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*Published in:*  
Journal of Neurophysiology

*DOI (link to publication from Publisher):*  
[10.1152/jn.00211.2019](https://doi.org/10.1152/jn.00211.2019)

*Publication date:*  
2019

*Document Version*  
Accepted author manuscript, peer reviewed version

[Link to publication from Aalborg University](#)

*Citation for published version (APA):*

Mrachacz-Kersting, N., Kersting, U. G., de Brito Silva, P., Makihara, Y., Arendt-Nielsen, L., Sinkjaer, T., & Thompson, A. K. (2019). Acquisition of a Simple Motor Skill: Task-Dependent Adaptation and Long-Term Changes in the Human Soleus Stretch Reflex. *Journal of Neurophysiology*, 122(1), 435-446.  
<https://doi.org/10.1152/jn.00211.2019>

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# Acquisition of a Simple Motor Skill: Task-Dependent Adaptation and Long-Term Changes in the Human Soleus Stretch Reflex

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**Keywords:** human, stretch reflex, plasticity, operant conditioning

1 **Abstract**

2 Changing the H-reflex through operant conditioning leads to CNS multi-site plasticity and can affect  
3 previously learned skills. In order to further understand the mechanisms of this plasticity, we operantly  
4 conditioned the initial (M1) component of the soleus stretch reflex. Unlike the H-reflex, the stretch  
5 reflex is affected by fusimotor control, comprises several bursts of activity resulting from temporally  
6 dispersed afferent inputs, and may activate spinal motoneurons via several different spinal and  
7 supraspinal pathways.

8 Neurologically normal participants completed six baseline sessions and 24 operant conditioning sessions  
9 in which they were encouraged to increase (M1up) or decrease (M1down) M1 size. Five of eight M1up  
10 participants significantly increased M1; the final M1 size of those 5 participants was  $143 \pm 15\%$   
11 (mean $\pm$ SE) of the baseline value. All eight M1down participants significantly decreased M1; their final  
12 M1 size was  $62 \pm 6\%$  of baseline. Similar to the previous H-reflex conditioning studies, conditioned  
13 reflex change consisted of within-session task-dependent adaptation and across-session long-term  
14 change. Task-dependent adaptation was evident in conditioning session 1 with M1up and by session 4  
15 with M1down. Long-term change was evident by session 10 with M1up and session 16 with M1down.  
16 Task-dependent adaptation was greater with M1up than with the previous H-reflex up-conditioning. This  
17 may reflect adaptive changes in the muscle spindle sensitivity, which affects the stretch reflex but not the  
18 H-reflex. Because the stretch reflex is related to motor function more directly than the H-reflex, M1  
19 conditioning may provide a valuable tool for exploring the functional impact of reflex conditioning and  
20 its potential therapeutic applications.

21

22 **New & Noteworthy**

23 Since the activity of stretch reflex pathways contributes to locomotion, changing it through training may  
24 improve locomotor rehabilitation in people with CNS disorders. Here we show for the first time that  
25 people can change the size of soleus spinal stretch reflex through operant conditioning. Conditioned  
26 stretch reflex change is the sum of task-dependent adaptation and long-term change, consistent with H-  
27 reflex conditioning yet different from it in the composition and amount of the two components.

28

## 29 **Introduction**

30 In animals and humans, an operant conditioning protocol can increase or decrease the size of the  
31 Hoffman reflex (H-reflex), which is produced by a wholly spinal pathway (Wolpaw, 1987; Chen and  
32 Wolpaw, 1995; Carp et al., 2006; Thompson et al., 2009). Acquisition of this simple skill can alter the  
33 pattern of muscle activation during walking in people and animals with spinal cord injury (Chen et al.,  
34 2011; 2014; Thompson and Wolpaw, 2015). Accumulating evidence suggests induction of complex  
35 plasticity at many sites within the nervous system. Some of these changes clearly underlie the new skill  
36 of a larger or smaller H-reflex while others are likely to be compensatory changes that prevent the  
37 plasticity responsible for the new skill from interfering with pre-existing behaviors (Wolpaw and Lee,  
38 1989; Wolpaw, 2010).

39 The H-reflex is elicited by weak electrical stimulation of the muscle nerve; it is often viewed as an  
40 electrical analogue of the spinal stretch reflex, which can be increased or decreased by operant  
41 conditioning (Wolpaw et al., 1983c; Evatt et al., 1989). Stretch reflex conditioning has several  
42 advantages as a research model. First, the H-reflex is elicited mainly by synchronous activation of  
43 primary afferent (group-Ia and large group-II) fibers, thus the afferent volley is minimally dispersed  
44 when it arrives at the spinal motoneurons (Burke et al., 1983). In contrast, the stretch reflex can be  
45 elicited by a rapid joint rotation, which produces a temporally dispersed activation of afferent fibers, the  
46 same afferent may even fire several times (Matthews, 1972). The afferents are activated in a manner  
47 similar to that occurring during natural movement (e.g., the stretch reflex is less affected by presynaptic  
48 inhibition than is the H-reflex (Morita et al., 1998)). Second, unlike the H-reflex, the stretch reflex is  
49 affected by muscle spindle sensitivity, and thus by changes in gamma drive (Matthews, 1972; Arris and  
50 Henneman, 1980; Matthews 1981). This provides a stretch reflex conditioning protocol an additional  
51 mechanism for changing reflex size. Third, unlike the H-reflex, the stretch reflex comprises several  
52 successive peaks of excitation. The first (M1) is generated mainly by group Ia afferents. Later peaks (M2  
53 and M3) have contributions from Group Ib and II afferents (M2; (Schieppati and Nardone, 1997; Dietz,  
54 1998; Schieppati and Nardone, 1999; Dietz and Duysens, 2000; Grey et al., 2001; Sinkjaer et al., 2004;  
55 af Klint et al., 2010)) and from transcortical pathways (M3; (Marsden et al., 1973; 1977; Capaday et al.,  
56 1991; Palmer and Ashby, 1992; Petersen et al., 1998; Mrachacz-Kersting et al., 2006)). The pathways  
57 responsible for these later peaks contribute to normal movement. Thus, the impact of M1 conditioning  
58 on these pathways should also be monitored.

59 The present study set out to demonstrate the feasibility of operant conditioning of the soleus M1 stretch  
60 reflex, to characterize the time course of M1 stretch reflex changes in conditioning responders, and to  
61 compare those to the previous soleus H-reflex conditioning. It introduces a novel stretch reflex  
62 conditioning protocol for the human soleus muscle based on the H-reflex conditioning protocol of  
63 (Thompson et al., 2009). Each conditioning session began with a set of control trials in which feedback

64 was not provided. Thus, in contrast to previous stretch reflex conditioning studies in monkeys and  
65 humans (Wolpaw et al., 1983b; Wolpaw and O'Keefe, 1984; Evatt et al., 1989; Wolf and Segal, 1996),  
66 this study aimed to differentiate the task-dependent adaptation in reflex size that occurs within each  
67 conditioning session from the long-term change that develops over many sessions and affects pathway  
68 function outside of the conditioning paradigm. While task dependent adaptation is attributable to rapid  
69 plasticity in the cortex (Thompson et al., 2009), long-term change reflects plasticity in the spinal cord  
70 (Wolpaw, 1997; 2007). This study also assesses the effects of M1 conditioning on the M2 component of  
71 the stretch reflex. The results and their differences from H-reflex conditioning provide new insights into  
72 the mechanisms and wider effects of spinal reflex conditioning.

## 73 **Materials and Methods**

74 *Participants:* Fourteen participants (8 women, 6 men; ages 19-35 years) provided written informed  
75 consent for the study. Three participated in both up and down-conditioning protocols, with at least six  
76 months between the two protocols. Since up- and down-conditioning are physiologically different  
77 phenomena (Carp and Wolpaw, 1994; 1995; Carp et al., 2001a; 2001b; Wolpaw and Chen, 2001), the  
78 first direction of conditioning would not affect the second direction of conditioning (Thompson et al.,  
79 2009). All participants were free of any known physical or neurological disorders. Approval for the  
80 study was provided by the scientific ethics committee for Nordjylland (Reference Number: N-  
81 20120044). The study was performed in accordance with the Declaration of Helsinki.

