Effects of electrical stimulation of dorsal raphe nucleus on neuronal response properties of barrel cortex layer IV neurons following long-term sensory deprivation

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Abstract: Objective To evaluate the effect of electrical stimulation of dorsal raphe nucleus (DRN) on response properties of layer IV barrel cortex neurons following long-term sensory deprivation. **Methods** Male Wistar rats were divided into sensory-deprived (SD) and control (unplucked) groups. In SD group, all vibrissae except the D2 vibrissa were plucked on postnatal day one, and kept plucked for a period of 60 d. After that, whisker regrowth was allowed for 8-10 d. The D2 principal whisker (PW) and the D1 adjacent whisker (AW) were either deflected singly or both deflected in a serial order that the AW was deflected 20 ms before PW deflection for assessing lateral inhibition, and neuronal responses were recorded from layer IV of the D2 barrel cortex. DRN was electrically stimulated at inter-stimulus intervals (ISIs) ranging from 0 to 800 ms before whisker deflection. **Results** PW-evoked responses increased in the SD group with DRN electrical stimulation at ISIs of 50 ms and 100 ms, whereas AW-evoked responses increased at ISI of 800 ms in both groups. Whisker plucking before DRN stimulation could enhance the responsiveness of barrel cortex neurons to PW deflection and decrease the responsiveness to AW deflection. DRN electrical stimulation significantly reduced this difference only in PW-evoked responses between groups. Besides, no DRN stimulation-related changes in response latency were observed following PW or AW deflection in either group. Moreover, condition test (CT) ratio increased in SD rats, while DRN stimulation did not affect the CT ratio in either group. There was no obvious change in 5-HT_{2A} receptor protein density in barrel cortex between SD and control groups. **Conclusion** These results suggest that DRN electrical stimulation can modulate information processing in the SD barrel cortex.

Keywords: sensory system; barrel cortex; 5-hydroxytryptamine receptors

1 Introduction

Whiskers, as sensory organs, are important for tactile

exploration, equilibrium, orientation, spatial learning, *etc.*^[1-3]. The representation of the whiskers in the rat somatosensory cortex is magnified relative to those of other body parts, since it forms histologically distinctive regions named “barrels”^[4].

Experience-dependent plasticity, an important characteristic of cerebral cortex, is present not only during neonatal development, but throughout the whole life^[5,6]. The plastic-

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ity of cortex representations has been studied at various ages, including early postnatal, adolescent, and adulthood stages^[7,8].

Serotonergic system influences cortical plasticity and plays different roles in regulating the function of somatosensory cortex^[9]. Serotonergic innervation of the cortex originates from the dorsal raphe nucleus (DRN) and the median raphe nucleus (MRN) of the medulla. The thin axons of DRN neurons and thick axons of MRN neurons project to the cortex^[9]. It is known that the serotonergic afferents originating from DRN start to innervate cortex on embryonic days 15 and 16^[10]. The rodent sensory cortex contains a variety of serotonergic receptors, including 5-HT_{1A} and 5-HT_{2A}^[11,12]. It is likely that 5-HT_{2A} receptors mediate the effects of dorsal, but not median raphe projections^[12].

Our previous study has reported that the electrical stimulation of DRN modulates response properties of barrel cortex neurons to whisker stimulation^[13]. However, little is known about how DRN activation changes the response properties of barrel cortex following long-term sensory deprivation, or which receptors are involved in this process. The present study was designed to assess the effect of electrical stimulation of DRN on the response properties of barrel cortex neurons after long-term sensory deprivation, and to study whether 5-HT_{2A} receptor activity in the cortical barrels was involved in the plasticity process.

