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RESEARCH ARTICLE

Effects of stimulus intensity and auditory white noise on human somatosensory cognitive processing: a study using event-related potentials

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Abstract
Exposure to auditory white noise has been shown to facilitate cognitive function. This phenomenon is often called stochastic resonance, and a moderate amount of auditory noise has been suggested to benefit individuals in hypodopaminergic states. Previous studies using psychophysic methods reported that stochastic resonance was sensitive to stimulus intensity; however, the relationship between neural activities elicited by different stimulus intensities and auditory white noise has not yet been clarified Thus, the present study aimed to investigate the effects of stimulus intensity (Experiment 1) and auditory white noise (Experiment 2) on behavioral data (reaction time (RT), the standard deviation of RT, and error rates), and the N140 and P300 components of event-related potentials (ERPs) in somatosensory Go/No-go paradigms. The subjects had to respond to the somatosensory stimuli by pressing a button with their right thumb only after presentation of the Go stimulus. In Experiment 1 with four different stimulus intensity levels, the peak latencies of N140 and P300 became shorter, and the peak amplitudes of N140 and P300 were enhanced with increases in stimulus intensity. In Experiment 2 with weak and mild intensities under auditory white noise and control conditions, the amplitudes of Go-P300 and No-go-P300 were enhanced by white noise, irrespective of weak and mild intensities, during Go/No-go paradigms. Auditory white noise did not significantly affect the amplitude of N140 or the latencies of N140 and P300. These results suggest the presence of a characteristic cross-modal stochastic resonance in neural substrates utilizing somatosensory ERPs.

Keywords ERP · Go/No-go · Stochastic resonance · P300

Introduction

Previous studies reported that exposure to auditory white noise facilitates cognitive processing, such as the shortening of reaction times (RT) during arithmetic tasks (Usher and Feingold 2000), and improvements in the detection of a weak visual signal (Manjarrez et al. 2007) and tactile, visual, auditory, and cross-modal perception (Manjarrez et al. 2007; Lugo et al. 2008). This phenomenon is frequently called 'cross-modal stochastic resonance'. Recent studies demonstrated that these noises are more critical for inattentive school children, including those with attention-deficit hyperactivity disorder (ADHD) (Söderlund et al. 2010). These findings led to the hypothesis that the neural mechanisms of stochastic resonance are related to dopaminergic neuromodulation originating from the substantia nigra/ventral tegmental area (SN/VTA) of the midbrain because ADHD is characterized by a dysfunctional and hypoactive dopamine system (Sikström and Söderlun 2007).

The present study used event-related potentials (ERPs) to clarify the effects of auditory white noise on the neural activities of motor execution and inhibition in Go/No-go paradigms. Two components, a negative deflection approximately 140–300 ms (N2) after stimulus onset and a positive deflection at approximately 300–600 ms (P3), elicited in No-go trials were larger than the ERPs recorded in Go trials (Falkenstein et al. 1999; Kamijo et al. 2004; Roche et al. 2005). In somatosensory Go/No-go paradigms, the amplitude of the No-go-N140 component was more negative

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than that of the Go-N140 component, and the amplitude of No-go-P300 was larger than that of Go-P300 (Nakata et al. 2004, 2005, 2015; Hatem et al. 2007; Shibasaki et al. 2017).

We previously investigated the effects of auditory white noise exposure on ERPs during somatosensory Go/No-go paradigms (Ohbayashi et al. 2017). We showed that the peak amplitudes of Go-P300 and No-go-P300 in ERP waveforms were significantly larger under 55 dB than 45 and 65 dB conditions, suggesting that exposure to auditory white noise at 55 dB changes the neural activation of motor execution and inhibition processing. We also set 2.0 times the sensory threshold as the stimulus intensity. Previous studies using psychophysic methods showed that stochastic resonance was sensitive to stimulus intensities across different modalities (Manjarrez et al. 2007; Lugo et al. 2008). Manjarrez and colleagues (2007) demonstrated that the detection of a weak visual signal was an inverted U-like function of the different intensity levels of auditory white noise, while this relationship was not observed when a strong visual signal was used. However, the relationship between ERP responses elicited by different stimulus intensities and a certain level of auditory white noise (stochastic resonance) remains unclear, whereas the relationship between ERP responses elicited by a certain stimulus intensity level and different levels of tactile white noise has been elucidated (Méndez-Balbuena et al. 2015). Therefore, the relationship between the ERP responses of motor execution and inhibition and different stimulus intensities need to be clarified. To the best of our knowledge, previous ERP studies using Go/No-go paradigms have not yet focused on this issue. In Experiment 1 of the present study, we set four conditions: 1.1, 1.5, 2.0, and 2.5 times the sensory threshold, to investigate the effects of stimulus intensity levels on somatosensory ERPs during Go/No-go paradigms. In Experiment 2, we examined the relationship between the ERP responses of motor execution and inhibition elicited by weak and mild stimulus intensities, and a certain level of auditory white noise. We hypothesized that the effects of auditory white noise on somatosensory ERPs were different between weak and mild stimulus intensities, even if cross-modal stochastic resonance was sensitive to stimulus intensities during cognitive Go/No-go paradigms.

Materials and methods

Participants

Sixteen normal subjects (ten females, six males, mean age 25.6 years, range 20–40 years) participated in Experiment 1, and fourteen normal female subjects (mean age 21.8 years, range 20–23 years) participated in Experiment 2. Four of the subjects who participated in Experiment 1 also participated in Experiment 2. None of the subjects had a history of neurological or psychiatric disorders. Informed consent was obtained from all subjects. The present study was approved by the Ethical Committee of Nara Women's University, Nara city, Japan. Experiments were conducted in accordance with the Declaration of Helsinki.

Experiment 1

Task and procedure

Go/No-go paradigms involve a continuously presented series of Go stimulus to which the subjects respond as rapidly as possible and No-go stimulus to which the subjects do not respond. Some studies sometimes used the frequency of Go stimuli ($\geq 75\%$) to create a prepotent tendency to respond that must then be inhibited for the No-go stimulus (Bokura et al. 2001; Huster et al. 2010). However, in this ERP study, we designed Go and No-go stimuli with the same probability to avoid the effects of stimulus probability and minimize differences in response conflict between event types (Braver et al. 2001; Nakata et al. 2015). To record somatosensory ERPs, the Go stimulus was delivered to the second digit of the left hand, and the No-go stimulus to the fifth digit of the left hand with ring electrodes. The electrical stimulus used was a current constant square wave pulse of 0.2 ms in duration. Subjects had to respond to the stimulus by pushing a button with their right thumb as quickly as possible only after presentation of the Go stimulus. Go and No-go stimuli were presented in a random order, with the interval of presentation being fixed at 2 s. RT was measured for the Go stimulus. Each session comprised 80 epochs of stimulation, which included 40 epochs for the Go stimulus and 40 for the No-go stimulus. Subjects kept their eyes open and focused on a small fixation point positioned in front of them at a distance of approximately 1 m throughout each task. The error rate, which included commission (i.e., error pushing with No-go stimulus) and omission (i.e., a slow response or no pushing with Go stimulus) errors, was calculated in each session. In a practice run, subjects were instructed to perform the Go/No-go paradigms for ten stimuli before recording.

