EFFECTS OF WHEAT BRAN FIBER AND CARBOHYDRATE SOURCE ON GLUCOSE TOLERANCE, SERUM CHOLESTEROL AND LIPOGENIC ENZYME ACTIVITY IN WEANLING RATS

by

JOSEPH DUDLEY MATTHEWS

B.S., Kansas State University, 1977

A MASTERS THESIS

Submitted in partial fulfillment of the

requirements for the degree

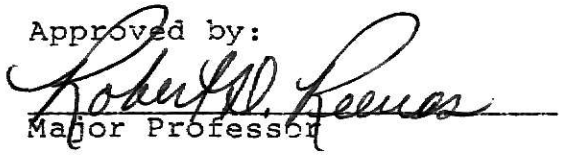
MASTER OF SCIENCE

Department of Foods and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1980

Approved by:



Major Professor

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to Dr. Robert D. Reeves, Assistant Professor, Department of Foods and Nutrition, for all his help as a research advisor, instructor and friend.

I would also like to thank Dr. Gur Ranhotra and Dr. Dan Upson for serving on my graduate committee and for their respective contributions in nutrition and physiology.

There are many others who have contributed in one way or another to the success of this research among them are Jean Craig, Susan Davis, Dr. Holly Fryer, Dr. Arthur D. Dayton and Tina Shrimplin. To these and others in the graduate workroom, I offer my heartfelt thanks.

I especially want to thank Jane, who married me in spite of the fact that I spent so many evenings with research rats instead of with her. And a special thank you is given to my parents and family for their encouragement and support throughout the years.

Finally, I must acknowledge Jesus Christ, my personal Savior and friend, whose presence in my life has given me the ability to set and achieve my goals.

INTRODUCTION

Dietary fiber has been a subject of research and discussion during the past few years. This surge of interest was sparked by the discovery that individuals in underdeveloped areas of the world have a much lower incidence of certain diseases than those in the industrialized nations. These disorders include ischemic heart disease, appendicitis, diverticular disease, gall bladder disease, varicose veins, deep vein thrombosis, hiatus hernia, and tumors of the large bowel. These differences may be due in part to the relatively higher level of fiber in the diets of those individuals in developing countries (1).

More recently dietary fiber has been prescribed, comgined with complex digestible carbohydrate, for relief of the symptoms of diabetes mellitus. The symptoms include impaired glucose tolerance, poor insulin response and the many progressive complications associated with that disease: hypercholesterolemia, atherosclerosis, increased lipogenesis and obesity (1, 3). The disease parameters of these conditions have been shown to be treatable to some extent by the incorporation of high levels of fiber into the diet (3). That this food category can be used as a prophylactic measure against diabetes mellitus and its associated complications is theoretical only, and the degree of effect in diabetic as well as normal individuals needs clarification.

The study to be presented is the first in a series of proposed investigations in this laboratory aimed at determining the effect of wheat bran fiber and carbohydrate source on carbohydrate and lipid metabolism and other metabolic parameters in normal and diabetic rats. Weanling rats will serve as the experimental model.

This study was specifically designed to investigate the influence of wheat bran carbohydrate type on selected hepatic lipogenic enzymes, serum cholesterol, glucose tolerance and food efficiency during a 6-week feeding trial in weanling rats.

LITERATURE REVIEW

Mechanisms of Fiber Action

Fiber effects large bowel function by causing an increase in fecal output, dilution of colonic contents, decreasing gut transit time and changing the colonic metabolism of minerals, nitrogen, and bile acids (4).

The actions of fiber probably are not due to its increased ability to hold water. *In vitro* water-holding capacity of 17 dietary fiber preparations, mainly food materials, bulk laxatives, and gel-forming polysaccharides, were compared to changes in fecal weight when these same fiber preparations were fed to human subjects. Overall, an inverese relationship ($r = 0.88$) between water-holding capacity and fecal weight was found. Bran had the lowest water holding (4.2g/g) and the largest fecal weight changes (117%) (5). This is probably because fiber is extensively degraded in the gut by colonic microflora (4). The major component of feces is bacteria (6). Coarse bran is more effective in increasing fecal weight than ground bran in humans ($P < 0.01$) (7). Though bran is the most widely known source of fiber as a fecal bulking agent, other dietary fiber sources have been noted for this effect. In a study conducted in 1979 (8) a diet in which half of the fiber came from fruits and vegetables and the rest came from bread and cereal products, fecal weight increased by 115 g/day and mean gut transit time was decreased by 18

hours. Gut transit time and fecal bulk are probably due to stimulation of the paristaltic response and possibly other unknown factors.

Glucose Tolerance

A relationship between dietary carbohydrate and glucose tolerance has been suspected for several years but the role of fiber in glucose tolerance is a relatively recent area of research.

In 1976 Harland, et al (9) reported significantly increased fasting blood glucose in both diabetic and control rats after three weeks on diets containing 10% Solka-floc, Alphacel, wheat bran, alfalfa, sugar cane pulp, or sugar beet pulp as sources of fiber. They found feeding Solka-floc, Alphacel and wheat bran increased fasting blood glucose in the normal animals. A 40% increase in the blood glucose levels of the diabetic rats fed all fiber sources was observed compared to the diabetic animals fed the control diet. The findings of Harland, et al (10) indicate a negative effect of fiber relative to fasting glucose. However a later study by the same researcher (10) failed to confirm these results.

A similar study the following year addressed the problem of glucose tolerance. The study, conducted by Anderson and Lin (2), included normal and diabetic rats. Diets were low in carbohydrate from mixed sources (32 g/100 g feed), high carbohydrate sucrose (72 g/100 g

feed) and high carbohydrate starch-bran (72 g/100 g feed). The starch-bran diet contained 60 grams of starch and 12 grams of wheat bran per 100 grams of feed. Protein was variable with 46 g/100 g feed in the low carbohydrate diet, 18 g/100 g feed in the high carbohydrate-sucrose diet and 12 g/100 g feed in the starch-bran diet. Fat was 12% by weight in the low carbohydrate diets and 6% in the high carbohydrate diets. Dietary fiber in the low carbohydrate diet was provided by cellulose at 6% and by wheat bran in the starch-bran diet also at 6%. High carbohydrate diets were associated with slightly higher plasma glucose levels in the diabetic and normal animals. Glucose tolerance was not changed by any of the test parameters. From the results of this study, the effects of carbohydrate type and fiber level in the diet are unclear.

The fasting plasma glucose elevation with high fiber diets combined with an apraently unchanged glucose tolerance could be due to residual fiber in the small intestine slowing the transit of absorbable carbohydrate across the brush border or simply delaying gastric emptying. This hypothesis was tested by Peng and Tsai (11) by administering locust bean gum with glucose solution for a glucose tolerance test. In this study rats were all fed a stock diet and then given glucose in a 10% solution or this solution with added locust bean gum at 1%, 1.5%, or 2.5%. They found that at 45 minutes post prandial the 2.5% gum solution produced a lower serum glucose level than in

control rats receiving a glucose solution without added gum. The same solution produced a higher serum glucose at 90 and 135 minutes compared to the controls which received no gum with the glucose load. All animals dosed with the glucose plus 2.5% gum solution had a significantly larger amount of glucose retained in the stomach than the controls 75 minutes after dosing. The researchers concluded that the reduction in postprandial hyperglycemia was a result of reduced rates of food emptying from the stomach into the intestine of the rats when locust bean gum was given with the meal.

The lack of effect of dietary fiber on glucose tolerance is supported by Albrink et al (12) who found no effect on human glucose tolerance when a high-fiber starchy diet was fed. These results and others led to the assumption that fiber in general has no effect on glucose tolerance but may raise fasting glucose due to delayed gastric emptying time (13). In addition there may be some slowing of absorption rate within the small gut though no overall malabsorption of glucose occurs when guar gum is given with the meal.

> The XI International Congress of Nutrition concluded: Dietary fibers modify glucose absorption and may be of clinical usefulness in the prevention and treatment of adult onset diabetes mellitus and hypoglycemia and obesity. (14)

Anderson and Ward (15) have gone one step further by suggesting that insulin therapy may be discontinued in some patients with adult onset diabetes when such patients are placed on a high carbohydrate and high-fiber diet. But Mann and Simpson (16) claim that only patients who are not truly insulin dependent can be considered for such diet therapy. These statements were not supported by research data but other literature cites this attitude.

These studies support the single action of fiber hypothesis concerning glucose in the blood. That action is a slowing of the gastric emptying and perhaps slowed absorption from the small intestine. If this is the only mechanism then fiber should be mixed thoroughly with the food (17, 18).

In a 1978 study Munoz (19), using several sources of fiber, including wheat bran, showed that there is another factor involved in fiber modification of glucose tolerance. They found improved glucose tolerance in men on high-fiber diets and a significant correlation between the area under the oral glucose curves and the levels of total and LDL cholesterol. The conclusion was that glucose tolerance can be improved by consumption of certain sources of dietary fiber.

Taylor (20) supported this stating that perhaps there is a factor in addition to slower gastric emptying time affecting blood glucose in a glucose tolerance test by fiber administered with the glucose load. He found

alterations in glucose tolerance tests varied with the type of fiber used.

These studies indicate an effect of dietary fiber and carbohydrate source on glucose tolerance. Indicated is an increased fasting glucose in animals fed high-fiber diets over a long period of time. Results of studies on the effect of fiber fed over a long period of time on glucose tolerance are not conclusive.

Effects of Dietary Fiber and Carbohydrate Source on Serum Cholesterol

Atherosclerosis is the most common clinical and anatomic type of circulatory disease and accounts for nearly half of all deaths from coronary heart disease (CHD) (21). Lipid deposition, particularly cholesterol and its esters, in the tunica intima of arterial walls increases in young adults. Smooth muscle and connective tissue proliferate and surround the lipid deposit narrowing the vessel (22). Calcification may convert the resulting sclerotic plaques into rigid pipestem conduits reducing the elasticity of the vessels. This results in an increase in range between systolic and diastolic blood pressures and a general increase in blood pressure. Total occlusion rarely occurs unless there is an accompanying infarct or collateral circulation has developed sufficiently to supply the ischemic areas (21).

Epidemiological studies in the 1950s and 1960s showed coronary heart disease patients to have consistently higher

serum cholesterol concentrations than control patients (23, 24). In 1971, following a 20 year longitudinal study, Kennel et al (25) found serum cholesterol to be the most consistent indicator of risk of developing atherosclerosis. An elevated level of serum cholesterol is a risk factor in the development of atherosclerosis. Dietary patterns leading to hypercholesterolemia are not as easy to define as the condition itself.

Increased dietary cholesterol has been shown to elevate serum cholesterol and produce atherosclerotic lesions in many species of laboratory animals (26). In humans a nearly linear relationship between dietary cholesterol intake and elevation of serum cholesterol concentration up to a daily intake of about 400 mg has been reported (27). Above that level of intake the relationship is no longer linear (28). An average of 600 mg cholesterol per day is injested by Americans (29).

Until recently polyunsaturated fatty acids (PUFA) have been advocated for their ability to decrease serum cholesterol when incorporated into the diet in place of saturated fats. Some evidence now contradicts this because of possible side effects of PUFA in the diet. PUFA have been shown to actually increase cholesterol absorption (30) and have been implicated in aging, carcinogenesis and increased propensity for gallstone formation (31).

Not all animals or humans exhibit the same susceptibility to hypercholesterolemia induced by diet. Animals

that have a high response to dietary cholesterol are called hyperresponders and those with low responses are called hyporesponders (32). Even in the absence of dietary cholesterol hypercholesterolemia, resulting in atherosclerosis and coronary heart disease may develop due to genetically determined metabolic defects (33). Because of tremendous variations in individual responses to dietary cholesterol restriction by the general population is not advocated, however in certain high risk individuals, hyperesponders, including those with elevated blood pressure, diabetics and the obese, dietary therapy should be instituted to prevent further provocation (25).

The variations among individuals within a given species have been attributed to several factors: 1) differences in the feedback control of cholesterol sysnthesis, 2) variability in cholesterol absoroption, 3) differences in bile acid excretion and 4) diversity and quanity of intestinal bacterial flora. The latter three of these factors have been postulated to be directly altered by the presence of varying amounts of fiber in the diet and type of carbohydrate consumed.

Leveille and Sauberlich (34) conducted a study in which rats were fed pectin in a high-cholesterol diet. Carbon-14 labelled cholesterol absoption was depressed by the dietary pectin. The investigators further believed that the majority of the hypocholesterolemic effect was due to an increased fecal bile excretion with the high pectin,

high carbohydrate diet.

In a similar study in which the type of fiber was not defined Balmer and Zilversmit (35) found no inhibition of carbohydrate absorption by the presence of high-fiber vs. purified diets. They did, however, find a decreased plasma carbohydrate concentration and attributed the difference to an increased fecal steroid excretion. In a rat study (36) using lignin vs. cellulose and a low-fiber diet, rats fed lignin showed reduced bile acid absorption compared to the other two diets. This reduced bile acid absorption did not result in a lower cholesterol level in the serum however.

Jenkins (37), in 1975, compared the effects of pectin, guar gum and wheat fiber on serum cholesterol. The mean serum carbohydrate levels fell by 36.6 mg per 100 ml while the human subjects were taking guar gum and a fall of 29.2 mg per 100 ml was seen in subjects receiving pectin. With wheat fiber a rise of 6.7 mg per 100 ml was seen in serum cholesterol. In another study Jenkins (38) found no change in serum cholesterol and triglycerides with guar gum. The difference in the results of these two studies was not explained.

In 1979 Jenkins (18) tested dietary guar gum in patients with type II (a or b) hyperlipidemia and found a fall of 10.6% ($P < 0.01$) in serum cholesterol. No change was seen in serum triglyceride levels. Jenkins et al (39) has further concluded that the fecal bulking action of dietary fiber is independent of its hypocholesterolemic

effect.

The administration of guar gum has been a problem for researchers due to its difficulty of incorporation into acceptable food products. In most preparations it has been unpalatable and often resulted in nausea. This problem has been approached with some success by incorporation into a crispbread which has been reported to have the same cholesterol lowering effects as other guar preparations (84).

Pectin, citrus pulp, soy hulls, rice hulls and wheat bran were compared for their ability to increase bile acid and sterol loss in feces of rats. The greatest effect was seen in rats fed pectin and to a lesser extent citrus pulp. Soy hulls, rice hulls and wheat bran had little effect on fecal bile acid and sterol excretion (41).

The effect of wheat bran on cholesterol metabolism is not clear. In a human study in which 0.5 g wheat bran per kg body weight per day for four weeks was given, a decrease in serum cholesterol and triglycerides of 10% and 24% respectively were noted (42). In many studies no effect of wheat bran is seen on serum or plasma cholesterol but the type of wheat bran is not always given (9, 43, 44). The type of wheat could be responsible for the variation in reported effects of wheat bran on cholesterol metabolism. A human study by Munoz (45) found a 12% reduction in plasma cholesterol with hard red spring wheat bran and no significant effects from soft white wheat (AACC) bran.

Van Beresteyn et al (19) studied lipid metabolism in

obese Zucker rats. They found no effect of wheat bran (type not indicated) on plasma cholesterol though an increased rate of cholesterol and bile acid excretion was noted. This suggests a compensatory mechanism of increased hepatic cholesterol synthesis on changes within the cholesterol pools in the body.

In cholesterol fed rats, fibrous mill-fractions, shorts and bran, were effective in preventing the elevation of plasma and liver cholesterol levels (46).

Hepatic Lipogenic Enzyme Activity

One of the primary metabolic drives in the body in times of excess caloric intake is the conversion of surplus carbohydrate to fat for storage in large and expandable adipocytes. These adipocytes are located in characteristic locations throughout the body. The majority of the carbohydrate to fat conversion occurs in hepatocytes. From the liver this lipid is tranfered via the blood to the storage depots or adipocytes. The resulting increase in blood lipids may be in itself pathogenic. The other obvious result is obesity.

The conversion of carbohydrate to fat occurs when large amounts of citrate build up in the mitochondria. The citrate is exported to the cytoplasm where citrate lyase cleaves it into exaloacetate and acetyl Co A. The oxaloacetate is converted to malate by malate dehydrogenase in the cytoplasm. The acetyl Co A is immediately available for

fatty acid production. This biosynthesis requires reduced NADP. NADPH is formed in the pentose phosphate pathway which occurs in the cytoplasm of hepatocytes, erythrocytes, adipocytes, cells of the kidney and to a lesser extent in skeletal muscle. NADPH is also produced in the cytoplasm when malate is oxidized in the presence of malic enzyme. The regulation of the pentose phosphate pathway occurs mainly where D-glucose-6-phosphate is dehydrogenated. This enzyme is increased when excess cellular glucose is present as in high carbohydrate diets.

Three enzymes which are indicative of lipogenesis are: malate dehydrogenase (E.C. 1.1.40), citrate lyase or citrate cleavage enzyme, and D-glucose-6-phosphate dehydrogenase (E.C. 1.1.1.49). For instance, a ten fold increase of glucose-6-phosphate dehydrogenase is noted in the liver when a person passes from a starved state to an overfed state of adequate carbohydrate (47).

