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Cardiotrition® Booster Role in Post Myocardial Infarction (MI) Recovery

Post-ischemic myocardial recovery is primarily limited by cellular energy depletion, mitochondrial dysfunction, and oxidative stress—key pathophysiological mechanisms that are not sufficiently addressed by conventional hemodynamically active therapies. Although metabolic agents such as trimetazidine improve myocardial metabolic efficiency, their support of ATP production remains indirect, and they do not specifically target mitochondrial function or provide direct intracellular cytoprotecting against oxidative stress.

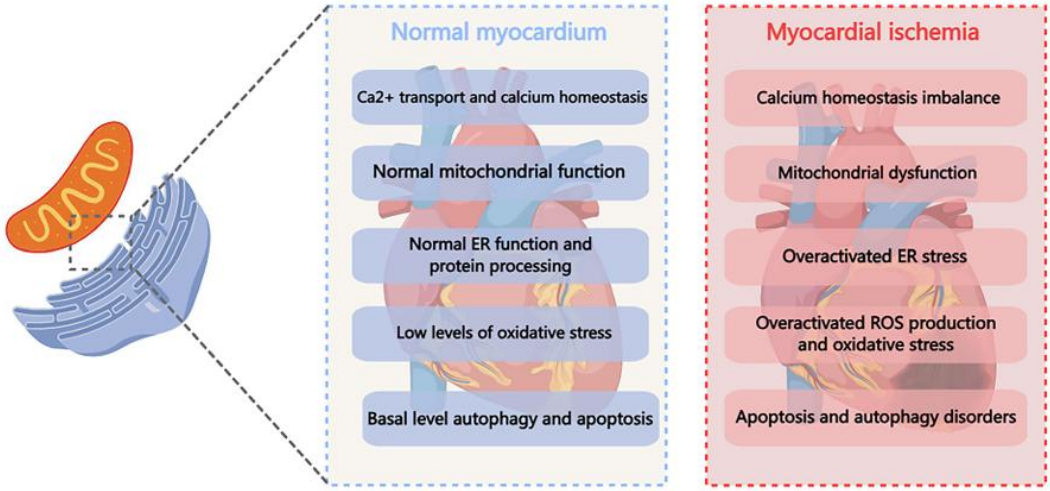
Cardiotrition® Booster is developed as an add on therapy for post-angina protocol specifically designed to support myocardial bioenergetics while protecting against oxidative stress. Unlike conventional formulations of CoQ10 and alpha-lipoic acid, which are limited by poor absorption and suboptimal myocardial delivery, Cardiotrition® Booster utilizes advanced liposomal encapsulation combined with CardioDrone® targeting technology

This delivery platform is engineered to preferentially transport active compounds to cardiomyocytes and mitochondria, particularly under conditions of ischemic stress.
Through this targeted intracellular approach, Cardiotrition® Booster promotes rapid ATP replenishment and reduces oxidative damage. Importantly, the formulation is hemodynamically neutral, making it suitable for patients with bradycardia or borderline blood pressure where escalation of traditional anti-anginal therapies may be limited.

As an adjunct to standard cardiovascular care, its use is associated with improved exercise tolerance, faster recovery following PCI and cardiac surgery, enhanced functional capacity, and additional protection in metabolically compromised myocardium, including diabetic heart disease. Overall, Cardiotrition® Booster serves as a mechanistically distinct, cytoprotective add-on therapy designed to work alongside standard post-angina and post-myocardial infarction treatments, directly supporting myocardial energy metabolism and cellular resilience.

ABSTRACT

Post-myocardial infarction recovery is limited by mitochondrial dysfunction, ATP depletion, and oxidative stress. Cardiotrition® Booster is a targeted metabolic adjunct designed to support myocardial bioenergetics and cytoprotecting using advanced liposomal delivery and CardioDrone® heart-targeting technology to enhance intracellular delivery of coenzyme Q10, alpha-lipoic acid, and acetyl-L-carnitine. In vitro evaluation in H9c2 cardiomyocytes demonstrated improved mitochondrial membrane potential and reduced mitochondrial superoxide production compared to untreated controls. These findings support Cardiotrition® Booster as an adjunct approach for enhancing mitochondrial function and cellular resilience following ischemic cardiac injury



Reduced ATP availability, oxidative imbalance, and early myocardial vulnerability are rooted in a combination of impaired oxygen supply and mitochondrial dysfunction. As the myocardium encounters ischemic stress—characterized by hypoxia, metabolic disruption, and altered redox signaling—mitochondrial ATP generation becomes less efficient, calcium homeostasis deteriorates, and reactive oxygen species begin to accumulate. This leads to impaired energy turnover, ionic instability, loss of cellular resilience, and reduced capacity for recovery.

