

Enhanced Somatosensory Inhibition Sharpens Hand Representation and Sensorimotor Skills in Pianists

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Dexterous motor skills, like those needed for playing musical instruments and sports, require the somatosensory system to accurately and rapidly process somatosensory information from multiple body parts. This is challenging due to the convergence of afferent inputs from different body parts into a single neuron and the overlapping representation of neighboring body parts in the somatosensory cortices. How do trained individuals, such as pianists and athletes, manage this? Here, a series of five experiments with pianists and nonmusicians (female and male) shows that pianists have enhanced inhibitory function in the somatosensory system, which isolates the processing of somatosensory afferent inputs from each finger. This inhibitory function was assessed using a paired-pulse paradigm of somatosensory evoked potentials in electroencephalography, which measures the suppressive effect of a first stimulus [i.e., conditioning stimulus (CS)] on the response to a subsequent second stimulus. We found that pianists and nonmusicians showed an inhibitory response to the sequential stimuli to the peripheral somatosensory nerve at the wrist when the CS was intense. However, only pianists exhibited an inhibitory response to a weak CS, indicating enhanced inhibitory function in pianists. Additionally, the CS increased the information content segregating individual fingers represented in the cortical activity evoked by passive finger movements and improved the perception of fast multifinger sequential movements, specifically for pianists. Our findings provide the first evidence for experience-dependent plasticity in somatosensory inhibitory function and highlight its role in the expert motor performance of pianists.

Key words: hand representation; multivariate pattern analysis; paired-pulse inhibition; pianists; somatosensory

Significance Statement

Fine motor skills, such as playing musical instruments, rely on the somatosensory system to process somatosensory information from multiple body parts. How does the somatosensory system process inputs from different body parts separately and with less interference? This study discovered enhanced inhibitory function in the somatosensory system of expert pianists, which contributes to isolating finger representation in the somatosensory processing and thereby improve the perception and execution of fast and complex multifinger movements. The present findings demonstrate that extensive musical training strengthens inhibitory processing in the somatosensory system, which underlies pianists' remarkable finger dexterity.

Introduction

Over centuries, the virtuosity of musicians and athletes has fascinated audiences. These dexterous motor skills commonly require fast and skillful control of multiple body parts. For example, pianists control bimanual finger movements with high temporal

and force precision at high speeds to produce beautiful music (Münste et al., 2002). During these movements, afferent somatosensory signals from multiple fingers are input into the nervous system within a short period, and they can interfere with each other. This is due to the architecture of the nervous system; first, cortical representation overlaps between multiple body parts in the sensorimotor cortex (Ejaz et al., 2015), and second, some sensory inputs from different body parts converge into identical neurons (Iwamura et al., 1980, 1993; Trzcinski et al., 2023). How do experts solve this problem and perform fast, complex movements with high precision? Previous studies have demonstrated that the distance of cortical representation in the somatosensory cortex (S1) between fingers can be plastically adapted through training of the use of a robotic finger (Kieliba et al., 2021), is larger in string instrument players than in untrained individuals (Elbert et al., 1995), and is smaller in musicians who have focal hand dystonia

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(Weise et al., 2012). This suggests that the increased distance of cortical body representation is one candidate mechanism to circumvent the interference of somatosensory information across different body parts. However, this idea is not fully validated since recent studies failed to replicate these findings; instead, they demonstrated unchanged cortical hand representation in the S1 in pianists (Ogawa et al., 2019) and dystonia patients compared with healthy participants (Sadnicka et al., 2022).

Another possibility is the enhancement of inhibitory functions in the somatosensory system. The inhibitory functions in the somatosensory cortices play roles in isolating somatosensory information in both spatial and temporal dimensions. A paired-pulse paradigm of somatosensory evoked potentials (SEPs) is a standard method to investigate the inhibitory functions in the somatosensory processes. This paradigm assesses the suppressive effect of a somatosensory peripheral nerve stimulus, a conditioning stimulus (CS), on cortical activity evoked by a subsequent second stimulus, usually called paired-pulse suppression (PPS). Previous studies have demonstrated that PPS is reduced by aging (David-Jürgens and Dinse, 2010; Cheng and Lin, 2013) and is aberrant in patients with focal hand dystonia (Tamura et al., 2008; Antelmi et al., 2017). Thus, we hypothesized that PPS is also plastically adapted in trained individuals who can perform dexterous motor skills. However, it is an index of the amount of inhibition induced by the CS but does not provide information about how the inhibitory activities modulate information processed in the somatosensory system. Multivariate pattern analysis is a strong tool for examining information content represented in cortical activities by quantifying the distance of cortical activity patterns between the two conditions. In the present study, we combined the paired-pulse paradigm, multivariate pattern analysis, and exoskeleton robot to examine how inhibitory activity in the somatosensory system modulates subsequent somatosensory information processing. Specifically, the CS to a somatosensory peripheral nerve was paired with passive finger movements to examine how inhibitory activity modulates the distance of cortical activation patterns evoked by passive movements between fingers.

Pianists are a unique population suitable for addressing the learning-dependent adaptation of inhibitory function in the somatosensory system. Playing the piano requires high temporal accuracy of movements with highly coordinated and independent control of multiple digits (Münste et al., 2002; Altenmüller and Furuya, 2016), and pianists utilize somatosensory information for fine control of finger movements (Hirano et al., 2020a,b; Hirano and Furuya, 2024). Here, we examined the experience-dependent plasticity of somatosensory inhibitory functions by comparing neurophysiological and behavioral indices between pianists and musically untrained individuals. A series of neurophysiological and behavioral experiments, with both univariate and multivariate analyses, revealed firstly that pianists have enhanced somatosensory inhibitory function that sharpens the cortical somatosensory representation of fingers and secondly that the activation of this enhanced inhibitory function improved the perception and execution of fast and complex finger movements by pianists.

Materials and Methods

Participants

A total of 54 healthy pianists (several pianists participated in different experiments; one participated in four experiments, four participated in three experiments, and six participated in two experiments) and 57 healthy nonmusicians participated in this study. All pianists majored in piano performance at a musical conservatory and/or had extensive and continuous

private piano training under the supervision of professional pianists and/or piano professors. Nonmusicians had no experience practicing playing any musical instrument for >5 years. All participants gave their written informed consent before participating in the experiments. All experimental procedures were carried out in accordance with the Declaration of Helsinki and were approved by the ethics committees of Sony.

Procedure

Experiment 1. This experiment aimed to assess the validity of recruiting pianists in this study as a model with a superior ability to perceive fast multifinger movements. Fifteen expert pianists [23.1 ± 3.6 years old (mean \pm SD), 11 females] and 15 nonmusicians [24.2 ± 3.1 years old (mean \pm SD), 9 females] participated in Experiment 1 and were asked to perform a perceptual test. They wore an exoskeleton robot hand (Furuya et al., 2023) on the right hand during this test. The robot hand can produce flexion and extension movements on the metacarpophalangeal joint of five digits. In this test, the second to fifth digits were passively flexed from 0 to 45° at a constant speed of 450 degree/s with a randomized order except for the order of 2-3-4-5 and 5-4-3-2 (i.e., one of the 22 possible sequences was randomly chosen; 2, index finger; 3, middle finger; 4, ring finger; 5, little finger). Participants were asked to identify the order of the passive finger movements and to respond to the perceived order by typing on a keyboard with their left hand using the same fingering as the perceived order. The time interval between the onsets of each finger flexion in the sequential passive movement varied across trials, which was determined using an adaptive Bayesian staircase method (King-Smith et al., 1994) to assess the perceptual threshold at which a participant was able to identify the order of a sequence of passive finger movements with 80% accuracy. We first specified a uniform distribution as the prior knowledge for the probability of thresholds and the Weibull function with a slope factor of 3.5 as the psychometric function. This slope factor was commonly used in previous studies, and the error between the actual and estimated values did not significantly affect the accuracy of the threshold estimate (Madigan and Williams, 1987). The probability density function was updated in each trial based on the discrimination result, the stimulus intensity of that trial, and the psychometric function. The stimulus intensity and the final threshold estimate were chosen as the mean of the latest probability density function. In this test, the chance level was 1/22 because the sequence on a given trial was randomly selected from 22 possible sequences. Therefore, we set the gamma value in the Weibull function to this chance level. The test consisted of 50 trials.

Experiment 2. This experiment was conducted to test whether the inhibitory function in the somatosensory system, as assessed by the paired-pulse paradigm of SEP, differs between pianists and nonmusicians and, if so, whether the ulnar or median nerve is responsible for the group difference of the inhibitory function. The two nerves innervate the different muscles and tactile regions in the hand. The ulnar nerve distal to the wrist mainly innervates the intrinsic hand muscles of four fingers (second to fifth digits), which means that the proprioceptive afferents from the muscles involved in the movement of the four fingers pass through the ulnar nerve. In contrast, the median nerve distal to the wrist mainly innervates the muscles involved in the thumb movement. The thumb is frequently used in daily life, whereas playing musical instruments requires the dexterous control of not only the thumb but also the other fingers. Thus, we hypothesized that the amount of PPS would differ between the two groups when tested through the ulnar nerve rather than the median nerve. Fourteen expert pianists [22.5 ± 3.4 years old (mean \pm SD), 12 females] and 14 nonmusicians [22.3 ± 1.7 years old (mean \pm SD), 11 females] participated in this experiment.

