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Elevated Systemic Oxidative-Nitrosative Stress and Cerebrovascular Function in Professional Rugby Union Players: The Link to Impaired Cognition

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Abstract : Introduction and aims: Sports-related concussion (SRC) represents a significant and growing public health concern in rugby union, yet remains one of the least understood injuries facing the health community today. Alongside increasing SRC incidence rates, there is concern that prior recurrent concussion may contribute to long-term neurologic sequelae in later-life. This may be due to an accelerated decline in cerebral perfusion, a major risk factor for neurocognitive decline and neurodegeneration, though the underlying mechanisms remain to be established. The present study hypothesised that recurrent concussion in current professional rugby union players would result in elevated systemic oxidative-nitrosative stress, reflected by a free radical-mediated reduction in nitric oxide (NO) bioavailability and impaired cerebrovascular and cognitive function. Methodology: A longitudinal study design was adopted across the 2017-2018 rugby union season. Ethical approval was obtained from the University of South Wales Ethics Committee. Data collection is ongoing, and therefore the current report documents result from the pre-season and first half of the in-season data collection. Participants were initially divided into two subgroups; 23 professional rugby union players (aged 26 ± 5 years) and 22 non-concussed controls (27 ± 8 years). Pre-season measurements were performed for cerebrovascular function (Doppler ultrasound of middle cerebral artery velocity (MCAv) in response to hypocapnia/normocapnia/hypercapnia), cephalic venous concentrations of the ascorbate radical (A•-, electron paramagnetic resonance spectroscopy), NO (ozone-based chemiluminescence) and cognition (neuropsychometric tests). Notational analysis was performed to assess contact in the rugby group throughout each competitive game. Results: 1001 tackles and 62 injuries, including three concussions were observed across the first half of the season. However, no associations were apparent between number of tackles and any injury type ($P > 0.05$). The rugby group expressed greater oxidative stress as indicated by increased A•- ($P < 0.05$ vs. control) and a subsequent decrease in NO bioavailability ($P < 0.05$ vs. control). The rugby group performed worse in the Ray Auditory Verbal Learning Test B (RAVLT-B, learning, and memory) and the Grooved Pegboard test using both the dominant and non-dominant hands (visuomotor coordination, $P < 0.05$ vs. control). There were no between-group differences in cerebral perfusion at baseline (MCAv: 54 ± 13 vs. 59 ± 12, $P > 0.05$). Likewise, no between-group differences in CVRCO2Hypo (2.58 ± 1.01 vs. 2.58 ± 0.75, $P > 0.05$) or CVRCO2Hyper (2.69 ± 1.07 vs. 3.35 ± 1.28, $P > 0.05$) were observed. Conclusion: The present study identified that the rugby union players are characterized by impaired cognitive function subsequent to elevated systemic-oxidative-nitrosative stress. However, this appears to be independent of any functional impairment in cerebrovascular function. Given the potential long-term trajectory towards accelerated cognitive decline in populations exposed to SRC, prophylaxis to increase NO bioavailability warrants consideration.

Keywords : cognition, concussion, mild traumatic brain injury, rugby
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