2 Materials and methods

2.1 Animal preparation A total number of 35 juvenile male Wistar rats were employed in the present study. They were divided into control ($n=16$) and sensory-deprived (SD) ($n=19$) groups. Rats were housed under a constant 12:12 h dark/light cycle, with free access to food and water. Whisker plucking was performed by applying slow strain to the base of vibrissa^[14]. All the experiments were conducted according to the guidelines on ethical standards for investigation of animals, which were approved by the Animal Experimentation Ethic Committee of Kerman Neuroscience Research Center (EC/KNRC/86-44). In SD animals, all the whiskers on the left side of the face, except the D2, were plucked on the day of birth. Whiskers were checked every other day for regrowth, and were plucked when necessary during a 60-d period. Af-

ter that, a further period of 8-10 d was allowed for whisker regrowth to approximately 10 mm, after which the recording was performed. Whiskers in control rats were all left intact.

2.2 Extracellular single unit recording For electrophysiological single unit recording, the animals were anesthetized with urethane (1.2 mg/kg, i.p.) and mounted on stereotaxic apparatus (Stoelting, USA). The depth of anesthesia was monitored throughout the experiment by testing the absence of spontaneous movement of whiskers and absence of response to hindpaw pinch and eye blink reflex. When necessary, additional doses of urethane (10% of induction dose) were administered to maintain constant anesthesia. A 3×3 mm² craniotomy window was made to expose the right posteromedial barrel subfield (1-4 mm posterior and 4-7 mm lateral to bregma)^[14], and the dura over the somatosensory area was not removed.

2.3 Stimulations A stimulating electrode was inserted into the DRN 7.8 mm posterior and 1.8 mm lateral to the midline. It was advanced 6.2 mm with an angle of 18° relative to midline, to avoid bleeding from sagittal sinus^[13,15]. The cortical surface was covered with 3% warm agar solution. Tungsten microelectrodes (1 MΩ) were slowly advanced perpendicular to the cortical surface and positioned into layer IV (600–900 μm) of the D2 barrel by a microdriver. It was kept still for about half an hour to reshape the cortex. Extracellular spike signals were amplified 10 000 times, filtered at 0.3-10 KHz with a band-pass filter (WPI, Dam-80), and sent to an amplitude window discriminator for single spike separation. Spike shapes were visualized through a storage oscilloscope with a 10-ms delay line circuit. The whisker whose displacement elicited the strongest neuronal response^[16] with the shortest latency was identified as the principal whisker (PW) (D2 in this study). The D1 whisker located caudally to the PW was chosen as the adjacent whisker (AW). Before recording, all whiskers on the left side of the snout were trimmed to the length of 10 mm, and the trimmed D2 and D1 whiskers were inserted 1-2 mm into 2 small-diameter glass tubes that were attached to miniature speakers. The stimulus waveforms consisted of ramp-and-hold trapezoids, which produced 500 μm displacement (deflection of whiskers was about 500 μm in height) for a duration of 200 ms^[13]. The amplitude of displacements was

calibrated using a microscope, with a scale being built into one of the eyepieces. For DRN stimulation, twisted bipolar steel electrode (0.125 mm in diameter, WPI) was used. The electrical stimulation of DRN consisted of trains of 10 pulses with 0.3 ms duration, and 75–200 μ A current (adjusted to be subthreshold in terms of directly affecting barrel neuron's spontaneous discharge), which was delivered with a frequency of 40 Hz using a stimulus isolator (WPI, A365).

To assess the effect of DRN stimulation on barrel neuronal responses, 2 different protocols were used. One is that the PW and AW were deflected individually to yield information about excitatory receptive fields, and the other is that the AW was deflected 20 ms before PW deflection (combined whisker deflection paradigm) to investigate the strength of surround inhibition^[2]. Consistent with both excitation and inhibition intervals (0–800 ms) for DRN^[17], DRN stimulation was applied at constant intervals of 0, 50, 100, 200, 400 and 800 ms before PW or AW deflection or only before PW deflection in combined whisker deflection paradigm. Whiskers were also deflected without DRN stimulation to establish a baseline response (indicated by inter-stimulus intervals “without”). Each deflection with or without DRN stimulation was repeated 40 times with a frequency of 0.5 Hz, in a random order.

