Mitochondrial dysfunction is the hallmark of numerous diseases including cardiac ischemia, Alzheimer’s disease, Parkinson’s disease, liver disease, cancer, diabetes, and Lou Gehrig’s disease. This is primarily due to the aging defects caused by the mitochondria and the production of Reactive Oxygen Species (ROS). Currently, there is limited research analyzing the whole mitochondrial network to demonstrate differences in older and younger patients. The objective of this review is to analyze collected mitochondrial proteomic data to detect the proteomic divergence within the aged heart relative to the younger heart. Specifically, understanding the key differences between the Subsarcolemmal mitochondrial (SSM) and the Interfibrillar mitochondria (IFM) was investigated. This review focused on proteomic data achieved from the mouse model using LC-MS/MS data. Proteomics satisfies this exigence by enabling accurate, system-wide quantitative analysis of protein supply. In addition, cutting-edge proteomics methods reveal how proteins perform their functions in convoluted interaction networks where minute alterations that occur can be detected from early pathological states. The proteomic differences in the SSM and IFM reveal evidence for decreases in the rate of oxidative phosphorylation and inactivity in the AMPK pathways which reduces the yield of ATP molecules. Metformin treatment was seen to increase ATP yield making it a potential mitigator of cardiac injury. A further genetic experiment could be conducted to trace genes that might be upregulated or downregulated in the mitochondrial genome. Reversing the effects of mitochondrial aging can increase lifespan and decrease the likelihood of diseases to occur at old age.

Abstract

Mitochondrial Free Radical Theory of Aging & Cardiac ischemia

Age related decrease in oxidative phosphorylation was present in the IFM and not the SSM. Therefore, the IFM mitochondria yield much less ATP molecules than the SSM through age. Metformin, a common drug for diabetes improved respiration rates in the aged mice. Therefore, metformin could be a potential protein drug that could mitigate the effects of age related OXPHOS damage. The heart muscle uniquely houses two distinct subpopulations of mitochondria known as the Subsarcolemmal mitochondria (SSM) and the Interfibrillar mitochondria (IFM). They both differ in their biochemical properties and relative location. Studies analyzing how both subpopulations react with age and cardiac ischemia will be the primary lens in this review.

Conclusions

There are significant differences in how the SSM and IFM mitochondrial subpopulations react to cardiac ischemia and age related changes. The IFM mitochondria are less resistant to damage through age than the SSM. In addition, metformin treatment proved to mitigate the damage of the IFM mitochondria and reduce cardiac injury through age. Metformin treatment also yields more ATP molecules in the aged mouse than aged mice without metformin treatment. Future experiments analyzing the whole proteomic structure of the mitochondria would be extremely beneficial in tracing the exact age-related disparity between the SSM and IFM. Then, results could show concentration increases and decreases through age which could then be adjusted through drugs to increase longevity and prevent painful deaths.

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Results & Data Analysis

Mitochondrial Free Radical Theory of Aging & Cardiac ischemia

Cardiac ischemia occurs when fatty plaque clogs arteries and stops normal blood flow leading to heart muscle death. To fix the issue,identifying procedures are carried out and the sudden resumption of blood flow causes reperfusion damage to nearby tissue.

Mitochondrial Free Radical Theory of Aging & Cardiac ischemia

Mitochondria are responsible for age related complications of cardiac ischemia and age related mortality rates of older ischemic patients.理解 this study has found directly which was form a 24 month mouse.

Conclusion

There are significant differences in how the SSM and IFM mitochondrial subpopulations react to cardiac ischemia and age related changes. The IFM mitochondria are less resistant to damage through age than the SSM. In addition, metformin treatment proved to mitigate the damage of the IFM mitochondria and reduce cardiac injury through age. Metformin treatment also yields more ATP molecules in the aged mouse than aged mice without metformin treatment. Future experiments analyzing the whole proteomic structure of the mitochondria would be extremely beneficial in tracing the exact age-related disparity between the SSM and IFM. Then, results could show concentration increases and decreases through age which could then be adjusted through drugs to increase longevity and prevent painful deaths.