

**Characterization of perceived muscle soreness and prediction of skeletal muscle markers of
damage following a bout of high intensity functional resistance training**

Lauren Elliott

University of Wyoming

Mentor:

Dr. Evan C. Johnson, PhD

Assistant Professor of Exercise Physiology

Hydration Laboratory

Department of Kinesiology and Health Promotion

University of Wyoming

Program:

Wyoming INBRE & UW Honors

May 1st, 2019

INTRODUCTION:

Skeletal muscle damage during exercise is caused by repeated eccentric motions. The damage increases as exercise volume and intensity increases, resulting in soreness and fatigue (Proske & Morgan, 2001). The muscle pain associated with skeletal muscle damage is sub-optimal for athletes as it may hinder them i

When skeletal muscle cell components enter the bloodstream, such as those measured by plasma osmolality, symptoms of muscle pain, weakness, and myoglobin in the urine may manifest (Torres et al. 2015). One hypothesis is that skeletal muscle damage releases skeletal muscle cell components into the bloodstream that sensitizes the nociceptors, leading to increased pain following a difficult exercise bout (Proske & Morgan). As such, it is important to understand the pain experienced by athletes during intense, long duration exercise as these exercise bouts can lead to skeletal muscle damage. Identifying predictors of severe skeletal muscle damage, such as patterns in pain perception or plasma osmolality, may reduce potential risks associated with high volume or extended duration workouts, such as High Intensity Functional Resistance Training (HIFRT) workouts.

Therefore, the purpose of this project is to determine if pain perception is related to markers of skeletal muscle damage following a standardized bout of High Intensity Functional Resistance Training (HIFRT). When athletes complete a bout of HIFRT, such as the *will* experience skeletal muscle breakdown due to the large number of repeated repetitions necessary for completion of the workout. However, each individual will have a unique response to the mechanical stressors, leading to a unique pain experience and change in plasma osmolality due to training status. This will allow us to examine if a relationship exists pain perception and plasma osmolality.

METHODS:

Nineteen subjects participated in the study, consisting of 13 males and 6 females (25.8±6.5 years old, 174.4±7.9 cm tall, with an average body weight of 77.9±13.7 kg, and reported an average training experience of 3.5±1.3 years). Each of the subjects completed the

standardized HIFRT

300 air squats, and a one mile run. Participants were asked to visit the lab during a series of five time points: 24 hours before exercise, immediately before exercise, immediately after exercise, 24 hours after exercise and 48 hours after exercise. At each time point, participants completed the Short Form McGill Pain Questionnaire and a blood draw (Melzack 1987). The Short Form McGill Pain Questionnaire consisted of 15 pain descriptors ranked on a scale of 0 to 3, a Visual Analog Scale (VAS) 100 mm long, and a Present Pain Intensity Scale (PPIS) scored 0-5 in order to monitor overall perceived muscle soreness (Melzack 1987). The Short Form McGill Pain scale was filled out independently by each participant in order to gain a more complete understanding of each participants perceived muscle soreness following the exercise bout (Melzack 1987). Blood samples obtained from the blood draw were used to measure plasma osmolality. The blood samples were collected in Lithium Heparin coated tubes, placed on a tube rocker for four minutes, and then centrifuged for ten minutes at 2300 RPM at 7 degrees (Beckman Coulter, Germany). Then, 250 μ L of the sample was aliquoted into a microcuvette and placed in the osmometer, which determined the osmolality via freezing point depression (Advanced Instruments Inc., Massachusetts, USA). The osmolality of the sample was then used as an indirect marker of skeletal muscle damage, as significant elevations in plasma osmolality may be due to higher concentrations of skeletal muscle cell components released into the bloodstream.

A repeated measures ANOVA test (RM-ANOVA) was conducted on pain scores derived from the McGill Short Form Pain Questionnaire as well as the mean plasma osmolality from each time point to determine main effects of time on each variable. Post-hoc t-tests with corrections for multiple comparisons were used to determine differences of post-workout time points from the immediate pre-workout scores. Additionally, a stepwise linear regression

analysis was conducted using SPSS to determine the strength of the relationship between post-exercise VAS and plasma osmolality at various time points. All statistical analyses were conducted using SPSS statistics software (SPSS Inc., Chicago, IL, USA).

