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# Effect of gabazine on sensory stimulation train evoked response in mouse cerebellar Purkinje cells

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Cerebellar Purkinje cells (PCs) respond to sensory stimulation *via* climbing fiber and mossy fiber-granule cell pathways, and generate motor-related outputs according to internal rules of integration and computation. However, the dynamic properties of sensory information processed by PC in mouse cerebellar cortex are currently unclear. In the present study, we examined the effects of the gamma-aminobutyric acid receptor A ($GABA_A$) antagonist, gabazine, on the stimulation train on the simple spike firing of PCs by electrophysiological recordings method. Our data showed that the output of cerebellar PCs could be significantly affected by all pulses of the low-frequency (0.25 -2 Hz) sensory stimulation train, but only by the 1st and 2nd pulses of the high-frequency ($\geq$ 4 Hz) sensory stimulation train. In the presence of gabazine (20 μM), each pulse of 1 Hz facial stimulation evoked simple spike firing in the PCs, but only the 1st and 2nd pulses of 4 Hz stimulation induced an increase in simple spike firing of the PCs. These results indicated that $GABA_A$ receptor-mediated inhibition did not significantly affect the frequency properties of sensory stimulation evoked responses in the mouse cerebellar PCs.

## 1. Introduction

The cerebellar PCs receive an extensive variety of projections and provide the principal output from the cerebellar cortex to deep cerebellar nuclei (Palay and Chan-Palay 1974). The mossy fibers and the climbing fibers are two different types of excitatory afferents, which are considered to transfer distinct information into the cerebellar cortex. The climbing fiber input derives from the inferior olive and activates the PCs directly by firing complex spikes (Ecces et al. 1967; Ito 1984). In contrast, the mossy fiber input comes from the pontine nuclei and reticular nucleus, and synapses onto granule cells, which in turn send their output through ascending axons, and subsequently the parallel fibers, into the molecular layer (Ecces et al. 1967; Ito 1984; Holtzman et al. 2006). Under *in vitro* conditions, electrical stimulation of granule cells or parallel fibers can excite PCs, and evoke simple spike firing of PCs (Eccles et al. 1967; Meek 1992; Dunbar et al. 2004; Gao et al. 2003; Qiu et al. 2008). However, the spike firing of PCs is difficult to induce by natural stimulation (Bell and Grimm 1969; Bower and Woolston 1983; Cohen and Yarom 1998; Eccles et al. 1972; Kolb et al. 1997). Bower and Woolston (1983) found that peripheral stimuli induce patch-like patterns of excitation in PCs. Later, the peripheral stimuli-induced patch-like patterns of excitation in PCs was assumed to be above the region of the activated granule cell layer (Lu et al. 2005; Brown and Ariel 2009). We recently found that air-puff stimulation of the ipsilateral whisker pad failed to evoke spike firing, but induced a $GABA_A$ receptor-mediated inhibition of PCs (Chu et al. 2011a, b).

The inhibitory interneurons of the cerebellar cortex molecular layer include basket and stellate cells, which receive excitatory input from parallel fibers and inhibitory input from other interneurons (Palay and Chan-Palay 1974; Häusser and Clarck 1997; Llano and Gerschenfeld 1993; Mittmann et al. 2005). Molecular interneurons have recently been divided into stellate-type and basket-type interneurons (Bower 2010; Sultan and Bower 1998; Mann-Metzer and Yarom 1999; Jörntell and Ekero 2002). Stellate-type interneurons provide dendritic inhibition, which is predicted to specifically counterbalance the parallel fiber excitation in local regions of the PC dendrites (Jaeger and Bower 1999; Jaeger et al. 1997), resulting in less direct effects on the spiking output of PCs (Santamaria et al. 2007). Basket-type interneurons offer somatic inhibition, which is powerful and rapid (Donato et al. 2008; Sakaba 2008), and results in direct effects on the spiking output of PCs by inhibition of the soma and initial segment of PCs (Donato et al. 2008; Huang et al. 2007; Santamaria et al. 2007; Bower 2010). We previously found that tactile face stimulation evokes a $GABA_A$ receptor-mediated field potential response in the molecular layer (Chu et al. 2011; Cui et al. 2014). In addition, whole-cell recordings indicated that tactile face stimulation evokes rapid excitation in molecular layer interneurons, and inhibition occurring at later latencies in PCs in mouse cerebellar cortex Crus II (Chu et al. 2012). These results suggest that the molecular layer interneuron network plays a critical role during sensory information processing in the cerebellar cortex (Chu et al. 2012).

