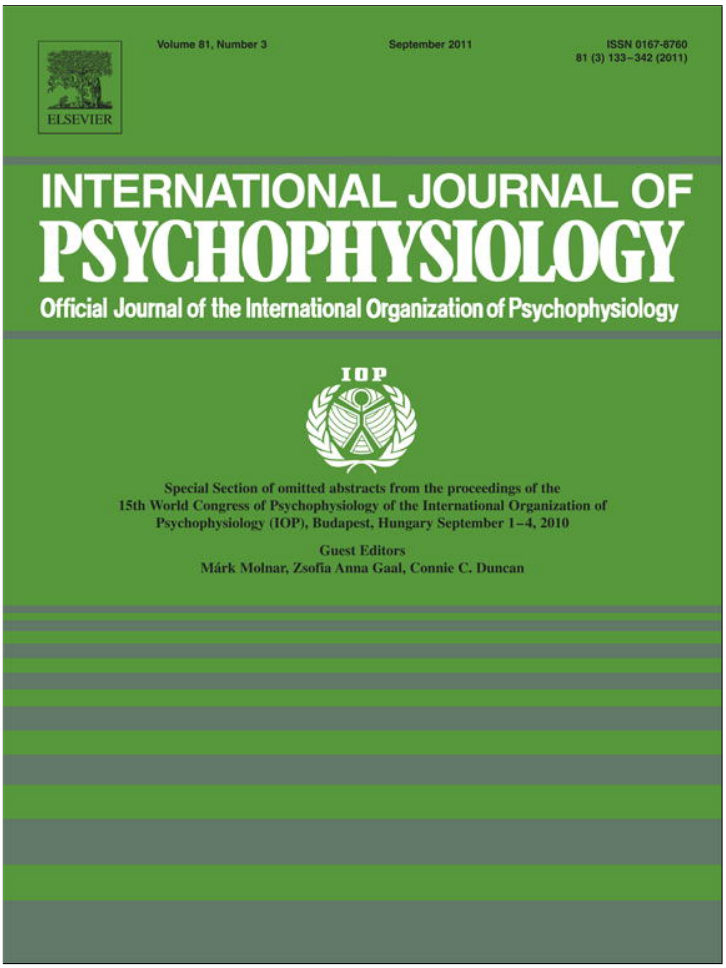

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Effects of sleep deprivation on auditory change detection: a N1-Mismatch Negativity study

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ABSTRACT

The effects of sleep deprivation on neural activity underlying stimulus change detection are still debated. The aim of this study was to investigate the effects of sleep deprivation on the relationship between N1 refractoriness and Mismatch Negativity (MMN) as indexes of different stages of change detection. Respectively, N1 represents the sensory feature trace creation with stimulus repetition and MMN represents the memory-based detection of deviance in a new incoming stimulus. Event-related potentials (ERPs) were recorded from 22 healthy participants during a passive auditory oddball task after a night of normal sleep and after a night of total sleep deprivation (TSD). Importantly, stimulus presentation was organized as a train of 10 stimuli, so that N1 refractoriness could be measured as amplitude decrease with stimulus repetition within each train. Results showed that N1 refractoriness and MMN were not affected by TSD suggesting that the change detection process was preserved in our paradigm. However, the overall N1 amplitude increased after TSD, an effect that may be related to an enhancement of cortical excitability.

1. Introduction

Sleep deprivation is associated with many sleep disruptive disorders and, more generally, with poor sleep hygiene. It has been associated with a decrease in performance involving several cognitive functions (Pilcher and Huffcutt, 1996; Banks and Dinges, 2007), such as attention (Drummond et al., 2001) and memory (Harrison and Horne, 2000).

Change detection processes are believed to be pre-attentive in nature, and therefore can be performed even when attention is reduced (Näätänen, 1990; Sussman, 2007). Moreover, they may be responsible for involuntary shifts of attention toward potentially dangerous stimuli (Näätänen, 1990; Schröger, 1996). In this context, the effects of sleep deprivation on change detection have captured the interest of many researchers. However, changes in the neural activity associated with stimulus processing and change detection after sleep deprivation are still debated.

Stimulus change detection is usually studied by means of the auditory oddball task. In this paradigm, a "standard" stimulus is repeatedly presented in a sequence and is occasionally replaced by a "deviant" stimulus differing from the standard stimulus along one physical feature (e.g. frequency, duration, etc.). According to the model developed by Näätänen and coworkers (Näätänen, 1990; Näätänen and Winkler, 1999; Näätänen et al., 2005), during the repetition of the standard tone, a representation of the standard stimulus feature is created. When a deviant stimulus is presented, the deviant stimulus features are compared with the representation of the previously presented standard stimuli, leading to the detection of change.

