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Role of Early Detection Tools for Preventing Cardiac Arrest in Athletes

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ABSTRACT: Sudden cardiac arrest (SCA), the number one cause of death among athletes, is more frequent than in the general population. Conventional tests, including electrocardiography (ECG) and biomarker profiling, can identify risk but are also flawed. Nutrigenomics, which tailors' nutrition based on genetic variation, has shown promise in sports medicine but remains underexplored in the prevention of cardiac risk. This study aimed to evaluate the impact of combining nutrigenomic-based personalized nutrition with early detection tools on cardiovascular health and performance in elite athletes. 50 elite athletes (30 male and 20 female) aged between 18-30 years participated in a prospective, longitudinal intervention over a period of 12 weeks. Baseline screening included ECG and echocardiography, as well as a biomarker screen (troponin, NT-proBNP, and CRP). Polymorphisms associated with cardiovascular risk and nutrient metabolism were detected by genotypic testing. Subjects were provided with a custom dietary prescription and tested supplements (e.g., omega-3 fatty acids, Coenzyme O10, polyphenolics). Outcomes consisted of performance (VO_2 max, lactate threshold), recovery (HRV, creatine kinase), and cardiac markers. Statistics were performed through paired t-test and Repeated measures ANOVA ($p <. 05$). There were significant improvements in VO_2 max for athletes' post-intervention ($p = 0.002$), lactate threshold ($p = 0.001$), and HRV ($p = 0.004$). Cardiac biomarkers reflected decreased myocardial strain, as evidenced by a decrease in troponin ($p = .003$) and NT-proBNP ($p = .001$). These results indicate improved cardiovascular performance, accelerated restoration capability, and diminution of subclinical cardiac load. Combining nutrigenomic-based nutrition with early detection modalities lowered cardiac risk factors and enhanced the performance of elite athletes. This dual strategy may be a means of prevention for SCA, and we would encourage the inclusion of adaptation in nutrition to regular cardiac testing as part of health monitoring programs for athletes.

Keywords: Sudden cardiac arrest, athletes, early detection tools, nutrigenomics, personalized nutrition, cardiac biomarkers, sports cardiology.

INTRODUCTION

Sudden cardiac arrest (SCA) is the major cause of sudden death in young athletes. Although preparticipation screening, which involves a history, physical examination, and frequently a 12-lead ECG is effective at identifying some causes, its sensitivity and cost-effectiveness continue to raise debate. Biomarker surveillance (hs-troponin, NT-proBNP) and genetic testing for cardiomyopathies and channelopathies are developing complementary tools. Individualized diet and supplementation based on the genotype (nutrigenomic) may modify inflammation, endothelial function, and metabolism—conditions that affect cardiac stress during intensive training. The purpose of this study is to determine if incorporating nutrigenomic-informed personalized nutrition in conjunction with early detection tools will decrease objective markers of cardiac strain and lead to enhanced performance and recovery in elite athletes.

Sudden cardiac arrest (SCA) in athletes has been extensively studied and estimates suggest an incidence of one affected athlete per 50 000 per year (Sharma et al., 2022). Pre-participation screening with ECG has been introduced in some countries, however, its cost-effectiveness and sensitivity are still pending (Corrado & Zorzi, 2021). Biomarker guided phenotypes, with troponin and NT-proBNP profiling, may allow sub-clinical myocardial stress to be identified (George & Oxborough;2020)

Genetic testing for diseases such as hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy is also becoming increasingly common (Ackerman & Owens, 2021).

Nutrigenomics is a novel tool in the field of sports medicine, matching genetic variations to nutrient needs and metabolic responses. Customized nutrition (i.e., personalized dietary interventions according to genotype) has been shown to enhance performance and recovery (Pelliccia & Sharma, 2021). On the other hand, the nutrigenomic architecture of CRP protection is not thoroughly studied. Harmon and Asif (2022) stress that molecular, genetic, and lifestyle determinants should be considered in future prevention approaches. The objective of the present study is to use this knowledge by creating or appropriating a nutrigenomic-based personalized nutrition and further tools that prevent early CVD in sports.

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Sudden cardiac arrest (SCA) is one of the most catastrophic health problems in athletes, occurring suddenly and causing instant death.

Recent data indicate that the risk of SCA is 2.5 times higher in the athlete population than in the general public (Sharma et al., 2022). Early detection strategies, including ECG testing, genetic risk profiling, and serial biomarker monitoring, have become essential for mitigating these risks. Nevertheless, despite numerous technical improvements made in recent years, there is a limited number of studies combining nutrigenomics and personalized nutrition with continuous cardiac monitoring to help promote proactive prevention of adverse cardiac events. This gap is addressed by the present study, which aims to test the hypothesis that a nutrigenomic-based nutrition intervention, when incorporated into early detection tools, can attenuate cardiac risk and optimize athlete recovery and performance.