We set four conditions with different stimulus intensities: 1.1, 1.5, 2.0, and 2.5 times the sensory threshold. We confirmed that participants did not feel any pain under any condition. Two runs were performed for each condition (i.e., eight runs in total). The order of conditions was randomized for each subject and counterbalanced across all subjects.

Experiment 2

Task and procedure

The setting for Go/No-go paradigms was the same as that in Experiment 1. We set two intensities and two conditions:

(1) a control condition with weak intensity, (2) an auditory white noise condition with weak intensity, (3) a control condition with mild intensity, and (4) an auditory white noise condition with mild intensity. Weak intensity was set for 1.1 times the sensory threshold, and mild intensity for 2.0 times the sensory threshold. A portable CD player including the sound source of white noise was set in front of participants at a distance of approximately 1 m. The noise level was set at 55 dB, which was measured around subjects' ears using a sound level meter, as described previously study (Ohbayashi et al. 2017). Prior to ERP recording, subjects were asked to listen to the white noise (i.e., auditory white noise condition with weak intensity, and white noise condition with mild intensity) being tested for 5 min to become familiar with the noise level. Go/No-go paradigms were then started. We set a 10-min break after each condition to avoid aftereffects. Two runs were performed for each condition (i.e., eight runs in total). The order of conditions was randomized for each subject and counterbalanced across all subjects.

Electroencephalography recording

Electroencephalography (EEG) was recorded with Ag/AgCl disk electrodes placed on the scalp at Fz, Cz, Pz, C3, and C4, according to the International 10–20 System. Each scalp electrode was referenced to linked earlobes. To reject eye movements or blinks exceeding 100 μV, an electrooculogram was recorded bipolarly with a pair of electrodes placed 2 cm lateral to the lateral canthus of the left eye and 2 cm above the upper edge of the left orbit and analyzed online. We also checked all raw data off-line, and if clear artifacts not exceeding 100 μV (ex. unexplained noise) were recorded, the trails were eliminated from averaging. Impedance was maintained at less than 5 kohm. All EEG signals were collected on a signal processor (Neuropack MEB-2200 system, Nihon-Kohden, Tokyo, Japan). The analysis epoch for ERPs was 600 ms, including a prestimulus baseline period of 60 ms. The bandpass filter was set at 0.1–50 Hz and the sampling rate was 1000 Hz. The peak amplitudes and latencies of N140 and P300 were measured at 110–230 and 240–500 ms, respectively. Amplitudes were measured baseline-to-peak. Slow responses exceeding 800 ms and incorrect responses were eliminated from averaging. As behavioral data, RT, the standard deviation (SD) of RT (i.e., response variability), and commission (i.e., error pushing with No-go stimulus) and omission errors (i.e., slow response or no pushing with Go stimulus) were evaluated for each condition.

Statistical analysis

In Experiment 1, behavioral data for RT, the SD of RT, and commission and omission error rates were separately submitted to analyses of variance (ANOVA) with repeated measures using Intensity as a factor (1.1, 1.5, 2.0, and 2.5 times). Data for the amplitudes and latencies of N140 and P300 in ERPs were separately submitted to a three-way repeated measures ANOVA with Intensity, Stimulus (Go, and No-go), and Electrode (Fz, Cz, Pz, C3, and C4) as factors. In Experiment 2, behavioral data for RT, the SD of RT, and commission and omission error rates were separately submitted to a repeated measures ANOVA using Condition (control and auditory white noise) and Intensity (weak and mild) as factors. Data for the amplitudes and latencies of N140 and P300 in ERPs were separately submitted to a four-way repeated measures ANOVA with Condition, Intensity, Stimulus, and Electrode as factors.

In all repeated measures factors with more than two levels, we tested whether Mauchly's sphericity assumption was violated. If the result of Mauchly's test was significant and the assumption of sphericity was violated, the Greenhouse–Geisser adjustment was used to correct sphericity by altering the degrees of freedom using a correction coefficient epsilon. When a significant main effect was identified, Tukey's HSD post hoc multiple-comparison was employed to identify specific differences. Significance was set at $p < 0.05$.

Results

Experiment 1

ANOVAs for RT showed the significant main effect of Intensity [Greenhouse–Geisser correction: F (1.405, 21.080) = 23.216, $p < 0.001$, $\varepsilon = 0.468$]. The post hoc test showed that RT was significantly earlier in the 1.5, 2.0 and 2.5 intensities than in the 1.1 intensity ($p < 0.001$, respectively). ANOVAs for the SD of RT showed the significant main effect of Intensity [F (3, 45) = 13.467, $p < 0.001$]. The post hoc test showed that the SD of RT was significantly smaller in the 1.5, 2.0, and 2.5 intensities than in the 1.1 intensity ($p < 0.01$, $p < 0.001$, and $p < 0.001$, respectively). ANOVAs for omission errors demonstrated the significant main effect of Intensity [Greenhouse–Geisser correction: F (1.624, 24.358) = 5.384, $p < 0.05$, $\varepsilon = 0.541$]. The post hoc test showed that omission errors were significantly smaller in the 2.0 and 2.5 intensities than in the 1.1 intensity ($p < 0.01$, respectively). ANOVA for commission errors showed no significant main effect (Table 1).

