The Relation between Nutritional Factors and Multiple Sclerosis: An Egyptian Study

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ABSTRACT

Background: No single environmental exposure has been consistently identified as a causal factor in MS. It has been suggested that nutrition and food patterns may play a role in the aetiology of MS. Objective: To explore the nutritional pattern of MS Egyptian patients in comparison with that of controls. Methods: The relation between nutritional factors and MS was studied among 30 MS patients and 30 matched controls in neurology department Cairo University between June 2006 and January 2007. Dietary information was collected by employing 24 hours recall method and food frequency questionnaire. Results: MS patients were underbuilt with lower BMI as compared to controls. Also patients seem to consume less vitamin A, B₁, C, carbohydrates, proteins and more fat. A significant negative correlation was found between patient disability as measured by EDSS and iron and B₁ intake. We found no statistical significant difference between patients and controls in zinc, iron, calcium, vitamin A, B₂, C carbohydrates, fat and protein on one hand and patient disability. Conclusion: The study generally supports a protective role for antioxidants and increased risk of MS with fat consumption. (Egypt J. Neurol. Psychiat. Neurosurg., 2008, 45(2): 321-330)

INTRODUCTION

Multiple sclerosis (MS) is an autoimmune neurological chronic illness that usually begins in young adulthood. To date, little is known about the etiology of (MS).¹

Although a role for genetic factors is suggested by a 10-50 fold greater incidence of MS in relatives of MS patients, variations in its prevalence across continents and alteration in the risk of acquiring MS among children of immigrants to reflect the rates of the adoptive country²³ suggest a role of lifestyle and local environmental factors.¹ Increased risk has been associated with viral infections⁵, higher socioeconomic status⁴, and dietary habits.⁷⁸

The involvement of nutritional factors in the etiopathogenesis of multiple sclerosis is currently being investigated. Notwithstanding the huge amount of data present in the literature, the possible etiological or protective role of nutrients with regard to the disease remains debatable. The epidemiological data suggest an association between multiple sclerosis and nutrition; the populations that take in a higher quantity of foods of animal origin seem to be the most affected. A role of saturated fatty acids in the etiopathogenesis of myelinic damage has been hypothesized.⁹

However, with thorough local and international literature search, it has been realized that there is insufficient literature on studies looking at nutrition and MS in Egypt. The present study aims to explore the nutritional pattern of MS Egyptian patients in comparison with that of normal controls.

METHODOLOGY

This is a case-control study that has been conducted at department of neurology Cairo University during the period June 2006 to January 2007. It comprised two groups. The patients group comprised 30 MS patients (9 males and 21 females). Their diagnosis was based on McDonald criteria."
Among MS patients, 8 (26.7%) were of the SPMS type, while 22 (73.3%) were of the RRMS type all in remission. The control group comprised 30 age-matched (9 males and 21 females) apparently healthy subjects.

Patients who were on any type of restrictive diet for hypertension, diabetes, lipid abnormalities or otherwise were excluded from the study. Patients whose swallowing difficulties has an impact on food type or consistency were also excluded.

Dietary information was collected by trained interviewers relying on two retrospective methods of dietary assessment: The 24 hour recall and food frequency questionnaire (FFQ). In the 24 hour recall respondent recalls all foods and beverages consumed in a given 24-hour period and reports them to the interviewer, who probes to get additional details on portion sizes, frequency and forgotten items. On the other hand the FFQ designed by the members of nutrition and food science department faculty of home economics, Al-Azhar University - and represents a modified version of Willet questionnaire - to determine usual dietary intakes during the year prior to the interview including food likes and dislikes. The questionnaire asks for the accurate frequency of consumption of various foods per day, week or month, in the year before diagnosis of cases, or one year before interview for controls. It contains three sets of pictures of foods in terms of small, medium and large portions for all food items to help visualize portions consumed. Daily energy and nutrient intake (e.g. protein, fats, vitamins, minerals, etc.) were calculated using food composition tables of national nutrition institute(exported computer software).

- Weight and height of participants were measured. Body mass index (BMI) was calculated as weight (in kg) divided by squared height (in meter\(^2\)).
- The clinical type of MS was identified based on McDonald criteria.
- The Krutzke expanded disability status scale (EDSS) was assessed on all MS patients.