This process is reflected in two components of electroencephalographic event-related potentials (ERPs): the N1 generated by the repeatedly presented standard stimulus and by the deviant stimulus, and the Mismatch Negativity (MMN) generated by the deviant stimulus presentation.

N1 is a negative wave peaking at about 50–150 ms after sound presentation with peak amplitude at the vertex and polarity inversion over inferior lateral regions (Peronnet et al., 1974). When an auditory stimulus is repeatedly presented with a short inter-stimulus interval (ISI) of a few seconds, N1 amplitude decreases at every repetition (Davis et al., 1966). The shorter the ISI, the stronger the decrease becomes (Mäntysalo and Näätänen, 1987; Ritter et al., 1968). This effect, called refractoriness, reflects a decrease of stimulus-specific responsiveness of N1 generators (Näätänen, 1988) and is thought to index the formation of the sensory feature trace of the standard stimulus (Näätänen and Picton, 1987).

MMN is a negative deflection elicited 100–250 ms after stimulus onset, in response to a change in one or more attributes of repetitive auditory stimulation (Näätänen and Winkler, 1999). MMN is usually calculated by subtracting the ERPs elicited by the standard stimulus from the ERPs elicited by the deviant stimulus, although it has been shown that this difference wave is a compound of a "genuine" MMN component and a N1 increase to the deviant sound (Jacobsen and

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Schröger, 2001; Jacobsen et al, 2003). MMN is believed to provide a physiological measure of memory-based stimulus discrimination. Along this line, MMN may be generated by a conflict of the current deviant sensory input with the active memory representation of previous stimulation (Watson et al., 2007) possibly through predictive models encoding stimulus regularity (Winkler, 2007). Therefore, the MMN can be elicited as long as the memory representation of standard stimulation is available for the matching process (Näätänen, 1992; Korzyukov et al., 1999; Schröger, 2007). Accordingly, several studies have shown that MMN decreases in amplitude with longer ISI and is generated for intervals up to 10–20 s (Mäntysalo and Näätänen, 1987; Sams et al., 1993; Sabri and Campbell, 2001).

Previous studies investigating the effects of sleep deprivation on change detection have found inconsistent results (Dikaya et al., 1992; Raz et al., 2001; Lee et al., 2004). Several studies have shown that MMN is unaffected by sleep quality in healthy subjects and patients (Salmi et al., 2005; Gosselin et al., 2006; Naumann et al., 2001), during wake–sleep transition (Ruby, et al., 2008; Winter et al., 1995), and after one night of sleep deprivation (Dikaya et al., 1992). On the other hand, a decrement in the MMN amplitude has been reported after short total sleep deprivation (Raz et al., 2001; Sallinen and Lyytinen, 1997) and during wake–sleep transition (Nashida et al., 2000; Sabri et al., 2000; Nittono et al., 2001; Sabri et al., 2003).

Whereas most studies have focused only on the MMN, some have also investigated the effects of sleep deprivation on the N1 amplitude. Raz et al. (2001), for example, showed that the N1 amplitude increases after one night of sleep deprivation, suggesting an association between a decrease in N1 refractoriness and MMN amplitude decrease. In fact, a lack of decrease in N1 amplitude over repeated stimulations would result in increased N1amlitude averaged across all standards. Although plausible as a mechanism, this issue has never been approached in a thorough way. Moreover, at odds with the results of Raz and colleagues, a later study reported no changes in N1 amplitude after sleep deprivation in an oddball paradigm (Lee et al., 2004).

The aim of the present study was to investigate the effects of total sleep deprivation on change detection *separately* on the N1 elicited by repeated standard stimulation and on the MMN generated by the deviant stimulus presentation. In particular, we investigated the creation of a sensory feature trace with standard repetition and the fading of the stimulus discrimination process with increasing ISIs. We developed a new auditory oddball paradigm based on the study of Cowan et al. (1993), in which stimuli were presented in trains with specific ISIs. We measured the N1 refractoriness with the repetition of the standard tone as an index of the sensory features trace creation, and the modulation of MMN amplitude with different ISI as an index of change detection.