82 *Operant conditioning study overview:* After attending a familiarization session (see *Familiarization*  
83 *session* below), each participant completed 6 baseline sessions and 24 up-conditioning or 24 down-  
84 conditioning sessions that occurred at a pace of 3 times per week. Figure 1 shows the operant  
85 conditioning session schedule and the set-up for stretch reflex elicitation. In each session, after preparing  
86 for the soleus and tibialis anterior (TA) EMG recording (see the section below), the soleus H-reflex/M-  
87 wave recruitment curve was obtained during natural standing with a stable level of soleus and TA  
88 background EMG activity. Then, 245 trials of stretch reflexes were elicited while the sitting participant  
89 produced approximately 10% maximum voluntary contraction (MVC) level of soleus EMG activity and  
90 no activation of the TA with the right lower leg fixed on the custom-made apparatus (Figure 1B) (see  
91 *Session protocol* below). In baseline sessions, all 245 reflexes were elicited without any feedback on  
92 reflex size. In contrast, in each conditioning session, the first 20 reflexes were elicited without any  
93 feedback on reflex size, and then 225 conditioning reflexes were elicited. In these 225 conditioning  
94 trials, the participant was asked to increase (up-conditioning) or decrease (down-conditioning) the size of  
95 M1 reflex with the aid of visual feedback, which showed after each perturbation whether the resulting  
96 reflex was larger (for up-conditioning) or smaller (for down-conditioning) than a criterion value. Soleus  
97 and TA background EMG levels were kept stable throughout data collection. In order to avoid session-  
98 to-session variability in the location of electrodes, the positions of all electrodes were mapped in relation  
99 to landmarks on the skin (e.g., moles or scars) during the familiarization session. To prevent the potential  
100 diurnal variation in reflex size from affecting the results, each participant's sessions always occurred at  
101 the same time of day (i.e., within the same 3-hr time window). A typical baseline or conditioning session  
102 took about 1.5 hour.

103 [Figure 1 near here]

104 *EMG recording:* Electromyographic (EMG) activity was recorded with custom-made amplifiers and  
105 surface Ag/AgCl electrodes (Medicotest 720-01-K) placed over the belly of the right soleus (SOL) and  
106 tibialis anterior (TA) muscles in accordance with the recommendations of (Cram and Criswell, 2011) to  
107 optimize recording from these muscles and avoid contamination from other muscles. EMG activity was

108 amplified using custom made EMG amplifiers, filtered at 20 Hz- 2kHz, and digitized (2 kHz) with  
109 scientific software Mr. Kick II 2.3 (Knud Larsen, Center for Sensory-Motor Interaction, Aalborg  
110 University, Denmark) and stored for later off-line analysis.

111 *Familiarization session:* All participants attended one familiarization session in which the experimental  
112 procedures were explained and implemented. This was to ensure that participants were comfortable with  
113 the electrical stimuli and perturbations that would elicit a stretch reflex (Figure 1C). During this session,  
114 the participants were asked to perform the maximal isometric voluntary SOL contraction (MVC) while  
115 standing. The instructions were to rise up on the toes as rapidly as possible and to hold this for 1 s. This  
116 was repeated twice and the best effort (quantified by the rectified SOL EMG amplitude) was defined as  
117 the MVC. Absolute soleus EMG amplitude range for 5-15% MVC (i.e., centering 10% MVC) level  
118 determined during the familiarization session was used for all stretch reflex measurements in the  
119 familiarization session and in all subsequent sessions. During stretch reflex trials, participants were  
120 asked to tonically activate the right SOL to produce this pre-set level of absolute EMG activity while  
121 sitting in a custom-made apparatus chair (Figure 1B) with their right TA silent. EMG electrode locations  
122 were mapped in relation to permanent marks on the skin (e.g., moles and scars) so that they could be  
123 maintained the same throughout the rest of the study for each participant.

124 *Custom joint-rotation device and stretch reflex elicitation:* During all stretch reflex measurements,  
125 participants were seated in a custom-made chair that was fixed to the floor, with their knee joint flexed at  
126 approximately 60° (Figure 1B). The right foot was fixed to a servo-controlled electrical actuator, such  
127 that the anatomical ankle axis of rotation was closely aligned with the fulcrum of the actuator and the  
128 foot rested on a footplate. This position minimized both hip and knee movement, ensuring that the  
129 movement of the actuator was transmitted solely to the ankle joint. This knee position also minimized  
130 the possible influence of gastrocnemius activity on the soleus stretch reflex; the gastrocnemius muscle is  
131 biarticular with two heads arising from just above the femoral condyles, and act both to flex the knee  
132 joint and to plantarflex the ankle joint. The left foot was placed on a custom-made plate that extended  
133 from the actuator such that the left leg was in the same starting position as the right leg. The angular  
134 position of the actuator was monitored with an angular displacement transducer (Transtek DC ADT  
135 series 600). To elicit the stretch reflex, 6° of dorsiflexion rotation was applied at 175°/s with randomly  
136 varying intervals of 5-7 s, when the participants had maintained a background SOL contraction of 5-15%  
137 MVC for at least 2 s.

138 *Session protocol:* In each of the baseline and conditioning sessions, first, an H-reflex/M-wave  
139 recruitment curve was measured. Then, 245 stretch reflex trials were performed. Exact session  
140 procedures are described here.

141 After EMG electrode placement, an H-reflex/M-wave recruitment curve was obtained while the  
142 participants stood upright and provided the pre-set level (i.e., 5-15% of MVC level, determined during

143 the familiarization session) of background activation in SOL. Using an isolated stimulator (Noxitest IES  
144 230), monopolar stimulation of the tibial nerve of the right leg was produced with the cathode (PALs  
145 Platinum round electrode, model 879100, 3.2 cm diameter, Axelgaard Man. Co. Ltd.) in the popliteal  
146 fossa and the anode (PALs Platinum rectangular electrode, model 895340, 7.5 x 10 cm, Axelgaard Man.  
147 Co. Ltd.) on the anterior aspect of the knee at the level of the patella. The cathode location was adjusted  
148 to maximize the soleus M-wave. Stimuli were delivered every 5–7 s if the background level of SOL  
149 activation had been maintained at the pre-set level (i.e., 5-15% MVC) for at least 2 s. Stimulus intensity  
150 was increased in 5-mA increments, with three to four stimuli at each level, until an M-wave [with the  
151 size >50  $\mu$ V] was observed; this was deemed the motor threshold (MT). Stimulus intensity continued to  
152 increase until M-wave peak-to-peak amplitude plateaued; this was defined as  $M_{\max}$ . Then, the scientific  
153 software Mr. Kick II 2.3 (Knud Larsen, Center for Sensory-Motor Interaction, Aalborg University,  
154 Denmark) was used to control the output of the stimulator such that 10 different stimulation intensities  
155 (up to that producing  $M_{\max}$ ) were applied randomly with three stimuli at each intensity. Stimuli were  
156 delivered every 5–7 s only if the background level of activation had been maintained at the required  
157 level for at least 2 s. The peak-to-peak values of the H-reflex and M-wave for each trial was extracted  
158 and the recruitment curves were constructed. Typically, the same range of stimulus intensities were used  
159 for all baseline and conditioning sessions. The same stimulus location was maintained throughout the  
160 study for each participant.

161 Next, the soleus stretch reflexes were elicited in one block of 20 control trials followed by three blocks  
162 of 75 control trials (in a baseline session) or 75 conditioning trials (in a conditioning session). During  
163 control trials, no feedback was provided as to M1 size, and participants were not asked to increase or  
164 decrease it. During conditioning trials (i.e., three sets of 75 trials in each of 24 conditioning sessions),  
165 participants were asked to either increase (M1up) or decrease (M1down) the size of the M1 component  
166 of the stretch reflex. Immediate visual feedback was provided indicating whether the trial was a success  
167 (i.e., whether M1 size was above (M1up) or below (M1down) a size criterion (Figure 1D)).