EEG recording and preprocessing

Electroencephalography (EEG) signals were recorded from the CP3 electrode according to the international 10/20 system (actiChamp plus and actiCAP snap, Brain Products; impedance <10 k Ω). The reference electrode was placed at Fz, and the sampling rate was 2.5 kHz. The off-line analysis was performed using EEGLAB (Delorme and Makeig, 2004) and a custom-made MATLAB script. For off-line analysis, EEG signals

were preprocessed by bandpass filtering (0.5–300 Hz) and line noise removal (EEGLAB cleanLineNoise function).

Electrical stimulation

An electrical stimulus (a rectangular pulse of 1 ms duration) was applied to the median and ulnar nerves at the wrist using a constant current stimulator (DS4, Digitimer) via a paired bar-type electrode to evoke the SEP. Before the experiment, we assessed the perceptual detection threshold of electrical stimulation for both nerves. The detection threshold was the lowest electrical current at which a participant reported sensation. Then, we determined the motor threshold, the lowest electrical current that evokes a visible twitch of muscles innervated by each of the ulnar and median nerves (abductor pollicis brevis for the median nerve, first dorsal interosseous muscle for the ulnar nerve).

Paired-pulse paradigm of SEPs

Two electrical stimuli were applied to the nerve in succession (Fig. 2A). We set the intensity of the first stimulus, called the CS, to either 0.95 times the perceptual detection threshold (i.e., CS_{PT} , weak intensity) or 0.95 times the motor threshold (i.e., CS_{MT} , high intensity). The second stimulus, the test stimulus (TS), was set at an intensity of 0.95 times the motor threshold to elicit a clear SEP. The interstimulus interval (ISI) between the CS and TS was set at 5, 10, 20, 30, 40, and 50 ms. In addition, there were conditions in which only the CS_{PT} or only the TS was presented. In total, EEG responses were recorded for 14 stimulus conditions (6 ISI * 2 different intensities of CS, single- CS_{PT} , and single-TS) for each of the median and ulnar nerve conditions. Each stimulus condition was repeated 250 times at a rate of 2.5 Hz. The different ISIs and intensities were presented stimulus-by-stimulus in a randomized order in both nerves.

The preprocessed EEG data were segmented in epochs –50 to 100 ms relative to the stimulus onset, and segments containing artifacts were removed (EEGLAB functions `pop_jointprob.m`, `pop_rejkurt.m`; both SD, 3; amplitude during prestimulus period >50 μ V). The segmented data were then superimposed to increase the signal-to-noise ratio. For the paired-pulse SEP, potentials evoked by the CS may overlap in time with potentials evoked by the subsequent TS. To remove this overlap, we subtracted the SEP waveform obtained in the CS-only condition from that obtained in the paired-pulse conditions. Specifically, the SEP waveform in the single- CS_{PT} condition was subtracted from that obtained in the paired-pulse conditions with CS_{PT} after adjusting for stimulus onset timing, and the SEP waveform in the paired-pulse conditions with CS_{MT} was subtracted from that obtained in the single-TS condition. We then determined that the negative peak component appeared ~20 ms after the stimulus onset and that the positive peak appeared ~25 ms after the stimulus onset and calculated the peak-to-peak amplitude between the two components. We calculated the difference in the peak-to-peak amplitude of the SEP between the paired-pulse and single-TS conditions to quantify the effect of a preceding CS on somatosensory processes evoked by a subsequent TS.

Experiment 3. This experiment tested the replicability of the group difference in the PPS observed in the former experiment and examined the functional role of the inhibitory effect, which can be activated by the ulnar nerve stimulation, on the perception of fast sequential finger movements. Fourteen expert pianists [22.6 ± 1.8 years old (mean \pm SD), 11 females] and 14 nonmusicians [22.4 ± 1.9 years old (mean \pm SD), 11 females] participated in Experiment 3 consisting of the SEP assessment and a perception test. The PPS in the CS_{PT} condition was assessed using the same procedure and equipment as in Experiment 2. During the perception test, participants wore the exoskeleton robot hand (Furuya et al., 2023) on their right hand. In this test, the finger joints of the robot hand were passively flexed with one of the predetermined sequences (1-3-2-5 or 1-3-5-2; 1, thumb; 2, index finger; 3, middle finger; 5, little finger) at a constant angular velocity of 450 degrees/s. After the passive movements, the participants were asked to press one of the two buttons put in front of them to identify the sequence in which their fingers were moved by the robot hand (i.e., a two-alternative forced choice task). The time interval

between the onsets of each finger flexion in the sequential passive movement varied across trials, which was determined using the adaptive Bayesian staircase method (King-Smith et al., 1994), to quantify the discrimination threshold at which a participant was able to discriminate the order of a sequence of passive finger movements with 80% accuracy. There were two conditions in this experiment. In the first condition, a weak electrical pulse (intensity just below the perceptual detection threshold, 1 ms square pulse) was applied to the ulnar nerve at the onset of each passive finger flexion using the constant current stimulator via a bar-type electrode (i.e., the stimulation was applied four times in a single passive sequential movement as the sequence used in this test consisted of four successive finger flexions). In the second condition, no electrical stimulation was provided. The two conditions appeared randomly in a trial-by-trial manner, and the corresponding probability density function was updated based on the correctness of the response. Each condition consisted of 75 trials.

Experiment 4. This experiment examined the effect of the inhibitory effect activated by the conditioning ulnar nerve stimulation on the distance in the cortical activity between the hand digits. Fourteen pianists [22.0 ± 2.2 years old (mean \pm SD), 10 females] and 14 nonmusicians [23.8 ± 3.4 years old (mean \pm SD), 8 females] participated in Experiment 4. In this experiment, we recorded the EEG signals during passive finger movements.

Passive finger movements

One of the five metacarpophalangeal joints of the right hand was flexed from 0 to 45° at a constant speed of 450 degrees/s using the exoskeleton robot hand. The same finger was extended from 45 to 0° at the same angular velocity 500 ms after flexion. In addition, there was another condition in which both the ring and little fingers were flexed simultaneously (i.e., dual condition). The time interval between successive finger flexions was randomly determined from 2 to 3 s. The number of passive movements was 200 for each digit and dual condition. In half of the trials, a weak electrical pulse (intensity just below the perceptual detection threshold, 1 ms square pulse) was applied to the ulnar nerve at the wrist at the onset of each passive flexion (i.e., ES condition) using the constant current stimulator via a paired bar-type electrode. In contrast, no electrical stimulation was applied in the remaining trials (i.e., noES condition). To avoid the contamination of artifacts originating from eye movements in EEG data, we asked participants to close their eyes and rest during the EEG recording.

EEG recording and preprocessing

EEG signals were recorded with 64 active electrodes according to the international 10/20 system (actiCHamp plus and actiCAP snap, Brain Products; impedance <10 k Ω). The reference electrode was placed at Fz, and the sampling rate was 1 kHz. The off-line analysis was performed using EEGLAB and a custom-made script using MATLAB. For off-line analysis, EEG signals were preprocessed by downsampling (250 Hz), low-cut filter (0.5 Hz), line noise removal (EEGLAB cleanLineNoise function), bad channel rejection, artifact subspace reconstruction (EEGLAB `pop_clean_rawdata` function, burst criterion, 40; window criterion, 0.25; burst rejection, on), channel interpolation, rereference to the average, and independent component analysis (ICA) with the Picard algorithm. Components extracted from the ICA were classified using a machine learning algorithm implemented in the EEGLAB `IClabel` function (Pion-Tonachini et al., 2019). ICA components classified with <5% probability of brain activity and >50% probability of muscle activity were removed from the data.

The preprocessed EEG signals were segmented according to the onset of passive finger flexion, which was extended from –200 to 400 ms and then averaged across trials to obtain the SEP. To estimate the equivalent current dipole location of the SEP, we performed a dipole source modeling that was implemented in EEGLAB (i.e., `dipfit_erppeeg` function). Because we did not record the actual position of EEG electrodes and individual MRI data on the head structure, we used the group-averaged SEP data and a boundary-element model as a head model for this analysis.

Multivariate pattern analysis

We performed the multivariate pattern analysis where the pairwise dissimilarity distance of multichannel EEG signals between individual digits was calculated for each sample of the target time window. The preprocessed EEG signals were segmented by the onset of passive finger flexion, which was extended from -200 to 400 ms. We randomly divided the segments of each finger into 10 pseudoblocks (i.e., each block contained a maximum of 10 segments per condition). We then estimated the SEP for each finger and condition in each pseudoblock using a linear model consisting of the finger (i.e., five fingers), condition (i.e., noES or ES), and their interaction as fixed effects. The model also includes the mean amplitude over the baseline period (i.e., -200 to 0 ms) and the interactions between the baseline and finger and between the baseline and condition as covariates (Alday, 2019). The estimated SEP amplitudes at each sample in each pseudoblock were whitened by the covariance of the residuals of the linear model (i.e., multivariate noise normalization) to minimize the effects of noise on the dissimilarity distance. The dissimilarity values of the prewhitened channel-wise SEP amplitude patterns between all possible pairs of digits for each sample were measured using the cross-validated squared Euclidean distance (this is the same distance measure as the crossnobis distance reported in the previous study; Walther et al., 2016). The dissimilarity of a sample t between digits i and j (i.e., d_{ij}^t) was obtained as follows:

$$d_{ij}^t = \frac{1}{M(M-1)} \sum_m \sum_{n \neq m} (\beta_{i,m}^t - \beta_{j,m}^t)(\beta_{i,n}^t - \beta_{j,n}^t)^T / P,$$

where M is the number of pseudoblocks (10 in this experiment), $\beta_{i,m}^t$ represents the prewhitened channel-wise SEP amplitude pattern evoked by the i th digit movement of sample t in the m th pseudoblock, and P is the number of sensors. This analysis was repeated for every sample and digit pair. Furthermore, this procedure was repeated 20 times by randomly reassigning the segments into 10 pseudoblocks, and the resultant 20 dissimilarity time courses for each digit pair were averaged. The multivariate noise normalization and the cross-validated squared Euclidean distance were calculated according to previous studies (Walther et al., 2016; Ritchie et al., 2021). Due to the cross-validation procedure, the expected value of the distance was zero (or less) if two patterns were not significantly different from each other and greater than zero if the two patterns were different. Finally, the dissimilarity time courses were averaged over all pairs of digits.

