Title: Neural gain induced by startling acoustic stimuli is additive to preparatory activation.

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Authors and Affiliations

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Abstract

Loud acoustic stimuli presented during movement preparation can shorten reaction time and increase response forcefulness. We examined how efferent connectivity of an agonist muscle to reticulospinal and corticospinal pathways, and the level of prepared movement force, affect reaction time and movement execution when the motor response is triggered by an intense acoustic stimulus. In Experiment 1, participants executed ballistic wrist flexion and extension movements of low and high force in response to visual stimuli. A loud acoustic stimulus (105dBa) was presented simultaneously with the visual imperative stimulus in probe trials. In Experiment 2, participants executed ballistic wrist flexion movements ranging from 10-50% of maximum voluntary contraction with a loud acoustic stimulus presented in probe trials. The shortening of response initiation was not affected by movement type (flexion or extension) or prepared movement force. Enhancement of response magnitude, however, was proportionally greater for low force movements and for the flexor muscle. Changes in peak force induced by the intense acoustic stimulus indicated that the neural activity introduced to motor program circuits by acoustic stimulation is additive to the voluntary neural activity that occurs due to movement preparation, rather than multiplicative.

Keywords: force; motor control; movement preparation; muscle; reaction time; StartReact effect

1.0 Introduction

Prepared motor responses can be triggered by the presentation of a loud acoustic stimulus (LAS). These triggered responses typically occur at shorter latencies and tend to be more forceful than would be produced voluntarily (Anzak et al., 2011; Honeycutt & Perreault, 2012; Kumru & Valls-Solé, 2006; Marinovic et al., 2015; Valls-Solé et al., 1999), potentially a product of increased neural activation being introduced to motor program circuits when a LAS is presented (Carlsen et al., 2012; Jaskowski et al., 1995; Marinovic et al., 2013; Marinovic et al., 2014a; Marinovic et al., 2014c; Marinovic et al., 2017; Tresilian & Plooy, 2006; Ulrich et al.,1998). It is unclear how characteristics of the prepared motor response, such as the forcefulness or muscle used to execute the response, may impact how much (or how quickly) LAS-evoked neural activity is injected into motor program circuits. One manner in which LAS-evoked activity may be modulated is via the strength of efferent reticulospinal connections that project to the agonist muscle. For example, as the reticular formation is implicated in both the startle reflex and voluntary motor control pathways, rather than the typical pathways used for voluntary motor control, early triggering of prepared responses may occur through a shorter pathway mediated by the reticular formation (Honeycutt et al., 2015; Rothwell et al., 2002; Yeomans & Frankland, 1995). Therefore, the amount of neural activity introduced to motor program circuits by a LAS and speed at which this activity is evoked may be expected to be greater when the agonist muscle has stronger efferent connectivity to the reticular formation. Differences in the shortening of RT via intense acoustic stimuli have been found between movements that engage muscles which have more reticulospinal projections, such as the biceps brachii, and muscles which have greater corticospinal connectivity, such as the first dorsal interosseous (Carlsen et al., 2009; Honeycutt et al., 2013). However, these differences in RT shortening between muscles may be a product of the functionality of movements that were tested, rather than muscle

connectivity (Marinovic & Tresilian, 2016; Castellote & Kofler, 2018). Other studies (Marinovic et al., 2014b) found no effect of muscle connectivity on RT shortening via intense acoustic stimuli, however, a comparison of movements which engage muscles that are functionally and anatomically similar remains to be tested.

While these aforementioned studies have investigated how preparation for movement of different muscles affects how quickly LAS-evoked activity is injected into motor program circuits, they have not examined how muscle connectivity affects the amount of activity that is accumulated after the presentation of a LAS, nor how the amount of force that is prepared for a movement may impact the level or speed of LAS-evoked activation. We hypothesised the level of activation evoked in motor program circuits by a LAS is affected by the amount of force prepared for a response in one of two ways. First, the amount of activation injected into motor program circuits by a LAS will increase as the amount of force prepared for a motor response increases, in which the enhancement of response magnitude scales with the required force of the prepared movement. This enhancement of response magnitude in a multiplicative manner would suggest that as prepared response force increases, a larger network of motor neurons are engaged which can subsequently be more easily recruited by the LAS. Alternatively, the amount of activation injected into motor program circuits by a LAS may be constant regardless of the amount of force prepared for a motor response, so that the enhancement of response magnitude remains constant with increasing prepared response force. Such enhancement of response magnitude in an additive manner would suggest activation introduced via functional connections between the auditory cortex and primary motor cortex (M1) (Marinovic et al., 2014c), or more directly from the brainstem to the spinal cord, converges with the activation associated with the prepared response. Furthermore, in flexors and extensors of the wrist – muscles which differ in their corticospinal and reticulospinal connectivity (Cheney & Fetz, 1980; Clough et al., 1968; de Noordhout et al.,

1999; Fetz & Cheney, 1980; Godfrey et al., 2013; Koganemaru et al., 2010; McMillan et al., 2004; Palmer & Ashby, 1992; Park & Li, 2013; Vallence et al., 2012), we investigated how muscle connectivity may impact the level or speed of LAS-evoked neural activation by examining how our proposed multiplicative or additive effects, and/or the shortening of RT, may be modulated by efferent connectivity to the reticular formation. As the reticular formation has been proposed to mediate the shortening of RT via intense sensory stimulation, we predicted a greater enhancement of response magnitude and shortening of RT would be observed in muscles which are more strongly connected to the reticular formation.

2.0 Method

2.1 Participants

Twenty-four participants (19 female) volunteered to participate in Experiment 1 (mean age = 22.5, *SD* = 5.41, range = 19 - 45). A second sample of 26 volunteers (19 female) was recruited for Experiment 2 (mean age = 22.85, *SD* = 4.48, range = 18 – 34). Participants in both experiments were self-reportedly right-handed, with normal or corrected-to-normal vision, and no apparent or known auditory impairments, neurological conditions, or injuries which could have affected their performance in the experiments. Informed, written consent was obtained from all subjects prior to participation, in accordance with the Declaration of Helsinki and approved by Curtin University's local human research ethics committee.

