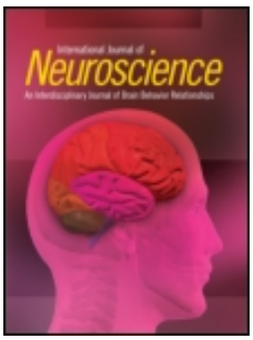

International Journal of Neuroscience

ISSN: 0020-7454 (Print) 1543-5245 (Online) Journal homepage: http://www.tandfonline.com/loi/ines20

Cerebellar transcranial direct current stimulation does not alter motor surround inhibition

Anna Sadnicka, Panagiotis Kassavetis, Tabish A. Saifee, Isabel Pareés, John C. Rothwell & Mark J. Edwards

To cite this article: Anna Sadnicka, Panagiotis Kassavetis, Tabish A. Saifee, Isabel Pareés, John C. Rothwell & Mark J. Edwards (2013) Cerebellar transcranial direct current stimulation does not alter motor surround inhibition, International Journal of Neuroscience, 123:6, 425-432, DOI: 10.3109/00207454.2012.763165

To link to this article: http://dx.doi.org/10.3109/00207454.2012.763165

Accepted author version posted online: 08 Jan 2013.
Published online: 11 Mar 2013.

International Journal of Neuroscience, 2013; 123(6): 425–432

ISSN: 0020-7454 print / 1543-5245 online
DOI: 10.3109/00207454.2012.763165

informa
healthcare

Cerebellar transcranial direct current stimulation does not alter motor surround inhibition

Anna Sadnicka, Panagiotis Kassavetis, Tabish A. Saifee, Isabel Pareés, John C. Rothwell, and Mark J. Edwards

Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology, University College London, London, United Kingdom

Motor surround inhibition (mSI) is one mechanism by which the central nervous system individuates finger movements, and yet the neuroanatomical substrate of this phenomenon is currently unknown. In this study, we examined the role of the cerebellum in the generation of mSI, using transcranial direct current stimulation of the cerebellum (cDC). We also examined intrasubject and intersubject variability of mSI. Twelve subjects completed a three session cross over study in which mSI was measured before and after (0 and 20 minutes) sham, anodal and cathodal cDC. mSI of the surround muscle (adductor digiti minimi) at the onset of flexion of the index finger was consistently observed. Anodal and cathodal cDC did not modulate the magnitude of mSI. For individual subjects (across the three sessions), the intrasubject coefficient of variation was 27%. Between subjects, the intersubject coefficient of variation was 47%. mSI was a stable effect in individual subjects across multiple sessions. This is an important observation and contrasts with other neurophysiological paradigms such as paired associative stimulation response, which exhibit great variability. In addition, we have quantified intrasubject variability of mSI, which will allow future therapeutic studies that attempt to modulate mSI to be adequately powered. We have not found evidence that the cerebellum contributes to the neuroanatomical network needed for the generation of mSI. Understanding the mechanisms of mSI remains a challenge but is important for disorders in which it is deficient such as Parkinson's disease and focal hand dystonia.

KEYWORDS: finger individuation, transcranial direct current stimulation, transcranial magnetic stimulation, motor control

Introduction

Surround (or lateral) inhibition is a term used to describe multiple phenomena throughout the nervous system in which neural signals to a central receptive field or target are facilitatory and eccentric signals are inhibitory. Within the motor system, it was first explored conceptually as a mechanism by which basal ganglia circuits selectively execute desired motor programmes [1]. Later, a potential neurophysiological measure of motor surround inhibition (mSI) was demonstrated; by stimulating the motor cortex using transcranial magnetic stimulation (TMS) at the onset of movement of the index finger, suppression in the size of responses of non-synergistic surround muscles was seen [2].

It is not known which neuroanatomical structures within the central nervous system are important for the generation of mSI. Some authors favour a neocortical mechanism following the observation that hemispheric dominance and task difficulty modulate the magnitude of mSI [3]. However, electrophysiological studies examining the dependency of mSI on dorsal and ventral premotor, and motor cortex interactions have failed to support this notion [4,5].

The cerebellum plays a major role in temporal encoding and coordination of movements, and deficiencies in hand control and individual finger movements are seen in patients with cerebellar disease [6]. It also has a net inhibitory effect on the cerebral cortex via the cerebello-dentato-thalamo-cortical pathway [6]. These characteristics make the cerebellum a suitable candidate to functionally contribute to the generation of mSI.

