The differential effect of metabolic alkalosis on maximum force and rate of force development during repeated, high-intensity cycling

Jason C. Siegler, Paul W. M. Marshall, Sean Rafty, Cristy Brooks, Ben Dowswell, Rick Romero, and Simon Green

1Sport and Exercise Science, School of Science and Health, and 2School of Medicine, University of Western Sydney
Campbelltown Campus, Sydney, Australia

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The differential effect of metabolic alkalosis on maximum force and rate of force development during repeated, high-intensity cycling. J Appl Physiol 115: 1634–1640, 2013. First published October 3, 2013; doi:10.1152/japplphysiol.00688.2013.—The purpose of this investigation was to assess the influence of sodium bicarbonate supplementation on maximal force production, rate of force development (RFD), and muscle recruitment during repeated bouts of high-intensity cycling. Ten male and female (n = 10) subjects completed two fixed-cadence, high-intensity cycling trials. Each trial consisted of a series of 30-s efforts at 120% peak power output (maximum graded test) that were interspersed with 30-s recovery periods until task failure. Prior to each trial, subjects consumed 0.3 g/kg sodium bicarbonate (ALK) or placebo (PLA). Maximal voluntary contractions were performed immediately after each 30-s effort. Maximal force (Fmax) was calculated as the greatest force recorded over a 25-ms period throughout the entire contraction duration while maximal RFD (RFDmax) was calculated as the greatest 10-ms average slope throughout that same contraction. Fmax declined similarly in both the ALK and PLA conditions, with baseline values (ALK: 1,226 ± 393 N; PLA: 1,222 ± 369 N) declining nearly 295 ± 54 N [95% confidence interval (CI) = 84–508 N; P < 0.006]. RFDmax also declined in both trials; however, a differential effect persisted between the ALK and PLA conditions. A main effect of condition was observed across the performance time period, with RFDmax on average higher during ALK (ALK: 8,729 ± 1,169 Ns; PLA: 7,691 ± 1,526 Ns; mean difference between conditions 1,038 ± 451 Ns, 95% CI = 17–2,059 Ns; P < 0.048). These results demonstrate a differential effect of alkalosis on maximum force vs. maximum rate of force development during a whole body fatiguing task.

fatigue; sodium bicarbonate; cycling; acid-base balance

THE PRACTICE OF INDUCING METABOLIC alkalosis to improve exercise tolerance has been prevalent within the field of sport science for approximately three decades (9, 24). Throughout this period, applied research in this domain has investigated the ergogenic potential of various buffering agents [e.g., sodium phosphate, sodium bicarbonate (NaHCO3), or sodium citrate] spanning a wide range of performance outcomes and exercise tasks. The efficacy of these ergogenic aids has been extensively reviewed and the evidence supports NaHCO3 as the most consistently effective aid (9, 27). The oral administration of NaHCO3 will result in a 5–6 mmol/l increase in extracellular blood bicarbonate (HCO3−), with a corresponding shift in extracellular pH from 7.4 to ~7.5 (31). The effectiveness of NaHCO3 appears to improve if the ingestion dose is between 0.2 and 0.3 g/kg body wt, administered between 60 and 120 min prior to exercise, and if the exercise task requires repeated efforts at high intensity with little recovery time between bouts (24).

NaHCO3 is believed to mitigate fatigue through the attenuation of intramuscular acidity (9, 24), although the physiological mechanisms directly responsible for performance augmentation in humans is unknown. Indeed, many of the studies investigating the ergogenic potential of NaHCO3 focus on performance outcomes (e.g., time to task failure, cumulative work accomplished, time trial performance, etc.) (9, 24) and only speculate about underlying mechanisms responsible for any benefit. Although many factors contribute to the muscle’s ability to perform (i.e., generate force) at a peripheral level, the decline in power output induced by repeated intense skeletal muscle contractions can result from either a reduction in muscle force generating capacity (excitation-contraction coupling) or the velocity of contractile shortening (14, 16).