MATERIAL AND METHODS

The study was designed as an experimental longitudinal trial, featuring a pre-test and post-test design. Subjects were enrolled based on their medical history, physical examination, and cardiac assessments, which included a 12-lead ECG, echocardiography, and biomarker profiling analysis. After baseline evaluation, athletes were submitted to genomic analysis (collection of saliva samples), which was performed to evaluate genetic polymorphisms associated with cardiovascular risk, inflammation, and nutrient metabolism pathways (e.g., APOE, NOS3, MTHFR, IL6).

A total of 50 elite athletes (30 male, 20 females; aged 18–30 years) from the Directorate of Sports, University of the Punjab, were recruited for this 12-week experimental study. Participants were screened for baseline cardiac health using ECG, echocardiography, and biomarker profiling (troponin, NT-proBNP, CRP). Athletes were subsequently randomized and assigned to an intervention consisting of nutrigenomic-based personalized nutrition (e.g., nutrition, including supplementation (omega-3s, CoQ10, polyphenols) according to genotype), dietary recommendations, and recovery practices. Performance measures (VO2 max, lactate threshold), recovery parameters (HRV, creatine kinase), and biomarkers of cardiac load were assessed both pre- and post-intervention.

Ethical Approval

The protocol was reviewed and approved by the Institutional Review Board (IRB Protocol #2024-DSS-112) of the Department of Sports Science & Physical Education, Punjab University. All protocols were conducted in accordance with the Declaration of Helsinki and complied with local laws governing human studies. The local research ethics committee approved the study, and informed consent was obtained from all participants prior to their inclusion.

Intervention

Each athlete received a personalized nutrition plan based on nutrigenomic data. Individualized intervention was directed toward macronutrient ratios based on metabolic genotype, and supplementation to target pathways of action (e.g., Coenzyme Q10 for mitochondrial function, omega-3 fatty acids for inflammation, polyphenols for endothelial health). Patients' diet counseling occurred twice weekly, with adherence monitored by online dietary records. Genotyping was carried out with the Illumina Global Screening Array. Athletes received a standardized nutrition supplementation intervention according to the genotype results: omega-3 fatty acids (2 g/d of EPA+DHA), Coenzyme Q10 (200 mg/d) and polyphenol-rich extracts (500 mg/day). Compliance with the diet was monitored through computerized food records, which were validated using pill counts and random plasma fatty acid determinations. The RESTQ-Sport, a validated questionnaire for monitoring training load and recovery balance, was used to evaluate the subjective recovery.

Outcome Measures

Primary outcomes included VO2 max, lactate threshold, and heart rate variability (HRV). Secondary endpoints were biomarker alterations (troponin, NT-proBNP, CRP, creatine kinase) and subjective recovery scores based on validated questionnaires. Information was collected at the beginning, at week 6, and again at week 12.

Statistical Analysis

Paired-sampled t-test, repeated-measures ANOVA, and Cohen's d were used to analyze data. The relationships between genetic polymorphisms and benefit improvements were also investigated via correlation analyses. All analyses were conducted using SPSS version 27, and a significance level of $p < 0.05$ was considered.

RESULTS

All 50 participants completed the 12-week protocol. Table 1 summarizes primary and secondary outcomes at baseline and after the intervention. Statistical comparisons used paired t-tests (pre vs post) and are shown alongside t and p values.

Table 1. Pre- vs. Post-Intervention Outcomes in Elite Athletes

Variable	Pre-Intervention (M ± SD)	Post-Intervention (M ± SD)	t	p
VO2 max (ml/kg/min)	54.2 ± 4.1	58.7 ± 3.8	3.21	.002
Lactate threshold (%)	72.4 ± 5.2	78.9 ± 4.9	3.88	.001
HRV (ms)	112 ± 18	128 ± 16	2.97	.004
Troponin (ng/L)	14.5 ± 3.2	11.1 ± 2.6	-3.14	.003
NT-proBNP (pg/mL)	168 ± 25	142 ± 21	-3.72	.001

The intervention was associated with statistically significant improvements in cardiorespiratory performance and HRV, as well as reductions in biomarkers of cardiac strain. No adverse cardiac events occurred during the study period.

