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Editorial

Importance of Antioxidants in Rheumatoid Arthritis

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Editorial

The exact cause of Rheumatoid Arthritis (RA) is unknown. A hypothesis regarding the onset of the disease is exposure of genetically susceptible individuals to environmental factors. Subjects with HLA-DR, PADI4 and PTPN22 alleles are more prone to RA disease [1]. Environmental factors could trigger the disease in genetically predisposed people. Among the factors, smoking has a significant role in increasing the risk of RA especially in individuals with anti-citrullinated protein antibodies (ACPA positive). Some bacteria (*E. coli, Streptococcus, Mycobacterium, Mycoplasma, and Helicobacter pylori*) and viruses (Epstein-Barr, parovirus and rubella) might also involve in the disease [2].

In RA disease in addition to the inflammatory cytokines and matrix metalloproteinases (MMPs), free radicals are also produced in excessive amounts which enhance the inflammatory pathways and interaction with host cells including chondrocytes, fibroblasts and osteoclasts [3-6]. Inflammatory responses are accompanied by generation of oxidant compounds *via.* inflammatory cells (neutrophils). Neutrophils in their plasma membrane have NADPH oxidase enzyme. With stimulation of the cells by bacteria and immune complexes, the enzyme produces a type of Reactive Oxygen Species (ROS) known as superoxide radicals (O_2^-). The radicals induce reactions of other ROS production [7,8]. Following the commencement of immune responses, free radicals can also be produced by monocytes, macrophages and granulocytes. In patients with RA, monocytes generate oxygen radicals 2.7 times higher than in healthy subjects [9-12]. Electron transport during oxidative phosphorylation in mitochondria is another origin of a type of ROS named to mtROS. Free radicals are consisted of not only ROS such as superoxide, peroxide and hydroxyl, but also include Reactive Nitrogen Species (RNS) [7,8].

Production of reactive oxygen and nitrogen species is a physiological process of the body's defense system against bacterial infections, but their incessant over expression leads to bone and cartilage destruction. Free radicals have the ability of attack to biological molecules including DNA, lipids, proteins and Low Density Lipoprotein (LDL); they cause loss of cellular integrity through damaging to unsaturated fatty acids in cell membranes, and functional changes in receptors and enzymes of the cells [13].

Antioxidant levels in synovial fluid and serum of RA patients is lower than healthy people; on the other hand, the continuous production of free radicals in inflamed joint gives rise to failure of antioxidant system (enzymatic and non-enzymatic) and further tissue damages [14]. ROS in particular mtROS trigger the expression of inflammatory cytokines including IL-6 and TNF-α through MAPK pathway [15]. It could be said that disease progression to some extent is due to increase in production of ROS and decrease in body's ability to scavenge them. Observed metabolites caused by free radicals in the synovial fluid of patients with RA are also confirming the above statements [9-12]. One of the metabolites of lipid peroxidation being increased in the serum and synovial fluid of RA patients is Malondialdehyde (MDA) [12].

Concentration of vitamin E (alpha-tocopherol), vitamin C, beta-carotene, and selenium and zinc antioxidants is lower in serum of RA patients compared to healthy individuals. Some studies have shown that consumption of the antioxidants have protective effects against tissue damage and may lead to clinical improvement in these patients [12,14]. Antioxidant compounds reduce inflammation by exertion their effects on the transcription factor of NF-κB, in RA patients [14]. Recently in our studies coenzyme Q_{10} (CoQ_{10}), a fat soluble antioxidant, showed a decreasing effect on serum MDA and proinflammatory cytokine of TNF-α [16]; it also improved the serum LDL concentration in RA patients [17].

Measurement of Total Antioxidant Capacity (TAC) can be used to evaluate the antioxidant status in serum. Decreased TAC indirectly reflects the increased activity of free radicals. Antioxidant defense system in RA patients encounters reduced levels of TAC and antioxidant enzymes (glutathione peroxidase, superoxide dismutase and catalase) and increased production of MDA. RA patients in our researches in comparison with healthy subjects, had a lower serum concentration of TAC and higher of MDA. One reason for this situation could be low intake of food antioxidants; as we found that dietary intake of zinc, vitamin C and E was lower than the recommended amounts in RA patients. Since the mentioned scavengers are a part of the TAC, their deficiency could increase lipid peroxidation.

Other studies have also shown that in people with RA dietary intake of micronutrients with antioxidant properties such as zinc, vitamin C, vitamin E, vitamin A and beta-carotene was low [18-21]. Loss of appetite and reduce the absorption and metabolism of dietary micronutrients would influence food intakes by patients with RA. The inability in food preparation and elimination of some foods owing to imaginary regimes might be other reasons for malnutrition in these patients [20,22]. Therefore, a review in diet of RA patients is necessary, and a nutritional education program that emphasizes the consumption of food sources of antioxidants would be helpful.