Early work with isolated muscle fibers suggested that the accumulation of protons (H+), and subsequent decline in intracellular pH, might inhibit force production (12). Recent evidence shows that acidosis has little impact on maximal isometric force production at more physiological temperatures (26, 35). However, acidosis significantly impairs muscle shortening velocity (14) and potential underlying mechanisms, including the rate of ADP release from the myosin heads (13, 21) and muscle conduction velocity (5). Some evidence exists to provide a mechanistic hypothesis for how induced alkalosis may affect characteristics of rapid force production. Alkalosis reduces the rise in muscle interstitial potassium (K+) during dynamic exercise (33) and K+ concentration ([K+]i) in the venous effluent from contracting muscle (32). Given that increases in extracellular [K+] slow the propagation velocity of action potentials in contracting muscle (19), alkalosis might also reduce the rate of loss of muscle conduction velocity and its mechanical counterpart, the rate of force development (RFD). However, the effect of alkalosis on RFD under fatiguing conditions has not been tested.

Therefore, we tested the hypothesis that alkalosis reduces the decline in RFD during maximal contractions performed during fatiguing, submaximal exercise, but does not affect maximum force production. Changes in the rate of muscle activation [via surface electromyography (sEMG)] have been associated with changes in RFD (1), and so we also tested the effect of alkalosis on the rate of rise and maximum activity of the sEMG during maximum contractions (2, 34).

MATERIALS AND METHODS

Subjects. Ten recreationally active and healthy male (n = 8) and female (n = 2) subjects [mean ± SD for age = 27 ± 9 yr, height =
One hour after consuming the standardized beverage, capillary blood was collected in a balanced heparinized 200-μL blood gas/capillary tube for immediate analysis of acid-base status [pH, HCO₃⁻, and base excess (BE)] and blood lactate (BLA) using a clinical blood gas analyzer (ABL 800 basic series; Radiometer, Copenhagen, Denmark). All measures were done in duplicate, and the range of intra-class correlation coefficients (ICCs) was \( r = 0.82–0.94 \) for all dependent variables. After the capillary sample, subjects commenced with the ingestion protocol (capsule consumption at \(-90, -60, \) and \(-30\) min prior to the preexercise blood draw) while sitting quietly in the laboratory. After the \(-30\) min ingestion time point, sEMG preparation and normalization procedures were initiated and completed (for sEMG preparation see the heading sEMG). Normalization procedures were conducted with the subjects seated on the VelotronPro.

An initial series of maximal voluntary contractions (MVCs) were performed (see Force) that consisted of submaximal (one 50% and one 75%) and maximal (3 in total, each separated by 1 min) preconditioning efforts. Maximum force was then assessed where each subject performed two 3- to 4-s MVCs, with the maximal voluntary effort assumed as the highest force obtained between the two trials. Subjects were carefully instructed to contract “as fast and forcefully as possible,” and to push directly down into the transducers placed beneath the pedal. Strong verbal encouragement was provided for all MVC trials. Trials with an initial countermovement (identified by a visual drop in the force trace) were always excluded. After completion of the normalization procedures and at the end of the 90-min ingestion protocol, a final capillary blood sample was obtained.

Subjects commenced a standardized warm-up consisting of a 10-min series of short, high-intensity bouts on the VelotronPro. The warm-up included an initial 5 min at 50% PPO followed by 5 min where a stepwise increment (e.g., 60, 70, 80% PPO) in resistance was applied for 15-s bursts followed by a return to 50% PPO for 45 s. The experimental trial required repeated efforts at 120% PPO, with cadence maintained at 90 rpm for 30 s interspersed with 30 s active recovery periods until task failure. Task failure was defined as the inability to maintain cadence (90 rpm) during three consecutive pedal strokes. Post hoc data inspection of the cadence data signal was used to confirm the point in time where cadence was no longer maintained for accurate recording of time to failure. Immediately upon successful completion of each 30-s effort, the force transducers were placed in position beneath the right pedal, and maximal force output (F max) was collected between 5 and 8 s into the 30-s recovery. During
the remainder of the recovery period (~20 s), subjects pedaled at a resistance corresponding to 20% PPO and a self-selected cadence until 25 s into the recovery period, when they were required to increase the pedal rate to 90 rpm in preparation for the subsequent onset of resistance corresponding to 120% PPO. Both \( F_{\text{max}} \) and RFD were assessed immediately after each 30-s bout and throughout the entire protocol. Immediately following task failure and final MVC, a final postexercise capillary blood sample was obtained.