Received 7 September 2012; revised 7 December 2012; accepted 30 December 2012.

Correspondence: Dr Mark J. Edwards, Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology, University College London, Box 146, National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, United Kingdom. Tel: 0044 203 4488749. E-mail: m.j.edwards@ucl.ac.uk

Previous work examining cerebellar brain inhibition (CBI), and individual finger movements demonstrated a non-specific decrease in cerebellar inhibition to active and surround muscles at the motor cortex at the onset of movement but no link between mSI and CBI [7]. However, CBI relies on a powerful (and painful) phasic non-topographically specific magnetic stimulation of the cerebellum that may not reveal subtle changes in paradigms such as mSI. In this study, we utilise cerebellar transcranial direct current stimulation (cDC), which has emerged as an important technique by which to enhance (anodal) or decrease (cathodal) cerebellar excitability [8]. The cerebellum is stimulated for 15 minutes, and changes in excitability are seen for at least 30 minutes [8]. This has been confirmed neurophysiologically (measuring CBI) and behaviourally (measuring rates of adaptation to sensory perturbations, a cerebellar-dependent learning task); anodal cDC increases CBI and leads to faster rates of adaptation and cathodal cDC decreases CBI [8,9]. In addition, cDC can be used to assess the cerebellar contribution to neurophysiological paradigms; recently, the cerebellum was shown to be a critical structure for the generation of motor cortex plasticity responses to paired associative stimulation (PAS) with an interstimulus interval of 25 ms [10].

Our hypothesis was that stimulatory anodal cDC would enhance mSI and cathodal cDC would impair mSI. Investigating techniques that may have the potential to modulate mSI is important for patients with disorders such as focal hand dystonia and Parkinson's disease in which impaired mSI is seen [11,12]. The multiple session design of this study gave us additionally the opportunity to assess intrasubject and intersubject variability of mSI.

Methods

Subjects

Twelve right-handed healthy subjects (mean age, 25 years; range, 19–35 years; 9 male) with no history of neurological or psychiatric disease participated in the study. Handedness was determined by the Edinburgh Handedness Inventory. Written informed consent was obtained from all participants, and the study was approved by the local ethics committee and conducted in accordance with the Declaration of Helsinki 2008.

Recording

Disposable surface electromyographic (EMG) electrodes were placed on the right first dorsal interosseus (FDI) and abductor digiti minimi (ADM) muscles, using a belly-tendon montage. The signal from the EMG electrodes was amplified (gain 1000), band-pass filtered (20–2000 Hz) (Digitimer D360 amplifier) and digitised at a sampling rate of 5 kHz and stored in a laboratory computer for off-line analysis by CED 1401 hardware and Signal software (Cambridge Electronic Design Ltd, Cambridge, UK).

Transcranial magnetic stimulation

Monophasic TMS pulses were delivered from a Magstim 200^2 stimulator. A figure-of-eight coil (external loop diameter of 9 cm) was held tangentially on the scalp at an angle of 45° to the midsagittal plane with the handle pointing laterally and posteriorly. Corticospinal tract excitability was measured as the peak-to-peak amplitude of the motor-evoked potential (MEP) generated by single-pulse TMS. TMS was applied to the motor "hot spot" of the right ADM muscle that was defined as the point where a magnetic stimulus of slightly suprathreshold intensity consistently elicited an MEP in ADM of the highest amplitude. This position was marked on a tight fitting neoprene cap to ensure consistent coil position during the experiment.

Cerebellar transcranial direct current stimulation

The cDC was applied to the cerebellum as previously described [8]. It was delivered with an intensity of 2 mA, using a DC stimulator through 25 cm^2 saline-soaked surface sponge electrodes (Eldith-Electro-Diagnostic & Therapeutic Systems GmbH, Germany). One electrode was centred on the right cerebellar cortex, 3-cm lateral to the inion and the other electrode was positioned on the right buccinator muscle [8]. Anodal or cathodal cDC was delivered over the cerebellum for 15 minutes. In the sham session, anodal cDC was applied for 30 seconds in order that a true sham condition was simulated (some subjects experience tingling at site of electrodes when stimulation is initiated). At the onset and offset of all interventions (anodal, cathodal and sham), current was changed in a ramp-like manner over 10 seconds. Subjects were supervised during cDC and listened to a radio documentary. They were asked to keep all movement, specifically finger movements, to a comfortable minimum.