RESULTS:

The RM-ANOVA output revealed significant main effects of time for eleven of the fifteen pain descriptors. H and all were significantly elevated when compared to baseline immediately following exercise (Individual time point differences are shown in Table 1.

Table 1. Effect of time across 15 pain descriptors and Visual Analog Scale derived from the Short Form McGill Pain Scale.

| Pain Descriptor | Immediately Post | 24 Hours Post | 48 Hours Post |
|------------------------|-------------------------|----------------------|----------------------|
| Throbbing | X | X | X |
| Shooting | | | |
| Stabbing | | | |
| Sharp | | | |
| Cramping | X | X | |
| Gnawing | | | |
| Hot-Burning | X | | |
| Aching | X | X | X |
| Heavy | X | | |
| Tender | | X | X |
| Splitting | | | |
| Tiring-Exhausting | X | X | |
| Sickening | X | | |
| Fearful | | | |
| Punishing-Cruel | X | | |
| Visual Analog Scale | X | X | X |

significantly elevated above baseline at that time point.

The RM-ANOVA also revealed a main effect of time for plasma osmolality as values were significantly elevated above baseline immediately following the workout. Individual h time point are shown in Table 2.

Table 2. Plasma Osmolality at all time points

| | Baseline | I-Pre | I-Post | 24h Post | 48h Post |
|------------------------------------|-----------------|--------------|---------------|-----------------|-----------------|
| Plasma Osmolality (mOsm/kg) | 287±3 | 287±4 | 290±6* | 287±4 | 287±4 |

* Indicates significantly different from baseline ($p < .050$).

The stepwise regression analysis revealed a significant relationship between VAS immediately after exercise and mean plasma osmolality 48h after exercise ($p=0.025$; Figure 1)

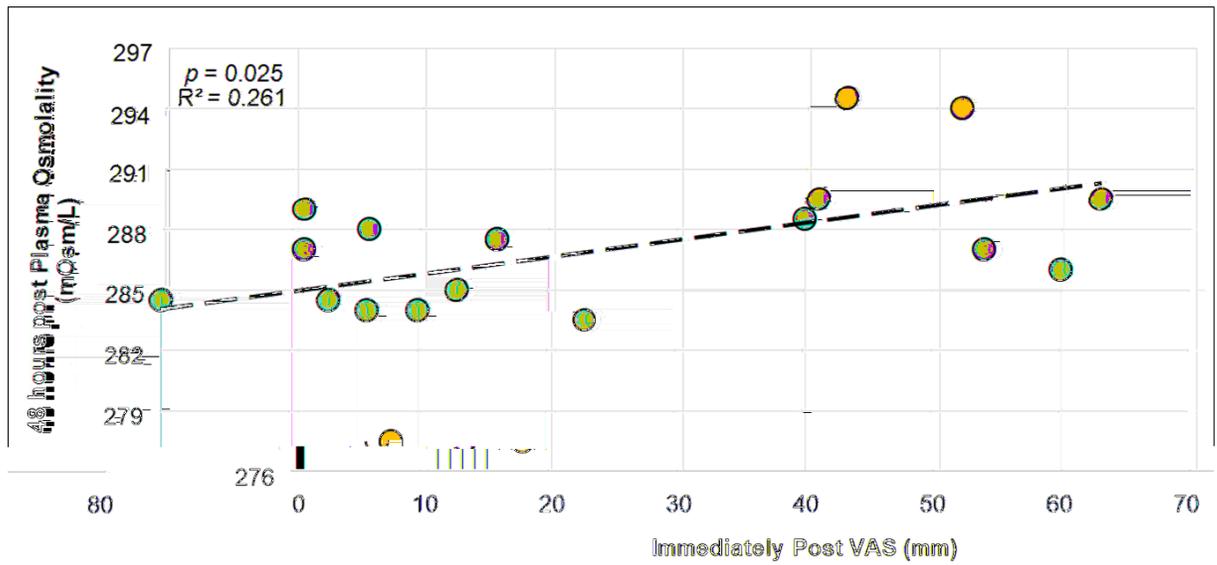


Figure 1. Regression relationship between VAS immediately post workout and plasma osmolality 48 hours post workout.