**Statistical Methods:**

Data were revised prior to computerized data entry. The Statistical Package of Social Sciences (SPSS, version 15.0) was used for statistical analysis. Descriptive statistics (i.e., frequency, percentage, mean and standard deviation) were calculated. Testing significance between the two study groups was applied using the unpaired t-test. Pearson’s correlation coefficient (r) was calculated between the EDSS and quantitative study variables. A statistically significant level was considered when p-value was less than 0.05.

**RESULTS**

Table (1) shows that both study groups were matched in their age (29.97±8.52 years vs. 31.53±4.7, for patients and controls respectively) and weight (62.17±11.06 vs. 69.77±9.10, for patients and controls respectively). However, the MS patients had significantly lower weight (p=0.003) and BMI (p=0.009). Among the patients group, the mean duration of disease was 4.73±1.82 years, while their mean EDSS was 4.21±1.96.

Table (2) shows that nutritional factors were generally less among MS patients than those for control subjects. Patients consumed significantly less vitamin A (p=0.01), less vitamin B1 (p=0.004), less vitamin C (p<0.001), less carbohydrates (p<0.001), less proteins (p=0.037) and less calories (p=0.023). MS patients showed higher intake of fats than their control (59.26±30.24 vs. 54.63±13.83, respectively). However, difference was not statistically significant.

Table (3) shows that as regard type of MS, there were no significant differences in all nutritional factors.

Table (4) shows that the EDSS correlated significantly and positively with age of patient (r=0.371, p=0.044), and duration of disease (r=0.597, p<0.001), while it correlated significantly and negatively with iron intake (r=−0.427, p=0.019), and vitamin B1 intake (r=−0.366, p=0.047).
Table 1. Characteristics of study subjects.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Patients Group</th>
<th>Control Group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ±SD</td>
<td>Mean ±SD</td>
<td></td>
</tr>
<tr>
<td>Age (in years)</td>
<td>29.97 ± 8.52</td>
<td>31.53 ± 4.70</td>
<td>0.381</td>
</tr>
<tr>
<td>Weight (in kg)</td>
<td>62.17 ± 11.06</td>
<td>69.77 ± 9.10</td>
<td>0.003</td>
</tr>
<tr>
<td>Height (in cm)</td>
<td>163.37 ± 3.22</td>
<td>165.33 ± 6.32</td>
<td>0.246</td>
</tr>
<tr>
<td>BMI (in kg/m²)*</td>
<td>23.26 ± 3.18</td>
<td>25.58 ± 3.46</td>
<td>0.009</td>
</tr>
<tr>
<td>Duration of MS (in years)</td>
<td>4.73 ± 1.82</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>EDSS**</td>
<td>4.21 ± 1.96</td>
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</tbody>
</table>

* Body Mass Index  ** Expanded Disability Status Scale

Table 2. Comparison between study groups as regard food constituents.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Patients Group</th>
<th>Control Group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ±SD</td>
<td>Mean ±SD</td>
<td></td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>13.43 ± 4.89</td>
<td>14.65 ± 3.31</td>
<td>0.261</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>14.16 ± 3.15</td>
<td>15.06 ± 5.05</td>
<td>0.413</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>685.02 ± 180.23</td>
<td>706.28 ± 261.49</td>
<td>0.715</td>
</tr>
<tr>
<td>Vitamin A (microgram)</td>
<td>993.20 ± 320.73</td>
<td>1251.12 ± 424.89</td>
<td>0.010</td>
</tr>
<tr>
<td>Vitamin B1 (mg)</td>
<td>1.26 ± 0.40</td>
<td>1.75 ± 0.80</td>
<td>0.004</td>
</tr>
<tr>
<td>Vitamin B2 (mg)</td>
<td>2.75 ± 1.65</td>
<td>2.31 ± 1.73</td>
<td>0.318</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>46.87 ± 14.63</td>
<td>63.84 ± 6.10</td>
<td>0.003</td>
</tr>
<tr>
<td>Carbohydrates (gram)</td>
<td>257.38 ± 103.72</td>
<td>414.69 ± 171.76</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fats (gram)</td>
<td>59.26 ± 30.24</td>
<td>54.63 ± 13.83</td>
<td>0.449</td>
</tr>
<tr>
<td>Proteins (gram)</td>
<td>72.44 ± 24.54</td>
<td>87.19 ± 28.75</td>
<td>0.037</td>
</tr>
<tr>
<td>Calories</td>
<td>1857.67 ± 649.50</td>
<td>2329.70 ± 898.48</td>
<td>0.023</td>
</tr>
</tbody>
</table>

Table 3. Comparison between secondary progressive multiple sclerosis (SPMS) and relapsing-remitting multiple sclerosis (RRMS) patients as regard food constituents.