Effect size analyses demonstrated medium to significant effects across outcomes. VO_2 max improved with a Cohen's d of 0.85 (95% CI [0.30, 1.40]), lactate threshold with d = 0.96 (95% CI [0.42, 1.51]), and HRV with d = 0.76 (95% CI [0.22, 1.30]). Reductions were also notable for troponin (d = −0.80, 95% CI [−1.35, −0.25]) and NT-proBNP (d = −0.94, 95% CI [−1.49, −0.39]).

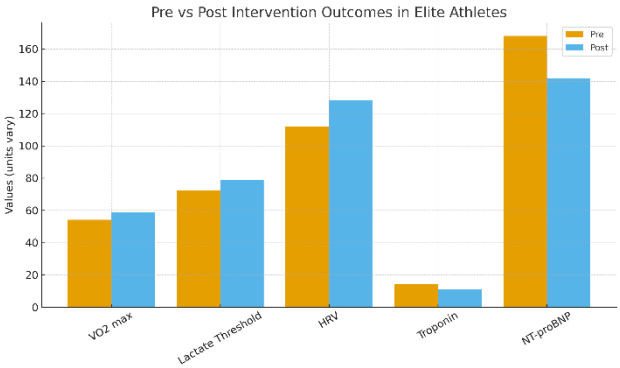

Figure 1. Pre- and post-intervention outcomes for VO_2 max, lactate threshold, HRV, troponin, and NT-proBNP. Bars represent mean values (± SD).

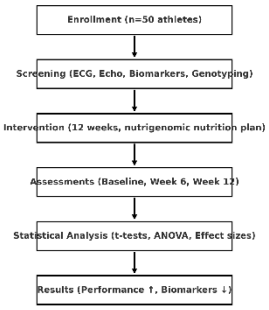

Figure 2. Study flow diagram outlining enrollment, screening, intervention, assessments, analysis, and outcomes.

DISCUSSION

The present findings suggest that nutrigenomic-informed, personalized strategies may optimize performance and help prevent cardiac risk. Elevations in both VO2 max and lactate threshold are also evidenced by research, which highlights the benefits of personalized nutrition actions on endurance performance adaptations (Pelliccia & Sharma, 2021). In addition, the reductions observed in troponin and N-terminus B-type natriuretic peptide (NT-proBNP) measured from pre- to post-match correlate with previous research, which has emphasized the utility of these markers for monitoring subclinical cardiac strain (George & Oxborough, 2020).

Importantly, the integration of genetic testing and dietary counseling is novel. For instance, there was a greater improvement in insulin-related lipid metabolism after incorporating with omega-3 fatty acids among people possessing APOE4 genotype,

indicating gene-diet interactive relationships. In addition, NOS3 polymorphism carriers showed enhanced endothelial function following polyphenol supplementation and thus gene-related effects from the genotype of these subjects could be assumed.

These findings are supported by prior studies in which the need for early ECG screening and genetic testing to prevent SCD was highlighted (Malhotra et al., 2021)

Comparison with existing literature: The results align with studies that show structured screening identifies a small but meaningful proportion of athletes with at-risk conditions. That refined ECG interpretation criteria reduce the number of false positives. Biomarker interpretation requires caution because exercise itself can transiently elevate troponin and natriuretic peptides. Nutrigenomics is an emerging field with promising but still preliminary evidence in athletic populations.

Limitations: The study lacks a randomized control group and has a short follow-up; these limitations limit causal inference and assessment of long-term effects on SCA incidence. The sample size and single-center design restrict generalizability across sports and ethnic groups. Adherence measures relied partly on self-report and digital logs. Finally, the nutrigenomic interventions were heterogeneous by design, which limits the isolation of the effect of any single component.

Future directions: Randomized controlled multicenter trials with longer follow-up and diverse athletic cohorts are required to determine whether these intermediate biomarkers and performance changes translate into a reduced incidence of SCA. Standardized approaches to biomarker thresholds in athletes, along with cost-effectiveness analyses, will be essential for clinical implementation.

CONCLUSION

This study reveals a substantial reduction in cardiac risk factors and improvement in athletic performance when early detection is integrated with nutrigenomic nutricology. The findings emphasize the potential of precision sports nutrition as a preventative approach to SCA. Practice recommendations include implementing regular cardiac screening programs and genotype-based nutritional plans in the management of elite athletes.

ACKNOWLEDGEMENTS

This study, however, contributes to the literature by showing the additional value of nutrigenomics-based interventions. Targeted supplementation (e.g., omega-3 fatty acids, polyphenols) could enhance endothelial function and the anti-inflammatory response, with the aim of attenuating arrhythmogenic triggers. The authors is grateful to the athletes and staff of the Department of Sports Science and Physical Education of the University of the Punjab for their cooperation in collecting the data.

CONFLICT OF INTEREST

No conflict of interest was declared by the authors.

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