2.2 Procedures – Experiment 1

Participants were seated in front of a 22" Samsung SyncMaster 2233LCD monitor (120 Hz refresh rate, 1680x1050 resolution), at a distance of 0.8m. This monitor was used to present visual stimuli to participants during the experiment. Subjects were asked to respond to visual targets by making ballistic wrist movements in two directions, flexion and extension, with their right (dominant) hand and forearm secured in a custom-made wrist device housing a six

degree of freedom force/torque sensor (JR3 45E15A-I63-A 400N60S, Woodland, CA; see de Rugy et al. (2012); pictured in Figure 1). This device held the hand and forearm in a neutral position throughout the experiment, recorded forces produced by wrist movements, and controlled a cursor which was presented visually on screen. The device was tightened snugly to the hand and forearm of each participant to prevent any time delay between muscle activation and recording of force. The experimental setup is pictured in Figure 1. All participants completed the same procedures as follows.

Insert Figure 1 here

Due to the more frequent use and subsequent strength of flexor muscles in comparison to extensor muscles (Salonikidis et al., 2011), differences in muscle strength within individuals were controlled in order to allow comparison between these two muscles. Therefore, a maximum voluntary contraction procedure was completed for both flexion and extension movements prior to the start of the experiment (see Selvanayagam et al., 2016). In this procedure, subjects made six (three flexions, three extensions) isometric maximum voluntary contractions of the wrist toward a target for three seconds and the peak force (Newtons, N) was measured. The maximum voluntary contraction was calculated as the average peak force of the three contractions for each type of movement. Subsequently, this data was used to determine the degree of force required to reach low and high force targets during the experiment. These targets were represented as 20% or 60% of maximum voluntary contraction, depending on the force requirement of the relevant trial.

Participants were next trained in the movements that were required in the experiment, until they were able to accurately initiate movements within 250ms after the imperative

stimulus (IS). 224 experimental trials were completed, half of which constituted a block of flexion movements and the remaining half required extension movements. The order in which participants completed flexion or extension movements was counterbalanced. As shown in Figure 1, each trial began with the presentation of the word “relax”, indicating to the participant to keep their hand stationary for the start of the trial. A cursor which could be controlled by moving the hand then appeared in the centre of the screen. During each trial, a circular warning signal (WS) was presented, indicating the impending presentation of the IS. The colour of the WS and text presented at the top of the screen (“low” or “high”) indicated the force requirement of the trial (see Figure 1). Participants were instructed to prepare to move during this period. After two seconds (±200ms), the IS appeared and participants responded with a ballistic wrist movement, aiming to stop the movement once the target had been reached. The inter-stimulus interval (WS - IS) was randomly determined from a Gaussian distribution. The IS appeared as a yellow circle in place of the warning cue. The size of the target and distance to reach the target remained consistent regardless of trial type, whereas the position of the target (on the left or right side of the screen) depended on the movement direction of the trial – extension or flexion. 25% of trials were probe trials, in which a LAS was presented as an accessory stimulus pseudo-randomly with the IS, so that two consecutive trials could not occur as probe trials. Visual and auditory stimuli were presented using Cogent 2000 graphics running in Matlab 8.4. Participants were instructed to ignore the LAS when it was presented. At the end of each trial, feedback was presented on screen indicating RT, so as to maintain participant motivation and encourage quick responses. In probe trials, this feedback was not presented so as to prevent subjects becoming aware of the study’s aims and modifying their responses.

2.3 Loud Acoustic Stimulus

In probe trials, a LAS was presented in brief (50ms with a rise and fall time <1.5ms) white noise bursts. The peak amplitude of the stimulus was measured using a Bruel and Kjaer sound level meter (Type 2205, A weighted; Brüel & Kjaer Sound and Vibration Measurement, Naerum, Denmark). Limits of the neuromuscular system may often complicate interpretations of data in regard to the early release or vigour of responses elicited by acoustic stimuli. That is, RT flooring effects or ceiling effects in the production of force may limit meaningful observations, particularly when comparing muscles in this context. Subsequently, we employed a LAS at two intensities during Experiment 1; a high intensity stimulus at 105dBa, and a lower, albeit still startling stimulus at 90dBa. We chose to incorporate a lower intensity stimulus that would still be capable of eliciting a startle response in the case that the StartReact effect relies on mechanisms that rely on activation of startle circuits (see Carlsen et al., 2007). The LAS was generated by the soundcard of the computer used to run experiments, and presented binaurally through stereophonic active noise cancelling headphones (Bose QC25).

2.4 Procedures – Experiment 2

The same procedures from Experiment 1 were used in Experiment 2, with the following exceptions. Only flexion movements were employed in this experiment, and as such, no extension trials were contained in the maximum voluntary contraction, practice, or experimental trials. In addition, five force levels (10%, 20% 30%, 40%, and 50% of maximum voluntary contraction) were required in the experiment. All participants completed 230 trials, making up five blocks of 46 trials each. Each block was randomised to one of the five required force levels, with the required force of each block indicated by the colour of the warning stimulus, as well as text presented at the start of each trial and block. 20% of trials were probe trials in which a LAS was presented at one intensity, 105dBa.

2.5 Data reduction and analysis

Median RT, peak rate of force development, and peak force were subjected to statistical analyses. RT was measured as the time difference between the presentation of the IS and movement onset, expressed in milliseconds (ms). For each trial, the full time series of force data was collected from the force/torque sensor and sampled at 2000Hz using a National Instruments data acquisition device (NI USB-6229). Estimations of the time of movement onset were determined from the tangential speed time series using a two-stage algorithm suggested by Teasdale et al., (1993). The derivative of the torque signal over time was used to measure the peak rate of force development in Newtons per second (N/s). To measure the extent of facilitation that occurred due to the LAS, differences and ratios between probe and control trials were calculated. Differences in RT are reported, calculated as $RT_{LAS} - RT_{Control}$. To examine the extent of change in movement execution that was elicited by the LAS, ratios are reported for rate of force development and peak force. For peak force, this was calculated as ($Peak\ Force_{LAS}$ / $Peak\ Force_{Control}$). The same was calculated for peak rate of force development.

Cases in which subjects responded prematurely before the presentation of the IS (anticipatory response), or responded with a significant delay after the IS (indicating insufficient movement preparation) were identified prior to data analysis and subsequently removed (Whelan, 2008). These cases were identified as a RT <50ms or a RT >1000ms. In Experiment 1, this resulted in the removal of 143 trials in total (2.66% of all trials). 152 trials were removed from Experiment 2 (2.54% of all trials).

