A COMPARATIVE PILOT ANALYSIS OF CELLULAR RESPONSES TO LOW-INTENSITY RESISTANCE EXERCISE WITH AND WITHOUT BLOOD FLOW RESTRICTION

Jessica M. Moon, Paola M. Rivera, Kadie R. Drahos, Blake W. Johnson, Trevor J. Dufner, and Adam J. Wells

Cellular Exercise Physiology Lab (CEPL)

Exercise Physiology, Intervention, and Collaboration (EPIC) Lab

Institute of Exercise Physiology and Rehabilitation Science (IEPRS), University of Central Florida

Introduction

Blood flow restriction (BFR) training has been shown to achieve comparable muscle strength and hypertrophy gains to traditional high-load resistance training, while utilizing significantly lower loads in combination with venous occlusion[1]. As such, lower loads and comparable positive outcomes make BFR an attractive option for those who are unable to perform high-load resistance exercise (RE). High-intensity RE has been shown to initiate a cascade of immune responses that are essential for the restoration of normal physiological function and muscle growth[2]. However, the mechanisms underpinning the efficacy of BFR remain elusive with several theories hypothesizing that the increased metabolic stress BFR induces, may stimulate muscle growth via increased recruitment of fast-twitch muscle fibers[3], increased inflammatory and endocrine response[4], and cellular swelling[5] amongst others. Considering the involvement of the immune system in muscle recovery and hypertrophy, an examination of immune responses to low-intensity exercise alone, as well as with BFR, is necessary to isolate the specific effect that BFR may have compared to low-load exercise alone.

The purpose of this study was to examine the effects of low-intensity RE with and without BFR on cellular immune responses of inflammation, focusing on clinically relevant markers to assess systemic inflammation.

Methodology

Participants: Fourteen recreationally active women

- BFR (n = 8)
  - 21 ± 1.4yrs., 164.5 ± 6.9cm, 72.2 ± 13.3kg
- RE (n = 6)
  - 22.2 ± 3.6yrs., 164.3 ± 7.3cm, 80.4 ± 30.2kg

Design:

- Randomized parallel group design
  - Two experimental trials over an 8-week training program.

Training Program:

- Two days per week consisting of leg press, leg extensions, leg curls
  - 1 x 30 repetitions & 3 x 15 repetitions with 30 seconds rest between sets
- BFR: Cuffs inflated at 60% of their total occlusion pressure

Experimental Trials:

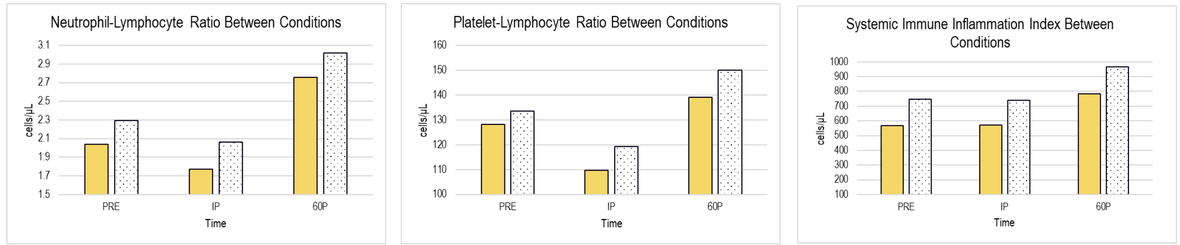
- ET1 was performed during the first training session of week 2 and consisted of a pre-exercise (PRE) blood sample, a training session, and two post-exercise blood samples: immediately post- (IP) and 60-minutes post-exercise (60P).
- ET2 was conducted in an identical fashion to ET1 during the first training session of week 8.