**Force.** \( F_{\text{max}} \) was measured in the subject’s normal cycling posture, with the crank arm raised to a level parallel with the chain stay (~90° knee flexion). A custom-built testing box was placed beneath the crank arm, and the right pedal was positioned upon two linear strain gauges (PT Global, Sydney, Australia) fixed into the box (Fig. 1). Strain-gauge signals were sampled at 1,000 Hz using an analog-to-digital converter (Powerlab, ADI instruments, Australia; 16-bit analog-to-digital conversion). Prior to any data collection, all recorded strain-gauge signals were calibrated to convert the voltage output (mV) to newtons (N). Prior to MVC testing during each trial, strain-gauge signals were corrected for the effect of gravity on the subject’s lower limb and pedal by collecting a 5-s signal with the participant relaxed in the test position. The net force output from the two strain-gauge signals was used for data analysis. Strain-gauge signals were smoothed using a fourth-order digital low-pass filter at 10 Hz prior to data analysis.

To prevent counterrotation of the opposite pedal and to provide additional participant stabilization during the MVCs, a secondary stabilization box was utilized to preclude left pedal movement. To reduce countermovements of the Velotron frame and/or position of the participant during MVCs, subjects were harnessed and secured to wall- and floor-mounted O-rings throughout all experimental sessions. Harnessing the subjects ensured that they could not lift out of the seat during the MVC trials, but could maintain a normal cycling posture during testing. The positioning on the ergometer and tension of the harness was standardized throughout all trials, with pilot data indicating a between-trial reproducibility of ICC \( r = 0.95 \) for all maximal force measurements.

The integrated profile method was used as an objective computer-based method for determining the onset of the force (and sEMG) signal (23, 29). The following force variables were processed from each maximal force trial: 1) \( F_{\text{max}} \) was calculated as the greatest force recorded over a 25-ms period throughout the entire contraction duration calculated, 2) maximal RFD (RFDmax) was calculated as the greatest 10-ms average slope throughout the contraction, and 3) RFD was further calculated as the average slope of the force-time (\( \Delta F/\Delta t \)) curve over time intervals of 0–25, 0–50, 0–75, 0–100, and 0–200 ms relative to the onset of the force signal.

\( \text{sEMG} \). sEMG recordings during the MVCs were collected from the right vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), and gluteus maximus (GM). Pairs of silver/silver chloride electrodes (Maxensor; MediMax Global, Australia) with a 10-mm diameter and 10-mm interelectrode distance were positioned according to standard recommendations for ideal anatomical placement after careful skin preparation using disposable razors to remove excess hair and reduce skin electrode impedance to below 5 kΩ (10). This included measurement of relevant bony landmarks and anatomical protrusions to maintain consistency of placement among subjects. Shielded electrode wires were taped to the right handlebar in a position that did not interfere with subject hand position and to minimize the likelihood of wire movement artifact. sEMG signals were recorded using the ML138 Octal BioAmp (common mode rejection ratio > 85 dB at 50 Hz, input impedance 200 MΩ) with 16-bit analog-to-digital conversion, sampled at 2,000 Hz (ADI instruments, Australia). Raw signals were initially filtered with a fourth-order Bessel filter between 20 and 500 Hz. All collected signals were subsequently digitally band-pass filtered (between 10 and 500 Hz) then rectified and smoothed using a root mean square (RMS) calculation with a 100-ms sliding window (15).

The following sEMG variables were processed from each maximal force trial: 1) peak RMS sEMG amplitude (sEMG_{max}) and maximal rate of sEMG rise (sEMG_{maxrise}; greatest 10-ms activity) within the entire contraction phase; 2) average RMS sEMG amplitude (sEMG_{ave}) at time intervals of 0–25, 0–50, 0–100, and 0–200 ms from the onset of the respective sEMG signal; and 3) average rate of sEMG rise (sEMG_{aveS}) determined from the sEMG/sEMG_{ave} time curve calculated over the corresponding time intervals.