We performed the cluster permutation test to assess the interactive effect between the group and condition (i.e., ES and noES conditions) on the average dissimilarity time course. First, we calculated the average dissimilarity time course difference between the ES and noES conditions for each group. A two-sample t test was then performed on the differential dissimilarities between the two groups for all samples. All samples with a $p < 0.05$ were selected. Selected samples were connected into connected sets according to temporal adjacency. Cluster-level statistics were calculated by taking the sum of the t values within each cluster. In the next step, the group label for each participant was shuffled. A two-sample t test was then performed on the differential dissimilarities between the shuffled groups for all samples. We identified the clusters and selected the cluster with the largest cluster t value. We repeated this procedure 10,000 times, and we obtained a permutation distribution. Finally, we calculated the proportion of clusters that resulted in a larger t value in the permutation distribution than the observed clusters. This proportion is the Monte Carlo significance probability, which also corresponds to a p value.

Somatosensory surround inhibition

To examine whether the surround inhibitory circuits in the somatosensory system are responsible for the effect of the CS on the behavior and interdigit dissimilarity, we assessed somatosensory surround inhibition using a dual-SEP technique. The SEP waveform evoked by the simultaneous flexion of the ring and little fingers (i.e., dual SEP) was compared with the arithmetic sum of two SEPs (i.e., sum SEP) evoked by individual passive movements of the two fingers. We focused on the CP3 electrode,

placed just above the sensorimotor area. The SEP evoked by passive movement is typically characterized by several components. A previous study demonstrated that the first component, observed at ~ 100 ms, originates from activities of the somatosensory cortex. Therefore, we quantified the peak amplitude of the first component and then compared it between the dual SEP and the sum SEP.

Experiment 5. This experiment was conducted to test whether the inhibitory activity evoked by the weak ulnar nerve stimulation enhances the production of fast and complex sequential finger movements performed by pianists. Fourteen pianists [23.0 ± 3.3 years old (mean \pm SD), 11 females] participated in this experiment. They were asked to strike piano keys using their right hand in the following order: 1-3-4-2-3-5 (1, thumb; 2, index finger; 3, middle finger; 4, ring finger; 5, little finger). They repeated the sequence production as fast and accurately as possible for 20 s. During this task, electrical pulses (1 ms square wave) with an intensity just below the PT were delivered to the right or left ulnar nerve at a frequency of 10 Hz. In the control condition, no stimulation was delivered during the task. One stimulation device (DS4, Digitimer, Inc.) was used to stimulate the ulnar nerve of the right hand, and another device (DS7A, Digitimer) was used to stimulate the ulnar nerve of the left hand. Both devices delivered electrical pulses via a pair of Ag/Cl electrodes placed on the wrist. Before the experiment, the pianists performed the task without any stimulation as a practice. They then performed the task three times for each condition in a randomized order (i.e., in a total of nine trials). The intertrial interval was set at 2 min to avoid muscle fatigue. We used a mute piano and instructed each participant to perform the task while listening to white noise through headphones worn on the ears and while the eyes closed. We calculated the number of erroneous keystrokes and the interkey-stroke interval (IKI) as performance indices.

Statistical analysis

A generalized linear mixed-effect model (GLME) implemented in the lme4 package (Bates et al., 2015) in R (<https://www.r-project.org>) was used to fit the data of each experiment. To determine the significance of the fixed effects, we performed Type 2 Wald χ^2 tests on the model using the ANOVA function implemented in the car package (Fox and Weisberg, 2019). Multiple comparisons were performed based on the estimated marginal means calculated using the emmeans package (Searle et al., 2023). The degrees of freedom of these comparisons were computed using the Kenward–Roger method, while p values were adjusted using Holm's method.

Results

Experiment 1: a superior motion perception in pianists

Figure 1 shows the perceptual threshold for discriminating the sequence order of passive finger movements obtained from the

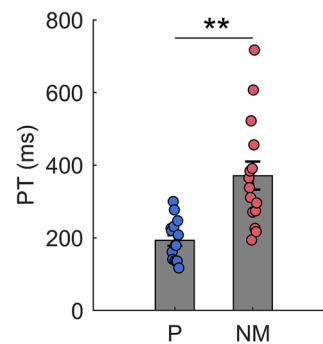


Figure 1. The perceptual threshold for identifying the order of passive multifinger movements obtained from pianists (P) and nonmusicians (NM). The vertical axis represents the timing interval between successive finger flexion motions at which a participant was able to identify the order of passive movements with 80% accuracy. Colored dots indicate data of individual participants. Mean \pm SEM. P vs NM, $**p < 0.01$.

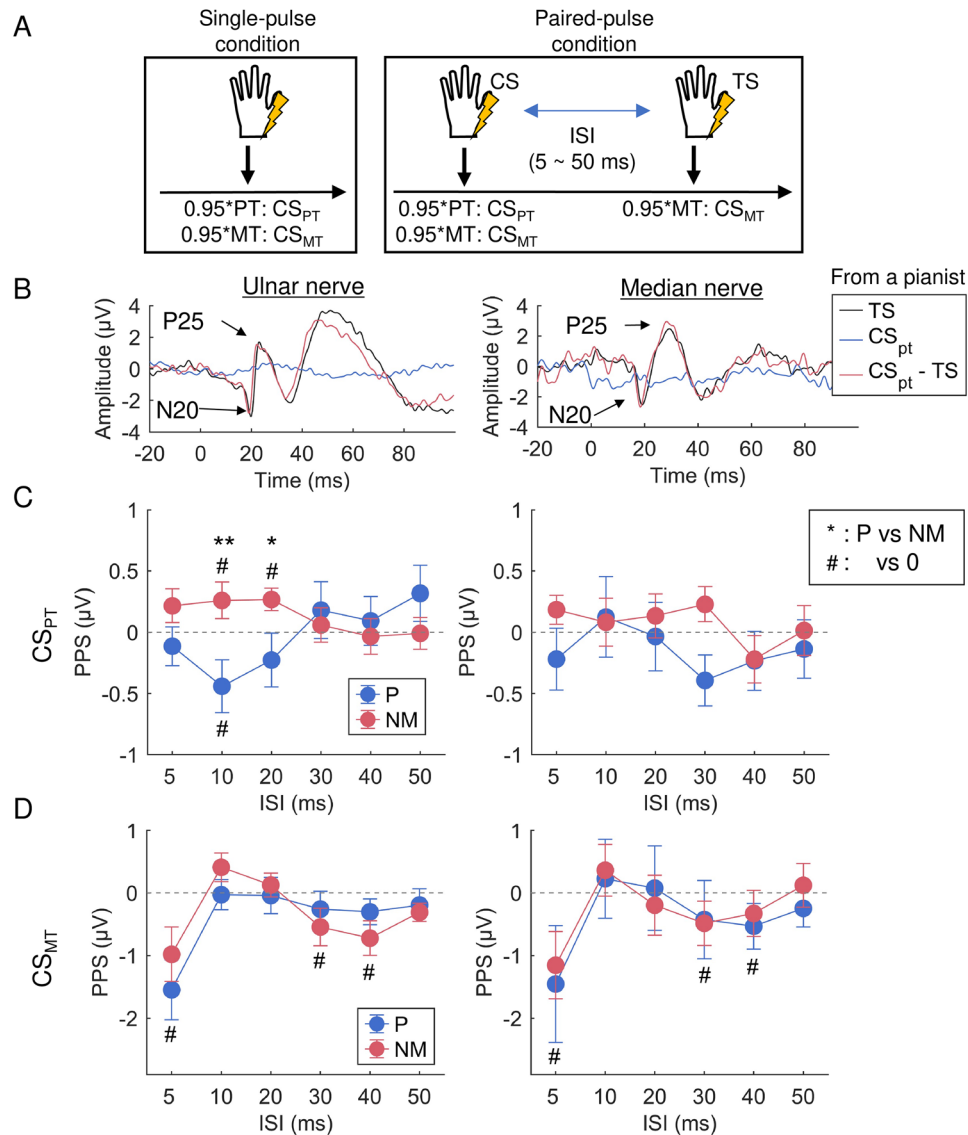


Figure 2. Effects of a leading somatosensory stimulus on the subsequent somatosensory processes. **A**, Paired-pulse paradigm of SEP. **B**, Typical SEP waveforms evoked by electrical somatosensory nerve stimulation. **C**, **D**, Effect of a CS on the N20-P25 amplitude of SEP. The horizontal axis indicates the ISI between the conditioning and test stimuli. The vertical axis represents the difference in the N20-P25 amplitude between the single-TS and paired-pulse conditions, an index of the PPS. The intensity of the CS was set just below the PT (**B**) and just below the MT (**C**). Blue and red lines represent results obtained from pianists and nonmusicians, respectively. The left and right panels represent the results obtained when the ulnar and median nerves were stimulated, respectively. Mean \pm SEM. Pianist versus nonmusician, * $p < 0.05$; ** $p < 0.01$; #, Bootstrapped 95% confidence interval of PPS does not cross 0.

pianists and nonmusicians. We found a significant difference in the threshold between the two groups ($t_{(28)} = -5.06$; $p = 2.36 \times 10^{-5}$), indicating that pianists have a superior ability to perceive the order of fast multifinger movements compared with nonmusicians.

