"OR029 : Prebiotics Supplementation Improves the Endothelial Dysfunction Induced by a Nutritional Depletion in N-3 Polyunsaturated Fatty Acids"

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ABSTRACT

Rationale: Nutritional disorders are often associated with a high risk to develop cardiovascular diseases, endothelial dysfunction being an early key marker. Changing the gut microbiota by prebiotics counteracts metabolic diseases. The work tests the hypothesis of a potential impact of prebiotics on the endothelial dysfunction induced by a nutritional depletion in n-3 polyunsaturated fatty acids (PUFA) in ApoE-/- mice. Methods: C57Bl/6J (WT) and ApoE-/- (KO) mice were fed a n-3 PUFA depleted-diet (DEF) for 12 weeks. For the last 15 days, WT and KO mice were or not supplemented with prebiotics (PRE). The vascular function was evaluated in mesenteric and carotid arteries. Circulating HbNO adducts were assessed by EPR. Results: The n-3 PUFA depletion induced an endothelial dysfunction in KO-DEF mesenteric arteries, as attested by a significant decrease of endothelial-dependent relaxation compared to WT DEF. Interestingly, PRE treatment in this group was able to improve the endothelial f...

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PREBIOTICS SUPPLEMENTATION IMPROVES THE ENDOTHELIAL DYSFUNCTION INDUCED BY A NUTRITIONAL DEPLETION IN N-3 POLYUNSATURATED FATTY ACIDS

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Rationale: Nutritional disorders are often associated with a high risk to develop cardiovascular diseases, endothelial dysfunction being an early key marker. Changing the gut microbiota by prebiotics counteracts metabolic diseases. The work tests the hypothesis of a potential impact of prebiotics on the endothelial dysfunction induced by a nutritional depletion in n-3 polyunsaturated fatty acids (PUFA) in ApoE−/− mice.

Methods: C57Bl/6J (WT) and ApoE−/− (KO) mice were fed a n-3 PUFA depleted-diet (DEF) for 12 weeks. For the last 15 days, WT and KO mice were or not supplemented with prebiotics (PRE). The vascular function was evaluated in mesenteric and carotid arteries. Circulating HbNO adducts were assessed by EPR.

Results: The n-3 PUFA depletion induced an endothelial dysfunction in KO-DEF mesenteric arteries, as attested by a significant decrease of endothelial-dependent relaxation compared to WT DEF. Interestingly, PRE treatment in this group was able to improve the endothelial function by restoring the endothelial-dependent relaxation. PRE treatment in KO-DEF mice also lead to a vessel enlargement, to an increased intima-media thickness in the 2nd order mesenteric arteries, to an increased basal tone and a better KCl-induced contractibility of mesenteric arteries, compared to non-supplemented KO DEF mice. More, circulating HbNO was increased in KO-DEF-PRE mice and an improvement of endothelial function was also observed in carotid arteries.

Conclusion: Our results demonstrated an outward remodeling and an improvement of the endothelial dysfunction in mesenteric arteries following prebiotic supplementation, probably due to activation of NO/NOS pathway. Data are in progress to evaluate how changes in the gut microbiota composition and function act on vascular function. This supports a novel therapeutic role for dietary prebiotics in the control of cardiovascular disease risk.

Disclosure of Interest: None Declared

Keywords: endothelial dysfunction, Prebiotics