2. Methods

2.1. Participants

Twenty-two undergraduate students (11 males, aged 20 to 29) participated in this experiment. Data from two participants was subsequently rejected due to excessive artefacts in the EEG signal. All participants reported no medical or psychiatric disease, or ongoing pharmacological treatment. All participants gave written consent, and were told that they were free to leave the experiment at any time. Moreover, to increase compliance to the experimental schedule, they received 75 euros once they completed both the experimental sessions. The study was approved by the Ethics Committee of Psychology Research of the University of Padova.

2.2. Task

The task was a passive auditory oddball paradigm during which participants read a book without paying attention to the sounds. Participants were monitored through a video camera and were engaged in conversation about the book at the end of the experiment, to make sure that they were compliant with the experiment requirements.

Acoustic stimuli were pure tones of 50 ms duration and 5 ms rise/fall time, generated by Cool Edit Pro 1.2 (Syntrillium Software Corporation) and delivered binaurally through an electrically shielded headphone (Sony DJ MDR-V150). The automated delivery of stimuli was controlled by a PC provided with E-Prime software (Psychology Software Tools, Inc.).

Auditory stimuli were delivered in trains of ten tones (9 standard and 1 deviant) separated by 10 s inter-sequence intervals. Each train was uniquely characterized by its own specific standard/deviant pair, ISI and position of the deviant stimulus within the sequence. Standard and deviant stimuli were different in frequency. Five of the nine different pairs of standard and deviant stimuli used by Cowan et al (1993) were chosen: 420–490 Hz, 510–595 Hz, 600–700 Hz, 690–805 Hz, 780–910 Hz. The deviant/standard frequency ratio was 7:6 in half of the trains (high pitch deviant and low pitch standard), and 6:7 in the other half (low pitch deviant and high pitch standard). In each train the ISI was 0.5 s, 1 s or 2 s. The deviant stimulus could occur in position 6, 8 or 10 of the sequence (see Fig. 1 for a schematic representation of trains of stimuli at different ISIs in which the deviant stimulus occurred in different positions). In order to avoid long-term memory effects, the trains had a quasi-random distribution in which two consecutive trains could not have the same frequency values.

We chose to present several pairs of standard/deviant stimuli so that the creation of the standard sensory trace took place independently for each stimulus train and without involving long term consolidation of the standard representation from previous presentation. Moreover, consecutive trains never had the same ISI in order to minimize the effect of temporal probability of the deviant presentation (Sabri and Campbell, 2001). 1800 stimuli in 180 trains (1620 standards and 180 deviants) were delivered for each recording session.

2.3. Procedure

The experiment consisted of four sessions. The first two sessions were recorded in the morning and in the evening after a normal night of sleep (control condition). Two weeks later, the same two sessions were recorded after a night of total sleep deprivation (sleep deprivation condition). Half of participants performed the control condition first, while the other half performed the sleep deprivation condition first. Participants completed a sleep diary during the week prior to the first experimental condition and during 2 weeks between the first and the second experimental condition in order to exclude the presence of sleep disorders or poor sleep hygiene (i.e., highly variable sleep schedules, sleep restriction, etc.).

Participants spent the night of sleep deprivation in the laboratory with a researcher monitoring their activity. They could watch movies,

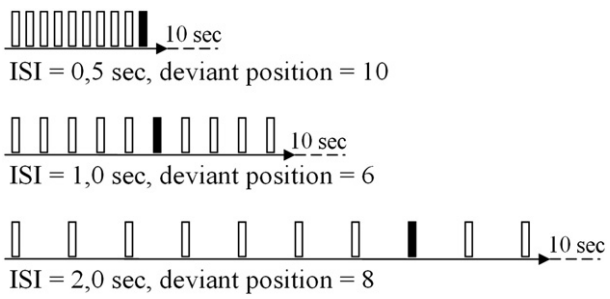

Fig. 1. Oddball paradigm. Schematic representation of short trains of stimuli. Each train included ten stimuli (rectangles), nine standard (white) and one deviant (black). The first five stimuli of each train were always standard stimuli. The deviant stimulus could appear in position 6, 8 or 10. Stimuli within trains were separated by one out of three different ISI (0.5, 1 or 2 s). Trains of stimuli were separated by an interval of 10 s.

listen to music, play video games, card and board games and interact with the researcher. They were not allowed to engage in heavy physical activity, assume caffeine and other stimulants or leave the laboratory area. On the day of electrophysiological recordings, participants could carry out their normal activities between sessions but they were asked to come back to the laboratory to sign a register every 3 hours in order to make sure they were compliant to experimental demands.