Figure 1 shows grand-averaged somatosensory ERPs across all subjects for each stimulus intensity. The results of ANOVAs for the amplitude of N140 showed the significant main effects of intensity [F (3, 45) = 11.108, $p < 0.001$], Stimulus [F (1, 15) = 20.242, $p < 0.001$], and Electrode [Greenhouse–Geisser correction: F (2.633, 39.499) = 10.442, $p < 0.001$, $\varepsilon = 0.658$], αvδ Iντεvσιτψ–Eλεχτροδε [Φ (12, 180) = 2.668, $p < 0.05$], Stimulus–Electrode [F (4,

Table 1 Behavioral data for each intensity with SE in Experiment 1

	×1.1	×1.5	×2.0	×2.5
RT (ms)	334 (20)	291 (15)	276 (13)***	271 (13)***
SD of RT (ms)	82 (5)	70 (6)*	62 (5)***	62 (5)***
Commission error (%)	0.6 (0.3)	1.0 (0.3)	0.3 (0.1)	1.0 (0.3)
Omission error (%)	3.4 (1.0)	1.6 (0.7)	0.9 (0.4)**	0.7 (0.4)**

A post hoc test showed that RT was significantly earlier in the 2.0 and 2.5 intensities than in the 1.1 intensity ($p < 0.001$, respectively). A post hoc test showed that the SD of RT was significantly smaller in the 1.5, 2.0 and 2.5 intensities than in the 1.1 intensity ($p < 0.01$, $p < 0.001$, and $p < 0.001$, respectively). A post hoc test showed that omission errors were significantly smaller in the 2.0 and 2.5 intensities than in the 1.1 intensity ($p < 0.01$, respectively)

* $p < 0.05$, ** $p < 0.01$, and *** $p < 0.001$)

60) = 9.154, $p < 0.001$], and Intensity–Stimulus–Electrode interactions [F (12, 180) = 2.051, $p < 0.05$]. The post hoc test for Intensity on each stimulus and electrode showed that the amplitude of Go-N140 was significantly more negative in the 2.0 and 2.5 intensities than in the 1.1 intensity at Fz ($p < 0.05$, respectively) and C3 ($p < 0.05$ and $p < 0.001$, respectively), and in the 1.5 intensity than in the 2.5 intensity at Pz ($p < 0.05$). The post hoc test for Intensity on each stimulus and electrode showed that the amplitude of No-go-N140 was significantly more negative in the 1.5, 2.0, and 2.5 intensities than in the 1.1 intensity at Fz ($p < 0.01$, $p < 0.001$, and $p < 0.001$, respectively), and in the 2.0 and 2.5 intensities than in the 1.1 intensity at Cz ($p < 0.01$, respectively), C3 ($p < 0.01$, respectively), and C4 ($p < 0.05$, respectively).

The same ANOVAs for the latency of N140 showed the significant main effects of Intensity [F (3, 45) = 6.163, $p < 0.01$] and Electrode [Greenhouse–Geisser correction: F (2.057, 30.858) = 10.347, $p < 0.001$, $\varepsilon = 0.4514$]. The post hoc test for Intensity on each stimulus and electrode showed that the latency of Go-N140 was significantly earlier in the 2.5 intensity than in the 1.1 intensity at C3 ($p < 0.05$) and C4 ($p < 0.05$). The post hoc test for Intensity on each stimulus and electrode showed that the latency of No-go-N140 was significantly earlier in the 2.5 intensity than in the 1.1 intensity at C3 ($p < 0.05$).

ANOVAs for the amplitude of P300 showed the significant main effects of Intensity [F (3, 45) = 7.015, $p < 0.01$] and Electrode [Greenhouse–Geisser correction: F (2.808, 42.119) = 33.270, $p < 0.001$, $\varepsilon = 0.702$], and Intensity–Stimulus [F (3, 45) = 3.032, $p < 0.05$] and Stimulus–Electrode interactions [Greenhouse–Geisser correction: F (2.068, 31.022) = 54.198, $p < 0.001$, $\varepsilon = 0.517$]. The post hoc test for Intensity on each stimulus and electrode showed that the amplitude of Go-P300 was significantly larger in the 2.0 than in the 1.1 intensity at Pz and C4 ($p < 0.05$, respectively). The post hoc test for Intensity on each stimulus and electrode showed that the amplitude of No-go-P300 was significantly larger in the 2.0 and 2.5 intensities than in the 1.1 intensity at Fz ($p < 0.05$ and $p < 0.01$, respectively), and in the 1.5, 2.0 and 2.5 intensities than in the 1.1 intensity at Cz ($p < 0.05$, $p < 0.01$, and $p < 0.001$, respectively), Pz ($p < 0.05$, $p < 0.01$, and $p < 0.001$, respectively), C3 ($p < 0.05$, $p < 0.05$, and $p < 0.01$, respectively), and C4 ($p < 0.01$, $p < 0.001$, and $p < 0.001$, respectively).

ANOVAs for the latency of P300 showed the significant main effect of Intensity [Greenhouse–Geisser correction: F (1.801, 27.027) = 21.772, $p < 0.001$, $\varepsilon = 0.575$] and a Stimulus-Electrode interaction [F (4, 60) = 4.785, $p < 0.01$]. The post hoc test for Intensity on each stimulus and electrode showed that the latency of Go-P300 was significantly earlier in the 2.0 and 2.5 intensities than in the 1.1 intensity at

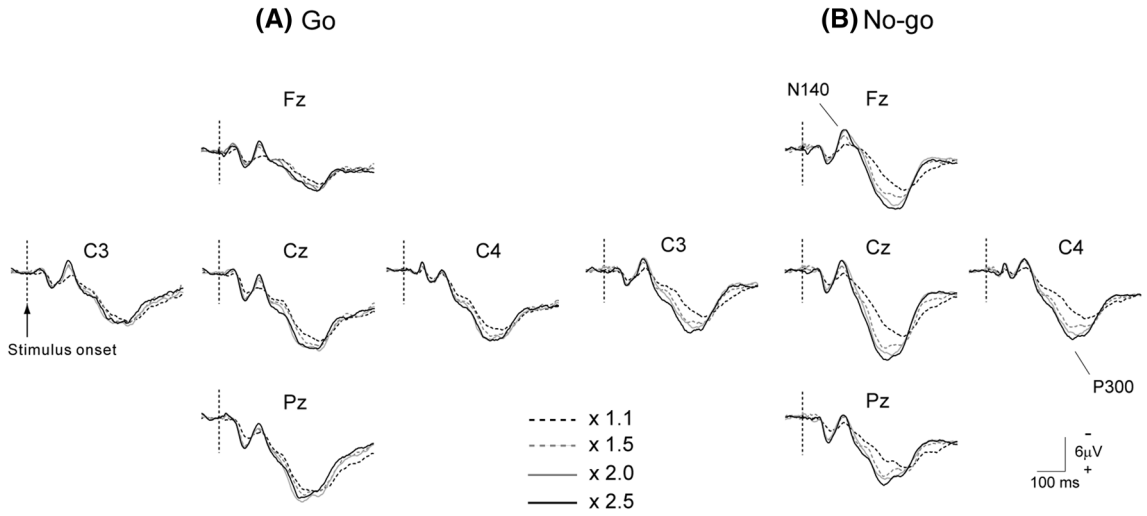

Fig. 1 Grand-averaged somatosensory ERP waveforms across all subjects in Experiment 1

