


# Muscle cocontraction following dynamics learning

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**Abstract** Coactivation of antagonist muscles is readily observed early in motor learning, in interactions with unstable mechanical environments and in motor system pathologies. Here we present evidence that the nervous system uses coactivation control far more extensively and that patterns of cocontraction during movement are closely tied to the specific requirements of the task. We have examined the changes in cocontraction that follow dynamics learning in tasks that are thought to involve single mechanical degree-of-freedom, at least two antagonistically sculpted feedforward adjustments to motor commands. We find that, even following substantial training, cocontraction varies in a systematic way that depends on both movement direction and the strength of the external load. The proportion of total activity that is due to cocontraction nevertheless remains remarkably constant. Moreover, long after indices of motor learning and electromyographic measures have reached asymptotic levels, cocontraction still accounts for a significant proportion of total muscle activity in all phases of movement and in all load conditions. These results show that even following dynamics learning in predictable and stable environments, cocontraction forms a central part of the means by which the nervous system regulates movement.

**Keywords** Motor learning · EMG · Impedance control · Muscle cocontraction

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well understood nor is manner in which cocontraction varies with the specific requirements of the task.

It has been shown that subjects use cocontraction control to offset the effects of destabilizing forces both under static conditions (Darainy et al. 2004) and during reaching movements (Burdet et al. 2001; Franklin et al. 2003a). Cocontraction control has been documented in the early stages of motor learning. It was shown that the cocontraction of muscles declines as learning progresses, both when subjects learn stable dynamics (Franklin et al. 2003b; Thoroughman and Shadmehr 1999) and for unstable dynamical tasks (Milner and Cloutier 1993). Little is known about the characteristics of cocontraction following

motor learning in stable environments. Apart from a general decline in cocontraction following learning, the manner in which cocontraction during movement is tuned to specific requirements of the task is unknown. In the present study, we have assessed the role of muscle cocontraction following adaptation to stable environmental dynamics in two situations. We have first examined the degree to which cocontraction during movement is modulated with the strength of the external force field. The modulation of cocontraction with the level of external force would be consistent with the idea that cocontraction controls neuromuscular disorder. Experimental procedures were is part of the way in which the nervous system compensates for the effects of external dynamics. A second focus is whether cocontraction is differentially tuned to offset the effects of loads associated with movements in different directions. Different muscle pairs are involved in moving each subject, the height of the seat was adjusted to produce in different directions and accordingly different patterns of muscle coactivation may be necessary to optimally support these movements. Apart from demonstrations under static conditions (Gribble and Ostry 1998; Gomi and Osu 1998), it is unknown whether the nervous system is capable of modulating the balance of cocontraction over different movement directions.

In the present study we have assessed the role of muscle cocontraction after extensive practice. We have used a center-out reaching task with a clockwise velocity-dependent curl field to gauge this effect (Shadmehr and Mussa-Ivaldi 1994). Two primary directions of movement were chosen for this study. Reaching movements in one direction required mostly elbow rotation (elbow movement condition) while movements in the second direction involved primarily shoulder rotation (shoulder movement condition). In each direction, subjects trained with two levels of force field strength consecutively. We obtained the measure of muscle cocontraction using a technique described previously (Shadmehr 1999; Gribble et al. 2003). This is a measure of co-occurring activity that is shared by an antagonist muscle pair and provides an estimate of cocontraction. We observed that even following adaptation and after considerable training, the cocontraction level varied with the strength of the force field. We also observed that movement direction had a substantial influence on the pattern of cocontraction. Moreover, cocontraction associated with these effects accounted for a substantial and remarkably constant portion of total muscle activation. These results show that even during wholly stable interactions with the environment, cocontraction is modulated with the level of the force field and also with the direction of movement. These findings suggest that coactivation control is an integral component

The experimental session was divided into a familiarization phase and an experimental phase. The electrodes were placed just prior to the experimental phase. In the familiarization phase of experiment, subjects were trained to make 15 cm reaching movements in 600–650 ms to two visual targets that were positioned just below the surface of the glass table. No forces were applied during this part of the experiment (null condition). One visual target was placed lateral to the subject at the left of the start point. The reaching movement to this target involved shoulder flexion and to a lesser extent elbow extension (30° combined shoulder flexion) and will be referred to as the elbow movement condition. A second target was placed in a diagonal direction, forward and to the right of the start point. Movement to this target primarily involved elbow extension (30° combined shoulder flexion) and will be referred to as the shoulder movement condition. Subjects were asked to move as straight as possible and audio-visual feedback of movement duration was provided at the end of each trial. In familiarization phase, two blocks of 50 trials each were carried out to each of the targets. Subjects had full view of the arm at all times during the experiment. Each trial started with a short beep and subjects were asked to reach the target in a single movement. They were also told that their reaction time was not the focus of the study and accordingly was not included in the measured movement duration. Following movement, when

the hand stopped inside a 1 cm diameter circular target zone, visual feedback of their movement duration was displayed on a monitor beside the robot. They also heard an audio feedback at the same time. The robotic arm moved the subject's hand back to the start point and after a 500 ms delay the next trial began.

Six shoulder and elbow muscle sites were identified and prepared for electrode placement. The muscles of interest were two single-joint shoulder muscles (pectoralis clavicular head and posterior deltoid), two muscles acting at the elbow (the double-joint muscle biceps long head and single-joint triceps lateral head) and two bi-articular shoulder and elbow muscles (biceps short head and triceps long head). Kinematic error was used to assess learning and was defined on a trial-by-trial basis, as the maximum perpendicular distance (PD) between the hand trajectory and a straight line that connected movement start and end.

