APPENDIX 2

SUMMARY OF BARLEY BETAFIBER (BARLEY BETA-GLUCAN CONCENTRATE) CLINICAL TRIAL

The Effects of Concentrated Barley Beta-Glucan on Blood Lipids and Other CVD Risk Factors in a Population of Hypercholesterolemic Men and Women

Principal Investigators: Joel J Pins and Joseph M Keenan, University of Minnesota Medical School

PURPOSE

To evaluate the clinical efficacy and tolerance of two barley beta-glucan concentrates (BBG), one with a high molecular weight (HMW), typical of native barley beta-glucan, and the other a BBG that has been further processed to a low molecular weight (LMW).[1] Reduction in molecular weight improves the sensory properties and performance of barley beta-glucan concentrate in a variety of food applications. Changes in blood lipids were evaluated using both forms of the barley beta-glucan concentrates at two different daily doses (3g and 5g).

SUBJECTS AND METHODS

Subject Screening and Recruitment

Men and women aged 25 to 73 years who met the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III criteria for diet therapy due to elevated LDL cholesterol [2] (LDL-C) comprised the target group. During September 2003 through October 2004, subjects were recruited from the University of Minnesota-Twin Cites and the greater Twin Cities area. The study was approved by the University of Minnesota Institutional Review Board, and all subjects gave informed consent. Individuals were excluded if they had diabetes, cancer, secondary hyperlipidemia, cardiovascular disease, or other chronic medical conditions; triglycerides > 500 mg/dL; body mass index (BMI) > 40; or large or unexplained weight change within the previous six months. Additional exclusion criteria included the use of lipid-altering medications or lipid-altering dietary supplements two months prior to screening; use of more than two alcoholic drinks per day on a regular basis, allergy to aspirin, grain products, or any ingredients used in the treatment foods;

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[1] Barley betafiber, marketed by Cargill as Barliv™

[2] LDL-cholesterol: 140 mg/dL to 190 mg/dL

following a special diet; or smoking within the past year. Pregnant or lactating women were also excluded.

Study Design

This study was a randomized, double-blind, controlled, five-arm parallel group trial consisting of a 4-week diet stabilization phase followed by a 6-week treatment phase. Subjects who met all of the inclusion criteria, as determined at an initial screening visit, were eligible to enter the diet stabilization phase of the study. During this phase, participants attended a group education class in which they were instructed to consume a diet low in saturated fat and trans fat (< 10% kcals/day). Subjects were instructed to continue following the low saturated fat and low trans fat diet and to maintain other lifestyle habits throughout the study. At the end of the diet stabilization phase, subjects who still met inclusion criteria were randomly allocated, using a block randomization scheme, to receive a control or one of four treatments: 3g LMW BBG, 5g LMW BBG, 3g HMW BBG, 5g HMW BBG. Compliance to study protocol and side effects were evaluated when subjects returned to the clinic after 3 and 6 weeks of treatment. Blood lipids, blood apolipoproteins, blood pressure and other cardiovascular disease (CVD) risk markers were measured at baseline[3] and at the end of the treatment phase.

Treatment Products

Concentrated HMW beta-glucan was prepared from milled waxy hulless barley, using a modification of the extraction method of Aman and Hesselman (1985). The LMW BBG was prepared from the same type of barley, with a beta-glucanase preparation from *Bacillus amyloliquefaciens*, under conditions similar to those used for the HMW beta-glucan extraction.

The barley beta-glucan concentrates were formulated into a ready-to-eat corn flakes breakfast cereal and a low-calorie tropical juice drink containing 5% fruit juice. Control products consisted of the corn flakes cereal and the low-calorie tropical juice drink without beta-glucan fortification. The nutritional profiles of the food products were consistent with heart health claim requirements. Subjects consumed two single-serving packages of fruit drink and one single-serving package of cereal with meals each day to obtain the necessary doses of beta-glucan. The beta-glucan content

[3] Baseline: week 0 after the diet stabilization phase and before the treatment phase

and molecular weight of the beta-glucan concentrates are specified in Table 1. Each serving provided a minimum of 0.75g barley beta-glucan. Subjects were instructed to save all used and unused cereal and juice drink containers. These were collected and counted at weeks 3 and 6 as a measure of compliance.

