Effect Of Eccentric Contraction-Induced Injury On Individual Quadriceps Muscles

MERCER UNIVERSITY
COLLEGE OF HEALTH PROFESSIONS

Chris Rawdon Ph.D.[1], Christopher Ingalls Ph.D.[2], Feng Yang Ph.D.[2], Jeffrey Otis Ph.D.[2], Kyle Brandenberger Ph.D.[2], Mekensie Jackson M.S.[2], Ryan Middleton M.S.[2]

[1]Mercer University, Macon, GA; [2]Georgia State University, Atlanta, GA

Mercer Health Sciences Center

INTRODUCTION

When exercise is unaccustomed, the repetitive loading can lead to a grade I injury of skeletal muscle often referred to as exercise-induced muscle injury. Research has shown that exercise-induced muscle injury can cause 25-50% reductions in maximal joint torque.[1] Strength deficits associated with exercise-induced muscle injury are normally assessed by measuring changes in joint torque which reflect the functional integrity of a given set of synergistic muscles. However, it is unknown whether individual muscles of a synergist group are injured to the same extent following injurious exercise. We hypothesized that the extent of injury among synergistic muscles is not uniform, and the primary cause of the weakness stems from the failure of muscle and not the ability of the nervous system to activate the muscle. We also presumed that muscle injury would alter balance (postural sway) and quadricep muscle activation patterns (electromyography [EMG]) during locomotion.

METHODS

15 healthy sedentary or recreationally active male subjects between 18 and 35 years old completed the study. Subjects performed either downhill running (DHR) on a treadmill for 60 min to induce injury (n=8) or level treadmill walking for 30 min as control (n=7). Before and after (immediately and 2-days) exercise, we measured 1) maximal voluntary contraction (MVC) torque of quadricep muscles (QMs) on a Biodex dynamometer, 2) torque produced by vastus medialis (VM) and vastus lateralis (VL) via electrical stimulation (20 and 80 Hz) 3) soreness of individual QMs, 4) QMs EMG root mean square (RMS) during running and MVCs, and 5) standing postural sway calculated by ground reaction forces.

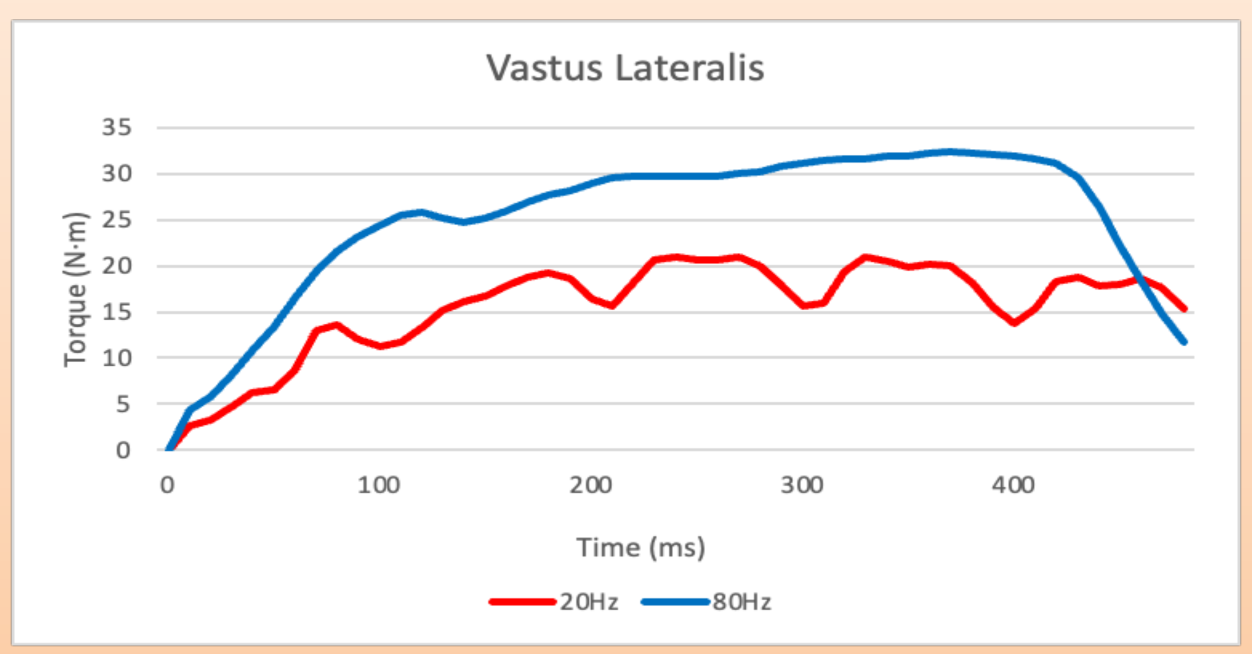

Figure 1. Torque produced by Vastus Lateralis during low (20 Hz) and high (80 Hz) frequency stimulation. Data collected from a single subject on a Biodex dynamometer during pilot study.