Motor task

Subjects were seated in a chair with their right hand resting in a relaxed position on a desk. They were asked to briefly depress a small button with the index finger

after a "go" signal (an auditory tone of 50 ms) with a self-paced delay. FDI is a synergist rather than a primary muscle for this movement, and previous studies have shown that this movement induces activation of FDI and suppression of the MEPs elicited in the ADM muscle [2]. Subjects were first asked to press with maximal force, and amplitude of mean EMG activity in FDI was noted. Subjects were then trained to perform the movement to the amplitude of 10% maximal EMG activity while visual feedback of the muscle activity was projected on a screen in front of them. Duration of the movement was approximately 100 ms. We favoured a short movement duration to facilitate production of a clean onset and offset of EMG activity as mSI has been found to be active only during the initiation of the movement and not later during tonic muscle contraction [2]. Subjects were also asked to keep the surround muscle ADM relaxed while they were performing the movement. Training was continued until subjects achieved consistent performance of the desired movement, and raw EMG signal in ADM muscle was not in excess of 100 μV.

Experimental design

Each subject took part in a cross over study, which consisted of each of the three types of stimulation (sham, cathodal or anodal) in **a** randomised order. Each session was separated by a week. Resting motor threshold (RMT) was measured and was defined as the lowest intensity [expressed as a percentage of maximum stimulus intensity (MSO)] that evoked a response of about 50 μV in the relaxed ADM in at least 5 of 10 trials [13]. The intensity of the stimulation was then set to evoke ADM MEPs with average peak-to-peak amplitude of approximately 1 mV at rest for the remainder of the experiment.

For the assessment of mSI, five states of self-triggered TMS were applied in a random order at variable intervals between EMG onset and TMS trigger (0, 50, 100, 200 ms and 5 seconds). This allowed us to assess the magnitude of mSI at time 0 ms and also assess if cDC induced changes in the timing profile of inhibition/mSI at later time intervals. The TMS pulse was triggered when EMG signal of right FDI rose above 100 μV. Twenty trials of 5 seconds (rest) and 15 trials of the other four intervals (0, 50, 100 and 200 ms) were collected. Five seconds after the onset of movement is considered to be sufficient for measurements at rest as no post-activation inhibitory or facilitatory effect are known to be active at this time [2].

Data analysis and statistics

For each subject, peak-to-peak MEP amplitude for each trial was measured off-line, and the mean MEP amplitude at rest and at each time interval was calculated. For each interval, mean MEP amplitude was then divided by mean rest MEP amplitude for the respective muscle (labelled in graphs as percentage of resting MEP). If the ratio is less than 1, there is evidence for mSI. When it is greater than or equal to 1, there is no mSI.

Unless otherwise stated, all results are expressed as mean $\pm$ standard error of the mean (SEM). We used SPSS software (version 19) for statistical analysis (SPSS Ltd., IBM, Armonk, NY, USA). Kolmogorov–Smirnov test was used to explore the normality of the data distribution, and Levene's test was used to explore the homogeneity of variance. Log10 transformation was performed when data were not normally distributed.

Repeated measures analysis of variance (rmANOVA) was used to confirm the presence of mSI in ADM and to assess the effects of cDC on the magnitude of mSI before and after stimulation. Bonferroni's correction for multiple comparisons was used for *post hoc t* tests. To quantify intrasubject and intersubject variability, the coefficient of variation (COV) was expressed as a percentage. The COV is the ratio of the standard deviation to the mean.

Results

All subjects completed the three sessions without any adverse events, and each experimental session lasted 2 hours.

Baseline measures

The mean stimulus intensity for RMT of ADM across the three sessions for all subjects was 41% of MSO ($\pm$2.3%). The stimulus intensity required for a 1-mV MEP in ADM ranged from 38% to 80% of MSO across subjects with a mean value of 57% ($\pm$3.4%). The mean stimulus intensity required for a 1-mV MEP in ADM was 137% of the RMT.

mSI present in ADM

Figure 1 demonstrates the profile of MEP sizes in the FDI and ADM muscles for each of the intervals tested. MEPs are expressed as percentage of resting MEP, and the group mean is derived from the individual mean of the three baseline measurements of mSI taken at each session. Log10 transformation was performed and the data satisfied the assumptions for parametric tests after the transformation. One-way rmANOVA revealed a significant effect of INTERVAL (0, 50, 100 and 200 ms) in the ADM muscle $F(3,7) = 22.84$, $p < 0.001$ and FDI muscle $F(3,7) = 15.84$, $p < 0.001$ (Figure 1).

