CHRD 2022: Abstract & Poster Submission Form

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Presenter Status

○ Undergraduate Students

○ Masters Student

◉ PhD Student

○ Post-Doctoral Fellows

○ Residents

○ Non-Trainee

Research Category

◉ Basic Science

○ Clinical

○ Community Health / Policy

Role in the project

☑ Design

☑ Perform Experiments

☑ Analyze Data

☑ Write Abstract

☐

Title

The pro-apoptotic effect of chronic contractile activity-induced extracellular vesicles on Lewis Lung Carcinoma (LLC) cells

Background

Regular exercise reduces tumor growth in vivo and in vitro, but the exact mechanisms have yet to be fully elucidated. Extracellular vesicles (EVs) are small, lipid membrane-bound structures that enclose biological cargo, and constitute an essential method of cellular communication. We have previously shown that chronic contractile activity (CCA) increases the concentration of skeletal muscle-derived EVs, and these in turn increased mitochondrial biogenesis in C2C12 myoblasts.

Objective

Here, we hypothesized that skeletal muscle-derived EVs post-CCA will mediate the anti-tumorigenic effects associated with chronic exercise.

Methods

C2C12 myoblasts were differentiated into myotubes, and electrically paced (3 hrs/day x 4 days @ 14 volts, C-PACE EM, IonOptix) to mimic chronic exercise. EVs were isolated from conditioned media from control and CCA myotubes using differential ultracentrifugation. Lewis lung carcinoma (LLC) cells were treated with control or CCA-EVs daily for 4 days. Cell count, viability, metabolism, and apoptosis was measured after treatment.

Results

CCA-EV treatment reduced cell count in LLC cells by ~6%, and cell viability by ~20% vs. control-EVs (p=0.03, N=6). There was no effect of CCA-EV treatment on metabolism, as measured by cytochrome c oxidase activity, Mitotracker Red staining and Complex I subunit-NDUFB8 expression. CCA-EV treatment increased the expression of pro-apoptotic protein markers: Bax by 24% and Bax/Bcl-2 ratio by 60% (p=0.03, N=6), and senescence marker HMGB1 by 48% (p=0.07, N=6) vs. control-EVs. The expression of other apoptotic proteins, p53, AIF, Bcl2, cytochrome c, caspase-3, and cleaved caspase-3 remained unchanged with CCA-EV treatment.

Conclusion

Our data show that CCA-induced skeletal muscle-EVs reduced cell count and viability, and increased the expression of some pro-apoptotic markers in LLC cells. This illustrates the potential of CCA-derived skeletal muscle-EVs in mediating the anti-tumorigenic effects of chronic exercise. More research is needed to investigate the mechanisms involved, and whether this persists across different cell lines/species.

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