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An Investigation of the Relationship Between Dietary Lipids and Serum Lipids.

(as it appears on title page of thesis)

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AN INVESTIGATION OF THE RELATIONSHIP BETWEEN DIETARY LIPIDS AND SERUM LIPIDS

A Thesis

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In Partial Fulfillment of the Requirements

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Sharon-Dawn Jo-Anne Vanin

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ABSTRACT

This thesis examined the relationship between dietary fat intake and serum lipid levels in a male sample of the population in Saskatoon, Saskatchewan. The total sample of 163 was subdivided into Normals (87) and patients with Ischemic Heart Disease (IHD) (76). All subjects were participants in an Exercise Research Project, and were between the ages of 35 and 60 years.

A 7-day Dietary History was used to evaluate the dietary habits. All subjects completed at least one dietary history. There were also 25 subjects who completed 2, and 2 subjects who did 3 histories. These histories were then coded and computer analyzed. Computer analysis utilized the Master Food Composition Table stored in computer memory. The table contained the nutritional breakdown of some 1,000 different food items. Included on the computer printout were the daily figures for Calories, Protein, Total Fat, Saturated Fat, Monousaturated Fat, Polyunsaturated Fat, and Cholesterol. The average weekly intake of the above mentioned nutrients was also computed.

The serum determinations were done by qualified technicians in a hospital laboratory. The figures from these determinations were obtained from the charts of the individual subjects. The other medical data necessary was also obtained from these charts.

The dietary and medical data were merged onto computer tape to facilitate analysis.

Normal subjects as compared to IHD subjects had higher intakes of cholesterol, total fat, saturated fat and calories. The IHD subjects had higher serum cholesterol and triglyceride levels than the Normals.

In this population sample studied by the author, the higher dietary intakes of fats did not appear to be associated with the increased serum lipid levels. However, the higher serum lipid levels were associated with the incidence of IHD. According to the literature reviewed, (7, 15, 29, 41, 43) the authors suggest an association between high dietary cholesterol and high serum cholesterol and that this in turn precipitates the occurrence of IHD.

This study compared present dietary fat intake with serum lipid levels and did not find a positive relationship.

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1. INTRODUCTION

1.1 Nature and scope of the work

The purpose of this thesis is to examine the relationship between dietary and plasma lipids in a male sample of the population in Saskatoon, Saskatchewan. Results of previous research indicate that high dietary intakes of cholesterol and saturated fats cause an increase in the plasma levels of cholesterol, and this in turn relates to a high incidence of Ischemic Heart Disease. (3, 25, 54). The author intends to determine if such a relationship exists in the population sample studied.

Elevated plasma lipids have been classified as responsible for increasing the risk of developing IHD. The Framingham Study (14) identified high serum cholesterol as an important risk factor.

The dietary information for this thesis was obtained from Diet Histories completed by participants in an Exercise Research Project. The histories were completed by 163 male subjects between the ages of 35 to 60 years. This total includes 87 Normal subjects and 76 subjects with IHD.

The author revised the initial diet history booklet to develop one which was more suitable for obtaining necessary dietary information. The author also enlarged the Master Food Composition Table used in analyzing the diet booklets.

1.2 Description of Ischemic Heart Disease (IHD)

Ischemic Heart Disease (IHD) is the major cause of death in the Canadian population and is responsible for more than 48,000 deaths annually. (30)

There are 2 coronary arteries which supply the heart with blood. IHD results from a deficiency of blood flow to the myocardium due to obstruction of one of the coronary arteries. The major cause of this obstruction is the incrustation of the blood vessels with atheromatous plaques. These plaques most often contain cholesterol and, in the later stages, may also contain substantial amounts of calcium. IHD is usually manifested clinically as either angina pectoris or myocardial infarction.