In ADM *post hoc* paired sample *t* tests of raw MEP data at rest (5 seconds) and during movement (0, 50,

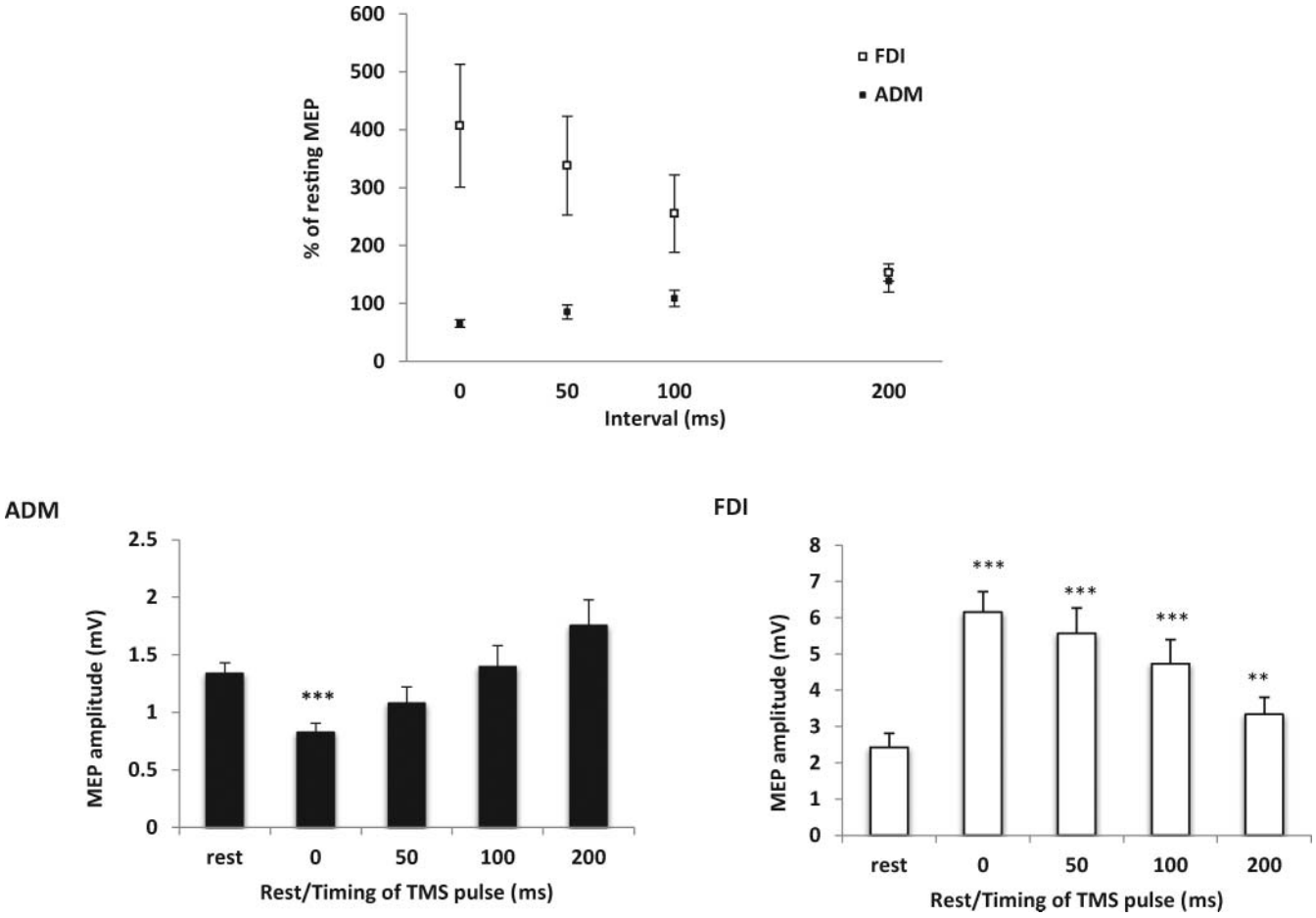

Figure 1. Profile of mSI. This figure demonstrates the group mean of the individual means across the three baseline sessions. In the upper panel, the normalised data are shown for both muscles. Raw MEP data are given for individual muscles below. The surround muscle ADM is significantly inhibited at time interval 0 ms. Note the reduction of variability in the ADM muscle MEPs (as indicated by the error bars demonstrating the standard error). The active muscle FDI is facilitated at the onset of movement and the later time intervals tested ($^{**}p \leq 0.01$; $^{***}p \leq 0.001$).

100 and 200 ms) revealed that mSI was present at time interval 0 ms, thus MEPs in ADM were significantly inhibited at time interval 0 ms $t(11) = 4.93$, $p < 0.001$. There was no significant inhibition of ADM at the other time intervals, and it can be seen from Figure 1 that the MEP size gradually increases. Only one subject had a mean ADM MEP amplitude at the onset of the movement (interval 0 ms), which was not less than the resting MEP (mean mSI = 1.12 ± 0.04 across three baseline sessions). The MEP was still suppressed in this subject (as there is an increase in spinal excitability at 0 ms [2]), but it is not by definition inhibited.