Pz ($p < 0.01$ and $p < 0.001$, respectively), in the 2.5 intensity than in the 1.1 intensity at C3 ($p < 0.01$), in the 2.0 and 2.5 intensities than in the 1.1 intensity at C4 ($p < 0.05$ and $p < 0.001$, respectively), and in the 2.0 and 2.5 intensities than in the 1.5 intensity at C4 ($p < 0.05$ and $p < 0.001$, respectively). The post hoc test for Intensity on each stimulus and electrode showed that the latency of No-go-P300 was significantly earlier in the 1.5, 2.0, and 2.5 intensities than in the 1.1 intensity at Fz ($p < 0.05$, $p < 0.01$, and $p < 0.01$, respectively) and Cz ($p < 0.01$, $p < 0.001$, and $p < 0.001$, respectively), and in the 2.0 and 2.5 intensities than in the 1.1 intensity at Pz ($p < 0.01$ and $p < 0.001$, respectively), C3 ($p < 0.001$ and $p < 0.05$, respectively), and C4 ($p < 0.001$ and $p < 0.01$, respectively).

The average values for the peak amplitudes and latencies of N140 and P300 are shown in Table 2.

Experiment 2

ANOVAs for RT showed the significant main effect of Intensity [F (1, 13) = 10.961, $p < 0.01$], indicating that RT was earlier in the mild intensity than in the weak intensity. ANOVAs for the SD of RT showed the significant main effect of Intensity [F (1, 13) = 5.300, $p < 0.05$], indicating that the SD of RT was smaller in the mild intensity than in the weak intensity. ANOVAs for omission errors demonstrated the significant main effect of Intensity[F (1, 13) = 17.428, $p < 0.01$], suggesting that omission errors were lower in the mild intensity than in the weak intensity. ANOVA for commission errors showed no significant main effect or interaction (Table 3).

Figure 2 shows grand-averaged somatosensory ERPs across all subjects for each stimulus intensity. The results of ANOVAs for the amplitude of N140 showed the significant main effects of Stimulus [F (1, 13) = 25.281, $p < 0.001$] and Electrode [Greenhouse–Geisser correction: F (1.905, 24.762) = 10.676, $\varepsilon = 0.476$, $p < 0.01$], and a Stimulus-Electrode interaction [Greenhouse–Geisser correction: F (2.838, 36.895) = 11.698, $\varepsilon = 0.710$, $p < 0.001$]. The same ANOVAs for the latency of N140 showed the significant main effects of Condition [F (1, 13) = 6.001, $p < 0.05$] and Electrode [Greenhouse–Geisser correction: F (1.976, 25.682) = 9.376, $\varepsilon = 0.494$, $p < 0.01$]. However, further analyses of the effects of Condition on each intensity and electrode did not detect

Table 2 Mean values for N140 and P300 with SE in Experiment 1

	Go					No-go				
	Fz	Cz	Pz	C3	C4	Fz	Cz	Pz	C3	C4
N140 amplitude (μV)										
×1.1	− 0.3 (0.5)	1.2 (0.7)	1.0 (0.7)	− 0.6 (0.5)	0.2 (0.7)	− 1.8 (0.4)	− 0.6 (0.6)	0.0 (0.6)	− 1.0 (0.4)	− 1.3 (0.4)
×1.5	− 1.4 (0.6)	0.5 (1.0)	1.5 (0.9)	− 1.6 (0.5)	− 0.1 (0.7)	− 3.2 (0.5)	− 1.7 (0.7)	− 1.0 (0.5)	− 2.1 (0.4)	− 2.1 (0.5)
×2.0	− 2.0 (0.6)	− 0.3 (0.9)	0.5 (0.8)	− 1.8 (0.5)	− 0.6 (0.6)	− 3.7 (0.5)	− 2.8 (0.8)	− 1.5 (0.8)	− 2.9 (0.5)	− 2.7 (0.7)
×2.5	− 2.1 (0.7)	− 0.6 (1.1)	0.0 (0.9)	− 2.5 (0.7)	− 0.5 (0.8)	− 4.2 (0.5)	− 2.7 (0.7)	− 1.0 (1.0)	− 2.6 (0.6)	− 2.6 (0.5)
	#		#	#		#	#		#	#
N140 latency (ms)										
×1.1	159 (6)	160 (6)	159 (6)	160 (5)	150 (6)	165 (7)	152 (6)	152 (6)	154 (5)	140 (3)
×1.5	159 (7)	153 (7)	150 (4)	149 (5)	139 (4)	156 (6)	144 (5)	149 (4)	147 (4)	136 (3)
×2.0	152 (6)	147 (5)	146 (4)	138 (2)	138 (4)	158 (6)	147 (5)	153 (5)	149 (4)	136 (3)
×2.5	146 (5)	141 (4)	145 (4)	142 (2)	134 (2)	154 (5)	143 (4)	147 (4)	142 (2)	136 (3)
				#	#				#	
P300 amplitude (μV)										
×1.1	6.5 (1.0)	12.0 (1.5)	13.3 (1.1)	9.0 (1.0)	10.5 (1.2)	7.7 (0.8)	11.6 (1.1)	9.5 (0.8)	8.5 (0.9)	8.5 (0.9)
×1.5	7.0 (0.9)	12.8 (1.5)	13.7 (1.2)	9.5 (1.0)	11.3 (1.2)	9.2 (1.1)	14.3 (1.6)	11.3 (1.2)	10.3 (1.2)	10.6 (1.2)
×2.0	7.6 (1.0)	13.9 (1.7)	15.1 (1.4)	10.1 (1.2)	12.4 (1.3)	10.0 (1.6)	14.8 (1.8)	11.5 (1.2)	10.3 (1.5)	11.2 (1.6)
×2.5	7.5 (1.1)	13.4 (1.8)	14.3 (1.3)	9.4 (1.2)	12.0 (1.4)	10.8 (1.4)	15.5 (1.7)	12.2 (1.3)	11.2 (1.3)	11.8 (1.4)
					#	#	#	#	#	#
P300 latency (ms)										
×1.1	343 (9)	352 (8)	348 (10)	350 (8)	342 (9)	360 (10)	350 (10)	359 (12)	354 (11)	356 (10)
×1.5	342 (11)	342 (12)	327 (13)	337 (10)	339 (8)	326 (10)	316 (10)	326 (10)	334 (10)	332 (9)
×2.0	319 (11)	330 (7)	318 (10)	333 (7)	315 (9)	320 (9)	299 (7)	308 (10)	308 (6)	313 (7)
×2.5	331 (12)	333 (11)	308 (9)	319 (9)	302 (6)	314 (8)	292 (7)	304 (11)	325 (11)	316 (7)
			#	#	#	#	#	#	#	#

