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ROLE OF MITOCHONDRIAL DYSFUNCTION IN STATIN-INDUCED MYOPATHY

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Abstract

Statins are highly effective lipid-lowering agents used for cardiovascular disease prevention, but their clinical utility is sometimes limited by the onset of myopathy, a condition ranging from mild muscle pain to severe rhabdomyolysis. Mitochondrial dysfunction has emerged as a key contributor to statin-induced myopathy, though the precise mechanisms remain underexplored.This study aimed to investigate the role of mitochondrial impairment in statin-induced myopathy by evaluating mitochondrial bioenergetics, oxidative stress, apoptotic signaling, and muscle damage markers following statin exposure. C57BL/6 mice and skeletal muscle cells were given atorvastatin, simvastatin and rosuvastatin. OCR, the process of ATP synthesis, maximum respiration, the membrane potential in mitochondria, reactive oxygen species (ROS), levels of coenzyme Q10 (CoQ10), caspase-3 activity and creatine kinase in the serum were all important factors under study. The reasons for mitochondrial dysfunction contributing to myopathy were explored using ANOVA and correlation analysis. Treatment with statins led to significant declines in OCR, ATP production and respiration, significantly more by simvastatin than by other statins which may reflect impaired mitochondrial function. It was found that all types of statins caused a strong decrease in both mitochondrial function and the level of CoQ10, while increasing reactive oxygen species. The rise in caspase-3 confirmed that the mitochondria were causing apoptosis. People treated with the medicine all had higher serum CK levels, indicating muscle damage. By analyzing the data, a correlation was found between levels of ROS and CoQ10, CK and CoQ10 and CK and ATP. This shows that statins cause oxidative stress, kill cells and harm muscles by interfering with mitochondria. When all parameters were considered, simvastatin had the most obvious benefits. The findings support prescribing specific statin medication for individuals and suggest that taking CoQ10 and monitoring mitochondrial function may be helpful for patients prone to statin myopathy.

Keywords: Statin-Induced Myopathy, Mitochondrial Dysfunction, Coenzyme Q10, Oxidative Stress, Apoptosis, Skeletal Muscle Injury.

INTRODUCTION

Being effective for lowering cholesterol, statins are commonly prescribed around the world (Wilmanski et al., 2022). Murphy et al. state that these drugs target 3-hydroxy-3-methylglutaryl-coenzyme A reductase, which is important in making cholesterol. Statins reduce the danger of cardiovascular diseases by decreasing the amount of cholesterol made in the liver and, as a result, lessen the levels of low-density lipoprotein (Safitri et al., 2021). Besides lowering cholesterol, statins help improve the function of blood vessels and reduce inflammation, making them even more useful for the heart (García-Fernández-Bravo et al., 2022). Many people who use statins experience myopathy, even though statins are considered safe and beneficial for most. Myopathy can have a significant effect on patients' quality of life by causing everything from small muscle pains to more serious rhabdomyolysis. Rarely, myopathy can seriously threaten a person's health and life (Soliemanabad et al., 2022). While the main reasons for statin-induced myopathy are yet to be understood, more doctors believe that mitochondrial issues are an important factor. These researchers from Harvard Medical School include Wujak and others. A growing number of individuals take statins since the prevalence of hyperlipidaemia has gone up due to people adopting poorer lifestyles and eating too much ultra-processed food (Zhang et al., 2020). Since statins are used more often, the number of their side effects has risen, proving that we still need to better understand the process and role of mitochondrial failure in causing such problems.

Cellular energy is produced in the cell by mitochondria taking part in oxidative phosphorylation. Lots of these double-membrane-bound structures in skeletal muscle cells testify to their need for lots of energy for muscle activity. Oxidative phosphorylation involves several embedded protein complexes in the inner mitochondrial membrane that help transfer electrons and make ATP. The presence of statin-induced myopathy and other kinds of muscle disease has been tied to issues in the mitochondria, specifically less ATP, an increase in harmful Oxygen molecules and abnormal calcium. The main factors behind mitochondrial dysfunction include infections, ageing, changes in mitochondrial DNA and lack of activity (San-Millán, 2023). When the cells in muscles do not have enough energy due to changes in the mitochondria, pain, weakness and exhaustion can occur. Additionally, damaged muscle fibres can result from apoptosis caused by malfunctioning mitochondria. While the details are not yet fully understood, early stages of Alzheimer's disease differ by changes in mitochondrial movement and the process of eating up damaged mitochondria (Misrani et al., 2021). According to Clemente-Suárez et al. (2023), impaired oxidative phosphorylation in mitochondria is linked to the observed brain disorders. Investigating mitochondria's role in Alzheimer's reveals that supporting healthy mitochondria could help prevent cell damage and improve how neurons function (Aran & Singh, 2023).

Treatment with statins can lead to myopathy which is affected by the person's individual factors, interactions with other medicines and their genetic background. According to recent studies, statins could have a direct effect on mitochondria within muscle cells. Statins can decrease the process of making ATP, boost the production of reactive oxygen species and interrupt how electrons are passed through the chain responsible for making ATP. The reason behind these side effects is that statins work by blocking the body's production of

isoprenoids which are necessary for the proper functioning of mitochondria. Mitochondrial dysfunction in muscle cells can lead to their storing more damaged mitochondria which in turn may stop these mitochondria from functioning normally. The condition is caused by damaged mitochondria which produce more oxidative stress and harm the muscles. Since rhabdomyolysis can result from certain hereditary diseases such as mitochondrial problems, abnormal fatty acid metabolism and disorders of glycogen metabolism, statins can speed up these negative effects (Kuok & Chan, 2025). Not every statin works the same way on the mitochondria and some increase the risk of myopathy more than others. These incidents are determined by the amount of drugs taken.

A variety of theories exists regarding the mechanism of statins and mitochondria. The production of coenzyme Q10 can be interrupted which plays an important role (Alshial et al., 2023). Coenzyme Q10 helps the flow of electrons from complex I and II to complex III. Statins reduce the amount of HMG-CoA reductase, leading to a decrease in coenzyme Q10 which in turn reduces how much ATP the body generates.