Certain characteristics closely related to the risk of developing IHD are described in the following discussion. (14)

Major risk factors include serum cholesterol, blood pressure level and cigarette smoking. The higher the serum cholesterol level, the greater is the risk of developing IHD. Above 260 mg%, there is twice the risk of developing IHD, while below 220 mg%, there is less than one-half the standard risk. (standard risk rate is generally 6 deaths/1,000 persons/year). Increased blood pressure (systolic 160 mm Hg and over, and diastolic more than 95 mm Hg) results in an increased risk of developing IHD. (14) Cigarette smoking appears to be related to the development of myocardial infarction and sudden death. The

results are not cumulative however, and those who have quit, have the same low rate of development of IHD as those who have never smoked.

Minor risk factors include electrocardiographic (ECG) abnormalities, overweight and reduced vital capacity. Those with ST-T wave ECG changes develop IHD more frequently than those without it. (ST -- depolarization of the heart, T -- polarization) Obese people have an added risk of mortality from IHD. Men with vital capacities below 3 litres had about twice the risk of developing IHD as those with vital capacities 4 litres or above. The relationship between vital capacity and IHD development has not yet been clarified.

When more than one of these factors is present, the risk of developing IHD increases still further. It is possible to determine the relative susceptibility to IHD of various subgroups of the population based on the presence of one or more high risk characteristics.

1.3 Epidemiology

The 1971 World Book Dictionary defines epidemiology as "the branch of medicine dealing with the distribution of disease and cause of the spread of diseases in a community". (60)

More than 1,400,000 Canadians of all ages have some form of cardiovasular disease. More than half of all deaths each year are due to heart and circulatory disease and about 35% of these occur among people 45 to 64 years of age. (61) In 1969, 48,394 Canadians died of IHD, which represented 31% of all deaths. (20) In 1968 in Saskatchewan,

there were 2266 deaths due to IHD. (20) This was 30% of all deaths.

Ischemic Heart Disease has a multifactorial etiology. The causative factors include high blood pressure, high serum cholesterol, obesity, high dietary cholesterol, too little physical exercise, diabetes, cigarette smoking, tension, stresses, and heredity. This thesis is mainly concerned with the role of diet, so this risk factor will be discussed further.

There is a higher rate of IHD in countries and cultures that are more advanced economically. In these countries the diets are high in calories, high in animal and saturated fats, high in cholesterol, and high in "empty" calories from refined foods.

In under-developed countries where the diets are low in total calories from animal fats, low in cholesterol and low in empty calories; the serum cholesterol is found to be low and atherosclerotic heart disease is rare. (3) Epidemiological studies have shown that in populations which have an elevated serum cholesterol, the indidence of IHD is greater. (29)

1.4 Diet and IHD

Diet is one of the risk factors of IHD. (13) The Framingham Study, an epidemiological investigation of cardiovascular disease, carried out in Framingham, Massachussetts, U.S.A., considered diet as one of the risk factors. Participants in this study were followed for 15 years, from 1950 to 1965. No changes were knowingly made in their life style.

The Framingham Study stated that even though a relationship was not found between dietary and serum cholesterol levels, this did not mean that serum cholesterol could not be changed by diet. Simply because they did not find a significant relationship between dietary and serum cholesterol levels, this did not mean that high dietary cholesterol did not cause elevated serum cholesterol levels in a high percentage of the North American population.

The results did not refute that differences in serum cholesterol levels in different races may be related to diet. Orientals have a low serum concentration of cholesterol and triglyceride and a lower incidence of IHD than do people in Western countries. Their fat consumption is only 15% of the total calories and this is mostly in the form of polyunsaturated fatty acids. Western cultures derive approximately 45% of their calories from fat, and this is mostly saturated animal fat. (34)

Leren (41), Turpeinen (57), Dayton (16), and Christakis (10), all suggest that altering the diet of healthy coronary-prone population groups lowers the serum lipid levels, if the diet is maintained, and this also reduces the incidence of atherosclerotic complications. When other factors, such as calories and total fat were kept constant, substituting saturated animal and dairy fats by polyunsaturated fats and oils, significantly lowered serum cholesterol levels, and reduced the mortality from clinical atherosclerosis. (43)

A food pattern to reduce serum cholesterol and maintain a reduced level was outlined by Brown. (6) Principals of the diet include low total fat, low saturated fats, less than 350 mg. of cholesterol per day, and a high polyunsaturated fat content.