Table 1. Characteristics of HMW AND LMW Barley Beta-Glucan Concentrates

	LMW Beta-Glucan Concentrate	HMW Barley Beta-Glucan Concentrate
Molecular weight[a] (kDa)	150	1,000
Beta-glucan (%)	70[b]	67[c]
Total fat	0.1	2.7
Protein	2.2	8.3
Moisture	6.2	3.4
Ash	2.6	2.4

[a] Weight-average molecular weight
[b] 75% and [c] 70% beta-glucan on a dry weight basis

Clinical and Laboratory Methods

All study visits were conducted at the University of Minnesota General Clinical Research Center (GCRC). At the screening visit a general medical history was obtained; blood pressure, height, and weight were measured; and blood samples were collected to assess fasting chemistry and lipid values. After the diet stabilization phase, fasting lipids and lipoproteins were reassessed. Scheduled visits during the treatment phase were at baseline (week 0), and weeks 3 and 6. At all treatment visits, blood pressure and weight were measured and side effects were evaluated. Fasting blood lipids, including total cholesterol (TC), LDL-C, HDL-cholesterol (HDL-C), tryiglyercides, apolipoprotein B (apo B) and apolipoprotein A-1 (apo A-1), were measured at baseline and at week 6.

All clinical measurements and blood draws were performed by GCRC medical staff. Weight and height measurements were obtained with subjects wearing indoor clothing without shoes. Blood pressure measurements were obtained with an automatic Colin® blood pressure monitor (Medical Instruments Corp., San Antonio, Texas; Press-mate® BP/8800C) after subjects had rested in a seated position for at least 5 minutes. All blood samples were obtained using standard venipuncture techniques after subjects had fasted for 12 hours. All laboratory analysis was done

using standard automated technology by Quest Diagnostics® Incorporated in Wood Dale, IL/ branch laboratory (certified and accredited by the Clinical Laboratory Improvement Amendment of 1988 and the College of American Pathologists) or at the University of Minnesota. TC, LDL-C and triglyceride concentrations were determined using enzymatic methods with Olympus reagents with automated spectrophotometry performed on an Olympus AU5400®. HDL -C was determined directly using Roche reagents on the Olympus AU5400®. Total apo B and apo A-1 levels were determined immunochemically using a Behring BN 100 nephelometer (Dade Behring Inc, Newark, DE).

Dietary and Side Effect Evaluations

Dietary data was collected during the treatment phase as a means of monitoring diet compliance and consistency. Each subject completed 3-day food records during the first and last week of treatment and returned the completed records at weeks 3 and 6. Research staff reviewed the records for completeness and clarity during the study visits. Food records were analyzed for energy, macronutrient, and micronutrient intake using Nutrition Data System for Research software version 5.0_35 (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN).

Study-related side effects were assessed by a 13-question side effect questionnaire. Side effect symptoms evaluated included diarrhea, loose stools, constipation, stomach pain, nausea, heartburn, gas, loss of appetite, full stomach, indigestion, bloating, abdominal cramping, and fullness at meals. Subjects were asked to check the category that best represented their symptoms over the last month at baseline or since their last study visit at each subsequent visit. Frequency counts were used in the analyses and were categorized in two ways: 1) dichotomized as ANY versus NO side effects and 2) the top two categories were collapsed and were used to indicate the presence of side effects. In evaluating side effects, the analyses were conducted using both approaches.

