CHRD 2021: Abstract & Poster Submission Form

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Research Category:

- ◉ Basic Science
- ○ Clinical
- ○ Community Health / Policy

What was your role in the project?

- ☐ Design
- ☑ Perform Experiments
- ☑ Analyze Data
- ☑ Write Abstract
- ☐

Presenter Status:

- ○ Undergraduate Students
- ○ Masters Student
- ◉ PhD Student
- ○ Post-Doctoral Fellows
- ○ Residents
- ○ Non-Trainee

Title

Skeletal muscle extracellular vesicles are altered with chronic exercise and can transmit exercise-associated metabolic reprogramming

Background

Chronic exercise evokes positive systemic benefits such as an increase in mitochondrial content/activity termed mitochondrial biogenesis (MitoB). This can be potentiated in part by the release of myokines from skeletal muscle. Myokines can be packaged within extracellular vesicles (EVs), which are small lipid membrane-bound structures that enclose biological cargo, and constitute an essential method of cellular communication. Previous work indicates exercise increases plasma EV yield, but the effects of exercise on skeletal muscle-derived EVs are poorly understood.

Objective

We hypothesize that exercise alters muscle-EV biophysical profile (size, zeta potential or stability, yield), and that these muscle-EVs can transmit the pro-metabolic effects of exercise.

Methods

Mouse myoblasts (muscle cells) were differentiated into myotubes, and electrically paced (3h/day x 4days @14V, C-PACE EM, IonOptix) to mimic exercise in vitro. EVs were isolated (N=8) from conditioned media from control and stimulated myotubes using differential ultracentrifugation. Isolated EVs were characterized biophysically. Myoblasts were treated with control or stimulated EVs daily for 4 days (N=6), and analyzed for changes in MitoB, cell count and viability.

Results

EV size was 74% smaller in stimulated vs. control EVs ($p<0.05$). Size distribution analysis revealed that stimulated EVs were enriched with 100-150 nm small EVs, while control EVs were enriched with 200-250 nm ($p<0.05$). Zeta potential was 1.2X lower, and protein yield unchanged in stimulated vs. control EVs ($p<0.05$). Myoblasts treated with stimulated EVs had 1.2X higher cell count and 56% higher cytochrome c oxidase activity, a marker of MitoB, vs. cells treated with control EVs. Stimulated EV treatment had no effect on cell viability.

Conclusion

Our data show that stimulation evoked the release of more stable, and smaller sized muscle-EVs that increased MitoB, and cell count in non-exercised cells. This novel and exciting finding supports the potential of exercise-derived skeletal muscle-EVs in mediating the pro-metabolic effects of exercise.

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