SWANK MULTIPLE SCLEROSIS NEWSLETTER

MARCH 1989 NUMBER 36

• FROM THE OFFICE OF ROY L. SWANK,MD.,PH.D.
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Multiple sclerosis: the lipid relationship

Roy L. Swank, MD, PhD, and Aagot Grimsgaard

In this issue of the newsletter we are printing exerpts fr om a recent publication concerning our 35 year study of the relation of fats to M.S. This study was published in the American Journal of Clinical Nutrition, vol. 148, pages 1387-93, 1989. Special written permission was obtained by the American Journal of Clinical Nutrition. American Society for Clinical Nutrition to reprint of this material.

Introduction

A direct relationship of consumption of saturated fat of animal origin to the frequency of multiple sclerosis(MS) was first suggested by Swank in 1950 (1). This was supported by a study of the incidence and nutrition of MS in Norway(2) and by statistical analysis of similar data from many geographical areas by Alter et al (3).

To test this hypothesis Swank undertook an investigation of the effects of a low-fat diet on patients with MS. This study, now in its 36th year, was reported several times, the first time in 1953, the last in 1970 (4-6). It revealed a marked decrease in the frequency of exacerbations of the disease (6), a rate of deterioration about half that observed in untreated cases at the Mayo Clinic by Maclean and Berkson (6), and death rates in patients on low-fat diet much lower than those reported by other investigators (6).

The present study reports the relationship of different levels of fat and oil consumption to the development of disability and the frequency of death. The influence of severity of MS at entry to the study and of delay of starting treatment are also examined.

Subjects and methods

Most MS cases (72%) were diagnosed in the inpatient services of the Montreal Neurological Institute; 22% were diagnosed by qualified consultants in Veterans Administration hospitals in Canada and northern New York State and referred to RLS. Six percent were first seen and evaluated by RLS.

An exacerbating-remitting neurological disease with two or more episodes was required for diagnosis, with evidence from history and examination that the central nervous system had sustained lesions disseminated both in time and space. In all cases the standard diagnostic tests available at that time(pneumograms, myelograms, spinal fluid examination,and electroencephalograms) had failed to rule out the probability of MS and in all but two cases, subsequent clinical events confirmed the diagnosis of an exacerbating-remitting neurological disease, assumed to be MS.

At each visit the patients were interviewed, the clinical state of their disease was evaluated, and the contents of their diets were estimated from oral questions and written records, which the patients prepared for 1-2 wks, before each visit. Initially, patients were seen every 2 wks, then once a month, and finally every 3-6mo until July 1954. During each visit the patients were interviewed and examined and their diets were evaluated. After 1954 they were seen once a year until 1972 and they reported to us by letter every 3-4 mo. They were contacted by phone and letter between 1972 and 1977 and interviewed again on 1977. Since then they have

Description of neurological grades(neurogrades, NGs)

0 Normal performance and normal neurological examination, frequent periods of fatigue, occasional exhaustion.

1 Normal performance physically and mentally,neurological signs present, frequent fatigue, periodic exhaustion.

2 Mildly impaired performace but actively ambulant, neurological signs present, usually working part- or full-time, fatigue and periodic exhaution, occasionally slight memory impairment

3 Severly impaired performance but ambulant; few working, usually part-time; neurological impairment usually widespread; slight memory impairment frequently present

4 Wheelchair, memory often impaired

5 Confined to bed and chair

6 Deceased

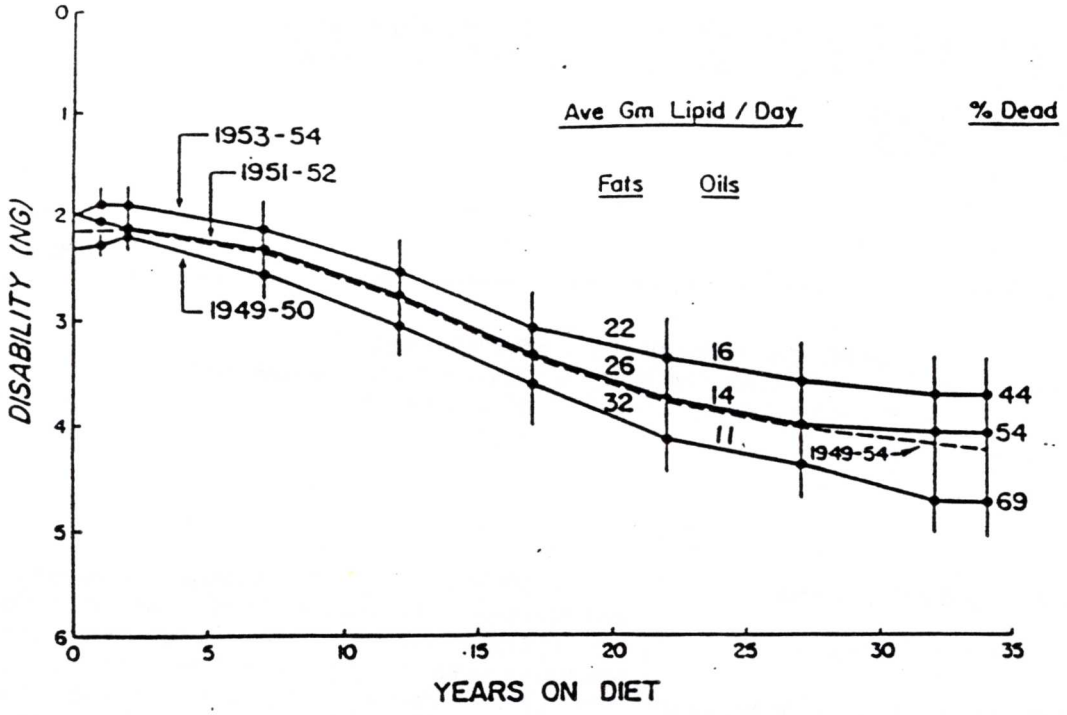


FIG 1. **Patients divided in to groups by date of entry into study. The average rate of progression of the disease and total percentage of deaths at the end of the study for all 150 patients (1949-54) are shown by the dashed line. The same information for the first 54 patients to enter the study (1949-50), next 53 patients to enter the study (1951-52), and the final 43 patients to enter the study (1953-54) are each shown by solid lines. The average intakes per day of fats and of oils are indicated above the solid lines for each group of patients. The vertical lines for each solid line indicate 95% confidence limits.**

reported irregularly by at least once a year by letter, the last time in January of 1985.

The diet

From 1949 to mid-1951 the fat intake, which before dieting had been aprox. 125g/d, was reduced to 20-30g of fat mostly from milk and other animal sources. In 1951 butter fats and hydrogenated oils were eliminated and animal fat were limited to 15 g/d. Five grams of cod liver oil and vegetable oils that remained fluid at room temperature were added and allowed to vary from 10 to 40 g/d as desired by the patients. Margarines, hydrogenated peanut butter, and all shortenings were disallowed. The diet contained from 60 to 90 g of protein mostly fish and seafood, whitemeat of chicken and turkey cooked with the skin removed, skim milk, and all lean meats, and occasional egg, vegetables, cereals, and nuts. The fats and oils in all meat items and eggs had been accounted for. Fats are defined as those lipids containing fatty acids that are solid at room temperature (20-22 C) and below. They are mostly of animal origin but the lipids in coconuts and palm oils, which are usually referred to as oils, are actually fats by our definition and are excluded from the low-fat diet. The oils that are unsaturated are vegetable in origin except for fish oils. All oils are fluid at room temperature and many (the polyunsaturated) are also fluid at refrigerator temperature. As the study progressed commercially processed foods and pen fattened beef had to be monitored to prevent unwanted increases in fat intake. Carbohydrates were consumed as needed to meet the energy requirements of the patients.