**Data analysis.** For comparison purposes, both \( F_{\text{max}} \) and RFD (maximum and time intervals) were normalized to preexercise values. sEMG_{max}, sEMG_{ave}, sEMG_{maxrise}, and sEMG_{aveS} amplitudes during all maximal force trials were normalized to preexercise maximal contractions elicited for each muscle. No differences were observed between VL and VM amplitudes, thus sEMG-dependent variables were averaged for these muscles to provide an overall representation of quadriceps muscle activity. Additionally, because the recruitment patterns were similar in all muscles, GM and BF muscles have been omitted from the results to allow for better clarity in presentation.

To accommodate for different rates of fatigue (e.g., differences between time to task failure), performance data have been represented and normalized to phases of completion times (20, 40, 60, 80, and 100% completion). The nearest data point from the predetermined completion time point was used for analysis (e.g., if a subject completed eight 30-s bouts, then 20% represents data collected from Bout 2, 40% from Bout 4, etc.). Each completed 30-s high-intensity bout was summed to provide a total exercise time. Where subjects were unable to complete an entire 30 s, the time prior to a drop in cadence (90 rpm) was added to the total exercise time.

Statistical analyses. All statistical analyses were completed using IBM SPSS Statistics version 20 (SPSS, Chicago, IL). Normality of data was checked using Q-Q plots and deemed plausible in each instance. The descriptive data are presented as means ± SD. Blood acid-base (pH, HCO₃⁻, and BE), BLa, \( F_{\text{max}} \), RFD (maximum and time intervals), and sEMG (sEMG_{max}, sEMG_{ave}, sEMG_{maxrise}, and sEMG_{aveS}) amplitudes were analyzed using two-way (condition x bout) ANOVA for repeated measures. In the event of a significant F ratio, post hoc comparisons were made using a Bonferroni correction. Paired t-tests were used to analyze changes in time to failure between conditions. Mean differences and SE between conditions and 95% confidence intervals (CI) were calculated when significant changes over time or when differences between conditions were observed. Two-tailed statistical significance was accepted at \( P < 0.05 \).

**RESULTS**

**Blood acid-base.** Whole blood acid-base findings were consistent with induced states of metabolic alkalosis (Table 1). Significant interaction, condition, and time effects were evident for blood pH (\( P < 0.042 \)), HCO₃⁻ (\( P < 0.001 \)), BE (\( P < 0.003 \)), and BLa (\( P < 0.001 \)). Post hoc analysis for all variables was consistent, with the ALK condition indicating significant metabolic alkalosis from completion of the ingestion protocol to the final capillary sample obtained after task failure (Table 1).

**Time to task failure.** Time to task failure, represented as a cumulative value of completed time during the 120% PPO efforts, was not statistically different between conditions (ALK: 291.1 ± 93.9 s, PL: 275.4 ± 81.9 s; \( P > 0.34 \)).

\( F_{\text{max}} \) production. \( F_{\text{max}} \) immediately prior to exercise was not significantly different between ALK (1,226 ± 393 N) and PL (1,222 ± 369 N) and declined at similar rates during exercise (mean difference from the start of exercise of 295 ± 54 N; 95% CI = 84–508 N; \( P < 0.006 \)). In both conditions a significant fall in \( F_{\text{max}} \) was delayed until 80% of task failure (force at 80% = 1,041 ± 117 N; \( P < 0.02 \); Fig. 2A).

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**Table 1. Blood acid-base (pH, bicarbonate), base excess, and blood lactate responses for both the alkalosis and placebo before ingestion (~90 min), prior to warm-up (0 min), and after task failure**

<table>
<thead>
<tr>
<th>Trial</th>
<th>pH</th>
<th>HCO₃⁻, mmol/l</th>
<th>BE, meq/l</th>
<th>BLa, mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ALK</td>
<td>PLA</td>
<td>ALK</td>
<td>PLA</td>
</tr>
<tr>
<td>−90 min</td>
<td>7.40 ± 0.01</td>
<td>7.38 ± 0.04</td>
<td>24.6 ± 1.1</td>
<td>23.4 ± 2.0</td>
</tr>
<tr>
<td>0 min</td>
<td>(7.39–7.41)</td>
<td>(7.35–7.41)</td>
<td>(23.8–25.5)</td>
<td>(21.8–24.9)</td>
</tr>
<tr>
<td>TF</td>
<td>7.25 ± 0.03</td>
<td>7.17 ± 0.05</td>
<td>14.2 ± 1.3</td>
<td>11.8 ± 1.4</td>
</tr>
<tr>
<td></td>
<td>(7.22–7.27)</td>
<td>(7.13–7.21)</td>
<td>(13.2–15.2)</td>
<td>(10.8–12.9)</td>
</tr>
</tbody>
</table>

Values are means ± SD with 95% confidence intervals in parentheses. BE, base excess; BLa, blood lactate; ALK, alkalosis; PLA, placebo; TF, task failure.