<table>
<thead>
<tr>
<th>Variables</th>
<th>SPMS (n=8)</th>
<th>RRMS (n=22)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ±SD</td>
<td>Mean ±SD</td>
<td></td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>11.10 ± 4.28</td>
<td>14.27 ± 4.912</td>
<td>0.118</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>13.05 ± 3.90</td>
<td>14.57 ± 2.83</td>
<td>0.250</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>619.11 ± 163.52</td>
<td>708.98 ± 183.54</td>
<td>0.233</td>
</tr>
<tr>
<td>Vitamin A (microgram)</td>
<td>1173.06 ± 199.45</td>
<td>927.79 ± 334.65</td>
<td>0.063</td>
</tr>
<tr>
<td>Vitamin B1 (mg)</td>
<td>1.07 ± 0.43</td>
<td>1.33 ± 0.38</td>
<td>0.132</td>
</tr>
<tr>
<td>Vitamin B2 (mg)</td>
<td>2.25 ± 1.13</td>
<td>2.94 ± 1.79</td>
<td>0.322</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>46.10 ± 18.00</td>
<td>47.16 ± 13.69</td>
<td>0.865</td>
</tr>
<tr>
<td>Carbohydrates (gram)</td>
<td>249.76 ± 131.83</td>
<td>260.15 ± 95.04</td>
<td>0.813</td>
</tr>
<tr>
<td>Fats (gram)</td>
<td>58.21 ± 25.90</td>
<td>59.64 ± 32.22</td>
<td>0.911</td>
</tr>
<tr>
<td>Proteins (gram)</td>
<td>71.75 ± 14.14</td>
<td>72.69 ± 27.66</td>
<td>0.929</td>
</tr>
<tr>
<td>Calories</td>
<td>1660.16 ± 710.82</td>
<td>1929.50 ± 627.59</td>
<td>0.324</td>
</tr>
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</table>
Table 4. Correlation with Expanded Disability Status Scale in patients group.

<table>
<thead>
<tr>
<th>Variables</th>
<th>R</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (in years)</td>
<td>0.371</td>
<td>0.044</td>
</tr>
<tr>
<td>Duration of disease (in years)</td>
<td>0.597</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight (in kg)</td>
<td>0.141</td>
<td>0.456</td>
</tr>
<tr>
<td>Height (in cm)</td>
<td>0.318</td>
<td>0.086</td>
</tr>
<tr>
<td>BMI (in kg/m²)</td>
<td>-0.021</td>
<td>0.914</td>
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<tr>
<td>Zinc (mg)</td>
<td>0.353</td>
<td>0.055</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>-0.427</td>
<td>0.019</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>0.114</td>
<td>0.547</td>
</tr>
<tr>
<td>Vitamin A (microgram)</td>
<td>-0.206</td>
<td>0.275</td>
</tr>
<tr>
<td>Vitamin B1 (mg)</td>
<td>-0.366</td>
<td>0.047</td>
</tr>
<tr>
<td>Vitamin B2 (mg)</td>
<td>0.121</td>
<td>0.525</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>0.315</td>
<td>0.090</td>
</tr>
<tr>
<td>Carbohydrates (gram)</td>
<td>0.256</td>
<td>0.173</td>
</tr>
<tr>
<td>Fats (gram)</td>
<td>0.241</td>
<td>0.200</td>
</tr>
<tr>
<td>Proteins (gram)</td>
<td>0.084</td>
<td>0.660</td>
</tr>
<tr>
<td>Calories</td>
<td>0.344</td>
<td>0.063</td>
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</table>

