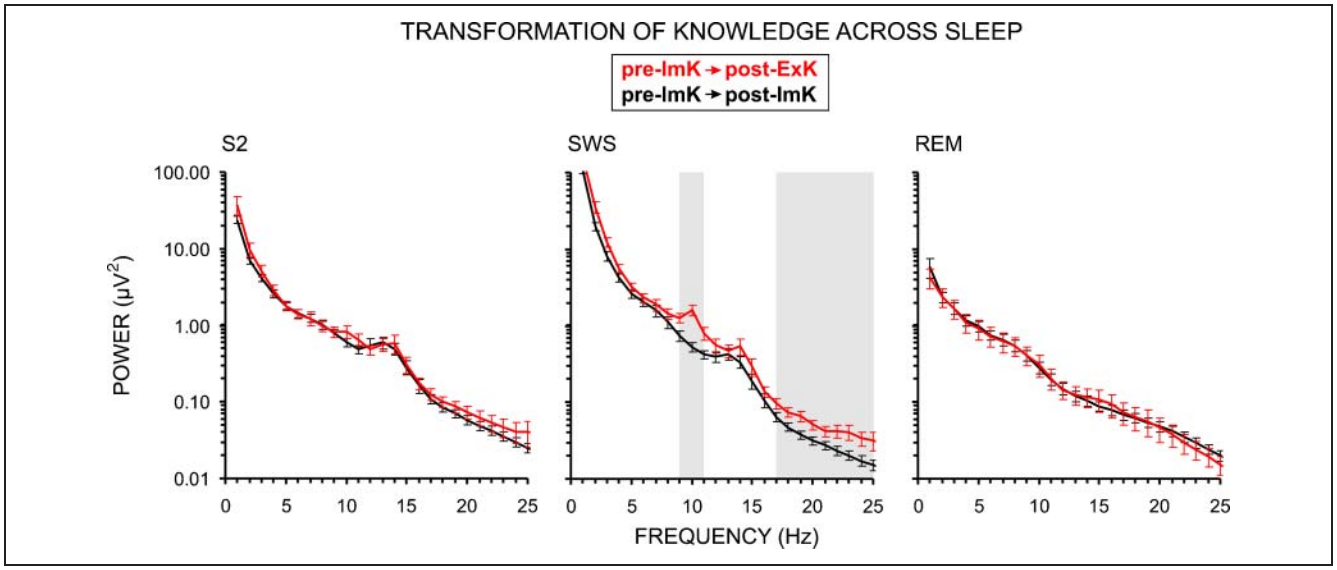

Figure 4. Power spectrum of sleep EEG for participants who showed transformation of knowledge across sleep. Participants with ImK before sleep who became solvers after sleep ("transformers," "pre-ImK → post-ExK") are presented in red. These 7 participants are compared with the 14 participants who showed ImK both before and after sleep ("nontransformers," "pre-ImK → post-ImK"), presented in black. Displayed is the grand average power spectrum (across electrodes C3 and C4) for the three sleep stages: S2, SWS and REM. Shaded areas indicate frequency ranges of significant difference between groups. Standard error bars are presented for each frequency bin.

other post-ExK participants, that is, to those solvers who had not acquired ImK before sleep (pre-NoK to post-ExK, $n = 6$, Table 2A). Consistent with the preceding analysis, ANOVA revealed a group difference in the 8–12 Hz band, with higher power on the right side for transformers with ImK before sleep relative to transformers without ImK before sleep (Hemisphere × ImK $F(1/11) = 15.3$, $p = .002$; effect of ImK at C3: $F(1/11) = 0.17$, $p = .7$; at C4: $F(1/11) = 4.9$, $p = .05$).

Despite the fact (reported below) that early- and late-night groups did not differ in broadband power, we aimed to further exclude the possibility that these effects of knowledge transformation in the alpha band were produced by differences between early- and late-night groups (because most transformers belonged to the early-night group). Thus, we made an additional comparison of early- versus late-night group by using only NoK subjects, none of whom were included in the main analysis of transformers versus nontransformers. These were the pre-NoK subjects (Table 2A, $n = 22$) and the post-NoK subjects (Table 2B, $n = 3$), pooled together ($n = 25$) for the sleep group comparison. No effect of sleep group was found in the ANOVA ($F(1/24) = 2.7$, $p = .1$). This was confirmed by nonsignificant Sleep Group effects in the MANOVAs ($F(1/24) = 0.5$–3.5, $.5 \geq p \geq .07$); the tendency reflected by these results rather went to the direction opposite to the critical effect.