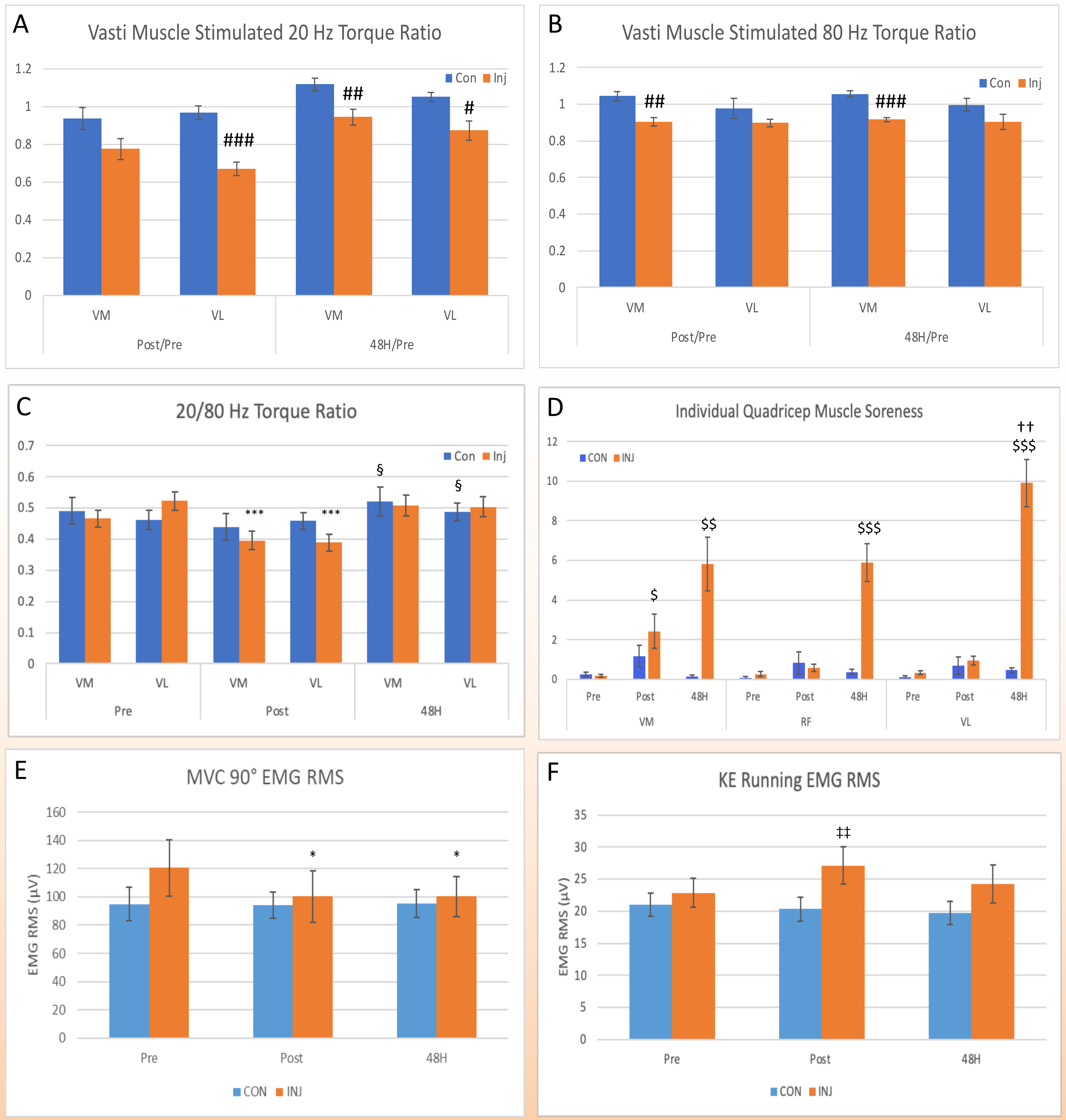

Figure 2. All values are mean ± SEM. A) 20 Hz and B) 80 Hz normalized to Pre for individual muscle torque for the control and injury group for the vastus medialis (VM) and vastus lateralis (VL). #, Significantly less than Control ($p \le 0.05$). ##, Significantly less than Control ($p \le 0.01$). ###, Significantly less than Control ($p \le 0.001$). C) 20/80 Hz torque ratio for both groups at baseline (Pre), immediately following (Post), and 48 hours (48H) following either walking (Con) or downhill running protocols (Inj). ***, Significantly less than Pre (and 48H $p \le 0.001$). §, Significantly greater than Post ($p \le 0.05$). D) Individual quadriceps muscle soreness values collapsed across legs. $, Significantly greater than Pre ($p \le 0.05$). $$, Significantly greater than Pre ($p \le 0.01$). $$$, Significantly greater than Pre ($p \le 0.001$). †† Significantly greater than VM and RF ($p \le 0.01$). E) Electromyography (EMG) Root mean square (RMS) of non-dominant knee extensors during maximal voluntary contractions MVCs at 90°. *, Significantly less than Pre ($p \le 0.05$). F) Running EMG RMS collapsed across both legs. ‡‡, Significantly Significantly greater than Pre & 48H ($p \le 0.01$).