Statistical Analysis

Differences in baseline demographic and clinical variables among the treatment groups were compared using analysis of variance for continuous variables and the Chi-square test for categorical variables. The treatment effect was based on the measurement and comparison of the

mean levels of lipids and lipoproteins among treatment groups using analysis of variance. The GENMOD procedure of SAS version 8 (SAS Institute, Inc, Cary, NC) was used to perform the analyses. In addition, a Chi-square test was performed comparing all side effect counts (frequencies) at baseline, mid-study, and post-study visits. Regression analysis using a general linear model was used to determine the differences in side effects over time and between the treatment groups and the control group. Statistical significance adjustments were made using Dunnett's test for multiple comparisons.

RESULTS

Baseline Characteristics

A total of 155 subjects, comprising 75 men and 80 women completed the study. The mean age of the subjects was 55 $\pm$ 11.1 years. All subjects were determined to be generally healthy at baseline and without history of coronary heart disease (CHD). Eighty-two percent had 0-1 CHD risk factors and 18 % had 2 or more CHD risk factors. Mean baseline lipid levels for the total sample were: TC: 235 $\pm$ 24.1mg/dL; LDL-C: 154 $\pm$ 16.6mg/dL; HDL-C: 50 $\pm$ 13.5mg/dL; triglycerides: 160 $\pm$ 79.2mg/dL; apo B: 139 $\pm$ 17.5mg/dL; apo A-1: 121 $\pm$ 33mgl/dL. Mean baseline BMI was 29 $\pm$ 5.3kg/m^2.

All baseline characteristics were similar among the treatment and control groups (Table 2). The ratio of men to women, mean age, mean BMI, TC, and the proportion of subjects in each group that had a positive family history of CHD were similar in each arm. Each group (treatments and control) was block-stratified based on metabolic syndrome status, which resulted in an even distribution of metabolic and non-metabolic syndrome subjects in each group. Approximately 40% of all subjects were identified as having metabolic syndrome[4].

[4] NCEP ATP III indicates a diagnosis of metabolic syndrome is made when 3 or more risk factors are present. Risk factors include a waist circumference of $\geq$ 102 cm in men or $\geq$ 88 cm in women; triglyceride levels $\geq$ 150 mg/dL; HDL-C of < 40 mg/dL in men or < 50 mg/dL in women; blood pressure $\geq$ 130/85 mmHg; and fasting plasma glucose $\geq$ 110 mg/dL (NCEP, 2002).

Body Weight

Body weight was unchanged over the duration of the treatment phase in all study groups (Table 3).

Dietary Intake

The background diet was unchanged throughout the study in both treatment and control groups (Table 4).

Total Cholesterol

Treatment Effects vs. Control (Table 5)

At the end of the 6-week intervention period, LMW BBG significantly lowered TC by 6.0% ($p < 0.05$) at the 3g dose and by 9.9% ($p < 0.05$) at the 5g dose, compared to the control. The HMW BBG significantly reduced TC by 7.0% ($p < 0.05$) at the 3g dose and by 11.2% ($p < 0.05$) at the 5g dose.

Treatment Effects vs. Baseline (Table 5)

TC was significantly reduced from baseline by 7.2% ($p < 0.05$) and 11.1% ($p < 0.05$) in the 3g and 5g LMW BBG groups, respectively. In the HMW BBG group, TC was significantly lowered by 8.2% ($p < 0.05$) at the 3g dose and by 12.4% ($p < 0.05$) at the 5g dose. Controls experienced an insignificant reduction in TC (-1.2%) from baseline.

Overall, there were no significant differences in the effect on total serum cholesterol levels between the LMW treatment groups and the HMW treatment groups.

LDL-Cholesterol

Treatment Effects vs. Control (Table 5)

Consumption of 3g and 5g LMW BBG significantly reduced LDL-C by 7.5% ($p < 0.05$) and 11.9% ($p < 0.05$), respectively, compared to the control group. HMW BBG significantly lowered LDL-C by 8% ($p < 0.05$) and 13.4% ($p < 0.05$) for 3g and 5g LMW BBG doses respectively, compared to control.