**DISCUSSION**

For persons with multiple sclerosis (MS), good nutrition has the potential to enhance quality of life. Evidence has shown that optimal nutrition can reduce the risk of developing secondary conditions associated with MS and decrease the risk of developing other chronic diseases.16-19

Since the cure for MS remains elusive, patients seek alternative methods that may alleviate symptoms or delay disease progression. Unfortunately, patient decisions are not always based on sound nutritional recommendations. As a result, macronutrient and micronutrient deficiencies can develop, thereby placing patients at risk of adverse secondary conditions or malnutrition.20

Conversely, dietary supplementation with exceedingly high doses of macronutrients or vitamins and minerals is not only a risk for toxicity, but can also lead to deficiencies due to competitive binding to enzymes and transport proteins.20

The present study indicated that MS patients were generally under-built (significantly less BMI than control). This indicates that individuals with lower BMI are at higher risk of developing MS, to what extent this is a real association is not clear. It is possible that the presence of disease may have contributed to a hypermetabolic state and consequent lowering of body weight.1

This is consistent with a case-controlled study, in which 197 newly diagnosed MS cases were recruited over a four-year period and were matched with 202 controls for age and gender. The cases had significantly lower body mass index (BMI) than the controls.1

These findings can be explained by the fact that during the course of MS, secondary conditions such as bowel problems, dysphagia and reduced appetite may lead to unintentional weight loss and malnutrition. Factors that decrease appetite in patients with MS include fatigue and medications.17,19

Our MS patients seemed to consume more fats than controls. The increasing risk of MS with a high animal and saturated fat intake, as observed in this study, is consistent with findings from other studies.
Since the early 1950’s, it has been proposed that high consumption of saturated fat SFA, particularly from animal fat may be involved in the etiology and course of MS. Since lipids are the main component of the myelin sheath, the imbalance in the proportion of saturated to unsaturated fatty acids is postulated to be responsible for modifying the stability of the myelin sheath or for increasing platelet aggregation, with hypoxia in the microcirculation in the central nervous system and subsequent perivascular demyelination.21-26

Several case-control studies have suggested a positive association between MS and high saturated and animal fat (meat, milk, butter, eggs) consumption.27-29

The findings from these studies appear to be consistent, suggesting that lower consumption of saturated fat (i.e. <20 g/day) is related to significantly fewer exacerbations, slower deterioration, and lower death rates. Patients consuming 10–15 g per day or less of saturated fat had improved energy levels and less fatigue. On the other hand, saturated fat intake of approximately 20–42 g per day was associated with increased disability and three times the death rate.1,10,31

However, several population-based case-control studies, including a large prospective cohort study, have found that MS patients and controls did not differ significantly in fat intake, or that MS patients consumed less fat than controls prior to onset, suggesting that fat consumption does not increase the risk of MS.32

Approximately one-third of nervous tissue consists of polyunsaturated fatty acids (PUFA). PUFA are described as linoleic acid, or omega–6 fatty acid (n–6), and alpha-linolenic acid or omega–3 fatty acid (n–3). The body cannot synthesize them; therefore they must be obtained in the diet. PUFAs may not only exert immunosuppressive actions through their incorporation in immune cells but also may affect cell function within the CNS.33-35

In a Double Blind controlled study 36 involving 312 patients as well as in other studies36-39, it appears that there is a trend suggesting that the addition of omega six and omega three Polyunsaturated Fatty Acids to the diet of patients with Multiple Sclerosis results in a reduction of the severity and frequency of relapses in a two year period.

However, in patients with a progressive chronic form of the disease, polyunsaturated fatty acids did not demonstrate any effect on the progression of the lesions.9

Furthermore, according to Farinotti et al.40, PUFAs seem to have no major effect on the main clinical outcome in MS (disease progression), and does not substantially affect the risk of clinical relapses over 2 years.

PUFA are easily attacked by free radicals. Vitamin E acts as an antioxidant, while vitamin C is involved in the regeneration of vitamin E41,42. Polysaturated fatty acid (PUFA) and antioxidant deficiencies have been observed in MS patients. In our study, MS patients were less nourished than the control subjects, in their intake of vitamin C. Our results are in accordance with Ghadirian et al., who reported in a case-control study that higher intake of foods containing vitamin C was negatively associated with risk of MS.