In FDI, there was significant enhancement of MEP amplitudes at all of the time intervals (0, 50, 100 and 200 ms) compared with rest (0 ms $t(11) = -8.77$, $p < 0.001$; 50 ms $t(11) = -5.46$, $p < 0.001$; 100 ms $t(11) = -4.27$, $p = 0.001$; 200 ms $t(11) = -3.45$, $p = 0.005$).

Effect of cDC on mSI

To explore the effect of cDC on mSI, we looked at the magnitude of mSI at 0 ms in the muscle ADM at each of the time points measured (baseline, T0, T20) (Figure 2A). rmANOVA with factors TIME (baseline, T0, T20) and cDC (sham, anodal, cathodal) revealed no significant effect of TIME [$F(2,10) = 1.09$, $p = 0.35$], cDC [$F(2,10) = 1.03$, $p = 0.38$] or their interaction [$F(4,8) = 1.05$, $p = 0.39$]. There was also no significant effect of cDC on MEP profile at any of the other intervals tested (50, 100 or 200 ms) (Figure 2B–D). On the basis of these results, we conclude that the cerebellum does not seem to have a role in the generation of mSI.

Intrasubject and intersubject variability of mSI

To quantify variability of mSI, we examined mSI seen in ADM at the onset of index finger movement (interval

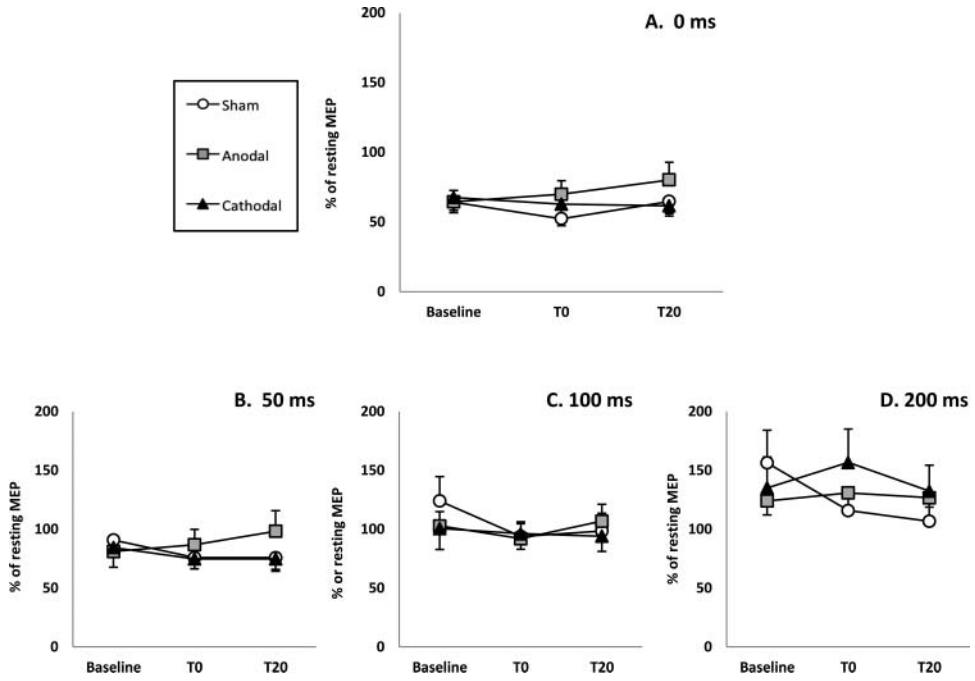

Figure 2. Effect of sham, anodal and cathodal cDC on the magnitude of mSI in ADM. There was no significant modulation of the magnitude of mSI by cDC.

