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

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## Introduction

Intense physical exertion, such as that experienced during collegiate football training, often leads to skeletal muscle damage and systemic physiological stress. This is reflected by elevations in blood and urine biomarkers associated with muscle, liver, and kidney function. While these elevations are typically part of the normal adaptation to training, persistent or extreme deviations may indicate overtraining or risk for conditions such as rhabdomyolysis.

Monitoring biomarkers like creatine phosphokinase (CPK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), blood urea nitrogen (BUN), and creatinine provides valuable insight into recovery and overall athlete health. However, reference values derived from clinical settings may not fully capture the physiological norms of elite athletes.

This study aimed to characterize acute changes in biomarkers following a preseason scrimmage among collegiate football players. By analyzing levels at baseline, immediately post-exercise (IPE), and 24 hours post-exercise (24hP), we sought to better understand exercise-induced muscle, hepatic, and renal stress and the timeline of biomarker recovery. These findings can help inform evidence-based recovery protocols and support athlete health and performance.

## Objective and Significance

To characterize acute changes in biomarkers of muscle damage, renal function, hepatic stress, and metabolic balance in collegiate football players following intense exercise, and to evaluate biomarker recovery over a 24-hour period.

Understanding how key physiological biomarkers respond to and recover from high-intensity activity can help identify athletes at risk of prolonged stress or incomplete recovery. This data provides critical insight for designing individualized training and recovery protocols to optimize performance, reduce injury risk, and improve athlete health monitoring in contact sports.

## Methods

Collegiate American football players (n = 20) participated during the preseason training period. All participants provided written informed consent, and the study was approved by the university's IRB.
Data were collected across three time points: baseline (BL), immediately post-exercise (IPE), and 24 hours post-exercise (24hP). Each session followed a standardized, high-intensity football scrimmage designed to simulate competitive game conditions. Venous blood and urine samples were collected at each time point. The biomarker panel included indices of muscle damage (CPK, LDH, myoglobin, TNNI1), renal stress (creatinine, BUN), hepatic function (AST, ALT, bilirubin), and metabolic shifts (phosphorus, uric acid). Linear mixed models were used to assess changes over time and associations between biomarkers. Pearson correlation and regression analyses were used to assess relationships between CPK and ancillary biomarkers. Significance was set at $p < 0.05$.

## Results

| | Time Point | | | | | |
|---|---|---|---|---|---|---|
| | BL | | 24h P | | IP | |
| | Mean | Std Dev | Mean | Std Dev | Mean. | Std Dev |
| Blood Urea Nitrogen (mg/dL) | 17 | 2 | 18.25 | 3.24 | 19.31 | 2.15 |
| Creatinine (mg/dL) | 1.2 | 0.21 | 1.25 | 0.20 | 1.54 | 0.26 |
| Uric Acid (mg/dL) | 5.61 | 1.40 | 6.02 | 1.45 | 6.41 | 1.58 |
| Phosphorus (mg/dL) | 4.61 | 0.44 | 3.59 | 0.50 | 3.76 | 0.53 |
| Total Bilirubin (mg/dL) | 0.72 | 0.16 | 0.91 | 0.30 | 0.99 | 0.26 |
| CPK (IU/L) | 267.67 | 205.30 | 726.53 | 430.56 | 968.25 | 549.76 |
| LDH (IU/L) | 143 | 30.77 | 182.56 | 23.32 | 229.5 | 29.16 |
| AST (IU/L) | 22.89 | 4.23 | 33.38 | 6.32 | 40.56 | 9.16 |
| ALT (IU/L) | 21.78 | 3.38 | 28.13 | 5.61 | 32.5 | 6.86 |
| UR Creatinine (mg/dl) | 270.24 | 158.85 | 266.75 | 122.04 | 416.69 | 196.41 |
| Serum Myoglobin (ng/mL) | N/A | N/A | 0.30 | 0.39 | 1.54 | 1.68 |
| Urine Myoglobin (ng/mL) | N/A | N/A | 0.35 | 0.58 | 14.27 | 32.70 |
| Serum Troponin (ng/mL) | 0.65 | 0.79 | 0.39 | 0.54 | 0.84 | 0.81 |

**Table 1.** Descriptive statistics for biomarkers of muscle damage, renal function, liver stress, and metabolic shifts at baseline (BL), immediately post-exercise (IP), and 24 hours post-exercise (24hP). Values are presented as mean ± standard deviation. Notable trends include large increases in CPK, LDH, AST, and ALT immediately post-exercise, with partial recovery by 24hP. Creatinine, BUN, and uric acid showed modest post-exercise elevations, while phosphorus declined. Serum troponin levels also rose above baseline at IP, indicating skeletal muscle strain. N/A indicates data not collected at that time point.

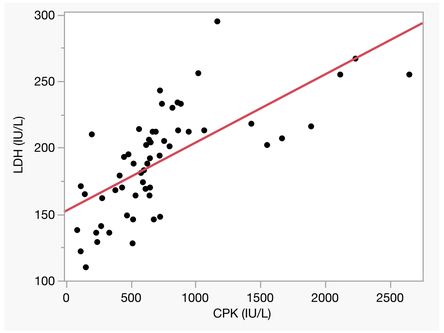


**Figure 1.** Linear regression analysis of lactate dehydrogenase (LDH) and creatine phosphokinase (CPK) levels shows a significant positive correlation ($R^2 = 0.35$, $p < 0.0001$), suggesting that LDH is a moderately reliable marker of muscle damage following intense exercise. The fitted regression equation is LDH (IU/L) = 145.56 + 0.0518 × CPK (IU/L).