Antioxidants may support cellular defences in various ways, including radical scavenging, interfering with gene transcription, protein expression, enzyme activity and by metal chelation. Furthermore, antioxidant and PUFA treatment in experimental allergic encephalomyelitis, an animal model of MS, decreased the clinical signs of disease.33

On the other hand, a prospective study of two large cohorts of women reported that intake of vitamin C-rich foods (e.g., oranges and orange juice) was not associated with a reduced risk of MS.44

This study shows a protective effect of higher level of vitamin B1, which may be involved in the regulatory process of the nervous system, or act as an antioxidant. Our results are in accordance with Ghadirian et al., who reported in a case-control study that higher intake of foods containing vitamin B1 was negatively associated with risk of MS.

Our patients had a lower intake of vitamin A in their diet as compared to controls suggesting a protective effect for the vitamin. Other investigators found no protective effect for intake of vitamin A-rich foods.50. Similarly, a prospective cohort study indicated that higher intake of foods containing dietary carotenoids was unrelated to reduced risk of MS.44

In this study, we found that MS patients and controls did not differ in iron values; this is in agreement with other studies, in which all MS patients had iron values within normal limits.45
In this study and in accordance to Melo, we did not find significant differences between zinc levels in MS patients and control. On the contrary, other authors found that mean of zinc level was statistically significantly higher in patients than in the control group (P 0.001). Another study has shown that zinc level is significantly elevated in patients with multiple sclerosis (MS) in between attacks and that this level was dramatically decreased during a clinically documented exacerbation of MS. It has been suggested that mechanisms which govern cellular availability, compartmentalization of Zn, or the binding of Zn to cell surface membranes may be altered in patients with MS, according to disease activity. Difference in the results between studies might be due to the different stage of activity of the patients at enrolment in the study.

In this study, we did not find significant differences between vitamin B2 in MS patients and control. On the contrary, Ghadirian et al. reported in a case-control study that higher intake of foods containing vitamin B2 was negatively associated with risk of MS.

In the present study, MS patients had fewer intakes of carbohydrates than the control subjects. Carbohydrate supplies fuel in the form of glucose to the brain and red blood cells. It is the primary source of energy for all body functions and is required for processing other nutrients. A lack of carbohydrate in the diet can result in fatigue; a common symptom experienced by many MS patients. Adequate carbohydrate in the diet is fundamental to maintaining energy levels and mineral balance and deters the body from reliance on body fat stores and liver and muscle glycogen stores for energy.

Our Ms patients were less nourished than the control subjects, as regard the proteins. Protein, the body's building blocks, is required for growth, repair and replacement of tissue, collagen synthesis, formation of hormones, anti-bodies and enzymes, and is required for excitability and contractibility of neuromuscular tissue. Protein is especially important for patients with MS who are at risk for developing pressure ulcers, or who have experienced unintentional weight loss, resulting in loss of lean body mass. As regard total energy intake our patients had a lower energy intake as compared to controls. This finding is in contradiction to other authors, who found that patients with MS had a higher energy intake especially from animal fats. This difference might be due to variation in the severity of the disease between studies as measured by EDSS with the obvious impact of patient disability and lifestyle restriction on food intake. Other factors that may influence food intake and were not controlled in either study is cigarette smoking and mood disorders.

In the present study it was found that the EDSS correlated significantly and negatively with iron intake (r=-0.427, p=0.019), and vitamin B1 intake (r=-0.366, p=0.047).

In harmony with this relationship investigators have found that levels of plasma iron was significantly lower in hospitalized MS patients with pressure sores.

Other investigators found normal serum level of iron in patients with MS but a higher serum level of soluble transferring receptors (sTFR) assuming possible iron overload in MS brains leading to free radical generation and oxidative damage. In line with the latter finding it has been hypothesized that iron accumulates in MS patients leading to increase superoxide dismutase production. The latter combines with nitric oxide (NO) to form peroxinitrite an extremely powerful free radical that leads to myelin damage.

As mentioned earlier thiamine intake was negatively correlated with EDSS. It is known that deficiency of thiamine may contribute to nerve damage. Many years ago, researchers found that injecting thiamine into the spinal cord or using IV thiamine combined with niacin in people with MS led to reduction of symptoms.