At the beginning of each session, right before the electrophysiological recordings started, participants rated their perceived drowsiness by means of the Stanford Sleepiness Scale (Hoddes et al., 1973).

2.4. Data recording

Electroencephalographic (EEG) signals were recorded from five electrodes (Grass Gold 10 mm) placed according to the 10–20 system (Jasper, 1958) over Fz, Cz, Pz, right and left mastoids, and referred to the nose. Vertical and horizontal Electrooculographic (EOG) signals were recorded by electrodes placed 1 cm above and below the left eye and 1 cm out of eyes' canthi, in order to offline correct for ocular movement artefacts.

Signals were online filtered and amplified by a Grass Amplifier (MODEL 15RXi). The bandpass filter used for EEG recording was 0.1–30 Hz with a gain factor of 20000 (10000 for EOG). Signals were digitally sampled at 1000 Hz by Labmaster LAB RACK BNC Termination Panel 12 bit A/D board (resolution 6.1×10^{-2} μV) and recorded with VPM software. EEG and EOG signals were acquired in epochs beginning 100 ms before stimulus onset and lasting 450 ms.

2.5. Data analysis

The EEG data were analysed using Scan 4.3 software (Neuroscan Inc.). EEG was offline corrected for blinks based on vertical EOG signal. Epochs exceeding a voltage threshold of ± 70 μV in any EEG and EOG channels (3.67% in the control condition and 3.95% in the sleep deprivation condition) were rejected from analyses. Then epochs were baseline corrected and separately averaged for each condition.

The first 5 standards of stimulus trains were analysed to measure N1 amplitude. Averages were computed for each recording session for standard stimuli in position 1, 2, 3, 4 and 5 of the stimulus trains and for each different ISI separately. As N1 amplitude was maximal over Cz, we measured the negative peak values over this electrode in a time window between 75 and 150 ms after stimulus onset. A 4-way repeated measures ANOVA was performed to investigate N1: Sleep (control, deprivation) × Circadian (morning, evening) × Position (1, 2, 3, 4, 5) × ISI (0.5, 1, 2).

MMN was computed as the waveform originating from the sample by sample difference between the fifth standard stimuli in a stimulus train and the deviant tone of the same stimulus train. As previously explained this method has been widely used to measure the MMN although it is influenced by the N1 elicited by the deviant stimulus. The grand-average across all conditions showed that the MMN peaked over Cz and inverted polarity over mastoids. To perform statistical analysis, the signal was re-referenced to linked mastoids (Kujala et al., 2007) and MMN was measured as mean amplitude over 30 ms centred around the grand-average peak latencies in each condition. A 3-way repeated measures ANOVA was performed to investigate MMN component: Sleep (control, deprivation) × Circadian (morning, evening) × ISI (0.5, 1, 2 s).

A 2-way repeated measure ANOVA on Stanford Sleepiness Scale ratings was performed: Sleep (control, deprivation) × Circadian (morning, evening). Due to a technical issues, data from 19 participants was considered.

All statistical analyses were performed using Statistica 6 (StatSoft Inc.) software. Where appropriate the Greenhouse–Geisser correction was used. Post-hoc comparisons were calculated using Tukey's HSD correction for multiple comparisons.

3. Results

3.1. N1

We found many effects related to N1 that have previously been shown in other studies.

N1 amplitude decreased with the repetition of the standard stimulus within trains (Position main effect: $F_{4,\ 76}=80.73$, $\varepsilon=0.34$, $p<0.01$). Post-hoc comparisons showed a significantly higher N1 amplitude for the first position stimulus compared to the other four positions ($p<0.01$). N1 for the second position stimulus was also significantly higher than N1 for the fourth and the fifth positions ($p<0.01$).

N1 reduction with stimulus repetition was stronger when stimuli were presented closer in time. First, N1 was higher with longer ISIs (ISI main effect: $F_{2,\ 38}=49.85$, $p<0.01$). Post-hoc comparisons revealed a higher amplitude N1 for the 2.0 s ISI condition ($p<0.01$) compared with 0.5 and 1.0 s conditions. Moreover, this effect was modulated by the position of the standard stimulus within the train (Position × ISI interaction: $F_{8,\ 152}=8.58$, $p<0.01$). N1 amplitude was not different for the first stimulus of the train across ISI conditions; for all the other positions of the stimulus (between 2 and 5), N1 amplitude for the 2.0 s ISI condition was significantly higher compared with the 1.0 and 0.5 s ISI conditions ($p<0.01$).

