Multiple sclerosis and nutrition

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Benefits from any particular diet in multiple sclerosis (MS) have not yet been proven. It is, however, frequent that malnutrition may potentially exacerbate the symptoms of MS. There is some evidence that a high intake of saturated fat increases the incidence of MS. Epidemiological studies imply that unsaturated fatty acids may have a positive effect on the course of MS. However, the results of controlled studies are ambiguous. A meta-analysis of three small controlled clinical trials suggests a benefit from linoleic acid. Intake of Vitamin D is associated with a lower incidence of MS. In MS, the risk of osteoporosis is high, and prophylactic vitamin D and calcium should be considered at an early stage. The role of minerals, trace elements, antioxidants, vitamins or fish oil is unclear. The possible relationships between diet and MS have not been subjected to adequate study. It seems possible that in the future, diets or dietary supplements may become recommended forms of treatment for MS.

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Introduction

Publications on multiple sclerosis (MS) tend to begin almost ritually with the notion that the possibilities available to modern medicine have been revolutionized during the last decade and that today a wide range of effective treatments are available. Many patients, however, perceive the reality differently: they are suffering from a disease of unknown origin, which cannot be healed; whose symptoms are difficult to treat, and whose course, despite all of the medical measures taken, frequently and inexorably worsens. It is, therefore, almost inevitable that many patients turn to alternative therapies for help. The majority of MS patients employ a variety of alternative therapies, and diet or dietary supplements constitute a large proportion of these measures.1,2 The main motive is not a general mistrust of medical science, but an understandable desire to try anything which might seem to offer the patient some hope of recovery. Moreover, complicated, time-consuming diets or other adjustments in lifestyle may provide a sense of control and initiative, restraining the feeling of being helplessly exposed to a mysterious disease.3 Such needs are largely ignored by the medical profession at large and create a breeding ground for profit-oriented doctors and other healers.

Although no specific benefit from a particular diet has been proven, numerous experimental, epidemiological and clinical studies suggest that nutritional factors may influence the incidence as well as the course of the disease. With this review, we intend to provide a brief overview of the epidemiological studies on diet and the incidence of MS and to discuss the possible effects of particular diets and dietary supplements.

Diet as a possible risk factor for MS

The first clinical descriptions of MS date back to the end of the 19th century. Because of its distinctive clinical characteristics, it may be assumed that the disease was essentially nonexistent previous to that time. The incidence of MS is strikingly related to geographic latitude and generally increases with the distance from the equator.4,5 Dietary factors have repeatedly been suggested as a possible explanation for this phenomenon. However, there is, as yet, no definite proof that diet actually influences the incidence of MS.4,5

Methodological problems complicate investigations on the correlation between diet and multiple sclerosis. There are only a few prospective ecological studies available and most evidence is derived from case-control or population-based epidemiological studies. Population-based epidemiological studies indicate a variety of associations between MS and diet.5,6 More than 50 years ago, Swank et al. analysed the incidence of MS in geographically diverse regions in Norway.7 They found a
higher incidence in the inland areas compared with the coastal regions where the intake of fish was higher and the consumption of saturated animal fat was lower. In the USA, Agranoff and Goldberg linked the incidence of MS to geographic latitude and dietary habits. High consumption of milk and low average ambient temperatures were associated with a high incidence, whereas the intake of fish and unsaturated fat seemed to be of a preventive nature. Alter et al. analysed epidemiological data from 22 countries. The prevalence of MS correlated positively with the intake of total energy, fat, oil and protein. Above all, the intake of animal fat was associated with the prevalence of MS (a correlation coefficient of 0.7). Knox analysed mortality statistics from 20 countries. Mortality from MS correlated with the consumption of a variety of foodstuffs of animal origin (meat, milk, butter, eggs) and refined sugar. The strongest correlation was with the total fat intake. Butcher studied the association between the consumption of milk and the prevalence of MS. He found regional correlations as well as temporal changes related to the consumption of milk. For example, the incidence of MS in Japan rose between 1950 and 1969 in parallel with increased consumption of milk. Lauer et al. evaluated the incidence of MS in numerous states of the USA. The intake of meat, dairy products and low average temperatures were each independently associated with the incidence of MS. Esparza et al. confirmed these results. In their analyses of epidemiological data from 38 countries, a high intake of animal fat and geographic latitude were related to the incidence of MS. In summary, most population-based epidemiological studies suggest an association between the incidence of MS and the intake of saturated fat of animal origin.