The incorporation of fiber into the diet may alter the lipids in the body from both intrinsic and extrinsic sources. In addition, sucrose and amylose could produce different levels of lipogenic enzyme activity.

In 1974 Heaton and Pomare (48) found a significant lowering of serum triglycerides in fourteen human subjects fed unprocessed wheat bran for four to nine weeks. The level of bran fed was a median dose of 38 grams per day. In contrast, Connell (49) found no such decrease in serum lipids in a similar human study. Jenkins (38) in 1975,

using six human subjects fed 36 g of wheat bran fiber per day for three weeks also found no change in the serum levels of triglyceride. Some other factor may be responsible for the disparity of results under seemingly similar circumstances.

A study using both diabetic and non-diabetic rats (9) and including 10% Solka-floc, Alphacel, wheat bran, alfalfa, sugar cane pulp, or sugar beet pulp found that even though diabetic rats had double the values for serum triglycerides as compared to normal rats, no differences in triglycerides could be attributed to fiber in either diabetic or control rats. But when carbohydrate is associated with fiber as in the starch-bran diets used by Anderson and Lin (2), as previously described, there is a lower plasma and liver triglyceride level reported in both control and diabetic rats. In addition the hepatic lipogenic enzyme activities in both normal and diabetic rats were decreased in animals fed the starch-bran feeds.

Anderson (50) applied the results from the rat study to a group of human patients with diabetes mellitus and vascular disease and found that the high carbohydrate (75%) diet with 70 grams of dietary fiber per day significantly decreased plasma triglycerides. But when the effects of fiber alone are considered the results are negative. Lin and Anderson (44) continued their studies and concluded that neither guar gum nor wheat bran lowers fasting triglycerides in the blood. This was supported by a study where

wheat bran fed to rats elicited no significant change in triglycerides over a one year test period (10).

One study, using diabetic patients on a control diet for 7 days followed by a high-carbohydrate and high-fiber diet for 16 days, found fasting serum triglycerides were not altered significantly on the high-carbohydrate and high-fiber diet (15). In contrast the same researcher two years earlier found that high-carbohydrate high-fiber diets lowered plasma triglycerides in similar patients (50). The differences were not explained.

Fasting serum triglycerides dropped approximately 15% when subjects were fed a high-fiber bread (12 slices daily) (51). But this figure was insignificant due to the high level of variability among the subjects.

Different fibers could have different effects on lipid metabolism. This is reasonable since the source of fiber has been shown to effect other parameters of digestion and metabolism differently. In another study by Lin Chen and Anderson (52) in which cholesterol-fed rats received either pectin, guar gum, oat bran or cellulose, triglycerides were lowest in rats receiving guar gum, oat bran and pectin compared to those fed cellulose ($P < 0.05$).

In summary, the effect of fiber and carbohydrate source on lipid metabolism is not agreed upon by researchers in the field.

METHODS AND PROCEDURES

Selection and Treatment of Animals

Experimental Protocol

This study was designed to investigate the effects of wheat bran fiber and carbohydrate type on glucose tolerance, serum cholesterol, hepatic lipogenic enzyme activity, food efficiency and fecal weight in male weanling rats. Food intake and body weight were also recorded during the study.

To achieve the stated objectives, three levels of dietary fiber: 0%, 6%, and 18% and two types of carbohydrate: cornstarch (amylose) or sucrose were combined in a 3 x 2 factorial arrangement with ten male weanling Sprague-Dawley rats[1] in each of the six dietary groups. This protocol is outlined in Table 1.

TABLE 1

EXPERIMENTAL PROTOCOL

Group	Fiber Level	Carbohydrate Type	Number of Rats
A	Low	Sucrose	10
B	Low	Amylose	10
C	Medium	Sucrose	10
D	Medium	Amylose	10
E	High	Sucrose	10
F	High	Amylose	10

[1]Gibco Animal Resource Laboratory, Madison, Wisconsin

The animals were individually housed in stainless steel cages with elevated stainless steel mesh floors and fronts. All rats were fed and watered ad libitum using inverted glass water bottles with rubber stoppers and stainless steel watering tubes. Stoppers were protected from the rats by a stainless steel plate attached to each cage. Feed cups were stainless steel with a stainless steel grid plate placed on top of the food to reduce waste. The grid plates were removed from the cups on the thirteenth day of the study because of injuries incurred by three of the rats. Damage to the premaxilla and the maxillary gingiva of the three rats required removal from the study. The feed cups were fastened to the fronts of the cages with spring type clips. Trays under the cages were lined with newspaper which was kept beyond the reach of the animals. The rats were randomly arranged in the cage rack.

The initial weight of each rat was recorded and subsequently noted every second day of the study. The mean starting weight of the rats used was 42.1 g with a standard deviation of 3.2 g and a range of 36.5 to 48.5 grams. The rats were blocked according to weight and assigned to dietary groups.

Laboratory lights were automated to turn on at 0700 and turn off at 1900. The temperature of the laboratory was kept at 24°C $\pm$ 4°C.

On the 28th day of the study the feed cups were

removed from the cage of one rat from each dietary group. These six rats were fasted for 12 hours prior to glucose tolerance testing (gtt). Following the fast, rats were moved to galvanized cages in a separate room for the gtt. After the gtt the rats were returned to their original cages and their original diets for seven days. Rats were then again fasted for 12 hours and a final blood sample was obtained at the time of sacrificing. Liver tissue was sampled for hepatic lipogenic enzyme activity. This procedure was repeated for all 60 rats.

Diets

Rats were fed purified diets prepared in advance. These diets were stored in polyethylene bags and refrigerated. Diets were formulated to achieve equal levels of protein, fat, minerals, and vitamins by mass in each diet. The major variables were fiber and carbohydrate levels and caloric density of the diets. Fiber and carbohydrate were manipulated to achieve diets with dietary fiber levels of 0%, 6%, and 18%. Diet compositions and calculations are shown in Tables 2 and 3 respectively.

Modifications in the amount of casein, corn oil, beef tallow, and carbohydrate were made whenever necessary to account for the lipid (5.22%), protein (14.3%), and absorbable carbohydrate (40.26%) contained in the bran. A complete analysis of the wheat bran is found in the appendix. The calculations of the protein, lipid, carbohydrate,

and fiber contents of each of the diets is shown in Table 3. Also shown is the caloric density of each diet.

The animals were started on the experimental diets at the same time but later testing procedures were staggered over a ten day period for convenience.

Tissue Sample Collection

Plasma for glucose tolerance. After the 12 hour fast approximately 2 ml of tail blood was drawn from the tail of each rat by lancing the tip of the tail longitudinally with a scapel. The wound was approximately 5 mm long. The blood was allowed to flow into heparinized tubes. This sample served as the fasting blood sample at 0 minutes. Following the initial sample the rats were injected with 4.2 mg sodium pentobarbital[1] in 0.07 cc solution per 100 g body weight and were administered 350 mg glucose[2] per 100 g body weight in a 35% solution with distilled water. The anaesthetic was injected intraperitoneally and the glucose solution was fed orally using a syringe with attached tygon tubing. Each rat was timed separately and blood samples similar to the first sample were drawn every 30 minutes. These tests corresponded to 0, 30, 60, and 90 minutes post-prandial. The blood thus obtained was centrifuged within

---

[1]60 mg sodium pentobarbital in 10% alcohol, 40% propylene glycol and water to 1 ml Diabutal™ from Diamond Laboratories Incorporated, Des Moines, Iowa 50304.

[2]Certified A.C.S. Dextrose (anhydrous), Fisher Scientific Company, Fairlawn, New Jersey 07410.

TABLE 2

DIET COMPOSITION

Dietary Component[1]	Diet[2] (grams/100 grams)					
	A	B	C	D	E	F
Vitamin Free Casein	22.50	22.50	20.33	20.33	15.87	15.87
dl Methionine	0.20	0.20	0.20	0.20	0.20	0.20
Beef Tallow	5.00	5.00	4.77	4.77	4.31	4.31
Corn Oil	5.00	5.00	4.77	4.77	4.31	4.31
Sucrose	62.30	---	49.83	---	25.00	---
Cornstarch	---	62.30	---	49.83	--	25.00
Wheat Bran[3]	---	---	15.10	15.10	45.30	45.30
Vitamin Mix[4]	1.00	1.00	1.00	1.00	1.00	1.00
Mineral Mix[5]	4.00	4.00	4.00	4.00	4.00	4.00

1. Diet components from Teklad Test Diets, Madison, Wisconsin 53711
2. A = 0% Fiber Sucrose D = 6% Fiber Amylose
   B = 0% Fiber Amylose E = 18% Fiber Sucrose
   C = 6% Fiber Sucrose F = 18% Fiber Amylose
3. A.A.C.C. certified foodgrade wheat bran from American Association of Cereal Chemists, St. Paul, Minnesota 55121
4. Vitamin Mix #40060, Teklad
5. Williams Briggs Modified Mineral Mix #71709, Teklad

TABLE 3

DIET CALCULATIONS

Nutrient	0% Fiber Diets	6% Fiber Diets	18% Fiber Diets
Protein	20.55% of Cal.	21.74% of Cal.	24.58% of Cal.
Casein	21.94 g/100 g	19.83 g/100 g	15.48 g/100 g
Wheat Bran	---	2.16 g/100 g	6.65 g/100 g
Fat	21.08% of Cal.	23.00% of Cal.	27.39% of Cal.
Corn Oil and Beef Tallow	10.00/g/100 g	9.55 g/100 g	8.6 g/100 g
Wheat Bran	---	0.79 g/100 g	2.36 g/100 g
Absorbable Carbohydrate	58.37% of Cal.	55.25% of Cal.	48.03% of Cal.
Cornstarch or Sucrose	62.30 g/100 g	49.80 g/100 g	25.00 g/100 g
Wheat Bran	---	6.08 g/100 g	18.25 g/100 g
Dietary Fiber			
Wheat Bran	---	6.07 g/100 g	18.21 g/100 g
Total Kcal/100 g	426.96	404.54	360.16

30 minutes after drawing. Centrifugation continued for 10 minutes at no less than 3500 RPM in a centrifuge[1] with free swinging buckets. The plasma was drawn off the packed cells and tested immediately for glucose.

Plasma for Cholesterol Testing

Seven days after the glucose tolerance test the rats were fasted 12-hours and sacrificed by cardiac exsanguination. No anaesthesia was used in this case because such compounds are normally detoxified in the liver causing an alteration in the liver enzymes (53). Blood for fasting plasma cholesterol was obtained from the rats by cardiac puncture. The blood was drawn by syringe with either an 18 or 20 gauge needle, placed in centrifuge tubes and centrifuged for 10 minutes at a minimum of 3500 RPM. The plasma was removed, placed in plastic test tubes and sealed with plastic caps and parafilm. The tubes of plasma were then frozen at -10°C for 2 months until analyzed for cholesterol.

Liver Biopsy for Tissue Sample

The liver was removed, rinsed immediately in ice water and placed in a small plastic petri dish on ice. Approximately 1 gram of the liver was homogenized in cold 0.25 M sucrose solution using a mechanical homogenizer.[2]

---

[1] IEC Clinical Centrifuge, Damon/IEC Division, Needham Heights, Massachusetts.

[2] Brinkman homogenizer (PT 10-35) with a stainless steel probe.

This solution was centrifuged[1] at 26,500 x g for 30 minutes at 4°C. The cytosol fraction was placed in individual plastic test tubes with sealing plastic caps and parafilm and frozen at -10°C for 3 months before assay for lipogenic enzyme activity.

Chemical Analysis

Glucose Determination

Plasma was tested for glucose using the glucose oxidase method of Fales (78-a) as modified by Cooper and McDaniel (54). The reaction was as follows:

1. $C_6H_{12}O_6 + H_2O + O_2 \xrightarrow{\text{glucose oxidase}} C_5H_6(OH)_5COOH + H_2O_2$

2. $H_2O_2 + C_6H_3(OCH_3)NH_2 \xrightarrow{\text{peroxidase}}$ oxidized chromogen + H_2O

3. Reaction is stopped with H_2SO_4(6N).

Proteins were removed from the plasma using barium hydroxide[2] and zinc sulfate[3] reagents. After centrifuging for 10 minutes at 3500 RPM the supernatant was removed and treated with a reagent containing glucose oxidase[4] which converted the D-glucose in the clear filtrate to gluconic acid. The resulting hydrogen peroxide was changed to oxygen and water by another enzyme in the solution,

---

[1] Sorvall automatic refrigerated centrifuge (Model RC2-B).

[2] Solutions of 0.12N barium hydroxide ($Ba(OH)_2 \cdot 8H_2O$) in free distilled water.

[3] Solution of 2.2% zinc sulfate ($AnSO_4 \cdot 7H_2O$) in CO_2 free distilled water.

[4] A 200 ml alequot of solution contained 250 mg glucose oxidase (Sigma Chemical, Type II).

peroxidase.[1] The oxygen thus released bonded to the orthodianisidine[2] a chromogenic compound also in the reagent. The intensity of the resulting color was proportional to the amount of oxygen released and therefore to the amount of glucose present in the original sample (54). The rose-pink color which resulted was measured in a spectrophotomer[3] at 540 mu at 25°C.

Total Cholesterol

Plasma samples from the sacrificed rats were removed from the refrigerator (-10°C) two months after autopsy and tested for total cholesterol using the ferric chloride - sulfuric acid reaction method of Tietz (55).

Proteins were precipitated and cholesterol dissolved by addition of isopropyl alcohol, mixing and centrifuging. Color development was achieved by addition of 3.0 ml glacial acetic acid and 0.3 ml iron reagent[4] to 1.0 ml of the isopropyl alcohol with dissolved cholesterol. At 30 second

---

[1] A 200 ml alequot of solution contained 50 mg peroxidase. RZ 0.3 (Sigma Chemical Company).

[2] One ml of a 1% ortho-dianisidine-methanol indicator solution was added to the enzyme solution. Sigma Chemical Company, St. Louis, Missouri 63178.

[3] Coleman double-beam spectrophotometer (124C) with autosampler (124-20000).

[4] Iron reagent was prepared by mixing 2.5 grams ferric chloride (lump) in 100 ml phosphoric acid with mortar and pestle. Phosphoric acid assay 85%.

intervals 3.0 ml concentrated sulfuric acid was added to one tube at a time and mixed with a vortex mixer.[1] After cooling for 10 minutes the absorbance was read against a reagent blank at 560 mu in an auto sampling spectrophotometer.[2]

Glucose-6-Phosphate and 6-Phosphogluconate dehydrogenasees

Hexose monophosphate shunt dehydrogenase activity (HMPD) was determined by the process of Glock and McLean (56). Reduction of NADP by shunt enzymes was measured by the change in optical density over time. The combined activity of D-glucose-6-phosphate, E.C. 1.1.1.49 and 6-phospho-D-gluconate, E.C. 1.1.1.44 was measured. The reactions were as follows:

Glucose-6-phosphate + NADP $\longrightarrow$ 6-phosphogluconate + NADPH + H.

6-phosphogluconate + NADP $\longrightarrow$ ribulose-5-phosphate + CO_2 + H + NADPH.

Glucose-6-phosphate + NADP $\longrightarrow$ 6-phosphogluconate + NADPH + H.

Reagent concentrations and volumes are listed in Table 5. Addition of glucose-6-phosphate started the reaction. A blank minus NADP was used for each determination.

---

[1] Thermolyne Mini Mix (Model M-16715).

[2] Coleman double-beam spectrohotometer (Model 124C) with autosampling attachment, Coleman (124-20000).

TABLE 4

REAGENT CONCENTRATIONS AND VOLUMES FOR GLUCOSE-6-PHOSPHATE DEHYDROGENASE AND 6-PHOSPHOGLUCONATE DEHYDROGENASE ACTIVITY

Reagent[1]	Concentration	Volume/Curvette Sample	Blank
Glycylglycine Buffer	0.25 M pH 7.6	0.5 ml	0.5 ml
$MbCl_2$	0.10 M	0.5	0.5
NADP	0.0015 M	0.2	---
Glucose-6-Phosphate	0.05 M	0.1	0.1
Enzyme Homogenate	1 - 10 dilution[2]	0.1	0.1
Distilled Water		1.1	1.3
Total Volume		2.5	2.5

1. Sigma Chemical Company, St. Louis, Missouri 63178.

2. This dilution was omitted but compensated for in later calculations.

NADP - malate dehydrogenase

The activity of NADP - malate dehydrogenase, E.C. 1.1.1.40, was determined by the method of Ochoa (57). Reagent concentrations and volume are shown in Table 6. The reaction for the determination was:

$$\text{L-malate}^- + \text{NADP}^+ \xrightarrow{Mn^{++}} \text{Pyruvate}^- + CO_2 + \text{NADPH} + H^+.$$

TABLE 5

REAGENT CONCENTRATIONS AND VOLUMES FOR MALIC ENZYME

Reagent[1]	Concentration	Volume/Curvette Sample	Blank
Glycylglycine Buffer	0.25 M pH 7.4	0.30 ml	0.30 ml
$MnCl_2$	0.05 M	0.06	0.06
NADP	0.000675 M	0.24	0.24
Malate	0.030 M pH 7.4	0.10	---
	1 - 10 dilution[2]	0.10	0.10
Distilled Water		2.20	2.20
Total Volume		3.0	3.0

1. Sigma Chemical Company, St. Louis, Missouri 63178.

2. This dilution was omitted but compensated for in later calculations.

Statistical Analysis

A 3 x 2 factorial design with three levels of fiber and two sources of carbohydrate was used in this study. Data was analyzed by computer for analysis of variance using an F-distribution with 5 degrees of freedom (58).