A series of linear mixed-effects models were conducted using the lmer function (lmerTest package; version 2.0-36; Kuznetsova et al., 2017) in RStudio (version 1.1.442; RStudio Team, 2015). These were conducted with trial type, muscle type, and required force

set as fixed factors and subjects set as a random factor, with Kenward-Roger approximation for degrees of freedom, in order to analyse the effects of trial type (control, low intensity LAS, high intensity LAS), muscle type (extension, flexion), and required force (low, high) on the facilitation of movement initiation and execution in Experiment 1. Along with these main effects, all interactions were tested in each linear mixed-effects model. In Experiment 2, analyses were conducted with required force (10%, 20%, 30%, 40%, and 50% of maximum voluntary contraction) and trial type (control, LAS) as fixed factors and subjects set as a random factor. Examination of residuals for each subject using Q-Q plots indicated no severe violations of the assumption of normality of residuals. Along with *F* values and *p* values of analyses, R^2 values, calculated using the r2beta function (r2glmm package; version 0.1.2; Jaeger et al., 2017) are reported to provide an estimate of effect sizes. Post-hoc tests were conducted using the emmeans function (version 1.3) with correction for multiple comparisons using the false-discovery rate procedure (Benjamini & Hochberg, 1995). To complement the traditional frequentist analyses and support inferences based on the null hypothesis, additional Bayesian linear models were conducted using the lmBF function from the BayesFactor package (version 0.9.12; Morey et al., 2018). The resulting Bayes factors (BF_{01}) are reported alongside *p* values of non-significant results of the main analyses.

As a further consideration for comparing the shortening of RT between muscles, it has been suggested that responses to a LAS fit two different mechanisms (Carlsen et al., 2007). The first may be the usual voluntary pathway for motor control, often without sternocleidomastoid (SCM) activity, and reduced shortening of response latencies – potentially representing a case of simple stimulus intensity and accessory stimulus effects (Marinovic & Tresilian, 2016). If a second mechanism exists, this would represent a distinct StartReact mechanism that produces the shortest response latencies, often – but not always - in the presence of SCM activity (Carlsen et al., 2009; Carlsen et al., 2007; Marinovic &

Tresilian, 2016). Therefore, if the StartReact effect does in fact follow a separate, distinct pathway from voluntary motor control pathways, muscles which have stronger connectivity to this pathway via the reticulospinal tract would be more likely to activate this pathway when a LAS is presented. To test this, a cumulative distribution function (CDF) was used to examine the effects of muscle type, as well as required force, at the 35th and 65th percentiles of RT for probe trials of both intensities. As reported by Leow et al. (2018), 35th and 65th percentiles were chosen for the CDF based on mean RT latencies of trials in the presence (35th percentile) or absence (65th percentile) of SCM activity reported by Honeycutt et al. (2013). While the presence or absence of SCM activity has previously been used in the literature in an attempt to separate responses which may or may not activate neurophysiological mechanisms responsible for the StartReact effect, this is not always reliable (Leow et al., 2018; Marinovic & Tresilian, 2016). For example, short latency responses indicative of the StartReact effect can be observed in the absence of SCM activity (Valls-Solé et al., 2005; Castellote et al., 2017). Furthermore, longer latency responses can occur in the presence of SCM activity (Marinovic & Tresilian, 2016). Therefore, examining trials based on RT percentile latencies, rather than the presence or absence of surface SCM activity, may be a more reliable method of capturing these two potential mechanisms in response to intense acoustic stimuli (Dean & Baker, 2017; Leow et al., 2018; Marinovic & Tresilian, 2016).

To estimate the pattern of neural activation that is accumulated due to the LAS with increasing force, two predicted patterns of the data were proposed. The first, representing a multiplicative effect of the LAS (Figure 2A) predicts an interaction of trial type and required force. Figure 2B depicts an additive effect of the LAS and predicts no such interaction. As such an effect relies on observation of the null hypothesis, the data may further be explored with the equations and model presented below.

Insert Figure 2 here

The peak force of responses executed in control trials may be described by the following equation, where A is equal to the slope. If responses are biased to present with more or less force than is required by the task, A < 1 or A >1. If there is no bias, then A may be observed as a random variable with a mean = 1.

$$\text{Equation 1: } (\text{Peak force})_{\text{Control}} = A \times (\text{Required force})$$

If the effect of the LAS on response force is multiplicative, and the multiplying factor is denoted as B, then B x A equates to the slope of LAS trials. When B > 1, this results in a slope steeper than control trials, as depicted in Figure 2A. The equation for LAS trials is therefore described as:

$$\text{Equation 2: } (\text{Peak force})_{\text{LAS}} = B \times A \times (\text{Required force})$$

These equations may then be used to determine how LAS-evoked neural activation is introduced to the system. For example, calculating ratios of LAS to control trials, as denoted by Equation 3, results in the multiplying factor, B. This is depicted in the flat line representing ratios in Figure 3A.

$$\text{Equation 3: } \frac{(\text{Peak force})_{\text{LAS}}}{(\text{Peak force})_{\text{Control}}} = B$$

Furthermore, differences in response force for the multiplicative predication may be represented by Equation 4. When B > 1, the gradient (A (B – 1)) is positive and corresponds with a slope that linearly increases with required response force, as depicted in the "differences" slope in Figure 3A.

Equation 4: $(\text{Peak force})_{LAS} - (\text{Peak force})_{Control} = BA(\text{Required force}) - A(\text{Required force})$

$= A(B - 1)(\text{Required force})$

Alternatively, if the effects of the LAS on response force are additive, this may be represented by Equation 5, where F is equal to a constant force added to responses by the LAS, as depicted by a LAS slope parallel to the control slope in Figure 2B.

Equation 5: $(\text{Peak force})_{LAS} = A \times (\text{Required force}) + F$

Given an additive model, ratios of LAS to control trials are calculated in Equation 6 to determine the neural activation that is introduced to the system by the LAS. This gives a non-linear curve with increasing required force as depicted by the slope for ratios in Figure 3B.

Equation 6: $$\frac{(\text{Peak force})_{LAS}}{(\text{Peak force})_{Control}} = \frac{(\text{Peak force})_{Control} + F}{(\text{Peak force})_{Control}}$$

If differences between LAS and control trials are calculated for the additive model, as in Equation 7, the constant F is derived, equal to the amount of force added to the response by the LAS. This is depicted by the flat line for differences in Figure 3B.