However, these findings are not confirmed by the majority of the population-based case-control studies which have failed to identify a relationship between the intake of fat or meat and the incidence of MS. Only a few case control studies found any association between the incidence of MS and the intake of meat, fat of animal origin, saturated fat or dairy products. The case-control studies have yielded mixed results (overview and discussion of the methodological problems see Lauer et al.). A large number of differing risk factors were identified including the consumption of brain, eggs and confectionery, young potatoes, alcohol, smoked meat products, and, in an Italian study, a high intake of pasta, bread, horse meat, minestrone, coffee and tea. As far as breast feeding is concerned, there have been a number of conflicting results. As preventive factors, some individual studies suggest the intake of vitamin D (see below) and a high consumption of vegetables. Remarkably, most studies found no correlation with the consumption of fruit or vegetables.

The evidence from the epidemiological studies is inconsistent and difficult to interpret. Many studies suggest an association between the incidence of MS and the intake of saturated fat of animal origin. However, any causal relationship remains to be proven.

Nutritional status in MS patients

Dietary habits and the nutritional status in MS patients have not been extensively studied. Individual findings and our own experience suggest that many patients suffer from various forms of malnutrition; obesity, weight loss, or vitamin deficiency are not unusual. Studies on the nutritional status in MS depend heavily on the patient group selected. In a population of unselected patients—many of them had been only recently diagnosed and were only slightly or moderately disabled—the nutritional status was comparable with that of the population in general. With severely disabled patients, the proportion of patients suffering from malnutrition and weight loss increases. Hewson et al. documented the food intake of 11 patients over seven days. In comparison to a healthy control group, the patients had a smaller intake of calories; however, the composition of the food was no different. Timmerman and Staffenboom analysed the food intake of women with MS over three days. When compared with current recommendations, the patients consumed an inadequately small amount of carbohydrates, fibres, vitamin E, calcium and zinc. In contrast, their intakes of saturated fat, protein, vitamin A and C, folic acid and iron were greater. The reliability of these studies is limited as the patient groups were heterogeneous, and it can be assumed that only those patients who are relatively moderately disabled or who are in receipt of excellent care participate in trials of this type.

Williams et al. examined 20 severely disabled patients; 10 of them suffered from pressure sores. For both groups, the intakes of energy, folic acid, vitamin D, iron and zinc were below recommended values. All of the patients had relatively low levels of albumin, zinc and iron. The patients with pressure sores had significantly lower serum levels of iron and zinc. The authors concluded that the prevalence of malnutrition in severely disabled MS patients is high, and that deficiencies in zinc and iron make them more susceptible to pressure sores. However, in another study, the serum levels of copper, selenium and zinc were within the normal range.

Obesity is frequent in MS patients. Immobility and subsequent low energy expenditure, steroids, anti-depressants, and a boring, inactive daily life are amongst the causal factors. On the other hand, obesity and an eating pattern consisting of a few heavy meals per day can aggravate fatigue syndromes, cause complications such as pressure sores or thrombosis, or may worsen disabilities already existing.

With increasing disability, weight loss, malnutrition and cachexia are frequently found. In addition to MS-specific factors such as dysphagia or adynamia, drugs potentially contribute to the development of malnutrition. Dysphagia is a common and often overlooked symptom in MS patients; its actual prevalence may be underestimated. Baclofen (nAUSEA, sedation), Metoclopramid (diarrhoea) or antibiotics are drugs which typically contribute to lack of appetite. Malnutrition itself can aggravate fatigue, muscular weakness or spasms and is
often neglected as an aetiologic cofactor of these common MS symptoms. 29
To summarize, nutritional imbalances in MS are common and may have a negative effect on the course of the disease, as well as, more importantly, the patient’s quality of life. For the individual patient, an interdisciplinary approach including dietary advice, occupational, speech and physical therapy is required.