RESULTS AND DISCUSSION

Body Weight Gain, Feed Intake, Food Efficency Ratio and Fecal Weights

Body weight gain. There were differences noted in the ending body weights of the dietary groups. The rats were initially blocked by weight so beginning mean group weights were not significantly differnt. Beginning and ending weights are seen in Table 6 and body weight gain appears in Figure 1 and Table 6. Mean daily weight gain was higher in low-fiber animals than high-fiber animals ($P < 0.005$). The different types of carbohydrate used in the diets resulted in no significant differences in weight gain.

These findings support the hypothesis of Trowell (59) that high-fiber helps prevent obesity and contradicts findings by Johnson and Chang (60) and Silman (61).

This study found a moderate effect of fiber in reducing body weight gain and no effect from carbohydrate source on weight gain.

Feed intake. Measured semi-daily, feed intake was highest in rats fed high-fiber diets when compared to medium-fiber diets ($P < 0.05$) and low-fiber diets ($P < 0.05$) measured in grams per day. When measured by volume rather than mass, feed intake differences were more marked with high-fiber fed rats consuming more than medium-fiber ($P < 0.0001$) and low-fiber ($P < 0.0001$) fed rats. In addition, when measured by volume medium fiber diets were

TABLE 6

INITIAL AND FINAL BODY WEIGHT AND WEIGHT GAIN

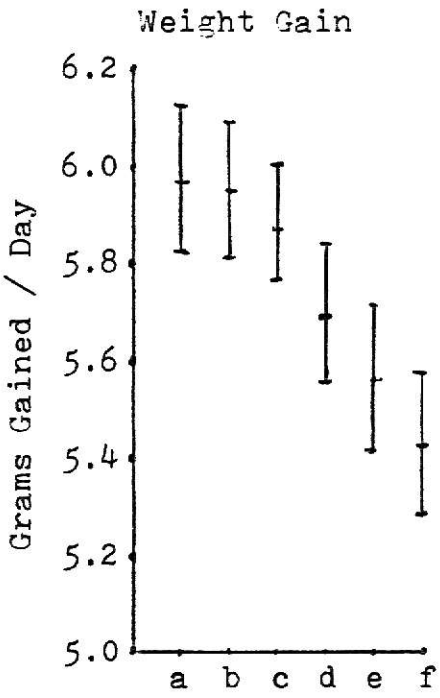
(Mean ± Standard Error, N = 10)

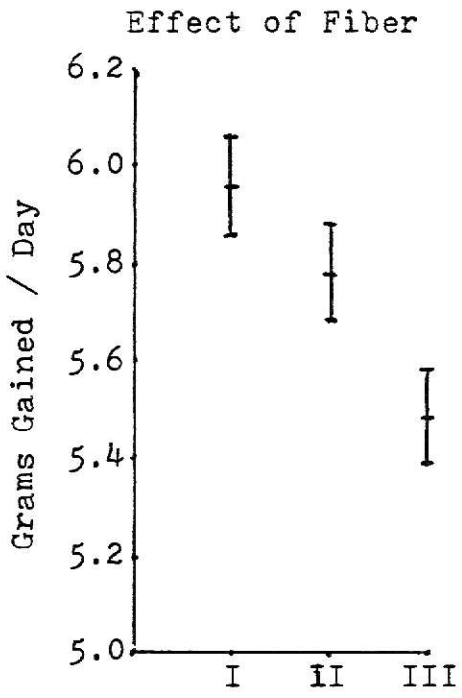
Diet	Initial Weight	Initial Weight	Weight Gain grams/day[2]
Low Fiber			
Sucrose[1]	42.0 ± 0.2	277.9 ± 11.9	6.0 ± 0.2b
Amylose	41.9 ± 0.2	258.4 ± 1.11	6.0 ± 0.2[b]
Medium Fiber			
Sucrose	42.0 ± 0.2	271.8 ± 11.1	5.9 ± 0.2[b]
Amylose	42.0 ± 0.2	256.1 ± 11.1	5.7 ± 0.2
High Fiber			
Sucrose	42.6 ± 0.2	261.4 ± 11.1	5.6 ± 0.2
Amylose	41.8 ± 0.2	246.9 ± 11.1	5.4 ± 0.2[a]

1. n = 1

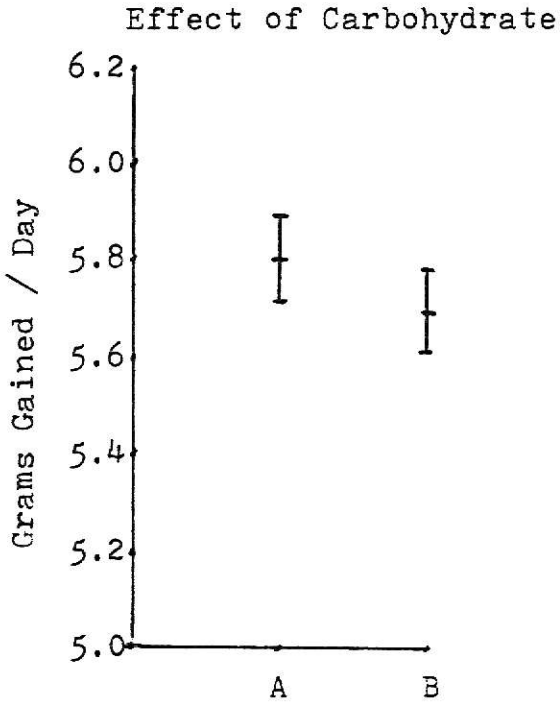
2. Values in the same column with different superscripts are significantly different at the $P < 0.05$ level.

Figure 1. Mean Daily Weight Gain





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets



consumed in greater quantities than low-fiber diets ($P < 0.01$).

When feed intakes were compared on the basis of caloric consumption per day the above situation was reversed due to the greater caloric density of low-fiber diets. High-fiber diets provided the fewest calories per day followed by medium-fiber diets and the most calories per day were consumed by rats on the low-fiber diets ($P < 0.05$). Caloric consumption was determined by calculation of calories per gram of feed and then multiplied by the grams of feed consumed per day.

Rats on a high-fiber diet tend to consume a greater volume of food and a greater mass of food than rats on lower fiber diets and at the same time consume fewer calories daily. While this does not support the hypothesis that rats consume food for calories rather than mass or volume it does not contradict that hypothesis either. Animals must consume a greater quantity by mass and volume of high fiber food to achieve the same caloric intake they would receive on a low fiber diet. This higher volume could be greater than the physical capacity of the animal. Rats may eat for calories, however they may also be limited by their own capacities.

Food efficiency ratio. The change in body mass divided by the daily caloric intake when multiplied by 100 is called the food efficiency ratio (FER). The FER is a

measure of the ability of the animal to convert calories to body mass.

In this study low-fiber diets and medium-fiber diets were less efficient than high-fiber diets ($P < 0.05$). This is a result of considerably fewer calories being consumed by high fiber fed rats and a weight gain only slightly lower than low and medium-fiber rats. Rats on a low-fiber diet consumed 56.1 kcal per day and high-fiber fed rats consumed only 45.5 kcal per day.

According to Kelsey et al fecal loss of energy is increased by higher levels of dietary fiber (62). This would depress values of food efficiency for animals on high-fiber diets. This did not prove to be the case in this study. Fat pad measurements were not made in the present study so it is not certain whether the increased weight gain per unit energy intake was due to an increase in lean body mass or to fat deposition. Hepatic lipogenic enzyme activity was measured, however, and will be discussed later in this discussion.

Dry fecal weight. Feces were collected from each animal for one week and desicated to find the average daily dry fecal weight. As expected high-fiber diets resulted in the highest average daily dry fecal weights followed by medium and lastly low-fiber diets ($P < 0.0001$). For comparison, see Table 8 and Figure 6.

TABLE 7

FEED INTAKE BY MASS, VOLUME, AND KILOCALORIES (kcals), DIET CALORIC DENSITY, AND FOOD EFFICIENCY RATIOS (FER)

Diet	Mean Daily Feed Intake g/day	Mean Daily Feed Intake cm^3/day	Mean Daily Feed Intake kcal/day	Diet Caloric Density	FER[3]
Low Fiber[2]					
Sucrose	13.2 ± 0.4	16.5 ± 0.7^{c}	56.9 ± 1.6^{d}	4.2696	10.5 ± 0.4^{c}
Amylose	12.9 ± 0.4^{a}	16.8 ± 0.7^{c}	55.2 ± 1.5^{bd}	4.2696	10.8 ± 0.4^{c}
Medium Fiber					
Sucrose	13.2 ± 0.4	19.3 ± 0.7^{d}	51.9 ± 1.5^{b}	4.0454	11.6 ± 0.4^{abc}
Amylose	13.2 ± 0.4	17.9 ± 0.7^{cd}	52.1 ± 1.6^{b}	4.0454	10.9 ± 0.4^{bc}
High Fiber					
Sucrose	14.2 ± 0.4^{b}	28.9 ± 0.7^{b}	46.1 ± 1.5^{a}	3.6016	12.0 ± 0.4^{ab}
Amylose	13.9 ± 0.4	25.7 ± 0.7^{a}	44.9 ± 1.6^{a}	3.6016	12.2 ± 0.4^{a}

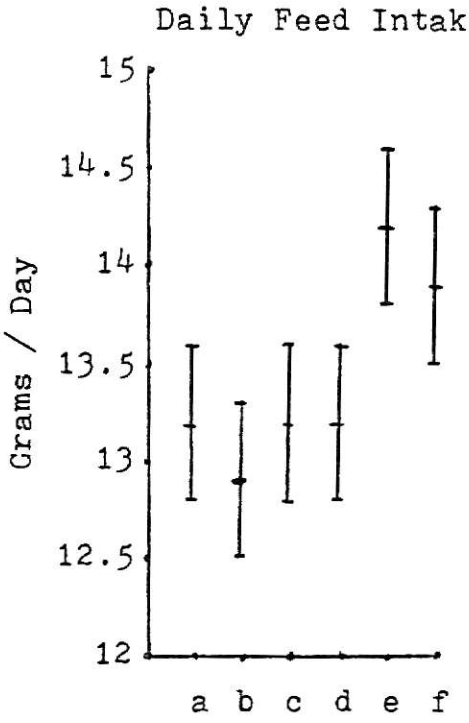
1. Values in the same column with the same superscripts are not significantly different at the $P < 0.05$ level, other values are significant at this level.

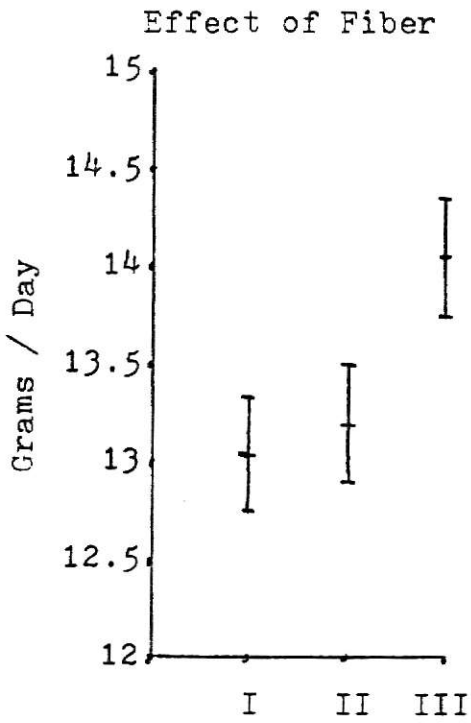
2. n = 9, all others: n = 10

3. FER: food efficiency ratio = $\frac{\text{body mass gain}}{\text{kcal}}$ x 100

4. Experimental period was 41 days.

Figure 2. Daily feed intakes by mass





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets

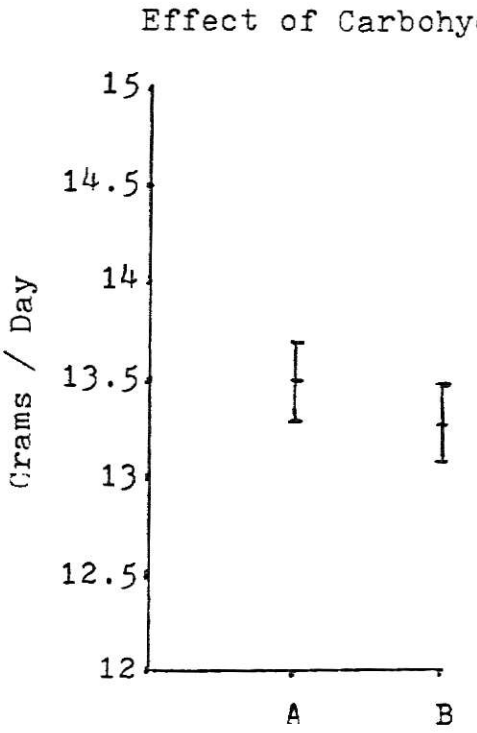
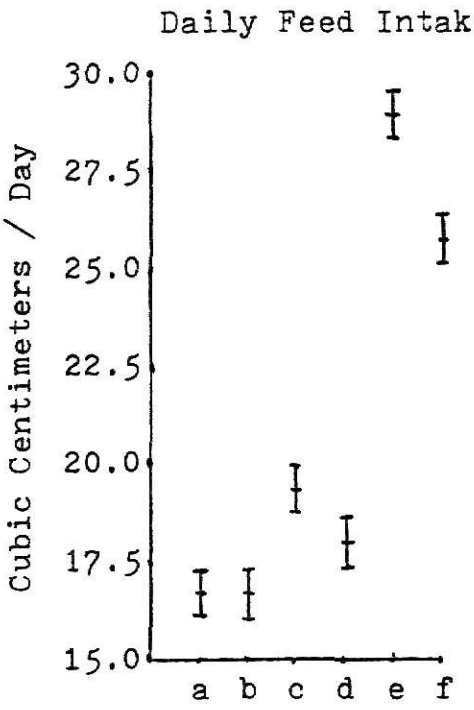
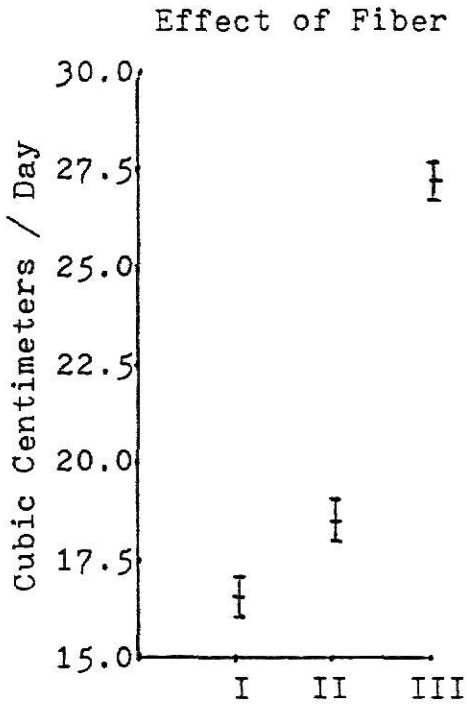