RESULTS

- MVC torque was significantly reduced immediately (25.3%) and 2-days (14.0%) after DHR, whereas torque was unchanged after level walking.
- Immediately following DHR, 20 Hz stimulated torque relative to baseline was lower ($p = 0.054$) in the VL (67.1 ± 0.4%) than the VM (77.5 ± 0.6%). 20 Hz stimulated torque deficit remained lower ($p = 0.051$) in the VL (87.5 ± 0.4%) than the VM (94.5±0.4%) 48 hours after DHR. There were no statistical differences ($p \ge 0.810$) in the 80 Hz torque deficit (89.7 ± 0.4% to 91.5 ± 0.2%) for the injured vasti muscles.
- Immediately after DHR, the 20/80 Hz torque ratio decreased ($p = 0.001$) by 20.6% across both muscles. No muscle interaction ($p \ge 0.109$; Observed Power = 0.442) was present for 20/80 Hz torque ratio.
- At 48 hours, the VL experienced greater ($p \le 0.004$) soreness than RF and VM in the DHR group.
- After DHR (immediately and 2-days), MVC RMS across the QMs was significantly reduced by 16.8% immediately following injury.
- Running RMS of the QMs increased immediately after DHR.
- Postural sway length increased by 2.1% from baseline ($p = 0.024$) immediately after DHR while it decreased 11.6% ($p = 0.017$) in the control group at 2-days. The postural sway returned to baseline levels for the DHR group at 48H.

CONCLUSIONS

Our study has confirmed differential injury across synergists muscles due to significantly higher soreness in the VL than the RF and VM at 48H for the downhill running group. This is the first study of its kind to compare torque produced both low-frequency (20 Hz) and high-frequency (80 Hz) stimulations across the individual muscles of a synergist muscle group in humans. DHR caused a significant loss of intrinsic force capacity compared to walking controls. There was a trend towards differential submaximal (20 Hz) torque loss between the VM and VL muscles for the injury groups immediately ($p = 0.054$) and 48 hours ($p = 0.051$) following the downhill run. Reduced activation (i.e., RMS) of the quadriceps (r^2= 0.45; $p < 0.001$) and intrinsic force depression (i.e., 20 & 80 Hz torque) (r^2= 0.186; p= 0.025) account for the decreases in MVC torque after DHR. Muscle injury from DHR disrupted standing balance and normal muscle activation patterns during running.

PRACTICAL APPLICATIONS

It has been previously proposed that unbalanced and asymmetric activities can create differential fatigue across muscles and thereby a kinetic imbalance could result in musculoskeletal injury.[2] Strength imbalances of a working muscle group can promote instability of the joint[3,4,5] and this instability could potentially lead to injuries such as ligament sprains or tendinopathies. Any exercise or activity that causes differential strains on a muscle group, may result in leaving a person more susceptible to a secondary injury due to altered joint mechanics.

REFERENCES

1. Warren, G. L., Ingalls, C. P., Lowe, D. A., & Armstrong, R. B. (2002). What mechanisms contribute to the strength loss that occurs during and in the recovery from skeletal muscle injury?. Journal of Orthopaedic & Sports Physical Therapy, 32(2), 58-64.
2. Kumar, S. (2001). Theories of musculoskeletal injury causation. Ergonomics, 44(1), 17-47.
3. Yaggie, J. A., & McGregor, S. J. (2002). Effects of isokinetic ankle fatigue on the maintenance of balance and postural limits. Archives of physical Medicine and Rehabilitation, 83(2), 224-228.
4.Fitzgerald, G. K., Piva, S. R., & Irrgang, J. J. (2004). Reports of joint instability in knee osteoarthritis: its prevalence and relationship to physical function. Arthritis care & research, 51(6), 941-946.
5. Khalaj, N., Vicenzino, B., Heales, L. J., & Smith, M. D. (2020). Is chronic ankle instability associated with impaired muscle strength? Ankle, knee and hip muscle strength in individuals with chronic ankle instability: a systematic review with meta-analysis. British journal of sports medicine, 54(14), 839-847.