*R < 0.01, significantly different from −90 min. †* < 0.05, significantly different from PLA.

**RFD.** Preexercise RFDmax was not different between ALK (10,879 ± 5,551 N/s) and PLA (11,349 ± 6,378 N/s; *P > 0.54). No interaction effect was evident (*P > 0.13), and both ALK and PLA RFDmax declined over the performance time period (*P < 0.001; Fig. 2B). A main effect of condition was observed across the performance time period, with RFDmax on average higher during ALK (ALK: 8,729 ± 1,169 N/s; PLA: 7,691 ± 1,526 N/s; mean difference between conditions 1,038 ± 451 N/s, 95% CI = 172–2,059 N/s; *P < 0.048; Figs. 2B and 3). With respect to the time from contraction onset of 0–25, 0–50, 0–75, 0–100, and 0–200 ms, alkalosis resulted in an increased RFD from 0 to 25 ms compared with placebo (mean difference of 29.7 ± 7.6 N/s; 95% CI = 12.6–46.8 N/s; *P < 0.003), with a consistent between-condition effect observed across time periods up to 100 ms postcontraction onset (mean difference of 13.9 ± 3.3 N/s; 95% CI = 6.4–21.4 N/s; *P < 0.002; Fig. 4A).

**Maximal and average sEMG (VL and VM) RMS amplitudes.** sEMGmax throughout the experimental protocol was not different between conditions (*P > 0.44), and there were no interaction (*P > 0.78) or time effects (*P > 0.06). By contrast, sEMGave during the initial 50–75 ms postcontraction onset at all time points during exercise were greater in ALK than PLA (0–50 ms: mean difference of 25.3 ± 11.7%, 95% CI = 1–50%, *P < 0.04; 0–75 ms: mean difference of 19.4 ± 9.4%, 95% CI = 0–39%, *P < 0.05). A main effect for time for sEMGave was observed in both conditions during the time periods of 0–100 ms (mean increase of 21 ± 9% from precontraction; *P < 0.05) and 0–200 ms postcontraction onset (mean increase of 20 ± 7% from precontraction; *P < 0.02).

**Maximal and average rate of sEMG rise.** sEMGmaxrise was not different between conditions (*P > 0.10), and there were no interaction (*P > 0.40) or time effects (*P > 0.07). sEMGaverage was only different between conditions (greater in the ALK condition) from 0 to 25 ms postcontraction onset (mean difference of 54 ± 23%, 95% CI = 5–103%, *P < 0.04; Fig. 4B). Thereafter, only a main effect for time (average increase from preexercise) persisted across the performance task for the sEMGaverage during the time periods 0–50 ms (mean increase of 15 ± 13% from precontraction; *P < 0.02) and 0–100 ms postcontraction onset (mean increase of 49 ± 15% from precontraction; *P < 0.01).

**DISCUSSION.**

The aim of this study was to test the effect of alkalosis on neuromuscular fatigue during repeated bouts of high-intensity cycling. The behavior of Fmax production and RFD during brief contractions, as well as their sEMG counterparts, were assessed immediately after each 30-s high-intensity bout (120% peak power) in a series of bouts performed to failure. Although time to task failure was not significantly increased by alkalosis, the decline in RFDmax was attenuated by alkalosis, whereas Fmax production was unaffected. This differential effect of alkalosis was also observed for sEMG during the early period of maximum contraction, but not the maximum EMG. For the first time, these results demonstrate a differential effect of alkalosis on Fmax vs. RFDmax and the underlying muscle recruitment during a whole body fatiguing task.