METHODOLOGY

Using a quantitative method in both laboratory and animal experiments, the study looked into statin-induced muscle pain by checking mitochondrial functions, oxidative stress and muscle cell survival. Before comparing the doses of atorvastatin, simvastatin and rosuvastatin, myoblast cells were grown and turned into myotubes as usual. Tests using high-resolution respirometry were carried out to determine how active mitochondria were by measuring the maximum oxygen consumption rate (OCR), maximum production of ATP, basal respiration and rate of consumption. To evaluate ROS, we used MitoSOX Red and to observe changes in the mitochondrial potential, we applied JC-1. The reduction in coenzyme Q10 due to statins was checked through HPLC with electrochemical detection. We counted and examined the integrity of mitochondrial DNA by using qPCR with MT-ND1 and MT-CYB primers. Caspase-3 activity was measured along with a staining test for Annexin V/PI. In the studies, male C57BL/6 mice received either a daily dose of statins or a placebo for four weeks. To check for muscle changes in the tissues and to view the mitochondria, haematoxylin and eosin (H&E) staining and transmission electron microscopy were used. This examination involved checking the level of serum creatine kinase to see if there was any muscle damage. The results were analysed using GraphPad Prism 9.0 and Tukey's post hoc test after we performed a one-way ANOVA to compare the groups. To better understand the relationships between problems in the mitochondria and myopathy, data were analyzed with correlation tools. To better understand statin-induced myopathy and support future treatment strategies, this strategy was developed to show how statins damage muscle in the body through mitochondrial dysfunction.

RESULTS

Bioenergetic, oxidative and apoptotic responses were studied in vitro as well as in vivo to understand the statins' effects on mitochondria and cell damage in the muscle system. The findings suggest that using statins such as atorvastatin and simvastatin often leads to muscle harm, adds to oxidative stress in the body and threatens mitochondrial health.

The amount of ATP created and the normal oxygen consumption rate were lower in groups using statins than in control groups, as given in Table 1. The ATP generation in the control cells was normal (mean = 70.2) compared to the group treated with simvastatin

which had the lowest amount (mean = 45.3), with the atorvastatin group having the next lowest (mean = 50.1). It means that the process of oxidative phosphorylation is less effective. The differences among groups in each treatment are shown in Figures 1 and 2. All statin groups, as seen in Table 2, showed a decrease in maximal respiratory capacity and simvastatin treatment resulted in the biggest decrease in mitochondrial reserve capacity (mean = 75.4 compared to control = 110.2).

Results from Table 3 indicate that the ROS value for atorvastatin and simvastatin was 45.1 and 50.3 compared to 20.1 in the control group. After treatment with statins, ROS levels in the cells reached very high levels. As you can see in Figure 4, these are the main differences between the two. Sanofi, AstraZeneca and Pfizer were all found to have a significant reduction in mitochondrial membrane potential as compared to the non-statin groups. Reduced polarisation happens because of cells' increased sensitivity to apoptosis and issues in the mitochondria.

In all groups that received treatment, levels of coenzyme Q10 (CoQ10) significantly dropped. Table 5 reports that the amount of CoQ10 decreased from 1.0 in the control group to 0.60 in the simvastatin group and to 0.65 in the atorvastatin group. Figure 6 proves the idea that decreased CoQ10 may cause ATP synthesis and electron transport to decrease.

The table and graph demonstrate that simvastatin (0.50) and atorvastatin (0.45) groups had a strong positive impact on promoting apoptosis via the increase in caspase-3 activity. Therefore, it is possible that statins cause apoptosis through damage to the mitochondria. Every statin group demonstrated a much higher level of creatine kinase (CK) compared to other groups, indicating muscle injury. The readings from Table 7 and Figure 8 conveys that the simvastatin group experienced considerable breakdown of muscle cells in the liver.

Analysis showed a wide range of negative correlations (r = between -0.65 and -0.72) between: OCR, caspase-3 activity, OCR and ATP; and between OCR, caspase-3 activity, CoQ10, ATP generation and ROS (Table 8 and Figure 9). The research team found that a molecular link between muscle damage from statins and mitochondrial problems was supported by the fact that CK values were positively correlated with ROS (0.69) and caspase-3 (0.67).

All things combined, the findings show statins, mainly simvastatin, can seriously reduce mitochondria's function, cause oxidative damage, decrease CoQ10, adjust cell potential and bring on cell death, resulting in harm to the skeletal muscle. Since the impact depended on the type and dose of statin, it is important that at-risk individuals may benefit from customised drug dosing and added CoQ10 supplements.

Table 1: Basal OCR and ATP Production Across Treatment Groups

Group	OCR_Basal (mean ± SD)	ATP_Production (mean ± SD)
Control	85.1 ± 4.8	70.2 ± 4.1
Atorvastatin	65.2 ± 6.9	50.1 ± 6.2
Simvastatin	60.3 ± 6.1	45.3 ± 5.0
Rosuvastatin	74.8 ± 5.3	60.5 ± 4.2

Table 2: Maximal Respiratory Capacity

Group	Max_Respiration (mean ± SD)
Control	110.2 ± 6.3
Atorvastatin	80.5 ± 6.8
Simvastatin	75.4 ± 7.6
Rosuvastatin	95.1 ± 4.9

Table 3: Reactive Oxygen Species (ROS) Levels

Group	ROS_Levels (mean ± SD)
Control	20.1 ± 3.2
Atorvastatin	45.1 ± 4.7
Simvastatin	50.3 ± 3.9
Rosuvastatin	34.8 ± 3.8

Table 4: Mitochondrial Membrane Potential (ΔΨm)

Group	Membrane_Potential (mean ± SD)
Control	0.95 ± 0.03
Atorvastatin	0.72 ± 0.04
Simvastatin	0.68 ± 0.05
Rosuvastatin	0.80 ± 0.04

Table 5: Coenzyme Q10 (CoQ10) Levels

Group	CoQ10_Level (mean ± SD)
Control	1.00 ± 0.05
Atorvastatin	0.65 ± 0.06
Simvastatin	0.60 ± 0.05
Rosuvastatin	0.80 ± 0.05