In the experimental phase of study, subjects were randomly divided into two groups of five subjects each. The first group was tested first with movements to the lateral target (shoulder movement condition) and then repeated the same procedure with movements to the diagonal target (elbow movement condition). The second group of subjects did the same task but in opposite order. Five blocks of reaching movements were carried out to each target.

Blocks 1 and 2 involved 50 trials each and were carried out under null conditions (only Block 2 was recorded). Block 3 involved 150 trials under low force conditions (see below). A fourth block (Block 4) also involved 150 trials under high force conditions. A final block of 50 trials was carried out under null conditions to test for after-effects.

EMG data and kinematics were recorded simultaneously. EMG magnitudes of antagonist muscles under static conditions. As a normalization factor we used the mean muscle activity from the high force condition during a 200 ms window from 500 to 300 ms before movement onset (averaged over the 300 trials in both movement directions). As a control, we repeated the normalization procedure using the mean EMG activity in a 200 ms window starting 300 ms after the end of movement. We also repeated the normalization procedure based on measures of maximum voluntary cocontraction that were recorded prior to the experimental measurements. In both cases, the results obtained were qualitatively similar to those reported above.

Clock-wise velocity-dependent forces were used during force-field trials. Equation 1 shows the forces that were applied to the subject's hand.

$$\begin{bmatrix} f_x \\ f_y \end{bmatrix} = \beta \begin{bmatrix} 0 & 1 \\ -1 & 0 \end{bmatrix} \begin{bmatrix} v_x \\ v_y \end{bmatrix} \tag{1}$$

In this equation  $f_x$  and  $f_y$  are the commanded force to the robot,  $v_x$  and  $v_y$  are hand velocities in Cartesian coordinates and  $\beta$  gives the field strength.  $\beta$  was set at 7 N s/m for the low force-field condition and 17 N s/m for the high force-field condition. In 20% of randomly chosen force-field trials, the robot motors were turned-off and subjects were tested under null conditions (catch trials).

### Data analysis

Position signals were sampled at 200 Hz, low-pass filtered at 20 Hz and numerically differentiated to produce the velocity signals. EMG signals were sampled at 1,000 Hz and digitally band-pass filtered between 20 and 450 Hz. EMG signals were then full-wave rectified and the root-mean-square of the signal was calculated over a sliding window of 100 ms. A measure of baseline EMG activity was removed from all signals on a muscle-by-muscle basis. The measure was obtained by recording a single 30-s trial at the beginning of the experimental phase, during which the subject rested at the center of workspace and did nothing. For each trial, the EMG signals and kinematics data were time-aligned at movement start. Movement onset was scored at the time that tangential hand velocity exceeded 20 mm/s. Movement end was also scored when the hand tangential velocity fell below the 20 mm/s. Kinematic error was used to assess learning and was defined on a trial-by-trial basis, as the maximum perpendicular distance (PD) between the hand trajectory and a straight line that connected movement start and end. We also assessed performance by computing a learning index (Hwang et al 2003), LI, a measure that takes into account both force-field trials and catch-trials and, hence, corrects for possible differences in performance due to differences in the action of the force field. The LI is defined as:

$$LI = \frac{|PD_{catch}|}{|PD_{catch}| + |PD_{ff}|} \tag{2}$$

where  $PD_{ff}$  is the maximum perpendicular distance from a straight line under force field conditions and  $PD_{catch}$  is the same measure for catch trials. The learning index ranges from 0.0 early in training to an upper limit of 1.0 under conditions of complete adaptation.

Following the removal of baseline activity, measures of EMG were normalized on a muscle-by-muscle basis for each subject separately. The normalization serves to equate EMG magnitudes of antagonist muscles under static conditions. As a normalization factor we used the mean muscle activity from the high force condition during a 200 ms window from 500 to 300 ms before movement onset (averaged over the 300 trials in both movement directions). As a control, we repeated the normalization procedure using the mean EMG activity in a 200 ms window starting 300 ms after the end of movement. We also repeated the normalization procedure based on measures of maximum voluntary cocontraction that were recorded prior to the experimental measurements. In both cases, the results obtained were qualitatively similar to those reported above.

We have not used the more usual technique of normalizing EMG relative to maximum voluntary contraction (MVC). In order to obtain a normalized measure of muscle cocontraction (as opposed to individual muscle contraction), we needed a reference that equated for EMG levels in antagonist muscles when the arm is cocontracted in statics. Conventional MVC measures are obtained for each muscle separately and MVC values for antagonist muscle pairs are unlikely to result in static equilibrium.