#Significant differences by a post hoc test for Intensity on each electrode

Table 3 Behavioral data for each condition with SE in Experiment 2

	Weak control	WN	Mild control	WN
RT (ms)	313 (18)	309 (21)	283 (20)	287 (21)
SD of RT (ms)	71 (5)	71 (5)	65 (6)	66 (6)
Commission error (%)	0.5 (0.2)	0.8 (0.3)	0.5 (0.3)	0.9 (0.3)
Omission error (%)	2.5 (0.7)	3.4 (0.7)	0.4 (0.2)	1.2 (0.5)

ANOVAs for RT, the SD of RT, and omission errors showed the significant main effect of Intensity, but no significant main effect of condition on commission errors

WN white noise condition

the significant differences in the latencies of Go- and No-go-N140 between the auditory white noise and control conditions at all intensities and electrodes.

ANOVAs for the amplitude of P300 showed the significant main effects of Condition [F (1, 13) = 19.177, $p < 0.01$], Intensity [F (1, 13) = 8.568, $p < 0.05$], and Electrode [F (4, 52) = 19.746, $p < 0.001$] as well as Condition–Stimulus [F (1, 13) = 5.235, $p < 0.05$], Intensity–Stimulus [F (1, 13) = 11.966, $p < 0.01$], Stimulus–Electrode [F (4, 52) = 26.028, $p < 0.001$], and Intensity–Stimulus–Electrode interactions [F (4, 52) = 3.920, $p < 0.01$]. Further analyses of the effects of Condition on each intensity and electrode demonstrated that the amplitude of No-go-P300 in the weak intensity was significantly larger under the auditory white noise condition than under the control condition at Fz [F (1,

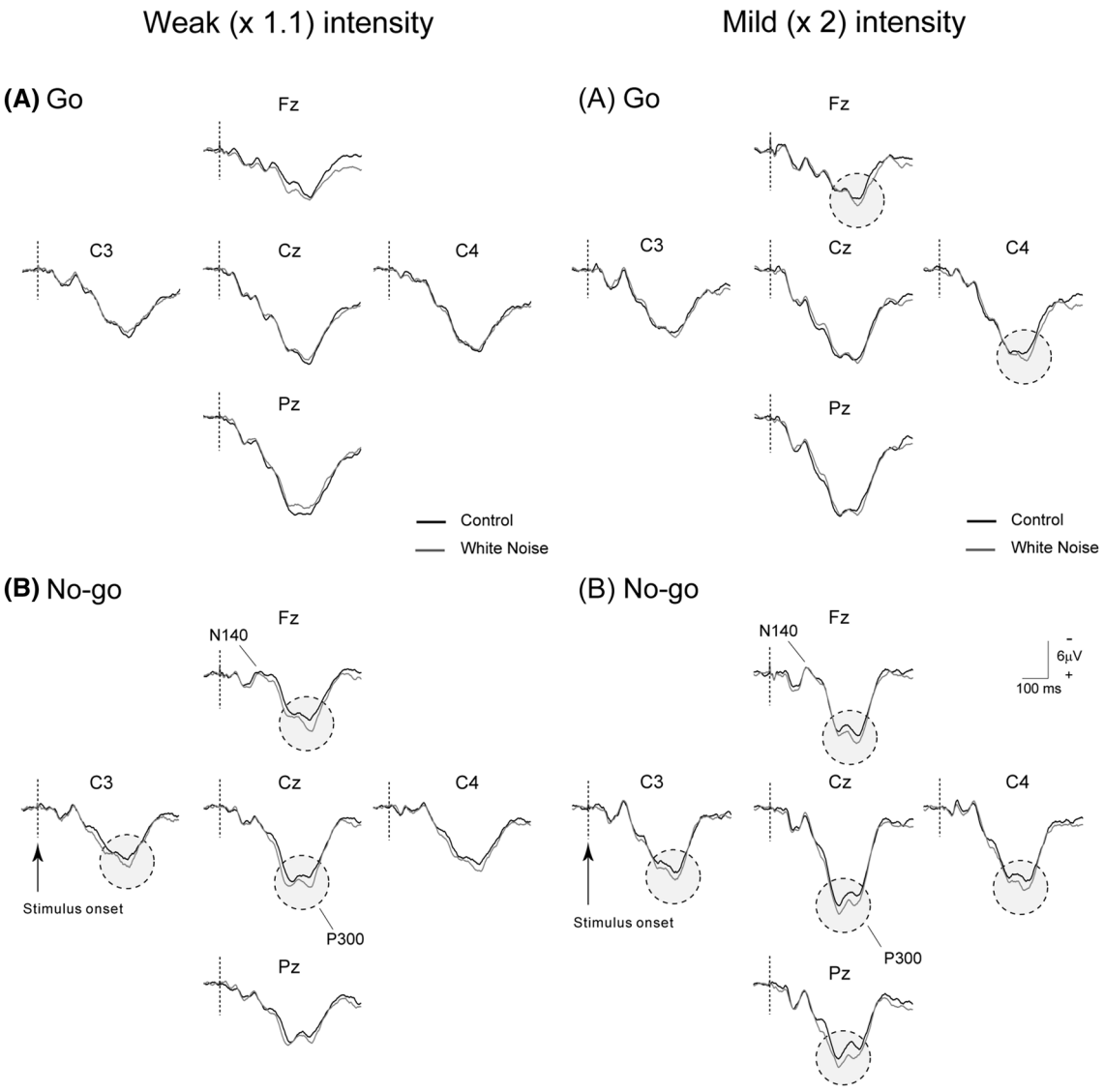

Fig. 2 Grand-averaged somatosensory ERP waveforms across all subjects for each intensity in Experiment 2. Gray circles indicate significant differences in the amplitude of P300 between the auditory white noise and control conditions

13) = 9.982, $p < 0.01$], Cz [F (1, 13) = 5.375, $p < 0.05$], and C3 [F (1, 13) = 5.330, $p < 0.05$]. In addition, the amplitude of Go-P300 in the Mild intensity was significantly larger under the auditory white noise condition than under the control condition at Fz [F (1, 13) = 4.751, $p < 0.05$] and C4 [F (1, 13) = 6.231, $p < 0.05$], and the amplitude of No-go-P300 in the mild intensity was significantly larger under the auditory white noise condition than under the control condition at Fz [F (1, 13) = 6.321, $p < 0.05$], Cz [F (1, 13) = 19.719, $p < 0.01$], Pz [F (1, 13) = 14.043, $p < 0.01$], C3 [F (1, 13) = 12.161, $p < 0.01$], and C4 [F (1, 13) = 10.914, $p < 0.01$].