Equation 7: $(\text{Peak force})_{LAS} - (\text{Peak force})_{Control} = F + (\text{Peak force})_{Control} - (\text{Peak force})_{Control}$

$= F$

Insert Figure 3 here

As the presence of an additive LAS effect as depicted in Figure 2B cannot be determined by an absence of an interaction of trial type and required force – evidence for the null hypothesis, we compared data from Experiment 2 to our multiplicative and additive

models presented in Figure 3. In this analysis, both ratios of probe to control trials (Peak $Force_{LAS}$ / Peak $Force_{Control}$) and differences between probe and control trials (Peak $Force_{LAS}$ - Peak $Force_{Control}$) for peak force data in Experiment 2 were calculated to determine the fit of the data to each model, using polynomial contrasts with the contr.poly function (R Core Team, 2016). The multiplicative hypothesis would be supported by a linear increase in peak force differences and no change in peak force ratios as force increases (see Figure 3A). Alternatively, an additive hypothesis would be supported by no change in peak force differences and a reciprocal decrease in peak force ratios as required force increases (see Figure 3B).

3.0 Results

3.1 Experiment 1

3.1.1 Maximum Voluntary Contraction

Our maximum voluntary contraction procedure indicated a statistically significant difference between flexors and extensors in their force production capabilities, $t_{(23)} = 6.12$, $p < .001$, $d = 1.25$, with flexors showing greater maximum voluntary contraction force ($M = 107.15$ N, $SD = 38.31$) compared to extensors ($M = 83.23$ N, $SD = 29.36$).

3.1.2 Facilitation of Response Initiation

A statistically significant main effect of trial type indicated RT was shortened by the presentation of the LAS, $F_{(2, 253)} = 203.77$, $p < .001$, $R^2 = .617$. Control trial RT ($M = 194.59$ ms, SD = 20.17) was significantly reduced by both the high intensity ($M = 147.93$ ms, $SD = 20.12$ and low intensity LAS ($M = 150.99$ ms, $SD = 22.25$), with the difference between both probes and control trials resulting in $p < .001$ in post hoc tests. Analysis of the difference between control trials and LAS trials at both intensities indicated no significant main effects or interactions for RT (see Figure 4A). Subsequent Bayesian analysis of the non-significant

effect of muscle type, $F_{(1, 161)} = .98$, $p = .323$, $R^2 = .006$, yielded a Bayes Factor (BF_{01}) of 4.53. This is substantial evidence (Jeffreys, 1961) for the null hypothesis of differences between these muscles in their shortening of RT via a LAS.

Our CDF analysis (Figure 4B) for all probe trials averaged across both intensities indicated mean RTs at the 35th percentile = 137.07 ms, SD = 15.62. At the 65th percentile mean RT = 162.64 ms, SD = 24.63. A recent meta-analysis comparing RTs based on SCM activity reports a mean difference in RT latency between SCM+ and SCM- trials of -16.9 ms (Leow et al., 2018). Our results indicate a mean difference of -25.57 ms between fast and slow latency responses. Therefore, our analysis based on CDF latencies appears to converge with recent studies adopting the same procedure. No interactions or main effects beyond LAS intensity were statistically significant in analyses of our CDF. Importantly, the main effect of muscle type for the fast percentile did not reach statistical significance, $F_{(1, 69)} = .45$, $p = .506$, $R^2 = .006$. Again, subsequent Bayesian analysis showed substantial support (Jeffreys, 1961) for this null hypothesis, $BF_{01} = 4.42$.

Insert Figure 4 here

3.1.3 Enhancement of Response Execution

Peak rate of force development showed a main effect of trial type, $F_{(2, 253)} = 18.54$, $p < .001$, $R^2 = .128$, indicating the presentation of the LAS increased response vigour. Rate of force development showed the greatest increase in the high intensity LAS condition ($M = 622.93$ N/s, $SD = 226.08$). The low intensity LAS condition ($M = 606.32$ N/s, $SD = 218.58$) showed some enhancement of rate of force development, with control trials showing the lowest rate

of force development (M = 501.66 N/s, SD = 223.35) (see Figure 5A). Post hoc tests indicated the difference in peak rate of force development between control trials and probe trials of both intensities was statistically significant, $p < .001$. Similarly, peak force increased from control trials (M = 37.98 N, SD = 20.78) in both the high intensity (M = 42.75 N, SD = 19.47), and low intensity LAS conditions (M = 42.64 N, SD = 19.92), shown by a main effect of trial type on peak force, $F_{(2, 253)} = 3.61$, $p = .028$, $R^2 = .028$ (see Figure 5B). Post hoc analysis of the difference in peak force between control trials and high intensity probe trials ($p = .033$) and low intensity probe trials ($p = .033$) was statistically significant. The interaction of trial type and required force for peak force was not statistically significant, $F_{(2, 253)} = .17$, $p = .841$, $R^2 = .001$.

Due to the inherent difference between flexors and extensors in their force production capabilities, with flexors showing greater response magnitude regardless of trial type, the raw values of peak rate of force development and peak force were adjusted as a ratio of probe to control trials to examine the proportional facilitatory effects of a LAS on movement execution. Flexors showed larger ratios of peak rate of force development (M = 1.34, SD = .31) compared to extensors (M = 1.22, SD = .28) with a statistically significant main effect of muscle type on ratios of peak rate of force development, $F_{(1, 161)} = 14.89$, $p < .001$, $R^2 = .085$. Furthermore, a main effect of required force was found to be significant, $F_{(1, 161)} = 7.90$, $p = .005$, $R^2 = .047$, with larger ratios for low force movements (M = 1.32, SD = .34) compared to high force movements (M = 1.24, SD = .26). Ratios for low force movements were larger regardless of the intensity of the acoustic stimulus, with a non-significant main effect of LAS intensity, $F_{(1, 161)} = 1.15$, $p = .285$, $R^2 = .007$. Additionally, the interaction of required force and muscle type was significant, $F_{(1, 161)} = 3.94$, $p = .049$, $R^2 = .024$. The difference between low and high force ratios was statistically significant for flexors, ($p = .002$), but not for extensors ($p = .56$) (see figure 5C). Similar effects were noted for the ratios of peak force.