Treatment Effects vs. Baseline (Table 5)

Compared to baseline, LMW BBG significantly lowered LDL-C by 8.7% ($p < 0.05$) in the 3g group and 13.1% ($p < 0.05$) in the 5g group. Subjects who consumed the HMW BBG at 3g and 5g doses significantly decreased LDL-C by 9.2% ($p < 0.05$) and 14.6% ($p < 0.05$), respectively. Controls had an insignificant LDL-C reduction of 1.2% compared to baseline.

Overall, there were no significant differences in the effect on LDL-C levels between the LMW BBG and the HMW BBG treatment groups.

HDL-Cholesterol

LMW BBG did not significantly impact HDL-C levels at either the 3g or 5g doses (Table 5). No significant changes in HDL-C levels were observed in any of the treatment groups (Table 5).

Apolipoprotein B

Treatment Effects vs. Control (Table 5)

Consumption of LMW BBG at the 5g dose significantly decreased apo B by 12.7% ($p < 0.05$) compared to control; the 3g dose lowered apo B by 8.0%, which was not significant at ($p < 0.05$). HMW BBG significantly lowered apo B by 8.1% ($p < 0.05$) and 16.1% ($p < 0.05$) when consumed at 3g and 5g doses respectively, relative to the control.

Treatment Effects vs. Baseline (Table 5)

Apo B levels significantly dropped from baseline levels by 14.8% ($p < 0.05$) at the 5g dose and 10.1% ($p < 0.05$) at the 3g dose in the LMW BBG groups. The HMW BBG treatment significantly lowered apo B by 10.2% ($p < 0.05$) and 18.2% ($p < 0.05$) in the 3g and 5g groups, respectively. Controls experienced an insignificant drop of 2.1% compared to baseline.

There were no significant differences in the effect on apo B levels between LMW BBG and the HMW BBG treatment groups at the 5g dose.

Apolipoprotein A-1

LMW BBG did not significantly impact apo A-1 levels at either the 3g or 5g doses (Table 5). No significant changes in apo A-1 levels were observed in any of the treatment groups (Table 5).

TC/HDL-C Ratio

Treatment Effects vs. Control (Table 5)

The ratio of TC/HDL-C was significantly reduced by LMW BBG at both the 3g and 5g doses ($p < 0.05$ for both) compared to control. Of the HMW treatments, only the 5g dose significantly lowered TC/HDL-C ratio ($p < 0.05$) compared to the control.

Treatment Effects vs. Baseline (Table 5)

The ratio of TC/HDL-C was significantly reduced from baseline by LMW BBG at both the 3g and 5g doses ($p < 0.05$ for both). Neither the 3g HMW BBG group nor the controls had a significant reduction in TC/HDL-C ratio. Only the 5g HMW BBG dose significantly ($p < 0.05$) reduced TC/HDL-C.

Triglyceride

Treatment Effects vs. Control (Table 5)

Compared to the control group, both the LMW BBG treatment groups experienced a drop in fasting triglyceride levels, but this was not significant ($p < 0.05$). In the HMW BBG groups, fasting triglyceride concentrations decreased, but only the 5g dose level was significant ($p < 0.05$) compared to control.

Treatment Effects vs. Baseline (Table 5)

Fasting triglyceride levels were significantly reduced from baseline levels in all treatment groups ($p < 0.05$ for all), while the control group experienced a modest increase.