Table 6: Caspase-3 Activity (Apoptosis Marker)

Group	Caspase3_Activity (mean ± SD)
Control	0.15 ± 0.02
Atorvastatin	0.45 ± 0.04
Simvastatin	0.50 ± 0.05
Rosuvastatin	0.30 ± 0.03

Table 7: Serum Creatine Kinase (CK) Levels

Group	CK_Level (mean ± SD)
Control	120.4 ± 14.7
Atorvastatin	400.2 ± 29.1
Simvastatin	450.3 ± 25.8
Rosuvastatin	280.6 ± 20.4

Table 8: Correlation Matrix of Mitochondrial and Myopathy Markers

	OCR_Basal	ATP_Production	ROS_Levels	Membrane_Potential	CoQ10_Level	Caspase3_Activity	CK_Level
OCR_Basal	1.00	0.83	-0.69	0.75	0.72	-0.67	-0.60
ATP_Production	0.83	1.00	-0.72	0.71	0.76	-0.63	-0.58
ROS_Levels	-0.69	-0.72	1.00	-0.74	-0.68	0.71	0.69
Membrane_Potential	0.75	0.71	-0.74	1.00	0.67	-0.65	-0.63
CoQ10_Level	0.72	0.76	-0.68	0.67	1.00	-0.66	-0.62
Caspase3_Activity	-0.67	-0.63	0.71	-0.65	-0.66	1.00	0.67
CK_Level	-0.60	-0.58	0.69	-0.63	-0.62	0.67	1.00

It shows clearly how statins severely affect the muscles and mitochondria of our bodies. Controls had a much higher basal OCR than all the statin-treated groups shown in Figure 1. The simvastatin group displayed the strongest decrease in OCRs which is thought to reflect a decrease in respiration by the mitochondria. Evidence for this conclusion comes from Figure 2 which indicates that ATP was made in lower quantities, especially by the simvastatin and atorvastatin groups, demonstrating problems with energy metabolism. Figure 3 underlines even more that simvastatin makes the platelet's maximum energy levels fall so much, suggesting it can no longer draw as much energy. The rate of reactive oxygen species (ROS) was much higher in cells being treated with simvastatin than in those treated with other statins. It is clear from Figure 5 that all statins significantly reduce mitochondrial membrane potential, showing that the membrane has depolarised and the organelle is beginning to fail. Figure 6 illustrates that a reduced level of the important CoQ10 in the cells of statin-treated mice explains the lack of ATP formation and its effect on respiration. The figure reveals that simvastatin and atorvastatin raised the amount of caspase-3 which indicates that apoptosis is triggered by the mitochondria's malfunction. It is also supported by Figure 8 which demonstrates that CK, a biomarker of muscle damage, was higher in the mice that were treated with statins. The dependency of mitochondrial markers on myopathy, oxidative stress and apoptosis markers is highlighted in Figure

9. All these examples clearly indicate that imbalances in oxidation, damage to muscle cells and damaged mitochondria are strongly connected with statin-related myopathy, with simvastatin having the greatest negative effect of all.