Fat and fatty acids

The hypothesis that a modification of the intake of fat might influence the course of MS is derived from pathophysiological considerations, and the results from animal experiments and epidemiological studies suggesting an increased incidence of MS in populations with a high intake of saturated fat. Polysaturated fatty acids, in particular, omega-3 fatty acids, produce various immunomodulatory and anti-inflammatory reactions which may influence the course of the disease. 34 Vegetable fats constitute the main source of unsaturated fat.

In the European and North American diet, omega-6 unsaturated fatty acids predominate. Important essential fatty acids from dietary sources are alpha-linolenic acid (omega-3) and linoleic acid (omega-6). To a large extent, omega-3 fatty acids are also derived from eicosanoids (eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)). From these fatty acids, the same enzyme systems synthesize various other unsaturated fatty acids, which serve as the main precursors of leukotrienes and prostaglandins. If the intake of omega-3 fatty acids is increased, they will partially replace the omega-6 fatty acids within the cell membranes. 35 Omega-3 and omega-6 fatty acids are the substrates of identical enzymes for the synthesis of prostaglandins. Therefore, an increased intake of omega-3 fatty acids, e.g., with eicosanoids from fish oil, slows the production of the proinflammatory leukotrienes and prostaglandins synthesized from arachidonic acid. In a small study of 20 MS patients, Gallai et al. demonstrated that omega-3 fatty acids actually decreased the serum levels of various proinflammatory cytokines. 36 However, these mechanisms are complex, and the traditional concept that omega-3 fatty acids are anti-inflammatory, and omega-6 fatty acids are proinflammatory, is only partially true, and indeed, obsolete and simplistic. 37 38 In high concentrations, omega-3 fatty acids have anti-coagulatory effects and cause bleeding complications. In Western countries, the intake of omega-6 fatty acids is relatively too high when compared with omega-3 fatty acids. The most important sources of omega-3 fatty acids are a number of vegetable oils and sea fish.

Omega-6 fatty acids

Several authors found low levels of linoleic acid in the blood, blood cells and cerebrospinal fluid (CSF) of patients with MS. 39 41 However, the results vary. 42 44 In the brain tissue of MS patients the levels of linoleic acids and other polysaturated fatty acids are low. 45 A deficiency in linoleic acid exacerbates the symptoms of the experimental allergic encephalomyelitis (EAE). 46 47 While substitution with linoleic acids acts in a protective fashion. 48 49 In addition to the evidence from the epidemiological studies, these results provided the theoretical basis for clinical studies with linoleic acid.

There have been three randomized intervention studies with linoleic acid (Table 1). All of the studies were relatively small, and the statistical power too small to be able to detect any resultant mild therapeutic effects. Sunflower oil was used as the source of linoleic acid. No study showed any effect on relapse rate and the degree of disability. In two studies, significant differences in the severity and duration of the relapses in favour of active treatment were noted. 50 51 The third study did not show any difference at all between the treatment groups. 52 Thus, these studies do not justify recommending therapy with linoleic acid. However, regarding the pilot character of the study and the small number of patients, the results should not be interpreted as being entirely negative. Possibly, the relative small amount of linoleic acid used in these trials was not the optimum dose, and a larger dose would produce better results.

In a meta-analysis of these trials, Dworzak et al. detected significant beneficial effects, more pronounced in the group of patients with mild disability (EDSS 0–2). 53 In the group of severely disabled patients (EDSS 3–6), the positive effects were less consistent, but still visible. Within the mildly disabled patients, the intervention group showed a slower progression of disability (P=0.08) and a better relapse score (severity and dura-