Although previous results indicate that cerebellar PCs exhibit responses to sensory stimulation, the frequency property of sensory information processing in mouse cerebellar cortex are currently unclear, as well as the resulting patterns of PC output, are poorly understood. Therefore, here we studied the dynamic

characterization of sensory stimulation-evoked responses PCs by electrophysiological recording and pharmacological methods. We found the output of cerebellar PCs could be significantly affected by all pulses of the low-frequency (0.25 -2 Hz) sensory stimulation train, but only by the 1st and 2nd pulses of the high-frequency ($\geq$ 4 Hz) sensory stimulation train. With blockade of $GABA_A$ receptor activity, each pulse of 1 Hz facial stimulation evoked simple spike firing in the PCs, but the other pulses of stimulation still did not significantly affect the output of the PCs except the 1st and 2nd pulses. These results indicated that $GABA_A$ receptor-mediated inhibition did not significantly affect output of cerebellar PCs during sensory information processing in the mouse cerebellar cortex. Our findings suggest that the cerebellar PCs respond preferentially to low-frequency sensory stimulation regardless of $GABA_A$ receptor activity.

## 2. Investigations and results

### *2.1. Frequency properties of facial stimulation-evoked extracellular responses in PCs*

We used the air-puff stimulation train (10 ms, 10 pulses) at 0.25 Hz, 1 Hz, 2 Hz and 4 Hz to examine the frequency properties of facial stimulation-evoked extracellular responses in PCs. As shown in Fig. 1A, when the stimulation train was at 0.25 Hz, 1 Hz and 2 Hz, each pulse of the stimulation train induced a pause of simple spike firing in the PC. In order to understand the effect of the stimulation train on the output of PCs, we calculated the ratio of stimulation-induced pause and mean value of inter-spike interval (ISI) (time of pause/ISI). When the frequency of stimulation train was at 0.25 Hz, the time of pause/ISI value of the 10th response was $2.23 \pm 0.16$, which was similar to the value of the 1st response ($2.21 \pm 0.14$; $P = 0.85$, n = 6; Fig. 1B). When the frequency of the stimulation train was at 1 Hz, the time of pause/ISI value of the 10th response was $1.70 \pm 0.12$, which was significantly longer than the normalized ISI value ($0.99 \pm 0.02$; $P = 0.002$, n = 6), but shorter than the value of the 1st response ($2.14 \pm 0.19$; $P = 0.03$, n = 6; Fig. 1B). When the frequency of the stimulation train was at 2 Hz, the time of pause/ISI value of the 10th response was $1.32 \pm 0.10$, which was significantly longer than the normalized ISI value ($1.00 \pm 0.03$;; $P = 0.01$; n = 6), but significantly shorter than the ratio of the 1st response ($2.20 \pm 0.22$; $P = 0.026$, n = 6; Fig. 1B). When the frequency of the stimulation train was at 4 Hz, the time of pause/ISI value of the 10th response was $1.01 \pm 0.10$ (n = 6), which was significantly shorter than the value of the 1st response ($2.14 \pm 0.16$; $P = 0.002$, n = 6; Fig. 1B), but was not significantly longer than the normalized ISI value ($1.01 \pm 0.01$; $P = 0.83$, n = 6). These data indicated that the output of cerebellar PCs could be significantly affected by all pulses of the low-frequency (< 4 Hz) sensory stimulation train, but only by the 1st and 2nd pulses of the high-frequency ($\geq$ 4 Hz) sensory stimulation train. This suggests that the cerebellar PCs generate sensory-related output that is limited to low-frequency sensory stimulation.