2. LITERATURE REVIEW

2.1 Dietary cholesterol and serum cholesterol

Brunner (7) stated that the low cholesterol and low caloric intake of the Yemenite Jews was responsible for the low cholesterol levels and low incidence of IHD.

Dayton (15) showed in his experimental group (who had been on a low cholesterol diet) that when the regular diet was resumed, a rise in serum cholesterol resulted. This rise was comparable in magnitude to the fall seen at the start of the study.

Hegsted (29) states that a high level of serum cholesterol is closely associated with IHD, and that serum cholesterol can be lowered with a low fat - low cholesterol diet.

Leren (40) in a 5 Year Study compared the effects of a normal Norwegian intake and a low fat intake. The diet group had an average reduction in serum cholesterol of 29% and had fewer infarcts and fewer deaths than the control group. (29, 40) This indicates a positive benefit from dietary treatment in patients with a previous myocardial infarction. This study involved men 30 to 67 years of age who had survived a myocardial infarction. The low fat diet had reduced cholesterol and saturated fat intake. (39% of total calories).

Little (43) suggests that reduction of cholesterol ingestion to less than 500 mg/day lowers serum cholesterol significantly.

Bierenbaum (2) found in his study with men 30 to 50 years old who had past myocardial infarctions, when placed on a low saturated fat-low cholesterol diet, they had a reduction in serum cholesterol levels.

The Framingham Study found no indication of a relationship between dietary cholesterol and serum cholesterol. (34)

Keys (38) found in several experiments that the serum cholesterol level is related to the square root of the concentration of cholesterol in the diet.

2.2 P/S ratio

The P/S ratio refers to the proportion of polyunsaturated fatty acids as compared to the saturated fatty acids in the diet. To find the complete diet ratio, the total polyunsaturated and total saturated fatty acids are determined. These totals are expressed as a ratio.

Williams (59) states that if all of the other nutritional needs are cared for, the ratio of saturated to unsaturated fats is not important. He regards extreme emphasis on the use of polyunsaturated fats to be unwarranted.

Brown (6) suggests that polyunsaturated and saturated fatty acids must meet certain limits in order to be effective.

Keys (37) believes that if the P/S ratio is 2/1, this will help lower serum cholesterol.

Pinckney (55) states that excessive polyunsaturates may be potentially toxic. He quotes such side effects as increased incidence of cancer and premature aging.

A study done in London by Little (56) showed that men surviving one myocardial infarction who followed a diet low in saturated fats and high in polyunsaturates had a 22% fall in the initial serum cholesterol of 272 mg %.

2.3 Role of dietary saturated fats.

Keys (37) incriminated fats, particularly saturated fats in the development of IHD. He developed a formula to permit estimation of the effect of any diet on serum cholesterol level: (38)

$\Delta chol = 1.3\ (2\Delta S' - \Delta P) + 1.5\Delta Z$

S' — % of total cals. from sat. f. a. (omitting stearic which has no cholesterol increasing effect.)

P — % of total cals. from polyunsaturated fatty acids

Z — square root of dietary cholesterol (mgm/1000 cals.)

Hegsted (29) in his study agreed with Keys, in demonstrating that saturated fatty acids elevate serum cholesterol, whereas polyunsaturated fatty acids reduce serum cholesterol. To be effective in lowering serum cholesterol, there should be twice as much polyunsaturated fatty acid in the diet as there are saturated fatty acids. (29, 43).

Malmros (46), in experiments with rabbits showed that lesions caused by a highly saturated coconut fat diet disappeared on a polyunsaturated corn oil diet.

In practically all countries there is a clear relationship between the amount of fat and the amount of saturated fat consumed. Thus, it may be the composition of the dietary fat that is critical (saturated as compared to polyunsaturated), and not the amount of dietary fat. (29)

Dalderup (12) proved that he could decrease serum cholesterol by using a special margarine with a polyunsaturated fatty acid content of over 50%.