168 *Visual feedback:* Visual feedback provided to the participant is essentially the same as the one used in  
169 the previous H-reflex conditioning studies (Thompson et al., 2009; 2013; Makihara et al., 2014), except  
170 that the feedback targeted the M1 response, instead of the H-reflex.

171 A screen ~1.5 m in front of the participant provided visual feedback on the ongoing SOL EMG activity  
172 level (left) and the size of the M1 component of the SOL stretch reflex (right), which occurred typically  
173  $39 \pm 2$  ms after the onset of perturbation (Figure 1C). The background EMG panel (Figure 1D) was the  
174 same for both control and conditioning trials. The shaded area of the background EMG panel represented  
175 the target window (i.e., corresponding to the 5-15%MVC range, determined during the familiarization  
176 session), within which the SOL EMG activity had to be maintained prior to reflex elicitation. The bar

177 indicated the SOL EMG level in real-time and was updated every 100 ms; it was green if the EMG level  
178 stayed within the shaded area and red if it did not.

179 The M1 panel (Figure 1D) differed between control trials, during which the participant was not asked to  
180 modify M1 size, and conditioning trials, during which the participant was asked to increase (M1up) or  
181 decrease (M1down) M1 size. During control trials, the shaded area of the M1 panel indicating the range  
182 of M1 sizes that satisfy the reward criterion, was set as large as possible so that all trials with various M1  
183 sizes would be registered as “success”. During conditioning trials, this shaded area covered only the  
184 upper (for M1up) or lower (for M1down) portion of the panel; with M1up, the bottom border of the  
185 shade represented the reward criterion, while with M1down the top border represented the reward  
186 criterion. For how the reward criterion was calculated, see the paragraph below. When M1 size satisfied  
187 the criterion (i.e., when the top of the bar (i.e., M1 size) got in the shaded area), the bar was green,  
188 indicating success; when M1 size did not satisfy the criterion (i.e., the top of the bar got out of the  
189 shaded area), the bar was red, indicating failure. This feedback appeared 200 ms after the imposed ankle  
190 rotation began.

191 As described in previous studies (Thompson et al., 2009; 2013; Makihara et al., 2014), the reward  
192 criterion level was based on the average reflex size for the previous block of trials. Thus, in each  
193 conditioning session, the criterion level for the first block of 75 conditioning trials was based on the  
194 immediately preceding block of 20 control trials, and the criterion levels for the second and third blocks  
195 of conditioning trials were based on the immediately preceding block of 75 conditioning trials. The  
196 criterion level was calculated such that if M1 sizes for the new block were similar to those for the  
197 previous block, 50–60% of the trials would be successful (Chen and Wolpaw, 1995; Thompson et al.,  
198 2009). The thick horizontal line represents the average M1 size for the six baseline sessions. Thus, the  
199 participants also received information as to their current performance in relation to their average initial  
200 M1 size. The percentage of successful trials within the current block was displayed at the bottom of the  
201 screen and updated after each trial, while the number of completed trials was shown at the top of the  
202 screen.

203 *Data analysis:* To calculate M1 size for each participant’s session, M1 size was defined as a root mean  
204 square (RMS) value of the rectified SOL EMG in the M1 window minus an RMS value for 100 ms of  
205 pre-perturbation period. For each participant, the M1 window was determined as a 10-ms window  
206 including the M1 peak (the first peak response that occurred around 45 ms after perturbation onset) by  
207 visual inspection (e.g., Figure 1C). Then, the average size was calculated for the 20 within-session  
208 control trials, each block of 75 trials, and for all three blocks of 75 trials together. Values were expressed  
209 as a percentage of their average values for the six baseline sessions. The size of M2, the second set of  
210 peak responses that occur around 60 ms post perturbation onset, was also calculated in a similar way.

211 The M2 window was typically around 58-68 ms after perturbation onset, whereas a typical M1 window  
212 was around 42-52 ms (see Figure 1C).

213 To determine for each participant if the conditioning (M1up or M1down) had been successful, the  
214 average M1 size for the three 75-trial blocks of conditioning trials in the last six conditioning sessions  
215 (sessions 19-24) were compared to that for the three blocks of 75 control trials of the six baselines by a  
216 single-tail *t*-test. A significant change in M1 size ( $p < 0.05$ ) in the direction of conditioning (i.e., increased  
217 for M1up, decreased for M1down) defined successful conditioning.

218 Regardless of whether the data are from a baseline session or a conditioning session, for each participant,  
219 M1 sizes from all three 75-trial blocks were averaged together and called “conditioned M1,” and M1  
220 sizes from the 20 control trials were averaged together and called “control M1.” The final effect of the  
221 protocol on the conditioned M1 size was calculated by averaging the M1 size for the three 75-trial  
222 blocks of conditioning sessions 22–24 and expressing the value as a percentage of the average M1 size  
223 for the three 75-trial blocks of the six baseline sessions. The final effect on the control M1 size was  
224 calculated by averaging the M1 size for the 20 control trials of conditioning sessions 22–24 and  
225 expressing the value as a percentage of the average M1 size for the first 20 trials of the six baseline  
226 sessions. To assess the time course of changes, a repeated-measures ANOVA was used to evaluate  
227 conditioned and control M1 sizes across successive 6-session bins (i.e., baseline sessions 1–6 and  
228 conditioning sessions 1–6, 7–12, 13–18, and 19–24). Comparable procedures were used to assess the  
229 impact of the conditioning protocol on the M2 component of the stretch reflex. This procedure was  
230 chosen over characterizing the learning via a function, based on the inter-session variability of the  
231 reflexes.

232 In order to assess the session-to-session variability in EMG recording condition, the peak-to-peak  $M_{\max}$   
233 and the peak-to-peak  $H_{\max}$  were calculated from the recruitment curve measured at the beginning of each  
234 session. To assess the stability of background EMG activity across sessions, the SOL and tibialis anterior  
235 background EMG were calculated for each session, along with M1 and M2 sizes. These values were  
236 evaluated with a repeated-measures ANOVA, in the same way as the time course evaluation for M1 and  
237 M2 changes.

238

## 239 Results

### 240 Stability of the $M_{\max}$ , $H_{\max}$ , and background EMG

241 In order to ensure that M1 changes over sessions were not due to inter-session differences in electrode  
242 placements, we measured the  $M_{\max}$  at the beginning of every session. Across all M1up participants, the  
243  $M_{\max}$  averaged  $7.7 \pm 0.2$  (SE) mV during the baseline sessions and  $7.7 \pm 0.2$  mV during the conditioning  
244 sessions. One-way repeated measures ANOVA revealed no significant difference across the sessions  
245 ( $F_{(4,28)} = 0.91$ ,  $p = 0.47$ ). Similarly, the M1down group showed no significant difference across sessions  
246 ( $F_{(4,28)} = 1.01$ ,  $p = 0.42$ ;  $M_{\max}$  baseline sessions:  $6.5 \pm 0.8$  mV; conditioning sessions:  $6.5 \pm 0.8$  mV).  $H_{\max}$   
247 did not change significantly across sessions for either the M1up ( $F_{(4,28)} = 0.899$ ,  $p = 0.48$ ) or the M1down  
248 group ( $F_{(4,28)} = 0.47$ ,  $p = 0.76$ ); it averaged  $3.3 \pm 0.2$  mV for M1up and  $2.8 \pm 0.4$  mV for M1down during  
249 the baseline sessions and  $3.5 \pm 0.2$  mV for M1up and  $2.8 \pm 0.2$  mV for M1down during the conditioning  
250 sessions. SOL background EMG also remained stable throughout the study;  $F_{(4,28)} = 0.335$ ,  $p = 0.852$  for  
251 the M1up group;  $F_{(4,28)} = 0.338$ ,  $p = 0.850$  for the M1down group, by one-way repeated measures  
252 ANOVA. TA background EMG remained at resting level (i.e.,  $< 5 \mu\text{V}$ ) in both groups throughout the  
253 study. Overall, SOL  $M_{\max}$  and  $H_{\max}$ , and SOL and tibialis anterior background EMG values all remained  
254 within  $\pm 10\%$  of the baseline values throughout the study. Stability of these values in the M1up and  
255 M1down groups of participants in whom conditioning was successful is displayed in Figure 2 and  
256 summarized in Table 1.