Results

The average progression of the disease in the 150 patients is indicated by the dashed line in figure 1. The duration of disease before the study was 6.2±.6y and age was 34.1±8.9y. Eighty females and 70 males entered the study. Six patients were lost and were not used to determine the percentage deaths. During the study 81 patients(40 males and 41 females) are known to have died (56%) and the neurological deterioration increased from a NG of 2.2±.92 to 4.3±2.1. The fat intake for the group was 26 ±12g and oil intake was 13±7g. The fat intake increased from 23±12 to 31±18g from beginning to end of the first 22y. The oil intake remained stable at 13±7g/d during the same period.

Influence of the year of entry to the study on disability and deaths

The 150 patients were divided into three groups of 54, 53, and 43, respectively, depending upon the time of entry to the study, 1949-50, 1951-52, and 1953-54. The rate of deterioration and percentage deaths in each group at the end of the study are shown in Figure 1. The first group(before-study duration of MS 6.5±6.0y)suffered the most rapid deterioration and highest percentage deaths, the last group(before-study of MS 5.1±5.8y) suffered the

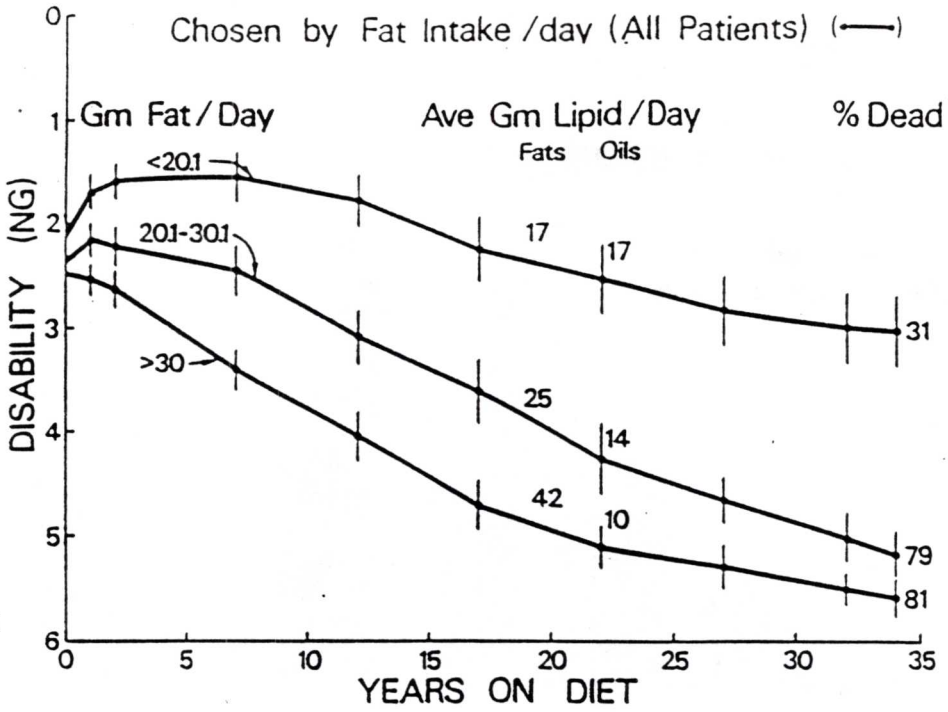


FIG 2. **Patients grouped by fat intake per day: average rate of deterioration of MS with 95% confidence limits(vertical lines) and percentage of deaths at the end of the study for three groups of patients (solid lines). The average daily fat and oil intakes are indicated above each solid line, and percentage deaths at the end of each solid line.**

lowest average deterioration and lowest percentage deaths. The before-study duration of the intermediate group was 4.7±6.7 y.) The clinical deterioration and deaths correlated directly with the average daily consumption of fat with a statistical significance $p < 0.01, 0.01$, and 0.05 for the three groups, respectively, and the duration and severity of disease before entry to the study with a significance of $p < 0.05$. The correlation was indirect for oil consumption.

Influence of fat intake

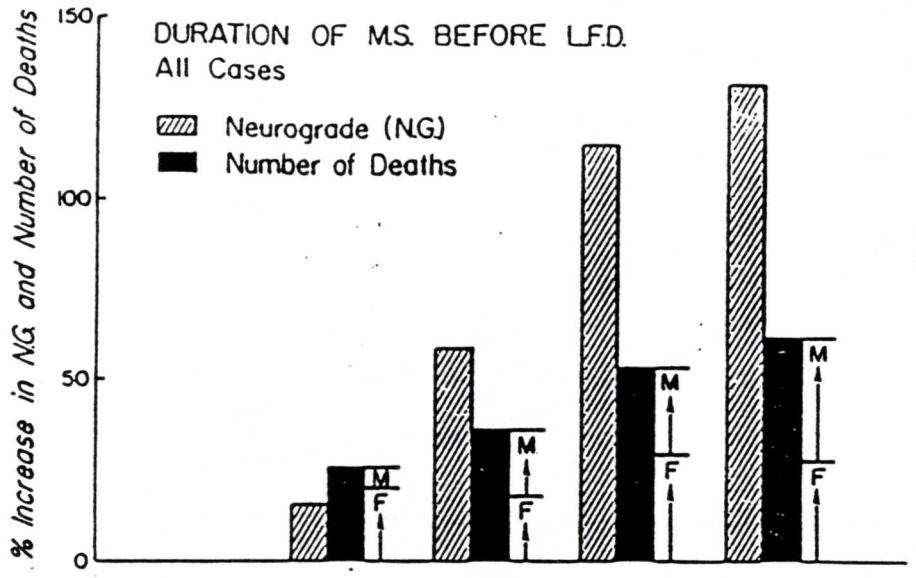
The close direct relationship of deterioration and of deaths at the end of the 34-y study to the amount of the fat intake in the 150 patients is shown in figure 2(solid lines). Note the sharp increase in mortality and morbidity that attended the increase in fat from 17 to 25g. Seventy patients (26 men and 44 women) consumed less than or equal to 20 g fat/d(17±2.0g). Thirty-one percent died (7 men and 15 women). The average deterioration was slight,indicated by the NG increase from 2.22±1.1 to 2.9±2.3 (or 0.7). Thirty-seven patients (19 males and 18 females) consumed 20.1 - 30.1 g fat/d (25±3.0g). Seventy-nine percent died (13 females and 16 males) and a sharp increase in average NG from 2.4±0.8 to 4.8±1.3(or 2.6) indicated a marked average neurological deterioration. Thirty-seven patients(20 males and 17 females) consumed more than 30 g fat/d (42±6g). Eighty-one percent died (17 males and 13 females) and the NG increased from 2.5±0.7 to 5.5±1.7 (or 3.0). Chi-square tests between deaths and average fat intakes from each group were significant ($p < 0.001$). The average duration of MS before entry to the study were 4.8±5.3, 6.7±6.7, and 8.0 ±6.7y, respectively, for the three groups of patients.