0 ms) (Table 1). Intrasubject variation of mSI (range of mSI responses exhibited by a single subject) as assessed by COV had a mean value of 27% (range, 14%–48%). Intersubject variability (different subjects) had a mean value of 44% (range, 40%–46%).

Discussion

Motor surround inhibition was clearly demonstrated across subjects; at 0 ms, there was consistent and statistically significant inhibition of MEPs in ADM. The study design allowed three measures of mSI on different sessions in the same subjects, and mSI was confirmed to be stable within subjects. Given the intrinsic variability of MEPs, this marks out the measurement of mSI a robust and reproducible TMS paradigm. This is in contrast to some other commonly used electrophysiological paradigms. For example, a common measure of motor cortex plasticity is PAS in which repetitive pairing of median nerve stimulation and TMS pulses to the motor cortex lead to facilitation of MEPs in APB [14]. However, if individual PAS responses are displayed, it is seen that some subjects have facilitatory responses while others have inhibitory responses to PAS. Furthermore, if PAS is tested in the same subjects at another session, the direction of the MEP response may change, subjects can switch between facilitators and inhibitors and vice versa [15]. This is not seen with mSI when tested across the three sessions and quantified by the COV (Table 1). This reemphasises the importance of the deficiency of mSI seen in diseases of motor control such as focal hand dystonia and Parkinson's disease [16]. Attempting to modulate the strength of mSI, as in this study, remains an important potentially therapeutic goal in neurophysiological studies of mSI.

The mSI is defined as the functional inhibition of surround muscles seen during the movement initiation phase (and just before and during the first phase of EMG onset) [16]. The mechanisms of how and where it is generated are less well characterised. At the spinal level, there is a non-spatially selective facilitation at these time points (shown by F-wave and H-reflex studies), and thus mSI is thought to reflect a supraspinal control mechanism [2]. We find no evidence that modulating the excitability of the cerebellum in isolation can change the magnitude of mSI. This adds to previous work examining CBI, which did not find a functional link between mSI and CBI [7]. In addition, no association between activity in premotor cortex (both ventral and dorsal) and mSI has been demonstrated [4,5]. It may be that mSI is a fundamental inhibitory mechanism within the nervous system, and subtle alteration of the activity of one of the nodes within the mSI network does not allow a meaningful change in mSI to be observed. Alternatively, the genesis of mSI may reside within other areas such as the basal ganglia nuclei. It should be possible in the future to explore this hypothesis by

Table 1. Intrasubject and intersubject variability of mSI exhibited in ADM muscle at the onset of movement (interval 0 ms). Values are shown for each session before any stimulation. Each measure of mSI is given as a ratio of mean resting MEP for ADM (normalised values). Intrasubject and intersubject variability are compared using the coefficient of variation (COV).

Subject	1	2	3	4	5	6	7	8	9	10	11	12	Intersubject COV (each session)	Mean intersubject COV
mSI (session 1)	0.29	0.39	0.47	0.56	0.62	0.42	0.84	0.94	0.41	1.02	0.80	0.94	40%	44%
mSI (session 2)	0.46	0.42	0.72	0.37	0.44	0.66	0.65	0.51	1.17	0.56	0.52	1.26	45%	
mSI (session 3)	0.35	0.31	0.26	0.53	0.46	0.56	0.78	0.82	0.81	0.89	1.20	1.14	46%	
Mean mSI for each subject	0.37	0.37	0.48	0.49	0.51	0.55	0.76	0.76	0.80	0.82	0.84	1.12		
Intrasubject COV (individual values)	24%	15%	48%	22%	19%	22%	13%	29%	47%	28%	41%	14%		
Mean ($n = 12$) Intrasubject COV							27%							

measuring mSI in patients with Parkinson's disease or dystonia before and after deep brain stimulation.