ANOVAs for the latency of P300 showed the significant main effects of Intensity [F (1, 13) = 10.055, $p < 0.01$] and Electrode [F (4, 52) = 5.756, $p < 0.01$].

The average values for the peak amplitudes and latencies of N140 and P300 in Experiment 2 are listed in Table 4.

Discussion

We investigated the effects of stimulus intensity and auditory white noise on behavioral data and N140 and P300 in somatosensory Go/No-go ERPs in Experiments 1 and 2. We separately discussed each effect below.

In the behavioral data of Experiment 1, RT became shorter and the SD of RT was smaller with increase in stimulus intensity (Table 1). RT is an important measure for understanding sensorimotor performance in humans (Schmidt 2001), and is defined as the time from stimulus onset to a response, including components such as stimulus evaluation and response selection. Response variability has been identified as an important factor for evaluating the speed and accuracy of movement. It is often calculated as the SD of RT, indicating variability in the time from stimulus onset to the response (Nakata et al. 2012). Our results

Table 4 Mean values for N140 and P300 with SE in Experiment 2

	Go					No-go				
	Fz	Cz	Pz	C3	C4	Fz	Cz	Pz	C3	C4
N140 amplitude (μV)										
Weak intensity										
Control	0.0 (0.6)	2.9 (0.7)	2.6 (0.7)	0.0 (0.6)	1.1 (0.7)	− 1.7 (0.5)	0.1 (0.6)	0.3 (0.6)	− 1.4 (0.5)	− 1.1 (0.7)
WN	0.7 (0.5)	2.9 (0.8)	2.3 (0.7)	− 0.3 (0.7)	1.2 (0.7)	− 1.4 (0.5)	0.0 (0.8)	0.1 (0.8)	− 1.3 (0.6)	− 1.1 (0.8)
Mild intensity										
Control	− 0.1 (0.5)	2.7 (0.8)	2.3 (0.9)	− 1.0 (0.7)	1.0 (0.8)	− 2.1 (0.6)	− 0.3 (0.9)	− 0.1 (0.7)	− 1.6 (0.7)	− 0.9 (0.9)
WN	− 0.2 (0.6)	2.6 (0.9)	2.4 (0.8)	− 0.9 (0.7)	1.1 (0.9)	− 2.4 (0.5)	− 0.6 (0.7)	− 0.3 (0.7)	− 1.6 (0.6)	− 1.6 (0.8)
N140 latency (ms)										
Weak intensity										
Control	171 (8)	161 (8)	147 (4)	155 (6)	146 (5)	164 (7)	158 (8)	163 (8)	157 (7)	151 (7)
WN	161 (7)	150 (7)	141 (2)	147 (4)	137 (2)	162 (7)	151 (7)	151 (7)	151 (5)	136 (3)
Mild intensity										
Control	156 (8)	151 (7)	146 (6)	142 (3)	138 (4)	163 (7)	156 (6)	155 (6)	155 (7)	142 (6)
WN	158 (7)	150 (6)	149 (6)	145 (4)	141 (4)	152 (6)	149 (6)	147 (6)	148 (6)	141 (6)
P300 amplitude (μV)										
Weak intensity										
Control	7.6 (1.2)	14.3 (1.3)	14.9 (1.3)	10.0 (0.6)	12.4 (1.2)	8.0 (1.0)	12.2 (1.4)	9.5 (1.3)	8.8 (1.0)	9.5 (1.1)
WN	8.5 (1.0)	14.4 (1.2)	14.6 (1.1)	10.1 (0.6)	12.2 (1.0)	9.8 (0.9)**	13.6 (1.2)*	10.8 (1.0)	9.9 (0.9)*	10.5 (1.1)
Mild intensity										
Control	7.9 (1.1)	14.0 (1.0)	14.7 (1.5)	10.0 (0.7)	12.8 (1.2)	10.5 (1.2)	14.7 (1.6)	11.1 (1.3)	10.4 (1.2)	11.6 (1.3)
WN	8.9 (1.2)*	14.7 (1.5)	15.3 (1.6)	10.5 (0.8)	13.9 (1.3)*	11.8 (1.3)*	16.6 (1.7)**	12.8 (1.4)**	11.7 (1.2)**	12.9 (1.3)**
P300 latency (ms)										
Weak intensity										
Control	336 (10)	328 (8)	321 (11)	331 (9)	330 (8)	323 (11)	316 (10)	315 (10)	330 (12)	330 (10)
WN	309 (13)	317 (12)	315 (12)	319 (13)	324 (11)	333 (9)	317 (12)	313 (10)	333 (10)	331 (10)
Mild intensity										
Control	318 (10)	301 (11)	301 (10)	317 (11)	306 (9)	314 (11)	316 (10)	298 (12)	304 (12)	325 (11)
WN	321 (9)	312 (10)	304 (11)	330 (10)	302 (9)	302 (9)	291 (10)	276 (6)	310 (8)	309 (12)

Control condition vs. White noise (WN) condition, * $p < 0.05$ and ** $p < 0.01$

showed that RT and the SD of RT were affected by stimulus intensity, which was consistent with classical psychophysic data (Teichner 1954). Omission errors were also smaller with increase in stimulus intensity. These results suggest that somatosensory perception, which involves a chain of events consisting of phenomena such as detection, memory, discrimination, categorization, and decision-making (Romo et al. 2012), was affected by stimulus intensity.

In ERP waveforms in Experiment 1, the peak latencies of N140 and P300 became shorten and the peak amplitudes of N140 and P300 were enhanced by increase in stimulus intensity (Fig. 1; Table 2). Previous studies using classical oddball paradigms reported that N1 (N140) and P300 in target stimuli were affected by stimulus intensity in auditory (Sugg and Polich 1995; Fjell and Walhovd 2003) and somatosensory (Nakajima and Imamura 2000) stimuli. P300 is generally considered to involve an endogenous component, which is related to the amount of attentional resources allocated to the stimulus (Duncan et al. 2009). However, these previous findings also suggested that ERP waveforms in target stimuli consist of not only endogenous but also exogenous components. In the present study, since the latencies and amplitudes of N140 and P300 were modulated by an increase in the stimulus intensity, neural activities for Go (target and motor execution) and No-go (non-target and motor inhibition) stimuli during Go/No-go paradigms should include endogenous and exogenous components.