As in the preceding analyses, we controlled whether the transformation group differences in the alpha and beta bands were specific to sleep by conducting the same comparison between groups for the alpha and beta bands during NRT performance before and after sleep. These analyses did not yield any effects of transformation group ($F(1/19) < 2.7$, $p > .2$) nor any interactions with early or late night or other factors ($p > .5$).

To provide a link to previously reported results of sleep spindle activity in the 8–12 Hz band during SWS (Marshall et al., 2006), an additional analysis compared the frequency of occurrences of distinct 0.5–3.5 sec packages within the 8–12 Hz range during SWS between "transformers" and "nontransformers." A Knowledge Transformation × Hemisphere ANOVA revealed a significant effect of Knowledge Transformation ($F(1/19) = 6.8$, $p = .019$), reflecting a higher spindle density in transformers ($7.24 \pm 0.32 \times 30\ s^{-1}$) than in nontransformers ($6.22 \pm 0.22 \times 30\ s^{-1}$). This effect stemmed from a difference only at C4 ($F(1/19) = 8.6$, $p = .009$, in contrast at C3 $F(1/19) = 0.6$, $p = .44$), which was also reflected by a significant Knowledge Transformation × Hemisphere interaction ($F(1/19) = 4.4$, $p = .05$). An additional control analysis revealed a nonsignificant effect of knowledge transformation on EMG activity during SWS ($F(1/19) = 1.9$, $p = .2$).

Overall Differences in EEG between Early versus Late Sleep

Sleep data of the early- and late-night groups are presented in Table 1. As a final control for possible differences of the frequency profile between early and late sleep within each sleep stage, power values of the five frequency bands of sleep EEG were entered to a common ANOVA, separately for each sleep stage (S2, SWS, REM) with the between-subject factor sleep group (early-night group, $n = 26$, vs. late-night group, $n = 20$). No differences in band-specific EEG power were found between the sleep groups for SWS, S2 or REM sleep ($p > .1$ for each analysis both for the main effect of Sleep Group and for interactive effects of the Sleep Group with repeated measures factors band and hemisphere) indicating that spectral EEG characteristics of these sleep stages did not differ between first and second half of the night.

In each sleep stage, EEG power was overall higher at the left than the right electrode ($F(1/44) = 6.9$–9.1, $.01 \geq p \geq .004$ for the main effect of Hemisphere). Only for SWS was there a significant Sleep Group × Hemisphere × Band interaction ($F(4/174) = 3.0$, Greenhouse–Geisser corrected $p = .04$): Whereas the left versus right prevalence persisted for delta and theta (hemisphere, $F(1/44) > 4.2$, $p < .05$) without differences between groups (Sleep Group × Hemisphere, $F(1/44) < 1.8$, $p > .2$) laterality differences disappeared in the late-night group for the alpha, sigma, and beta bands during SWS, indicated by significant Sleep Group × Hemisphere interactions, $F(1/44) > 4.8$, $p < .05$ in separate analyses for each of these three bands.

DISCUSSION

The present study explored whether and how acquisition, transformation, and generation of knowledge is reflected in neurophysiological parameters during sleep occurring between two sessions of the task. This was done by assessing spectral sleep EEG characteristics of different sleep stages. Major results demonstrated that specifically the EEG characteristics of SWS were predictive for ExK generation and transformation after sleep.

Most intriguingly, specific patterns of spectral power during SWS were associated with the emergence of ExK after sleep on the basis of ImK acquired before sleep: A peak at ~10 Hz was only present in the SWS spectra of "transformers," that is, postsleep solvers who had already accumulated ImK about the hidden structure of the NRT before sleep. No such spectral peak was observed for either those participants who had gained ImK before sleep but did not become solvers after sleep, or those ones who became solvers after sleep without having gained ImK before sleep. The transformers' higher power in the 8–12 Hz band was also accompanied by a higher 8–12 Hz spindle density. Thus, spectral ~10 Hz activity during SWS was extracted here as a neurophysiologic sleep signature of implicit-to-explicit abstract knowledge transformation.

The relevant frequency range of 8–12 Hz of sleep EEG is lower than the frequency ranges reported traditionally for slow (~12 Hz, or <13 Hz) and fast (~14 Hz, or >13 Hz) sleep spindles (De Gennaro & Ferrara, 2003; Anderer