At the synaptic level a gamma amino butyric acid (GABA)ergic mechanism for mSI has been proposed largely based on animal work [16]. In humans, proving the link between GABAergic circuits and mSI is less certain. No functional link has been shown between mSI and short-interval intracortical inhibition and cortical silent period, which are indirect markers for $GABA_A$ and $GABA_B$ receptor function, respectively [2,17]. Other inhibitory projections to M1 are reduced at the onset of movement and do not consistently demonstrate the action specific modulation of muscle excitability unique to mSI (long-interval intracortical inhibition, short-latency afferent inhibition, long-latency afferent inhibition, interhemispheric inhibition, CBI) [2,7,16].

There is increasing evidence that mSI is an adaptive phenomena. It has previously been shown that mSI is more pronounced in the dominant hemisphere, is stronger with low force levels and starts earlier with increasing task difficulty [3,18]. More recently, it has been demonstrated that the magnitude of mSI is increased by carefully timed vibration training [19]. Conversely, 30 minutes of finger exercises with synchronised movements of the index and little finger in contrast to little finger movements alone reduces the magnitude of mSI, perhaps blurring individuation of digits as measured by mSI or implicating a role for fatigue on mSI modulation [20].

The failure of cDC to modulate mSI was surprising. We believe cDC to be an excellent tool to explore the functional network that contributes to mSI; indeed, in the visual cortex, anodal cDC has recently been found to change surround suppression, a comparable paradigm to mSI in the visual system [21]. It is an interesting question whether the degree of adaptation of mSI may be increased or decreased by stimulation techniques; one might expect cDC to modify the adaptation seen with vibration training.

Further characterisation of mSI remains a challenging field. It is worth restating that the first study of mSI found comparable amounts of inhibition in ADM when the paradigm is triggered by mouth or leg movement (risorius: 77%; tibialis anterior: 68%) [2]. This finding has never been replicated but suggests a less spatially specific mechanism for mSI than is currently discussed, particularly when mSI is mentioned in the context of models of focal hand dystonia. In addition, the current literature freely moves between using the term surround inhibition as a cellular mechanism in the senses, neurophysiological mechanism in motor (mSI) and sensory systems (somatosensory-evoked potentials [22]), as a mechanism for selecting motor programmes [23] and as an explanation for psychophysical phenomena [24]. To move away from a purely descriptive term that represents the capability of organisms to attach saliency to inputs or produce specific commands, we must examine the similarities and differences between surround inhibition at each hierarchical level and modality to understand its mechanisms further.

A limitation of our study is that subtle differences in experimental conditions across the three sessions may have lead to incorrect acceptance of the null hypothesis that the cerebellum does not functionally contribute in the generation of mSI (both subject dependent, e.g. level of attention to task and experimental, e.g. differences in placement position of TMS coil). We considered increasing the number of subjects but as no trend was seen in our 12 subjects we consider the acceptance of the null hypothesis to be correct.

Conclusions

We find mSI to be a robust electrophysiological phenomenon with minimal intrasubject variability over the three sessions in this study. Quantification of intrasubject variability in this study will allow future therapeutic studies that attempt to modulate mSI to be adequately powered. We do not find evidence to suggest that the cerebellum contributes to the neuroanatomical network necessary for the generation of mSI. We have reviewed the current literature on mSI and identify important future challenges in the field that need further investigation so that the physiology of mSI and its deficit in certain diseases is more clearly understood.

Declaration of Interest

The authors report no conflict of interest. The authors alone are responsible for the content and writing of this paper.

Dr Sadnicka is funded by the Guarantors of Brain with an Association of British Neurologists Clinical Training Fellowship and has received financial support from the Dystonia Medical Research Foundation and the Movement Disorders Society. Dr Kassavetis is funded by Parkinson's UK and has received financial support from the Dystonia Medical Research Foundation. Dr Saifee is funded by a fellowship awarded by the National Institute for Health Research. Dr Pareés is funded by the Fundación Alfonso Martín Escudero and has received support from the Anglo-Spanish Society. Prof Rothwell has current grant support from the European Union FP7, Tourette Association, Dystonia Medical Research Foundation, The Stroke Association and The Royal Society. Dr Edwards is funded by a fellowship awarded by the National Institute for Health Research.