257 [Figure 2 and Table 1 near here]

### 258 M1 and M2 stability in the baseline sessions

259 All participants completed six baseline sessions in each of which 245 control reflexes were elicited.  
260 There was no significant difference in average M1 size between the initial 20 control trials and the  
261 subsequent 225 control trials in either the M1up or M1down group (all participants included, two-tailed  
262 paired t-test,  $p = 0.99$  for each group). M1 size did not differ significantly among the three 75-trial  
263 blocks nor across the baseline sessions (block  $\times$  session interaction,  $p = 0.13$  (M1Up) and  $p = 0.99$   
264 (M1down)).

265 There was no significant difference in average M2 size between the initial 20 trials and the subsequent  
266 225 trials in either the M1up or M1down (two-tailed paired t-test,  $p = 0.99$  for both types of training).  
267 M2 size did not differ significantly among the three 75-trial blocks nor across the baseline sessions  
268 (block  $\times$  session interaction,  $p = 0.41$  (M1Up group) and  $p = 0.47$  (M1down group)).

### 269 The effect of conditioning on the size of the conditioned M1 reflex

270 As noted above, for each session, the data for the three 75-trial blocks were combined to calculate the  
271 average conditioned M1 reflex size, which is referred to as “the conditioned M1 size” and is expressed

272 as a percentage of the participant's average M1 size for the three 75-trial blocks of the six baseline  
273 sessions. Across all participants (i.e., N=8 for M1up and N=8 for M1 down) there was a significant  
274 change between the baseline session and the last six conditioning sessions (paired t-test, one-tailed,  $p =$   
275  $0.02$  and  $p < 0.001$  for M1up and M1 down respectively). By the analysis described in Methods,  
276 conditioning was successful in five of eight M1up participants and in all eight M1down participants. In  
277 the other three M1up participants, M1 size did not change significantly. Of the three participants who  
278 completed both the M1up and M1down protocols, two were successful in both while one was  
279 unsuccessful in M1up conditioning and subsequently (>6 months later) was successful in M1down  
280 conditioning.) Figure 3A shows the final conditioned M1 sizes (defined as the average conditioned M1  
281 sizes for sessions 22-24) for the eight M1up and the eight M1down participants. The filled symbols  
282 represent the successful and the open symbols the unsuccessful participants. Since the main aim of this  
283 study was to characterize the time course of M1 and M2 changes in the responders of M1 conditioning,  
284 the rest of this presentation focuses primarily on the data from the 13 successful participants (5 M1up  
285 and 8 M1 down).

286 [Figure 3 near here]

287 The bottom panels of Figure 4 show the average rectified SOL EMG data of the three 75-trial blocks for  
288 one baseline session and the last conditioning session in single participants. The top panels show the  
289 ankle angle during these stretch reflex trials. Figure 5A summarizes the time course of M1 size changes  
290 for the successfully conditioned participants of both groups; each symbol indicates the group average  
291 ( $\pm$ SE) for each session. The conditioned M1 size increased in the M1up group ( $F_{(4,16)} = 4.46$ ,  $p = 0.013$ ;  
292 and decreased in the M1down group ( $F_{(4,28)} = 18.79$ ,  $p < 0.001$ ) (top panel). The final conditioned M1  
293 size was  $143 \pm 15$ SE % for the M1up group and  $62 \pm 6$ % for the M1down group. To aid in assessing the  
294 time course of M1 changes, the time course of H-reflex changes using data from (Thompson et al., 2009)  
295 is displayed in Figure 5B.

296 [Figure 4 and 5 near here]

### 297 **The effect of conditioning on the size of the control M1 reflex**

298 As defined in Methods, the M1 reflex obtained in the first 20 control trials of the control sessions and the  
299 20 control trials of the conditioning sessions is referred to as the "control M1." The middle panel of  
300 Figure 5A shows the time course of control M1 size changes across six baseline and 24 conditioning  
301 sessions for the M1up and M1down participants; each symbol represents the group mean ( $\pm$ SE) for each  
302 session, expressed as a percent of its baseline value. The control M1 size increased in the M1up group  
303 ( $F_{(4,16)} = 3.09$ ,  $p = 0.046$ ; and decreased in the M1down group ( $F_{(4,28)} = 6.72$ ,  $p = 0.001$ ). The final control  
304 M1 size was  $126 \pm 18$ (SE)% of baseline for the M1up group, and  $75 \pm 8$ % of baseline for the M1down  
305 group.

306 It appears that the control M1 requires more time to change, compared to the conditioned M1 reflex. For  
307 the M1up group, the conditioned M1 size was consistently above baseline values from conditioning  
308 Session 1 on, while the control M1 did not exceed the baseline value until Session 10. For the M1down  
309 group, the conditioned M1 was consistently below the baseline by Session 4 while it was not until  
310 Session 16 that the control M1 was consistently below baseline. Although the onset was delayed, the  
311 control M1 change was obvious towards the end. Figure 6 shows a typical example of control M1  
312 change with M1down conditioning.

313 [Figure 6 near here]

### 314 **Within-session task-dependent adaptation in the M1 reflex**

315 To quantify the task-dependent change in the M1 reflex from control trials to conditioning trials, for  
316 each session, the control M1 size was subtracted from its conditioned M1 size (the bottom panel, Figure  
317 5A). A within-session difference in the correct direction (i.e., positive for the M1up group and negative  
318 for the M1down group) is referred to as task-dependent adaptation (Thompson et al., 2009). It appears in  
319 session 1 for M1up participants and by session 4 for M1down participants, and it remains about the same  
320 over the remaining sessions. Its average final (sessions 22-24) values are  $24 \pm 9\text{SE} \%$  and  $-15 \pm 4\%$  for the  
321 M1up and M1down groups, respectively. Notably, the amount of task-dependent adaptation with up-  
322 conditioning was larger with M1up than with HRup ( $p=0.01$  by unpaired t-test for sessions 1-24). Figure  
323 7 shows an example of within-session task dependent adaptation in a typical M1up participant.

324 [Figure 7 near here]

325 Table 1 summarizes for the M1up and M1down participants the changes in control and conditioned M1  
326 reflexes and their within-session difference (i.e., task-dependent adaptation) across the conditioning  
327 sessions grouped into four 6-session bins (C1-6, C7-12, C13-18 and C19-24). To delineate the  
328 similarities and differences between M1 conditioning and H-reflex conditioning, the data from this study  
329 are presented with the previous H-reflex conditioning data (Thompson et al., 2009). Significant  
330 differences from the average of the six baseline sessions are indicated by asterisks. Table 1 verifies the  
331 control/conditioned M1 differences in the onset of the impact of conditioning. For the M1up group the  
332 within-session difference is significant from the first bin of 6 sessions on, while the conditioned M1 is  
333 significantly different from the second bin of 6 sessions and the control M1 is not significantly different  
334 until the fourth (and final) bin. For the M1down group, the within-session difference is significant from  
335 the third 6-session bin, the conditioned M1 is significantly smaller from the second 6-session bin and the  
336 control M1 is not significantly different until the fourth bin.

### 337 **The effect of M1 conditioning on the size of the M2 reflex**

338 Similar to the M1 size calculation, the M2 values from the three 75-trial blocks were combined to  
339 calculate the average M2 size for each conditioning session; this is referred to as “the conditioned M2”

340 size and expressed as a percentage of the participant's average M2 size across the six baseline sessions.  
341 M1 conditioning changed M2 size in the direction of M1 conditioning in 4 of the 8 M1up participants  
342 and in 5 of the 8 M1down participants. For the whole group, the final conditioned M2 did not change  
343 significantly in the successful M1up group (N=5,  $p = 0.28$ , by paired t-test) or successful M1down group  
344 (N=8,  $p = 0.16$ ), (Figure 3B). Final conditioned M2 size averaged  $120 \pm 32\%$  for the successful M1up and  
345  $89 \pm 10\%$  for the successful M1down group, respectively. Final control M2 size was  $118 \pm 32\%$  for the  
346 successful M1up and  $99 \pm 5\%$  for the successful M1down group, respectively. The final M2 within-  
347 session change was  $2 \pm 14\%$  and  $-10 \pm 9\%$  for the M1up and M1down groups, respectively. Table 2  
348 summarizes the changes in conditioned M2, control M2, as well as M2 within-session change across the  
349 four 6-session bins of conditioning sessions (C1-6, C7-12, C13-18 and C19-24).