Sixty-six patients had MS less than 3 years upon entry to the study. Thirty-five consumed less than or equal to 20 g fat/day, NG increased from 2.0±1.1 to 2.3±2.0, and 21% died. Fourteen consumed between 20.1 and 30.1 g fat, the NG increased from 2.4±0.8 to 4.4±1.5, and 50% died. Seventeen consumed greater than 30 g fat/day, the NG increased from 2.3±0.75 to 5.3±1.7,and 75% died. The average fat and oil intakes of the patients in each group were similar to the consumption of fats and oils by the corresponding groups, which included the entire 150 patients shown in Figure 2. Eleven patients, each of whom had MS less than 2 years and an initial NG of 1.0, consumed less than or equal to 20.1 g fat/day(average 18g). They experienced but one death (9%) and exhibited an average change in NG from 1.0±0.0 to 0.90±0.53 during the 34-y period.

Although most patients consuming greater than 20 g fat suffered significant increases in disability and deaths, six patients (three female and three male) behaved differently. The average NG in this small group(7%) on entry to the study was 2.2±0.75. During the course of the study, two patients remained the same at NGs 2.0 and 1.0, one deteriorated from 2.0 to 3.0, and three improved, one from 2.0 to 0.0 and two from NG 3.0 to 2.0(average change 1.7±1.0) The average fat intake of the six patients was 38g/d/ and the oil intake was 13 g/d.

Influence on the disease of delays in treatment

In a previous paper (6) it was observed that delays in treatment tended to decrease the benefits of low-fat diet in



Duration of M.S. (yrs)	0-1	0-2	2-5	>5
average	0.39	0.82	3.5	11.5
Number of cases	31	50	37	63
% Female	64	56	57	49
% Male	36	44	43	51
Average age before L.F.D.	30.0	30.5	30.6	38.9
Average fat/day (gms)	23.1	24.8	24.0	29.6
% N.G. 1 + 2	64	64	80	44

FIG 3. Duration of MS before low-fat diet: number of deaths and the percentage increases in disability that occured in patients whose treatment by low-fat diet was delayed. The relative number of female and male deaths are shown to the right of the death (black) columns by vertical arrows. Patients in the 0-1 columns are included in the 0-2 columns.

MS. Additional support of this view is presented in figure 3. The number of deaths and severity of deterioration increased in direct relation to the duration of disease before diet. The average age of patients and average consumption of fat per day were similar in patients in whom delays in treatment were 0-1, 0-2, and 2-5 y, yet the mortality and morbidity steadily increased. Note that there was a preponderance of minimally disabled patients (NG 1 and 2) in the 2-5 y delayed-treatment group yet there was an increase in deaths and deterioration in this group consistent with the belief that early diagnosis and treatment are important to maintain the well-being of patients with MS. Those in which the delay in treatment exceeded 5 y showed an even greater morbidity and mortality; this could have been partly due to the increased age and fat intake of this group and possibly to the reduced number of females in the group. Correlations between death and deterioration (NG change) and duration of disease before entry to study were significant(p0.001).

Effect of gender on course of disease

The speed of deterioration and the percentage of deaths were slightly greater in males than females. Females also tended to eat slightly less fat than males. Females in all NGs appeared to tolerate MS better than males did but chi-square and t-test results of $p < 0.20$ to $p < 0.30$ indicated weak statistical significance. The differences in fat intake also were of a low order of significance ($p < 0.20$)

Influence of oil consumption

Oil intake tended to be indirectly(inversely) related to the intake of fat and to the rate of clinical deterioration and percentage dead at the end of study (fig 1 and 2). Correlation coefficients of oil to fats increased gradually from the relatively weak indirect correlation of -0.24($p < 0.02$) during the first 2 y on the diet to a stronger indirect correlation of -0.62($p < 0.001$) during the 22nd year.

Causes for death

One hundred forty-four patients were followed to death or to the end of the study. Eighty-one (56%) died. The causes

ne disease, such as urinary tract or pulmonary infections; 12 (3%) died from vascular disease of heart or brain; 7 (4.8%) died from cancer of bowel, breast, or prostate gland; and 3 (2.0%) died from MS complicated by tuberculosis, alcoholism, or a major operation.

If the non-MS deaths were excluded from consideration, the percentage deaths for those eating less than or equal to 20 g fat/day would be 20% instead of 31% (fig 2) and those eating greater than 20 g fat/day, 62 instead of 80%. Because of the uncertainty in patients with disabling MS, deaths from all causes were included in the calculations of the percentage of deaths in this paper.

Discussion

Although the fat intake varied, our patients consumed significantly less saturated fatty acids than the approx. 125 g/d that they had been accustomed to. The entire group, and particularly those who consumed less than or equal to 30 g fat/day, experienced marked decreases in the frequency of exacerbations(6). It was, therefore surprising that only those consuming less than or equal to 20 g fat/day (average 17 g) suffered little deterioration and few deaths. Those who consumed an average of either 8 or 24 g fat more than this exhibited sharp increases in deterioration and in the number of deaths. This suggests that with few exceptions, MS patients are very sensitive to or intolerant of saturated fatty acids and that a high animal- fat intake may be an important factor in the mechanism of the disease. The implication of this should not be overlooked in the dietary treatment of MS. To assure the best clinical results, fat intake must be kept very low, in our experience less than 15 g/d exclusive of fractional fats, which should not exceed approx 5 g/d.

The six patients (7%) who consumed greater than 20 g fat/day and who failed to demonstrate this high degree of sensitivity to consumed fat may be benign cases of MS similar to those reported by MacKay an Hirano (10) and by Lehoczky and Holasy-Lehoczky (11). The latter investigators estimated that approx. 4% of their 2000 cases fell into the benign category.

Duration of disease before treatment also influenced the outcome of the disease in our patients. Both males and females who started on diet soon after onset of symptoms did better than those who started later (fig 3). Although both sexes were influenced in this way, females appeared to deteriorate more slowly than did similarly disabled males, possibly because the females tended to consume less fat than the males did. In addition, the outlook for severely disabled patients upon entry to the study was previously shown to be worse than for those with minimal disability (6).

The oil intakes bore an indirect relationship to the rates of deterioration and number of deaths as well as to the fat intakes. This appears to be consistent with the suggestion of Sinclair (12), supported by Thompson and collaborators (13-15), that the casual factor in MS is a relative deficiency of essential fatty acids rather than consumption of an excess of saturated fatty acids as suggested by Swank (1). Swank believes that the oils were beneficial to the MS patients, and made it easier for them to follow the low-fat diet by broadening their choices of foods. The oils seemed to make patients feel better and increased their energy but it is doubtful that oils were essential to their health. This belief is supported by the relatively low consumption of oils during the first few years of dieting, indicated by the weak negative correlation coefficient of oils to fats. Moreover, during the first 2 y no oil was added to the low- fat diet yet the exacerbation decreased 80% (from one attack per person per 1 y to an average of one attack per person per 5 y)(Fig 1)(6).

Large animal-fat (saturated fatty acid) meals are known to cause aggregation of blood cells, slowing of the circulation and a reduction in the oxygen available in the brain (20-26). This tendency to detain blood in the microcirculation could cause acidification of surrounding area, activate intracelluar lysing enzymes, and thereby increase the permeablility of the blood- brain barrier. Toxic components in normal as well as MS plasma (27-31) could permeate to the surrounding neurons as previously suggested (6, 9, 32, 33) and increase the tendency for their destruction. The narcosis and occasional increase in symptoms and signs of exacerbating MS cases after infusions of normal plasma would appear to be consistent with one step of such a series of events (32).

Summary

Although patients in the 35 year study do well on the very lowest fat intake (17 grams & below) we have found that most patients have more energy if the saturated fat intake is kept below 15 grams per day. Some patients are even more sensitive to fat and must keep their fat intake below 5-10 grams daily to maintain the highest evergy and stamina level. During the first year on diet we advise patietns to consume less than 10-15 grams of fat daily. This seems to result in a more rapid response to the diet. When under stress or experiencing symptoms we advise patients to reduce their saturated fat intake to less than 10 grams daily.