Table 1 Randomized studies with unsaturated fatty acids

<table>
<thead>
<tr>
<th>Patients (n)</th>
<th>Therapy</th>
<th>Placebo</th>
<th>Duration</th>
<th>Primary outcome</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milik, 1973 40</td>
<td>Sunflower oil (17.2 g linoleic acid/day)</td>
<td>Olive oil (0.4 g linoleic acid/day)</td>
<td>24 months</td>
<td>EDSS</td>
<td>Trend towards fewer relapses and significantly milder relapses with sunflower oil</td>
</tr>
<tr>
<td>Paty, 1976 41</td>
<td>Sunflower oil (17 g linoleic acid/day)</td>
<td>Olive oil (1 g linoleic acid/day)</td>
<td>30 months</td>
<td>EDSS</td>
<td>No difference</td>
</tr>
<tr>
<td>Bates, 1978 40</td>
<td>Linoleic acid (3 or 23 g/day)</td>
<td>Oleic acid (4 or 16 g/day)</td>
<td>24 months</td>
<td>Number, duration, severity of relapses: Clinical deterioration</td>
<td>No significant difference. Trend in favour of high-dose linoleic acid</td>
</tr>
<tr>
<td>Bates, 1988 41</td>
<td>Fish oil (1.7 g EPA, 1.1 g DHA)</td>
<td>Olive oil</td>
<td>24 months</td>
<td>EDSS</td>
<td>No significant difference. Trend in favour of fish oil</td>
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tion) \( (P = 0.001) \). These promising results should justify a larger clinical trial. Unfortunately, this trial has never been undertaken, probably due to the high costs of such a trial and the absence of any commercial interest. Linoleic acid in vegetable oils is readily available in every supermarket and has, therefore, no commercial potential as a pharmaceutical drug whatsoever.

In some countries, evening primrose oil is a popular alternative MS treatment. Evening primrose oil is a vegetable oil rich in gamma linolenic acid, an omega-6 fatty acid. After a randomized study did not show any positive effects, no further studies with evening primrose oil have been undertaken. The dietary needs for omega-6 fatty acids can be satisfied much more easily and cheaply with other vegetable oils.

**Omega-3 fatty acids**

In cardiovascular diseases, there is an increasing body of evidence that omega-3 fatty acids reduce the risk of cardiovascular events and sudden death. A large study demonstrated a reduction in cardiovascular mortality from supplementation with omega-3 fatty acids after myocardial infarction. The American Heart Association recommends the intake of omega-3 fatty acids for all patients with coronary heart disease. In various autoimmune diseases, beneficial effects from omega-3 fatty acids are also assumed.

Where MS is concerned, there is not enough data available to be able to confirm the beneficial effect of omega-3 fatty acids. Large controlled studies do not exist. Some investigations found low levels of omega-3 fatty acids in patients with MS. In contrast to omega-6 fatty acids, in experimental environments, omega-3 fatty acids have not been extensively studied, and the results vary. In the large Nurses’ Health Study, there was no association between the incidence of MS and the intake of omega-3 fatty acids from fish.

Nordvik et al. treated 16 newly diagnosed patients with omega-3 fatty acids. They observed a reduction in the relapse rate and an improved EDSS. However, this small study was neither randomized nor controlled. Similar results were already published in 1986 in a small sample of 10 patients. Because of the obvious weaknesses of these studies, the results can hardly be used for practical recommendations.

A large, randomized, placebo-controlled study with 292 patients showed no significant differences between active treatment with fish oil (containing high amounts of omega-3 fatty acids) and a placebo (Table 1). However, there were nonsignificant trends in almost all of the results in favour of fish oil. The therapeutic effects were arguably reduced because the patients in the placebo group received dietary advice to increase their intake of polyunsaturated omega-6 fatty acids. From an ongoing randomized study on the effects of a fat-reduced diet with supplementation with omega-3 fatty acids, only provisional data from the first 23 patients is available, suggesting beneficial effects from active treatment.

Today, there is still not enough data available to recommend omega-3 fatty acids for MS. However, due to the generally accepted beneficial effects for cardiovascular diseases, the consumption of fatty fish and vegetable oils containing omega-3 fatty acids should be encouraged, independent of the possible effects on MS.

It should also be noted that large amounts of omega-3 fatty acids can create unwanted side effects. High dosages of omega-3 fatty acids (>3 g/day) increase the risk of bleeding complications due to their anti-coagulatory effects. Other side effects include gastrointestinal symptoms, increased LDL-cholesterol levels and hyperglycaemia in patients with diabetes. Therapy with omega-3 fatty acids should, therefore, only be undertaken under medical supervision. The consumption of fish alone will hardly produce concentrations that might lead to unwanted side effects. Irrelevant from the medical point of view, but disappinting and not unproblematic in daily life, is a distinctly fishy smell, which may occur after the intake of large amounts of fish oil.