We obtained a measure of cocontraction as follows. On shoulder movement condition and elbow and biarticular each trial and for each antagonistic pair of muscles (for cocontraction in the elbow movement condition. example, biceps long head and triceps lateral head), the We tested six additional subjects in a control study that minimum normalized muscle activity (from the two EMG involved only the shoulder movement condition, and was signals) was calculated at each point in time (Thorouhman conducted without catch trials. Subjects first completed 450 and Shadmehf1999; Gribble et al.2003). This measure of trials under null conditions followed by 450 further trials in cocontraction was calculated over the course of movement in the high force condition. EMG and movement kinematics to yield a cocontraction trajectory for all trials in the null, were recorded as described above. We verified that per- low force and high force conditions. formance had reached asymptotic levels by dividing the

We also computed a measure of reciprocal activation for experiment into bins of 25 trials each. A repeated measures each trial and each pair of antagonist muscles. The measure ANOVA assessed differences in both cocontraction and of reciprocal activation was the total activity in each movement curvature over the course of the null and force- muscle pair minus the activation in each muscle due to held trials in the control study.

muscle cocontraction. The total activity in each muscle pair was thus the sum of reciprocal activation plus two times muscle cocontraction (reflecting the contribution of Results cocontraction to the measured activity of each individual muscle). The contribution following learning of muscle The aim of this study was to assess the role of cocon- cocontraction to total muscle activity was calculated as traction following adaptation to a novel dynamic follows (averaged over the final 25 trials in each experi- environment. In order to quantify learning we calculated mental condition, not including catch-trials and the the maximum perpendicular deviation (PD) of the hand immediately following trial):

$$C = \frac{2coc}{2coc + recip} \quad (3)$$

where  $C$  is the proportion of total muscle activity due to low force-held conditions, 150 trials under high force- cocontraction,  $coc$  is the contribution of each muscle to held conditions and 50 final after-effect trials). The cocontraction and  $recip$  is the total reciprocal activation as occurrence of catch-trials shown in red was random in the defined above. actual experiment however for visualization purposes they

For purposes of statistical analysis, each individual co are plotted at equal intervals. It can be seen that move- contraction trajectory was divided to four parts, 250 to 50 ments are straight during null held trials. When the force- ms before movement onset, 50 ms before movement onset held is unexpectedly introduced, the hand path deviates to maximum tangential hand velocity, maximum velocity from a straight line but with practice the deviation is to end of movement and a 200 ms interval immediately reduced. As in other studies of force-held learning even at following movement end. Repeated measures ANOVA and asymptote some curvature remains (Lackner and Dizio Bonferroni corrected post hoc tests were used to evaluate 1994; Caithness et al.2004). The hand path is deviated in statistical differences. the opposite direction during catch trials and the magni- tude of the after-effect increases as a function of load.

We also classified muscles according to their role in Following the completion of the low force condition movement and cocontraction measures were divided of subjects rest for 5 min and then start the high force on this basis. We refer to the cocontraction activity of antag- subjects rest for 5 min and then start the high force on- onistic muscles that are involved in the generation of condition in which the direction of force-held remains the movement as prime mover cocontraction, and the cocon- same but the magnitude is increased. As in the low force traction activity of antagonistic muscles at the largely condition, the hand initially deviates from a straight line. stationary joint as stabilizer cocontraction. Previous studies Curvature is reduced with practice, however, the effect of have demonstrated that activity in biarticular muscles is the force-held is not fully removed and some residual closely related to movement and torque at the elbow (Gomi, curvature is observed that is greater in magnitude than in and Osu1998; Gribble and Ostry1998). Therefore, we the low force condition. The final block of the experiment have grouped double joint muscles with elbow muscles for involves 50 trials under null held conditions. Here one purposes of this analysis. Thus, we grouped together as can observe a considerable after-effect that gradually stabilizers, the shoulder cocontraction values in the elbow returns to null held levels. Figure 1b shows the perpen- movement condition and the elbow and biarticular cocon- dicular deviation for the elbow movement task. The effect traction values in the shoulder movement condition. We is qualitatively similar to that observed in the shoulder grouped as prime movers, shoulder cocontraction in the movement condition.

**Fig. 1** Maximum perpendicular deviation (PD) during the four phases of the experiment. Mean values across subjects ( $\pm$ SE) are shown. **a** Shoulder movement condition. **b** Elbow movement condition. PD reaches asymptotic levels well before the end of training

