N-3 FATTY ACIDS DO NOT IMPROVE ENDOTHELIUM-DEPENDENT VASODILATION IN RAT THORACIC AORTA WHEN ASSOCIATED TO ACUTE EXERCISE

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Introduction: n-3 polyunsaturated fatty acids (PUFA) are known to have beneficial effects in coronary artery disease. Whether PUFA are beneficial on endothelial function is controversial. The purpose of this study was to determine whether PUFA result in an additional effect to acute exercise on endothelial function in rats.

Methods: The study was carried out with 2 groups of Sprague Dawley male rats subjected for 4 weeks to a diet based on PUFA (n=20) or standard diet (n=20). In each group 10 rats acted as sedentary controls. The 10 others ran on a treadmill (15° incline). The running speed was progressively increased until animal was exhausted. Animal was then sacrificed and endothelial function was assessed by measuring isometric tension in rings of thoracic aorta.

Acetylcholine (ACh, 10⁻⁶ mol/L and 10⁻⁵ mol/L) induced vasorelaxation was examined in aortic rings precontracted with phenylephrine (PE, 10⁻⁷ mol/L). Response to ACh was also studied in presence of 5.10⁻⁵ mol/L of N⁹⁶⁹;-nitro-L-arginine methyl ester (L-NAME). Finally, vascular responses to nifedipine (10⁻⁸ mol/L and 10⁻⁵ mol/L), a L-type Ca²⁺ channel antagonist, were examined.

Results: The present study showed that response to ACh (10⁻⁶ mol/L) was significantly improved after acute exercise in standard diet group (89.6 ± 7.25% vs 123.9 ± 9.87%, p=0.01) and in PUFA diet group (86.9 ± 11.65% vs 124.1 ± 5.65%, p=0.02). The same effect was found in response to ACh (10⁻⁵ mol/L), in standard diet group (92.9 ± 6.77% vs 128.6 ± 9.2%, p=0.01) and in PUFA diet group (96.9 ± 10.23% vs 125.6 ± 5.75%, p=0.01). When PUFA supplementation was compared to standard diet no significant change was found in response to both concentrations of ACh, either at rest or after an acute exercise. Pretreatment of rings with L-NAME inhibited the ACh-mediated vasorelaxation in all groups. Response to nifedipine (10⁻⁸ mol/L) in PE precontracted rings was significantly improved after acute exercise only in PUFA supplemented rats (-3.6 ± 2.02% vs 5.11 ± 2.79%, p=0.01) but not in standard diet ones. Response to nifedipine (10⁻⁵ mol/L) was enhanced similarly after acute exercise in both standard (27.82 ± 5.56% vs 54.29 ± 5.26%, p=0.01) and PUFA diets (27.82 ± 5.56% vs 50.03 ± 3.78%, p=0.03).

Conclusion: Acute exercise improves endothelium sensitivity to ACh while PUFA supplementation alone or associated to exercise has no effect on endothelium-dependent vasorelaxation. However, PUFA may potentiate acute exercise effect on smooth muscle cell relaxation via L-type Ca²⁺ channel modifications.

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