Bierenbaum (2) found that the degree of unsaturation of fats did not appear to influence serum cholesterol.

2.4 Dietary carbohydrate

In various studies, sucrose has been implicated as being the causative dietary factor in the development of IHD. In Yudkin's studies, the prevalence of heart disease appeared to be more closely related to sugar intake than to fat intake. (62, 63, 64) However, Elwood et al (21) found that there was no evidence in men with IHD that they had a higher sucrose intake than did others. Yudkin, (13) unlike other workers, suggests that patients who had myocardial infarctions consumed two times as much sugar as did the controls. He found this in 2 studies, one in 1964 and one in 1967. (63, 64) Burns-Cox et al (8) found in a study with hospital patients admitted with myocardial infarctions that they did not take significantly more sugar than controls. Epidemiological studies from New York,

Oslo, and Helsinki, showed a positive relationship between low saturated fat and low sugar consumption, low serum cholesterol and decreased incidence of IHD. (63)

It appears that the serum lipids can be changed by alterations in dietary fat or sugar. (63) Different types of carbohydrate have different effects on plasma triglyceride concentrations. (13) The composition of serum lipid after several days on a low fat diet appears to be dependent on the type of carbohydrate consumed. (45) Lipid is formed from carbohydrate in the absence of dietary fat. The 2-Carbon portions of carbohydrate are precursors of triglyceride. (42)

2.5 Percentage of body fat

Serum cholesterol rises with a gain in weight, even though the subject is on a low cholesterol diet.

Keys (33) stated that each 1% rise in calories above the caloric balance raises the serum cholesterol level 2 mg%. The rising cholesterol level reaches a plateau after 10 weeks, even though the positive calorie balance and weight gain continue much longer. If a positive caloric balance is prevented. Overnutrition in general, rather than intake of dietary fat, may be responsible for IHD.

Nestel (18) stated that in obesity the production rate of cholesterol is increased. This is maybe due to synthesis by the adipose tissue itself.

Miettinen (48) stated that obesity is associated with

an increased rate of cholesterol synthesis in man. Obese subjects produced daily, 20 mg. cholesterol / Kgm of adipose tissue; compared to controls who produced 12 mg. / Kgm. Therefore, overweight patients generally have higher serum cholesterol levels.

2.6 Literature review summary

The authors in agreement that dietary cholesterol increases serum cholesterol include D. Brunner (7), S. Dayton (15), D. Hegsted (29), P. Leren (41), J. Little (43), and M. Bierenbaum (2).

Serum cholesterol can be increased with high dietary intakes of saturated fats. The preceeding statement was agreed on by L. Dalderup (12), A. Keys (37, 38) and D. Hegsted (29). M. Bierenbaum (2) did not agree that saturated fats were the cause of increased serum cholesterol levels.

Several authors agreed that a high P/S ratio (2:1) lowers serum cholesterol. These authors include H. Brown (6), L. Dalderup (12), D. Hegsted (29), A. Keys (37, 38), P. Nestel (50), and H. Malmros (46). The authors disagreeing that a high P/S ratio was important were R. Williams (59), E. Pinckney (55), and M. Bierenbaum (2).

An increase in the percentage of body fat may increase serum lipids. A. Keys (38), P. Nestel (50), and A. Miettinen (48) found an increase in the serum cholesterol fraction.

Dietary carbohydrate may increase the serum lipid level. J. Yudkin (64) found that serum cholesterol and triglyceride were in-

creased with high carbohydrate diets. I. Macdonald and D. Braithwaite (44) duplicated several of Yudkin's experiments.

Several epidemiologic studies on humans have been carried out and found a positive relationship between dietary and serum cholesterol. Such studies included those by P. Leren (4) in Norway in 1966; by J. Little (43) in Canada in 1965; by O. Turpeinen (57) in Finland in 1958; and by G. Christakis (10) in the United States in 1957.