Figure 3. Daily feed intake by volume





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets

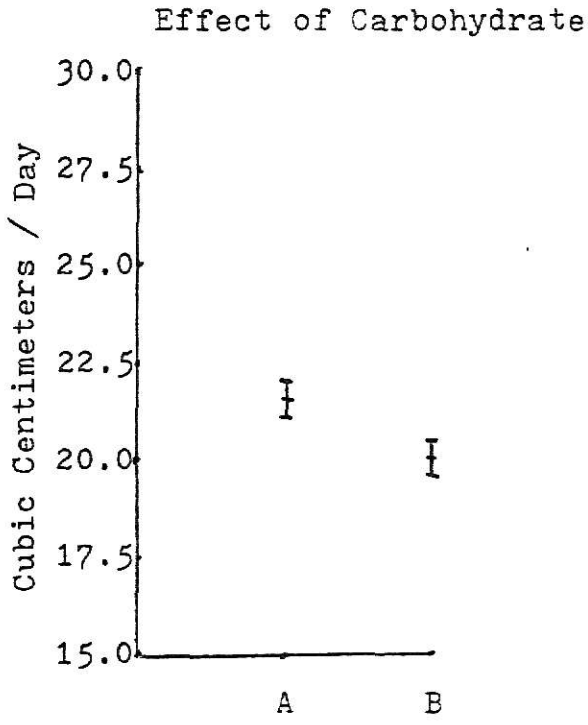
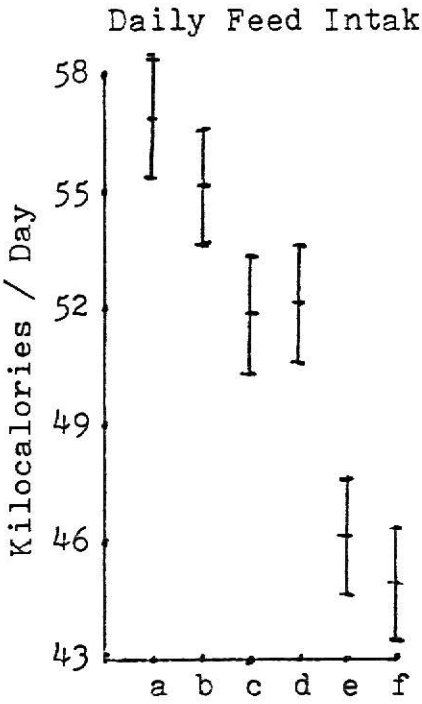
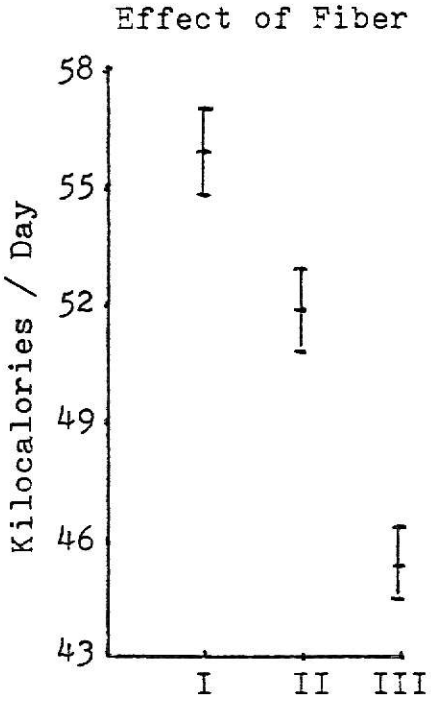


Figure 4. Mean daily caloric consumption





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets

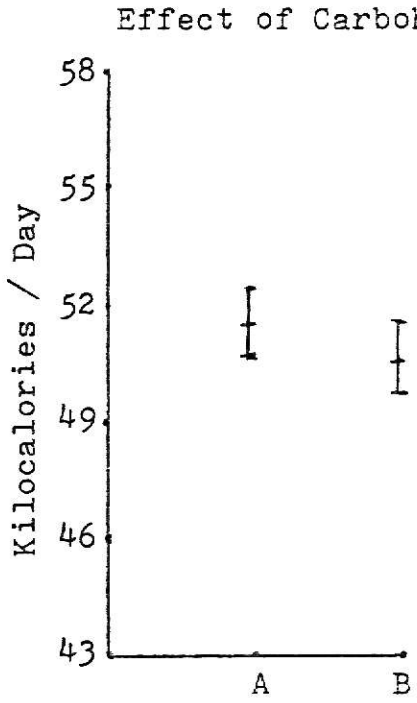
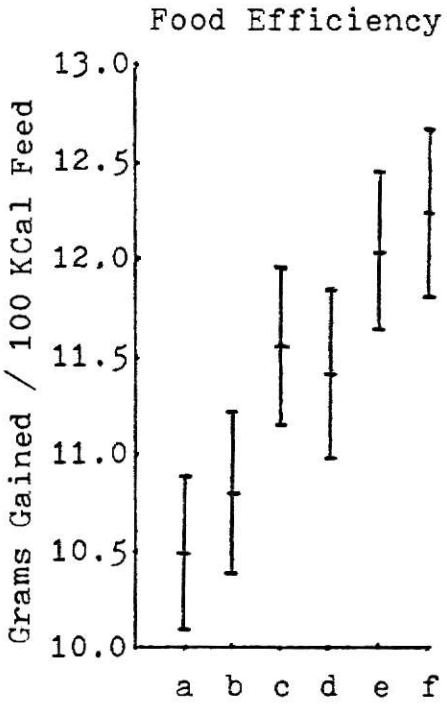
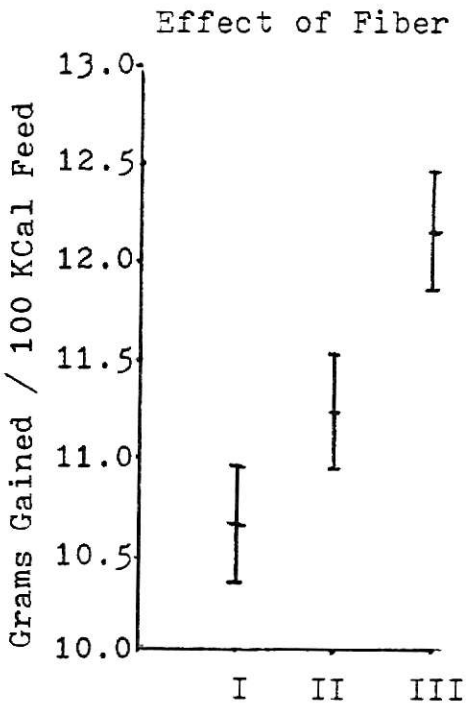


Figure 5. Food efficiency





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets

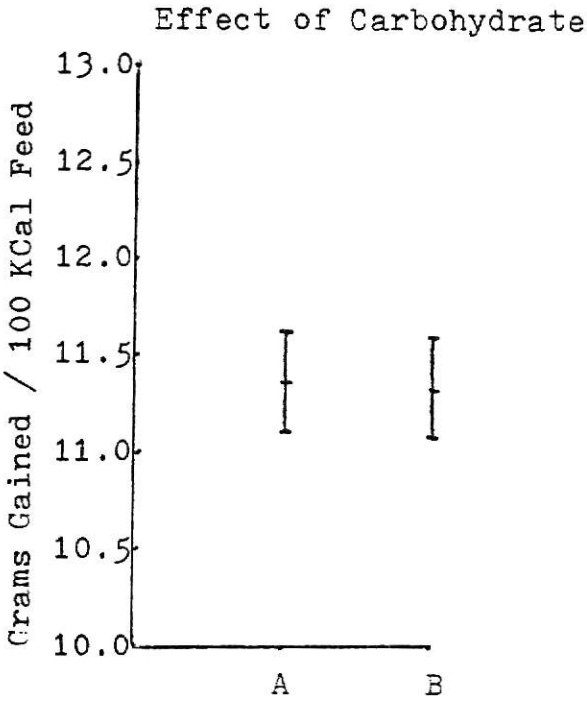


TABLE 8

MEAN DAILY DRY FECAL WEIGHT

(n = 10, fourth week)

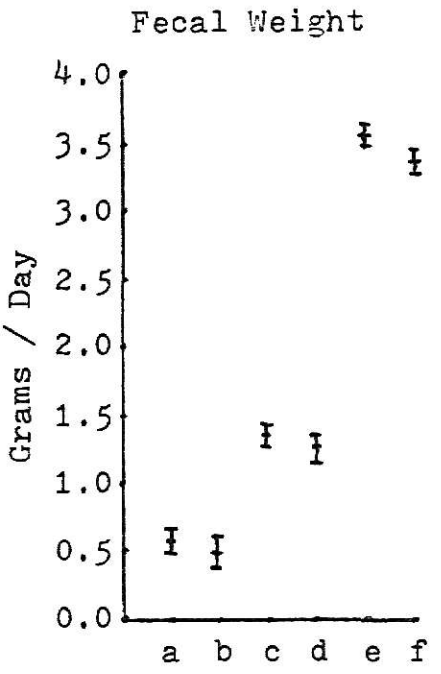
Diet	Fecal Weight in grams/day
A. Low fiber sucrose[2]	0.56 ± 0.08^{b}
B. Low fiber amylose[2]	0.50 ± 0.08^{b}
C. Medium fiber sucrose	1.35 ± 0.08^{c}
D. Medium fiber amylose[2]	1.29 ± 0.08^{c}
E. High fiber sucrose	3.56 ± 0.08^{a3}
F. High fiber amylose[2]	3.32 ± 0.08^{a3}

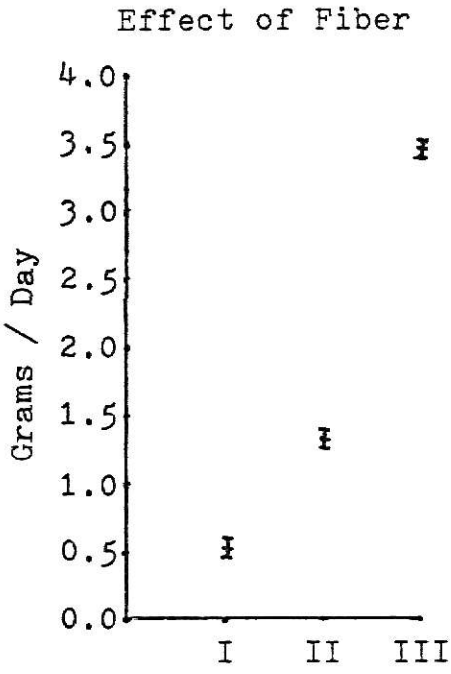
1. Values in the same column with the same superscripts are not significantly different at the $P < 0.0001$ level, other values are significant at this level.

2. n = 9

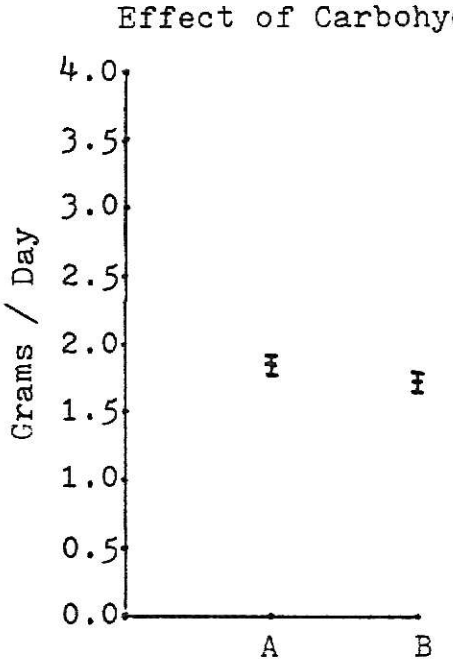
3. High fiber sucrose and high fiber amylose are significantly different at the $P < 0.05$ level.

Figure 6. Mean daily dry fecal weight





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets



The levels of fiber incorporated in the diets were 0%, 6%, and 18%. If the fecal weight of the rats receiving 0% fiber is considered to be obligatory, or the minimum possible fecal output, and that value is subtracted from the fecal weight of the 6% and 18% fiber fed rats, then the 6% and 18% fiber diets are found to yield approximately 1 and 3 grams additional feces per day:

medium fiber rat feces	1.32 g	High fiber rat feces	3.44 g
- low fiber rat feces	0.53 g	low fiber rat feces	0.53 g
	.79 g		2.91 g

0.79 g : 2.91 g is approximately 1 :3

This indicates a direct proportionality between dietary fiber and dry fecal weight.

This study has shown that dry fecal weight is proportional to the level of dietary fiber incorporated into the feed.

Glucose Tolerance, Serum Cholesterol, and Hepatic Lipogenic Enzymes

Glucose tolerance. Glucose tolerance was tested after four weeks on the experimental diets. Results of these tests appear in Table 9 and Figures 7 and 8. Though differences were not significant, amylose fed rats exhibited a lower mean blood glucose than sucrose fed rats at all test periods. The mean blood glucose levels between dietary groups were relatively close at all times but exhibited wide variability between individuals within each dietary group. Low-fiber animals had the lowest blood glucose levels at fasting and 90 minute test times ($P < 0.05$). The low fiber rats also had the lowest blood glucose levels at 30 and 60 minutes postprandial but these differences were not significant.

These results do not concur with the majority of the literature concerning the effects of fiber on glucose tolerance (63, 64, 65, 66, 67, 68, 69).

In a human study by Jeffery's and Macdonald (63) wheat bran significatly ($P < 0.01$) improved glucose tolerance at 60, 90, and 120 minutes compared within subjects to the control. The disparity could be due to more fiber being retained in the human upper small intestine after fasting when compared to rats. This is important because the physiological effects of dietary fiber on parameters of glucose tolerance are thought to be manifested in the upper small intestine since it is here that usable carbohydrate is

TABLE 9

GLUCOSE TOLERANCE DATA[1, 2]
(Four weeks on experimental diets, n = 10)

Diet	0 minutes	30 minutes	60 minutes	90 minutes
Low Fiber				
Sucrose[3]	103.6 ± 11.4	222.4 ± 20.5	157.2 ± 12.0[4]	149.8 ± 9.4
Amylose[3]	84.8 ± 11.4	197.2 ± 20.5	163.7 ± 11.2	128.3 ± 9.4
Medium Fiber				
Sucrose	125.6 ± 10.7	250.5 ± 19.2	187.4 ± 10.6	173.9 ± 8.8
Amylose[4]	105.5 ± 12.2	229.0 ± 21.9	160.8 ± 12.0	145.8 ± 10.0
High Fiber				
Sucrose[3]	113.9 ± 11.4	227.8 ± 20.5	180.2 ± 11.2	151.9 ± 9.4
Amylose[3]	127.2 ± 11.4	224.3 ± 20.5	183.1 ± 11.2	173.8 ± 9.4