Consistent with the ratios of rate of force development, larger ratios for low force movements (M = 1.22, SD = .26) compared to high force movements (M = 1.14, SD = .20) were observed, with a significant main effect of required force on peak force ratios, $F_{(1, 161)} = 7.93$, $p = .005$, $R^2 = .047$. Again, low force movements showed larger ratios regardless of LAS intensity, with a non-significant main effect of trial type for ratios of peak force, $F_{(1, 161)} = .03$, $p = .868$, $R^2 = .000$. In addition, the interaction of muscle type and required force was statistically significant for peak force ratios, $F_{(1, 161)} = 4.40$, $p = .037$, $R^2 = .027$, with a statistically significant difference between low and high force ratios for flexors, $p = .004$, but not extensors, $p = .735$ (see Figure 5D).

Insert Figure 5 here

3.2 Experiment 2

3.2.1 Facilitation of Response Initiation

RT was facilitated by the LAS, with probe trials showing significantly shorter RTs (M = 147.35 ms, SD = 26.18) compared to control trials (M = 194.42 ms, SD = 21.52), as shown by a statistically significant main effect of trial type on RT, $F_{(1, 225)} = 473.16$, $p < .001$, $R^2 = .678$. Analysis of differences between probe and control trials for RT showed no effect of force, $F_{(4, 100)} = 1.56$, $p = .190$, $R^2 = .059$ (see Figure 6).

Insert Figure 6 here

3.2.2 LAS Induced Activation

Peak force was enhanced at the presentation of the LAS, with a main effect of trial type, $F_{(1, 225)} = 25.10$, $p < .001$, $R^2 = .1$ (see Figure 7). Similarly to the enhancement of response magnitude by the LAS in Experiment 1, the interaction of trial type and required force was not statistically significant, $F_{(4, 225)} = .168$, $p = .955$, $R^2 = .003$.

Insert Figure 7 here

As our analysis failed to indicate a significant interaction of trial type and required force, the data provided direct evidence against our proposed multiplicative LAS effect (Figure 2A), but not against the additive LAS effect (Figure 2B). Therefore, we examined the fit of the data to our additive model (Figure 3B) by calculating ratios ($\text{Peak Force}_{\text{LAS}}$ / $\text{Peak Force}_{\text{Control}}$) and differences ($\text{Peak Force}_{\text{LAS}}$ - $\text{Peak Force}_{\text{Control}}$) of peak force over trial types. Ratios of peak force (Figure 8A) showed a significant main effect of required force, $F_{(4, 100)} = 6.35$, $p < .001$, $R^2 = .203$. This effect of force was not significant for peak force differences (Figure 8B), $F_{(4, 100)} = .846$, $p = .499$, $R^2 = .033$. Polynomial contrasts showed a linear effect of force for peak force ratios, $t_{(100)} = -4.73$, $p < .001$. There was no such linear effect for peak force differences, $t_{(100)} = -1.06$, $p = .289$, indicating a decreasing trend for peak force ratios and a negligible change in the calculated differences of peak force as the required force in the task was increased. These results support our additive model presented in Figure 3B.

Insert Figure 8 here

4.0 Discussion

4.1 Reductions of response latency do not differ between muscles

We observed large (≈50 ms) reductions in RT in both low intensity and high intensity LAS conditions in comparison to control trials. In this shortening of RT, no difference between flexion and extension movements emerged. It has been argued that there are two potential mechanisms that underlie responses to a startling stimulus. We conducted a CDF analysis (see Leow et al., 2018) in an effort to capture these two potential mechanisms and determine whether longer latency responses masked our ability to observe differences between muscles in their shortening of RT. Our analysis indicated no difference in response latency between muscle types in the fastest percentile of RT analysed (35%). Moreover, we obtained substantial support for the absence of differences in RT between muscle types from our Bayesian analysis, suggesting connectivity of agonist muscles of the wrist to reticulospinal pathways is not a critical factor in the magnitude of RT shortening via intense acoustic stimulation. Although we tried to avoid a floor effect by using a stimulus with lower intensity than in typical StartReact studies, it is possible that even at 90dBa there was little room for RTs to reduce much further. Future studies may want to consider employing even lower intensities (e.g., 80dBa). However, the magnitude of the shortening of RT observed in our data is arguably less than that observed by others. We have observed a mean difference in RT between probe and control trials of 46.66ms, as compared to a mean difference of 60.1ms reported in Leow et al.'s (2018) mini meta-analysis. In fact, some studies within this meta-analysis (e.g. Tresch et al., 2014) report a mean difference of approximately 90ms. The larger shortening of RT in previous studies may be related to their higher intensity of the acoustic stimulus (128dB in Tresch et al., 2014).

4.2 Muscle connectivity affects enhancement of response magnitude

To examine the facilitatory effects of a LAS on response magnitude, in Experiment 1 ratios of probe to control trials were analysed for peak rate of force development and peak force. Ratios for low force movements were larger than high force movements for peak rate of force development and peak force. This difference was observed for both low and high intensity probe conditions, indicating that this effect was not likely due to high force movements being limited in their facilitation by ceiling effects. Flexors showed a greater rate of force development and peak force enhancement compared to extensors. Interestingly, our maximum voluntary contraction procedure indicated lower force production capabilities for extensors in comparison to flexors. As a function of required force being a percentage of maximum voluntary contraction for each muscle, our task required the execution of weaker extension responses compared to flexion responses. Given low force responses showed greater enhancement of response magnitude in response to the LAS, it may be expected extensors would show a greater increase of response force and vigour. On the contrary, our data indicated a greater enhancement of response magnitude for the stronger flexor muscles. Given extensors have been shown to have greater corticoneuronal connections (Cheney & Fetz, 1980; Clough et al., 1968; de Noordhout et al., 1999; Fetz & Cheney, 1980; Palmer & Ashby, 1992), flexors being more amenable to the effects of the LAS may suggest increased facilitation of reticulospinal pathways by the LAS; thereby indicating a subcortical mechanism for movement execution that is activated by intense acoustic stimuli and bypasses cortical circuits. However, if this were the case, a similar effect would be expected for our RT data, with shorter RT for muscles that have greater connectivity to reticulospinal pathways. Alternatively, in contrast to earlier reports of greater corticoneuronal contributions to extensors, recent neurophysiological reports have indicated greater functional corticospinal