350 [Table 2 near here]

351 **Discussion**

352 This is, to our knowledge, the first demonstration of operant conditioning of the M1 component of the  
353 human soleus stretch reflex. The reflex changed without alterations in background EMG, initial muscle  
354 length, or imposed perturbation. M1up conditioning was successful in five of eight participants and  
355 M1down conditioning in all eight participants. M1 size increased to  $143 \pm 15\%$  of its initial value in the  
356 successful M1up participants and decreased to  $62 \pm 6\%$  in the M1down participants. These success rates  
357 are similar to those for soleus H-reflex and biceps stretch reflex conditioning in animals (Wolpaw, 1983;  
358 Wolpaw et al., 1983a; Wolpaw, 1987; Chen and Wolpaw, 1995; Carp et al., 2005) and humans (Evatt et  
359 al., 1989; Wolf and Segal, 1996; Thompson et al., 2009).

360 The results are particularly notable in two respects. First, they confirm the two-phase acquisition of an  
361 operantly conditioned spinal reflex increase or decrease, hypothesized in 1984 (Wolpaw and O'Keefe,  
362 1984) and first documented in 2009 (Thompson et al., 2009). Second, they assess the impact of M1  
363 conditioning on the M2 component of the stretch reflex. Because the stretch reflex is more directly  
364 related to motor function than the H-reflex, both of these contributions illuminate the implications of  
365 spinal reflex conditioning for understanding normal motor function and for developing protocols that can  
366 address the reflex abnormalities associated with spinal cord injury, stroke, or other chronic disorders.

367 **Task-dependent adaptation and long-term change in M1**

368 As in soleus H-reflex conditioning (Thompson et al., 2009), the data reveal a two-phase phenomenon;  
369 task-dependent adaptation within conditioning sessions (phase 1) and long-term change across  
370 conditioning sessions (phase 2). However, the time courses of these two phases differed from those  
371 found for the soleus H-reflex (Thompson et al., 2009). The task-dependent adaptation for a conditioning  
372 session was defined as the conditioned M1 size (M1 size for the 225 conditioning trials) minus the  
373 control M1 size (M1 size for the 20 control trials). Thus, it shows the immediate effect of asking the  
374 participant to increase (or decrease) M1 and providing immediate feedback as to whether the size  
375 criterion was met. In contrast, long-term change was indicated by the increase (or decrease) in the  
376 control M1 size over conditioning sessions. Thus, it assesses the persistent effect of the conditioning  
377 sessions.

378 In the present results, task-dependent adaptation first appeared in conditioning session one (M1up) or  
379 four (M1down) and remained stable over the remaining sessions. Long-term change appeared in session  
380 10 (M1up) or session 16 (M1down) and grew gradually over the remaining sessions. The difference  
381 between the M1up and M1down groups in the onset of task specific adaptation and long-term change  
382 (also seen for soleus H-reflex conditioning (Thompson et al., 2009)) is further evidence that up-  
383 conditioning and down-conditioning are not mirror images of each other but have different mechanisms  
384 (Wolpaw, 2007; Thompson et al., 2009). The development of the two phases over the course of up- and

385 down-conditioning with M1 or H-reflex conditioning (Thompson et al., 2009) are summarized in Table  
386 1. M1up conditioning produces greater task-dependent adaptation and less long-term change (although  
387 insignificant) than does H-reflex up-conditioning; in contrast, M1down conditioning produces  
388 comparable task-dependent adaptation and greater (although insignificant) long-term change compared  
389 to H-reflex down-conditioning. Overall, for both task-dependent adaptation and long-term change, the  
390 time courses of development are similar for M1 and H-reflex conditioning.

391 How do we interpret the differences and similarities between M1 and H-reflex conditioning? The H-  
392 reflex is referred to as the electrical analog of the M1 stretch reflex. A principal difference is that the H-  
393 reflex bypasses the muscle spindle while M1 is affected by the sensitivity of the spindle and thus by  $\gamma$ -  
394 motoneuron activity. This may constitute an extra degree of freedom available for participants as they  
395 learn to change M1 size. Muscle spindle excitation is affected by initial muscle length (Matthews, 1972)  
396 and muscle background activity (Marsden et al., 1976; 1983). Both of these variables were maintained  
397 stable throughout the study; thus, it is unlikely that they contributed to the changes. It is possible that  
398 alterations in  $\gamma$ -motoneuron activity contributed to M1 change, as suggested for monkey biceps M1  
399 conditioning (Wolpaw and O'Keefe, 1984).

400 Another potential site of change is the Ia-synapse, known to be modulated by presynaptic inhibition  
401 (Eccles et al., 1962; Stein, 1995). This inhibition is influenced by corticospinal, reticulospinal and  
402 vestibulospinal pathways (Iles, 1996; Meunier and Pierrot-Deseilligny, 1998; Pierrot-Deseilligny and  
403 Burke, 2012; Baldissera et al., 1981) and is task-dependently modulated (Hultborn et al., 1987; Hultborn  
404 and Meunier, 1987; Stein and Capaday, 1988; Stein, 1995; Côté and Gossard, 2003). Morita et al.  
405 (Morita *et al.*, 1998) reported evidence that the stretch reflex is less sensitive to presynaptic inhibition  
406 than the H-reflex; this is not consistent with the present finding that the conditioned M1 change is  
407 greater than the H-reflex change. In the present study, M1up produced a larger within-session M1  
408 increase (task-dependent adaptation), for which the most plausible mechanism is a change in presynaptic  
409 inhibition (Capaday and Stein, 1987). Indeed, a release of presynaptic inhibition at the segmental level  
410 has been implicated to be a mechanism responsible for the increase in H-reflex and stretch reflex size  
411 during the Jendrassik maneuver (Dowman and Wolpaw, 1988; Zehr and Stein, 1999). However, in the  
412 current study, participants were instructed to maintain the upper body (including facial muscles) relaxed  
413 throughout all the reflex trials. Thus, changes in presynaptic inhibition via the Jendrasski maneuver are  
414 unlikely to have occurred here. In addition to presynaptic inhibition, animal studies, which would  
415 capture long-term physiological and/or anatomical changes, provide substantial evidence of altered  
416 motoneuron properties, in GABAergic terminals on motoneurons, and possibly in oligosynaptic afferent  
417 pathways (Wolpaw, 1997; 2010; Thompson and Wolpaw, 2014). Further studies are needed to identify  
418 the mechanisms underlying M1 and H-reflex conditioning and the extent to which they differ.

419 **The effect on the M2 component of the soleus stretch reflex**

420 For M1up and M1down groups, the changes in M1 did not have large or consistent effects on M2 size.  
421 This differs from stretch reflex conditioning in the biceps brachii M1, which produced significant  
422 changes in M2 (Wolf et al., 1995). Since the M2 of upper limb muscles is likely generated through  
423 transcortical pathways (Crago et al., 1976; Marsden et al., 1977; Rothwell et al., 1986; Goodin et al.,  
424 1990; Capaday et al., 1991; Thilmann et al., 1991; Palmer and Ashby, 1992), it might be more  
425 comparable to the M3 for lower limb muscles (Petersen et al., 1998; Mrachacz-Kersting et al., 2006).  
426 (Note that the M3 was not measurable in the present study in which the conditioning protocol was  
427 administered in sitting participants.) The M2 of the soleus muscle is thought to be largely mediated by  
428 group Ib and/or group II afferents (Schieppati and Nardone, 1997; Dietz, 1998; Schieppati and Nardone,  
429 1999; Dietz and Duysens, 2000; Grey et al., 2001; Sinkjaer et al., 2004; af Klint et al., 2010). Thus, the  
430 soleus M2 is not comparable to the M2 of the biceps brachii.