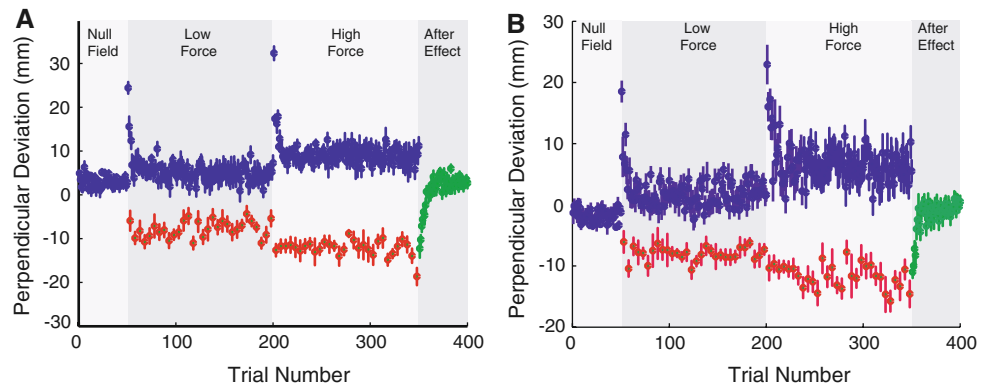


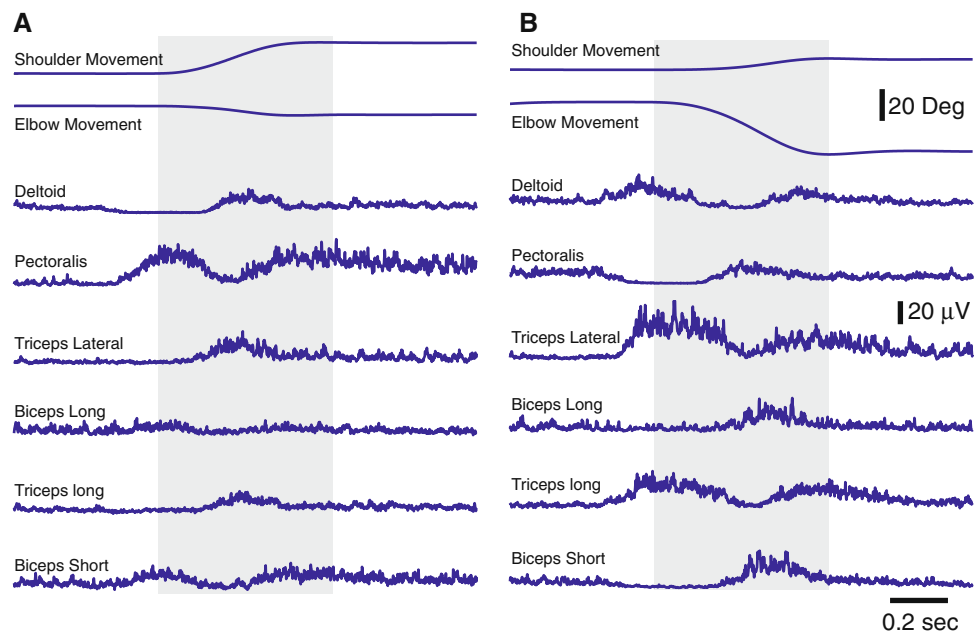
Figure 1 shows that performance reached asymptotic levels in all conditions. This was examined quantitatively by assessing changes in perpendicular deviation over the course of the trial. For this purpose, we divided the last 50 trials of the low and high force conditions into five bins of ten trials each (catch trials were excluded). A repeated measures ANOVA found no differences in mean curvature over this set of 50 trials, although, as noted above, curvature was reliably higher in the high force condition ( $P < 0.01$ ). This analysis thus shows that performance had reached asymptotic levels long before the end of training.

The larger perpendicular deviation that is observed in high force trials at the end of the learning phase is accompanied by larger perpendicular deviation in catch trials. Thus, while limb deflections due to hand velocity are greater, the amount of learning as assessed by the magnitude of the catch trials curvature is greater as well. To assess possible differences in the amount of motor learning with force-dependent strength, we calculated a learning index, LI, for both the low force and the high force conditions (Hwang et al. 2003). The computation was carried out on a per subject basis using PD measures for the last 10% of trials in each condition.

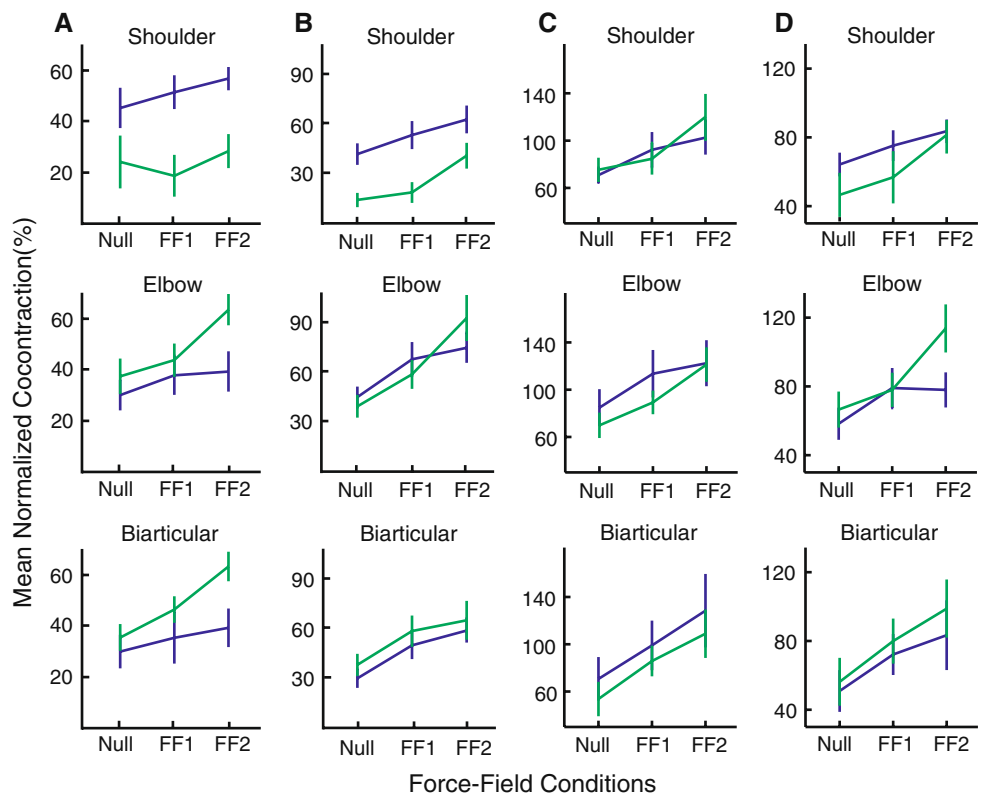
Differences in the LI were tested using a two-way repeated-measures ANOVA. We found that the LI was similar in low and high force conditions ( $P = 0.19$ ), averaging 0.65 and 0.61 for the shoulder movement condition and 0.71 and 0.66 for the elbow movement condition ( $P = 0.24$  for shoulder vs. elbow movement). This suggests that in each movement direction subjects learned approximately the same percentage of the force-dependent magnitude.