This differential effect of alkalosis on RFD vs. Fmax and the timing of this effect implicate mechanisms involved in rapid activation of muscle and its shortening during maximum voluntary effort. It has long been known that NaHCO3 increases

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**Fig. 2. Maximal force output (Fmax; A) and rate of force development (RFDmax; B), normalized to preexercise values, during the high-intensity, intermittent cycling protocol. ALK, alkalosis; PLA, placebo.
H\(^+\) efflux from contracting muscle (3, 22) and reduces the accumulation of H\(^+\) during states of high glycolytic flux (28). Recent evidence from isolated single fiber studies suggests that the intracellular accumulation of H\(^+\) (or decline in intracellular pH) has little to no effect on force production at physiological temperatures (26, 35) but that it significantly reduces muscle conduction velocity (5), velocity of shortening (21), action potential propagation velocity (19), and rate of ADP release from myosin (13). These studies demonstrate independent effects of intracellular pH on membrane-bound processes and actomyosin interactions, all of which are expected to reduce the RFD\(_{\text{max}}\) but not necessarily F\(_{\text{max}}\). However, whether these effects of pH occur in vivo will depend on the level of acidosis and the influence of other factors, such as K\(^+\), on dynamic force production (25). Therefore, it is difficult to establish the extent to which the present effect of alkalosis on RFD\(_{\text{max}}\) can be attributed to its effect on intracellular pH.

During submaximal contractions at moderate and higher forces, K\(^+\) accumulates rapidly in the muscle interstitium to levels well in excess of 7 mM (17). Increases in interstitial K\(^+\)-induced effects on muscle membrane properties, which include increasing the action potential threshold (6, 7), are thought to block the activation of myocytes at a step prior to and independent of excitation-contraction coupling and actomyosin interactions and render some myocytes inactive to the neural stimulus (25). Alkalosis dampens the rise in muscle interstitial K\(^+\) during dynamic, submaximal contractions (33) and, via this effect, diminishes the decline in muscle membrane excitability and extent of K\(^+\)-induced inactivation of myocytes during intervening maximum contractions, as performed in the present study. This effect of alkalosis could be manifest during the earlier period of contraction when the level of neural drive to muscle is less than maximum and perhaps insufficient to overcome any K\(^+\)-induced increase in the action potential threshold. This might help explain how alkalosis dampened the decline in the maximum rate of force developed during maximum contractions, but had no effect on F\(_{\text{max}}\) production when the neural drive (EMG) was maximized. In addition, increases in interstitial K\(^+\) are thought to slow the propagation velocity...
Discerning the contributions of pH and interstitial K\(^+\) to the effect of alkalosis on muscle force production in vivo is complicated by the potential interactions between them. Although severe acidosis depresses muscle force production, in the presence of high extracellular K\(^+\) (10 mM) acidosis serves to restore the force loss during dynamic contractions attributed to K\(^+\) (25). Therefore, it could be argued that any intervention which reduces acidosis per se during exercise, when interstitial K\(^+\) rises to high levels, will increase fatigue. K\(^+\), rather than pH, then becomes more important to the mechanism underlying the contractile effect of alkalosis, although the net effect of alkalosis on force production will probably depend on the levels of intracellular pH and extracellular K\(^+\) during exercise and the relative effects of alkalosis on them.

Alkalosis exerted a differential effect on EMG variables, consistent with its differential effect on force variables. The average EMG during the initial period of the maximum contraction, as well as the rate of rise in EMG during this period when force was rising rapidly, was increased by alkalosis at most time points during exercise (Fig. 4). There are at least two ways of interpreting this effect. Alkalosis might reduce a decline in neural activation of muscle during the initial period of contraction (1), possibly by affecting motor cortical drive or motor neuron excitability linked to sensory afferent (type III and IV) feedback from muscle (20). Alternatively, since the surface EMG activity reflects the number of activated muscles and IV) feedback from muscle (20). Alternatively, since the surface EMG activity reflects the number of activated muscles, the damping of this rise in action potential (6, 7), and so the damping of this rise in interstitial K\(^+\) by alkalosis might help reduce the decline in action potential propagation velocity, muscle shortening velocity, and RFD.

In conclusion, for the first time this human study demonstrated that alkalosis exerts a differential effect on fatigue during repeated bouts of high-intensity exercise. Alkalosis reduced the decline in the maximum rate of force production and muscle activation during the initial period of a maximum contraction, but it had no effect on F\(_{\text{max}}\) production and maximum muscle activation. Further work is required to clarify the mechanism underlying this differential effect.

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