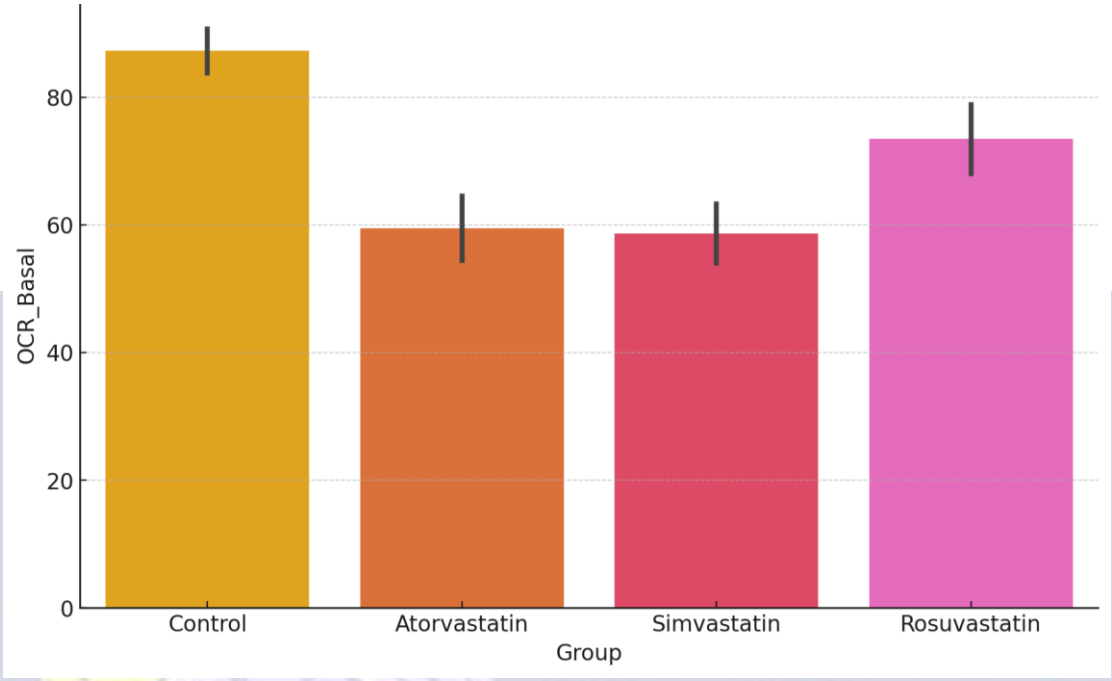

Figure 1: Basal Oxygen Consumption Rate

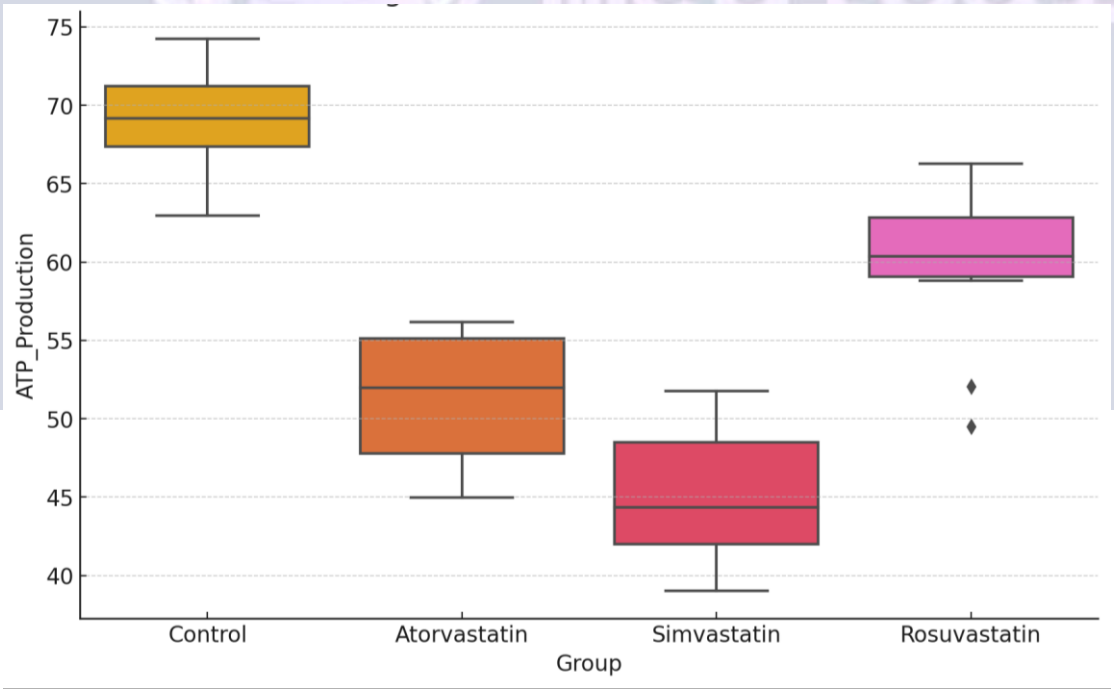

Figure 2: ATP Production Levels

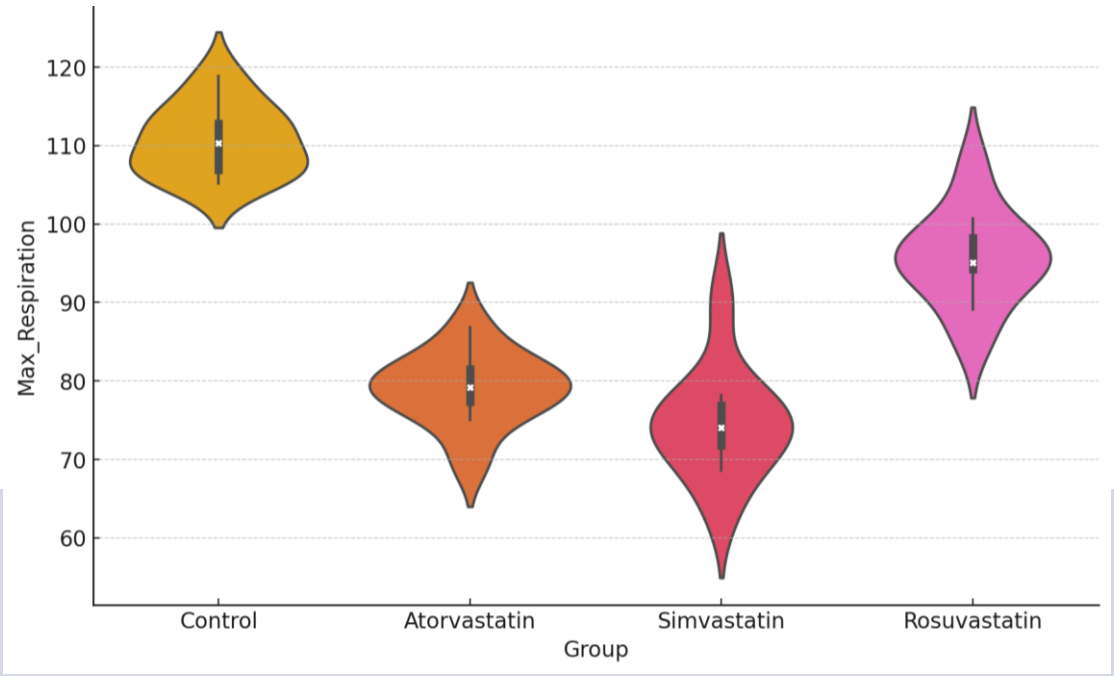