Experiment 2: enhanced the paired-pulse inhibition in pianists

The purpose of Experiment 2 was to examine the difference in somatosensory inhibitory function between pianists and nonmusicians. Here, we conducted the paired-pulse paradigm of SEP by stimulating the ulnar and median nerves and compared the PPS between the two groups. There were no significant differences in the peak-to-peak amplitude of SEP between pianists and nonmusicians in either nerve in the single-TS condition (median, pianist = 4.84 ± 3.78 μ V; nonmusician, 3.58 ± 2.45 μ V; $t_{(26)} = 1.01$; $p = 0.32$; ulnar, pianist = 4.21 ± 2.24 μ V; nonmusician, 3.44 ± 1.99 μ V; $t_{(26)} = 0.93$; $p = 0.36$). Figure 2C illustrates the PPS

at each ISI for ulnar and median nerve stimulation in the CS_{PT} condition. The GLME model (Gaussian distribution: link, identity) revealed a significant second-order interaction effect (group*ISI*nerve, $\chi^2_{(5)} = 12.49$; $p = 0.03$) on the PPS, indicating that the effects of a weak preceding stimulation on subsequent somatosensory processes varied depending on the group, ISI, and nerve. We reanalyzed the data from the median and ulnar nerve stimulation conditions to simplify the results. For the median nerve stimulation condition, a GLME model yielded that neither the main effects nor their interaction were significant (group, $\chi^2_{(1)} = 1.02$; $p = 0.14$; ISI, $\chi^2_{(5)} = 4.28$; $p = 0.51$; interaction, $\chi^2_{(5)} = 5.09$; $p = 0.41$). In contrast, there was a significant interaction between the group and ISI effects for the ulnar nerve stimulation condition (group*ISI, $\chi^2_{(5)} = 14.84$; $p = 0.01$). Post hoc comparison revealed a significant difference in the PPS between the two groups at ISIs of 10 ms ($t = -2.81$; $p = 0.006$) and 20 ms ($t = -1.98$; $p = 0.049$). We then tested whether the suppression at each ISI

significantly differed from 0. We found that the bootstrap 95% CI of the PPS at an ISI of 10 ms did not cross the 0 in the pianists (−0.87 to −0.06) and nonmusicians (0.04–0.61). In contrast, for an ISI of 20 ms, the bootstrap 95% CI of the PPS exceeded 0 in the nonmusicians (0.08–0.42) but not in the pianists (−0.69 to 0.14). These results showed that the PPS induced by the CS_{PT} modulated subsequent somatosensory processes differently in pianists and nonmusicians. Specifically, pianists and nonmusicians showed inhibitory and facilitatory effects, respectively.

Figure 2D illustrates the effects of CS_{MT} on the N20-P25 amplitude of the SEP across different ISIs for ulnar and median nerve conditions. A GLME model (Gaussian distribution: link, identity) yielded a significant fixed effect of the ISI ($\chi^2_{(5)} = 49.37$; $p = 1.868 \times 10^{-9}$) but not the group ($\chi^2_{(1)} = 0.05$; $p = 0.82$), nerve ($\chi^2_{(1)} = 0.007$; $p = 0.94$), or their interaction effects (all, $p > 0.05$), indicating that the effect of CS_{MT} stimuli on the SEP amplitude varied across ISIs, but did not differ between groups and nerves. For both nerves, the upper bound of the bootstrap 95% CI of the PPS did not reach 0 for ISIs of 5 ms (ulnar, −1.97 to −0.74; median, −2.84 to −0.60), 30 ms (ulnar, −0.82 to −0.04; median, −1.44 to −0.02), and 40 ms (ulnar, −0.88 to −0.23; median, −1.00 to −0.04), indicating significant inhibitory effects of the preceding CS_{MT} on the subsequent somatosensory processes for these ISIs in both nerves.

These results showed that the paired-pulse paradigm successfully induced inhibitory activity in the somatosensory processing in both groups and nerves when the CS was intense (i.e., CS_{MT}). In contrast, the CS_{PT} (i.e., weak CS) inhibited the SEP evoked by the TS only in the pianists but not the nonmusicians. This was only evident in the ulnar nerve but not the median nerve.

Experiment 3: a weak CS to the ulnar nerve improves the perception of complex finger movements in pianists

Experiment 2 demonstrated that weak conditioning stimulation of the ulnar nerve differently modulates subsequent somatosensory processes between pianists and nonmusicians. However, it remains unclear whether the inhibitory/facilitatory activities induced by the conditioning stimulation simply attenuate or amplify neural activities evoked by somatosensory afferent inputs or whether it has functional roles in behaviors, specifically in the perception of finger movements. To examine this, we paired the weak conditioning stimulation to the ulnar with passive finger movements and tested the effect of the conditioning stimulation on the perception of complex sequential movements involving multiple fingers.

First, we examined the effects of weak ulnar nerve stimulation on subsequent somatosensory processing using EEG, as in Experiment 2, to test whether we could replicate the results observed in the former experiment in other participants. Figure 3A shows the PPS at different ISIs in the two groups. A GLME model (Gaussian distribution: link, identity) revealed a significant fixed effect of the group but not ISI or their interaction on the PPS (group, $\chi^2_{(1)} = 6.09$; $p = 0.01$; ISI, $\chi^2_{(5)} = 3.92$; $p = 0.56$; interaction, $\chi^2_{(5)} = 4.82$; $p = 0.44$). For the pianists, the upper bound of the 95% bootstrap CI of the PPS was smaller than 0 only for an ISI of 10 ms (95% CI, −0.63 to −0.04), but not for the other ISIs (95% CI, 5 ms, −0.35 to 0.21; 20 ms, −0.31 to 0.18; 30 ms, −0.56 to 0.12; 40 ms, −0.48 to 0.03; 50 ms, −0.43 to 0.13). In contrast, for the nonmusicians, the lower bound of the bootstrap 95% CI was >0 for an ISI of 30 ms (95% CI, 0.12–0.49), but not for the other ISIs (95% CI, 5 ms, −0.09 to 0.47; 10 ms, −0.12 to 0.27; 20 ms, −0.29 to 0.37; 40 ms, −0.01 to 0.55; 50 ms, −0.06 to 0.57). The pianists showed the inhibitory

effect of CS_{PT} at an ISI of 10 ms in Experiments 2 and 3, whereas the nonmusicians showed the facilitatory effect with different ISIs between the two experiments. We combined the data from the two experiments to further confirm the impact of weak ulnar nerve stimulation on subsequent somatosensory processing. Then, we tested whether the bootstrap CI of PPS crossed 0. As this is the third time this analysis has been performed, we corrected for the alpha level (i.e., $0.05/3 = 0.0167$) and calculated a 98.33% CI of PPS. In the combined data (i.e., sample size = 27 for each group), we found that in pianists, the bootstrap CI of the PPS at an ISI of 10 ms did not exceed 0 (−0.71 to −0.11), indicating significant inhibition. In contrast, the bootstrap CI of PPS crossed 0 in all ISIs in the nonmusicians (5 ms, −0.01 to 0.46; 10 ms, −0.03 to 0.40; 20 ms, −0.13 to 0.36; 30 ms, −0.05 to 0.36; 40 ms, −0.13 to 0.37; 50 ms, −0.09 to 0.45). This suggests that the weak ulnar nerve stimulation robustly induced inhibitory activity in the somatosensory process between the two experiments in the pianists. In contrast, no stable effect was found in the nonmusicians.

To examine the functional roles of this inhibitory activity on the perception of fast and complex finger movements, we asked participants to perform the somatosensory perception test with and without receiving the weak conditioning stimulation of the ulnar nerve. Figure 3, B and C, shows the group means of the discrimination thresholds for the two conditions in the two groups and the difference in the threshold between the two conditions in both groups, respectively. A GLME model (Gamma distribution: link = identity) yielded a significant interactive effect between the group and condition on the discrimination threshold ($\chi^2_{(1)} = 7.84$; $p = 0.005$). In most pianists, the perceptual discrimination threshold was lower in the ES condition than in the noES condition (Fig. 3C). A post hoc test revealed that the stimulation significantly reduced the discrimination threshold in pianists (z -ratio, −2.20; $p = 0.028$). In contrast, the discrimination threshold did not differ between the two conditions in the nonmusicians; rather, it tended to be larger in the ES condition than in the noES condition (ratio, 1.77; $p = 0.077$). Thus, in pianists, the inhibitory activity induced by the weak conditioning stimulation to the ulnar nerve improved the perception of fast-complex multifinger movements.

A regression analysis also showed that the PPS at an ISI of 10 ms could significantly explain the difference in the discrimination threshold between the two conditions for pianists (Fig. 3D, left; adjusted $R^2 = 0.61$; $p < 0.01$) but not for nonmusicians (Fig. 3D, right; adjusted $R^2 = -0.04$; $p = 0.48$). In contrast, we found no significant correlation between the discrimination threshold in the noES condition and the PPS at an ISI of 10 ms (pianist, $R^2 = 0.063$; $p = 0.386$; nonmusician, $R^2 = 0.003$; $p = 0.846$).