431 A possible explanation why M2 did not change consistently, is a potentially mixed origin of M2.  
432 Because the distinction between M1 and M2 is based on their latencies, there is a possibility that delayed  
433 Ia excitation of motoneurons may contribute to M2, in addition to the excitation from Ib and/or group II  
434 afferents. With M1up conditioning, such delayed Ia excitation could decrease, as those motoneurons  
435 could start firing in the M1 time window. This explanation seems feasible in some, but not all  
436 participants, however. Another explanation is related to the functional relevance of M2 during the sitting  
437 task in the current study. It is unlikely that the M1 or M2 in the soleus would have significant function in  
438 the sitting posture, thus changes in M2 would not necessarily reflect systematic changes (compensatory  
439 or reactive plasticity (Wolpaw, 2010)). Future studies should condition the soleus stretch reflex during  
440 more functional tasks, such as standing as has been done for the soleus H-reflex conditioning (Thompson  
441 et al., 2009; Makihara et al., 2014).

#### 442 **Functional Implications**

443 Several types of afferents generate the stretch reflex; Group Ia and II afferents arising from muscle  
444 spindles, Group Ib afferents from Golgi tendon organs, and cutaneous afferents. Providing perturbations  
445 such as the ankle joint rotations used here, is one way to probe these pathways and quantify their role  
446 during tasks such as walking. These perturbations activate the afferents in a manner similar to what  
447 occurs during normal behaviour: they are dispersed in time and the same afferents may be activated  
448 several times. Additionally, and unlike the H-reflex, the stretch reflex size is affected by the sensitivity  
449 of the muscle spindle, and thus by descending gamma drive. Conditioning of the stretch reflex thereby  
450 provides a more natural paradigm into the adaptations of the underlying circuitry.

451 Appropriate soleus H-reflex conditioning leads to a return to a more normal gait pattern in spinal cord  
452 injured animals and humans (Chen et al., 2006; Manella et al., 2013; Thompson et al., 2013). This  
453 favourable effect on locomotion results from the H-reflex changes triggering much wider beneficial  
454 plasticity (Thompson et al., 2013; Thompson and Wolpaw, 2014). While the H-reflex depends mainly on

455 the Ia afferent pathway, Group Ib and II afferents appear to play a prominent role in generating soleus  
456 locomotor activity in humans (Schieppati and Nardone, 1997; Dietz, 1998; Schieppati and Nardone,  
457 1999; Dietz and Duysens, 2000; Grey et al., 2001; Sinkjaer et al., 2004; af Klint et al., 2010). Thus,  
458 operant conditioning of the soleus stretch reflex, which engages these other pathways, might prove to be  
459 a more efficient and/or effective therapeutic approach than H-reflex conditioning.

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603 **Funding:** This work was supported in part by Villum Kann Rasmussen Foundation and Spar Nord Fond  
604 of Denmark, the US National Institute of Neurological Disorders and Stroke [NS069551 to AKT], and  
605 the US National Institute of General Medical Sciences [GM104941, Institutional Development Award  
606 (IDeA) to Binder-MacLeod].

607 **Acknowledgements:** The authors would like to thank Dr. Jonathan R. Wolpaw for providing his  
608 invaluable advice and comments on the manuscript on behalf of the National Center for Adaptive  
609 Neurotechnologies (NIH/NIBIB, P41EB018783).

610

611 **Table 1:** Soleus and tibialis anterior background EMG during stretch reflex trials and the soleus  $M_{\max}$   
 612 and  $H_{\max}$  values during standing for each of successive 6-session blocks.

	Group	B1-6	C1-C6	C7-C12	C13-C18	C19-C24
<b>Soleus EMG (<math>\mu\text{V}</math>)</b>	M1up	15.8±1.7	15.8±1.4	15.2±1.6	14.9±1.7	15.6±1.8
	M1down	16.4±2.8	16.4±2.7	15.7±2.6	16.3±2.8	16.1±2.5
<b>TA EMG (<math>\mu\text{V}</math>)</b>	M1up	3.6±0.4	4.2±0.2	3.9±0.3	3.8±0.4	4.0±0.5
	M1down	3.6±0.3	3.9±0.3	3.8±0.4	3.8±0.4	3.7±0.4
<b>Soleus <math>M_{\max}</math> (mV)</b>	M1up	8.1±0.6	8.2±0.7	8.5±0.6	8.4±0.7	8.4±0.7
	M1down	6.7±0.9	6.7±0.9	6.7±0.9	6.6±0.9	6.7±0.9
<b>Soleus <math>H_{\max}</math> (mV)</b>	M1up	3.9±0.5	4.1±0.6	4.3±0.5	4.3±0.6	4.3±0.7
	M1down	2.7±0.4	2.7±0.6	2.7±0.6	2.6±0.5	2.6±0.5

613 All values are mean  $\pm$  SE for successful M1up or M1down participants, and are expressed as percentage  
 614 of baseline values. None of the values from conditioning sessions are significantly different from the  
 615 values from baseline sessions.

616

617

618 **Table 2:** M1 reflex values for all successful M1up and M1down participants compared to H-reflex  
 619 values for all successful HRup and HRdown participants from the study by Thompson et al. (2009) for  
 620 each of successive 6-session blocks.

	Group	C1-C6 (%)	C7-C12 (%)	C13-C18 (%)	C19-C24 (%)
<b>Conditioned reflex</b>	M1up	116.9±6.8*	136.8±12.7*	145.7±22.4*	141.2±17.2*
	HRup	115.6±6.2	122.4±5.5*	127.6±7.5*	137.3±8.6*
<b>Control reflex</b>	M1up	96.6±4.3	109.5±5.6	118.4±11.2*	117.2±10.9*
	HRup	106.4±6.0	106.7±3.7	116.5±4.9	128.2±5.4*
<b>Within-session change</b>	M1up	20.3±6.2*	27.2±8.5*	27.3±12.5*	24.0±7.9*
	HRup	9.2±5.4	15.6±4.1*	11.6±4.2*	-12.4±6.0*
<b>Conditioned reflex</b>	M1down	92.9±5.1	84.9±6.2*	74.4±5.9*	62.1±7.1*
	HRdown	93.4±4.0	81.7±4.4*	75.1±4.9*	72.3±5.3*
<b>Control reflex</b>	M1down	100.0±5.8	96.8±4.2	89.2±6.5	77.5±5.5*
	HRdown	97.1±1.8	95.5±2.8	90.5±4.5	86.7±5.9*
<b>Within-session change</b>	M1down	-7.5±3.8	-11.9±5.2	-14.8±5.0*	-15.3±4.0*
	HRdown	-3.8±3.2	-13.9±3.2*	-14.4±5.1*	-14.4±6.3*

621 Values represent the average  $\pm$  SE and are expressed as percentage of baseline values.

622 \* Significant differences from the six baseline sessions ( $p < 0.05$ , LSD *post hoc* after repeated measures  
 623 ANOVA).

624 \* H-reflex values are from Thompson et al., (2009).

625

626 **Table 3:** M2 stretch reflex values for all successful M2up and M2down participants for each of  
 627 successive 6-session blocks.

	Group	C1-C6 (%)	C7-C12 (%)	C13-C18 (%)	C19-C24 (%)
<b>Conditioned M2 reflex</b>	M1up	119.4±17.5	126.3±30.1	126.8±28.2	122.7±23.6
	M1down	110.4±7.7	104.8±11.5	97.5±9.3	89.9±9.6
<b>Control M2 reflex</b>	M1up	111.2±14.6	129.1±30.9	139±40.2	124.4±26.3
	M1down	111.4±3.9	110.5±7.7	100.1±8.3	97.4±6.5
<b>Within-session change</b>	M1up	8.2±9.3	-2.8±12.4	-12.2±16.5	-1.7±11.6
	M1down	-1.0±5.7	-5.7±4.8	-2.6±8.3	-7.5±6.8

628 Values represent the average  $\pm$  SE and are expressed as percentage of baseline values.

629 \* Significant differences from the six baseline sessions ( $p < 0.05$ ).