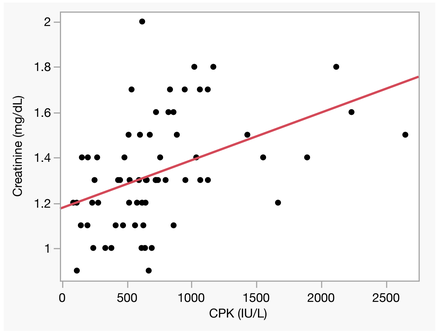


**Figure 2.** Linear regression analysis of creatinine (mg/dL) and creatine phosphokinase (CPK) (IU/L) reveals a statistically significant positive correlation ($R^2 = 0.19$, $p = 0.0002$), suggesting that elevated muscle damage is associated with transient renal stress. The regression equation is Creatinine = 1.18 + 0.00021 × CPK.

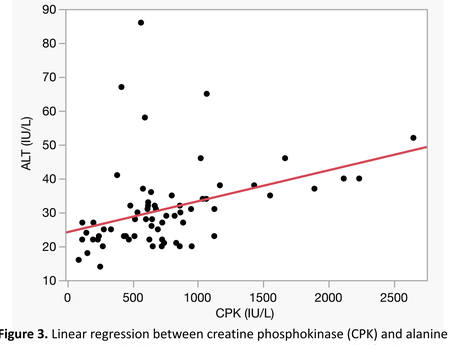


**Figure 3.** Linear regression between creatine phosphokinase (CPK) and alanine aminotransferase (ALT). CPK was positively associated with ALT ($R^2 = 0.14$, $p = 0.0020$), indicating a moderate relationship between muscle damage and hepatic stress. This suggests that ALT may partially reflect exercise-induced muscle stress, although with less predictive strength than AST or LDH.

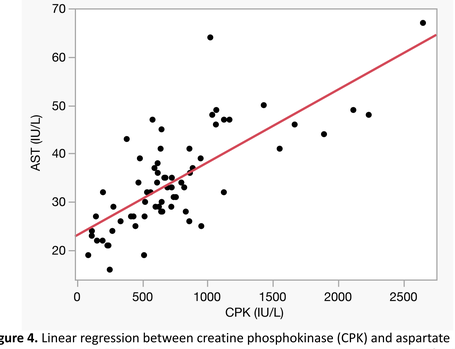


**Figure 4.** Linear regression between creatine phosphokinase (CPK) and aspartate aminotransferase (AST). CPK showed a strong positive association with AST ($R^2 = 0.56$, $p < 0.0001$), suggesting that AST is a reliable indicator of muscle damage following intense exercise. The strength of this relationship highlights AST's utility in monitoring muscle stress and recovery dynamics in athletes.

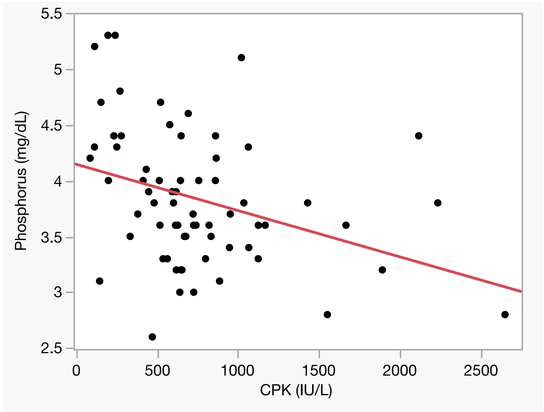


**Figure 5.** Linear regression between creatine phosphokinase (CPK) and phosphorus levels. A statistically significant inverse relationship was observed ($R^2 = 0.12$, $p = 0.0041$), indicating that as CPK increases, phosphorus decreases. This finding suggests a potential metabolic shift involving phosphate utilization during periods of high muscle stress and may reflect increased ATP turnover in response to intense exercise

## Discussion and Limitations

- Biomarker Patterns: Most biomarkers peaked immediately post-exercise (IPE) and declined by 24h post (24hP), yet many remained elevated—indicating ongoing physiological stress and incomplete recovery in some athletes.
- Key Correlations:
  - CPK strongly correlated with LDH ($R^2 = 0.35$) and AST ($R^2 = 0.56$), reinforcing their value as muscle damage indicators.
  - CPK also correlated with creatinine ($R^2 = 0.18$), indicating transient renal stress.
  - A novel finding was the inverse relationship between CPK and phosphorus ($R^2 = 0.10$, $p = 0.0165$), suggesting metabolic shifts during muscle stress.
- Implications: Persistent elevations in muscle, liver, and kidney biomarkers—despite partial recovery at 24h—emphasize the importance of individualized monitoring to optimize recovery, performance, and injury prevention.
- Limitations:
  - No follow-up beyond 24hP to assess longer-term recovery.
  - Moderate sample size; further research needed with larger cohorts and extended recovery windows.
- Takeaway: Monitoring biomarker trends (rather than single time points) may offer better insight into athlete readiness and inform evidence-based training protocols.

## Conclusion

Intense physical activity in collegiate football athletes led to significant, measurable changes in biomarkers of muscle damage, renal stress, and hepatic load. While most markers showed partial recovery by 24 hours post-exercise, several remained elevated, highlighting individual variability in physiological stress responses and recovery rates. Strong correlations between CPK and markers like LDH, AST, and creatinine underscore the interconnected effects of muscle breakdown on other organ systems. These findings support the value of routine biomarker monitoring to guide recovery strategies, reduce injury risk, and enhance athlete health and performance.