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Testosterone effects in the mesenteric vascular bed: ROS production, COX activation and leukocyte migration

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Aim: The mechanisms whereby testosterone acts in the cardiovascular system are yet to be completely described. However, oxidative stress and inflammation seem to be key players in the effects of testosterone. We sought to determine whether testosterone induces leukocyte migration and the mechanisms involved in such effect. We hypothesized that testosterone induces leukocyte migration via NADPH oxidase-driven reactive oxygen species production and cyclooxygenase-dependent mechanisms.

Method and Result: Sixteen weeks-old Wistar rats were firstly treated intraperitoneally with single 5mL injections of either saline, sodium salicylate (1.25x10-3mol/L), flutamide (androgen receptor antagonist, 10-6 mol/L), apocynin (NADPH oxidase inhibitor, 3x10-4mol/L) or NS398 (COX2 inhibitor, 10-3 mol/L) and then with testosterone (10-7 mol/L). Leukocyte migration was assessed 24 hours after testosterone administration by intravital microscopy. Serum levels of testosterone were measured by ELISA. NADPH oxidase activity and subunits expression were assessed by membrane fraction-dihydroethidium fluorescence and immunoblotting, respectively. Testosterone administration did not change the serum levels of endogenous testosterone and increased venular leukocyte migration to the adventia, NADPH oxidase activity and the expression of NADPH oxidase subunits in mesenteric vessels. These effects were blocked by flutamide. Sodium salicylate partially inhibited testosterone-induced leukocyte migration. Apocynin and NS398 also inhibited testosterone-induced leukocyte migration and NADPH oxidase activity.

Conclusion: Testosterone induces leukocyte migration via NADPH oxidase-dependent and COX2-related mechanisms and may contribute to inflammatory processes and oxidative stress associated with cardiovascular diseases.

Biography

Andreia Z Chignalia has completed her Ph.D. at the age of 28 years from University of Sao Paulo, Brazil. She did part of her Ph.D. training at University of Ottawa, Canada. Her post-doctoral training started at the University of Sao Paulo and she is currently a visitor postdoctoral Research Associate at the University of Illinois at Chicago. Her studies are mainly focused on redox-sensitive vascular effects testosterone. Besides significant papers in the redox-field, Chignalia is also a co-author of a book chapter in the area.

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