1. Glucose administered after 12 hour fast, 350 mg glucose/100 g body weight, in a 35% solution with distilled water.

2. Analyzed by glucose oxidase method of glucose assay (54).

3. n = 9

4. n = 8

Figure 7. Glucose tolerance data by diet

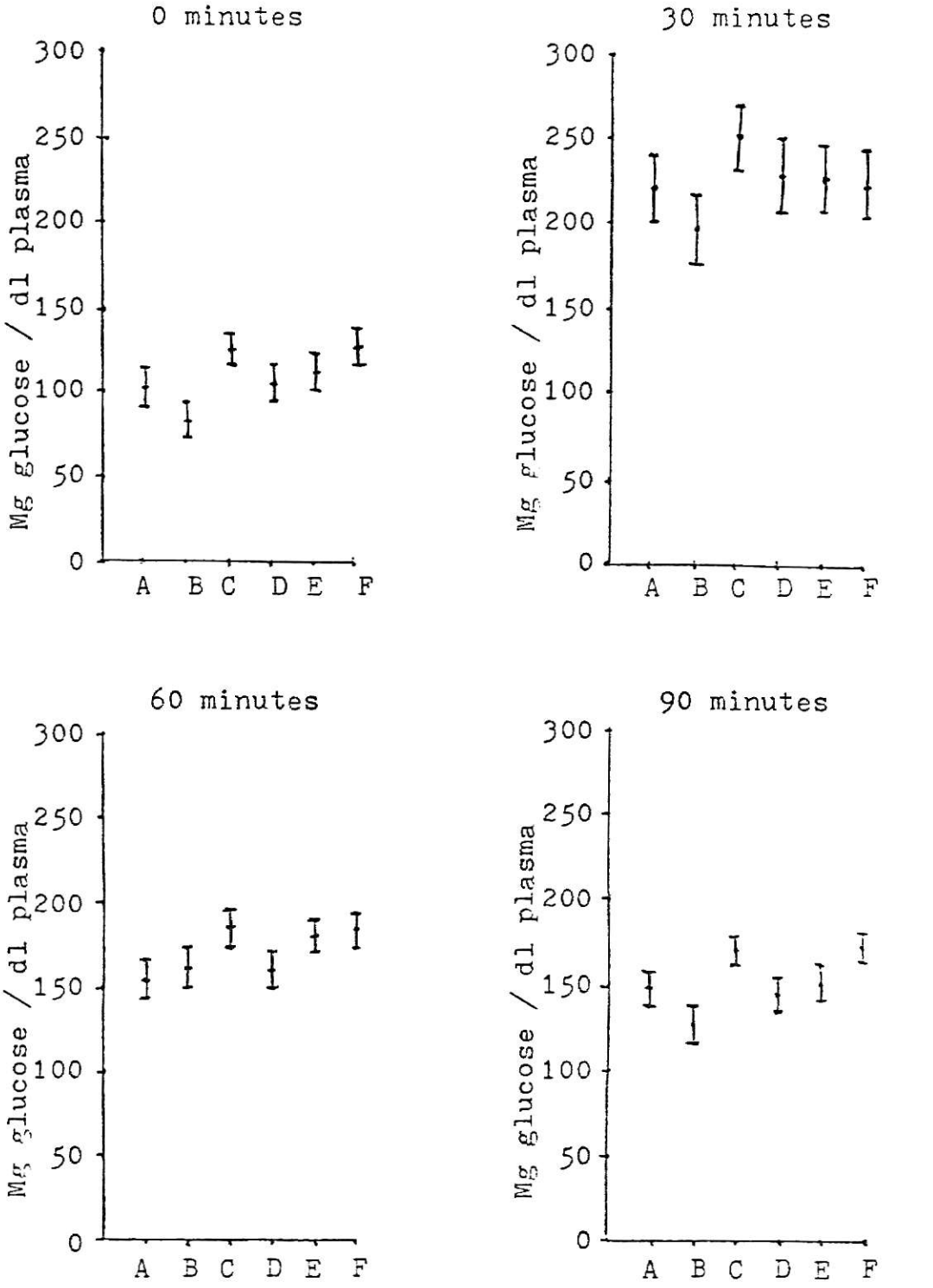


Figure 8. Glucose Tolerance By Carbohydrate Type

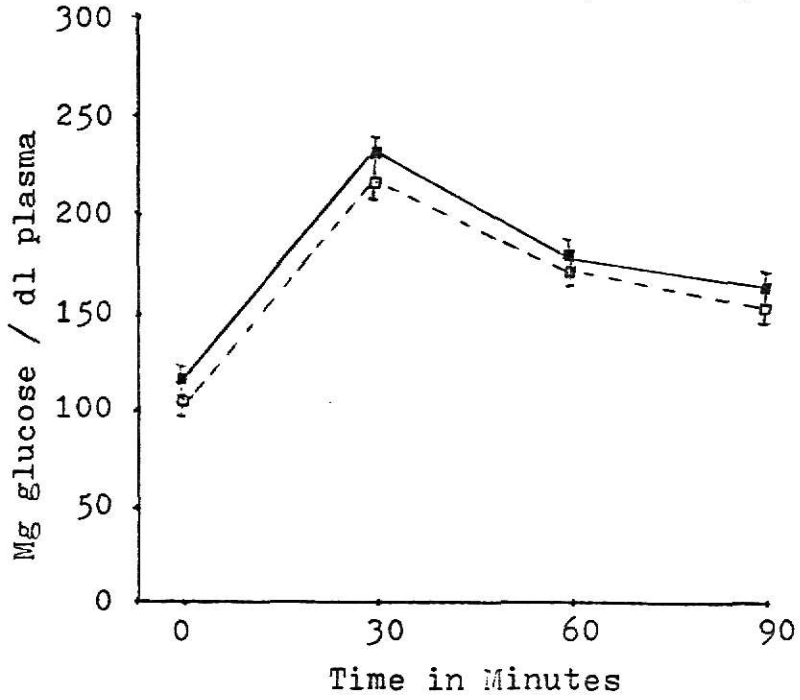
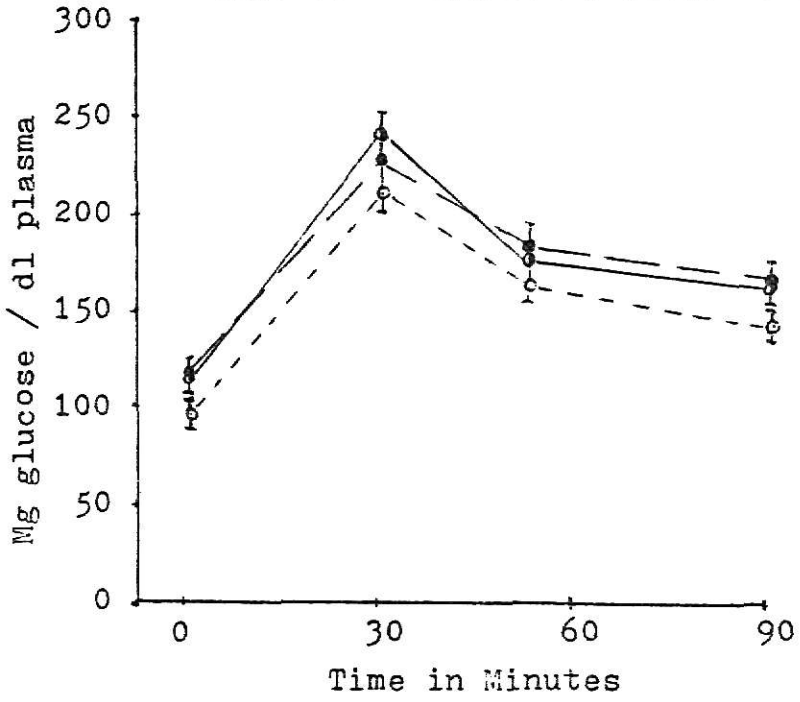
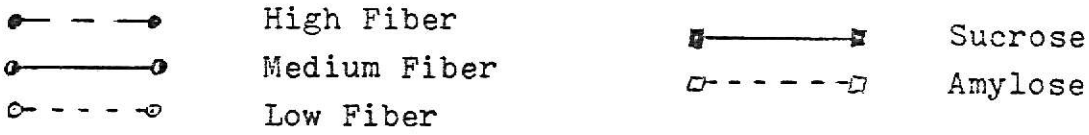


Figure 9. Glucose Tolerance By Fiber Level





absorbed (70). This supports the theory that fiber may decrease the bioavailability of utilizable carbohydrate. The decreased rate of diffusion of available carbohydrate to the absorptive villi of the small intestine is suggested by studies correlating reduction in blood glucose with the viscosity of the fiber ($r = 0.904$, $P < 0.02$) (66). Decreased availability of carbohydrate could prevent the steep increase in postprandial blood glucose and an insulin surge. These high insulin levels, stimulated by high blood glucose levels, could decrease satiety by producing rebound falls in blood glucose and a subsequent urge to consume more food, sooner. More cellular glucose would be shunted through the pentose phosphate pathway because of elevated intracellular levels of glucose. This in turn would increase lipogenesis and favor the deposition of body fat. This will be discussed further in this section under lipogenic enzyme activity in the liver.

Humans adapted to diets high in fiber show improved glucose tolerance despite loss of intralumenal fiber (69, 71). This indicates an effect other than slowed absorption rates from dietary fiber interference. This study failed to indicate any physiological change resulting from increased dietary fiber that would improve glucose tolerance in normal rats.

Serum cholesterol. Serum cholesterol was not altered by increased fiber in the diet. Furthermore, the source of

carbohydrate had no effect on serum cholesterol levels. This result supports findings by other researchers (50). Results of serum cholesterol tests are presented in Table 10 and Figure 10.

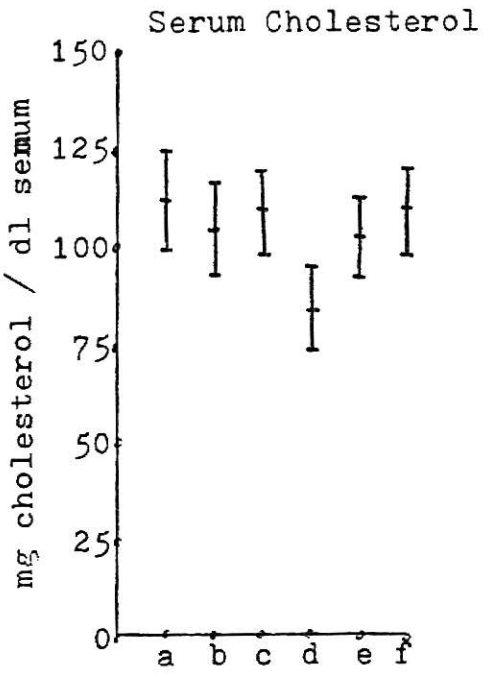
TABLE 10

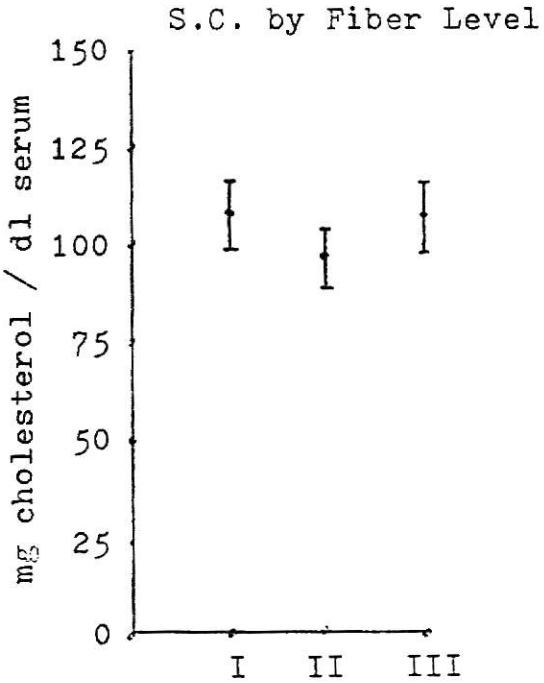
SERUM CHOLESTEROL
(six weeks, n = 10)

Diet	Serum Cholesterol mg/dl[1]
Low fiber	
Sucrose[2]	111.2 ± 12.6
Amylose[3]	105.0 ± 11.6
Medium fiber	
Sucrose[4]	109.2 ± 10.8
Amylose[4]	83.9 ± 10.8
High fiber	
Sucrose	101.9 ± 10.1
Amylose[3]	109.7 ± 11.6

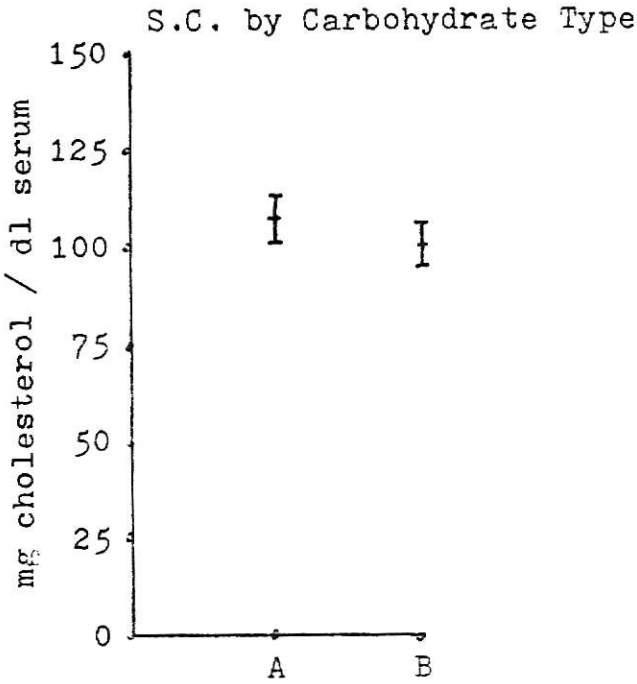
1. No values were significantly different.
2. n = 7
3. n = 8
4. n = 9

Figure 10. Serum Cholesterol Data (S.C.)





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets



Hepatic Lipogenic Enzyme Activity

Liver biopsy and enzyme analysis showed striking difference in both malic enzyme activity and hexose monophosphate shunt dehydrogenases activity between the diets tested.

Hepatic hexose monophosphate shunt dehydrogenases (HMPD). When the change in optical density of the enzyme reaction mixture per minute per gram of tissue was compared across the diets, low fiber sucrose had the greatest HMPD activity and high fiber amylose had the lowest HMPD activity. Fiber directly affected the HMPD activity with the low-fiber diets showing the highest activity, medium-fiber less activity, and high-fiber diets had the lowest HMPD activity ($P < 0.0002$). Low-fiber versus medium-fiber was significantly different at $P < 0.01$. Sucrose fed rats had greater HMPD activity than amylose fed rats ($P < 0.0001$). Results appear in Table 11 and Figure 11.

Hepatic malate dehydrogenase. The results of malate dehydrogenase tests were similar to HMPD test results. Low fiber diets resulted in the greatest activity followed by medium-fiber and lowest activity was seen in high-fiber diets ($P < 0.0005$). Medium-fiber diets resulted in activity that was lower than activity from low-fiber diets ($P < 0.01$). Amylose fed rats had a lower activity than sucrose fed rats ($P < 0.0001$). Results are shown in Table 11 and

Figure 12.

These results support the results of a study by Lin and Anderson (2). In their study Lin and Anderson found a lower activity of both Glucose-6-phosphate dehydrogenase and malic enzyme in rats fed starch-bran ration when compared to rats fed a sucrose diet. These results are not unexpected since sucrose would be more rapidly absorbed than amylose resulting in a more dramatic rise in insulin activity and increased intracellular glucose. Starch is decidedly less lipogenic enzyme stimulating than sucrose. The difficulty in interpretation concerns the effects of fiber.

If fiber is increased in the diet it clearly depresses lipogenic enzyme activity regardless of the nature of the absorbable carbohydrate present. It is difficult to ascribe this difference to any one factor since the change could be due to decreased absorption of carbohydrate or simply because a smaller total volume of carbohydrate is ingested when fiber is increased in the diet. The latter factor is probably the major determinant, however an actual fiber induced depression of lipogensis should not be overlooked as a possibility. The mechanism of such an action could be the reduction of the rate of absorption with a less dramatic increase in enzyme activity and therefore a slower increase in intracellular glucose. This gradual increase in glucose would have less of a stimulatory effect of the lipogenic apparatus.

TABLE 11

HEXOSE MONOPHOSPHATE DEHYDROGENASES (HMPD) ACTIVITY AND NADP-MALATE DEHYDROGENASE (MALIC ENZYME) ACTIVITY

Diet	HMPD Enzyme Activity[1] Mean ± S.E.	Malic Enzyme Activity[1] Mean ± S.E.
Low fiber		
Sucrose[2]	11.20 ± 0.79^{b}	3.71 ± 0.26^{d}
Amylose[3]	6.71 ± 0.84^{cd}	2.04 ± 0.28^{bc}
Medium fiber		
Sucrose	8.90 ± 0.74^{d}	2.82 ± 0.25^{e}
Amylose[3]	4.58 ± 0.84^{ac}	1.30 ± 0.26^{ac}
High fiber		
Sucrose	4.40 ± 0.74^{a}	1.41 ± 0.28^{ab}
Amylose[2]	2.60 ± 0.79^{a}	0.81 ± 0.26^{a}

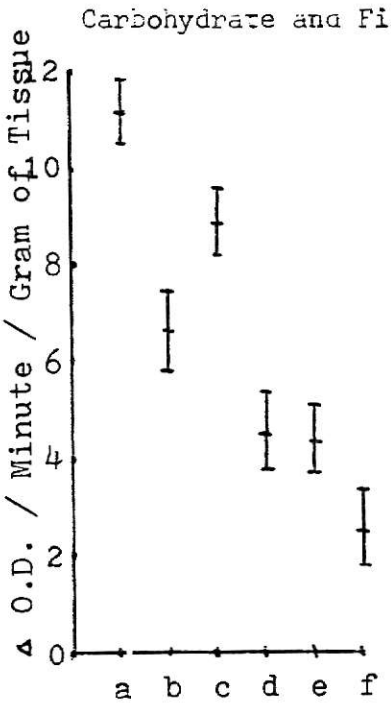
1. Change in optical density/minute/gram tissue

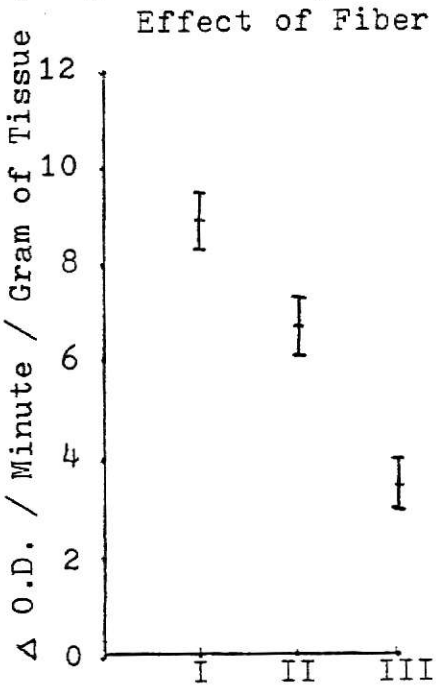
2. n = 9

3. n = 8

Values in the same column with the same superscripts are not significantly different at the $P < 0.05$ level.