Sleep deprivation modulated the generation of N1. Indeed, N1 amplitude increased after sleep deprivation as shown by the significant main effect of sleep ($F_{1,\ 19}=8.82$, $p<0.01$). The Sleep × Position effect was not significant, revealing a similar trend in N1 reduction with stimulus repetition between the sleep deprivation and the control condition (Fig. 2).

No other effects were found to be significant.

3.2. MMN

MMN recorded in the sleep deprivation condition and in the control condition are displayed in Fig. 3. No significant sleep effect was found either as a main effect or interacting with other variables. There was a tendency ($F_{2,38}=2.86$, $p=0.07$) for MMN amplitude to be influenced by ISI. No other effects were found significant.

3.3. Stanford Sleepiness Scale

Perceived drowsiness increased after sleep deprivation as shown by the significant main effect sleep ($F_{1,19}=51.94$, $p<0.001$).

4. Discussion

We found that N1 amplitude elicited by acoustic standard stimuli was increased by one night of total sleep deprivation and that this effect was independent from the N1 reduction with the repetition of the stimulus, i.e. N1 refractoriness. Moreover, we found no modification of MMN amplitude after sleep deprivation. Therefore, our results indicate that sleep deprivation modulates N1 amplitude independently from change detection processes as indexed by N1 refractoriness and MMN.

A general increase of the N1 amplitude during sleep deprivation is consistent with previous findings (Raz et al., 2001). Here we tested whether the increment on averaged N1 may be caused by the shortening of the refractory period of auditory cortex (Näätänen and Picton, 1987). We showed that the decrease of N1 amplitude associated with the repetition of standard stimuli was similar in both sleep deprivation and control conditions. Moreover, the ISI effect found for the standard stimuli (higher amplitude for longer intervals) was not affected by sleep loss. Therefore, our data demonstrate a possible dissociation between refractoriness modification and increases in N1 amplitude following sleep deprivation.

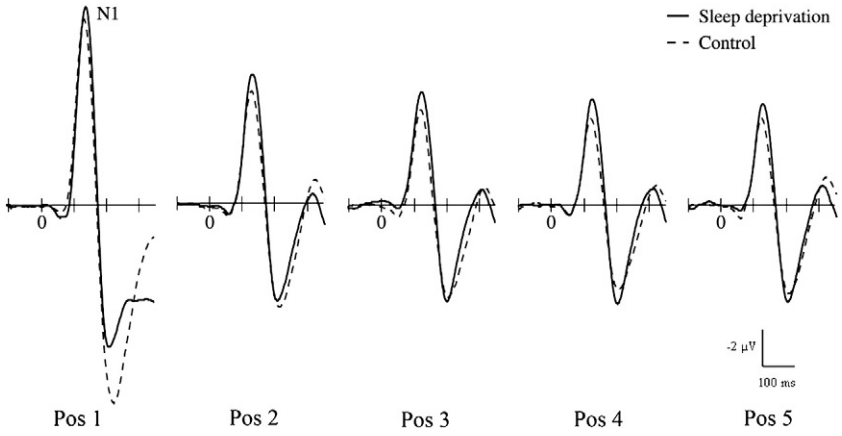

Fig. 2. N1 for standard stimuli. ERP waveforms recorded over Cz for standard stimuli in the first five positions of stimuli trains: Pos 1, Pos 2, Pos 3, Pos 4, Pos 5. Solid lines represent the sleep deprivation condition and dashed lines represent the control condition.

Even though sleep deprivation is also associated with drowsiness and lower arousal, it is unlikely that these variables induced an increase in N1 amplitude. Indeed, previous studies have shown that sleepiness (Ferrara et al., 1995) and low arousal (Näätänen and Picton, 1987) *reduce* rather than increase N1 amplitude.

A possible explanation for the increment of N1 after sleep deprivation could be a enhancement of cortical responses to acoustic stimuli. It is known that sleep deprivation can induce an increase in cortical excitability, probably related to modulations in the balance between inhibition and excitation at a cortical level (Scalise et al., 2006; Civardi et al, 2001; De Gennaro et al, 2007). Accordingly, a recent study has shown that the firing rate of cortical neurons increases with longer periods of wakefulness and decreases after being asleep (Vyazovskiy et al, 2009). Therefore, it is possible that sleep deprivation may induce an increase in excitability of N1 cortical generators which respond more strongly to auditory stimulation.