In addition, it has been suggested that nutritional intake influences the risk of patients acquiring MS. Optimal nutritional management is an integral component in holistic patient care that directly impacts on many disease-induced symptoms.

In conclusion, nutritional factors may have a significant role in the etiology, pathology and prognosis of MS. Nutritional intervention is a vital component in the overall management of patients with MS. There appears to be a consensus that following a diet low in saturated fat results in fewer...
exacerbations, slows deterioration, improves energy levels and reduces fatigue.

Polyunsaturated n–6 and n–3 fatty acids may have immunosuppressive and anti-inflammatory effects. Carbohydrate and protein are essential nutrients in maintaining energy levels; reducing fatigue.

We are aware of the importance of vitamins A, C and B1 to metabolic pathways and their antioxidant effect. Despite the current advances in conventional medical treatment, Medical treatment in conjunction with nutritional therapy can result in optimal symptom management for patients with MS.

However, our study has certain limitations; there are some intrinsic limits in the assessment of past diet. Not only are there inaccuracies in recall of past diet by any method but there are large intra-individual variations in diet which make the process of characterizing typical diet for an individual rather difficult. Also socioeconomic factors and religion both of which has their impact on dietary intake were not excluded as confounding factors in this study. In addition certain items e.g. vitamin D, linoleic acid and more specification of food items like vegetable or fruit fibre were not fulfilled. However, this study generally supports a protective role for components commonly found in plants (fruit/vegetables and grains) and an increased risk of high animal food intake. However, further investigations in similar populations are required to confirm which factors consistently contribute to increased or decreased risk of developing MS. This study was somewhat exploratory in nature due to a lack of sufficient published studies in Egypt. A large number of findings did not reach statistical significance due to small sample size, but it confirms a possible role of dietary factors in the causation of MS. With these findings in mind, further studies are required to accurately assess patients’ nutritional requirements during and after exacerbations.

Conclusion

Dietary intake and nutritional factors play a role in the pathogenesis and risk of MS. A protective role for antioxidant and a possible role for fats is suggested.

REFERENCE


54. Stern EL. The intraspinal injection of vitamin B1 for the relief of intractable pain, and for inflammatory and degenerative disease of the central nervous system. Am J Surg 1938; 34: 495.
لم يتمكن العلماء حتى الآن من التعرف على عوامل بيئية مسببة لمرض التصلب المتعدد. وقد اقترح أن أساليب التغذية والعادات الغذائية قد تلعب دورًا في العوامل المسببة لمرض التصلب المتعدد. وتمدّد الدراسة إلى التعرف على العوامل الغذائية في مرضى التصلب المتعدد. وممارستها في مجموعة ضابطة من الأشخاص.

وقد تم دراسة العلاقة بين العوامل الغذائية ومرض التصلب المتعدد من خلال 30 حالة من مرضى التصلب المتعدد و30 شخص من الأشخاص المشاركين في المرضى في العمر والجنس كمجموعة ضابطة، وكان ذلك في قسم الأعصاب بجامعة القاهرة، في الفترة ما بين يونيو 2006، ويناير 2007. وقد تم تدوين المعلومات الغذائية باستخدام طريقة التذكير على مدار 24 ساعة بواسطة تكرار الغذاء.

تبين من المقارنة بين مجموعة المرضى والمجموعة الضابطة النتائج التالية: يتميز مرضى التصلب المتعدد بمناخ طعام أقل من المجموعة الضابطة، وكذلك مستوى أقل من تأثير كتلة الجسم مقارنة بالمجموعة الضابطة. ظهر أن هناك تأثيرًا إيجابيًا بين علاج التصلب المتعدد ومقدار تناولهم للدهون والبروتينات بـ 2.1. لا يوجد فرق إحصائي بين المرضى والجموعة الضابطة في تناول الفيتامينات والكحول والكليوبس "2. وحدت الدراسة في إيجاد علاقة بين تناول الفيتامينات والكليوبس "2. ومواد البروتينات من ماهية وبعض فعالية تأثير الدراسة وجود دور وفائق لتناول مغذيات الأكاسد في مرضى التصلب المتعدد، وارتفاع نسبة الإصابة بالمريض مع تناول الدهون.