**Vitamin D**

Vitamin D epidemiological evidence and risk of osteoporosis. In MS patients, osteoporosis is a frequent, underdiagnosed and undertreated complication of a chronic vitamin D deficiency which may lead to additional morbidity. The majority of patients suffer from vitamin D deficiency. Nieves et al. analysed bone density and laboratory parameters of vitamin D metabolism in women with MS. The bone density and the mean 25(OH) vitamin D levels were much lower compared with a healthy control group. Forty per cent of the patients reported almost no exposure to sunlight.

In a study researching the progression in bone density over two years, the risk of fractures in the absence of major trauma was 10-fold, and the bone loss was seven times greater in MS patients compared with the control group.

In addition, a low intake of vitamin D, immobility, corticosteroid therapy and sun avoidance due to heat sensitivity and fatigue makes MS patients prone to osteoporosis. Thus, it would seem justifiable to recommend that preventive therapy with vitamin D and calcium should be generally established for MS patients very widely, and in particular for all postmenopausal women and patients receiving repeated corticoid therapy.

In everyday practice, this problem is often overlooked: 71% of the postmenopausal women with MS reported by Shabas et al. did not take vitamin D, and only 50% used calcium supplements.

**Vitamin D as a specific therapy**

A relationship between the geographic distribution of MS, exposure to sunlight, and vitamin D metabolism has been suspected for many years. In northern regions, winter sunlight is not sufficient to produce adequate amounts of vitamin D. With people who live in regions > 42° latitude and have a low intake of vitamin D, a relative vitamin D deficiency during four to six months of the year is common. People who have a particularly high exposure to sunlight suffer from a higher risk of melanoma, but their risk of suffering from MS is reduced. Clinical relapses as well as new lesions in...
the MRI appear most frequent in spring, when vitamin D storage and 25(OH) vitamin D levels are at their lowest. New lesions in the MRI appear most frequently two months after 25(OH) vitamin D levels were lowest. Vitamin D exerts numerous immunomodulatory effects. Vitamin D increases the proliferation of lymphocytes and reduces the production of proinflammatory cytokines. In experiments with animals, treatment with vitamin D reduces or prevents the symptoms of EAE. The exact mechanisms of this phenomenon have not yet been sufficiently well explained.

A recent prospective epidemiological study supports the hypothesis that vitamin D reduces the incidence of MS. In the US–American Nurses’ Health Study, women who took additional vitamin D supplements had a 40% lower risk of MS. However, women who used vitamin D supplements frequently also took other vitamins. This study analysed the association between the intake of vitamin D and the incidence of MS and provides no information on the possible effects of vitamin D in patients already manifesting the disease.

To date, there has not been sufficient evidence to recommend a therapy with vitamin D for MS. Interventional studies with vitamin D are rare. Goldberg et al. reported a reduction of the relapse rate after introducing a therapy containing calcium, magnesium and fish liver containing high amounts of Vitamin D. This study was uncontrolled and very small (11 patients). A methodologically better, MR-based pilot study, although equally small, showed no beneficial effects of a therapy with vitamin D.

Patients who wish to perform an experimental therapy with vitamin D should be made aware of the possible side effects of vitamin D. Subsequent to hypercalcaemia, vitamin D may produce cardiac arrhythmias and adynamia. However, the risk of these complications only arises when high amounts of vitamin D (>1000 μg/day) are consumed. Most dietary supplements, containing ≤100 μg vitamin D, are harmless. Renal diseases, sarcoldosis and hypocalcaemia are relative contraindications for vitamin D.