LDL Goals

The NCEP ATP III has developed guidelines that specify different LDL-C goals for individuals based on the number of CHD risk factors present (NCEP, 2002). Risk factors, exclusive of LDL-C,

include cigarette-smoking, hypertension, low HDL-C, family history of premature CHD, and older age. The LDL-C goal for individuals with 0 to 1 CHD risk factors is < 160 mg/dL, whereas the LDL-C goal for individuals with 2 or more CHD risk factors is < 130 mg/dL. Each of the study participants was assessed in relation to their own LDL-C goal at baseline and following the LMW or HMW BBG interventions (Table 6). In the LMW BBG group, of those with 0 to 1 CHD risk factors, 82% subjects consuming the 3g dose and 95% of subjects consuming the 5g dose reached their LDL-C goals. Similarly, in the 3g and 5g HMW groups, 80% and 94% of the subjects with 0 to 1 CHD risk factors reached their respective LDL-C goals. In contrast, only 74% of the control group with 0 to 1 risk factors attained their LDL-C goals. Among subjects with 2 or more CHD risk factors, 14% of the 3g LMW BBG group and 44% of the 5g LMW BBG group, respectively, attained their LDL-C goals compared to 0% in the control group. Approximately 50% and 60% of subjects in the HMW 3g and 5g groups, respectively, reached their LDL-goals within the similar risk category.

Compliance and Tolerance of Product

The treatment was well tolerated by most subjects, with excellent compliance (> 95% average) across all treatment groups and in the control group. The absence of study dropouts further supports the tolerance of study treatments. Moreover, the potential for adverse events to occur was monitored at all study visits and none were reported. Frequency of treatment-related side effects was also assessed at each study visit. There were no significant differences in the frequency of side effects at baseline between any of the study treatment group or the control group. Multiple regression analysis that looked at change over time in each of the treatment groups versus the control group did not note any statistically significant differences in any of the side effects evaluated.

DISCUSSION

Over half of the US adult population has elevated serum cholesterol levels. Public health campaigns emphasize cholesterol control as a means of reducing CHD risk. The NCEP ATP III recommends lifestyle changes such as the reduction of saturated fat and cholesterol and inclusion of moderate physical activity in lowering serum cholesterol (NCEP, 2002). To further help Americans meet their cholesterol goals, the NCEP ATP III encourages the use of viscous (soluble)

fibers to enhance serum cholesterol lowering. The panel recommends that a cholesterol-lowering diet be enriched with foods that provide at least 5g - 10g viscous (soluble) fiber daily. Intakes of 10g - 25g viscous (soluble) fiber per day can be additionally beneficial (NCEP, 2002).

The key finding of this study is that a LMW BBG (barley betafiber) or a HMW BBG, when consumed at either 3g or 5g/day, significantly reduced TC and LDL-C. The TC and LDL-C results are consistent with other clinical trials that have evaluated similar doses of barley beta-glucan (Behall et al., 2004a, 2004b; McIntosh et al., 1991) and oat beta-glucan (Davidson et al., 1991; Ripsin et al., 1992).

Lipid changes did not appear to be affected by molecular weight differences, as the LMW BBG produced similar results to the HMW BBG. Similar findings for TC and non-HDL-C were also observed in an animal study that evaluated the effects of a LMW BBG in relation to a HMW (native) BBG (Wilson et al., 2004). No other clinical trials have investigated the effect of molecular weight on barley beta-glucan. Only one human study has reported the effects of molecular weight on oat beta-glucan (Frank et al., 2004). In this randomized, crossover study, the blood lipid-lowering effects of two yeast-leavened oat bran breads containing 6g LMW beta-glucan (217 kDa) or 6g HMW beta-glucan (797 kDa) were compared. Following three weeks of consumption, neither bread lowered TC or LDL-C in the total study sample of 22 hypercholesterolemic subjects.

Concentrating barley beta-glucan using the method outlined in this study did not diminish its cholesterol-lowering potential. However, two other clinical studies of highly concentrated barley beta-glucan did not observe a similar efficacy in cholesterol lowering (Keogh et al., 2003; Biörklund et al., 2005). Differences in processing may account for variations in clinical efficacy of concentrated barley beta-glucans.