At the core of these changes are three biological processes:

1. ATP Depletion and Bioenergetic Failure

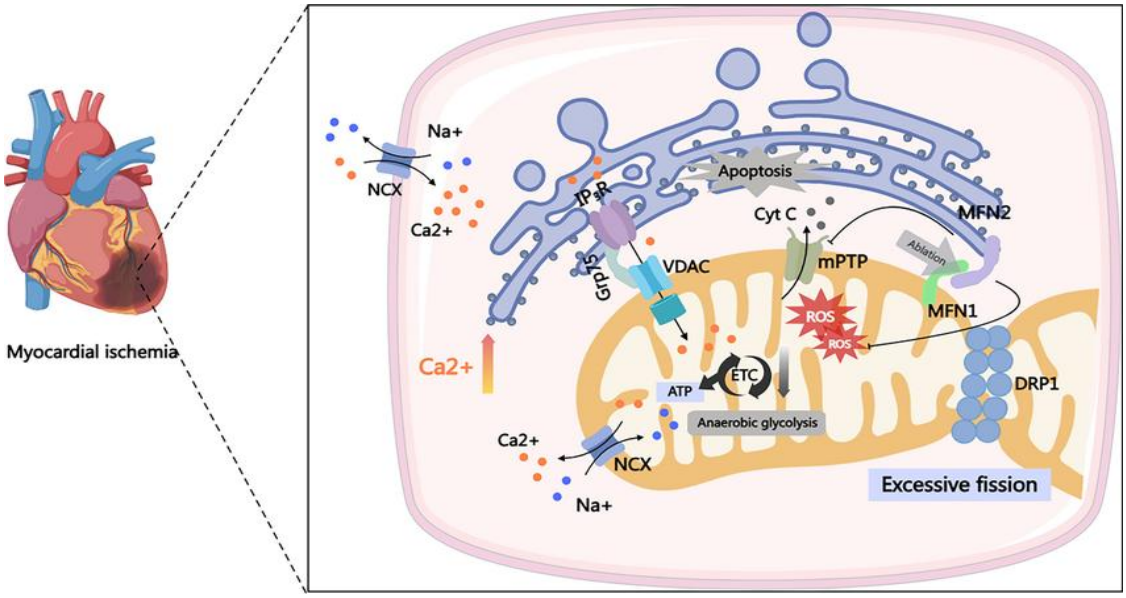
Myocardial ischemia causes a marked reduction in oxygen availability, leading to inhibition of the mitochondrial electron transport chain and suppression of oxidative phosphorylation. As a result, ATP production rapidly declines, forcing cardiomyocytes to rely on anaerobic glycolysis, an inefficient pathway associated with intracellular acidosis. ATP depletion impairs the activity of energy-dependent ion pumps, particularly SERCA and Na^+/K^+-ATPase, resulting in calcium accumulation and ionic imbalance. This early bioenergetic failure destabilizes mitochondrial function and sensitizes cardiomyocytes to injury.

2. Mitochondrial Dysfunction and Oxidative Stress Amplification

ATP depletion disrupts normal electron flow within the respiratory chain, promoting electron leakage from complexes I and III and increasing the generation of reactive oxygen species. Simultaneously, reduced ATP availability compromises endogenous antioxidant defenses, limiting the cell's ability to neutralize oxidative stress. Mitochondrial calcium overload further accelerates ROS production, creating a self-perpetuating cycle in which oxidative damage worsens mitochondrial dysfunction, leading to progressive ATP loss and declining cellular resilience

3. Reperfusion-Induced Oxidative Injury and Cell Death

During reperfusion, the abrupt reintroduction of oxygen to previously ischemic myocardium triggers a burst of ROS generation from structurally compromised mitochondria. This oxidative surge damages lipids, proteins, and DNA, while promoting the opening of the mitochondrial permeability transition pore. mPTP opening leads to dissipation of the mitochondrial membrane potential, irreversible ATP depletion, and activation of apoptotic and necrotic cell death pathways, ultimately expanding myocardial injury and limiting functional recovery



Technology Overview

Cardiotrition® is built on a multi-layered, patented delivery platform designed to overcome the limitations of conventional cardiac supplements. The formulation integrates advanced nano-carrier systems with organ-targeting ligands to ensure precise delivery, enhanced stability, and superior bioavailability.

At the core of this technology is CardioDrone®, a dual-ligand targeting system engineered to selectively guide active compounds toward cardiac cells while minimizing off-target distribution. This heart-specific targeting enhances therapeutic efficiency and reduces systemic exposure.