2.4 Experimental procedures and data analysis Spike units during the first 200 ms of each recording file (with no stimulation) were counted as spontaneous activity. Response magnitudes were calculated by counting the number of spikes from 5 ms to 55 ms after the onset of each deflection. The response latencies were considered as the post-deflection time bin when the neuronal responses exceeded mean+2SD of spontaneous activity. Whisker interaction was quantified with a condition test ratio (CT ratio) as previously reported^[16]. This ratio indicates the effect of conditioning stimulus (AW deflection) on the response of test stimulus (PW deflection), and has been defined as the ratio between the observed and the expected responses^[16]. At the end of the experiments, small electrolytic lesions were made in the sites of recording and stimulation in the D2 barrel and DRN (D2 barrel: 2–3 μ A for 10 s, DRN: 20 μ A for 20 s). A coronal section of DRN with electrical stimulation was shown in Fig. 1, as stained by Nissl,

Fig. 1 A photomicrograph showing the track of stimulating electrode insertion into dorsal raphe nucleus (DRN). Scale bar: 400 μ m.

and the track of stimulating electrode insertion into DRN was also demonstrated.

The animals were perfused trans-cardially with physiological saline followed by 4% paraformaldehyde. The brains were removed for histological analysis. Cytochrome oxidase staining was used to visualize barrels in tangential sections, and the Nissl cell body staining was for localizing lesions of DRN in the brainstem^[14,18]. Data were presented as means $\pm$ SEM and analyzed with *t*-test or repeated measures ANOVA (RMA) followed by two-tailed paired *t*-test as post test using SPSS software (version 17). *P* < 0.05 was considered to be statistically significant.

2.5 Immunoblot analysis Immunoblot analysis was performed to observe the changes in the distribution of 5-HT_{2A} receptor, the most abundant receptor of 5-HT^[19], in barrel cortex following sensory deprivation. In separate groups with similar treatment as discussed above, the dissected brain tissues from barrel field (marked through electrophysiological electrodes) were homogenized in ice-cold RIPA buffer using a tissue punch (2 mm in diameter). Equal amounts of samples were resolved electrophoretically in 9% SDS-PAGE gel and transferred to nitrocellulose membranes. After being blocked overnight at 4 °C, the membranes were incubated with anti-5-HT_{2A} [1:1 000; SR-2A (M-75): sc-50397, Santa Cruz, USA] or anti- β -actin primary antibody (1:1 000; Cell Signaling Technology, INC., MA, USA). After being washed, mem-

branes were incubated with the horseradish peroxidase-conjugated secondary antibody. The antibody-antigen complexes were detected using the ECL system and exposed to Lumi-Film chemiluminescence detection film. Lab work analyzing software was used to analyze the intensity of protein band. β -Actin served as a control.

3 Results

The percentages of changes in neuronal response magnitude to deflections of PW or AW after DRN electrical stimulation in both control and SD rats were listed in Table 1. In SD rats, PW-evoked responses increased with DRN electrical stimulation at inter-stimulus intervals (ISIs) of 50 and 100 ms (RMA followed by paired *t*-test, $P < 0.05$, Fig. 2A), compared with those at pre-stimulation condition. As shown in Fig. 2B, AW-evoked responses increased with DRN electrical stimulation at ISI of 800 ms in both control and SD rats (RMA followed by paired *t*-test, $P < 0.01$ for SD rats, $P < 0.05$ for control rats), compared with those at pre-stimulation condition. Moreover, whisker plucking induced an increase in neuronal response of layer IV barrel cortex neurons to deflection of the PW, and a decrease in responsiveness to deflection of the AW (Fig. 2A, B). DRN electrical stimulation at all ISIs reduced the difference in PW-evoked responses between control and SD rats (*t*-test, $P > 0.05$; Fig. 2A).