Based on the results of Experiment 1, we examined the effects of auditory white noise on somatosensory ERPs elicited by weak and mild stimulus intensities in Experiment 2. Our results showed that the amplitudes of Go- and No-go-P300 were significantly larger under the auditory white noise condition than under the control condition (Fig. 2; Table 4). We need to consider why auditory white noise enhanced the amplitudes of Go- and No-go-P300. We hypothesized dopaminergic neuromodulation. Dopamine neurons are mainly generated from the SN/VTA in the midbrain region. Rausch and colleagues (2014) using functional magnetic resonance imaging (fMRI) noted improvements of neuronal activation in the midbrain region while listening to auditory white noise, as well as enhanced connectivity between midbrain regions and the superior temporal sulcus (Rausch et al. 2014). The superior temporal sulcus belongs to a part of the temporoparietal junction, which is associated with the neuronal networks of Go- and No-go-P300 (Nakata et al. 2008a, b). Moreover, dopamine neurons are transmitted from the SN/VTA to the hippocampal region (Düzel et al. 2009). The hippocampal region is also one of the generators of P300 (Halgren et al. 1980; Nishitani et al. 1998). Therefore, we considered that some neural pathways for dopamine neuron release enhanced the amplitudes of Go- and No-go-P300. However, the present study did not evaluate dopamine release directly. Thus, in future studies, the hypothesis for dopaminergic neuromodulation during listening auditory white noise should be examined. Indeed, Salimpoor and colleagues (2011) showed endogenous dopamine release while listening to pleasurable music using positron emission tomography (Salimpoor et al. 2011).

We also need to consider why the effects of auditory white noise were more sensitive to the amplitude of No-go-P300 than that of Go-P300, because the number of electrodes showing significant differences between the auditory white noise and control conditions was clearly larger with the No-go stimulus than Go stimulus (Fig. 2; Table 4). This may be related to differences in generator mechanisms between Go- and No-go-P300. Our previous fMRI studies, which were performed using the somatosensory Go/No-go paradigm, showed that the strength of neural activity was greater with the No-go stimulus than Go stimulus in the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, anterior cingulate cortex, inferior parietal lobule, and caudate nucleus (Nakata et al. 2008a, b). Therefore, we speculate that these areas are readily affected by auditory white noise.

In addition, enhancements in the amplitudes of Go-P300 and No-go-P300 were observed under the weak and mild intensities. As discussed in the Introduction, previous studies using psychophysic methods demonstrated that stochastic resonance was sensitive to stimulus intensities across different modalities (Manjarrez et al. 2007; Lugo et al. 2008). We considered that this discrepancy was related to required tasks. Previous studies used sensory detection tasks with a sub-threshold stimulus, whereas the present study used cognitive Go/No-go paradigms with a supra-threshold stimulus. Therefore, the experimental findings cannot simply be compared. In addition, Méndez-Balbuena and colleagues (2015) used the visual evoked potential (VEP), which was elicited by a supra-threshold stimulus, and reported that the amplitude of the P100 component was modulated by tactile noise at an optimal level, compared with zero noise or a high mechanical noise level (Méndez-Balbuena et al. 2015). Further studies are needed to clarify the mechanisms responsible for the differences between the sensory and cognitive Go/No-go responses occurring simultaneously under sub-threshold conditions during different levels of auditory white noise.

In contrast, the amplitudes and latencies of Go- and No-go-N140 were not affected by auditory white noise. Previous studies reported that N140 was generated from several regions including the secondary somatosensory cortex, insula, cingulate cortex, and prefrontal cortex (Inui et al. 2003; Nakata et al. 2005; Kida et al. 2006). These findings indicated that the effects of auditory white noise did not affect the generator for Go-N140 or No-go-N140. Moreover, in the behavioral data of Experiment 2, no significant effects of auditory white noise were observed on

RT, the SD of RT, or error rates (Table 3), while the amplitude of P300 was modulated. Sikström and colleagues also recently reported no significant effects of auditory white noise on RT during visual Go/No-go paradigms (Sikström et al. 2016), and Treviño and colleagues showed no effects of background white pixel-noises on RT during visual discrimination tasks with a random dot motion stimulus (Treviño et al. 2016). Herweg and Bunzeck (2015), using some memory tasks, suggested that auditory white noise affected some parts of the cognitive function, rather than all cognitive process. Our results support these findings.

In conclusion, the present study investigated the effects of stimulus intensity and auditory white noise on behavioral data, and N140 and P300 in somatosensory Go/No-go paradigms. Behavioral data, including RT, the SD of RT, and omission errors, and the amplitudes and latencies of N140 and P300 were affected by stimulus intensities. Auditory white noise was shown to affect the amplitude of P300, irrespective of the stimulus intensity. Our results revealed a characteristic of neural substrates for stochastic resonance by utilizing somatosensory ERPs.

Compliance with ethical standards

Conflict of interest There are no conflicts of interest.

References

Bokura H, Yamaguchi S, Kobayashi S (2001) Electrophysiological correlates for response inhibition in a Go/NoGo task. Clin Neurophysiol 112:2224–2232

Braver TS, Barch DM, Gray JR, Molfese DL, Snyder A (2001) Anterior cingulate cortex and response conflict: effects of frequency, inhibition and errors. Cereb Cortex 11:825–836

Duncan CC, Barry RJ, Connolly JF, Fischer C, Michie PT, Näätänen R, Polich J, Reinvang I, Van Petten C (2009) Event-related potentials in clinical research: guidelines for eliciting, recording, and quantifying mismatch negativity, P300, and N400. Clin Neurophysiol 120:1883–1908

Düzel E, Bunzeck N, Guitart-Masip M, Wittmann B, Schott BH, Tobler PN (2009) Functional imaging of the human dopaminergic midbrain. Trends Neurosci 32:321–328

Falkenstein M, Hoormann J, Hohnsbein J (1999) ERP components in Go/Nogo tasks and their relation to inhibition. Acta Psychol 101:267–291

Fjell AM, Walhovd KB (2003) Effects of auditory stimulus intensity and hearing threshold on the relationship among P300, age, and cognitive function. Clin Neurophysiol 114:799–807