excitability for flexors compared to extensors (Godfrey et al., 2013; Koganemaru et al., 2010; McMillan et al., 2004; Park & Li, 2013; Vallence et al., 2012). The greater observed enhancement of response magnitude via the LAS for flexors may thereby be a product of response force being correlated with M1 activity (Ashe, 1997). Subsequently, muscles with stronger corticospinal connectivity may be more sensitive to changes in response force induced by an intense acoustic stimulus. This is in support of the differences we have observed between muscles in the facilitation of response magnitude but not latency in this task. This further emphasises the utility of considering dynamics of movement such as forcefulness and vigour, rather than just response latency alone. Consistent with this assertion, Vergilino-Perez et al. (2012) have reported a similar pattern of effects in gain and amplitude of saccades (see also Reuter et al., 2019), but not latency. Our maximum voluntary contraction procedure did indicate mechanical differences between the muscles we tested. It is therefore not possible to assert that the altered enhancement of response magnitude between muscles was solely due to innervation. However, our maximum voluntary contraction procedure likely limited the influence of mechanical differences between the muscles by setting the required force for each muscle at an equal proportion relative to the amount of force they were capable of exerting.

4.3 Multiplicative and additive models of neural activation accumulation

Examination of the enhancement of response magnitude induced by acoustic stimuli allows estimations of how neural activation may be introduced to motor program circuits. As in Figure 2A, an interaction of trial type and required force would be expected for peak force if a multiplying effect underlies LAS-induced neural activation. Given no such interaction of trial type and required force was observed, the data in Experiment 1 appeared to suggest an additive effect of the LAS. This was supported by the finding of higher ratios of peak force

for low force movements compared to high force movements, for flexion responses. This initial suggestion of an additive effect of the LAS appeared to be impacted by muscle connectivity, with larger change in response magnitude observed for flexors.

In Experiment 2, we aimed to further examine the nature of the neural gain introduced by the LAS with increasing force which we observed in Experiment 1. Peak force was analysed to examine the additional neural activation that is introduced to motor program circuits by the LAS. Again, no interaction of trial type and required force was observed, failing to support the multiplicative model of neural activation. To examine the pattern of this activation, we analysed both ratios of probe to control trials and differences between probe and control trials for peak force. The data (Figure 8) fit the additive hypothesis (Figure 3B). As required force increased, peak force differences remained constant, whereas ratios decreased. This suggests recruitment of motor neurons by the LAS remains constant as prepared force increases, and LAS-induced activation is additive to the signal prepared in motor program circuits. As LAS-evoked activity appears to be added at a constant rate and does not interact with the level of activation of the prepared response, this would suggest neural activity associated with temporal preparation converges with activation introduced to motor program circuits, potentially via functional connections between the auditory cortex and M1, or more directly from the brainstem to the spinal cord (Marinovic et al., 2013; Marinovic & Tresilian, 2016; Marinovic et al., 2014c). While it is important to note that this work represents examination of a theoretical model's fit to behavioural data, a body of work has suggested a linear increase of force output with neuronal firing (see Cheney & Fetz, 1980; Evarts 1968; Russo et al. 2018). It has previously been proposed that LAS induced activation is additive to the voluntary cortical activation that occurs during movement preparation, and that these two processes jointly contribute to the initiation command of the response (Marinovic et al., 2017; Maslovat et al., 2014). Here, similar to the compounded

activation that occurs for response initiation, we have shown that activation introduced by the LAS may add to voluntary activation in motor program circuits, and together these can contribute to the amplitude of the accumulated signal that determines the magnitude of response execution. Furthermore, the current study provides a novel report of the additive nature of the activation that is injected into motor program circuits by the LAS.

5.0 Conclusions

In summary, our data indicate that the shortening of RT via intense acoustic stimuli in wrist muscles is not impacted by connections of the muscle to corticospinal or reticulospinal tracts. However, flexor muscles showed a greater enhancement of movement execution by the acoustic stimulus, possibly due to their stronger functional corticospinal connectivity in comparison to extensors. Over increasing force requirements, the benefit of the LAS on peak force did not change, and was therefore proportionally greater for low force movements. These changes in peak force induced by the acoustic stimulus fit our additive activation model and indicate startle-related activation does not interact with the voluntary activation in motor program circuits, but rather adds to this voluntary drive at a constant rate. The results we report here support M1 involvement in the early triggering of motor actions via intense acoustic stimuli (see Marinovic & Tresilian, 2016), and indicate the forcefulness and vigour of responses elicited by intense sensory stimuli may be enhanced differentially depending on the type of movement prepared. These findings may have implications for potential therapeutic applications of the StartReact effect. For example, prepared motor actions which we have shown to receive more benefit from the acoustic stimulus may be the more promising targets for rehabilitative protocols in conjunction with intense sensory stimuli in order to retrain motor control in neurological conditions in which movement execution is impaired (Honeycutt et al., 2015; Marinovic et al., 2016).

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Figure Captions

Figure 1. (A) Diagram of experiment set up. Displays participant seated in front of a monitor with arm secured in the wrist device. Adapted from de Rugy et al. (2012). (B) Sequence of events requiring a low force flexion response.

Figure 2. (A) Multiplicative effect of LAS, where response force induced by the LAS scales with the amount of force prepared for the response. (B) Additive effect of LAS, where response force induced by the LAS is added at a constant rate to the prepared motor response.

Figure 3. (A) Multiplying factor hypothesis of LAS induced neural activation. Differences in peak force between probe and control trials increase, while ratios remain constant. (B) Adding factor hypothesis of LAS induced neural activation. Ratios of probe to control trials decrease, while differences remain constant.

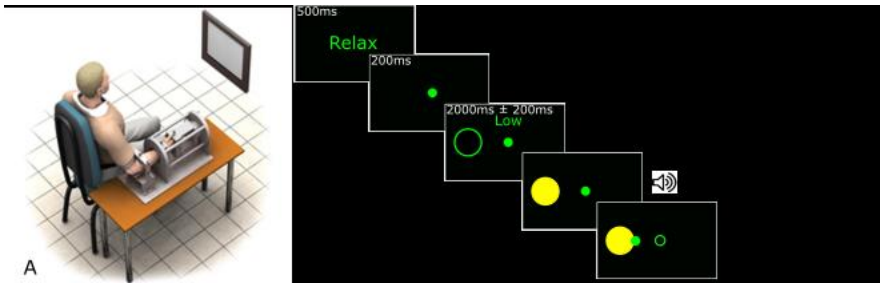
Figure 4. (A) Difference in RT between both probe conditions and control trials. (B) RT for fast (35th percentile) and slow (65th percentile) responses over different muscle types and force requirements.