In parallel, Innova3® molecular technology transforms key ingredients such as Coenzyme Q10 and Alpha-Lipoic Acid into highly stable, bioavailable forms. Molecular modification improves resistance to heat, UV degradation, and gastrointestinal breakdown, ensuring ingredient integrity throughout digestion.

Encapsulation within LipoXTEND® modified polymeric liposomes further protects the actives, enabling sustained release and prolonged mitochondrial support. This integrated nano-delivery approach allows Cardiotrition® to achieve superior absorption, extended plasma presence, and consistent cellular uptake compared to conventional formulations

Mechanism of Action

CARDIOTRITION® Booster It works by enhancing myocardial bioenergetics to support efficient energy production in heart cells, reducing oxidative stress to help protect cardiac tissue from free radical damage, and optimizing oxygen delivery and utilization to improve overall cardiac efficiency. Through these combined actions, CARDIOTRITION® Booster also helps support the maintenance of healthy heart tissue and may aid in preventing permanent structural changes associated with cellular stress and energy deficiency.

Material and Methods

Cell Culture Conditions

H9c2 cardiomyocytes were cultured in Dulbecco's Modified Eagle's Medium (DMEM) supplemented with 10% fetal bovine serum, 100 U/mL penicillin, 100 µg/mL streptomycin, and 40 µg/mL gentamycin. Cells were maintained at 37 °C in a humidified atmosphere containing 5% CO_2 and were used for experiments upon reaching ~~appropriate confluency. Cell~~s were treated with the test formulation Cardiotrition® Booster, containing coenzyme Q10, alpha-lipoic acid, and acetyl-L-carnitine.

Mitochondrial Membrane Potential (JC-1 Assay)

Mitochondrial membrane potential ($\Delta\Psi m$) was evaluated using the fluorescent probe JC-1. Following 24 hours of treatment, H9c2 cells were incubated with 10 µM JC-1 for 10 minutes at 37 °C. The staining solution was then removed, and cells were washed twice with phosphate-buffered saline (PBS). Fluorescence signals were acquired using an inverted fluorescence microscope at 20× magnification. Quantitative analysis of fluorescence intensity was performed using ImageJ software

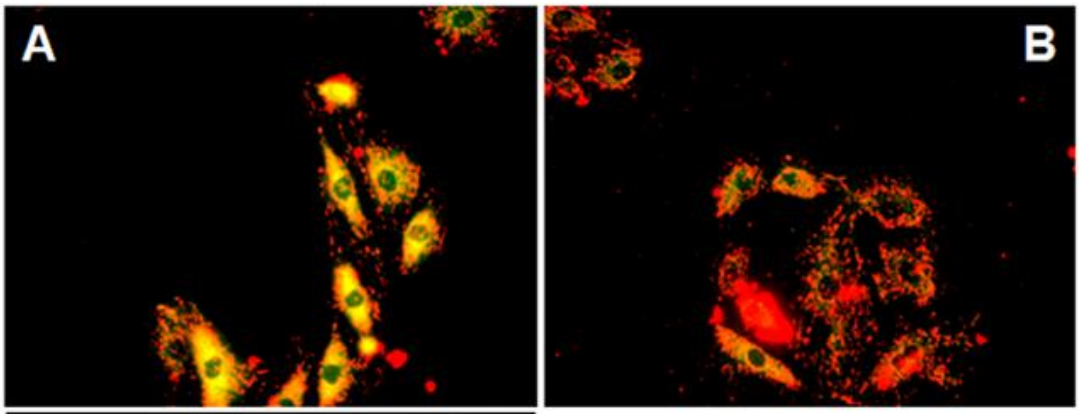
Induction of Oxidative Stress and MitoSOX™ Red Assay

Oxidative stress was induced by treating cells with 100 nM rotenone for 48 hours or 100 µM tert-butyl hydroperoxide (TBH) for 30 minutes. Following stress induction, mitochondrial superoxide production was evaluated using the mitochondria-targeted fluorescent probe MitoSOX™ Red. Cells were incubated with 5 µM MitoSOX™ Red for 20 minutes at 37 °C in the dark, washed with PBS, and fluorescence images were acquired using an inverted fluorescence microscope. Fluorescence intensity was quantified using ImageJ software and expressed as mean fluorescence intensity per cell.