Figure 2 shows performance under null force conditions for a representative subject. Mean shoulder and elbow rotation are shown along with the EMG activity of six elbow and biarticular muscles in each of these four inter-shoulder and elbow muscles. The data are time aligned to movement start and the highlighted area shows the period of movement. There is a clear burst of phasic muscle activity in agonist muscles before the onset of movement.

**Fig. 2** Shoulder and elbow rotation and mean EMG activity of six shoulder and elbow muscles for a representative subject. **a** Shoulder movement condition. **b** Elbow movement condition



**Fig. 3** During wholly stable interactions with the environment, cocontraction is modulated with the level of the force-field and also with the direction of movement. Mean across subjects ( $\pm$ SE) of shoulder, elbow, and biarticular cocontraction for shoulder movement direction (green) and elbow movement direction (dark blue). Note that for visualization purposes different scales have been used. **a** From 250 to 50 ms before movement. **b** A measure of 50 ms before movement to maximum velocity. **c** Maximum velocity to movement end. **d** A measure of 200 ms following movement end



A four-way repeated measure ANOVA followed by generating limb displacement as prime movers. The analysis showed that cocontraction increased with magnitude of the force-field ( $p < 0.001$ ). Cocontraction magnitudes at each force level were found to be reliably different from one another by post hoc tests ( $p < 0.02$  in all cases). Cocontraction magnitudes also varied over the course of

the movement ( $P < 0.001$ ). Cocontraction was lowest in the intervals preceding peak velocity and increased reliably to a maximum in the deceleration phase of movement (shown in Fig. 3). The cocontraction level at the end of movement was not reliably different from that in the deceleration phase ( $P = 0.08$ ).

The relative magnitude of cocontraction in stabilization versus movement related muscles also varied during movement ( $P < 0.01$ ). In the period prior to movement (Fig. 3a) and in the interval from movement start to peak velocity (Fig. 3b), post hoc comparisons showed that cocontraction magnitudes were reliably greater for muscles involved in joint stabilization than for muscles involved primarily in generating the movement ( $P < 0.01$  and  $P < 0.05$ , respectively). In the two subsequent intervals, from peak velocity onward, cocontraction levels were similar for stabilization muscles and prime movers ( $P = 0.24$ ,  $0.07$  respectively).

Muscle cocontraction accounted for a substantial portion of total activity in each muscle pair even following learning. Figure 4a shows a representative example of muscle activity in the shoulder movement condition (null field). The records display an interval from 500 ms before movement start to 500 ms after movement end. The activity for each muscle pair is shown separately. Flexor muscles are shown in green, extensors are in blue and the common portion due to cocontraction is the light blue shaded region.

We quantified the proportion of total muscle activity due to cocontraction,  $C$ , for each of the four time intervals shown in Fig. 3. Figure 4b shows this ratio in each of the four movement phases and the three force-field conditions (averaged over muscle pairs). Overall it can be seen that cocontraction accounted for approximately half of the measured muscle activity, except in the acceleration phase of movement where the contribution is less. It can also be seen the proportion of activity due to cocontraction is relatively constant in the different force conditions tested here and in different movement directions. Thus while both phasic muscle activity and cocontraction increase with level of the force-field the overall proportion is for the most part unchanged. Differences in  $C$  were assessed using a three-way repeated measures ANOVA followed by Bonferroni corrected post hoc tests. We found a significant change in the proportion of muscle activation due to cocontraction over the four time intervals of the movement ( $P < 0.001$ ). Cocontraction during the acceleration phase of movement was reliably less than in the three other intervals ( $P < 0.001$  in each case). Overall, cocontraction was found to account for 53, 36, 57 and 56% of the total activity in these intervals, respectively. The lowest value, in the acceleration phase of the movement, is due to the large phasic muscle activation associated with initiation of limb movement. We found that the proportion of cocontraction did not differ with force level ( $P = 0.06$ ). However there were reliable