Alternatively, the increment of N1 after sleep deprivation may be due to a form of attentional "leakage" as suggested by Raz et al. (2001). The failure to sustain attention to a primary attended task after sleep deprivation, e.g. reading a book, would induce a shift of attention to a secondary unattended task, e.g. the auditory oddball task. Unfortunately, despite we controlled participants' compliance to task requirements, we did not systematically take a measure of participants' reading ability after sleep deprivation and after one night sleep. Therefore, we cannot draw specific conclusions regarding the role of attentional leakage on N1 increments following sleep deprivation. Although some studies have found an MMN amplitude reduction following sleep deprivation, our data suggest that change detection may be unaffected after one night of total sleep deprivation. For example, we found no difference in MMN amplitude between sleep deprivation and the control condition. Moreover, the sleep deprivation condition did not induce a decrease of MMN amplitude with longer ISIs.

It should be noted that the MMN measured in this study is very likely to be a combination of a "genuine" MMN component and a N1 increase to the deviant sound (Jacobsen and Schröger, 2001; Jacobsen et al, 2003). The neural populations engaged by the deviant sound were probably less refractory than those engaged by the presentation of the fifth standard sound of the stimulus train.

The lack of sleep deprivation effects on MMN has been found in previous studies (Ruby, et al., 2008; Salmi et al., 2005; Gosselin et al., 2006; Naumann et al., 2001; Winter et al., 1995), but is not confirmed by other investigations (Raz et al., 2001; Sallinen and Lyytinen, 1997; Nashida et al., 2000; Sabri et al., 2000, 2003; Nittono et al., 2001). It is possible that inconsistent results across studies depend on the nature of the stimulation. Separate cortical sources have been individuated when the MMN is generated for different kinds of deviance, such as frequency, intensity and duration (Giard et al, 1995). Moreover, different effects have been found depending on the nature of variation in the oddball task. For example, Näätänen et al (1993) found that directing attention to a distracting task can reduce MMN for intensity deviations but not for frequency deviations. Therefore, different systems may be involved in MMN generation and they may be selectively affected by the state of the subject.

Another relevant variable may be the nature of the oddball paradigm and whether it involves long-term memory consolidation of the standard stimuli. In the classical oddball paradigm, in which the same standard stimuli is repeated during the task, it is possible to obtain a strong representation of the standard stimulus reinforced by long term memory consolidation processes (Cowan et al., 1993). Different studies (Ritter et al., 1998; Winkler et al., 1996; Winkler and Czigler, 1998) support the idea that the repetition of the standard stimulus can create a memory trace that represents not only the features of the stimulus but also the regularities of the stimulation sequence (e.g., the repetition of a tone, the alternation of two tones, some periodicity of the sound sequence, etc.). Unlike previous sleep

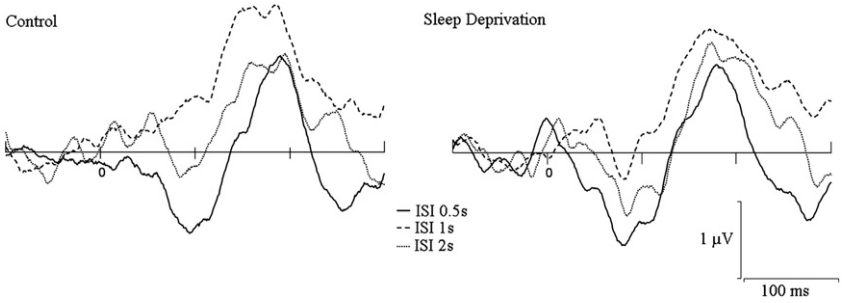

Fig. 3. Mismatch negativity. Differential waveforms resulting from the subtraction between deviant and standard stimuli over Cz for each ISI separately. Control condition is shown on the left and sleep deprivation condition is shown on the right.

deprivation studies, we used different trains and therefore we reduced the potential longer-term memory confound. In fact, the neuronal representation of the frequent standard stimulus and the timing regularity related to the inter-stimulus interval were created separately in each 10-stimuli sequence, as they could not be inferred from the previous trials. Based on these observations we can speculate that sleep deprivation may affect longer-term processes associated with memory trace formation. Further studies are needed to investigate this possibility.

In our study we have revealed the modulation of N1 by sleep deprivation, independently of stimulus repetition and change detection. We have also shown that under particular paradigm and stimulation conditions, sleep loss might not affect change detection. Our results open new questions on the effects of sleep deprivation on the neural processing underlying sensory processing of auditory stimuli. Our study shows how different methodological choices allow the investigation of different functional mechanisms involved in the formation of an auditory memory trace and matching processes.

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