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634 **Figure Legends**

635 **Figure 1: (A)** Study session schedule. Six baseline sessions are followed by 24 conditioning sessions, all  
636 at pace of 3 sessions per week. **(B)** The stretch reflex pedal. Participants are seated comfortably with  
637 both feet on separate foot plates. **(C)** The upper panel shows the change in ankle angle in degrees, the  
638 lower panel depicts the activity of the soleus muscle following a single imposed dorsiflexion  
639 perturbation. Several peaks may be seen labelled as M1 and M2. The grey shaded area visualizes the  
640 time window for the M1 for which the participants received feedback during the conditioning sessions.  
641 **(D)** Visual feedback. The feedback on the screen is comprised of two parts, the background EMG and  
642 the stretch reflex size both shown as bars. The shaded area in the left panel represents the pre-set range  
643 for the soleus background activity, which must be maintained for at least 2s by the participant for a  
644 stretch reflex trial to occur. The shaded area in the right panel represents the targeted range for the size  
645 of M1 component. During control trials, this right shaded area is set as large as possible since the  
646 participant is not training to modify the M1 size. During M1up conditioning trials, this shaded area  
647 appears in the upper half (i.e., a criterion level and above), based on the baseline sessions. In contrast,  
648 during M1down conditioning trials, this area appears in the lower half (i.e., a criterion level and below).  
649 Immediately after a stretch reflex trial occurs (i.e., 200 ms after perturbation onset), a vertical bar  
650 reflecting M1 size is displayed. When the bar height falls within the shaded area, the participant had a  
651 successful conditioning trial and the bar is depicted as green. If the bar height falls out of the shaded  
652 area, the bar becomes red and the trial is registered as an unsuccessful trial. This provides immediate  
653 feedback on M1 size to the participant for each single trial performed.

654 **Figure 2:** Soleus and TA background EMG and the soleus  $M_{\max}$  and  $H_{\max}$  values for all baseline and  
655 conditioning sessions in M1up **(A)** and M1down **(B)** participants in whom conditioning was successful.  
656 Each set of a symbol and error bars represents the average ( $\pm$ SE) value for successfully conditioned  
657 participants.  $N=5$  for M1up **(A)** and  $N=8$  for M1down **(B)**. Circles are for the soleus background EMG  
658 amplitude (in  $\mu$ V), squares are for the TA background EMG (in  $\mu$ V), diamonds are for the  $M_{\max}$  (in mV),  
659 and crosses are for the  $H_{\max}$  (in mV).

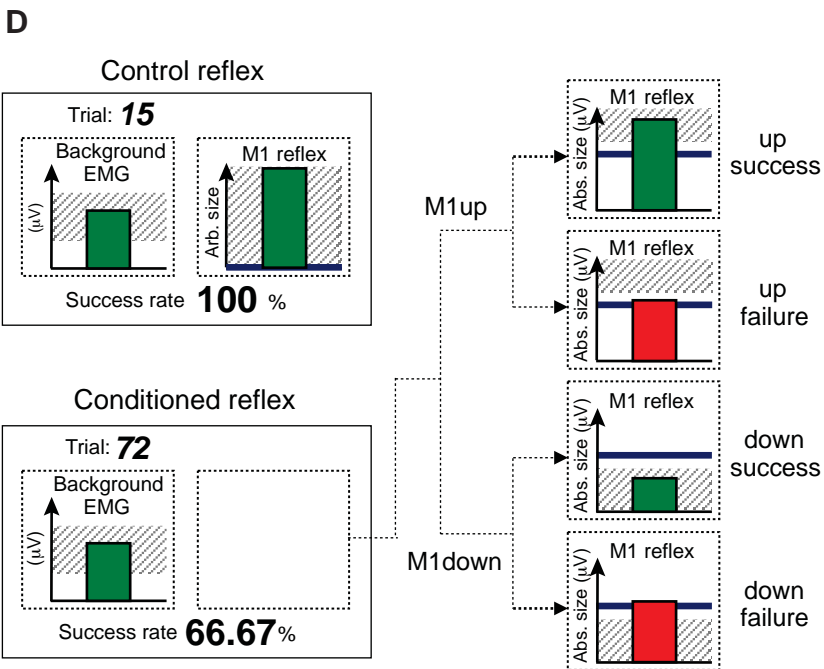
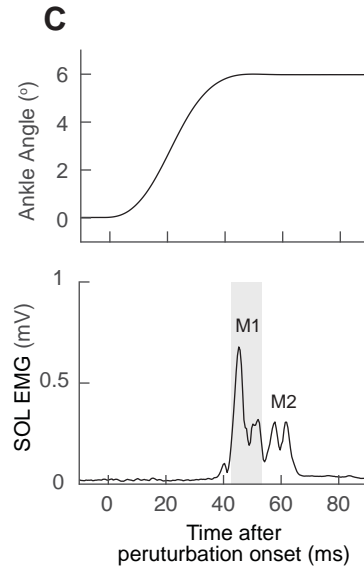
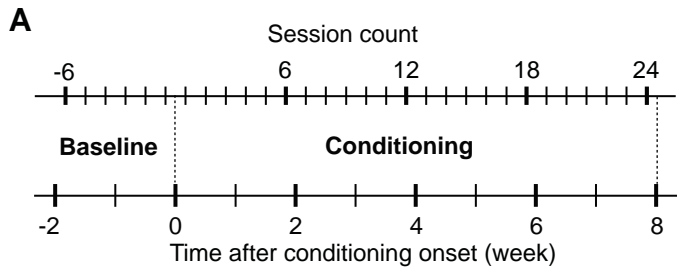
660 **Figure 3: (A)** The final conditioned M1 size for individual participants. The filled symbols represented  
661 successful participants in whom the average conditioned M1 for conditioning sessions 19-24 was  
662 significantly increased (5 of 8 M1up participants, upward triangles) or decreased (8 of 8 M1down  
663 participants, downward triangles) compared to the average baseline M1. The open symbols show the  
664 three unsuccessful participants (i.e., 3 of 8 M1up participants). **(B)** The final conditioned M2 size for  
665 individual participants. As for (A), the filled symbols represented the successful participants in whom  
666 the average conditioned M1 for conditioning sessions 19-24 was significantly increased (5 of 8 M1up  
667 participants, upward triangles) or decreased (8 of 8 M1down participants, downward triangles) compared  
668 to the average baseline M1.