*Bibliography not included : see Am. J. Clin. Nutr. vol.148, pages 1387-93, 1989.

Vitamins

If you have a lactose intolerance or unable to tolerate milk products for other reasons the following foods will help meet your Calcium requirements. Broccoli, Brewer's Yeast, spinich, green leafy vegetables, kale, beet, turnip, collard and mustard greens, filberts, salmon, soybeans, tempeh, tofu, and watercress. The RDA for most adults is 800 milligrams daily. Postmenopausal women require an increase to 1200-1500 milligrams daily. Older people require 800 milligrams daily and in some cases this may be to low. Many times older people will have difficulty digesting milk products and will have to rely on supplements. A multiple vitamin is usually not a large enough supplement. An additional 400 milligrams may be necessary. Caution should be taken not to exceed 1200-1500 daily because of the possiblility of producing kidney stones. Plenty of water should be consumed daily, 6-8 glassees, when taking a calcim supplement to guard against the risk of stones

The office will be closed from March 18th until March 27th Emergency calls will be answered.

THE OREGON HEALTH SCIENCES UNIVERSITY

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June 1, 1989

Dear Reader,

I have been involved in the "enigma" of M.S. half my life, since 1948. Three avenues of research have been pursued , the precipitating cause of the disease, the part played by the inherited metabolic fault , and the mechanism or genesis of the disease. This year we finished our investigation of the principle precipitating factor, high fat consumption, and have published the first of two papers with Aagot Grimsgaard on the relation of fat consumption to the disease (see newsletter # 36). A second paper with Barbara Dugan is in the process of publication. The "bottom line" from these studies is that the low fat diet arrests the progress of the disease in all but very few patients. Because of this it should be obvious that early diagnosis and institution of the low fat diet is important, if not imperative, if the best possible result is to be obtained. The other two aspects of our research have also been in progress since 1949-50. I believe that both have advanced to a stage which justifies a report to our readers so that they will have a better perspective of the overall thrust of our efforts.

In August of 1948, I began my studies in the library of McGill University in Montreal. At the same time I interviewed all the M.S. patients which visited the outpatient clinic. At the end of 4 months I had learned two important things. First, the frequency of M.S. was high in high fat consuming nations, and low in low fat consuming nations of the world, and second that in a number of patients the exacerbations or attacks of M.S. often developed abruptly much like cerebral strokes. By January 1st, with the help of Miss Eddy from the Nutritional Department of the Royal Victoria Hospital, I put together a low fat diet. Soon after, Aagot Grimsgaard joined me and with further help from Miss Eddy she assumed responsibility for the patients' diet. By the first week in January the clinical side of our study was underway, we had started to place patients on the low fat diet. By 1951, 2 years later, oils were added to the diet including cod liver oil (CLO), and fats were reduced to their present level of 15 grams/day. Since then the diet has remained unchanged.

One might ask why the fat intake was so low and why we added oil. We reduced the fat intake as low as we considered possible and at the same time practical. I now consider ourselves fortunate that we did. Oils were added for the essential fatty acids which are like vitamins and cannot be fabricated by the body. They need to be supplied. Also, the oils make dieting easier and tastier. You know the story from there on.

In April of 1949 I went to Europe to observe and study the effects of food shortages in occupied countries during the war. In Norway and Denmark the fat consumption was reduced more than 50%. In both countries the occurrence of new cases(incidence) of M.S. decreased. After the war when the fat intake in Norway returned to "normal" the incidence of M.S. more than doubled. These and other observations convinced me that the low fat diet

was an important cause of M.S. I also started an epidemiological study in Norway with Julie Bakker, Ola Lerstad and Axel Strom which gave further support for treatment with low-fat diet. Subsequently, Milton Alter and coworkers demonstrated a strong direct correlation between the frequency of M.S and the fat intake.

While in Europe I thought a good deal about the underlying reasons why saturated fats did, and unsaturated fats did not cause M.S. and formulated plans for more basic research. After returning home in July 1949, we fed dogs and humans large butterfat (saturated fat), and vegetable oil (unsaturated fat) meals and observed the changes which occurred in the blood microscopically with dark field illumination. Several hours after saturated fat meals the visible fat in the blood(Chylomicra) and the red blood cells began to clump (aggregate). These changes in red cells became maximum 6 to 9 hours after the meal when the chylomicra were disappearing. The red cells returned to normal about 12 hours after the meals. After inunsaturated oil meals these aggregations were absent.

Next, with Chester Cullen, I examined the living circulation in the cheek pouch of the hamster after butter fat meals. Beginning 3 hours after the meal the red cells started to clump and 1 to 2 hours later the circulation was remarkably slowed and often stopped. At 12 hours recovery would start and the following day the circulation returned to normal.

In Oregon these fat feedings experiments were continued. In hamsters we measured the oxygen content of brain after fat meals and found it reduced. After oil meals or after meals of protein or carbohydrates little or no change occurred in oxygen in the brain. The speed of the blood flow was measured and found to be slow after fat meals, and hamsters developed convulsions and abnormal electrocardiograms. We also examined the effects of infused large molecular weight substances and observed the same aggregation phenomena and occasionally paralysis in dogs, and a breakdown of the blood brain barrier in hamsters. Injection of serotonin also caused red cell aggregation and a breakdown in the blood-brain barrier in animals.

From these studies the following hypothesis was developed. The aggregation of red cells and slowing of the circulation lead to a spotty arrest of the circulation to the brain, and subsequent acidification of surrounding tissues. Breakdown of the blood- brain-barrier, leakage of toxic substances in the blood to the surrounding nerve tissues, and destruction of brain tissues follows. Since only a small percentage of the population of the U.S.A get M.S.(The maximum circa 0.2%) one must assume that there must be something unusual about people with M.S. This unusual property could be the presence of something toxic, or some important factor which is missing. Our studies indicate that something is missing. In other words there is a deficiency in the blood in this small group of patients. Since 4 to 6 % of patients with M.S. have blood relatives with M.S. it is assumed that the missing substance (deficiency) is inherited.

A search for this abnormal component or factor was begun in the early 1950's with Professor Quastel & Franklin using two-dimensional chromatograms. A year later I attempted to correlated chromatogram changes with neurological findings. In both studies chromatograms of normal subjects and M.S.

patients differed. At the time we were unable to carry this project further. Nearly 30 years later, using the red cell mobility test , well known to all of you, a new start was made. With Dr. Seamen, Cherry Tamblyn an Chip Zukowski, electrophoretic mobility of red cells was shown to be slower in M.S. patients than in normals. Furthermore, the difference was shown to be due to differences in the plasma rather than to red cell membranes. Red cells & even plastic beads incubated in normal plasma were more mobile than when placed in M.S. plasma. Finally when normal plasma was infused into M.S. patients whose red cell mobility was very slow, within hours the patients' condition improved, and the mobility returned to normal. This indicated that there was a deficiency in M.S. plasma which was temporarily restored to normal by infusion of normal plasma. Having shown that M.S. plasma is lacking one or more components it remains only to identify this or these factors.

In the past year Roy Garvin and I have been studying normal and M.S. plasma using standard techniques to separate the plasma protein components. We believe that we have shown that M.S. and normal plasma differ in several respects. It will be some months, however, before we will be able to identify the differences if actually they are present. If we are successful we will have to integrate the low fat diet, the aggregation of blood cells by fat meals, the missing plasma components and the pathological lesions with the symptomatology.