Biochemical Analysis

- Blood draws were obtained using standard phlebotomy techniques using a superficial forearm vein. A total of 4mL of blood was collected at each time point in 4-ml K2 EDTA Vacutainer Tubes (Becton Dickinson, Franklin Lakes, NJ).
- Whole blood samples were analyzed same-day using an automated hematology analyzer (Sysmex XN-450 Automated Hematology Analyzer).
- Samples were analyzed for complete blood counts which were used to calculate cellular inflammation markers of **neutrophil to lymphocyte ratio** (NLR = neutrophil counts/LYM counts, **platelet to lymphocyte ratio** (PLR = platelet counts/LYM counts), and **systemic immune-inflammation index** (SII = platelet counts × NEUT counts/LYM counts).

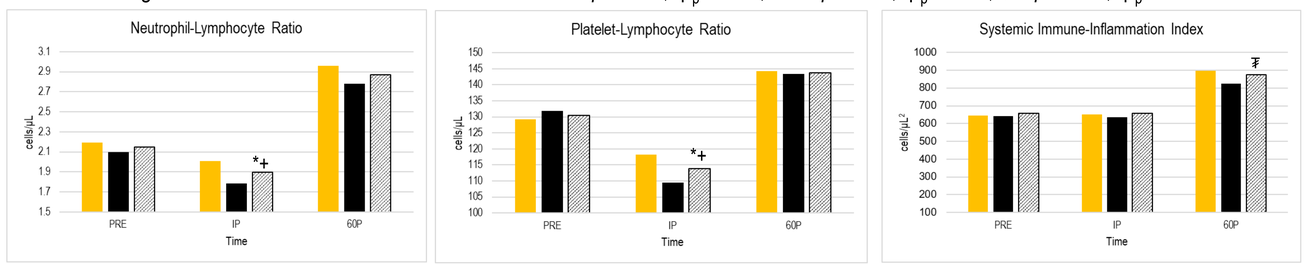
Results

- No significant interactions, main effect of group, or main effect of ET were reported for any clinical outcome variable (p's >.05).



Changes in clinical outcomes between conditions collapsed across Experimental trials.

- Significant main effects for time were observed: **NLR:** p = .022, η^2_p = .357; **PLR:** p = .011, η^2_p = .402; **SII:** p = .050, η^2_p = .280



Changes in clinical outcomes collapsed across groups. * = significantly different than PRE; + = significantly different than 60P; ∓ = significantly different than IP

Practical Applications

Given that low-load RE, with or without BFR, does not significantly differ in immune response, this form of exercise may potentially be useful for those who are immunocompromised, those who have chronic inflammatory conditions, or for practitioners looking to design effective and safe protocols for patients to prevent muscle atrophy while avoiding excessive immunological stress.

Further, this investigation provides insight and advances our knowledge of the physiological processes involved in BFR interventions by confirming that there are comparable immunological mechanisms between BFR and non-BFR exercise.

Statistical Analysis

- An EM algorithm was used to impute any missing values for the dependent variables.
- All missing data were deemed to be missing completely at random (MCAR), as determined by Little's MCAR test.
- Data imputation was performed for each subscale (same variable along ET time points) for each treatment group independently.
- Normality was assessed using the Shapiro-Wilk test for each group independently.
- Separate three-way (Group [BFR, RE] x Time [PRE, IP, 60P] x ET [Wk-2, Wk-8]) repeated measures ANOVA were used to examine changes in cellular inflammation markers (NLR, PLR and SII) between groups over time.
  - If the assumption of Sphericity was violated a Greenhouse-Geisser correction was applied.
  - In the event of a significant interaction Fisher's LSD post hoc analyses were used for pairwise comparisons.
- Significance for all analyses was accepted at an alpha level of $p \leq 0.05$.

Conclusions

- There were no significant differences in cellular immune responses to low-load RE between the BFR and RE only groups, however the low-load RE regimen induced notable acute changes in clinical cellular inflammation markers.
- Clinical markers of NLR and PLR, although altered by the exercise intervention, remained within normal ranges, whereas increases in SII at 60P indicate acute inflammation, albeit below values suggesting substantial systemic inflammation.
- There were no significant cellular adaptations to the exercise bout following 8 weeks of training.

Acknowledgements

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