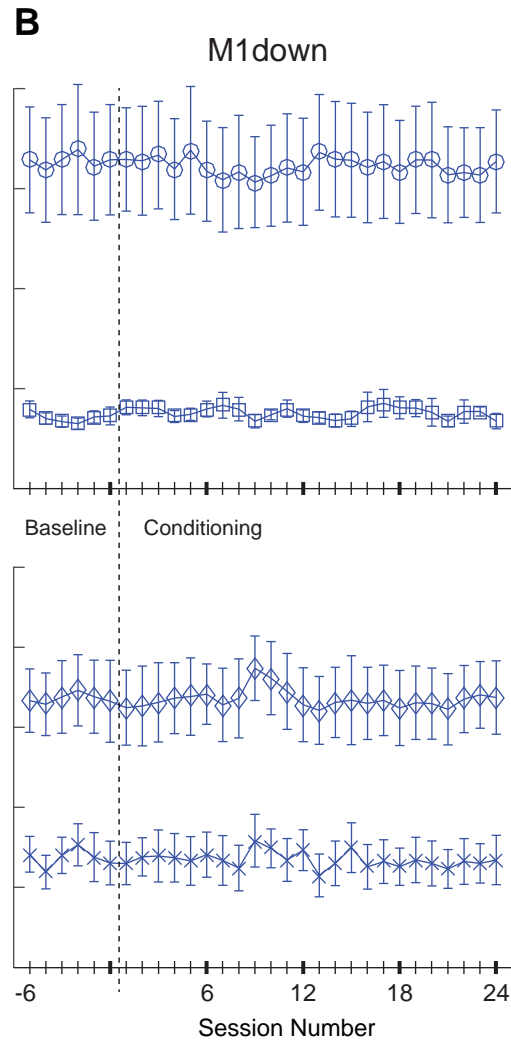
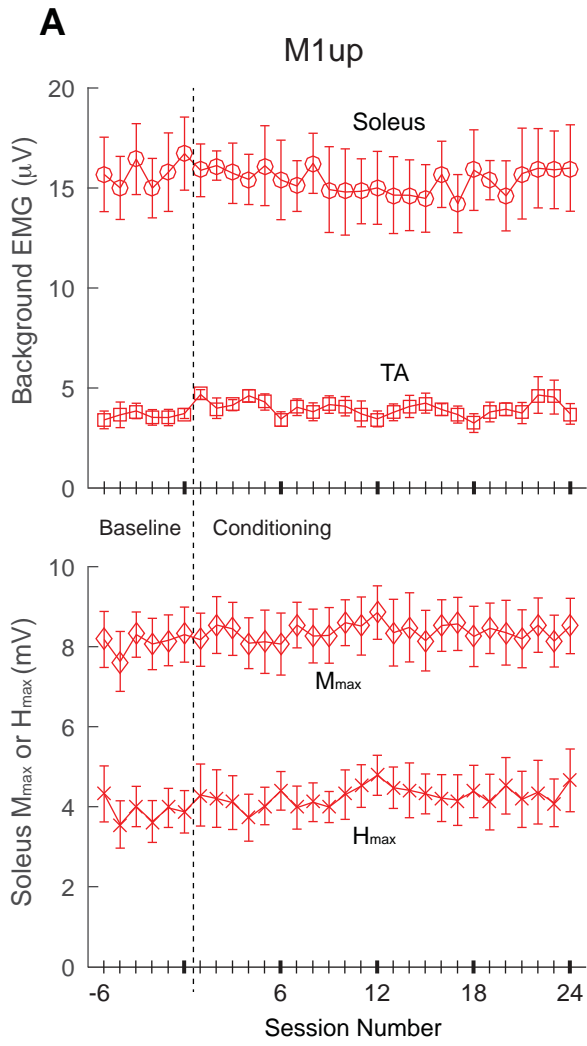
669 **Figure 4:** Average conditioned and control stretch reflexes for a representative participant of the M1up  
670 **(A)** and M1down **(B)** group. The upper panels show the change in ankle angle in degrees, the lower  
671 panels depict the average rectified SOL EMG data of the three 75-trial blocks for one baseline session  
672 and the last conditioning session in single participants. The dotted black traces represent data from a  
673 single baseline session while the red (M1up) and blue (M1down) traces represent data from the last  
674 conditioning session.

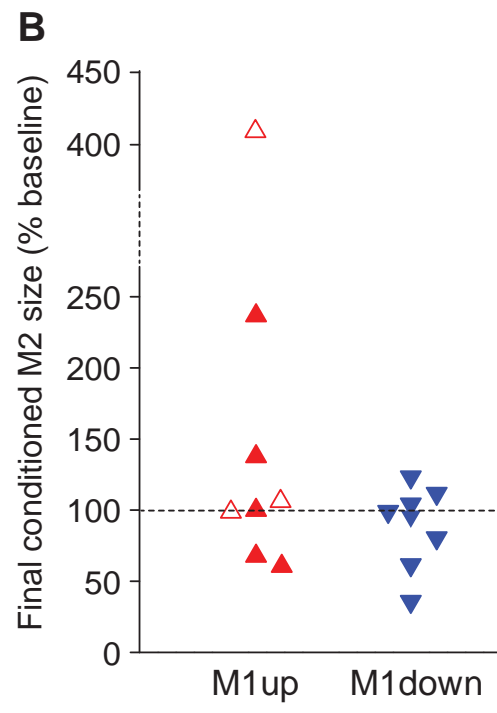
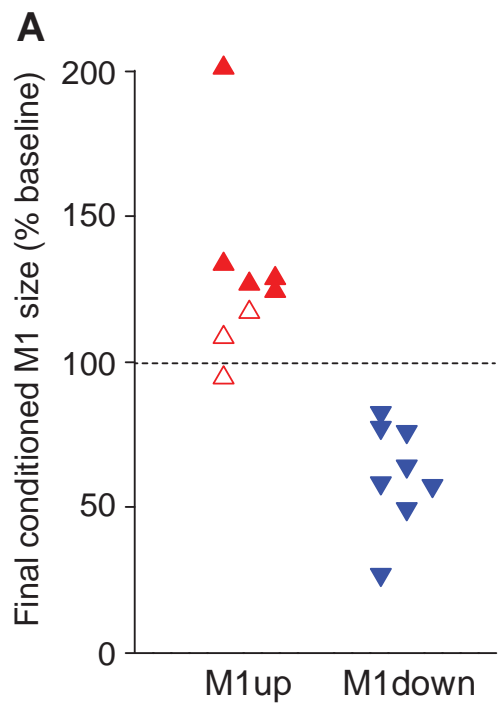
675 **Figure 5:** Average ( $\pm$ SE) M1 sizes and H-reflex sizes (from Thompson et al., 2009) for all successful  
676 M1up/HRup (red upward triangle) and M1down/HRdown (blue downward triangle) participants for all  
677 baseline and conditioning sessions. **(A)** upper panel: Average conditioned M1 size, middle panel:  
678 Average control M1 size and lower panel: within sessions change (average conditioned minus control  
679 M1 size). **(B)** upper panel: Average conditioned H-reflex size, middle panel: Average control H-reflex  
680 size and lower panel: within sessions change (average conditioned minus control H-reflex size). The  
681 vertical dotted line separates the baseline from the conditioning sessions.

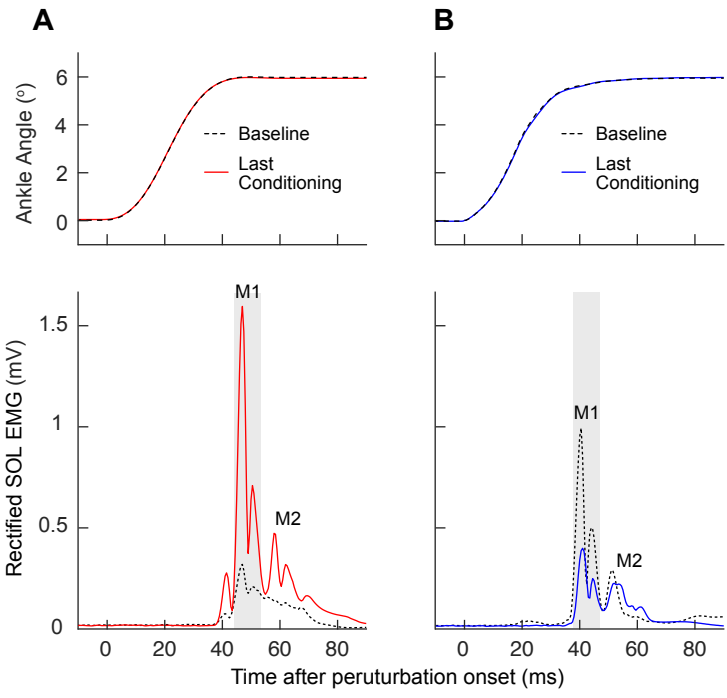
682 **Figure 6:** Control M1 change with M1down conditioning. Control SOL EMG during one baseline  
683 session (black trace) and the final conditioning session (blue trace) for n=1. Each trace is the average of  
684 20 trials.

685 **Figure 7:** Within-session task dependent adaptation with M1up conditioning. Control SOL EMG during  
686 the control trials (black trace, the first 20 trials where no visual feedback is provided) and the  
687 conditioning trials (red trace, the three blocks of 75 trials where feedback is provided in relation to the  
688 size of the M1 response) for n=1.









### A. M1 stretch reflex

### B. H-reflex

(from Thompson et al., 2009)