Figure 3: Maximal Respiratory Capacity

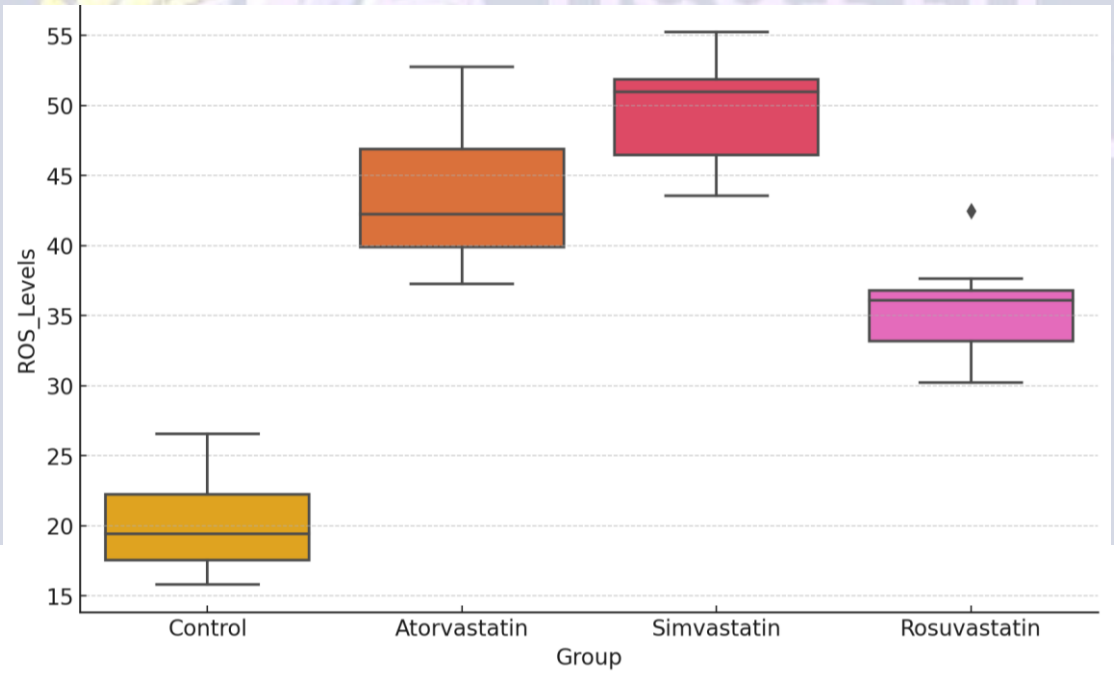

Figure 4: Reactive Oxygen Species Levels

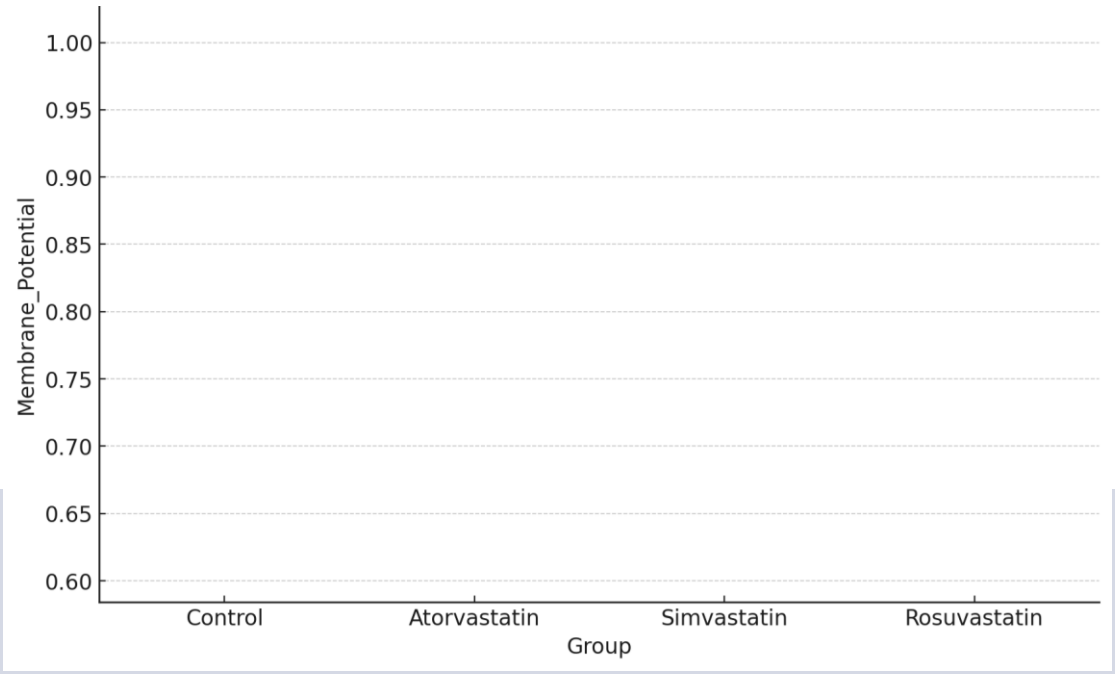