References

1. Klareskog L, Catrina AI, Paget S. Rheumatoid arthritis. Lancet. 2009; 373: 659-672.

2. Tobón GJ, Youinou P, Saraux A. The environment, geo-epidemiology, and autoimmune disease: rheumatoid arthritis. Journal of Autoimmunity. 2010; 35: 10-14.

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3. Tayar JH, Suarez-Almazor ME. New understanding and approaches to treatment in rheumatoid arthritis. Br Med Bull. 2010; 94: 201-214.

4. Brennan FM, McInnes IB. Evidence that cytokines play a role in rheumatoid arthritis. J Clin Invest. 2008; 118: 3537-3545.

5. Connell L, McInnes IB. New cytokine targets in inflammatory rheumatic diseases. Best Pract Res Clin Rheumatol. 2006; 20: 865-878.

6. Carrasco R, Barton A. Biomarkers of outcome in rheumatoid arthritis. Rheumatology Reports. 2010; 2: 26-38.

7. Veselinovic M, Barudzic N, Vuletic M, Zivkovic V, Tomic-Lucic A, Djuric D, et al. Oxidative stress in rheumatoid arthritis patients: relationship to diseases activity. Molecular and Cellular Biochemistry. 2014; 391: 225-232.

8. Stamp LK, Khalilova I, Tarr JM, Senthilmohan R, Turner R, Haigh RC. Myeloperoxidase and oxidative stress in rheumatoid arthritis. Rheumatology (Oxford). 2012; 51: 1796-1803.

9. Bae SC, Kim SJ, Sung MK. Inadequate antioxidant nutrient intake and altered plasma antioxidant status of rheumatoid arthritis patients. J Am Coll Nutr. 2003; 22: 311-315.

10. Pattison DJ, Symmons DP, Young A. Does diet have a role in the aetiology of rheumatoid arthritis? Proc Nutr Soc. 2004; 63: 137-143.

11. Ostrakhovitch EA, Afanas'ev IB. Oxidative stress in rheumatoid arthritis leukocytes: suppression by rutin and other antioxidants and chelators. Biochem Pharmacol. 2001; 62: 743-746.

12. Nourmohammadi I, Athari-Nikazm S, Vafa M, Bidari A, Jazayeri S, Hoshyarrad A, et al. Effects of antioxidant supplementations on oxidative stress in rheumatoid arthritis patients. Journal of Biological Sciences. 2010; 10: 63-66.

13. Finaud J, Lac G, Filaire E. Oxidative stress: relationship with exercise and training. Sports Med. 2006; 36: 327-358.

14. Pattison DJ, Winyard PG. Dietary antioxidants in inflammatory arthritis: do they have any role in etiology or therapy? Nat Clin Pract Rheumatol. 2008; 4: 590-596.

15. Naik E, Dixit VM. Mitochondrial reactive oxygen species drive proinflammatory cytokine production. J Exp Med. 2011; 208: 417-420.

16. Abdollahzad H, Aghdashi MA, Asghari Jafarabadi M, Alipour B. Effects of Coenzyme Q_{10} Supplementation on Inflammatory Cytokines (TNF-α, IL-6) and Oxidative Stress in Rheumatoid Arthritis Patients: A Randomized Controlled Trial. Archives of Medical Research. 2015; 46: 527-533.

17. Abdollahzad H, Alipour B, Aghdashi MA, Asghari Jafarabadi M. Coenzyme Q_{10} Supplementation in patients with rheumatoid arthritis: Are there any effects on cardiovascular risk factors? European Journal of Integrative Medicine. 2015; 7: 534-539.

18. Silva BN, Araújo ÍL, Queiroz PM, Duarte AL, Burgos MG. Intake of antioxidants in patients with rheumatoid arthritis. Rev Assoc Med Bras. 2014; 60: 555-559.

19. Cerhan JR, Saag KG, Merlino LA, Mikuls TR, Criswell LA. Antioxidant micronutrients and risk of rheumatoid arthritis in a cohort of older women. Am J Epidemiol. 2003; 157: 345-354.

20. Hejazi J, Mohtadinia J, Kolahi S, Bakhtiyari M, Delpisheh A. Nutritional status of Iranian women with rheumatoid arthritis: an assessment of dietary intake and disease activity. Women's Health. 2011; 7: 599-605.

21. Shaabani Y, Foroughi M, Rastmanesh R, Jamshidi A, Tajik N, Assadi O. Assessment of antioxidant nutrient intake and malondiadehyde plasma level in rheumatoid arthritis. ARYA Atherosclerosis. 2010; 5: 1-5.

22. Rennie KL, Hughes J, Lang R, Jebb SA. Nutritional management of rheumatoid arthritis: a review of the evidence. J Hum Nutr Diet. 2003; 16: 97-109.

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