The effects of sensory deprivation and DRN electrical stimulation on neuronal response latency were shown in Table 2. There was no significant difference in response latency to PW deflection between control and SD groups (*t*-test, $P > 0.05$), however, response latency to AW deflection increased in SD group (*t*-test, $P < 0.001$), compared to that in

control group. Besides, no response latency changes related to DRN stimulation were observed for PW or AW deflection in either group (RMA followed by paired *t*-test, $P > 0.05$, Table 2).

As illustrated in Fig. 3, CT ratio increased in SD rats compared to that in control group, while DRN stimulation did

Fig. 2 Effect of DRN stimulation with different ISIs on response magnitudes of barrel neurons to PW (A) or AW (B) deflection. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ vs the control. # $P < 0.05$, ## $P < 0.01$ vs without stimulation. $n=16$ in control group, $n=19$ in SD group. Here n indicated the number of neurons.

Table 1. Percentages of changes in response magnitude to deflection of PW or AW following DRN electrical stimulation with different intervals, compared with no electrical stimulation.

ISIs (ms):		0	50	100	200	400	800
Percentage of change (PW)	Control	8.2	17	13.8	18.9	18	7.5
	SD	0	8.4	9.5	6.9	5.8	5
Percentage of change (AW)	Control	8.5	23.2	22.6	18.2	21.7	34.6
	SD	16.3	17.7	51.4	69.3	58.6	61

PW: principal whisker. AW: adjacent whisker. ISIs: inter-stimulus intervals.

Table 2. Response latency to PW or AW deflection at different ISIs in SD and control groups

ISIs (ms)		Without	0	50	100	200	400	800
Response latency (ms)	Control	7.5 ± 0.2	7.6 ± 0.2	7.6 ± 0.2	7.4 ± 0.3	7.4 ± 0.3	7.4 ± 0.3	7.4 ± 0.2
	(PW) SD	7.5 ± 0.2	7.3 ± 0.2	7.4 ± 0.2	7.2 ± 0.2	7.2 ± 0.2	7.2 ± 0.2	7.4 ± 0.2
Response latency (ms)	Control	11 ± 0.3	11 ± 0.2	11 ± 0.3	11 ± 0.2	11 ± 0.4	11 ± 0.5	11 ± 0.5
	(AW) SD	14 ± 0.5***	14 ± 0.6***	14 ± 0.4***	14 ± 1.1***	13 ± 0.5***	14 ± 0.4***	13 ± 0.5**

** $P < 0.01$, *** $P < 0.001$ compared to the control with the corresponding ISI in the same whisker. $n=16$ neurons in control group, $n=19$ neurons in SD group. PW: principal whisker. AW: adjacent whisker.

Fig. 3 Effect of DRN stimulation with different ISIs on CT ratio of barrel cells. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ vs the control group with the corresponding DRN stimulation. $n=16$ in control group, $n=19$ in SD group. Here n indicated the number of neurons.

Fig. 4 Effect of sensory deprivation on 5-HT_{2A} receptor protein density. $n=7$ in control group, $n=8$ in SD group. Here n indicated the number of brain sections of barrel area and each of the sections was taken from a single rat.

not significantly affect the CT ratio in either group (except at ISI of 800 ms).

As shown in Fig. 4, sensory deprivation had no effect on the density of 5-HT_{2A} receptor protein in barrel cortex.

4 Discussion

The present study indicated that whisker plucking from the first day of birth could lead to both enlarged excitatory and weakened inhibitory responses in the layer IV barrel neurons, which is consistent with our previous study^[3]. Whisker plucking affects barrel neurons' response properties, and these data support previous findings in neonatally SD rats^[20].

Here we also found that CT ratio, a sign of altered inhibition-potential balance, increased due to sensory deprivation. After induction of plasticity in barrel cortex, it was likely that whisker plucking from the date of birth significantly decreased the inter-whisker suppression in the spared barrel. Therefore, the observed plasticity in the barrels may be due to the changed balance between inhibitory and excitatory responses and is in favor of disinhibition in the barrel cortex. It is also possible that a postnatal age-sensitive component of the inhibition-potential balancing system may be responsible for different CT ratios in the SD group. Other studies have shown that whisker plucking could down-regulate the GABAergic system and, as a result, diminish surround inhibition of AW-related barrel onto the PW-related barrel^[21].