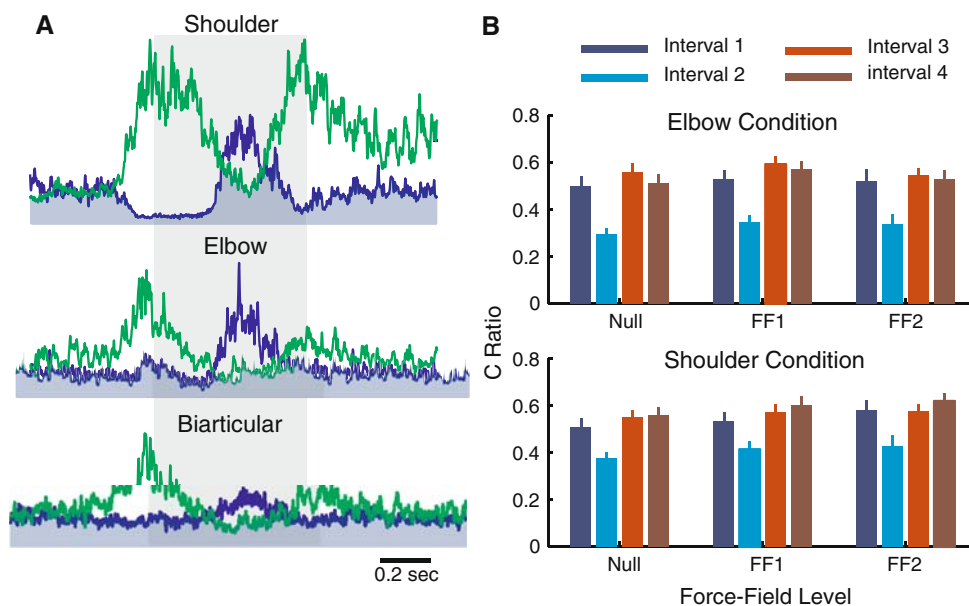


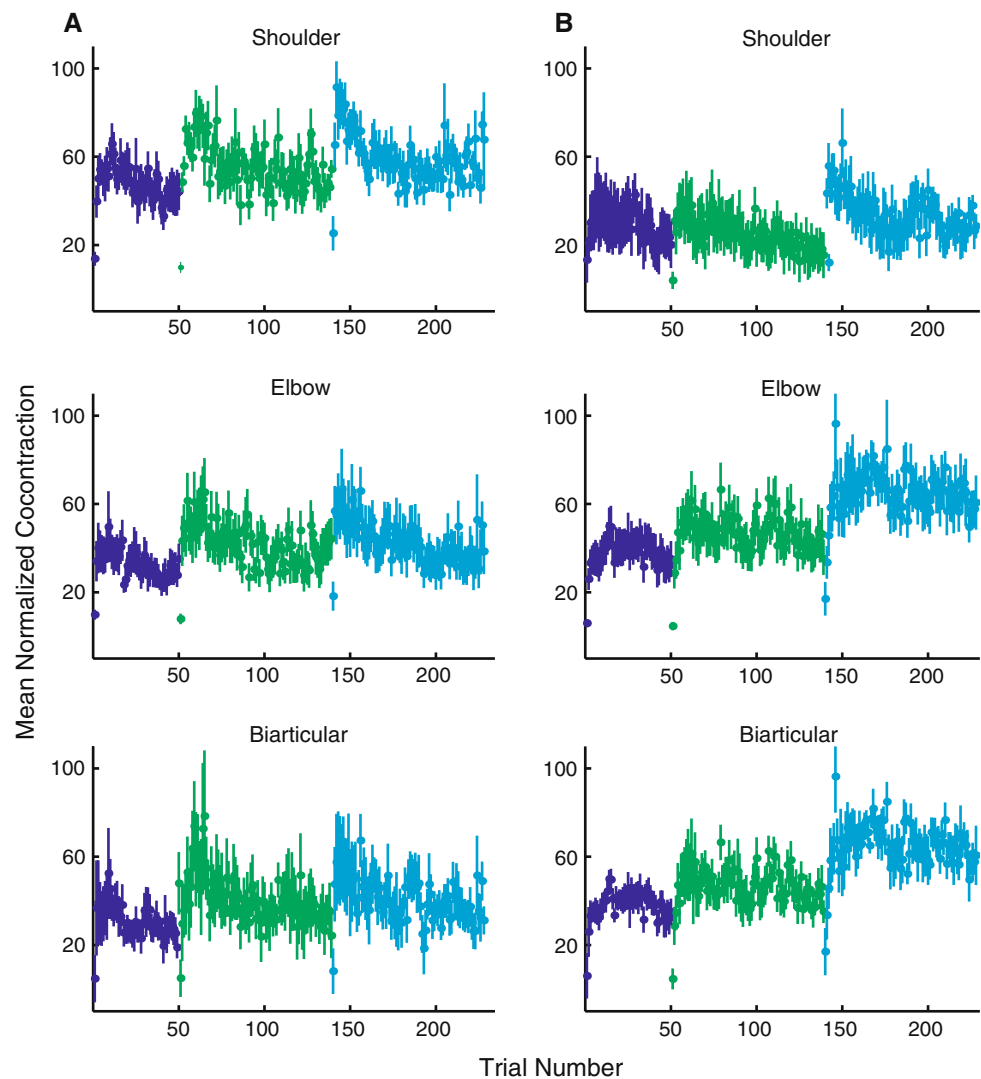
Fig. 4 Cocontraction accounts for a substantial proportion of total muscle activity. a Representative example of muscle activity in the shoulder movement condition for an individual subject (averaged over 20 null field trials). Agonist muscles are shown in green, antagonist muscles are in dark blue. The darker shaded area shows

the defined cocontraction activity for each muscle pair. The highlighted band indicates the period of movement. Mean across subjects  $\pm$  SE of the relative contribution of cocontraction to total muscle activity ( $C$  ratio), averaged over muscles. The figure shows the four movement intervals at each of the three force-field levels

differences in the proportion of cocontraction with movement direction ( $P < 0.01$ ). A greater proportion of cocontraction activity of the first 10% of trials (the very first trial was excluded) and the last 10% of trials (the last trial was excluded) was observed in the elbow movement condition but the differences were small (52.5 vs. 48.5%) ( $P < 0.05$ ) in each condition. However, there is still substantial cocontraction even in the plateau phase of learning. There were two other statistically reliable differences. Cocontraction during the acceleration phase of movement accounted for less of the total in the shoulder condition by assessing changes in the cocontraction level over the last 50 trials of force-field learning. As in our earlier assessment of movement curvature, we divided cocontraction measures over the last 50 force-field trials in each condition (53.7 vs. 59.3%) ( $P < 0.02$  in each case).