Figure 11. Hepatic Hexose Monophosphate Dehydrogenase Activity





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets

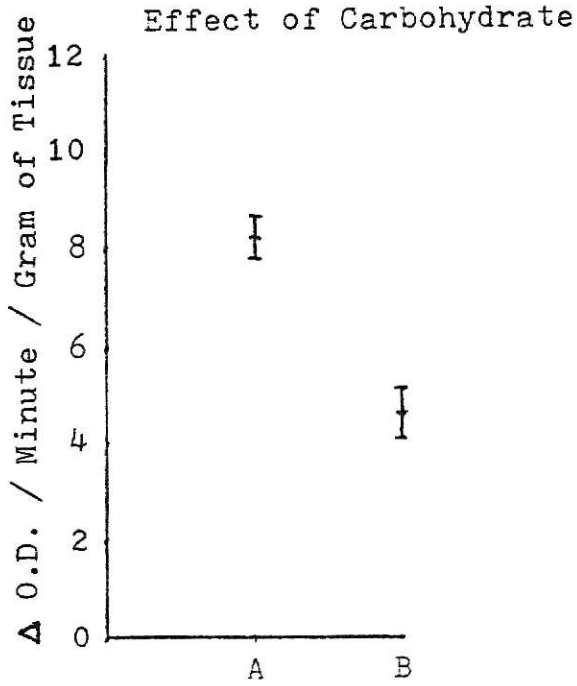
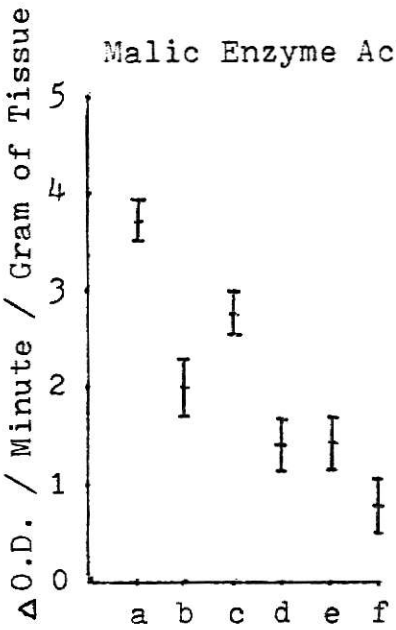
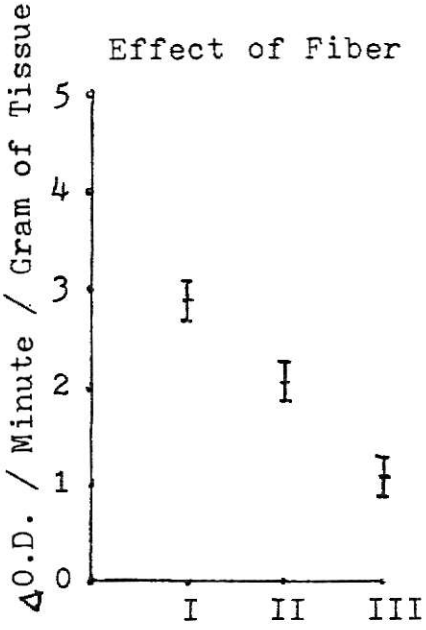
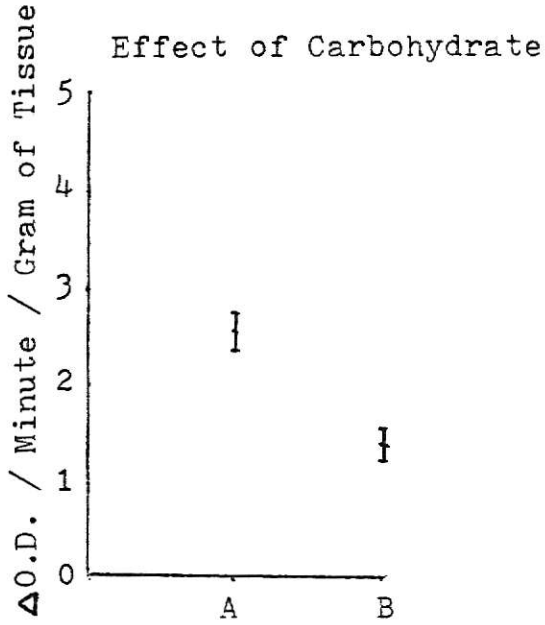


Figure 12. NADP-Malate Dehydrogenase Activity





a = low fiber sucrose
b = low fiber amylose
c = med fiber sucrose
d = med fiber amylose
e = high fiber sucrose
f = high fiber amylose
I = low fiber diets
II = med fiber diets
III = high fiber diets
A = sucrose diets
B = amylose diets



CONCLUSIONS

The following conclusions have been made from the results obtained in this study:

1. Increasing dietary fiber from wheat bran (soft white spring wheat) slightly reduces weight gain. Weight gain is essentially unchanged by changing from sucrose to amylose.

2. Rats fed high-fiber diets consumed a greater mass and volume of feed yet fewer calories than those fed lower-fiber diets. In addition, animals fed high-fiber diets have a higher food efficiency ratio than those fed lower-levels of fiber. Amylose fed rats consume smaller volumes than sucrose fed rats.

3. Dry fecal weight is directly proportional to dietary fiber when the fiber source is soft white spring wheat bran.

4. Glucose tolerance shows no trend of change with varying levels of fiber from wheat bran. Glucose tolerance curves may be slightly improved by amylose vs. sucrose but not significantly.

5. Serum cholesterol is not altered by wheat bran fiber from soft white spring wheat when it is fed to normal rats for five weeks.

6. Lipogenic enzyme activity is significantly decreased by what bran in the diet. In addition, lipogenic enzyme activity is lower in rats fed amylose than in

rats fed sucrose.

In conclusion, of the parameters studied, the greatest benefits from wheat bran in the diet are derived from its ability to: increase food efficiency, increase fecal output, and depress lipogenesis. Amylose is preferred to sucrose in the diet because amylose is less lipogenic.

SUMMARY

The influence of dietary fiber and carbohydrate source on selected parameters of lipid and carbohydrate metabolism was investigated. Male weanling rats, averaging 42.1 g, were fed ad libitum diets containing 20% protein, 10% fat and either 0%, 6%, or 18% fiber by weight with the balance comprised mainly of carbohydrate. Carbohydrate was provided by either cornstarch (amylose) or cane sugar (sucrose). The influence of the type of carbohydrate and the level of fiber incorporated into the diet were considered for the following parameters: 1) glucose tolerance, 2) weight gain, feed intake and food efficiency, 3) fecal output, 4) serum cholesterol and 5) hepatic lipogenic enzyme activity, specifically, malate dehydrogenase and hexose monophosphate dehydrogenases (HMPD).

The rats fed high-fiber diets consumed more feed by weight than the medium-fiber ($P < 0.05$) or low-fiber ($P < 0.05$) diet groups. The high-fiber groups also consumed more feed by volume than medium-fiber ($P < 0.0001$) or low-fiber ($P < 0.0001$) fed rats, however caloric intake in high-fiber groups was lower than medium-fiber and low-fiber ($P < 0.05$) diet groups. The rats fed high-fiber showed less weight gain than medium-fiber ($P < 0.05$) and low-fiber ($P < 0.0005$) groups and rats fed high-fiber had a greater food efficiency than medium-fiber ($P < 0.05$) or low-fiber

groups ($P < 0.05$). Fecal weight was directly proportional to dietary fiber level ($P < 0.0001$). The source of carbohydrate had no significant effect on the parameters discussed thus far.

No trend in glucose tolerance was observed due to carbohydrate source or fiber level. There were no significant differences in the serum cholesterol levels of animals on the diets tested.

The rats fed low-fiber diets had higher hepatic HMPD and malic enzyme activities than the medium-fiber ($P < 0.0005$) and high-fiber ($P < 0.0005$) groups. The medium-fiber group also showed a lipogenic enzyme activity greater than the high-fiber group ($P < 0.0005$). Sucrose fed rats consistently showed a higher hepatic HMPD ($P < 0.0001$) and a higher malic enzyme ($P < 0.0001$) activity than rats fed amylose.

Feeding wheat bran decreases weight gain, increases food efficiency and results in greater fecal weight. These same parameters are not altered by changing the source of carbohydrate from sucrose to amylose.

Carbohydrate source and wheat bran fiber do not significantly alter serum cholesterol or glucose tolerance. Hepatic lipogenic enzyme activity is lower in high-fiber and amylose containing diets than in lower fiber and sucrose containing diets.

LITERATURE

1. Burkitt, D. P., Walker, A. R. & Painter, N. S. (1974) Dietary fiber and disease. J. Am Med. Assoc. 229, 1068-1074.

2. Anderson, J. W. & Lin, W. (1977) Effects of high-sucrose or starch-bran diets on glucose and lipid metabolism of normal and diabetic rats. J. Nutr. 107, 584-585.

3. Trowel, H. (1978) Diabetes mellitus and dietary fiber of starchy foods. Am. J. Clin. Nutr. 31, S53-S57.

4. Stephen, A. M. & Cummings, J. H. (1980) Mechamism of action of dietary fibre in the human colon. Nature 84, 283-284.

5. Stephen, A. M. & Cummings, J. H. (1979) Water-holding by dietary fibre in vitro and its relationship to faecal output in man. Gut 20, 722-729.

6. Jawetz, E., Melnick, J. L. & Adelberg, E. A. (1980) Review of Medical Microbiology, 14th ed., Lange Medical Publications, Los Altos, California.

7. Frexinos, J. & Louis, A. (1978) Letters a la reduction. Effet sur le poids des sellers de trois, produits contenant des fibres alimentaires. Gastroenterol Clin. Biol. 2, 1055-1057.

8. Strasse-Wolthius, M., Hautvast, J. G., Hermus, R. J., Katan, M. B., Bausche, J. E., Reitberg-Brussaard, J. H., Velema, J. P., Zondervan, J. H., Eastwood, M. A. & Brydon, W. G. (1979) The effect of a natural high-fiber diet on serum lipids, fecal lipids, and colonic function. Am. J. Clin. Nutr. 32, 1881-1888.

9. Harland, B. F., O'Dell, R., & Prosky, L. (1976) Effect of dietary fiber on some metabolic responses in normal and diabetic rats. Fed. Proc. 35, 420 (abst.).

10. Prosky, L., Harland, B. F., O'Dell, R. G., & Stone, C. L. (1978) Year-long study of rats fed wheat bran and alphacel. Fed. Proc. 37, 755 (abst.).

11. Peng, B. & Tsai, A. C. (1978) Effect of locust bean gum on glucose tolerance in rats. Fed. Proc. 37, 542 (abst.).

12. Albrink, M. J., Newman, T. & Davidson, P. C. (1979) Effects of high- and low-fiber diets on plasma lipids and insulin. Am. J. Clin. Nutr. 32, 1486-1491.

13. Leeds, A. R. (1979) Gastric emptying, fiber and absorption. Lancet 2, 872.

14. Spiller, G. A. & Kay, R. M. (1978) Recommendations and conclusions of the dietary fiber workshop of the XI International Congress of Nutrition, Rio de Janeiro. Am. J. Clin. Nutr. 32, 2102-2103.

15. Anderson, J. W. & Ward, K. (1979) High-carbohydrate, high-fiber diets for insulin-treated men with diabetes mellitus. Am. J. Clin. Nutr. 32, 2312-2321.

16. Mann, J. I. & Simpson, H. C. (1980) Fiber, diabetes, and hyperlipidaemia. Lancet 1, 44.

17. Taylor, R. H., Goff, D. V., Wolever, T. M. & Fielden, H. (1979) Dietary fibre and diabetes. Lancet 2, 782.

18. Jenkins, D. J., Leeds, A. R., Slavin, B., Mann, J., Jepson, E. M. (1979) Dietary fiber and blood lipids: reduction of serum cholesterol in type II hyperlipidemi by guar gum. Am. J. Clin. Nutr. 32, 16-18.

19. Munoz, J. M., Sandstead, H. H., Jacob, R. A., Logan, G. M. & Dlevay, L. M. (1978) Effects of some cereal brans on glucose tolerance and plasma lipids of normal men. Fed. Proc. 37, 755 (abst.).

20. Taylor, R. H. (1979) Gastric emptying, fibre, and absorption. Lancet 2, 872.

21. Robbins, S. L. & Angell, M. (1976) Basic Pathology, 2nd ed., W. B. Saunders Co., Philadelphia, Pa.

22. Tejada, C., Strong, J. P., Montenegro, M. R., Restrepo, C. & Solberg, L. A. (1968) Distribution of coronary and aortic atherosclerosis by geographic location, race and sex. Lab. Investig. 18, 509-526.

23. Levine, S. A., Sprauge, H. B. & White, P. D. (1951) Clinical aspects of coronary heart disease. An analysis of 100 cases in patients 23 to 40 yeras of age with myocardial infarction. J. Am. Med. Assn. 146, 1291-1295.

24. Hatch, F. T., Reissell, P. K., Poon-King, T. M., Canellos, G. P., Lees, R. S. & Hagpian, L. M. (1966) A study of coronary heart disease in young men. Characteristics and metabolic studies of the patients and comparison with age-matched healthy men. Circulation 33, 679-703.

25. Kannel, W. B., Castelli, W. P., Gordon, T. & McNamara, P. M. (1971) Serum cholesterol lipoproteins and the risk of coronary heart disease: the Framingham study. Ann. Int. Med. 74, 1-12.

26. Katz, L. N. & Stamler, J. (1956) IN: Experimental Atherosclerosis (Thomas, C.C. ed.) Springfield, Ill.

27. Beveridge, J. M., Connell, W. F., Haust, H. L. & Mayer, G. A. (1959) Dietary cholesterol and plasma cholesterol levels in man. J. Biochem. Physiol. 37, 575-582.

28. Connor, W. E., Hodges, R. E. & Bleiler, R. E. (1961) The serum lipids in men receiving high cholesterol and cholesterol free diets. J. Clin. Investig. 40, 894-901.

29. Connor, W. E. (1961) Dietary cholesterol and the pathogenesis of atherosclerosis. Geriatrics 16, 407-415.

30. Reiser, R., Sorrels, M. F. & Williams, M. C. (1959) Influence of high levels of dietary fat and cholesterol on atherosclerosis and lipid distribution in swine. Circ. Res. 7, 833-846.

31. Melchior, G. W., Lofland, H. B. & Jones, D. C. (1974) Influence of dietary fat on cholelithiasis in squirrel monkeys. Fed. Proc. 33, 626 (abst.).

32. Loflanel, H. B., Clarkson, T. B., St. Clair, R. W. & Lehner, N. D. (1972) Studies on the regulation of plasma cholesterol levels in squirrel monkeys of two genotypes. J. Lipid Res. 13, 39-47.

33. Goldstein, J. L., Hazzard, W. R., Schrott, H. G., Bierman, E. L. & Motulsky, A. E. (1973) Hyperlipidemia in coronary heart disease: I. lipid levels in 500 survivors of myocardial infarction. J. Clin. Invest. 52, 1533-1543.

34. Leveille, G. A. & Sauberlich, H. E. (1966) Mechanism of the cholesterol-depressing effect of pectin in the cholesterol-fed rat. J. Nutr. 88, 209-214.

35. Balmer, J. & Zilversmit, D. B. (1974) Effects of dietary roughage on cholesterol absorption, cholesterol turnover and steroid excretion in the rat. J. Nutr. 104, 1319-1328.

36. Chang, M. L. & Johnson, M. A. (1978) Effect of lignin versus cellulose on the absorption of taucholic acid and lipid metabolism in rats fed cholesterol diets. Fed. Proc. 37, 542 (abst.).

37. Jenkins, D. J. (1975) Effect of pectin, guar gum and wheat fiber on serum cholesterol. Lancet 1, 1116.

38. Jenkins, D. J. (1975) Effect of wheat fiber on blood lipids, fecal steroid excretion and serum iron. Am. J. Clin. Nutr. 28, 1408-1411.

39. Jenkins, D. J., Reynolds, D., Leeds, A. R., Waller, A. L. & Cummings, J. H. (1979) Hypocholesterolemic action of dietary fiber unrelated to fecal bulking effect. Am. J. Clin. Nutr. 32, 2430-2435.

40. Jenkins, D. J., Reynolds, D., Slavin, B., Leeds, A. R., Jenkins, A. L. & Jepson, E. M. (1980) Dietary fiber and blood lipids: treatment of hypocholesterolemia with guar crispbread. Am. J. Clin. Nutr. 33, 575-581.

41. Pfeifer, J. J. & Karp, L. A. (1978) Comparative ability of dietary fibers to promote losses of fecal bile acids, sterols and fatty acids by the rat. Fed. Proc. 37, 735 (abst.).

42. VanBerge-Henegouwen, G. P., Huybreyts, A. W., VandeWerf, A., Demacker, P., Schade, R. W. (1979) Effect of a standardized wheat bran preparation on serum lipids in young healthy males. Am. J. Clin. Nutr. 32, 794-798.

43. Vahouny, G. V., Roy, T., Cassidy, M., Gallo, L. L., Kritchevsky, D., Story, J. & Treadwell, C. R. (1978) Dietary Fibers and lymphatic absorption of cholesterol in the rat. Fed. Proc. 37, 755 (abst.).

44. Lin, W. & Anderson, J. W. (1978) Effects of guar gum and wheat bran on lipid metabolism of rats. Fed. Proc. 37, 542 (abst.).

45. Munoz, J. M. (1979) Effects of some cereal brans and textured vegetable protein on plasma lipids. Am. J. Clin. Nutr. 32, 580-592.