### *2.2. Effects of $GABA_A$ receptor antagonist on facial stimulation train evoked responses in PCs*

We first tested the effects of gabazine (20 $\mu$M) on 1 Hz facial stimulation (10 ms, 10 pulses)-evoked response in PCs. Each pulse of 1 Hz facial stimulation evoked pauses of simple spike firing in the PCs under control conditions (Fig. 2A, B; upper panel), but evoked simple spike firing in the PCs in the presence of gabazine (Fig. 2A, B; lower panel). In the presence of gabazine, the 1st pulse of stimulation train increased the simple spike firing to $42.1 \pm 7.8$ Hz, which was significantly higher than the baseline ($19.6 \pm 2.1$ Hz; n = 6; $P = 0.002$); the 10th pulse of stimulation train increased the simple spike firing to $32.0 \pm 5.4$ Hz, which was significantly higher than the baseline ($19.6 \pm 2.1$ Hz; n = 6; $P = 0.016$; Fig. 2B, lower panel). Consistent with our previous studies (Chu et al. 2011a,b), the present results showed that 10 pulses of 1 Hz facial stimulation train induced inhibition of PC simple spike firing, but the stimulation train evoked simple spike firing in the presence of a $GABA_A$ receptor antagonist.

Moreover, we examined the effects of gabazine on 4 Hz facial stimulation (10 ms, 10 pulses) evoked responses in PCs. Under control conditions, with exception of the 1st and 2nd pulses of 4 Hz stimulation-that induced inhibition of PCs, the other pulses of stimulation had less effect on the output of the PCs (Fig. 3A, B; upper panel). With blockade of $GABA_A$ receptor activity, the 1st and 2nd pulses of 4 Hz stimulation induced an increase in simple spike firing of the PCs, but the other pulses of stimulation still did not significantly affect the output of the PCs (Fig. 3A, B; lower panel). These results indicated that $GABA_A$ receptor-mediated inhibition did not significantly affect output of cerebellar PCs during sensory information processing.

## 3. Discussion

The sensory information from mossy fibers transfers through the granule cell layer and molecular layer, and results in patterns of PC output spikes in the cerebellum. In the present study, we demonstrated the dynamic characterization of sensory stimulation-evoked extracellular response in PCs. We found that PCs respond preferentially to low-frequency sensory stimulation independent of $GABA_A$ receptor activity.

Cerebellar PCs are the most investigated neurons in the mammalian cerebellum, and are the focus of computation: receiving convergent projections from all other cortical neurons and providing the sole output from the cerebellar cortex (Palay and Chan-Palay 1974). Both the mossy fibers and the climbing fibers are considered to transfer sensory information to the cerebellar cortex (Armstrong and Drew 1980; Bower and Woolston 1983; Cook and Wiesendanger 1976; Palay and Chan-Palay 1974; Shambes et al. 1978). Our previous results showed that facial stimulation of the ipsilateral whisker pad failed to evoke complex spikes and simple spike firing, but induced a $GABA_A$ receptor-mediated pause of spike firing in PCs via the mossy fiber-granule cell pathway in the cerebellar cortex folium Crus II. This suggested that facial stimulation influences the output of PCs (Chu et al. 2011a). In this study, we examined the effects of a facial stimulation train at 0.25–4 Hz on the output of PCs, and addressed the frequency properties of cerebellar PCs in response to sensory stimulation. Our results showed that with frequencies of the stimulation train (10-pulse) at 0.25–2 Hz, the time of pause/ISI value of the ninth response was significantly longer than the normalized ISI value, indicating that each pulse of stimulation train induced a pause of simple spike firing of the PC. However, when the frequency of the stimulation train was at 4 Hz, with the exception of the 1st and 2nd pulses, the other pulses did not induce a significant pause of simple spike firing of the PC. The present results indicate that frequencies of sensory stimulation train $\geq$ 4 Hz significantly influence the output of PCs (except the 1st and 2nd pulses), suggesting that cerebellar PCs generate sensory-related output that is limited to low-frequency sensory stimulation in the intact cerebellar cortex.