Results

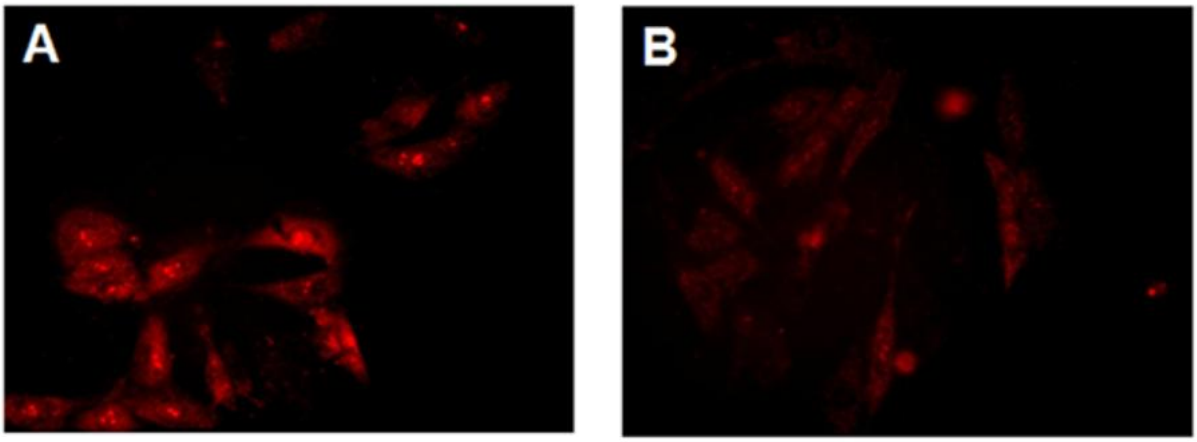
Mitochondrial Membrane Potential

Mitochondrial membrane potential was evaluated using JC-1 staining in H9c2 cells. As shown in Figure, control cells exhibited both red and green fluorescence, indicating a basal mitochondrial membrane potential(A). Cells treated with **Cardiotrition® Booster** for 24 hours showed a marked increase in red JC-1 fluorescence intensity and a significantly higher red/green fluorescence ratio compared with control cells (B), reflecting an increase in mitochondrial membrane potential.



Mitochondrial Superoxide Production

Physiological mitochondrial superoxide production was assessed using the MitoSOX™ Red fluorescent probe in H9c2 cells. As shown in Figure , control cells exhibited a detectable oxidized MitoSOX fluorescence signal (A). Cells treated with **Cardiotrition® Booster** for 24 hours displayed a visibly reduced MitoSOX fluorescence intensity compared with control cells



Conclusion

The recovery of the myocardium following an ischemic event remains a significant clinical challenge, primarily hampered by persistent mitochondrial dysfunction, bioenergetic failure, and unchecked oxidative stress. Conventional pharmacological interventions, while essential for managing hemodynamics, often fail to directly address these underlying cellular pathologies. The white paper herein presents Cardiotrition® Booster as a mechanistically distinct and evidence-based adjunct therapy designed to fill this critical gap.

By leveraging an integrated and patented delivery platform that combines CardioDrone® heart-targeting technology, Innova3® molecular transformation, and LipoXTEND® liposomal encapsulation, Cardiotrition® Booster is engineered to overcome the bioavailability and delivery limitations of conventional nutraceuticals. This sophisticated approach ensures the preferential intracellular transport of its key active compounds—coenzyme Q10, alpha-lipoic acid, and acetyl-L-carnitine—directly to the mitochondria of cardiomyocytes, particularly under conditions of ischemic stress.

The scientific rationale is robustly supported by the in vitro data presented. Treatment with Cardiotrition® Booster in H9c2 cardiomyocytes resulted in a significant increase in mitochondrial membrane potential ($\Delta\Psi m$), as indicated by a higher red/green fluorescence ratio in the JC-1 assay. This suggests an improvement in the energetic capacity and overall health of the mitochondria. Concurrently, the formulation demonstrated a clear cytoprotective effect by visibly reducing mitochondrial superoxide production, as measured by the MitoSOX™ Red assay. These findings confirm that Cardiotrition® Booster not only supports bioenergetic efficiency but also actively mitigates the primary drivers of oxidative damage, thereby breaking the self-perpetuating cycle of injury that limits post-ischemic recovery.

In conclusion, Cardiotrition® Booster represents a promising, mechanistically targeted nutritional intervention. Its ability to enhance ATP replenishment and reduce oxidative stress, coupled with its favorable hemodynamic neutrality, positions it as a valuable adjunct to standard post-myocardial infarction and post-angina care. By directly supporting myocardial bioenergetics and cellular resilience, Cardiotrition® Booster has the potential to improve functional outcomes, enhance exercise tolerance, and provide additional protection to the vulnerable, metabolically compromised myocardium. Future clinical studies are warranted to further validate these promising preclinical findings and to establish its definitive role in the management of ischemic heart disease.

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