Figure 5 shows changes in cocontraction over the course of the high and low force conditions into five bins of ten trials each (catch trials and the following force-field trial during the interval from 250 to 50 ms before the onset of movement are excluded). Over the course of these trials we found no statistically reliable differences in cocontraction activity (show a similar pattern). Null trials are shown in dark blue, cocontraction in the low force condition is shown in green (with catch trials and the first trial following the training), and cocontraction in the high force condition is shown in light blue. A repeated measures ANOVA ( $P > 0.4$ ) showed that cocontraction had thus reached asymptotic levels long before the end of the novel task.

**Fig. 5** Cocontraction declines with learning but there is persistent cocontraction even at asymptotic performance levels. Mean across subjects ( $\pm$ SE) of shoulder, elbow, and biarticular muscle cocontraction from 250 to 50 ms before movement. Null is in *blue*, low force is in *green* and high force is in *light blue* (catch trials and the first force-field trial afterwards are removed) **a** Shoulder condition. **b** Elbow condition

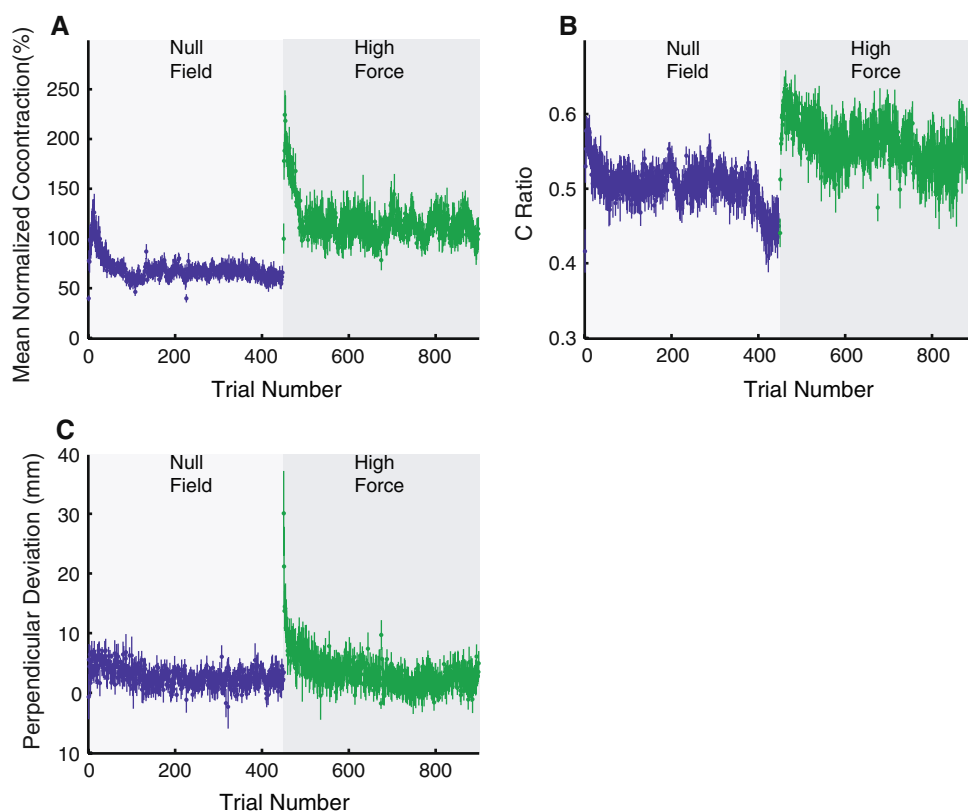




We conducted a control study with six new subjects to rule out the possibility that either insufficient learning or total muscle activity (in range of 50%). There were no catch-trials. The new subjects were tested over the course of 450 null movements and 450 high force movements, without any catch-trials. Figure 6a shows that the level of cocontraction is greater in the high force condition. Figure 6b shows that the proportion of total muscle activity due to cocontraction remains high regardless of fatigue in the present study we compared the mean IMDF force-field condition. Indeed, even in the absence of catch-trials, cocontraction accounts for about 50% of total muscle activity. Figure 6c shows that performance in the high force condition approaches null field levels within about 100 trials.

The order in which subjects performed the task requires that null field trials were followed by high force trials. Although using a two-way repeated measures ANOVA. We found that cocontraction was reliably greater under high force conditions, the possibility remains that muscle fatigue conditions ( $P < 0.001$ ). Using Bonferroni corrected pairwise comparisons, we found no differences in fatigue quantitatively. It is known that during isometric cocontraction over the first 100 training trials ( $P > 0.1$  in all cases). This shows that even after a longer period of training without catch-trials subjects consistently use cocontraction as part of the control that underlies movement.

**Fig. 6** Cocontraction remains high after extensive training in the absence of catch-trials. **a** Mean normalized cocontraction across subjects and muscle pairs ( $\pm$ SE) over the interval 250 ms before movement start to 200 ms after movement end. The level of cocontraction is greater in the high force condition. **b** Mean C ratio (proportion of total muscle activity due to cocontraction) averaged over subjects and muscle pairs ( $\pm$ SE). In both null and high force conditions, cocontraction accounts for about 50% of total muscle activity. **c** Mean perpendicular deviation ( $\pm$ SE) over the course of training. Performance in the high force condition approaches null field levels within about 100 trials



measures ANOVA (two directions of movement, three force levels) found no differences in the mean IMDF for the three force levels ( $P = 0.16$ ). Muscle fatigue can thus be ruled out as a potential source of the present results.