Our previous results showed that facial stimulation evokes spike firing in PCs in the absence of GABAergic inhibition (Chu et al. 2011a, b). Our results showed that blockade of $GABA_A$ receptor activity failed to improve the frequency properties of PCs in

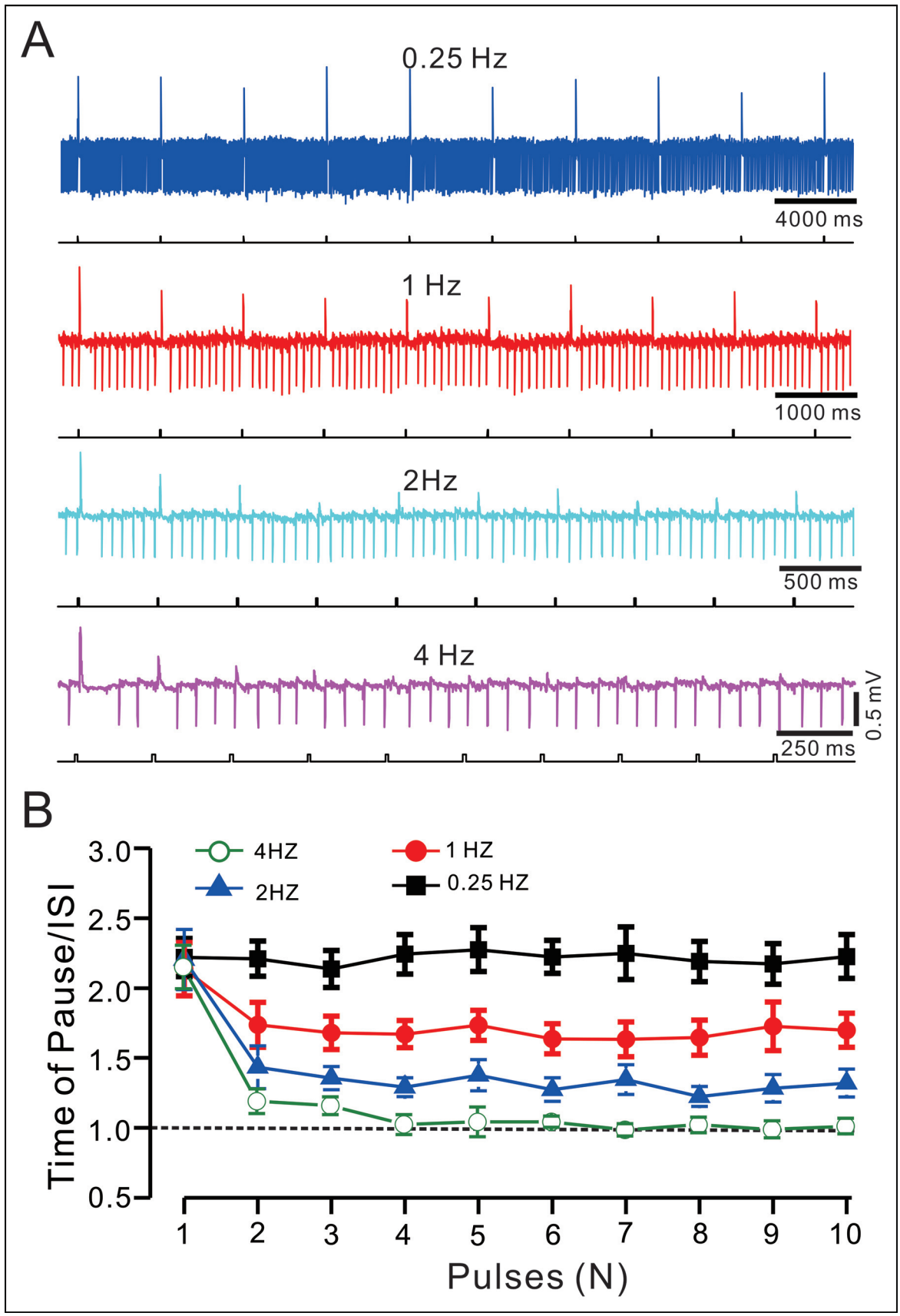


Fig. 1: Extracellular recordings showing the frequency properties of facial stimulation evoked responses in PCs. (A) Representative extracellular recordings showing the responses of PC evoked by 0.25 Hz, 1 Hz, 2 Hz, 4 Hz facial stimulation (10 ms, 10 pulses). Note that with exception of the 1st and 2nd pulses, the residuary pulses of 4 Hz stimulation train induced less effect on the spontaneous simple spike firing of PC. (B) Summary of normalized amplitude of the responses evoked by 10 pulses stimulation at 0.25 Hz (black; n = 7), 1 Hz (red; n = 7), 2 Hz (red; n = 6), and 4 Hz (blue; n = 6). Error bars indicate SEM.