et al., 2001; Zeitlhofer et al., 1997; Gibbs & Gibbs, 1950). Also, sleep spindles were not analyzed here with respect to spindle power. One major criterion for distinguishing between slow spindle activity and alpha rhythm related to microarousals during sleep is topography. Slow spindles have a frontal focus (Anderer et al., 2001), whereas alpha has a parieto-occipital focus (Cantero, Atienza, & Salas, 2002; Pivik & Harman, 1995). We had only the C3 and C4 recordings available for analysis. Hence, basing on topographic criteria, we cannot fully exclude the possibility that the transformers' 10-Hz peak reflects alpha activity because of microarousal states. But we consider this possibility rather implausible. First, the effect was restricted to the SWS stage. It appears rather improbable that microarousals occur during the deep Sleep Stage SWS but not during the lighter S2. On the contrary, this distinction between sleep stages provides support for the assumption that this activity is because of slow spindles because it is the SWS stage where slow spindles typically occur (Marshall et al., 2006; Mölle et al., 2004). Second, EMG activity during SWS was not higher in transformers than nontransformers, which would be expected in case of more microarousals (ASDA, 1992). Finally, ~10-Hz spindle density, computed according to established criteria for spindle identification, was higher in transformers than nontransformers suggesting an association of alpha power increase with slow spindle activity during SWS.

It may be suggested that the ~10-Hz oscillations in transformers reflect individual traits that may determine stable interindividual differences in sleep spindle-like characteristics (Schabus et al., 2006; De Gennaro & Ferrara, 2003). To test for this possibility, sleep EEG should have been recorded in a control night before any task performance. Unfortunately, no EEG was recorded during the adaptation night. On the other hand, no differences existed between transformation groups for the 8–12 Hz sleep EEG of Sleep Stage 2 and REM sleep, nor for wake EEG during NRT performance. Thus, the generation of ~10 Hz oscillatory patterns only during SWS appears as a dynamic spindle-related functional correlate of knowledge transformation.

The functional significance of 8–12 Hz activity during SWS has remained elusive until recently, when multichannel recordings during sleep have become more common, although the ~10-Hz feature of non-REM sleep has been described earlier (for review, see Pivik & Harman, 1995) either as a neuropathological "alpha–delta sleep" (Hauri & Hawkins, 1973) or as "alpha intrusion" by hyperarousable states during sleep (for review, see Salih et al., 2009). A re-examination of findings has revealed the physiological nature of this activity during SWS, emphasizing its distribution at frontal and central leads (Duckrow & Zaveri, 2005), in contrast to the faster spindles (12–16 Hz) that have a centro-parietal focus (e.g., supporting material in Mölle et al., 2004) and mainly occur during S2 sleep. Recently, Marshall et al. (2006) have demonstrated that the increase of SWS by slow oscillating direct current stimulation (0.75 Hz) specifically enhanced the spectral power of slow spindle (8–12 Hz) sleep EEG activity during SWS, which was also accompanied by an improvement in declarative memory performance after sleep. This result is in line with the present findings by providing strong evidence for the functional role of 8–12 Hz SWS oscillations for declarative (explicit) memory consolidation. Spectral 8–12 Hz activity of sleep EEG also has been regarded in the context of the use- or experience-dependent theory of sleep function stating that homeostatic responses during sleep can be determined by the specificity and duration of sensory overloading and learning in the preceding waking period (Cantero, Atienza, Salas, & Dominguez-Marin, 2002; Sejnowski & Destexhe, 2000; Borbely, 1982). Accordingly, a spectral 9.5–12.5 Hz component of non-REM sleep EEG of humans has been associated with processing in the motor system during sleep as it subserved the functional connectivity between the frontal–central cortex and BG (Salih et al., 2009). Consistent with the use-dependent theory, the current sleep EEG results indicated a left over right dominance of spectral power from delta, theta, and faster frequency bands, corresponding to a presleep functional involvement of the right hand during NRT learning (Huber et al., 2000; Kattler, Dijk, & Borbély, 1994). However, the effect of knowledge transformation on both the ~10-Hz activity and ~10-Hz spindle density observed in the present study was more prominent at the ipsilateral (C4) than contralateral (C3) electrode, which does not allow to attribute this effect to a prevalent engagement of local sensorimotor networks before sleep (Cajochen, Di Biase, & Imai, 2008). Moreover, Cantero, Atienza, Salas, et al. (2002) have found an increase of 8–12 Hz spectral power of SWS after an extensive exposure to environmental auditory stimulation. Yet, this increase could not be directly associated with use-dependent overarousal of local auditory networks. Rather, it reflected a global experience-based plasticity involving synaptic reorganization after novel (hippocampus-mediated) sensory experience (Cantero, Atienza, Salas, et al., 2002).

