Metformin is a leading pharmaceutical intervention to target the biology of aging in humans. Exercise is currently the gold standard intervention to improve muscle and metabolic health in aging. However, combining metformin with exercise prevents adaptations to mitochondrial function, insulin sensitivity, and muscle strength/size.

**Metformin alters the muscle transcriptome reducing the total number of up and down regulated genes by aerobic exercise training**

Data interpretation:
- *demonstrates AET is significantly different compared to both SED and AET+MET
- # demonstrates AET is significantly different to only AET+MET unless otherwise specified

**Summary**
- Metformin inhibits mitochondrial adaptions to aerobic exercise in mice similar to humans
- Metformin prevents decreased mitochondrial complex I site IF hydrogen peroxide emissions
- Metformin reduced total skeletal muscle gene expression following aerobic exercise training

Future work will expand on these preliminary findings by evaluating potential upstream/downstream mechanisms of metformin regulating mitochondrial adaptions to exercise.

Future work will broaden these findings to determine if age, sex, or antecedent metabolic health influences how metformin interacts on skeletal muscle with exercise.

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