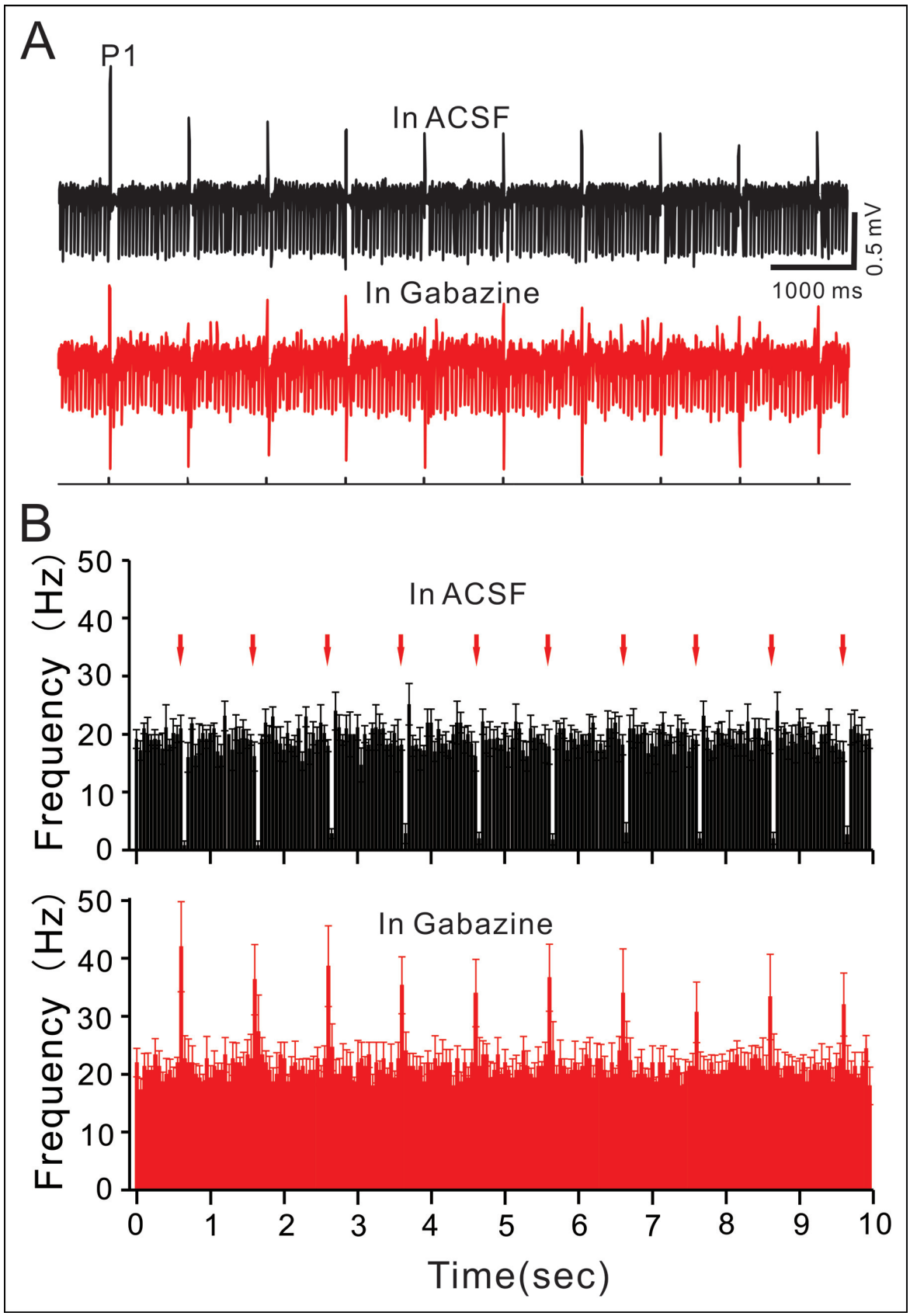


Fig. 2: Effect of GABAA receptor antagonist on 1 Hz facial stimulation evoked responses in PCs. (A) Representative extracellular recording traces showing the responses of PC evoked by 1 Hz facial stimulation (10 ms, 10 pulses) in ACSF (upper) and in the presence of gabazine (20 μM; lower). (B) Summary of data showing the effect of 1 Hz facial stimulation (arrows) on the time course of PC simple spike firing rate in ACSF (upper) and with gabazine (lower). Note that gabazine blocked 1 Hz facial stimulation-induced inhibition of simple spike firing, and revealed the stimulation-evoked simple spike firing. Error bars indicate ± SEM.