**Vitamin B₁₂**

The link between vitamin B₁₂ and MS has been under discussion for more than 50 years. Vitamin B₁₂ is a prerequisite for the synthesis of myelin, which has been postulated as an argument for the possible relationship between allegedly low vitamin B₁₂ levels and the incidence of MS. Furthermore, vitamin B₁₂ deficiency and MS share some clinical and MRI characteristics, indicating common pathophysiological mechanisms. Some authors reported a high prevalence of B₁₂ deficiency in patients with MS. However, most patients with MS have normal vitamin B₁₂ levels. Nijst et al. found low vitamin B₁₂ concentrations in the CSF of MS patients, while the serum levels were within normal limits. In addition to inadequate dietary intake, gastrointestinal malabsorption is not uncommon in MS and may contribute to vitamin B₁₂ deficiency. Immediately after corticosteroid treatment, the concentrations of folic acid and vitamin B₁₂ are decreased. It is also possible that many patients do not have an absolute vitamin B₁₂ deficiency, but suffer from a disturbance in the metabolism of vitamin B₁₂.

Controlled, large intervention studies with vitamin B₁₂ do not exist. A high-dose therapy with vitamin B₁₂ over six months in six severely disabled patients showed no clinical benefit. However, a reduction in the latency of the potentials noted during the course of the study was interpreted as an indication of the effectiveness of the therapy. In a placebo-controlled trial with 138 patients, Wade et al. found small, but insignificant, beneficial effects from a high-dose therapy of parenteral vitamin B₁₂ (combined with folate and l-phenylalanine, the 'Carli-Loher Regime').

Currently, there is no scientific basis to recommend supplementing the diet with vitamin B₁₂ apart from in the treatment of vitamin deficiencies. Vitamin B₁₂ deficiency in MS patients is not unusual. Therefore, these patients should be meticulously screened, in particular, as the typical neurological signs of vitamin B₁₂ deficiency may be imitated or aggravated by MS symptoms.

**Selenium**

Oxidative stress possibly plays a key role in the pathophysiology of MS. Therefore, because of its potent antioxidant properties, selenium may have a positive effect on the course of the disease. There is no reliable data available as to how many patients use selenium but our own experience suggests that a large proportion of MS patients use dietary supplements containing selenium, often combined with vitamins C and E, which also produce antioxidant effects. It is frequently claimed that selenium deficiency is both a common and important problem in MS. Indeed, clinical studies have yielded mixed results. In Finnish patients, the selenium content in the blood was markedly lower than in healthy control groups. Clauson et al. found low selenium levels and lowered glutathione peroxidase activities in the erythrocytes of MS patients. However, in a previous study some years earlier, the same authors described normal blood selenium levels. Compared with healthy control groups, the selenium concentrations in the erythrocytes of MS patients were even higher. Other authors also found higher or comparable selenium levels in the blood and erythrocytes of MS patients. The conclusion arrived at was that the decreased glutathione peroxidase activity found in the erythrocytes of MS patients, a surrogate marker of antioxidant capacity, is irrespective of the selenium levels, and is primarily the result of genetic factors.

An intervention study with selenium and vitamins C and E increased the activity of glutathione peroxidase in various blood cells of the patients treated. However, no beneficial clinical effects were determined.
An important aspect of every therapy with antioxidants is the question as to whether the substances are capable of passing the blood–brain barrier. In the presence of an intact blood–brain barrier, most antioxidants do not penetrate it. Analyses of selenium levels in whole blood or erythrocytes are, for that reason, of questionable relevance.

**Other antioxidant vitamins and compounds**

A study with 36 patients showed low levels of vitamin E in serum and CSF, but there was no association with clinical variables. A recent large epidemiological trial yielded no correlation between the intake of the antioxidants vitamin C, carotenoids and vitamin E and the incidence of MS. This study confirms several previous case-control studies which also found no correlation between the risk of MS and the intake of fruit and vegetables rich in vitamins A, C and E. These vitamins are harmful if their intake does not exceed the recommended maximum doses. Vitamin A should not be taken by women during pregnancy. There are no results available from clinical studies suggesting beneficial effects from vitamins A, C and E in MS.

In addition to vitamins, a large number of antioxidant compounds such as coenzyme Q10, alpha-lipoic acid and antioxidans from red wine or grape extracts are taken by MS patients. It is indisputable that these substances act as antioxidants. However, their effects on MS are entirely speculative. Some antioxidants have immunostimulatory effects which, theoretically, could also generate negative effects in MS. The consumption of antioxidants should be discouraged until valid clinical data on the safety and efficacy of these substances is available.