In addition there is the problem of early diagnosis, Tony Leckband is studying the streaming potential of plasma in M.S. and normal subjects. If the differences we suspect are present this test might assist in early diagnosis of M.S.

In general I feel that our work has reached a critical stage. The one item short in supply which is necessary to get us through this phase of our problem is adequate financing. To this end we are now soliciting financial assistance from those of you able and willing to help.

Sincerly yours,

Roy L. Swank

Roy L Swank , M.D., Ph.D.

RESEARCH FUNDING

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SWANK MULTIPLE SCLEROSIS NEWSLETTER

JUNE 1989 NUMBER 37

• FROM THE OFFICE OF ROY L. SWANK, MD.,PH.D.
• EDITOR: BARBARA BREWER DUGAN ASSISTANT EDITOR: BARBARA KALKHOVEN
• DESIGN COORDINATOR: ANTHONY J. LECKBAND

TRIBUTE: L.J. JACKSON

It is with great sorrow that we acknowledge the passing of Lavelle Jackson. Lavelle was a member of our reseach team for 30 years. He directed the Biocolloid research lab at Providence Hospital and was an intergral part of our research program. His expertise and assuring personality will be missed by many of you.

URINARY AND BOWEL PROBLEMS

FREQUENCY AND URGENCY OF URINATION. Leakage and loss of bladder control are common complaints of the M.S. patient particulary in patients with moderate to severe disability. These complaints may accompany an exacerbation or appear during stress and fatigue. They can also develop as a result of a bladder infection, or during exposure to heat. Women entering menopause may also experience these symptoms.

URINARY INFECTIONS are more common in female than male M.S. patients. The relative high frequency in females is often the aftermath of pregnancy and normal delivery of a baby. This event causes a relaxation of, and separation of the urogenital(pelvic) diaphram upon which the bladder rests. As a result the bladder sinks lower in the pelvis and urine tends to remain pooled after urination. Bacteria are often present in urine and if the urine is retained infection has an opportunity to develop.

PREVENTION AND TREATMENT: The first line of defense is to empty the bladder with each urination. This is facilitated by bending forward while pressing on the lower abdomen from the beginning to end of urination. If, the patient feels the urge to urinate again and does so it is a sign that the bladder was not emptied the first time. To check for residual urine (amount retained) measure the amount of retained urine, which can be obtained by catheterization after urination. This procedure can be done by a Urologist. If the amount of residual urine is more than 4 oz (100 cc) urinary infection is apt to occur. An increase in water consumption to at least 8 glasses daily and the addition of 1 glass of straight cranberry juice and Vitamin C 500-1000 mgs. daily helps to prevent infections. If infections are frequent and these preventative measures are not successful it may be necessary to catheterize oneself 2-3 times daily. This technique can be learned from a Urologist or Public Health Nurse. The procedure is not difficult for females unless coordination and normal feeling are seriously impaired. Self catheterization if done carefully and with reasonable cleanliness can be continued for years without causing infections. This not only helps alleviate bladder infection but can also be used in situations where bladder control is a problem.

IF INFECTION RESULTS antibiotic treatment will be necessary for 7 to 14 days. The urine should then be free of infection. If infection recurs it is advisable to continue the medication one day a week for six months. An alternate method is to take one-half the daily does of antibiotic for extended periods of time. I favor the former method for most M.S. patients. If at anytime infection seems to be developing, (burning on urination, increasing frequency and urgency of urination or fever has developed) start the daily routine of antibiotic treatment for two weeks. Before starting this treatment have a urine sample examined, but do not wait for complete analysis and bacterial sensitivity test before resuming therapy. If treatment is delayed, much longer treatment will be required. It is assumed that the patient will know from past experience that he or she is not allergic to the antibiotic and that previous infections have responded to the drug. If the infection does not respond in 1 week, one can shift to another antibiotic, one in which the bacteria were found sensitive. This can be determined from the test of the original urine sample.

MEDICATIONS: As a rule, infections respond to Ampicillin 500 mgs or Septra DS twice daily for 10 days. If not effective or if the patient is allergic to these medications there are a host of other antibiotics available on the market, one of which can be prescribed by your physician. Ditropan is commonly prescribed for frequency and urgency of urination. It is currently the most effective as well as practical drug for this symptom. It is dispensed in a 5 mg. tablet. The usual requirement is one tablet twice daily, the maximum is one tablet 4 times daily. I suggest that this medication be used only when bathroom privileges are inconvenient, such as when shopping or traveling. Ditropan will cause your mouth to be slightly

dry. There are other medications such as Banthine, Probanthine and related drugs for urinary frequency, but as a rule Ditropan is the drug of choice in M.S.

CONSTIPATION AND OTHER BOWEL CONCERNS can be a problem for patients. When the problem of constipation is moderate an increase in water intake to at least 8 glasses daily and a moderate increase in bran will often correct the problem. When these measures fail one can add the laxative described on page 59 of the 1987 book*. This usually leads to success especially if the water intake is also increased. As an alternative one can eat 4 to 8 dried prunes each night. The prunes contain a natural laxative substance. This is a simple chore and pleasant if one likes prunes. Finally if the stools are dry and hard, 1 tablespoon of mineral oil at bedtime will help. Excessive use of mineral oil can affect the absorption of the fat soluble vitamins A,D,K, and Carotene, therefore, it should be used only occasionally or for short periods of time. Regular use of prescribed or over the counter laxatives is discouraged because it can lead to a dependency. If many hard round stools are passed, and particularly if crampy pain is experienced usually in the left lower part of the abdomen one must consider the possibility of spasms in the lower bowel which hold up the passage of the bowel contents. This leads to drying of the stool and formation of nodules. This can often be relieved by medications which relieve the spastic colon. Tincture of Belladonna 10 to 20 drop in a small amount of water at bedtime often relaxes the obstruction and leads to success. If not increase to 30 drops the next night. There are other bowel relaxing medications, but Belladonna is inexpensive and safe. Expect slight dryness of the mouth.

* The MS diet book by Swank & Dugan, publ. by Doubleday & Co.

SEIZURES

Epileptic seizures with loss of consciousness, seizure activity, and an abnormal electroencephelogram (EEG) are rarely seen in M.S. patients. However, very short seizures without loss of consciousness, with normal EEG's occur in more than 5% of patients. The most common type of seizure consists of a sudden mental lapse lasting 1 to 3 seconds during which the patient cannnot speak and does not move. It may occur during a conversation and usually is not detected by the person being spoken to. The EEG will be normal even during the attacks. They may occur on occasion or frequently during a single day(as often as 25 times/d) and they continue to recur periodically for up to six months. Needless to say these seizures are upsetting to the patient even though they are not detected by others. A mental lapse seizure could be described as a brief aura which fails to progress to a classical seizure.

Occasionally a slight movement of an arm, or an abrupt feeling of weakness in the legs may accompany the seizures. However, they are recovered from quickly. Rarely have these brief seizures occured for longer than 6 months. Successful treatment has been Dilantin 100 mgs. three times daily. This treatment can be continued for 6 months then slowly discontinued. These brief "seizures" may recur later, but this has been rare.

TRIGEMINAL NEURALGIA (Tic Douloureux)

Is a painful affliction of one side of the face. The pain is lancing (knife like) and very severe, and occurs spontaneously or when certain areas of the face or gums are touched. Patients with this pain are rendered helpless during attacks of the pain. Repeat attacks of the pain may occur and last for weeks or months and then disappear for variable periods. The treatment of choice is Tegretol 200 mgs. 3 to 4 times a day. In a few cases some relief is obtained with Dilantin 100 mgs. 3 to 4 times daily and also with Lioresol 10 mgs. 4 times daily. During treatment with Tegretol blood test including liver function test should be monitered every 3 to 4 weeks for harmful effects. When the pain subsides as it usually does the medications should be dicontinued until the next attack.