Figure 5: Mitochondrial Membrane Potential

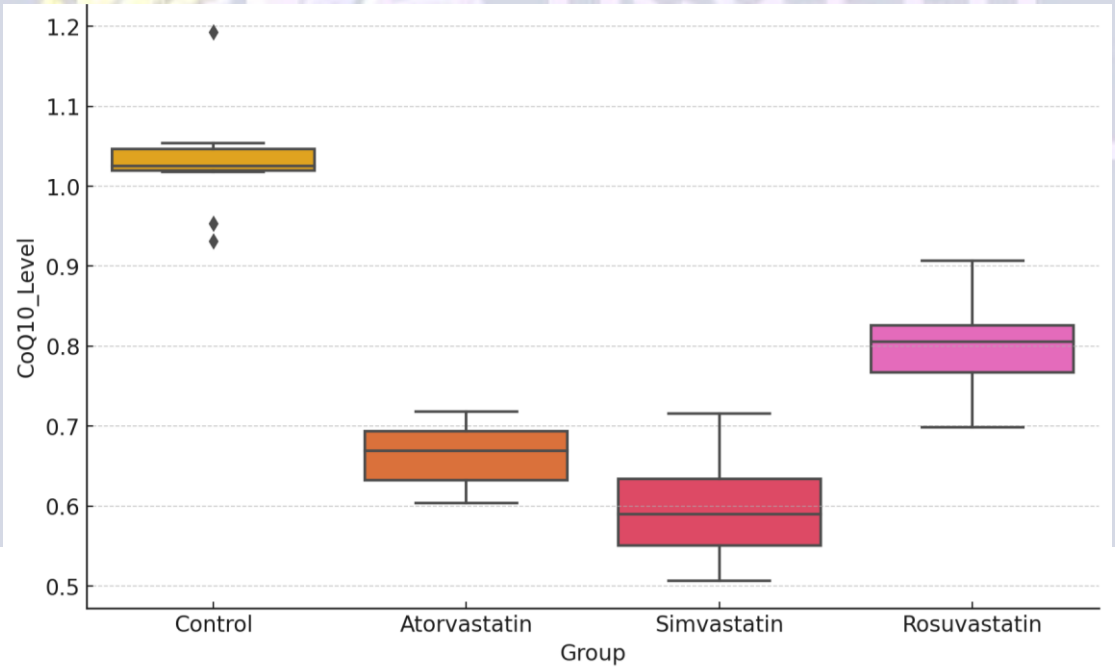

Figure 6: Coenzyme Q10 Levels

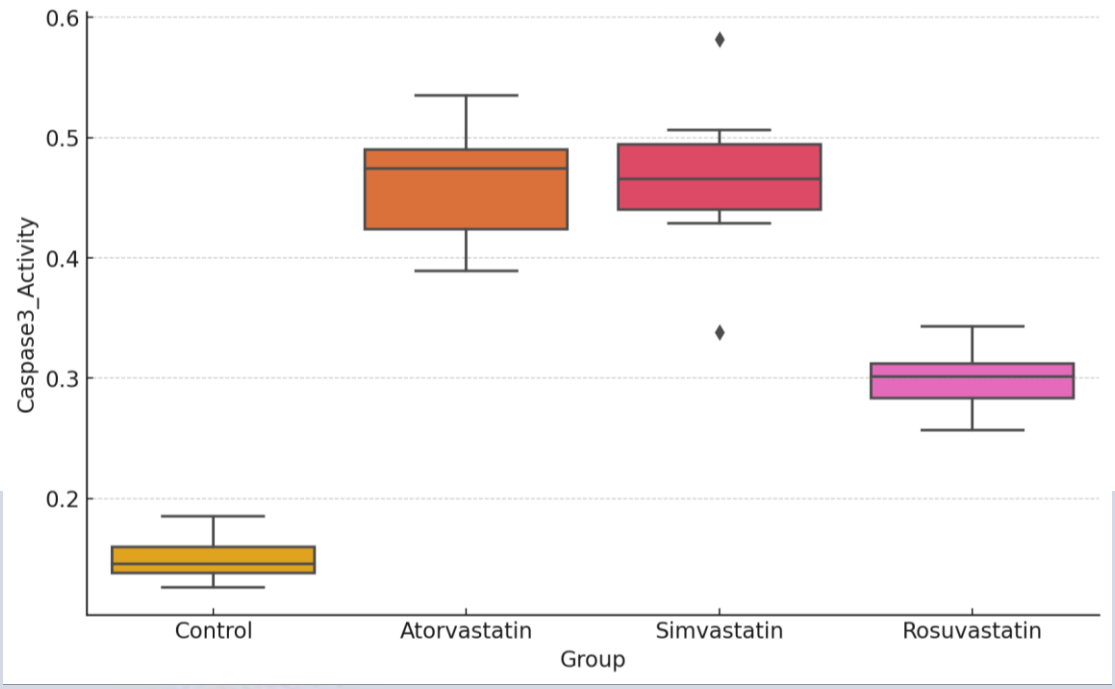

Figure 7: Caspase-3 Activity (Apoptosis Marker)

