Introduction

Blood flow restricted exercise (BFRE) with low loads has gained interest during the last years because it induces muscle hypertrophy to a similar extent as conventional heavy load strength training. Although it is highly debated, BFRE has recently been suggested to elicit considerable stress to exercising muscles and in some cases, muscle damage. The degree of stress caused by BFRE has, however, been poorly investigated at the cellular level.

The heat shock proteins (HSP), such as α-crystallin, are typical intracellular markers of cellular stress and known to translocate and accumulate in the affected areas within the cell. AIMS: To compare the acute and long-term effects of a failure (FA) vs submaximal (SU) BFRE protocols on α-crystallin response in exercising muscles.

Methods

Sixteen untrained men (18-45 yrs) completed 14 BFRE sessions divided into 2 blocks of 7 sessions in 5 days, interspersed by 10 days of rest (Fig. 1). Legs were randomly assigned to either FA (4 sets to voluntary failure) or SU protocol (30-, 15-, 15-, 15 reps) using unilateral knee extensions at 20% of 1RM with 30s rest between sets. BFRE was conducted with partial blood flow restriction (100 mmHg) induced by a 15 cm wide pressure cuff.

Biopsies from the m.vastus lateralis were collected before (Pre), 2h after the first session (+2h), during the rest period (+11d) and 10 days post intervention (+29d).

The HSP response investigated was changes in α-crystallin staining intensity on muscle cross sections analyzed by immunofluorescence. The staining intensity was measured using ImageJ (with a mean ± SD of 209 ± 170 fibers/time point).

Results

Relative to pre-exercise (100%), a significant increase in α-crystallin staining intensity (reflecting cytoskeletal bound proteins) was observed 2h after the first session of both BFRE protocols (FA: 234.7 ± 179.6%, P<0.0001 and SU: 173.7 ± 95.7%, P<0.05, respectively) (Fig. 2-3). There was no significant difference between protocols at any time point, but the acute response of α-crystallin tended to be larger in FA legs than SU legs. The overall change in α-crystallin staining intensity gradually decreased to baseline values during the rest period (FA: 142.6 ± 68.8% and SU: 118.2 ± 60.13%) and 10 days post intervention (FA: 91.10 ± 35.83% and SU: 80.70 ± 33.28%). Note that a large intersubject variability was observed, especially in the acute response (Fig. 4).

Discussion

The increase in α-crystallin staining intensity indicates that both FA and SU BFRE are able to induce cell stress and possible damage to cytoskeletal structures. The translocation of HSP to myofibrillar structures after low-load BFRE is probably related to ischemia rather than mechanical stressors. It should be noted that some subjects were not able to complete all the repetitions during SU. Consequently, our SU protocol was close to failure in the first training sessions. In addition, since fiber-type specific adaptations after BFRE have already been observed, one main perspective is to investigate α-crystallin in type 1 and type 2 fibers. In parallel to the immunohistological analyses, immunobLOTS and ELISA for HSP are carried out and should improve understanding of the acute and chronic HSP response after FA and SU training.

Conclusions

1. The results in this study suggest that cytoskeletal proteins are stressed after the first session of both low-load FA and SU BFRE protocols. 2. No accumulation of this small HSP in cytoskeletal structures seems to occur after a period of BFRE training.

References:

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