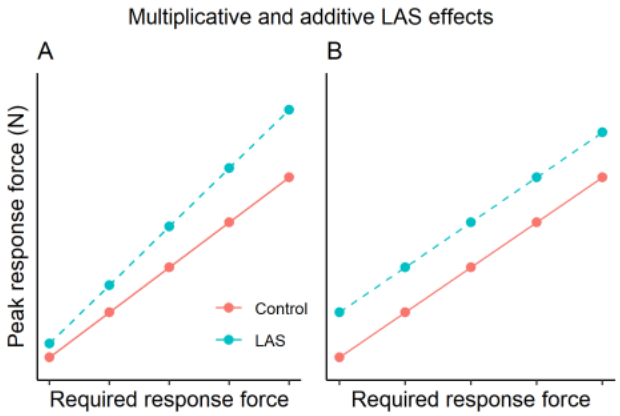
Figure 5. (A) Peak rate of force development over trial types, muscle types, and force levels. (B) Peak force over trials types, muscle types, and force levels. (C) Ratios of probe to control trials for rate of force development. (D) Ratios of probe to control trials for peak force. *** = $p < .001$, ** = $p < .01$, * = $p < .05$.

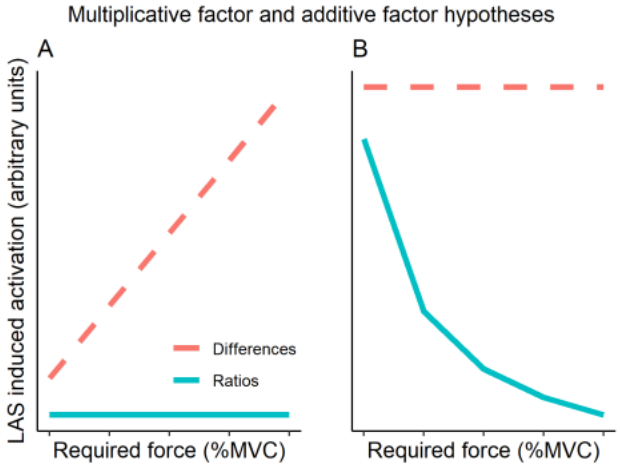
Figure 6. RT differences between probe and control trials over increasing force. Required force is represented as percentage of maximum voluntary contraction.

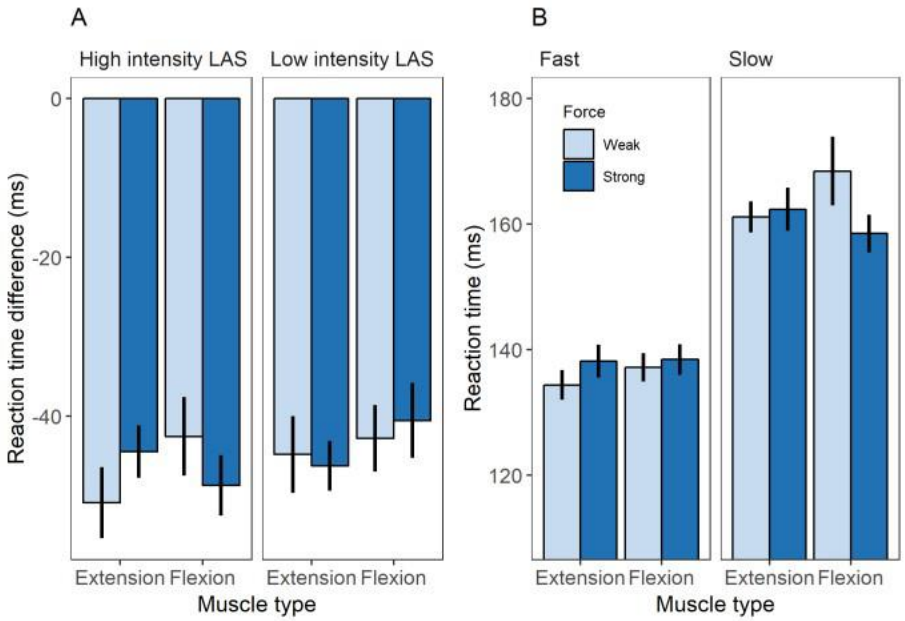
Figure 7. Peak force of responses over probe and control trials with increasing required force (percentage of maximum voluntary contraction).

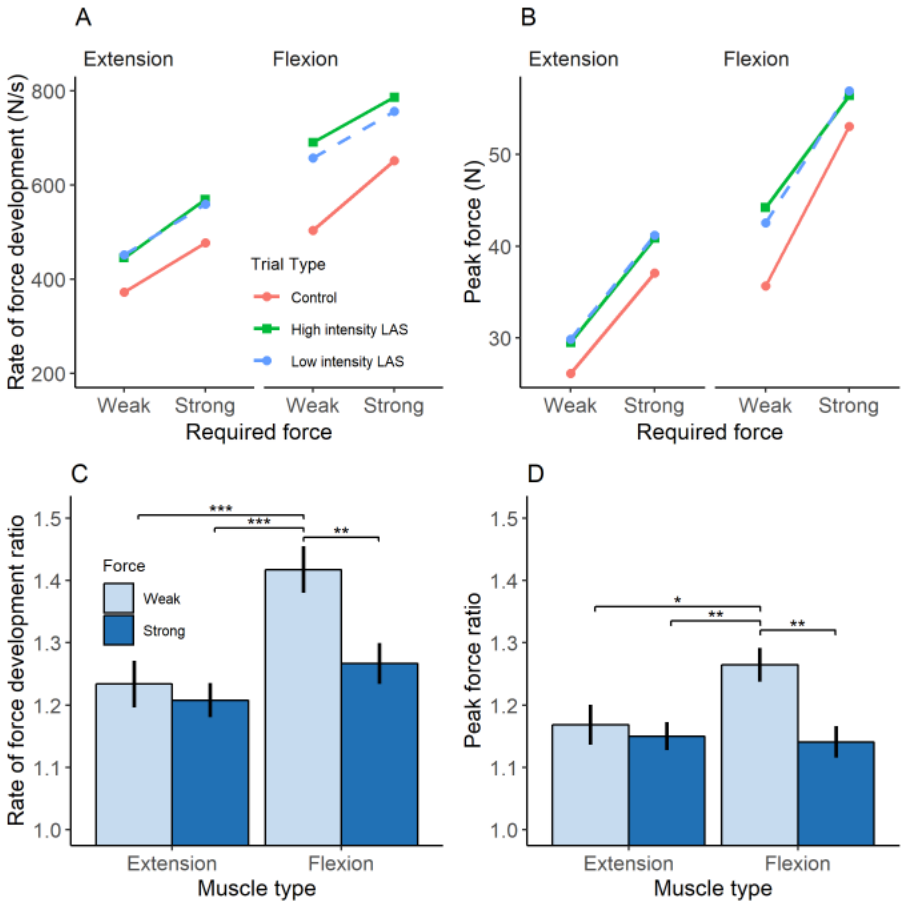
Figure 8. (A) Ratios of probe to control trials for peak force. (B) Differences between probe and control trials for peak force.