Experiment 4: a weak CS to the ulnar nerve increased the distance of cortical activity patterns between fingers and enhanced surround inhibition in pianists

Experiments 2 and 3 demonstrated that the weak conditioning stimulation to the ulnar nerve activates somatosensory inhibitory function and enhances the perception of a fast and complex multifinger movement, specifically in pianists. However, how the inhibitory activity induced by the conditioning stimulation enhances the perception remains unclear. Here, we combined the paired-pulse paradigm and MVPA to test the hypothesis that the CS spatially sharpens the representation of digits in the somatosensory processes. Previous fMRI studies demonstrated that passive finger movements evoke unique spatial activity patterns in the S1 for each digit (Arbuckle et al., 2022; Sanders

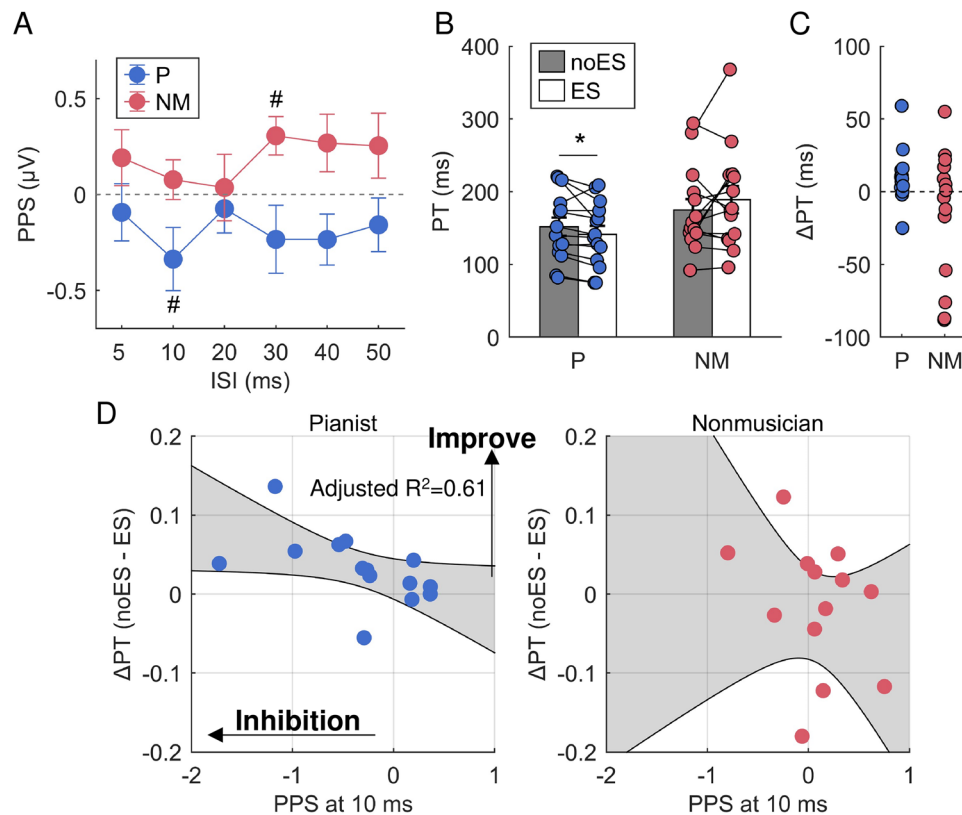


Figure 3. Effects of weak ulnar nerve stimulation on somatosensory perception. **A**, PPS assessed by paired-pulse SEP with weak ulnar nerve stimulation. Pianist versus nonmusician, $*p < 0.05$. **B**, Effect of weak ulnar nerve stimulation on somatosensory discrimination perception. The vertical axis represents the timing interval between each finger flexion at which a participant was able to discriminate the order of a sequence of passive finger movements with 80% accuracy. The left and right show the data obtained from pianists (P) and nonmusicians (NM). Colored thin lines indicate individual data. The filled box represents the discrimination threshold when no electrical stimulation was provided, whereas the white box represents the threshold when weak ulnar nerve stimulation was provided in the test. Mean \pm SEM. With ES versus without ES, $*p < 0.05$. **C**, The difference in the discrimination threshold between the ES and noES conditions. Each dot represents individual data. **D**, The correlation between the change in the discrimination threshold by the provision of ulnar nerve stimulation and the PPS at the ISI of 10 ms in pianists (left) and nonmusicians (right). The shaded area represents the 95% confidence interval. Note that the ΔPT was log-transformed.

et al., 2023). The uniqueness of the cortical activity was assessed using the MVPA, which calculates the dissimilarity of a pattern of multichannel cortical activity for all pairs of fingers. Based on this observation, in this experiment, we quantified the dissimilarity in the scalp distribution patterns of the SEP amplitude evoked by passive finger movements between digits and examined the effect of weak ulnar nerve stimulation on it. Figure 4 shows the group-averaged waveforms and topographic maps of SEP evoked by the passive movement of each digit. The dipole source localization analysis on the averaged data identified that the estimated dipole locations for the early components (i.e., latency, 80, 124 ms) were around the sensorimotor cortex (Fig. 4B), whereas the later component was around the vertex.

Figure 5A shows the group mean time course of the interdigit dissimilarity (i.e., crossnobis distance) of the SEP distribution patterns across 64 channels for each condition in pianists and nonmusicians. The interdigit dissimilarity increased ~ 60 ms after the onset of passive flexion. Interestingly, weak ulnar nerve stimulation increased the interfinger dissimilarity in the pianists but not nonmusicians. The cluster permutation test for interactive effects between the group and condition factors revealed a cluster that showed a significant interactive effect that extended from 108 to 132 ms after the onset of passive flexion (cluster $t = 20.09$; $p = 0.02$). We found that the interdigit dissimilarity was higher in the ES condition than in the noES condition for pianists but not for nonmusicians. Figure 5B shows the

representational dissimilarity matrix consisting of the dissimilarity values of all possible pairs of digits and conditions observed at 108–132 ms after the onset of passive flexion. To visualize this, we applied classical multidimensional scaling to the group-averaged representational dissimilarity matrix. Figure 5C shows the two-dimensional map of the relationship of dissimilarity of spatial SEP amplitude patterns across digits. This map shows the enlarged distance of somatosensory representation between digits in the ES condition in pianists. We further calculated dissimilarity values on a channel-by-channel basis using only adjacent channels to confirm the cortical origin of the interdigit dissimilarity. The adjacent channels were determined based on the Euclidean distance of the 3D layout of the electrodes. The dissimilarity values were averaged across samples between 108 and 132 ms and across participants, and then the resultant values were used to create a topographic map (Fig. 5D). This analysis demonstrated that the dissimilarity value was highest around the CP3 electrode, above the sensorimotor cortex contralateral to the stimulated hand. These results indicate that the inhibitory activity evoked by the weak ulnar nerve stimulation sharpens the individual finger representation in the early somatosensory process, specifically for pianists.

The former analysis revealed that the weak CS to the ulnar nerve increased the uniqueness of the cortical activity patterns of passive movements for each digit. The possible underlying mechanism is surround inhibition, in which somatosensory

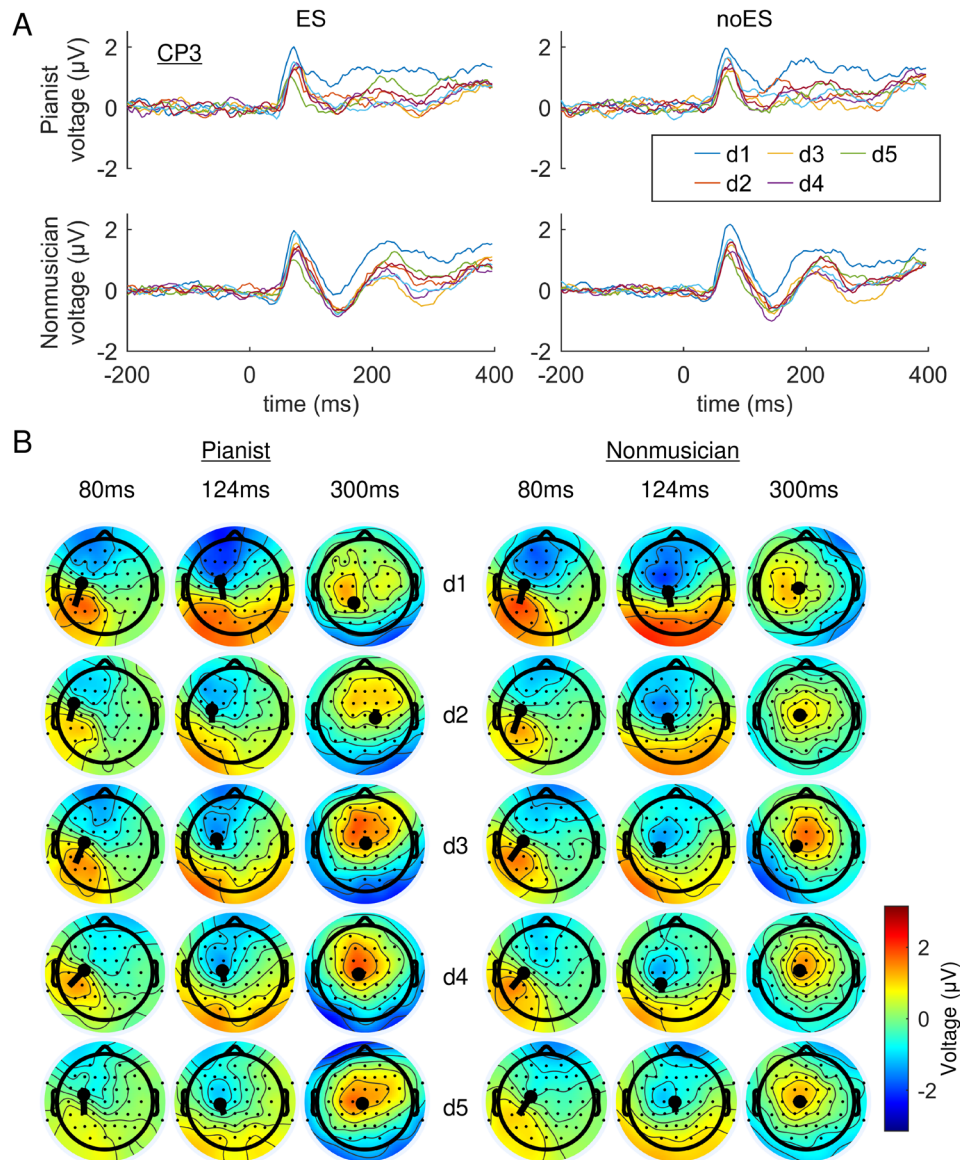


Figure 4. SEP waveform evoked by the passive finger movements and its topographical map. **A**, Group-averaged SEP waveforms at the CP3 electrode evoked by passive flexion of individual digits. The left and right panels show the SEP in the ES and noES conditions. **B**, The topographical map of the grand-averaged SEP at 80, 124, and 300 ms latency. The black line on each panel represents the estimated dipole location of each component.

