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Metabolomic biomarker candidates for Skeletal Muscle loss in the Collagen-Induced Arthritis (CIA) model

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Statement of the Problem: There is no consensus for diagnosis or treatment of RA muscle loss. We aimed to investigate metabolites in arthritic mice urine as biomarkers of muscle loss.

Methodology & Theoretical Orientation: DBA1/J mice comprised collagen-induced arthritis (CIA) and control (CO) groups. Urine samples were collected at 0, 18, 35, 45, 55, and 65 days of disease and subjected to nuclear magnetic resonance spectroscopy. Metabolites were identified using Chenomx and Birmingham Metabolite libraries. The statistical model used principal component analysis, partial least-squares discriminant analysis, and partial least-squares regression analysis. Linear regression and Fisher's exact test via the Metabo Analyst website were performed (VIP-score).

Findings: Nearly 100 identified metabolites had CIA vs. CO and disease time-dependent differences ($p<0.05$). Twenty-eight metabolites were muscle-associated: carnosine (VIPs 2.8×102) and succinyl acetone (VIPs 1.0×10) showed high importance in CIA vs. CO models at day 65; CIA pair analysis showed histidine (VIPs 1.2×102) days 55 vs. 65, histamine (VIPs 1.1×102) days 55 vs. 65, and L-methionine (VIPs 1.1×102) days 0 vs. 18. Carnosine was fatigue- (0.039) related, creatine was food intake- (-0.177) and body weight- (-0.039) related, and both metabolites were clinical score- (0.093; 0.050) and paw edema- (0.125; 0.026) related.

Conclusion & Significance: Therefore, muscle metabolic alterations were detected in arthritic mice urine, enabling further validation in RA patient's urine, targeting prognosis, diagnosis, and monitoring of RA-mediated muscle loss.

Biography

Paulo Vinicius Gil Alabarse has his expertise in Rheumatoid Arthritis and related muscle loss, as well as osteoarthritis. His research focus on searching for novel metabolic biomarker of muscle loss targeting diagnosis, follow up, and treatment response in order to improve individual disease progress and treatment response.