Given the prerequisite of ImK acquirement before sleep and related presleep hippocampal activation during implicit NRT learning (Rose et al., 2002, 2005) for oscillatory rhythmic ~10 Hz patterns to be generated in the transformers' sleep EEG, it may be speculated that this spectral feature in humans reflects readout of hippocampally stored information to the neocortex during sleep (Rasch et al., 2007), analogously to the neocortical part of the compound of hippocampal ripples and cortical spindles observed in recordings from animals (Mölle & Born, 2009; Mölle, Eschenko, Gais, Sara, & Born, 2009; Wierzynski et al., 2009; Mölle, Yeshenko, Marshall, Sara, & Born, 2006; Siapas & Wilson, 1998). These enhanced ~10-Hz cortical oscillations might therefore not only reflect the successful acquisition of ImK about relationships between predictive and predictable responses but also an additional neural reorganization of these hippocampus-dependent memories during SWS, which might have increased the probability of interactions with explicit processing systems.

Furthermore, it is worth noting that the effect was larger at the right than at the left site. The creative holistic insight mode of processing has been consistently demonstrated to depend on the functional predominance of right hemisphere activity (Yordanova et al., 2010; Kounios et al., 2008). Thus, we may assume that the present larger right-side effect reflects such right hemisphere engagement that might promote insight after sleep.

A second main finding of our study was that all participants who discovered the hidden structure of the NRT after sleep, no matter whether they had acquired ImK before sleep or not, manifested a significant increase in spectral beta activity during SWS. EMG analyses excluded the possibility of artificial beta increase because of contamination from myographic activity. Also, beta activity generated during NRT performance before or after sleep did not differentiate solvers from nonsolvers, indicating that beta increase during sleep did not reflect individual or sample differences in this frequency range that could have affected both the task-related and sleep EEG. Although this result was not expected, there is actually some evidence that enhanced high-frequency spectral EEG activity may subserve a mechanism associated with ExK generation. In terms of network activation, beta power increase during wake reflects enhanced local synchronization of functionally specified locally distributed networks (Pfurtscheller & Lopes da Silva, 1999) and synchronization of a widely distributed long-distance beta network was reported to be a marker of conscious access to the content of verbal material (Gaillard et al., 2009). Extending previous observations of increased coherence among different cortical regions by slow oscillations (Mölle et al., 2004), the current results of sleep-dependent beta power increase in solvers may therefore point to a stronger synchrony of local networks as a functional precursor of subsequent insight, which is potentiated specifically by SWS. Activation of neural beta processors during sleep may thus facilitate synchrony within a distributed beta network during subsequent wakefulness, supporting the conscious access to hidden NRT information in solvers. Another possible explanation is that spectral beta activity of sleep EEG was elevated in solvers relative to nonsolvers already before the occurrence of SWS. As far as some (though nonsignificant) beta elevation was detected for Sleep Stage 2 of solvers, an active information processing engaging neural beta networks might be a continuous mechanism activated after presleep learning in solvers (Perlis et al., 2004) rather than a mechanism specifically linked to SWS. However, no differences existed between solvers and nonsolvers in spectral beta power during light sleep (S1) of the first sleep cycle nor during active NRT processing before sleep, which emphasizes the specific associations of SWS beta activity with ExK generation after sleep.

In contrast to 8–12 Hz activity, 13–16 Hz (sigma) EEG activity increased during sleep in association with implicit learning before sleep. No such correlations with presleep implicit learning existed for the sigma power during NRT performance before or after sleep pointing to a specific sleep-related effect. Although EEG phenomena from the sigma frequency range (13–16 Hz) are analyzed typically during Sleep Stage 2 in relation with fast spindles (e.g., De Gennaro & Ferrara, 2003; Gais et al., 2002), it is demonstrated here that sigma power from each sleep stage (including REM sleep) correlated with implicit learning before sleep. Yet, this was only found for early-night sleep. One possible source of this early-night effect may come from the dominance of SWS, during which spindle activity is specifically reorganized in terms of grouping and synchronization by slow oscillations (Marshall et al., 2006; Mölle et al., 2002, 2004; Gais et al., 2002). However, the neurofunctional associations of preexisting implicit representations with sigma EEG power even in REM sleep and related synchronized fast spindle/sigma oscillations during sleep remains to be clarified.

In conclusion, the present study provides original evidence for the association between neuroelectric signals generated during SWS and the gain of ExK after sleep. EEG power in the alpha band (8–12 Hz) during SWS emerged as a specific marker of the transformation of presleep ImK to postsleep ExK, possibly reflecting increased slow spindle activity, whereas beta power increase during SWS was a marker of the gain of ExK after sleep, irrespective of presleep knowledge. Because no such EEG correlates of later insight were found during S2 and REM sleep, the results suggest that it is SWS and its associated neuronal memory reprocessing that lay the foundations for restructuring of task-related representations, eventually promoting the gain of ExK. It remains to be determined whether the EEG characteristics in the alpha and beta bands during SWS that correlated with performance differences in knowledge transition and generation after sleep, are indeed causal for these processes to occur after sleep.

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