inputs from one body part inhibit the excitatory activities induced by inputs from neighboring body parts (Gandevia et al., 1983; Tinazzi et al., 2000). It has been shown that simultaneous stimulation of two independent fingers (or nerves) suppresses the amplitude of the SEP compared with the arithmetic sum of the individual SEPs. This suppressive effect is an index of surround inhibition. In the present study, we compared the SEP amplitude evoked by single-finger movement and the simultaneous movement of two fingers. Figure 6A shows the SEP waveforms from around the somatosensory area evoked by the passive flexion of the ring and little fingers from one representative pianist and nonmusician. The right panels show the SEPs when a weak CS to the ulnar nerve was paired with the onset of passive movements, and the left panels show the SEPs without the conditioning stimulation. The yellow lines represent an arithmetic sum of the two individual SEPs (i.e., sum SEP), and the purple lines indicate the SEP evoked by the simultaneous passive flexion of the ring and little fingers (i.e., dual SEP). Figure 6B

shows the amplitude of the first component (i.e., P1 component) of the sum SEP, dual SEP, and the difference between the sum and dual SEPs in both groups. We found a significant interactive effect between the group and condition factors on the difference in the P1 amplitude between the sum and dual SEPs (GLME, Gaussian distribution: link, identity; group * condition, $\chi^2_{(1)} = 4.34$; $p = 0.037$). A post hoc test revealed that the difference in the P1 amplitude between the sum and dual SEPs was larger in the stimulation condition than in the no-stimulation condition in pianists ($t = -2.14$; $p = 0.04$) but not in nonmusicians ($t = 0.81$; $p = 0.43$). This supports our hypothesis that weak ulnar nerve stimulation activates the surround inhibition circuits of the somatosensory system in pianists.

Experiment 5: weak CS to the ulnar nerve facilitated the production of fast sequential finger movements

The final experiment examined whether the inhibitory activity evoked by the weak ulnar nerve stimulation enhances the

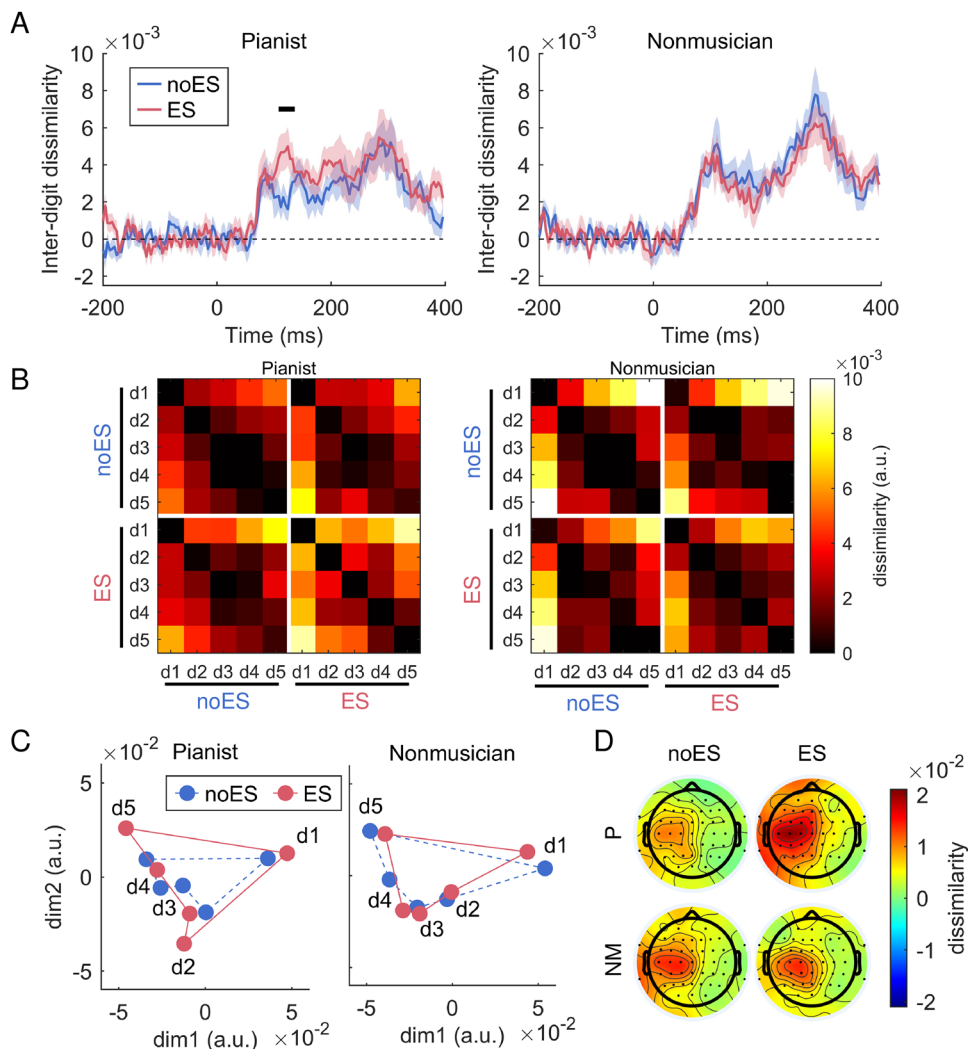


Figure 5. Effects of weak ulnar nerve stimulation on the subsequent proprioceptive processes. **A**, Effects of weak ulnar nerve stimulation on the average distance of spatial SEP amplitude patterns evoked by passive flexion of the MCP joint between two digits. The vertical axis represents the dissimilarity of spatial SEP amplitude patterns between two digits averaged across all possible pairs of digits. The blue lines represent the dissimilarity values obtained from the condition in which no electrical stimulation was provided, whereas the red line displays those obtained from the condition in which weak ulnar nerve stimulation was provided at the onset of the passive flexion of each digit. The horizontal line in the left panel indicates a cluster that shows significant differences in the dissimilarity between the two conditions ($p < 0.05$). The shaded area represents the SEM across participants. **B**, Averaged representational dissimilarity matrices (RDMs) across participants and the time within the significant cluster. The left and right panels show the RDMs obtained from pianists and nonmusicians, respectively. Each cell represents the dissimilarity of spatial SEP amplitude patterns between each corresponding pair of digits. **C**, 2D depiction of the data in **B**, using multidimensional scaling (MDS). MDS projects the higher-dimensional RDM into a lower-dimensional space while preserving the interdigit dissimilarity as accurately as possible. Blue dotted and red solid lines represent the data obtained from noES and ES conditions, respectively. MDS plots are purely for visualization purposes and were not used for statistical analysis. **D**, The topographical map of the dissimilarity value ~ 108 – 132 ms. The local dissimilarity values were computed based on the central and adjacent channels' SEP distribution.

production of fast and complex sequential finger movements performed by pianists. Figure 7A shows the number of erroneous strikes due to incorrect fingering (i.e., mistouches) made during the task in each condition. A GLME model with a Poisson distribution identified a significant main effect of condition on these data ($\chi^2_{(2)} = 13.16$; $p = 0.001$). A post hoc test revealed that the number of mistouches was significantly smaller when electrical stimulation was applied to the right ulnar nerve than in the other two conditions (right vs left, z -ratio, -2.64 ; $p = 0.017$; right vs no stimulation, z -ratio, -3.52 ; $p = 0.001$; left vs no stimulation, z -ratio, -0.89 ; $p = 0.376$). This reduction in the number of mistouches was not due to a decrease in keystroke speed because the IKI for correctly produced sequences did not differ across conditions (Fig. 7B, GLME, gamma distribution, log link function; condition, $\chi^2_{(2)} = 0.066$; $p = 0.968$). Thus, this experiment demonstrated that weak somatosensory stimulation improved

the fast and accurate execution of complex sequential finger movements in expert pianists.