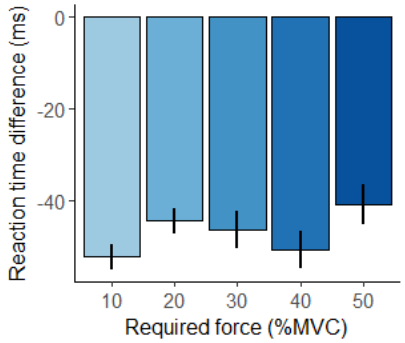


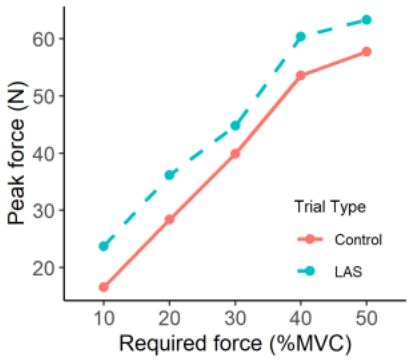


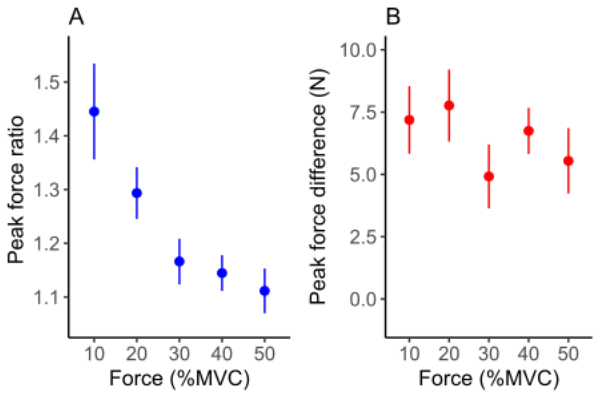




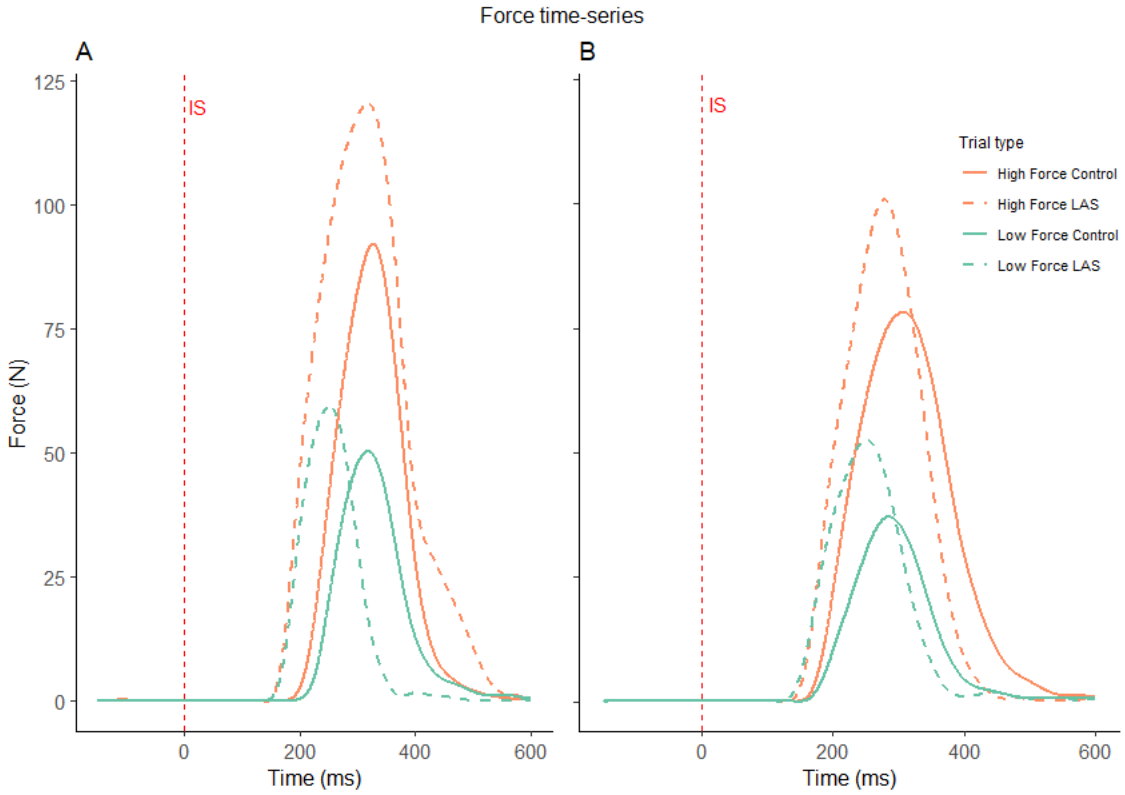








Supplementary Materials



Insert Supplementary Material 1 here.

Caption: Force plotted over the full time-series across Force Level (Low/High) and Trial Type (Control/LAS) for the median trial of each condition for a representative participant in Experiment 1. (A) = Flexion trials, (B) = Extension trials.