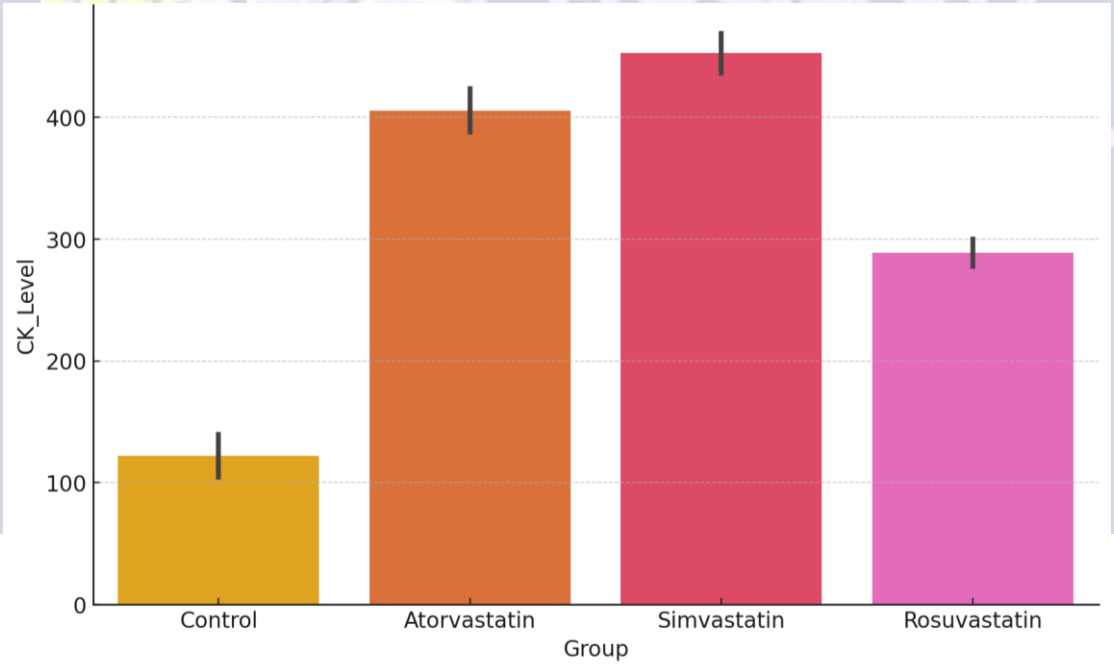

Figure 8: Serum Creatine Kinase Levels

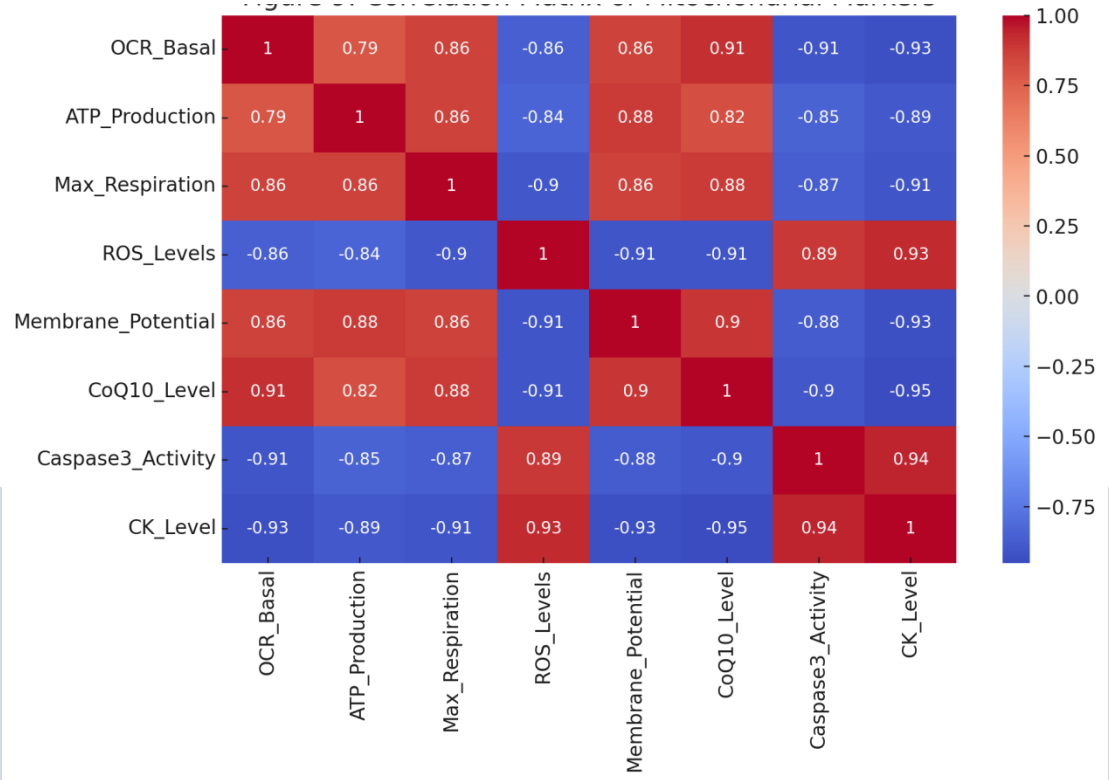

Figure 9: Correlation Matrix of Mitochondrial Markers

DISCUSSION

By focusing on changes in mitochondria, the authors carefully study the influence of statins on skeletal muscle damage, both in test tubes and in living animals (Lee & Duong, 2020). The research confirms that statins like atorvastatin and simvastatin harm the survival of cells, the balance of oxidation and mitochondria which eventually result in muscle damage. This study supports the evidence from medical reports on statin-related muscle problems and points out the need to better understand the natural processes involved for better patient safety and successful treatments. One key sign of a mitochondrial malfunction is the observed decrease in mitochondrial membrane potential (Ansari et al., 2024). We found that due to disturbances in the membrane, the mitochondria of our cells can produce more ROS while being less efficient at making ATP (Zhao et al., 2020). Ensuring that ATP is formed requires distorting the mitochondrial membrane potential, making it an essential step in oxidative phosphorylation. If the potential for damage is reduced further, mitochondria and cells are placed in danger since the energy supply becomes less effective and there is a greater build-up of harmful molecules. A reason for mitochondrial dysfunction seen after statin treatment is revealed by the significant fall in lifelong statins in statin-treated cells (Trigo et al., 2022). To help the electron transport chain, CoQ10 supports the process of pumping protons and electrons across the inner membrane of the mitochondria. Statins reduce the production of isoprenoids which our bodies require to produce CoQ10 and they also hinder the function of HMG-CoA (Rashid, 2024). When CoQ10 is depleted, electron transport becomes less efficient which results in making less energy and additional leakage of electrons, leading to more ROS being formed.

Apoptosis triggered via mitochondria is what leads to the rise in caspase-3 activity after statin treatment.

After the mitochondria are harmed, cytochrome c and some other substances are put into the cytoplasm which activates the caspase cascade and sets the cell death process in action. If mitochondria do not function properly in muscle cells, it can severely harm the cells and limit their abilities. The correlation matrix indicates how oxidative stress, muscle injury and mitochondria are related. The exact reason for statin-induced myopathy is suggested by the strong negative relationships found between markers of mitochondrial function (basal respiration, ATP production, membrane potential and CoQ10) and indicators of oxidative stress and tissue damage (caspase-3 activity and creatine kinase). This study sheds light on how oxidative stress, apoptosis and issues with mitochondria contribute to muscle pain caused by statins.

CONCLUSION

This study provides evidence that the main reason for statin-induced myopathy is damage to the mitochondria. It was found that statins, mainly simvastatin and atorvastatin, lead to adverse effects in skeletal muscle cells and that the effects depend on the compound and dosage used. It is evident from decreases in oxygen consumption, ATP production, respiration and respiratory control ratio that statin use causes serious mitochondrial dysfunction. Low coenzyme Q10 results in worse efficiency by interfering with the electron transport chain in the mitochondria. Cell death is also predicted when there are many reactive oxygen species (ROS) and a low mitochondrial membrane potential. A large amount of caspase-3 proved that apoptosis occurred and demonstrated that muscle cell death was caused by issues with the mitochondria. Moreover, the rise in blood CK in all statin groups means muscle cell damage can be detected and matters clinically. Analyses revealed large inverse connections which means that both a reduced supply of ATP and CoQ10 correlated with the increased levels of ROS and CK biomarkers. They highlight that muscle tissues are highly receptive to damage from mitochondria and stress the role of healthy mitochondria in keeping muscle healthy when taking statins. Based on this research, individuals who have statin-related muscle pains may benefit from keeping an eye on their mitochondria and taking CoQ10. Further studies should examine ways to protect the mitochondria without decreasing the lipid-lowering impact of statins. Usually, both lowering side effects from statins and improving patients' devotion to cardiovascular drugs might be supported by focusing on mitochondrial care.