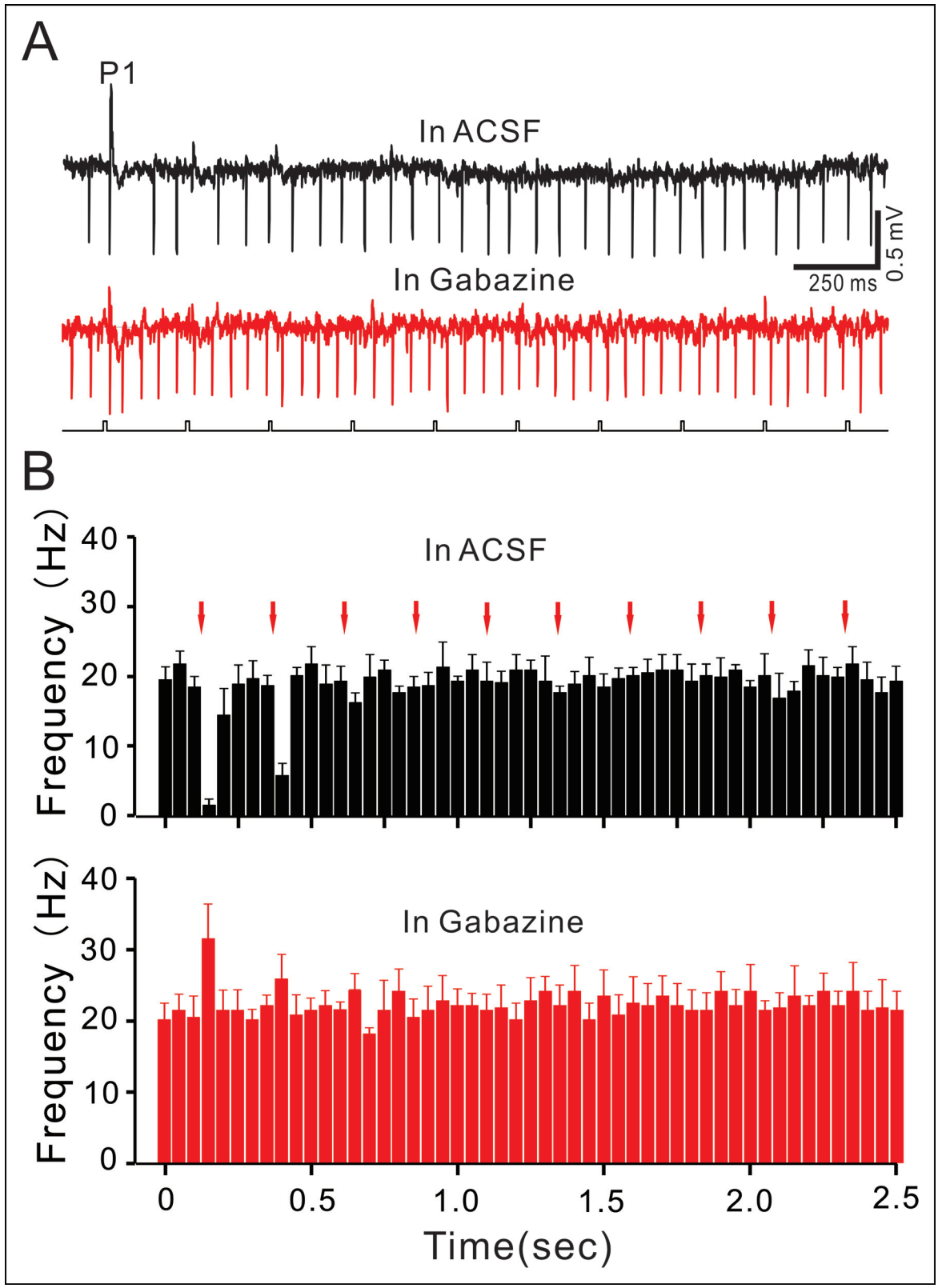


Fig. 3: Effects of GABAA receptor antagonist on 4 Hz facial stimulation evoked responses in PCs. (A) Representative extracellular recording traces showing the responses of PC evoked by 4 Hz facial stimulation (10 ms, 10 pulses) in ACSF (upper) and in the presence of gabazine (20 μM; lower). (B) Summary of data showing the effect of 4 Hz facial stimulation (arrows) on the time course of PC simple spike firing rate in ACSF (upper) and with gabazine (lower). Note that only the 1st and 2nd pulses of 4 Hz facial stimulation evoked simple spike firing of PC in the presence of gabazine. Error bars indicate ± SEM.

response to the sensory stimulation train. Instead of pauses of simple spike firing, each pulse of the stimulation train (10-pulse) at 0.25–2 Hz induced simple spike firing of the PC. With blockade of $GABA_A$ receptor activity, the first and second pulses of 4 Hz stimulation induced an increase in simple spike firing of the PCs, but the other pulses of stimulation did not affect the outputs of the PCs. These results suggest that the cerebellar PCs generate sensory-related output that is limited to low-frequency sensory stimulation, and regardless of $GABA_A$ receptor-mediated inhibition.

## 4. Experimental

Experimental procedures were approved by the Animal Care and Use Committee of Jilin University and were performed in accordance with the animal welfare guidelines of the National Institutes of Health (permit no. SYXK(Ji)2007-0011). The anesthesia and surgical procedures have been described previously (Chu et al. 2011a). In brief, 42 adult (6–8-week-old) ICR mice were anesthetized with urethane (1.3 g/kg body weight, intraperitoneal injection). Mice were tracheotomized to avoid respiratory obstruction, and fixed on a custom-made stereotaxic frame. After a watertight chamber was created, a 1-1.5 mm craniotomy was drilled to expose the cerebellar surface corresponding to Crus II. The brain surface was constantly superfused with oxygenated ACSF (mM: 125 NaCl, 3 