If Tegretol and other medications are not effective surgery can be considered. Several procedures are available. The nerve roots for the painful area are interrupted between the brain stem and the tigeminal ganglion. Tic Douloureux occurs in patients with M.S. and without M.S.. In M.S. the pain is often atypical. Therefore, it is wise if surgery is considered to employ a surgeon who has considerable experience with the condition. A related condition glossopharyngeal neuralgia rarely occurs. The pain from this condition occurs on oneside of the head invlving the throat and ear. The medical treatment is the same as for trigeminal neuralgia. The surgical treatment involves the roots of the glossopharyngeal nerve.

Both neuralgias are extremely painful. The pain is sharp and shooting and disabling when present. Sometimes this is misdiagnosed and teeth will be extraced unnecessarily.

DIETARY CONFUSION ??

For those patients who have not been seen in our office it is important to not confuse the Swank low fat diet with the National Heart Association Diet. The Swank diet is considerably lower in saturated fat. Although the National Heart Association Diet may reduce the frequency of exacerbations it fails to significantly retard or stop the progression of disability in M.S. patients.

ANSWERS TO YOUR QUESTIONS

1) Why do you not recommend the flu vaccination for M.S. patients ?

Patients with M.S. rarely have flu, and when they do , it's usually mild. Occasionally a patient will have a severe attack, but in our experience total recovery with no added disability results. The flu vaccination will often cause mild flu-like symptoms, therefore the patient who receives the vaccination will then suffer these symptoms unnecessarily.

2) What does Dr. Swank recommend for spastic legs?

Mild sedation in mild cases and in the more severe cases, Lioresol may be prescribed. Often leg exercises in the morning and massage will gain satisfactory results.

3) Is Physical Therapy beneficial to M.S. patients.?

Mild physical therapy can be helpful, but aggressive physical therapy can be very harmful. Active exercise should be stopped just before fatigue sets in. If this rule is followed gradual increase in strength can result.

4) Is it harmful to have more than 10 tsp. of oil (from fish, nuts, or vegetable oil) daily ?

In any unsaturated fat there is a small amount of saturated fat such as in pure vegetable oils. When a patient routinely exceeds 10 tsps. of oil daily they begin to accumulate a few extra grams of saturated fat. If your saturated fat intake is very low (0-5 grams) occasionally this will not hurt you. However, if you are reaching 15 grams daily and also have exceeded the 10 tsp. of oil daily you will exceed the 15 grams of saturated fat allowed.

MISC. OFFICE NEWS

Barb Kalkhoven will be on vacation the last 2 weeks in July. Dr. Swank and Barbara Dugan will be on vacation from August 7th to September 5th. the office will be open during this time. Bon Voyage' !!

Please remember to notify the office of any address changes or new telephone numbers. Also, if you are receiving 2 newsletters let us know.

We are beginning to put together a list of patients who wish to commicate with each other. There is still time to add your name. Send in an index card with your name and address, signed by you to serve as a release, with any other information that you would like to include.

Please remember that checks for cod liver oil are to be made out to O.H.S.U. Foundation Swank MS Research. Picked up in the office the cost is $14.00, mailed the cost is $16.50.

A reminder: We "respectively" ask that receivers of our newsletters make a $20.00 donation on a yearly basis. We spend a great deal of time on the newsletters to prepare and mail them out. You may have noticed that the format and design is different. New computer equipment has made the newsletter easier and more enjoyable to read. The unfortunate aspect to this is that our costs have risen for preparing the newsletter. The donation would be most appreciated. Checks are to be made out to O.H.S.U. Foundation Swank MS Research. Thank you!!

RECIPE

Vegetarian Enchiladas

1/2 cup Bulgar wheat

1/2 cup hot water

1 medium onion, chopped

1 green pepper, chopped

1 clove garlic, minced

1 8oz. can tomato sauce

1 tsp. salt

1/2 tsp. oregano

1/2 tsp. ground cumin

1/4 tsp cerly seed

3 cups cooked black beans or pinto beans

Sauce

2 15 oz. cans tomato sauce

1/2 tsp. ground cumin

Topping

1/2 cup sliced olives or sliced tomatoes or Soya Kaas cheese.

1 dozen corn or flour tortillas

Soak bulgar wheat in hot water until the water is absorbed. Saute' onion, green pepper & garlic. Add the bulgar wheat, tomato sauce, & seasonings. Simmer 15 minutes. Add cooked beans. Stir, then simmer 10 minutes to blend flavors. Test taste for seasoning.

Spread one-half tomato sauce in bottom of 9" x 13" pan. Place approximately 1/4 cup filling on each tortilla. Roll up and place, seamed side down, in pan. Top with remaing sauce.

Top with sliced olives, sliced tomatoes, and Soya Kaas cheese.

Bake at 350 degrees for 30 minutes, uncoverd.

Fat - Soya Kaas Cheese 1.3 grams per ounce

Oil - 2 tsp. per serving

SWANK MULTIPLE SCLEROSIS NEWSLETTER

OCTOBER 1989 NUMBER 38

• FROM THE OFFICE OF ROY L. SWANK,M.D. Ph.D.
• EDITOR: BARBARA BREWER DUGAN ASSISTANT EDITOR: BARBARA KALKHOVEN
• DESIGN COORDINATOR: ANTHONY J. LECKBAND

THOSE UNEXPLAINED FALLS

If you have experienced an unexplained fall or difficulty picking your foot up when climbing stairs you may be developing symptoms of "Foot Drop". Patients whose legs are weak or become weakened following extensive exercise, physical work, or times of increased nervousness and agitation may notice a tendency to "stub the toes". This symptom may be more noticeable when walking on thick carpets, climbing stairs or walking up a slight incline. Avoiding fatigue, which promotes increased leg weakness, will help reduce the weakness. If the weakness continues and falling develops support for the foot is necessary. If the problem is ignored serious injury can result from falls and injury to the ankle and foot. The trauma of a fall can also exacerbate the disease.

The suggestion of a leg brace may at first shock or depress the patient. However, it has been our experience that those patients experiencing these symptoms are greatly relieved after trying the brace. If the foot drop is minor an elastic ankle brace may be adequate. If you are falling and the toe of your shoe looks constantly worn more support will be necessary. A small, light weight plastic brace which fits into the shoe can be purchased at an orthopedic brace shop. This may require a prescription from your physician. This type of brace is secured to the back of the leg below the knee and bends forward at a right angle to support and slightly elevate the foot. This also raises the toes an prevents them from dragging. The brace adds support to each side of the ankle to prevent it from turning. The risk of falling is greatly reduced with this brace and walking becomes much easier.

HOW DO YOU FEEL ABOUT USING A CANE?

It is not our policy to suggest to patients the use of a cane unless it necessary. Most patients shy away from a cane thinking that it draws attention to their disability. On the other hand we have had many patients tell us they have been thought of as "drunk" because of their gait. Which draws more attention? If you are having instability and tend to stagger when walking, a cane may be very helpful. It will increase your stability and lessen the chance of a dangerous fall.