References

1. Mink JW. The basal ganglia: focused selection and inhibition of competing motor programs. Prog Neurobiol 1996;50(4):381–425.
2. Sohn YH, Hallett M. Surround inhibition in human motor system. Exp Brain Res 2004;158(4):397–404.
3. Beck S, Hallett M. Surround inhibition is modulated by task difficulty. Clin Neurophysiol (official journal of the International Federation of Clinical Neurophysiology) 2010;121(1): 98–103.
4. Beck S, Houdayer E, Richardson SP, Hallett M. The role of inhibition from the left dorsal premotor cortex in right-sided focal hand dystonia. Brain Stimul 2009;2(4): 208–14.
5. Houdayer E, Beck S, Karabanov A, et al. The differential modulation of the ventral premotor-motor interaction during movement initiation is deficient in patients with focal hand dystonia. Eur J Neurosci 2012;35(3):478–85.
6. Manto M, Bower JM, Conforto AB, et al. Consensus paper: roles of the cerebellum in motor control-the diversity of ideas on cerebellar involvement in movement. Cerebellum 2011;11(2):457–87.
7. Kassavetis P, Hoffland BS, Saifee TA, et al. Cerebellar brain inhibition is decreased in active and surround muscles at the onset of voluntary movement. Exp Brain Res 2011;209(3): 437–42.
8. Galea JM, Jayaram G, Ajagbe L, Celnik P. Modulation of cerebellar excitability by polarity-specific noninvasive direct current stimulation. J Neurosci 2009;29(28): 9115–22.
9. Galea JM, Vazquez A, Pasricha N, et al. Dissociating the roles of the cerebellum and motor cortex during adaptive learning: the motor cortex retains what the cerebellum learns. Cereb Cortex 2011;21(8):1761–70.
10. Hamada M, Strigaro G, Murase N, et al. Cerebellar modulation of human associative plasticity. J Physiol 2012; 590(10):2365–74.
11. Sohn YH, Hallett M. Disturbed surround inhibition in focal hand dystonia. Ann Neurol 2004;56(4):595–9.
12. Shin H-W, Kang SY, Hallett M, Sohn YH. Reduced surround inhibition in musicians. Exp Brain Res 2012;219(3):403–8.
13. Rossi S, Hallett M, Rossini PM, et al. Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. Clin Neurophysiol 2009;120(12):2008–39.
14. Stefan K, Kunesch E, Cohen LG, et al. Induction of plasticity in the human motor cortex by paired associative stimulation. Brain 2000;123(Pt 3):572–84.
15. Fratello F, Veniero D, Curcio G, et al. Modulation of corticospinal excitability by paired associative stimulation: reproducibility of effects and intraindividual reliability. Clin Neurophysiol 2006;117(12):2667–74.
16. Beck S, Hallett M. Surround inhibition in the motor system. Exp Brain Res 2011;210(2):165–72.
17. Poston B, Kukke SN, Paine R, et al. Cortical silent period duration and its implications for surround inhibition of a hand muscle. Eur J Neurosci 2012 (epub ahead of print)
18. Beck S, Schubert M, Richardson SP, Hallett M. Surround inhibition depends on the force exerted and is abnormal in focal hand dystonia. J Appl Physiol 2009;107(5):1513–8.
19. Kassavetis P, Saifee TA, Sadnicka A, et al. Adaptation of surround inhibition in the human motor system. Exp Brain Res 2012;222(3):211–7.
20. Kang SY, Hallett M, Sohn YH. Synchronized finger exercise reduces surround inhibition. Clin Neurophysiol 2012;123(11):2227–31.
21. Spiegel DP, Hansen BC, Byblow WD, Thompson B. Anodal transcranial direct current stimulation reduces psychophysically measured surround suppression in the human visual cortex. PLoS ONE 2012;7(5):e36220.
22. Tinazzi M, Priori A, Bertolasi L, et al. Abnormal central integration of a dual somatosensory input in dystonia. Evidence for sensory overflow. Brain 2000;123(1):42–50.
23. Mink JW. The Basal Ganglia and involuntary movements: impaired inhibition of competing motor patterns. Arch Neurol 2003;60(10):1365–8.
24. Blakemore C, Carpenter R. Lateral inhibition between orientation detectors in the human visual system. Nature 1970;228:37–41.