## Discussion

We have assessed characteristics of antagonistic muscle cocontraction over the course of dynamics learning. Subjects were given extensive practice in producing reaching movements to two visual targets. Each subject was tested in a null force condition followed by two force conditions, a low force and then a high force condition. Muscle coactivation, as assessed by the overlap of agonist and antagonist muscle activity was observed in all phases of movement throughout the course of training and accounted for a substantial proportion of total muscle activation even following learning. Importantly, cocontraction characteristic tuning has been observed, varied with the specific details of the task. In particular, cocontraction varied in magnitude with the strength of the force level and also varied with movement direction. In each direction, cocontraction changed over the course of movement such that before movement start and up to peak velocity, cocontraction in muscles involved primarily in joint stabilization was higher than in the prime movers. Following peak velocity cocontraction activity patterns in stabilizer muscles and prime movers was similar.

It has been suggested that a combination of feedforward and impedance control are involved in adaptation to both the stable and unstable dynamical environments (Franklin et al. 2003b). A number of studies have evaluated patterns of muscle activity in the early stages of learning (Franklin et al. 2003b; Thoroughman and Shadmehr 1999). These studies report an initial increase in activity for all muscles followed by a decrease in cocontraction as learning progresses. The data from the present study (Fig. 5) show a similar decline. The present study extends the previous findings by showing that the cocontraction which remains following motor learning is tuned to the requirements of the task such that it varies in magnitude in a systematic fashion both with force level and movement direction. Moreover, we observe that the relative contribution of cocontraction to total muscle activity remains essentially the same following learning at all force levels. This shows that cocontraction control is involved in all phases of learning and that the nervous system maintains a fairly constant balance of cocontraction and reciprocal activations similar to those observed for coactivation under static conditions. A second caveat is that cocontraction in the present study is estimated over time by computing the acceleration phase of the movement (from just before movement start to maximum velocity), we found that pairs of muscles were coactivated quite consistently accounted for 50–60% of total muscle activity. The reason that cocontraction percentages were not higher in the intervals just before and just after movement is the presence of phasic muscle activity both prior to movement onset and persistent activity following movement end (Suzuki et al. 2001). In the present dataset, the onset of phasic activity is clearly evident by 200 ms before the start of movement and is likewise present for several hundred milliseconds after the end of the movement (see Fig. 4). This accounts for the fact that the observed proportion of cocontraction in these intervals (250–50 ms before movement start and in the 200 ms following movement end) is comparable to that observed during movement.

The directional tuning of muscle cocontraction and corresponding directional changes to hand stiffness have been reported previously (Burdet et al. 2001; Darainy et al. 2004; Franklin et al. 2003b; Gomi and Osu 1998; Perreault et al. 2002). However, the studies in which this directional tuning has been observed, have involved either adaptation to an unstable dynamic environment or biofeedback of cocontraction that is presented to the subject. The patterns of shoulder and elbow cocontraction, and hence presumably, different underlying neural commands, are involved for different movement directions even when the interaction of the hand with the environment is stable. Cocontraction magnitudes were greater at the stationary joint. This difference presumably arises as a consequence of the biomechanics of the experimental design. The force level here produces torques that act primarily about the stationary joint. The observed difference in cocontraction between the stationary and moving joint may well reflect the need for greater stability under these conditions.

The way in which muscle cocontraction has been assessed in this study merits comment. The rationale for measuring overlap in EMG levels of antagonistic muscles as a measure of cocontraction is that the activity that is common to both muscles results in changes in impedance without producing accompanying changes in net joint torque. However the measurement of cocontraction in this manner also has certain limitations. First cocontraction as assessed in this way may in part reflect phasic muscle activation. This component is of course absent when cocontraction is assessed under stationary conditions. Nevertheless, even if the measured cocontraction in the present study derives in part from phasic activation, it acts to increase impedance and in this sense its effects are similar to those observed for coactivation under static conditions. A second caveat is that cocontraction in the present study is estimated over time by computing the minimum normalized EMG activity of antagonistic muscle pairs. For each muscle, EMG activity is normalized relative

to the observed EMG level under static conditions well before the movement onset. However, the normalization that serves to cancel out each opposite muscle torque under static conditions may require modification during movement. For example, changes in muscle moment arms along with length and velocity dependent differences in muscle force generating abilities (and the activity of other synergistic muscles) may influence torque and the actual level of muscle cocontraction. It is worth noting that measures taken in the interval preceding movement onset (Fig. 5a) are not contaminated by changes in muscle length and velocity. Similarly, measures from muscles that are primarily involved in joint stabilization are less affected.

We also wish to comment on a potential concern regarding the muscle activity level that we have used for EMG normalization. If EMG values in the interval used for normalization were small relative to those observed during movement, the effects of measurement error or noise could be greatly exaggerated. In the present study, EMG signals were normalized relative to the mean muscle activity in the high force condition during a 200 ms window before movement onset. The mean normalization factor (over subjects and muscles) was 1.7, whereas the mean baseline muscle activity was 1.7. Muscle activity in the interval used for normalization was thus almost 6.5 times the baseline activity level. By way of comparison, maximum EMG activity for the null held movements shown for a representative subject in Fig. 6 was around 40. Our normalization factor is thus large in relation to background signal levels and well within the range of signals that is applied to for normalization. It is thus unlikely that this procedure increases the effects of measurement error or noise.

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