

Dietary Supplementation in Multiple Sclerosis

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It is more than 50 years since epidemiologists and pathologists in Europe and North America suggested a relationship between dietary fat, particularly that in animal fat and the saturated fatty acids and the incidence and prevalence of multiple sclerosis. Neuropathologically a relative deficiency of polyunsaturated fatty acids was identified in the demyelinated lesions in brains from people with multiple sclerosis and, more significantly, in the normal appearing white matter from those same brains. The suggestion which followed was that a relative deficiency in polyunsaturated fatty acids in the diet was a predisposing factor to the development of multiple sclerosis and this led to the concept of correcting the presumed deficiency by dietary supplementation with polyunsaturated fatty acids.

The initial concept received some support from animal studies with experimental allergic encephalomyelitis in which there was evidence that both omega-6 polyunsaturated fatty acids and omega-3 polyunsaturated fatty acids could lessen the severity of the immune mediated disease. The relevance of these animal studies is now called into question by better understanding of the nature of the immune response in EAE when compared to MS.

There were several randomised, controlled clinical trials of intervention with polyunsaturated fatty acids during the 1970s and 1980s when the only available measurements of relapse rate, disease progression and activities of daily living provided uncertain and conflicting results. Most studies would be regarded as being too small, they were frequently performed in single centres, and the endpoints were not always appropriate.

Since 2000 there have been a few studies of polyunsaturated fatty acid supplementation which have again provided variable results. The most recent trial of omega-3 polyunsaturated fatty acids eicosapentaenoic and docosahexaenoic acids on clinical disease activity and the surrogate marker of MRI failed to show benefit. Modern studies are affected by the concomitant use of disease modifying therapies but there is some suggestion that polyunsaturates and antioxidants may affect relapse rate in people with RRMS. Antioxidants alone have been reported to affect progressive MS, both primary and secondary.

The presentation will discuss the early results and the limitations and problems in the design, monitoring and interpretation of trials in multiple sclerosis involving any form of dietary or vitamin supplementation.

References:

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