HYPEREXTENSION (BUCKLING) OF THE KNEE

When there is weakness of the legs there is an increased tendency for the knees to buckle backward(hyperextend). Eventually you may begin to feel the knee snapping back with each step. If this is allowed to continue the hyperextension will increase and can become a serious and painful problem. As soon as this effect or change is recognized, either by yourself or others, a tight elastic knee bandage, similar to what athletes often wear, may be applied to the knee for support. This will not completely stop the backward thrust of the knee, but will lessen the trauma and the wear and tear on the knee joint, muscles, tendons, and ligaments which support the knee and hold the leg and thigh bones together. If the hyperextension becomes very severe,and if ignored, injury to the knee can result. With a prescription from your physician an orthopedic brace shop can fit you with a supportive brace.

WATCH THOSE POUNDS

It is uncommon to see sudden weight gain in patients who are following diet closely. During exacerbation of disease the patients may first lose as much as 10 pounds and then during the recovery period gain back what they have lost plus a few additional pounds due to inactivity.

Those extra pounds not only increase existing symptoms such as leg weakness and instability ,but can produce joint problems down the road. It goes without saying that fatigue is more pronounced in patients who are overweight.

If you are unable to lose weight on your own, programs such as Weight Watchers, Weight Loss Clinic, and Tops are safe and effective. All of these programs will first contact your physician before initiating treatment. The diets used are low fat and can easily be adapted to the Swank diet.

If you choose to lose weight on your own we suggest the following suggestions.

1. Record your diet without making changes for one week.
2. Estimate the amount of calories consumed.
3. Reduce your caloric intake to 1,000- 1,500(women) 1,500- 2,000 (men). It may be necessary to reduce the caloric intake below these levels because of reduced basal metabolic rate due to inactivity.
4. Decrease your unsaturated fat (oil) intake to 4 teaspoons daily.
5. Decrease your portions of meat to 3 ounces.
6. If possible begin an exercise program 3 X per week.

DEFINITIONS:

Kilocalorie (kcal)- is the standard unit for measuring food energy. It is also referred to as Calorie(Cal).

One pound of fat is equivalent to 3,500 Calories. A reduction of 500 Calories per day will produce a weight loss of one pound per week.

Physical inactivity may be a major factor in obesity. Due to fatigue many patients are unable to do routine exercise. If you are not having activity of disease an exercise program developed to fit your needs 3 X per week is recommended. Refer to newsletter #34. Exercise will decrease body fat but at first may not decrease body weight. Initially exercise will increase muscle mass, which is more dense than fat, and your body weight may not change. A minimum of 2 months is needed to obtain any reduction in adipose (fat) tissue.

Studies have shown that exercise incorporated in a weight loss program may decrease appetite. Also, physical exercise may increase metabolic rate which counteracts the decrease in metabolic rate which occurs when dieting.

The most beneficial exercise for overweight patients is one which involves moving body mass such as swimming. Patients do not become overheated with this exercise.

Fats offer the highest caloric value. Protein and carbohydrates contain less than 1/2 as many calories as the same number of grams of fat. Following low fat diet carefully, reducing the saturated fat to less than 15 grams daily reduces the calories.

Vitamins and Mineral supplements are recommended during weight loss programs of less then 1200 Calories.

The following chart will give you energy costs of various activities. To calculate the amount of energy expended, first convert your weight in pounds to kilograms by dividing by 2.2.

EXAMPLE: 140 pounds divided by 2.2 = 64 kilograms

If your weight in kilograms is 64, multiply this by the number of kcal (Cal) in the right hand column of the table.

EXAMPLE: Archery kcal= 0.754

your weight: 64 kilograms X 0.754 = 48 kcals per 10 minutes of Archery

Energy Costs of Various Activities

Activity	kcal/kg/10 min
Bicycle on level roads	0.734
Bowling	0.975
Calisthenics	0.734
Canoeing, 2.5 mph	0.441
Carpentry	0.564
Chopping wood	1.101
Classwork, lecture	0.245
Cleaning windows	0.607
Conversing	0.269
Dancing	
moderately	0.612
vigorously	0.831
Dressing	0.466
Driving car	0.438
Driving motorcycle	0.531
Driving truck	0.342
Gardening, digging	1.365
Gradening, weeding	0.862
Golfing	0.794
House painting	0.514
Ironing clothes	0.627
Lying quietly	0.195
Making bed	0.572
Mopping floors	0.665
Mountain climbing	1.470
Pitching horseshoes	0.518
Playing Ping-pong	0.566
Playing tennis	1.014
Resting in bed	0.174
Rowing for pleasure	0.734
Running up grade (treadmill)	
7.00 mph	2.045
8.70 mph	2.273
11.60 mph	2.879
Showering	0.466
Sitting, eating	0.204
normally	0.176
playing cards	0.210
writing	0.268
Sleeping	0.172
Standing, light activity	0.356
normally	0.206
Sweeping floors	0.535
Swimming, for pleasure	1.454
Volleyball	0.505
Walking on level (treadmill)	
2.27 mph	0.513
3.20 mph	0.690
3.50 mph	0.733
Walking downstairs	0.976
upstairs	2.540
Washing and dressing	0.382
Washing and shaving	0.419

FOOD LIST*

Following is a limited listing of foods permissible on diet. Note that these foods may contain small amounts of fats or oil. Be prudent in your use of them.

MANUFACTURER	VARIETY
COLD CEREALS	
Nabisco	Shredded Wheat
Post	Grape-nutsBarbara's Breakfast biscuits
Nature's Path	Manna (Millet Rice Flakes, Multi Grain Flakes with Oat Bran & with Oat Bran and Raisins)
Kolln	Oat Bran Crunch
Health Valley	100% Natural Bran Cereal
	Oat Bran Flakes
	Oat Bran O's
	Blue Corn Flakes
	Stone Wheat Flakes
	Raisin Bran
	Fruit Lites
U.S. Mills	Uncle Sam
	Crispy Brown Rice
	Skinner's Raisin Bran
	Skinner's Low-Sodium Raisin Bran
Perky Foods	Crispy Brown Rice, Nutty Rice
Barbara's	Brown Rice Crisps
	Breakfast O's
	Raisin Bran
Kellog Co.	Nutri-Grain (Corn, Wheat, Nuggets ect.)
HOT CEREALS	
Quaker Oats	Quaker Oats
	Minute Quaker Oats
U.S. Mills	Instant Oat Meal
	Barley Plus
	Brown Rice Cream
Stone-Buhr Milling	Hot Apple Granola
	7-Grain Cereal
Moore's Mill	Old Fashioned Oats and other grain cereals
Golden Temple	Oat Bran
Barbara's	14 grains
Kashi Company	Kashi (some sesame seeds)
POTATOES	
Ore-Ida	Hash Browns
Bel-Air	Hash Browns
POPCORN	
ANY Unprocessed Popcorn (with no added ingredients)	
Weight Watchers'	Microwave Popcorn
Nature's Best	Natural Popcorn
RICE CAKES	
Quaker	RiceCakes (lighty salted)
H.J. Heinz Co.	Chico San (Millet, Buckwheat, ect.)
Hollywood	Mini Rice Cakes (Teriyaki, Apple, Cinnamon)
Hain Pure Food Co.	Mini Rice Cakes (Apple, Cinnamon)
Pacific Rice Prod.	Mini Crispys (Apple Spice, Raisin N'Spice, Italian Spice, Natural Sodium Free)
Westbrae	Teriyaki Rice Cakes
Lundberg Family Farms	Rice Cakes (Wild Rice, Wehani, Brown Rice, Mocha Sweet)
CRACKERS	
San-J	Tamari Brown Rice Crackers
Westbrae	Brown Rice Crackers
Ralston Purina Co.	Natural Ry-Krisp
Edward & Son	Baked Brown Rice Snaps
Hol-Grain	Brown Rice Lite Snack Thins
	Whole Wheat Lite Snack Thins
O. Kavli A/S	Kavli Norwegian Crispbread
Barbara's	Crackle Snax
	Lightbread
Wasabrod	Wasa Crispbread (Breakfast, Hearty Rye)
PRETZELS	
Laura's Scudder's Inc.	Mini-Twist Pretzels
	Pretzels Sticks
	Bavarian Pretzels
Blue Bell	Pretzel Stix
Fred Meyer	Pretzel
Ralph's	Bavarian
BURGER MIXES	
Fanastic Foods	Fantastic Falafil
	Nature Burger (with Sesame seeds)