Concerning the effect of DRN stimulation on barrel response properties, we observed that DRN electrical stimulation at all ISIs significantly attenuated the difference in PW-evoked responses between SD and control groups. This

means that DRN stimulation can modulate the responses of barrel cortex neurons. Patterning of response magnitude to AW deflection was not affected by DRN stimulation (except at ISI of 800 ms). These data are supported by our previous study^[13].

It has been recognized that neocortical pyramidal neurons receive indirect serotonergic inputs that modulate pyramidal cell activity through binding to 5-HT_{2A} receptors^[22]. In addition, cortical interneurons with 5-HT_{2A} receptor-binding sites also receive serotonergic inputs^[23]. Serotonergic projections on thalamo-cortical afferents play an important role in postnatal growth of barrel cortex^[24]. It has been reported that 5-HT participates in the process of fine-patterning of cortical barrels through interacting with thalamo-cortical afferents^[24]. Furthermore, several studies have indicated that 5-HT helps thalamo-cortical afferent development, which might be promoted by co-existent components in 5-HT vesicles, including neurotrophics such as thyrotropin-releasing hormone (TRH), growth-associated protein-43 (GAP-43), NADPH and substance P^[25].

In this study, whether the response properties of barrel neurons would be altered due to the long-term sensory deprivation was examined. Besides, by determining the density of 5-HT_{2A} receptor (the predominant receptor in layer IV), we attempted to know if this receptor was responsible for the altered response properties in barrels.

However, our results showed no changes in 5-HT_{2A} receptor density, suggesting that there may be other 5-HT receptor subtypes (like 5-HT_{2C}) responsible for the changes in the neuronal response properties following long-term cortical sensory plasticity. It is reported that serotonergic 5-HT_{2C} receptor is the main receptor subtype in cortical plasticity, although its density seems to be low in barrel cortex^[9].

Taken together, DRN activation can affect neural response properties of barrel cortex following neonatal sensory deprivation. The modulating effect of DRN electrical stimulation on response properties of barrel neurons, which was observed in layer IV of barrel cortex, may be due to the existence of different functionally-opposing 5-HT-binding sites over a variety of barrel cortex neurons.

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背缝神经核的电刺激对经过长期感觉剥夺的大鼠皮层 IV 层神经元的神经应答特性的影响

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摘要: 目的 本文旨在研究长期感觉剥夺后, 背缝神经核的电刺激对桶状皮层(barrel cortex) IV 层神经元的神经应答特性的影响。方法 雄性 Wistar 大鼠分为感觉剥夺组(sensory-deprived, SD)和对照组。SD 组大鼠从出生开始, 左半脸所有胡须(D2 胡须除外)均被拔除, 并维持 60 天。在之后的 8-10 d 内, 允许胡须重新生长。对背缝神经核给予电刺激, 之后偏转胡须, 记录 D2 桶状皮层 IV 层神经元的神经应答。结果 当给予的电刺激的间距为 50 ms 和 100 ms 时, 电刺激能增强 SD 大鼠主须(principal whisker, PW)偏转引起的神经应答, 而当给予的电刺激的间距为 800 ms 时, 电刺激能增强 SD 组和对照组大鼠邻近胡须(adjacent whisker, AW)偏转引起的神经应答。与对照组相比, 胡须的拔除能增强桶状皮层对 PW 偏转的应答, 而削弱其对 AW 偏转的应答。此外, PW 或 AW 偏转后, 在两组中均未发现与背缝神经核的电刺激相关的反应潜伏期的变化。与对照组相比, SD 大鼠的桶状皮层中 5-HT_{2A} 受体的表达水平无明显差异。结论 背缝神经核的电刺激能调节感觉剥夺的桶状皮层的信息处理。

关键词: 感觉系统; 桶状皮层; 5-羟色胺受体