46. Ranhotra, G. S. (1973) Effects of cellulose and what mill-fractions on plasma and liver cholesterol levels in cholesterol-fed rats. Cereal Chem 50, 358-363.

47. Montgomery, R. Dryer, R. L., Conway, T. W. & Spector, A. (1980) Biochemistry: A case oriented approach, 3rd ed., C.V. Mosby Co., St. Louis, Mo.

48. Heaton, K. W., Pomare, E. W. (1974) Effect of bran on blood lipids and calcium. Lancet 1, 49.

49. Connell, A. M. (1975) Absence of effect of bran on blood lipids. Lancet 1, 496.

50. Anderson, J. W. (1977) High polysaccharide diet studies in patients with diabetes and vascular disease. Cereal Foods World 22, 12-14.

51. Conley, T., Davis, C., Fealk, F., Hubbard, M., Hyszczak, O., Hyszczak, R., & Justice, B. (1978) Human serum cholesterol and triglycerides response to the consumption of a diet high in cellulose. Fed. Proc. 37, 542 (abst.).

52. Lin-Chen, W. & Anderson, J. W. (1979) Effects of plant fiber in decreasing plasma total cholesterol and increasing high-density lipoprotein cholesterol. Proc. Soc. Exp. Biol. Med. 162, 310.

53. Guyton, A. C. (1976) Textbook of Medical Physiology, 5th ed., W. B. Saunders Co., Philadelphia, London, Toronto.

54. Cooper, G. R. & McDaniel, V. (1970) Methods for the Determination of Glucose, American Society for Clinical Pathologists Commission on Continuing Education, Chicago, Ill.

55. Tietz, N. W. (1976) Modified Leffler method, 512-513 IN: Fundamentals of Clinical Chemistry, W. B. Saunders Co., Philadelphia, Pennsylvania.

56. Glock, G. & McLean, P. (1953) Further studies on the properties and assay of glucose-6-phosphate dehydrogenase and 6-phosphogluconate dehydrogenase of rat liver. Biochem. J. 55, 400-408.

57. Ochoa, S. (1955) Malic enzyme. IN: Methods in Enzymeology 1, 739-741.

58. Snedecor, G. W. & Cochran, W. G. (1976) Statistical Methods, 6th ed., Iowa State University Press, Ames, Iowa.

59. Trowel, H. (1977) Dietary Fibre: Metabolic and Vascular Disease, Norgine Ltd., London.

60. Johnson, M. A. & Chang, M. L. (1978) The hypocholesteremic effect of pectin as influenced by type of dietary fat in rats fed a cholesterol-containing diet. Fed. Proc. 37, 542 (abst.).

61. Silman, A. J. (1979) Cereal fibre, total energy intake, and obesity. Lancet 1, 905.

62. Kelsay, J. L., Behall,K. M. & Prather, E. S. (1978) Effect of fiber from fruits and vegetables on metabolic responses of human subjects 1. Bowel transit time, number of defecations, fecal weight, urinary excretions of energy and nitrogen and apparent digestibilities of energy, nitrogen, and fat. Am. J. Clin. Nutr. 31, 1149-1153.

63. Jefferys, D. B. & Macdonald, I. (1973) The effect of dietary fibre on the response to orally administered glucose. Proc. Nutr. Soc. 33, 11A-12A.

64. Jenkins, D. J., Goff, D. V., Leeds, A. R. Alberti, K. G., Wolever, T. M., Gassull, M. A. & Hockaday, T. D. (1976) Unabsorbable carbohydrates and diabetes: Decreased postprandial hyperglycemia. Lancet 2, 172-174.

65. Jenkins, D. J., Leeds, A. R., Gussull, M. A., Cochet, B. & Alberti, K. G. (1977) Decrease in postprandial insulin and glucose concentrations by guar and pectin.

66. Jenkins, D. J., Wolever, T. M., Leeds, A. R., Gassull, M. A., Haisman, P., Dilawari, J., Goff, D. V., Metz, G. L. & Alberti, K. G. (1978) Dietary fibres, fibre analogues, and glucose tolerance: importance of viscosity. Brit. Med. J. 1, 1392-1394.

67. Munoz, J. M., Sandstead, H. H., Jacob, R. A., Logan, G. M. & Klevay, L. M. (1977) Effects of dietary fiber on oral glucose tolerance, serum cholesterol and triglycerides. Am. J. Clin. Nutr. 30, 635 (abst.).

68. Munoz, J. M., Sandstead, H. H., Jacob, R. A., Logan, G. M. & Klevay, L. M. (1978) Effects of some cereal brans on glucose tolerance and plasma lipids of normal men. Fed. Proc. 37, 755.

69. Walker, A. R., Walker, B. F., & Richardson, B. D. (1970) Glucose and fat tolerances in Bantu children. Lancet 2, 51-52.

70. Southgate, D. A. (1973) Fibre and the other unavailable carbohydrates and their effects on the energy value of the diet. Proc. Nutr. Soc. 32, 131.

71. Wapnick, S., Wicks, A. C., Kanengoni, E. & Jones, J. J. (1972) Can diet be responsible for the initial lesion in diabetes? Lancet 2, 300-301.

APPENDIX

ILLEGIBLE

THE FOLLOWING DOCUMENT (S) IS ILLEGIBLE DUE TO THE PRINTING ON THE ORIGINAL BEING CUT OFF

ILLEGIBLE

ERICAN ASSOCIATION OF CEREAL CHEMISTS

Raymond J. Tarleton, Executive Vice President

0 Pilot Knob Road, St. Paul, Minnesota 55121 • Telephone 612/454-7250

Serving the Cereal Industry

67

AACC CERTIFIED FOOD GRADE WHEAT BRAN

Analytical data for the official AACC Certified Food Grade Wheat Bran R07-3691, are summarized below. These values, as is basis, are the average of duplicate analyses.

The various analyses were divided among the following labs, unless otherwise noted: Doty Laboratories, Inc., 1435 Clay Street, North Kansas City, Missouri 64116; Ingman Laboratories, Inc., 324 South Fourth Avenue, Minneapolis, Minnesota 55415; Medallion Laboratories, 9000 Plymouth Avenue North, Minneapolis, Minnesota 55427; and Research 900, 900 Checkerboard Square, St. Louis, Missouri 63188.

The bran was made from a commercial blend of white wheats (87.3% soft white and 12.7% club white), taken after the fifth break roll over a 22 wire to a bran duster with a 1250 micrscreen. The bran was immediately sent through an enzyme deactivation steamer with a residence time of about thirty seconds, exiting at 213-215^{o}F, and 17.2% moisture, and into an insulated screw conveyer which holds the hot bran for about 1.5 minutes. The bran was dried to 10.4% moisture and sacked into "scotchguard" three layer paper, one-layer 2 mil polyethylene bags. The bran is being held at 0^{o}F in Minneapolis.

Requests for samples and information should be made at the letter-head address. Bran is available at $2.00/lb. in 2.5 lb. quantities or at $1.50/lb. in 30 lb. bags, plus shipping costs.

AACC CERTIFIED FOOD GRADE WHEAT BRAN
R07-3691

Assay	Value[a] (as is basis)	Method[b]
Crude Fiber	8.91%	AOAC-7.050-7.054
Protein	14.3%	AACC-46-10
Moisture	10.4%	AACC-44-40
Fat (Acid Hydrolysis)	5.22%	AOAC-7.047
Ash	5.12%	AACC-08-01
Aerobic Plate Count	16,000/g	AACC-42-11
Acid Detergent Fiber	11.9%	AOAC-7.055-.057
Neutral Detergent Fiber	40.2%	See Below[c]
Lignin	3.2%	AOAC-7.058
Pectin	3.0%	See Below[d]
Water Holding Capacity	9.5 g/g	See Below[e]
Cutin	0.0%	USDA Hndbk #379, pp 9
Thiamine (B1)	0.78 mg/100 g	AOAC-43.024-.030
Riboflavin (B2)	0.39 mg/100 g	AACC-86-70
Niacin	20.9 mg/100 g	AOAC-43.044-.046
Pyridoxine(B6)	0.58 mg/100 g	AOAC-43.159-.164
Folic Acid	0.12 mg/100 g	JAOAC,48(6), 1230 (19
Pantothenic Acid	2.48 mg/100 g	AOAC-43.130-.138
Vitamin E	2.69 mg/100 g	Gas Chromatorgraphy[f
Choline	228 mg/100 g	AACC-86-45
Aluminum	5.0 ppm	AOAC-2.096-2.100
Arsenic	< 0.1 ppm	AOAC-25.012
Barium	45.07 ppm	AACC-40-70
Boron	4.5 ppm	APHA 13th ed, 107B (1
Cadmium	2.8 ppm	AACC-40-70
Calcium	0.12 %	AACC-40-21
Cobalt	39.2 ppm	AACC-40-70
Copper	15.6 ppm	AACC-40-70
Iron	122 ppm	AACC-40-70

AACC CERTIFIED FOOD GRADE WHEAT BRAN, R07-3691

Assay	Value[a] (as is basis)	Method[b]
Lead	2.3 ppm	AACC-40-70
Magnesium	0.43%	AACC-40-70
Manganese	80.0 ppm	AACC-40-70
Mercury	0.002 ppm	AOAC-25.103
Phosphorus	1.04%	AOAC-7.103
Potassium	1.38%	AACC-40-70
Silicon	35.0 ppm	
Selenium	0.1 ppm	See Below[g]
Sodium	0.10%	AACC-40-70
Zinc	54.5 ppm	AACC-40-70
Damaged Starch	3.74%	AACC-76-30A
Total Starch	17.4%	AOAC-14.031
Total Sugar As Invert	7.04%	AOAC-7.066
Pentosan	22.1%	See Below[h]
Phytic Acid	3.36%	See Below[i]
B-Sitosterol	123 mg/100 g	Gas Chromatography[f]
Campesterol	68.8 mg/100 g	Gas Chromatography[f]
Stigmasterol	11.2 mg/100 g	Gas Chromatography[f]
Aflatoxin	<10 ppb	JAOAC 56(4), 803 (1973)
Sanitation[j]	0	AACC-28-60
Pesticides, Phosphorus Containing	< 0.005 ppm	AOAC-29.-
Pesticides, Chlorine Containing	< 0.02 ppm	See Below[k]

Particle Size

ON US #10	1%			
#12	2%		#70	Trace
		Thru	#70	Trace
#14	5%			
#16	11%			
#18	13%			
#20	9%			
#30	33%			
#40	17%			
#50	8%			
#60	1%			

a. Abbreviations: mg - milligrams
ppm - parts per million
ppb - parts per billion

b. AOAC References are 12th edition (1975).

c. Neutral Detergent Giver. Method of Van Soest and Wine, JOAC, 50 (1), 50 (1967), modified by use of hog pancreatic anylase; AACC Fiber Methodology subcommittee.

d. Reported as % galacturonic acid. Medallion Labs modified procedure from: McCready and McComb, Analytical Chemistry, 24 (12), 1986 (1952) and; Bitter and Muir, Analytical Biochemistry, 4, 330 (1962).

e. Water Holding Capacity of the NDF fraction. By Kellogg Co.

f. Method by Medallion Laboratories.

g. Analysis by WARF Institute, Inc., Madison, Wisconsin, according to J. Ag. & Fd. Chem., 9 488 (1961).

h. Method by CPC International, Inc.

i. Modified method by Research 900. Adapted from: Biochem. Z., 64, 422 (1914).

j. Whole insects, insect fragments, whole larvae, larva fragments, rodent hairs, rodent excretion fragments, other contaminants.

k. Modified method by Research 900.

Initial body weight, final body weight, mean daily weight gain (grams/day), Food efficiency ratio (FER), mean daily feed intake (grams), mean daily feed intake (cm^3), mean daily feed intake (kcal)

Diet	Initial weight	Final weight	Mean Daily weight gain	Mean Daily Feed Intake g/day	cm^3/day	kcal/day	FER
Low Fiber Sucrose							
1	48.5	---	---	---	---	---	---
2	44.5	275.0	6.0	12.4	15.4	53.3	11.2
3	44.5	267.5	5.9	12.4	15.4	53.3	11.1
4	43.0	268.0	5.8	13.0	16.1	55.9	10.4
5	42.0	280.0	6.4	13.0	16.1	55.9	11.4
6	42.0	280.0	6.2	13.4	16.6	57.6	10.7
7	41.0	275.0	6.4	13.7	17.0	58.9	10.8
8	39.0	280.0	6.0	13.5	16.7	58.0	10.4
9	39.0	290.0	6.2	13.5	16.7	58.0	10.6
10	36.5	261.0	4.9	13.4	16.6	57.6	8.6
Mean $\pm$ SE	42.0 $\pm$ 0.2	277.9 $\pm$ 11.9	6.0 $\pm$ 0.2	13.2 $\pm$ 0.4	16.5 $\pm$ 0.7	56.8 $\pm$ 1.6	10.5 $\pm$ 0.4
Low Fiber Amylose							
1	46.5	289.0	5.9	13.3	17.3	57.2	10.3
2	45.0	285.5	6.2	12.6	16.4	54.2	11.5
3	44.5	291.5	6.5	12.4	16.1	53.3	12.2
4	43.0	290.0	6.4	13.6	17.7	58.5	10.9

Initial body weight, final body weight, mean daily weight gain (grams/day), Food efficiency ratio (FER), mean daily feed intake (grams), mean daily feed intake (cm^3), mean daily feed intake (kcal)

Diet	Initial weight	Final weight	Mean Daily weight gain	Mean Daily Feed Intake g/day	cm^3/day	kcal/day	FER
5	42.5	266.5	5.8	12.7	16.5	54.6	10.6
6	42.0	264.5	6.0	13.2	17.1	56.8	10.6
7	40.0	109.5	---	---	---	---	---
8	39.5	257.5	5.6	13.5	17.5	58.0	9.6
9	39.0	268.0	5.6	10.7	13.9	46.0	12.2
10	37.0	262.0	4.9	12.4	16.1	53.3	9.3
Mean ± SE	41.9 ± 0.2	258.4 ± 11.1	6.0 ± 0.2	12.9 ± 0.4	16.8 ± 0.7	55.2 ± 1.6	10.8 ± 0.4
Medium Fiber Sucrose							
1	48.0	273.0	5.3	13.1	19.2	51.6	10.3
2	45.5	282.0	6.4	8.9	15.1	35.1	18.0
3	44.0	258.0	5.8	12.4	18.2	48.9	11.8
4	43.0	262.5	5.9	13.4	19.7	52.8	11.2
5	42.5	269.0	6.0	13.1	19.2	51.6	11.7
6	41.5	285.0	6.5	14.2	20.9	56.0	11.6
7	41.0	298.0	6.5	15.1	22.2	59.5	10.9
8	39.5	240.0	5.0	12.9	18.9	50.8	9.9
9	38.5	278.0	6.0	14.4	21.1	56.7	10.7

Initial body weight, final body weight, mean daily weight gain (grams/day), Food efficiency ratio (FER), mean daily feed intake (grams), mean daily feed intake (cm^3), mean daily feed intake (kcal)

Diet	Initial weight	Final weight	Mean Daily weight gain	Mean Daily Feed Intake g/day	cm^3/day	kcal/day	FER
10	37.0	276.0	5.3	14.1	20.7	55.6	9.5
Mean ± SE	42.0 ± 0.2	271.8 ± 11.1	5.9 ± 0.2	13.2 ± 0.4	19.3 ± 0.7	51.8 ± 1.5	11.6 ± 0.4
Medium Fiber							
Amylose							
1	48.0	281.0	5.4	13.5	18.3	53.2	10.1
2	45.5	299.0	6.6	15.2	20.6	59.9	11.0
3	43.5	187.0	3.6	9.6	13.0	39.8	9.6
4	43.0	288.0	6.5	12.2	16.5	48.0	13.6
5	42.5	255.0	5.6	13.1	17.7	51.6	10.8
6	41.5	291.5	6.5	13.8	18.7	54.4	11.9
7	40.5	301.0	6.7	15.0	20.3	59.1	11.4
8	39.5	249.0	5.6	12.6	17.0	49.6	11.4
9	38.5	259.0	5.6	14.1	19.1	55.6	10.1
10	37.0	---	---	---	---	---	---
Mean ± SE	42.0 ± 0.2	256.1 ± 11.1	5.7 ± 0.2	13.2 ± 0.4	17.9 ± 0.7	52.1 ± 1.6	10.9 ± 0.4
High Fiber							
Sucrose							
1	48.5	292.0	6.2	15.9	32.6	51.5	12.0
2	45.5	270.5	5.7	13.6	27.9	44.1	13.1

Initial body weight, final body weight, mean daily weight gain (grams/day), Food efficiency ratio (FER), mean daily feed intake (grams), mean daily feed intake (cm^3), mean daily feed intake (kcal)