**Other minerals**

The role of calcium has been discussed above. Zinc deficiency may predispose patients to pressure sores. There is not enough data on iron or magnesium supplements, which are frequently taken by MS patients, to be able to draw valid conclusions.

**The Swank diet**

Worldwide, a large number of differing special diets for MS are propagated and followed by many patients. No clinical benefit from any of these diets has been proven. For most of these diets there is no scientific data available at all, and some of them are quite bizarre, based on dubious or plainly wrong pathophysiological considerations. The Swank diet is one example of the more popular diets based on pathophysiological considerations.

Using studies in Scandinavia which suggested a link between intake of saturated fatty acids and the incidence of MS as his argument, Swank developed a special diet for MS patients. The main aim of the Swank diet is a drastic reduction in saturated fat. No more than 15 g/day saturated fat is to be consumed, which means a radical change in the usual dietary habits in the USA and Europe. Fatty dairy products (>1% fat) are not allowed. Frequent seafood meals are recommended. In addition to the reduced fat intake, 15 g/day vegetable oil and 5 g/day cod liver oil are consumed (a source of omega-3 fatty acids and vitamin D). In contrast to other inventors of MS diets, Swank wished to provide his dietary therapy with a scientific justification and monitored the patients for many years. One hundred and forty-four MS patients adhering to the Swank diet were observed over 34 years.

Those patients who followed the dietary instructions strictly had a slower progression of disability and lower mortality than patients who ingested more than 20 g/day fat. The effects were most pronounced in patients who were only moderately disabled when treatment began. After commencing the diet, the relapse rate declined markedly (prior to the diet one relapse/year, after five years of diet 0.1 relapse/year). At first sight, these results look very promising. However, the validity of the study is limited, because it was neither controlled nor blinded or randomized. It can be assumed that there was a considerable selection bias, as patients who perceive no benefit from a particular therapy are not very likely to continue over a period of 34 years. Because of these methodological problems, the possible benefits from the Swank diet can be judged as proven. However, it can be assumed that strict adherence to the Swank diet will reduce cardiovascular mortality.

**Summary and recommendations**

MS is not a metabolic disorder. No beneficial effects from any particular diet have been proven. Therefore, the same recommendations concerning a well balanced diet apply equally as they do for the population in general. Malnutrition, vitamin deficiencies, obesity and weight loss are common and may exacerbate the clinical symptoms. There is no basis for recommendations to avoid particular foods (e.g., alcohol, meat, wheat/gluten, coffee, animal fat). Some dietary therapies, particularly those that exclude whole food groups, are potentially harmful as they can lead to malnutrition. In certain situations (constipation, fatigue, dysphagia, etc.) a modification of the nutritional habits may lead to an improvement in the clinical symptoms.

Epidemiological studies indicate a correlation between the consumption of animal fat and the incidence of MS. Moreover, there are abundant findings from experiments on animals and theoretical considerations to suggest beneficial effects from unsaturated fatty acids. A meta-analysis of three controlled trials suggested a clinical benefit from linoleic acid. Although there is as yet no definite proof for this recommendation, it would seem justifiable to satisfy the need for fat predominantly with high-quality vegetable oils with a high content of unsaturated fatty acids, and to take two or more seafood meals.
per week. It remains a subject of speculation whether supplementing with various minerals, trace elements, fish oil or vitamins is helpful. One exception is the prophylaxis of osteoporosis with calcium and vitamin D, which should be widely recommended. Diets that forbid dairy foods can provoke vitamin D deficiency and could consequently be harmful. Dietary supplements with no proof of their efficacy, which are freely available, should be viewed with great caution. However, in our experience it is frequently not helpful to actively discourage a patient from a certain diet or other alternative therapy if she or he strongly believes in its efficacy even if this is highly questionable or very improbable from a medical point of view. Usually, these therapies are quite harmless, although frequently quite expensive.

The possible relationships between MS and nutrition have not been adequately researched. There is not enough clinical trial data or reliable information available to advocate the use of any particular diet or dietary supplement as a disease-modifying therapy. However, it seems probable that, in the future, a modification in diet or dietary supplements might be recommended as a treatment for MS.

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