KCl, 1 $MgSO_4$, 2 $CaCl_2$, 1 $NaH_2PO_4$, 25 $NaHCO_3$, and 10 D-glucose) with a peristaltic pump (Gilson Minipulse 3; Villiers, Le Bel, France) at 0.4 ml/min. Rectal temperature was monitored and maintained at $37.0 \pm 0.2\,^{\circ}C$ by body temperature equipment. Extracellular recording from PCs were performed with an Axopatch-200B amplifier (Molecular Devices, Foster City, CA). The recording traces were acquired through a Digidata 1440 series analog-to-digital interface on a personal computer using Clampex 10.3 software. Recording electrodes were filled with ACSF, with resistances of 3-5 MΩ. Tactile stimulation on the ipsilateral whisker pad was performed by air-puff (10-500 ms, 50-60 psi) through a 12-gauge stainless steel tube connected to a pressurized injection system (Picospritzer® III; Parker Hannifin Co., Pine Brook, NJ). The picospritzer was synchronized with electrophysiology recording *via* a Master 8 controller (A.M.P.I.) and Clampex 10.3 software (Molecular Device, Foster City, CA, USA). All the air-puff stimulation protocols were edited using Master 8 controller, and delivered at 0.05 Hz. To examine the frequency properties of the sensory stimulation-evoked responses in granule cell layer, molecular layer and PCs, we prepared stimulation trains (10-pulse) at 0.25 Hz, 1 Hz, 2 Hz, 4 Hz, 8 Hz and 33 Hz (10 ms, 50-60 psi). Gabazine (SR95531) was added to the ACSF, and applied onto the cerebellar surface at 0.4 ml/min. Gabazine hydrobromide (6-imino-3-(4-methoxyphenyl)-1 (6H)-pyridazinebutanoic acid hydrobromide) was purchased from Sigma (Sigma Shanghai, China). Electrophysiological data were analyzed using Clampfit 10.3 software (Molecular Devices, Foster City, CA). All values are expressed as the mean ± SEM, and differences were evaluated by the Student's paired *t*-test or one-way ANOVA using SPSS (Chicago, IL) software. *P* values below 0.05 were considered to indicate a statistically significant difference between experimental groups.

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