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DOI: 10.1159/000490189**The Level of Monocyte Chemoattractant Protein-1 May Be Affected by Several Factors**

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Dear Editor,

I read with a great interest the study by Mazaherioun et al. [1] entitled “Beneficial Effects of n-3 Fatty Acids on Cardiometabolic and Inflammatory Markers in Type 2 Diabetes Mellitus: A Clinical Trial” [1]. The authors aimed to study the effect of supplementation with n-3 polyunsaturated fatty acids (PUFAs) on levels of circulatory monocyte chemoattractant protein-1 (MCP-1) and resistin in patients with type 2 diabetes mellitus. They demonstrated the improvement in MCP-1 levels and lipid profiles after supplementation with n-3 PUFAs and no change in the serum resistin levels. They concluded that the anti-inflammatory effects of n-3 PUFAs might be mediated by targeting MCP-1.

MCP-1 is a powerful chemokine. It is synthesized and released by cardiac, vascular, and renal cells after hemodynamic stimuli such as sheer stress, blood flow, or oxidative stress and humoral stimuli such as angiotensin II and endothelin-1 [2]. Resistin is an adipose tissue-derived protein involved in the development of insulin resistance [1]. The secretion of MCP-1 is affected by age, body mass index, exercise, increased serum triglyceride concentration, insulin resistance, diabetes mellitus, cigarette smoking, coronary artery disease, hypertension, thrombus formation, tuberculosis, recurrent miscarriage, multiple sclerosis, HIV-neurological complications, nephropathy, inflammatory bowel disease, allergic asthma, rheumatoid arthritis, retinopathy due to oxygen-induced injury, tumor neovascularity, ischemia-related neuronal death,

and drugs such as doxycycline, amlodipine, leukotriene receptor antagonists, and HMG-CoA reductase inhibitors [2]. It has been reported that genetic variations could be an important factor in the variability of plasma levels of MCP-1 [3]. In an experimental study, increased expression of MCP-1 was also shown in hypertrophic and failing hearts due to pressure overload in the aorto-caval fistula, ischemic and reperfused rat hearts, and ischemic or inflammatory renal diseases [4]. The data presented by Mazaherioun et al. [1] suggest that serum insulin levels, blood pressure, and echocardiographic measurements of patients could be beneficial in terms of assessment of insulin resistance and heart disease.

While the authors concluded that the anti-inflammatory effects of n-3 PUFAs might be mediated by targeting MCP-1, they did not study any inflammatory biomarkers such as C-reactive protein, interleukin-6, and tumor necrosis factor- α in addition to MCP-1 and resistin. Subclinical inflammation is not uncommon in patients with type 2 diabetes mellitus [5].

I submit that the results of the study by Mazaherioun et al. [1] would have been stronger if the above-mentioned confounding factors were considered. We could then make a confident assertion that the increased MCP-1 levels in patients with type 2 diabetes mellitus was due to supplementation with n-3 PUFAs.

References

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Editor's Note: The author was requested to submit a response but no response was offered.