Discussion

The present study examined the experience-dependent plasticity of an inhibitory function in somatosensory processing by comparing expert pianists and nonmusicians. We found that pianists have a superior ability to perceive fast multifinger movements than nonmusicians and exhibit enhanced inhibitory function in the somatosensory processing. A traditional and novel paired-pulse paradigm demonstrated that a weak CS to the ulnar nerve inhibited the subsequent somatosensory processes, increased the distance of cortical activity patterns evoked by passive finger movements, enhanced the surround inhibition, and improved the perception and control of fast multifinger movements,

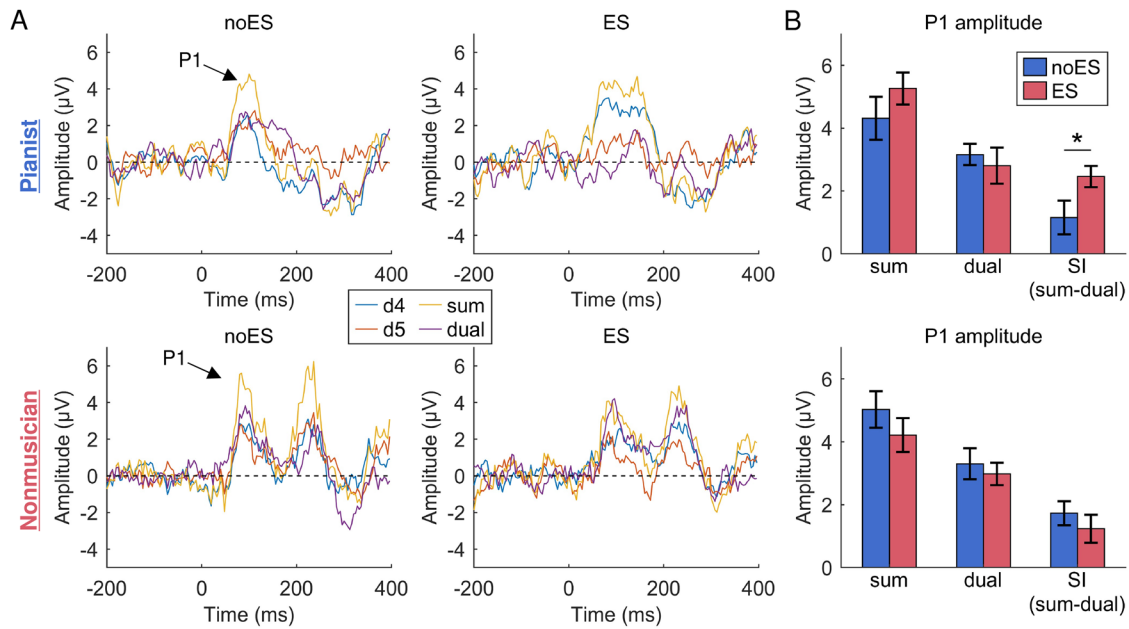


Figure 6. Effects of weak ulnar nerve stimulation on the surround inhibition (SI) of proprioceptive processes. **A**, Typical EEG waveforms evoked by passive finger flexion in a representative participant in each group. The blue and red lines represent the SEP evoked by the ring (d4) and little (d5) fingers, respectively. The yellow line indicates the arithmetic sum of the two SEPs (sum), and the purple line displays the SEP evoked by simultaneous flexion of d4 and d5 (dual). The top and bottom panels show the results obtained from a pianist and a nonmusician, respectively. We quantified the SI as the difference in the amplitude between the sum and dual SEPs. **B**, The group average of the amplitude and SI of the P1 component of sum and dual SEPs in both groups and both conditions. Mean \pm SEM. noES versus ES, $*p < 0.05$.

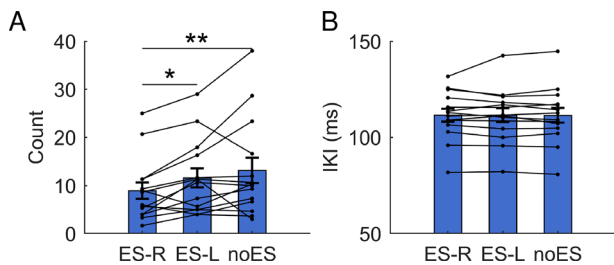


Figure 7. Effects of weak ulnar nerve stimulation on the performance of sequential finger movements. The group mean of the number of erroneous strikes due to incorrect fingering (**A**) and the IKI (**B**) during the repetition of the finger sequential movement for 20 s. Mean \pm SEM. Small dots and thin lines indicate individual data. Post hoc comparison, $**p < 0.01$; $*p < 0.05$.

specifically in pianists. These findings demonstrated novel neuroplastic mechanisms subserving the inhibitory function of somatosensory processing, which underlies outstanding abilities to perceive and execute fast and complex movements, characterizing experts who underwent extensive training of dexterous motor skills.

Inhibitory effect of weak ulnar nerve stimulation on somatosensory processes, specifically in pianists

We examined the inhibitory function in the somatosensory cortices using the paired-pulse paradigm of SEP. Previous studies demonstrated that PPS is plastically adapted by experiences and repetitive sensory inputs (Ragert et al., 2004; Höffken et al., 2007; Cheng and Lin, 2013). In both the pianists and non-musicians, a clear PPS was observed by the conventional paired-pulse method, in which the intensities of two stimuli were identical and close to the motor threshold in both the ulnar and median nerves. However, the group difference in the PPS was

only observed when testing it by the weak CS to the ulnar nerve. Specifically, applying CS_{PT} with an ISI of 10 ms inhibited the SEP only in pianists. This indicates a lower threshold for inducing inhibitory activity in pianists than nonmusicians, implying the experience-dependent plasticity of the inhibitory function. In contrast to pianists, the weak CS to the ulnar nerve facilitated the SEP evoked by the TS in nonmusicians. However, the effect differed between the two experiments. The statistical test failed to identify the difference in the SEP amplitude between the paired-pulse and single-TS conditions based on the combined data from the two experiments. Nonmusicians recruited in the present study might have a range of expertise outside of playing a musical instrument, such as sports and video games. Such a wide range of prior experience in nonmusicians may cause the PPS to be inconsistent between the two experiments.

We found no group differences in the PPS in the median nerve at either stimulus intensity. The median nerve innervates the skin of the thumb to the half of the ring finger, whereas the ulnar nerve innervates the skin of the remaining part of the ring finger to the little finger. In contrast, the ulnar nerve at the wrist innervates the muscles associated with the second to fifth digits, while the median nerve at the wrist innervates primarily the muscles related to the thumb. This suggests that the Ia afferents from muscle spindles (i.e., proprioceptive afferents) of the intrinsic hand muscles involved in the movement of the four fingers largely pass through the ulnar nerve. The thumb is frequently used in daily life and is anatomically and functionally highly independent of the other fingers (Lang et al., 2004; Kimoto et al., 2019). In contrast, playing the piano requires skilled control not only of the thumb but also of the other four fingers (Furuya et al., 2011). Such a unique demand for the four fingers during piano playing can explain neuroplasticity in the somatosensory system, which is responsible for the ulnar but not the median nerve in pianists.

A weak CS to the ulnar nerve activates surround inhibition

An essential role of somatosensory processing is to decode which body parts moved and how they moved from the somatosensory afferent information. Correcting ongoing movements based on incorrectly decoded sensory information can produce erroneous motor actions. This is a critical problem particularly for musicians and athletes. Previous studies demonstrated that cortical representation overlaps between various body parts in the sensorimotor cortex (Ejaz et al., 2015; Sadnicka et al., 2022) and that some sensory inputs from different body parts converge into identical neurons (Iwamura et al., 1980, 1993; Trzcinski et al., 2023). This may cause the sensory system to decode information from somatosensory inputs incorrectly when somatosensory information is inputted from different body parts within a short time. To avoid this, the somatosensory system must independently process the afferent information for each body part. Here, we showed the superior ability to perceive the fast multifinger movements in pianists compared with nonmusicians, suggesting that the pianist's nervous system processes somatosensory information from multiple fingers with less interference. The present study demonstrates that the enhanced somatosensory inhibitory function is a possible neural substrate for this superior perception. A CS to the ulnar nerve activated the inhibitory function, which increased the distance of the cortical activity evoked by passive movements between fingers. The latency of this effect was ~108–132 ms relative to the onset of movement. A previous study confirmed that the S1 and S2 are the origin of this component of SEP (Seiss et al., 2002), indicating that the inhibitory activity evoked by the CS modulates neural activity in the somatosensory cortices. We also found that the origin of the early component of SEP evoked by the passive movements was around the sensorimotor cortex. We also found that the CS enhanced surround inhibition. In addition, the stimulation lowered the perceptual threshold regarding the discrimination of the order of multifinger sequential movements. This perceptual enhancement and the amount of inhibition were correlated; pianists with stronger inhibition showed larger perceptual enhancement. The effects were specific to the pianists. These results indicate that pianists possess an enhanced inhibitory function that sharpens the somatosensory processing of individual fingers, which prevents the interference of cortical activities across fingers and enables pianists to perceive fast multifinger movements correctly. In contrast, we found no significant correlation between the order perception in the noES condition and the amount of inhibition. This may be due to the influence of multiple factors—such as somatotopic overlap, timing perception acuity, attention, arousal, and biomechanical finger independence—on the order perception of sequential finger movements, in addition to the inhibitory function within the somatosensory system. These factors could obscure the relationship between the variables when being analyzed without further control.