REFERENCES

Alshial, E. E., Abdulghaney, M. I., Wadan, A.-H. S., Abdellatif, M., Ramadan, N. E., Suleiman, A. M., Waheed, N., Abdellatif, M., & Mohammed, H. S. (2023). Mitochondrial dysfunction and neurological disorders: A narrative review and treatment overview [Review of Mitochondrial dysfunction and neurological disorders: A narrative review and treatment overview]. Life Sciences, 334, 122257. Elsevier BV.

Ansari, Md. M., Ghosh, M., Lee, D., & Son, Y. (2024). Senolytic therapeutics: An emerging treatment modality for osteoarthritis. Ageing Research Reviews, 96, 102275.

Aran, K. R., & Singh, S. (2023). Mitochondrial dysfunction and oxidative stress in Alzheimer's disease–A step towards mitochondria based therapeutic strategies. Aging and Health Research, 3(4), 100169.

Clemente-Suárez, V. J., Redondo-Flórez, L., Beltrán-Velasco, A. I., Ramos-Campo, D. J., Belinchón-deMiguel, P., Martínez-Guardado, I., Dalamitros, A. A., Yañéz-Sepúlveda, R., Martín-Rodríguez, A., & Tornero-Aguilera, J. F. (2023).

Mitochondria and Brain Disease: A Comprehensive Review of Pathological Mechanisms and Therapeutic Opportunities [Review of Mitochondria and Brain Disease: A Comprehensive Review of Pathological Mechanisms and Therapeutic Opportunities]. Biomedicines, 11(9), 2488. Multidisciplinary Digital Publishing Institute.

Franco-Obregón, A., Tai, Y. K., Wu, K. Y., Iversen, J. N., & Wong, C. J. K. (2023). The Developmental Implications of Muscle-Targeted Magnetic Mitohormesis: A Human Health and Longevity Perspective [Review of The Developmental Implications of Muscle-Targeted Magnetic Mitohormesis: A Human Health and Longevity Perspective]. Bioengineering, 10(8), 956. Multidisciplinary Digital Publishing Institute.

García-Fernández-Bravo, I., Torres-Do-Rego, A., López-Farré, A., Galeano-Valle, F., Demelo-Rodríguez, P., & Álvarez-Sala-Walther, L. A. (2022). Undertreatment or Overtreatment With Statins: Where Are We? [Review of Undertreatment or Overtreatment With Statins: Where Are We?]. Frontiers in Cardiovascular Medicine, 9. Frontiers Media.

Kuok, C. I., & Chan, W. K. Y. (2025). Rhabdomyolysis in Children: A State-of-the-Art Review [Review of Rhabdomyolysis in Children: A State-of-the-Art Review]. Children, 12(4), 492. Multidisciplinary Digital Publishing Institute.

Lee, G. X., & Duong, D. K. (2020). Rhabdomyolysis: evidence-based management in the emergency department. 22(12), 1.

Misrani, A., Tabassum, S., & Yang, L. (2021). Mitochondrial Dysfunction and Oxidative Stress in Alzheimer's Disease [Review of Mitochondrial Dysfunction and Oxidative Stress in Alzheimer's Disease]. Frontiers in Aging Neuroscience, 13. Frontiers Media.

Murphy, C. L., Deplazes, E., Cranfield, C. G., & Garcia, A. (2020). The Role of Structure and Biophysical Properties in the Pleiotropic Effects of Statins [Review of The Role of Structure and Biophysical Properties in the Pleiotropic Effects of Statins]. International Journal of Molecular Sciences, 21(22), 8745. Multidisciplinary Digital Publishing Institute.

Rashid, A. (2024). Untitled.

Safitri, N., Alaina, M. F., Pitaloka, D. A. E., & Abdulah, R. (2021). A Narrative Review of Statin-Induced Rhabdomyolysis: Molecular Mechanism, Risk Factors, and Management [Review of A Narrative Review of Statin-Induced Rhabdomyolysis: Molecular Mechanism, Risk Factors, and Management]. Drug Healthcare and Patient Safety, 211. Dove Medical Press.

San-Millán, I. (2023). The Key Role of Mitochondrial Function in Health and Disease [Review of The Key Role of Mitochondrial Function in Health and Disease]. Antioxidants, 12(4), 782. Multidisciplinary Digital Publishing Institute.

Soliemanabad, S. K., Rasouli, K., Zakariaei, Z., Soleymani, M., & Aliabadi, P. K. (2022). Rhabdomyolysis due to warfarin and atorvastatin combination therapy in a patient with ischemic heart disease: (A drug interaction). Annals of Medicine and Surgery, 75.

Trigo, D., Vitória, J., & Silva, O. A. B. da C. e. (2022). Novel therapeutic strategies targeting mitochondria as a gateway in neurodegeneration [Review of Novel therapeutic strategies targeting mitochondria as a gateway in neurodegeneration]. Neural Regeneration Research, 18(5), 991. Medknow.

Wilmanski, T., Kornilov, S. A., Diener, C., Conomos, M. P., Lovejoy, J. C., Sebastiani, P., Orwoll, E., Hood, L., Price, N. D., Rappaport, N.,

Magis, A. T., & Gibbons, S. M. (2022). Heterogeneity in statin responses explained by variation in the human gut microbiome. Med, 3(6), 388.

Wujak, M., Kozakiewicz, A., Ciarkowska, A., Loch, J. I., Barwiołek, M., Sokolowska, Z., Budny, M., & Wojtczak, A. (2021). Assessing the Interactions of Statins with Human Adenylate Kinase Isoenzyme 1: Fluorescence and Enzyme Kinetic Studies. International Journal of Molecular Sciences, 22(11), 5541.

Zhang, Q., Fan, X., Ye, R., Hu, Y., Zheng, T., Shi, R., Cheng, W., Lv, X., Chen, L., & Liang, P. (2020). The Effect of Simvastatin on Gut Microbiota and Lipid Metabolism in Hyperlipidemic Rats Induced by a High-Fat Diet. Frontiers in Pharmacology, 11.

Zhao, C., Hu, Y., Chen, H., Li, B., Cao, L., Xia, J., & Yin, Y. (2020). An in vitro evaluation of the effects of different statins on the structure and function of human gut bacterial community. PLoS ONE, 15(3).