Characterizing Biomarkers of Muscle Damage in Collegiate Football Players: Implications for Recovery and Athlete Health

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Background:
Intense physical activity, particularly during preseason training, places significant physiological stress on athletes, leading to muscle damage. This damage is often reflected in elevated levels of biochemical markers such as creatine kinase (CK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), blood urea nitrogen (BUN), and creatinine. Collegiate football players are especially susceptible to these changes due to the high intensity of their training regimens. Understanding the typical trends in these biomarkers is critical for distinguishing between normal recovery processes and conditions like exertional rhabdomyolysis, which may lead to serious systemic complications if untreated. Athletes with sickle cell trait (SCT) represent a subset of individuals with potentially heightened vulnerability to muscle stress and delayed recovery, necessitating a closer examination of their unique physiological responses.

Objective:
The primary objective of this study was to establish baseline and post-exercise reference ranges for key biomarkers of muscle damage in collegiate football players and to characterize their trends immediately post-exercise (IP) and 24 hours post-exercise (24h P). A secondary objective was to evaluate differences in biomarker responses between athletes with SCT and their non-SCT counterparts, providing insights into tailored recovery strategies.

Methods:
Biomarker levels were measured at three time points: baseline (BL), IP, and 24h P. Statistical analyses included paired t-tests to assess within-group changes over time and independent t-tests to compare between SCT and non-SCT groups. Correlations between biomarkers, such as CK and creatinine, were also evaluated to explore the relationship between muscle damage and kidney stress

Results:
Significant elevations in CK, LDH, AST, ALT, and creatinine were observed at IP, reflecting acute muscle damage and metabolic stress following exercise. By 24h P, most biomarkers showed partial recovery; however, levels of CK, LDH, AST, and ALT remained elevated, particularly in SCT athletes, suggesting prolonged physiological stress or delayed recovery. SCT athletes exhibited significantly higher peak and sustained biomarker levels than non-SCT athletes, with some exceeding clinically relevant thresholds. For example, CK levels were positively correlated with creatinine ($R^2 = 0.19$, $p < 0.001$), indicating kidney stress linked to muscle damage. Individual variability in biomarker responses was significant, emphasizing the need for personalized monitoring approaches.

Conclusion:
This study provides comprehensive reference data for muscle damage biomarkers in collegiate football players and underscores the importance of considering SCT status when designing training and recovery protocols. The findings highlight the prolonged recovery timelines in SCT athletes, suggesting that these individuals may benefit from modified exercise regimens and enhanced post-exercise monitoring. These insights can guide clinical and coaching practices aimed at optimizing performance and minimizing health risks in high-performance athletes.