Halgren E, Squires NK, Wilson CL, Crandall PH (1980) Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events. Science 210:803–805

Hatem SM, Plaghki L, Mouraux A (2007) How response inhibition modulates nociceptive and non-nociceptive somatosensory brain-evoked potentials. Clin Neurophysiol 118:1503–1516

Herweg NA, Bunzeck N (2015) Differential effects of white noise in cognitive and perceptual tasks. Front Psychol 6:1639

Huster RJ, Westerhausen R, Pantev C, Konrad C (2010) The role of the cingulate cortex as neural generator of the N200 and P300 in a tactile response inhibition task. Hum Brain Mapp 31:1260–1271

Inui K, Tran TD, Qiu Y, Wang X, Hoshiyama M, Kakigi R (2003) A comparative magnetoencephalographic study of cortical activations evoked by noxious and innocuous somatosensory stimulations. Neuroscience 120:235–248

Kamijo K, Nishihira Y, Hatta A, Kaneda T, Wasaka T, Kida T, Kuroiwa K (2004) Differential influences of exercise intensity on information processing in the central nervous system. Eur J Appl Physiol 92:305–311

Kida T, Wasaka T, Inui K, Akatsuka K, Nakata H, Kakigi R (2006) Centrifugal regulation of human cortical responses to a task-relevant somatosensory signal triggering voluntary movement. NeuroImage 32:1355–1364

Kopp B, Mattler U, Goertz R, Rist F (1996) N2, P3 and the lateralized readiness potential in a nogo task involving selective response priming. Electroencephalogr Clin Neurophysiol 99:19–27

Lugo E, Doti R, Faubert J (2008) Ubiquitous crossmodal stochastic resonance in humans: auditory noise facilitates tactile, visual and proprioceptive sensations. PLoS One 3:e2860

Manjarrez E, Mendez I, Martinez L, Flores A, Mirasso CR (2007) Effects of auditory noise on the psychophysical detection of visual signals: cross-modal stochastic resonance. Neurosci Lett 415:231–236

Méndez-Balbuena I, Huidobro N, Silva M, Flores A, Trenado C, Quintanar L, Arias-Carrión O, Kristeva R, Manjarrez E (2015) Effect of mechanical tactile noise on amplitude of visual evoked potentials: multisensory stochastic resonance. J Neurophysiol 114:2132–2143

Nakajima Y, Imamura N (2000) Relationships between attention effects and intensity effects on the cognitive N140 and P300 components of somatosensory ERPs. Clin Neurophysiol 111:1711–1718

Nakata H, Inui K, Nishihira Y, Hatta A, Sakamoto M, Kida T, Wasaka T, Kakigi R (2004) Effects of a go/nogo task on event-related potentials following somatosensory stimulation. Clin Neurophysiol 115:361–368

Nakata H, Inui K, Wasaka T, Akatsuka K, Kakigi R (2005) Somatomotor inhibitory processing in humans: a study with MEG and ERP. Eur J Neurosci 22:1784–1792

Nakata H, Sakamoto K, Ferretti A, Perrucci GM, Del Gratta C, Kakigi R, Romani GL (2008a) Somato-motor inhibitory processing in humans: an event-related functional MRI study. NeuroImage 39:1858–1866

Nakata H, Sakamoto K, Ferretti A, Perrucci GM, Del Gratta C, Kakigi R, Romani GL (2008b) Executive functions with different motor outputs in somatosensory Go/Nogo tasks: an event-related functional MRI study. Brain Res Bull 77:197–205

Nakata H, Sakamoto K, Kakigi R (2012) The relationship between reaction time and response variability and somatosensory No-go potentials. Eur J Appl Physiol 112:207–214

Nakata H, Sakamoto K, Honda Y, Kakigi R (2015) Temporal dynamics of neural activity in motor execution and inhibition processing. Eur J Neurosci 41:1448–1458

Nishitani N, Nagamine T, Fujiwara N, Yazawa S, Shibasaki H (1998) Cortical-hippocampal auditory processing identified by magnetoencephalography. J Cogn Neurosci 10:231–247

Ohbayashi W, Kakigi R, Nakata H (2017) Effects of white noise on event-related potentials in somatosensory Go/No-go paradigms. NeuroReport 28:788–792

Rausch VH, Bauch EM, Bunzeck N (2014) White noise improves learning by modulating activity in dopaminergic midbrain regions and right superior temporal sulcus. J Cogn Neurosci 26:1469–1480

Roche RA, Garavan H, Foxe JJ, O'mara SM (2005) Individual differences discriminate event-related potentials but not performance during response inhibition. Exp Brain Res 160:60–70

Romo R, Lemus L, de Lafuente V (2012) Sense, memory, and decision-making in the somatosensory cortical network. Curr Opin Neurobiol 22:914–919

Salimpoor VN, Benovoy M, Larcher K, Dagher A, Zatorre RJ (2011) Anatomically distinct dopamine release during anticipation and experience of peak emotion to music. Nat Neurosci 14:257–262

Schmidt RA (2001) Motor learning and performance. Human Kinetics, Champaign, USA, pp 55–87

Shibasaki M, Namba M, Oshiro M, Kakigi R, Nakata H (2017) Suppression of cognitive function in hyperthermia; from the viewpoint of executive and inhibitive cognitive processing. Sci Rep 7:43528

Sikström S, Söderlun G (2007) Stimulus-dependent dopamine release in attention-deficit/hyperactivity disorder. Psychol Rev 114:1047–1075

Sikström S, Jürgensen AM, Haghighi M, Månsson D, Smidelik D, Habekost T. Self-rated attentiveness interacts with transcranial direct current stimulation and noise stimulation in reaction time in a Go/No-Go task. Neural Plast. 2016; 2016: 5302538

Söderlund GB, Sikström S, Loftesnes JM, Sonuga-Barke EJ (2010) The effects of background white noise on memory performance in inattentive school children. Behav Brain Funct 6:55

Sugg MJ, Polich J (1995) P300 from auditory stimuli: intensity and frequency effects. Biol Psychol 41:255–269

Teichner WH (1954) Recent studies of simple reaction time. Psychol Bull 51:128–149

Treviño M, De la Torre-Valdovinos B, Manjarrez E (2016) Noise improves visual motion discrimination via a stochastic resonance-like phenomenon. Front Hum Neurosci 10:572

Usher M, Feingold M (2000) Stochastic resonance in the speed of memory retrieval. Biol Cybern 83:L11–L16