BEANS

Heinz	Vegetarian Beans
B&M	Vegetarian Beans
S&W	Maple Sugar Beans
Horizon	Refried Beans

COOKIES

Health Valley	All Brands without Hydrogenated Oils

SOUPS

Progresso	Minnestroni
	Lentel

SALAD DRESSING

Thompson Kitchens	Pritikin No-Oil Dressing (Ranch, Tomato, Russian, ect.)
WM Reily & Co.	Herb Magic (All No-Oil-Vinaigrette, Italian, Gypsy, Zesty Tomato, Creamy Cucumber)
American Health	El Morino (all No-Oil-Herbs & Spices, ect.)
Cook's Classic Ltd.	Cook's Classic (Italian Gusto only)
Kraft Inc.	Oil Free Italian
H.J. Heinz Co.	Weight Watcher's Dressing (Tomato, Vinaigrette, French)
Hain Pure Food Co.	No Oil Dressing Mix
Paul Newman's	Oil & Vinegar
Good Seasons	Italian (Dry)
Hidden Valley	Ranch (Dry)

SPAGHETTI SAUCE

Paul Newmans'	Spaghetti sauce
Pure & Simple Inc.	Johnson's Spaghetti Sauce
Trader Joe's	Trader Giotto's Italian Garden Fresh Vegetable Spaghetti sauce
Mrs. Johnson's	Spaghetti sauce
Westbrae	Ci Bella Pasta Sauce
Thompson Kitchens	Pritikin Spaghetti Sauce

CHEESE

American Natural Snacks	Soya Kaas (Cheddar, Jalap, Moz)
Rose International	Tofu Rella (Cheddar)
Diet & Health Products	Count Down Cheese
Sargento	Pot Cheese
Apple Farms	Quark (Soft Spreadable Cheese Cream Cheese)

ICE DESSETS

Dole Package Foods Co.	Fruit Sorbet
	Fruit N' Juice
	Sun Tops-Real Fruit Juice Bars
Eskimo Pie Co.	All Natural Pops (Fruit Punch, Cherry, Orange)
Frozfruit Corp.	Frozfruit (chunk strawberry, lemon, cantelope, lime, orange, banana, cherry, pineapple)
Tofutti Brands	Lite lite Tofutti (vanilla, cappucino chocolate-strawberry-vanilla)
Vitari	All flavors
Dole	All flavors

FROZEN YOGURT (NON FAT)

Honey Hills	All Flavors
Columbo Lite	All Flavors
TCBY	All Flavors

FROZEN DESSERT BARS

Weight Watchers	Fudge Cycle
	Dream Cycle
Schwan's	Chocolate Fudge Bar
Darigold	Fudge Cycle

*Note that many of these food items were taken from the May/ June Issue of the MCDOUGALL newsletter.

WHERE TO EAT OUT

In Portland:

Macheesemo Mouse

1200 SW Salmon	249-0002
715 SW Weidler	228-3491
3553 SE Hawthorne	232-6588
811 NW 23rd	274-0500

Comments: No cheese, No processed chicken.

Song of the Rose Cafe
1328 NW 23rd 224-0863

Abou Karim
221 SW Pine 223-5058

Al-Amir
223 SW Stark 274-0010

In Beaverton:

Nature's Fresh Northwest
4000 SW 117th 646-3824

Nonna Emilia Ristorante Italiano
17210 SW Shaw 649-2232

Macheesemo Mouse
10719 SW Bvtn Hillsdale Hwy
646-6000

Malones
14709 SW Teal Bvld 646-6759

OFFICE NEWS

As many of you know it sometimes is necessary to leave a message on our answering machine. This is necessary as we are limited in our office staff due to funding and office space. When leaving a message please speak slowly when giving your telephone number. If you are having a medical emergency ,state this in your message, otherwise it may be the next day before we can return your call depending on what time you leave your message. We would like to thank everyone for their patience and understanding as we try to meet everyone's needs as quickly as we can.

There is a new Free Parking facility on campus. It is located below the outpatient clinic and hospital south. Bring our ticket to the office for validation,

RECIPES

Turkey Jerky

1 pound boned and skinned turkey breasts
1 tablespoon salt
1/2 cup water
2 tablespoons firmly packed brown sugar
2 cloves garlic, minced or pressed, or 1/4 teaspoon garlic powder
1/2 small onion, minced, or 1/4 teaspoon onion powder
1 teaspoon pepper
1/2 teaspoon liquid smoke
Non stick cooking spray(Non fat)

Rinse meat and pat dry. Pull off and discard any fat and connective tissue. For easier slicing, freeze meat until it's firm but not hard. Cut into 1/8 to 1/4-inch-thick slices: cut breast piece with or against the grain.

In a bowl, stir together salt, water, brown sugar, garlic, onion, pepper, and liquid smoke. Add turkey and mix well. Cover and chill at least 1 hour or up to 24 hours; eat will absorb most of the liquid.

Depending opon drying method, evenly coat dehydrator racks (you need 3, each about 10 by 13 in.) or metal racks (to cover a 10 by 15 baking pan) with nonstick cooking spray.

Lift turkey strips from liquid, shaking off excess, and lay strips close together, but not overlapping, on racks.

In a dehydrator, arrange trays as manufacturer directs and dry at 140 F until a cool piece of jerky (removed from the dehydrator and let stand about 5 minutes) cracks and breaks when bent; this should take 41/2 to 5 hours.

In a oven set at 150 to 200 F, place pan on center rack; prop door open about 2 inches. Dry until a cool piece of jerky (see above) cracks and breaks when bent, 3 to 5 hours.

Let jerky cool on racks, then remove. Serve, or store in airtight containers in a cool, dry place up to 3 weeks, in refrigerator up to 4 months, or longer in the freezer. Makes about 7 ounces.

Teriyaki Turkey Jerky

Prepare turkey jerkey (recipe procedures) omitting salt and water. Add 1/4 cup soy sauce and 2 teaspoons worcestershire sauce.

Zucchini Carrot Cake

2 eggs
1 cup sugar
2/3 cup oil
1/4 cup all purpose flour
1 tsp baking powder
1 tsp baking soda
1 tsp cinnamon
1/2 tsp salt
1 cup carrots grated
1 cup zucchini grated & drained
1/2 cup nuts , walnut

Beat eggs with sugar until frothy. Gradually beat in oil. Add dry ingredients. Beat at high speen, 4 minutes. Stir in carrots & zucchini and nuts , pour into greased 9" square pan. Bake in 350 F oven about 35 minutes.

RESEARCH EFFORTS

We would like to take this opportunity to thank you for your contributions to our research. With your help, we may have identified a plasma protein involved in the metabolism of fat, Continued support is necessary if we are to carry this research further.

Sincere Thanks

Roy L. Swank M.D. ,Ph.D.

Make checks payable to: *O.H.S.U. FOUNDATION*
SWANK M.S. RESEARCH