Summary
Multiple sclerosis (MS) is a chronic and autoimmune disease of the central nervous system, leading to focal breakdown of the myelin sheath and axonal damage. There are two main forms of MS: relapsing-remitting (RRMS) and primary-progressive (PPMS). Both forms are inflammatory in nature, but disease-modifying therapies are currently available only for RRMS, not for PPMS.

Some years ago, we have introduced the idea that the lifestyle and dietary habits can influence the course of the disease. In fact, MS is a complex and multifactorial disease and it is acknowledged that environmental factors may contribute in some way to the disease and exacerbate or ameliorate its symptoms. In particular, the target of a nutritional intervention may be the control of the inflammatory status. This can happen in two ways: 1) modulating the inflammatory and metabolic activity of the human cell, and/or 2) by controlling the composition of gut microbiota and its inflammatory activity in the intestine. What increases inflammation are energy-dense Western-style diets, characterized by high salt, animal fat, red meat, sugar-sweetened drinks, fried food, alcohol, low fiber, and lack of physical exercise. The persistence of this type of diet, on one hand up-regulates the metabolism of human cells toward biosynthetic pathways, including the synthesis of pro-inflammatory molecules and, on the other hand, leads to a dysbiotic gut microbiota, alteration of intestinal immunity and low-grade systemic inflammation.

Conversely, exercise and calorie restricted diets based on the assumption of vegetables, fruit, legumes, and fish act on nuclear receptors and enzymes that up-regulate oxidative metabolism, while down-regulating the synthesis of pro-inflammatory molecules, and restoring or maintaining a healthy symbiotic gut microbiota. Anti-inflammatory dietary supplements, prebiotics and probiotics may be added to the diet to achieve a more robust effect of the nutritional intervention.

In a seven-month pilot study we investigated the effects of a calorie-restricted, semi-vegetarian diet and administration of vitamin D and other dietary supplements in 33 patients with RRMS, under therapy with IFN-beta and 10 patients with PPMS, with no therapy. At 0/3/6 months, patients had neurological examination, filled questionnaires and underwent anthropometric measurements and biochemical analyses. Serum fatty acids and vitamin D levels were measured as markers of dietary compliance and nutritional efficacy of treatment, whereas serum gelatinase levels were analyzed as markers of inflammatory status.

All patients had insufficient levels of vitamin D at baseline, but their values did not ameliorate following the administration of vitamin D equivalent to 914 I.U./day. Conversely, omega-3 polyunsaturated fatty acids increased already after three months, even under dietary restriction only.

After 6 months nutritional treatment, no significant changes in neurological signs were observed in any group, but physical and inflammatory conditions of the patients were improved. Only 4 patients withdrew from the study, indicating that the nutritional treatment was well accepted. Further nutritional clinical trials are needed, but in the meantime it may possible to provide nutritional guidance and physical activity opportunities to MS patients helping them to stay healthy. This may be especially relevant for PPMS, for which all efforts are still oriented only towards druggability and no attention is reserved to dietary habits and life style.
References