Diet	Initial weight	Final weight	Mean Daily weight gain	Mean Daily Feed Intake g/day	cm^3/day	kcal/day	FER
3	43.5	263.5	5.7	13.4	27.5	43.4	13.1
4	43.0	243.0	4.9	13.3	27.3	43.1	11.5
5	42.5	277.5	6.0	15.3	31.4	49.6	12.2
6	41.0	257.0	5.9	15.1	31.0	48.9	12.1
7	48.0	269.0	5.8	15.3	31.4	49.6	11.8
8	39.5	243.0	5.1	14.1	28.9	45.7	11.2
9	38.0	259.0	5.4	13.5	24.8	43.7	12.4
10	36.5	240.0	4.7	12.9	26.5	41.8	11.2
Mean $\pm$ SE	42.6 $\pm$ 0.2	261.4 $\pm$ 11.1	5.6 $\pm$ 0.2	14.2 $\pm$ 0.4	28.9 $\pm$ 0.7	46.1 $\pm$ 1.5	12.0 $\pm$ 0.4
High Fiber Amylose							
1	46.5	282.0	5.9	15.9	29.5	51.5	11.4
2	46.0	288.0	6.3	15.2	28.2	49.2	12.8
3	43.0	238.0	5.1	13.2	24.5	42.8	11.9
4	43.0	253.0	5.5	10.0	18.6	32.4	17.0
5	42.5	262.0	5.5	14.8	27.5	48.0	11.5
6	41.0	274.0	6.0	15.4	28.6	49.9	12.0
7	41.0	246.5	5.3	12.8	23.8	41.5	12.8
8	40.0	143.0	---	---	---	---	---

Initial body weight, final body weight, mean daily weight gain (grams/day), Food efficiency ratio (FER), mean daily feed intake (grams), mean daily feed intake (cm^3), mean daily feed intake (kcal)

Diet	Initial weight	Final weight	Mean Daily weight gain	Mean Daily Feed Intake g/day	cm^3/day	kcal/day	FER
9	38.0	240.0	5.0	13.8	25.6	44.7	11.2
10	37.5	242.5	4.6	13.7	25.5	44.4	10.3
Mean ± SE	41.8 ± 0.2	146 ± 11.1	5.43 ± 0.16	13.9 ± 0.4	25.7 ± 0.7	44.9 ± 1.6	12.2 ± 0.4

Mean Daily Dry Fecal Weight (grams)

Diet	Fecal Weight	Diet	Fecal Weight	Diet	Fecal Weight
Low Fiber Sucrose		Medium Fiber Sucrose		High Fiber Sucrose	
1	---	1	1.240	1	3.681
2	0.464	2	1.550	2	2.947
3	0.526	3	1.120	3	3.470
4	0.608	4	1.508	4	4.280
5	0.584	5	1.283	5	3.291
6	0.518	6	1.357	6	4.279
7	0.535	7	1.358	7	3.921
8	0.500	8	1.248	8	3.452
9	0.585	9	1.394	9	3.415
10	0.661	10	1.460	10	2.905
Mean $\pm$ SE	0.561 $\pm$ 0.085	Mean $\pm$ SE	1.352 $\pm$ 0.079	Mean $\pm$ SE	3.564 $\pm$ 0.079
Low Fiber Amylose		Medium Fiber Amylose		High Fiber Amylose	
1	0.450	1	1.280	1	3.614
2	0.443	2	1.503	2	3.703
3	0.456	3	0.775	3	2.889
4	0.415	4	1.615	4	2.994
5	0.458	5	1.298	5	3.194
6	0.504	6	1.263	6	3.761

Mean Daily Dry Fecal Weight (grams)

Diet	Fecal Weight	Diet	Fecal Weight	Diet	Fecal Weight
7	---	7	1.432	7	3.162
8	0.483	8	1.265	8	---
9	0.679	9	1.263	9	2.991
10	0.364	10	---	10	3.669
Mean ± SE	0.501 ± 0.085	Mean ± SE	1.294 ± 0.085	Mean ± SE	3.325 ± 0.085

Glucose Tolerance Test Results (mg glucose/dl plasma)

Diet	0 - Minutes	30 - Minutes	60 - Minutes	90 - Minutes
Low Fiber				
Sucrose				
1	---	---	---	---
2	92.7	493.0	188.2	124.1
3	113.9	203.4	---	118.5
4	101.2	191.5	77.8	106.0
5	91.2	191.2	122.1	95.5
6	138.6	196.6	166.7	164.9
7	140.6	206.3	185.2	171.0
8	86.7	206.1	167.4	189.3
9	56.7	170.9	174.8	188.3
10	107.1	167.5	180.7	169.3
Mean $\pm$ SE	103.6 $\pm$ 11.4	222.4 $\pm$ 20.5	157.2 $\pm$ 12.0	149.7 $\pm$ 9.3
Low Fiber				
Amylose				
1	100.0	96.7	133.9	127.1
2	52.6	380.9	231.7	115.6
3	94.4	186.3	127.6	137.1
4	49.1	223.7	150.9	127.4
5	73.6	156.0	139.6	110.0
6	121.4	151.3	159.4	144.0

Glucose Tolerance Test Results (mg glucose/dl plasma)

Diet	0 - Minutes	30 - Minutes	60 - Minutes	90 - Minutes
7	---	---	---	---
8	81.2	159.2	176.1	125.2
9	81.7	284.5	181.6	131.1
10	64.9	150.0	146.5	133.3
Mean ± Se	84.8 ± 11.4	197.2 ± 20.5	163.7 ± 11.2	128.3 ± 9.3
Medium Fiber Sucrose				
1	106.8	254.2	200.0	198.3
2	213.6	207.8	194.6	151.7
3	74.1	177.7	173.4	120.7
4	113.7	318.5	234.1	167.5
5	75.8	237.6	146.2	169.8
6	211.1	237.3	137.7	163.9
7	190.1	229.0	183.5	171.0
8	75.3	222.2	204.7	145.2
9	117.5	265.0	198.1	248.5
10	78.1	255.3	201.8	202.6
Mean ± SE	125.6 ± 10.7	250.5 ± 19.2	187.4 ± 10.6	173.9 ± 8.8

Glucose Tolerance Test Results (mg glucose/dl plasma)

Diet	0 - Minutes	30 - Minutes	60 - Minutes	90 - Minutes
Medium Fiber				
Amylose				
1	103.4	205.1	137.3	162.7
2	99.1	136.4	135.6	112.8
3	---	---	---	---
4	106.8	248.2	150.8	167.5
5	94.6	239.6	228.6	161.0
6	130.4	214.7	179.3	140.4
7	200.6	213.9	146.3	133.0
8	104.9	208.7	155.4	144.2
9	46.2	231.1	165.0	143.7
10	---	---	---	---
Mean $\pm$ SE	105.5 $\pm$ 12.2	229.0 $\pm$ 21.9	160.8 $\pm$ 12.0	145.8 $\pm$ 10.0
High Fiber				
Sucrose				
1	120.3	223.7	181.4	178.0
2	78.9	330.4	156.0	128.4
3	86.1	149.1	147.4	93.5
4	---	---	---	---
5	106.7	213.2	183.0	140.1
6	134.1	231.9	245.5	168.5

Glucose Tolerance Test Results (mg glucose/dl plasma)

Diet	0 - Minutes	30 - Minutes	60 - Minutes	90 - Minutes
7	219.6	280.3	175.7	204.4
8	88.6	198.3	222.6	159.7
9	103.9	172.8	142.7	149.5
10	89.5	257.9	168.4	154.4
Mean ± SE	113.9 ± 1.4	227.8 ± 20.5	180.2 ± 11.2	151.9 ± 9.3
High Fiber Amylose				
1	145.8	225.4	152.5	211.9
2	99.1	243.2	182.8	153.4
3	121.3	217.2	125.9	142.2
4	119.7	210.4	172.8	215.2
5	150.0	254.9	165.5	120.0
6	133.2	202.9	239.1	173.0
7	188.2	234.7	170.0	169.1
8	---	---	---	---
9	113.6	274.8	252.4	201.9
10	99.1	188.6	170.2	175.4
Mean ± SE	127.2 ± 11.4	224.3 ± 20.5	183.1 ± 11.2	173.9 ± 9.3

Cholesterol Data (mg cholesterol/dl serum)

Diet	Cholesterol	Diet	Cholesterol	Diet	Cholesterol
Low Fiber		Medium Fiber		High Fiber	
Sucrose		Sucrose		Sucrose	
1	---	1	97.7	1	71.6
2	---	2	97.8	2	102.2
3	70.9	3	---	3	105.2
4	118.7	4	124.6	4	165.9
5	164.2	5	98.5	5	41.8
6	66.4	6	123.9	6	139.6
7	---	7	97.8	7	120.0
8	159.0	8	141.0	8	118.7
9	103.0	9	130.6	9	78.4
10	95.5	10	51.5	10	76.9
Mean $\pm$ SE	111.2 $\pm$ 12.6	Mean $\pm$ SE	109.2 $\pm$ 10.8	Mean $\pm$ SE	101.9 $\pm$ 10.1
Low Fiber		Medium Fiber		High Fiber	
Amylose		Amylose		Amylose	
1	113.4	1	104.5	1	62.7
2	121.6	2	87.3	2	106.0
3	---	3	80.6	3	81.3
4	118.7	4	97.8	4	170.1
5	93.3	5	70.9	5	91.8

Cholesterol Data (mg cholesterol/dl serum)

Diet	Cholesterol	Diet	Cholesterol	Diet	Cholesterol
6	66.4	6	79.8	6	68.7
7	---	7	73.9	7	---
8	99.3	8	77.6	8	---
9	100.0	9	97.0	9	173.1
10	114.2	10	---	10	125.4
Mean $\pm$ SE	105.0 $\pm$ 11.6	Mean $\pm$ SE	83.9 $\pm$ 10.8	Mean $\pm$ SE	109.7 $\pm$ 11.6

Hepatic lipogenic enzyme activity (hexose monophosphate shunt enzymes and NADP-malate dehydrogenase)

Diet	HMPD OD/min/g. tis	Malate OD/min/g. tis.	Diet	HMPD OD/min/g. tis	Malate OD/min/g. tis
Low Fiber			Low Fiber		
Sucrose			Amylose		
1	---	---	1	4.38	1.38
2	12.34	1.95	2	6.72	2.20
3	15.18	4.87	3	---	---
4	11.01	4.81	4	4.80	1.44
5	16.94	4.03	5	3.79	1.84
6	12.06	5.02	6	8.52	2.19
7	11.65	4.43	7	---	---
8	11.14	4.26	8	9.07	3.21
9	7.52	2.49	9	8.30	2.78
10	4.55	2.17	10	7.74	1.55
Mean $\pm$ SE	11.20 $\pm$ 0.79	3.71 $\pm$ 0.26	Mean $\pm$ SE	6.70 $\pm$ 0.84	2.04 $\pm$ 0.28
Medium Fiber			Medium Fiber		
Amylose			Amylose		
1	5.97	1.85	1	2.67	0.57
2	9.95	3.12	2	5.79	1.52
3	3.04	1.42	3	4.08	0.61
4	8.78	1.78	4	8.30	2.09

Hepatic lipogenic enzyme activity (hexose monophosphate shunt enzymes and NADP-malate dehydrogenase)

Diet	HMPD OD/min/g. tis	Malate OD/min/g. tis.	Diet	HMPD OD/min/g. tis	Malate OD/min/g. tis
5	8.79	2.22	5	3.94	1.17
6	8.54	3.84	6	4.72	0.97
7	11.98	3.21	7	---	2.27
8	10.27	4.59	8	3.06	1.93
9	14.47	2.57	9	4.05	1.58
10	7.32	3.56	10	---	---
Mean ± SE	8.90 ± 0.74	2.82 ± 0.25	Mean ± SE	4.58 ± 0.84	1.39 ± 0.26
High Fiber Sucrose			High Fiber Amylose		
1	3.16	0.87	1	2.84	0.69
2	6.24	1.29	2	3.60	0.89
3	4.96	---	3	1.29	0.82
4	1.98	1.66	4	2.60	0.99
5	4.32	2.03	5	2.36	0.64
6	4.94	2.26	6	2.60	0.47
7	5.31	---	7	3.21	1.01
8	2.66	0.86	8	---	---
9	4.15	0.55	9	2.55	0.13
10	6.27	1.39	10	2.99	1.28
Mean ± SE	4.40 ± 0.74	1.41 ± 0.28	Mean ± SE	2.60 ± 0.79	0.81 ± 0.26

STATISTICS

Weight Gain:	High < Low fiber ($P < 0.005$) no significant difference due to carbohydrate source.
Fecal Weight:	High fiber > medium fiber > low fiber ($P < 0.0001$) no significant difference due to carbohydrate source.
Feed Intake: (weight)	High fiber > medium fiber ($P < 0.05$) high > low fiber ($P < 0.05$).
Feed Intake: (volume)	High > medium fiber ($P < 0.0001$) high fiber > low fiber ($P < 0.0001$) medium fiber > low fiber ($P < 0.01$).
KCAL's/Day:	High fiber < medium fiber ($P < 0.0001$) high fiber < low fiber ($P < 0.0001$) medium fiber < low fiber ($P < 0.05$).
Food Efficiency:	High fiber > low fiber ($P < 0.005$).
Cholesterol:	No significant differences.
Glucose 0-min.:	High fiber > low fiber ($P < 0.05$) high fiber amylose > low fiber amylose ($P < 0.05$) medium fiber sucrose > low fiber amylose ($P < 0.05$).
Glucose 30-min.:	No significant differences.
Glucose 60-min.:	No significant differences.
Glucose 90-min.:	High fiber > low fiber ($P < 0.05$). medium fiber > low fiber ($P < 0.05$). high fiber amylose > low fiber amylose ($P < 0.005$)

high fiber amylose > medium fiber amylose ($P < 0.05$)

medium fiber sucrose > low fiber amylose ($P < 0.001$)

medium fiber sucrose > medium fiber amylose ($P < 0.05$).

Malate: High fiber < medium fiber high fiber ($P < 0.005$)

amylose > sucrose ($P < 0.0001$).

HMP Shunt: High fiber < low fiber ($P < 0.0001$)

high fiber < medium fiber ($P < 0.0005$)

medium fiber < low fiber ($P < 0.01$)

amylose < sucrose ($P < 0.0001$).

EFFECTS OF WHEAT BRAN FIBER AND CARBOHYDRATE SOURCE ON GLUCOSE TOLERANCE, SERUM CHOLESTEROL AND LIPOGENIC ENZYME ACTIVITY IN WEANLING RATS

by

JOSEPH DUDLEY MATTHEWS

B.S., Kansas State University, 1977

ABSTRACT

Submitted in partial fulfillment of the
requirements for the degree

MASTER OF SCIENCE

Department of Foods and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1980

ABSTRACT

The influence of dietary fiber and carbohydrate source on selected parameters of lipid and carbohydrate metabolism was investigated. Male weanling rats, averaging 42.1 g, were fed ad libitum diets containing 20% protein, 10% fat and either 0%, 6%, or 18% fiber by weight with the balance comprised mainly of carbohydrate. Carbohydrate was provided by either cornstarch (amylose) or cane sugar (sucrose). The influence of the type of carbohydrate and the level of fiber incorporated into the diet were considered for the following parameters: 1) glucose tolerance, 2) weight gain, feed intake and food efficiency, 3) fecal output, 4) serum cholesterol and 5) hepatic lipogenic enzyme activity, specifically, amalate dehydrogenase and hexose monophosphate dehydrogenases (HMPD).

The rats fed high-fiber diets consumed more feed by weight than the medium-fiber ($P < 0.05$) or low-fiber ($P < 0.05$) diet groups. The high-fiber groups also consumed more feed by volume than medium-fiber ($P < 0.0001$) or low-fiber ($P < 0.0001$) fed rats, however caloric intake in high-fiber groups was lower than medium-fiber and low-fiber ($P < 0.05$) diet groups. The rats fed high-fiber showed less weight gain than medium-fiber ($P < 0.05$) and low-fiber ($P < 0.05$). Fecal weight was directly proportional to dietary fiber level ($P < 0.0001$). The source of carbohydrate had no significant effect on the parameters

discussed thus far.

No trend in glucose tolerance was observed due to carbohydrate source or fiber level. There were no significant differences in the serum cholesterol levels of animals on the diets tested.

The rats fed low-fiber diets had higher hepatic HMPD and malic enzyme activities than the medium-fiber ($P < 0.0005$) and high-fiber ($P < 0.0005$) groups. The medium-fiber group also showed a lipogenic enzyme activity greater than the high-fiber group ($P < 0.0005$). Sucrose fed rats consistently showed a higher hepatic HMPD ($P < 0.0001$) and a higher malic enzyme ($P < 0.0001$) activity than rats fed amylose.

Feeding wheat bran decreases weight gain, increases food efficiency and results in greater fecal weight. These same parameters are not altered by changing the source of carbohydrate from sucrose to amylose.

Carbohydrate source and wheat bran fiber do not significantly alter serum cholesterol or glucose tolerance. Hepatic lipogenic enzyme activity is lower in high-fiber and amylose containing diets than in lower fiber and sucrose containing diets.