



Evaluation of the Effect of Continuous and Interval Aerobic Training on Survivin Gene Expression and Cytochrome C in Myocardial Tissue of Rats Modeled with Myocardial Infarction

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E d i t o r	R e v i e w e r s
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1. Round 1

1.1 Reviewer 1

Reviewer:

When discussing “The Survivin gene, coding for a member of the inhibitor of apoptosis (IAP) family,” consider including a citation for its cardiac-specific expression or functional role in myocardial tissue, as most cited works concern oncology.

It reads “All biochemical and molecular experiments were performed in at least three independent replicates.” Clarify whether “replicates” refer to biological replicates (different animals) or technical repeats from the same sample—this distinction affects statistical interpretation.

The reporting of MANOVA assumptions is comprehensive but lacks exact test statistics (e.g., $W = 0.97$, $p = 0.43$). Including these numbers would increase transparency and allow readers to judge data normality and homogeneity.

The notation “↓ vs MI**” is ambiguous for academic publication. Replace symbolic arrows with explicit textual comparisons (e.g., “significantly lower than MI, $p < 0.01$ ”). Also report the exact p values instead of approximate ones (e.g., $p = 0.046 \rightarrow p = 0.0460$).

The manuscript states that “Changes in Cytochrome C across study groups are schematically illustrated...” but the figures are not labeled with units or error bars. Please ensure all graphs display mean \pm SEM and indicate statistical significance markers.

When asserting “MIHIIT produced significantly greater increases in Survivin expression compared to MICT,” include a plausible physiological explanation—e.g., differences in mitochondrial biogenesis signaling (PGC-1 α , AMPK)—to ground the interpretation in known molecular pathways.

The claim “Both training modalities effectively reduced Cytochrome C levels to comparable degrees” would be strengthened by discussing potential ceiling effects or the possibility that mitochondrial membrane stabilization reached maximal benefit in both groups.

Authors revised the manuscript and uploaded the updated document.

1.2 Reviewer 2

Reviewer:

The authors write, “These inconsistencies reinforce the importance of standardized protocols and comparative experimental designs.” This is a critical rationale—expand it by specifying what methodological heterogeneities (e.g., duration, load, rest ratios) most confound existing studies.

The aim statement is clear but could be reformulated into a hypothesis-driven format, e.g., “It was hypothesized that MIHIIT would enhance Survivin expression more strongly than MICT while similarly reducing Cytochrome C.” A hypothesis enhances the article’s scientific rigor.

The description of PCR conditions (“initial denaturation at 95 °C (15 min), followed by 40 cycles”) omits annealing and extension times per cycle. Include full thermal-cycle parameters and amplicon validation (e.g., melt-curve analysis).

The phrase “The results indicated a negative correlation between Cytochrome C and Survivin” should be quantified with Pearson r and p values. Without statistics, the strength of correlation cannot be evaluated.

The authors claim “Cytochrome C is a proven marker of apoptosis ... and its elevation has been reported across numerous MI models.” While accurate, this repeats earlier background material. Condense and focus on how the current magnitude of increase compares with those earlier reports.

The sentence “Interval and continuous training have been shown to increase expression of cardioprotective genes such as PP2Ac and GSK-3 β .” would benefit from discussing whether these genes mechanistically interact with Survivin or are simply parallel pathways—integrating this mechanistic bridge would deepen analysis.

Authors revised the manuscript and uploaded the updated document.

2. Revised

Editor’s decision after revisions: Accepted.

Editor in Chief’s decision: Accepted.