In addition to the perception, the weak conditioning stimulation enhanced the execution of fast multifinger sequential movements. Here, we emphasize that the performance level of our participants was very high, as evidenced by only a few mistouches at a repetitive keystroke rate above 9 Hz. Surprisingly, the conditioning stimulation further enhanced such a highly trained motor skill. The stimulation reduced the number of erroneous finger selections during movement without changing the movement speed. During the sequence execution, the somatosensory system must process incoming somatosensory afferents from different fingers and utilizes it to adjust motor commands (i.e., somatosensory–motor integration, closed-loop control). We previously

demonstrated that the somatosensory–motor integration function, as indexed by a modulatory effect of passive movements on the corticospinal excitability, is associated with the fingertip force fluctuation during a tapping task (Hirano et al., 2020a) and that the somatosensory perceptual training reduces the force fluctuation of repetitive keystrokes of a piano key (Hirano et al., 2020b; Hirano and Furuya, 2024). In the fast multifinger movements, abundant somatosensory afferents from different fingers are inputted into the somatosensory system. However, they interfere with each other due to the functional and structural overlap of somatosensory representation between fingers. This distorts the somatosensory–motor integration processes during the fast multifinger movements, which may lead to generating suboptimal motor commands. We postulate that activating the somatosensory inhibition by the conditioning stimulation prevented this interference, facilitating the sequence production. The CS inhibited the N20–P25 amplitude of SEP at the ISI of 10 ms and increased the interdigit dissimilarity of SEP patterns at a latency of ~132 ms relative to the onset of passive finger movement. This means that the CS could modulate the somatosensory activities from 30 ms to up to ~132 ms after the stimulation. In the sequential keystroke task, the CS was repetitively applied at 10 Hz, suggesting that the surround inhibition circuit would be activated throughout the task.

A limitation of this study is that several pianists participated in multiple experiments. We consider this overlap did not influence the results of this study. This is because each experiment was separated by at least 3 months, the neurophysiological measurements did not involve methods that could induce plasticity in the sensorimotor cortex, and the behavioral tasks have been used to assess motor and perceptual abilities, not to induce learning effects.

In summary, the present study addressed the experience-dependent plasticity of the inhibitory function of the somatosensory system. We identified that pianists have enhanced somatosensory inhibitory function. This inhibitory function corresponds to surround inhibition in the somatosensory cortices and plays an essential role in sharpening the representation of fingers in early somatosensory processing, specifically for pianists. These results demonstrate the experience-dependent plasticity of surround inhibition function in the somatosensory processes and its functional role in expert pianists' fast and complex movements.

References

- Alday PM (2019) How much baseline correction do we need in ERP research? Extended GLM model can replace baseline correction while lifting its limits. *Psychophysiology* 56:e13451.
- Altenmüller E, Furuya S (2016) *Brain plasticity and the concept of metaplasticity in skilled musicians*. pp 197–208. Cham: Springer.
- Antelmi E, Erro R, Rocchi L, Liguori R, Tinazzi M, Di Stasio F, Berardelli A, Rothwell JC, Bhatia KP (2017) Neurophysiological correlates of abnormal somatosensory temporal discrimination in dystonia. *Mov Disord* 32:141–148.
- Arbuckle SA, Pruszynski JA, Diedrichsen J (2022) Mapping the integration of sensory information across fingers in human sensorimotor cortex. *J Neurosci* 42:5173–5185.
- Bates D, Mächler M, Bolker BM, Walker SC (2015) Fitting linear mixed-effects models using lme4. *J Stat Softw* 67:1–48.
- Cheng CH, Lin YY (2013) Aging-related decline in somatosensory inhibition of the human cerebral cortex. *Exp Brain Res* 226:145–152.
- David-Jürgens M, Dinse HR (2010) Effects of aging on paired-pulse behavior of rat somatosensory cortical neurons. *Cereb Cortex* 20:1208–1216.
- Delorme A, Makeig S (2004) EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods* 134:9–21.
- Ejaz N, Hamada M, Diedrichsen J (2015) Hand use predicts the structure of representations in sensorimotor cortex. *Nat Neurosci* 18:1034–1040.

- Elbert T, Pantev C, Wienbruch C, Rockstroh B, Taub E (1995) Increased cortical representation of the fingers of the left hand in string players. *Science* 270:305–307.
- Fox J, Weisberg S (2019) *An R companion to applied regression*, Ed 3. Thousand Oaks, CA: Sage.
- Furuya S, Flanders M, Soechting JF (2011) Hand kinematics of piano playing. *J Neurophysiol* 106:2849–2864.
- Furuya S, Tanibuchi R, Nishioka H, Kimoto Y, Hirano M, Oku T (2023) Passive somatosensory training enhances piano skill in adolescent and adult pianists: a preliminary study. *Ann N Y Acad Sci* 1519:167–172.
- Gandevia SC, Burke D, McKeon BB (1983) Convergence in the somatosensory pathway between cutaneous afferents from the index and middle fingers in man. *Exp Brain Res* 50:415–425.
- Hirano M, Furuya S (2024) Active perceptual learning involves motor exploration and adaptation of predictive sensory integration. *iScience* 27:108604.
- Hirano M, Kimoto Y, Furuya S (2020a) Specialized somatosensory–motor integration functions in musicians. *Cereb Cortex* 30:1148–1158.
- Hirano M, Sakurada M, Furuya S (2020b) Overcoming the ceiling effects of experts' motor expertise through active haptic training. *Sci Adv* 6:eabd2558.
- Höffken O, Veit M, Knossalla F, Lissek S, Bliem B, Ragert P, Dinse HR, Tegenthoff M (2007) Sustained increase of somatosensory cortex excitability by tactile coactivation studied by paired median nerve stimulation in humans correlates with perceptual gain. *J Physiol* 584:463–471.
- Iwamura Y, Tanaka M, Hikosaka O (1980) Overlapping representation of fingers in the somatosensory cortex (area 2) of the conscious monkey. *Brain Res* 197:516–520.
- Iwamura Y, Tanaka M, Sakamoto M, Hikosaka O (1993) Rostrocaudal gradients in the neuronal receptive field complexity in the finger region of the alert monkey's postcentral gyrus. *Exp Brain Res* 92:360–368.
- Kieliba P, Clode D, Maimon-Mor RO, Makin TR (2021) Robotic hand augmentation drives changes in neural body representation. *Sci Robot* 6:7935.
- Kimoto Y, Oku T, Furuya S (2019) Neuromuscular and biomechanical functions subserving finger dexterity in musicians. *Sci Rep* 9:12224.
- King-Smith PE, Grigsby SS, Vingrys AJ, Benes SC, Supowit A (1994) Efficient and unbiased modifications of the QUEST threshold method: theory, simulations, experimental evaluation and practical implementation. *Vision Res* 34:885–912.
- Lang CE, Schieber MH, Karrer JH, Cheney PD (2004) Human finger independence: limitations due to passive mechanical coupling versus active neuromuscular control. *J Neurophysiol* 92:2802–2810.
- Madigan R, Williams D (1987) Maximum-likelihood psychometric procedures in two-alternative forced-choice: evaluation and recommendations. *Percept Psychophys* 42:240–249.
- Münste TF, Altenmüller E, Jäncke L (2002) The musician's brain as a model of neuroplasticity. *Nat Rev Neurosci* 3:473–478.
- Ogawa K, Mitsui K, Imai F, Nishida S (2019) Long-term training-dependent representation of individual finger movements in the primary motor cortex. *Neuroimage* 202:116051.
- Pion-Tonachini L, Kreutz-Delgado K, Makeig S (2019) ICLabel: an automated electroencephalographic independent component classifier, dataset, and website. *Neuroimage* 198:181–197.
- Ragert P, Becker M, Tegenthoff M, Pleger B, Dinse HR (2004) Sustained increase of somatosensory cortex excitability by 5Hz repetitive transcranial magnetic stimulation studied by paired median nerve stimulation in humans. *Neurosci Lett* 356:91–94.
- Ritchie JB, Lee Masson H, Bracci S, Op de Beek HP (2021) The unreliable influence of multivariate noise normalization on the reliability of neural dissimilarity. *Neuroimage* 245:118686.
- Sadnicka A, Wiestler T, Butler K, Altenmüller E, Edwards MJ, Ejaz N, Diedrichsen J (2022) Intact finger representation within primary sensorimotor cortex of musician's dystonia. *Brain* 146:1511–1522.
- Sanders ZB, Dempsey-Jones H, Wesselink DB, Edmondson LR, Puckett AM, Saal HP, Makin TR (2023) Similar somatotopy for active and passive digit representation in primary somatosensory cortex. *Hum Brain Mapp* 44:3568–3585.
- Searle SR, Speed FM, Milliken GA (2023) Estimated marginal means, aka least-squares means [R package emmeans version 1.8.4-1]. *Am Stat* 34:216–221.
- Seiss E, Hesse CW, Drane S, Oostenveld R, Wing AM, Praamstra P (2002) Proprioception-related evoked potentials: origin and sensitivity to movement parameters. *Neuroimage* 17:461–468.
- Tamura Y, Matsushashi M, Lin P, Ou B, Vorbach S, Kakigi R, Hallett M (2008) Impaired intracortical inhibition in the primary somatosensory cortex in focal hand dystonia. *Mov Disord* 23:558–565.
- Tinazzi M, Priori A, Bertolasi L, Frasson E, Mauguière F, Fiaschi A (2000) Abnormal central integration of a dual somatosensory input in dystonia: evidence for sensory overflow. *Brain* 123:42–50.
- Trzcinski NK, Hsiao SS, Connor CE, Gomez-Ramirez M (2023) Multi-finger receptive field properties in primary somatosensory cortex: a revised account of the spatiotemporal integration functions of area 3b. *Cell Rep* 42:112176.
- Walther A, Nili H, Ejaz N, Alink A, Kriegeskorte N, Diedrichsen J (2016) Reliability of dissimilarity measures for multi-voxel pattern analysis. *Neuroimage* 137:188–200.
- Weise D, Gentner R, Zeller D, Nagel A, Reinsberger C, Rumpf J-J, Classen J (2012) Focal hand dystonia: lack of evidence for abnormality of motor representation at rest. *Neurology* 78:122–128.