DISCUSSION:

From the analysis of the responses to the McGill Short Form Pain, eleven of the fifteen pain descriptors were effective in describing the pain perceptions experienced by the participants following the standardized bout of HIFRT. These pain descriptors should be used to monitor athletes who engage in HIFRT style workouts, as other pain words may not be as effective in describing the HIFRT pain experience. The VAS was also a useful tool in describing the overall pain perceptions related to HIFRT as it provided a generalized, overall understanding of the pain

experience following the HIFRT bout. Overall, this tells us that the workout did cause a measurable change in the participant's perception of pain.

Table 2 shows that plasma osmolality was significantly elevated from the baseline measure. It is known that Sodium, Blood Urea Nitrogen (BUN), and Glucose are the osmotically active particles in plasma (Calderon et al. 2015) which could contribute to the elevated plasma osmolality. Of those solutes, the concentration of Sodium is low in the skeletal muscle cell, so cell lysis would not significantly increase the concentration of Sodium outside the cell. Additionally, Glucose is used by the skeletal muscle cell as an energy source and would not be released into the bloodstream following skeletal muscle cell lysis. Therefore, urea is the only compound that would change plasma osmolality immediately following the workout. We hypothesize that the increase in plasma osmolality could be due to an increase in urea being released into the bloodstream by the damaged skeletal muscle cell, as urea is an essential component of the skeletal muscle cell.

Pain perception is related to markers of skeletal muscle damage as evidenced by the regression relationship seen in Figure 1. The individuals who perceived more pain immediately following the workout, determined by higher VAS scores, demonstrated higher plasma osmolality 48 hours after the workout. The delay in elevated plasma osmolality occurs as it takes time for skeletal muscle damage to develop. Previous research has shown that CK levels peak within 1-3 days following muscle injury (Huerta-Alardin et al. 2005). As such, the delay in elevated plasma osmolality was expected as it takes time for skeletal muscle damage to develop. As we hypothesize that a release of urea into the bloodstream is contributing to increased plasma osmolality, the urea may also contribute to increased pain perceptions. Urea in the bloodstream can be painful, as is the case with patients dealing with Gout, which is an accumulation of uric

acid crystals which causes joint discomfort (Dalbeth et al. 2016). This hypothesis is similar to that of Proske & Morgan (2001) as the urea released by the skeletal muscle would sensitize the nociceptor and lead to an increase in pain perception. However, future testing is needed to determine increased levels of urea in blood samples.

As the participants with higher VAS scores immediately following the workout are more likely to have elevated plasma osmolality 48 hours after the workout indicating skeletal muscle damage, these participants should be monitored closely for other signs of Rhabdomyolysis, such as darkening of the urine or muscle weakness, or signs of acute kidney injury. During future HIFRT workouts, these participants may benefit the most from scaling movements to prevent severe skeletal muscle damage which places them at risk of Rhabdomyolysis.

LITERATURE CITED:

Calderon, J.L., Bustos, F., Tuesta-Reina, L.R., Varona, J.M., Caballero, L and Solano F. Choice for best equation for plasma osmolality calculation: Comparison of fourteen formulae.

Clinical Biochemistry. 529-533 (2015).

Dalbeth, N., Merriman, T.R., and Stamp, L.K. Gout. *Lancet*. 2039-2052 (2016).

Huerta-Alardin, A.L., Varon, J., and Marik, P.E. Bench to bedside review: rhabdomyolysis- an overview for clinicians. *Critical Care*. 9. 158-169 (2005).

Melzack, R. The short form McGill pain questionnaire. *Pain*. 2. 194-197 (1987).

Proske, U. & Morgan, D.L. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *Journal of Physiology*. 537. 333-345 (2001).

Torres, P.A., Helmstetter, J.A., Kaye, A.M., and Kaye A.D. Rhabdomyolysis: pathogenesis, diagnosis, and treatment. *The Ochsner Journal*. 15. 58-69 (2015).