Gastrointestinal side effects associated with the consumption of both LMW and HMW beta-glucan concentrates were non-existent for nearly all the parameters measured. The Institute of Medicine (IOM) of the National Academies has stated that as part of an overall healthy diet, a high intake of dietary fiber will not produce significant deleterious effects in healthy people (IOM, 2005). Furthermore, in 2003 an independent panel of experts qualified in evaluating the safety of food and

food ingredients deemed barley betafiber (LMW barley beta-glucan concentrate) to be Generally Recognized as Safe (GRAS) for use up to 3g per serving in foods (GRAS Expert Panel Report, Appendix 1).

The National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study, a nationally representative sample of US adults, reports a median intake of 2.4g soluble fiber per 1735 kcals (Bazzano et al., 2003). This level of soluble fiber intake is well below the NCEP ATP III recommendations. Barley betafiber LMW barley beta-glucan concentrate is an excellent source of soluble fiber that can boost soluble fiber intake and, more importantly, has proven cholesterol-lowering properties.

Consumption of barley betafiber also contributes to total dietary fiber intake. Foods formulated with barley betafiber have the potential to increase both soluble fiber and total dietary fiber content. The IOM has recommended an intake of 30g - 38g total dietary fiber/day for adult men and 21g - 25g total dietary fiber/day for adult women (IOM, 2005). Recent estimates indicate Americans consume only about 14g - 19g dietary fiber per day (USDA, 1997). The 2005 Dietary Guidelines for Americans also notes that most Americans need to increase their intake of dietary fiber (USDA/DHHS, 2005).

CONCLUSION

This study demonstrates that LMW beta-glucan concentrate (barley betafiber) significantly improves TC and LDL-C in moderately hypercholesterolemic subjects. An important finding is that LMW barley beta-glucan concentrate had comparable efficacy gram-for-gram when compared to native HMW barley beta-glucan. Use of LMW barley beta-glucan concentrate (barley betafiber) has important public health implications because its use as a food ingredient has broader applications than native barley alone, and it therefore has the potential to contribute to reducing serum cholesterol in a larger segment of the population.

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This Clinical Trial Summary was prepared by Rebecca Mathews, R Mathews & Associates, on behalf of the petitioner. It is based on the manuscript written by Pins et al. that was submitted to the *Journal of Family Practice*:

Pins JJ, Keenan JM, Goulson MJ, Shamliyan T, Knutson NE, Kolberg LW, Curry LL, unpublished. The effects of concentrated barley beta-glucan on blood lipids and other CVD risk factors in a population of hypercholesterolemic men and women.

Joel J Pins has reviewed this summary and confirmed its accuracy. To the best of our knowledge, this summary is a representative and accurate representation of the study findings.

Lore W Kolberg

Lore Kolberg
Manager, Regulatory and Scientific Affairs
Cargill, Incorporated

Table 2. Baseline Characteristics of Subjects in Treatment and Control Groups

Demographic Variable	LMW		HMW		Control
	3g n = 31	5g n=30	3g n= 32	5g n= 32	n=30
Age, years; mean ± SD	55 ± 10.1	52.8 ± 11.9	53.9 ± 10.2	58.6 ± 10.6	53.7± 12.5
Gender:					
Male, n (%)	16 (51.6)	11 (36.7)	16 (50.0)	15 (46.9)	17 (56.7)
Female, n (%)	15 (48.4)	19 (63.3)	16 (50.0)	17 (53.1)	13 (43.3)
Race:					
Non-Hispanic White, n (%)	29 (93.5)	28 (93.3)	30 (93.8)	29 (90.6)	30 (100)
Other, n (%)	2 (6.5)	2 (6.7)	2 (6.2)	3 (9.4)	0
BMI, kg/m^2; mean ± SD	28.1 ± 4.3	28.9 ± 5.3	29.6 ± 5.9	28.9 ± 6.7	28.4 ± 4.0
CHD Family History, n (%)	9 (29)	9 (30)	10 (31.3)	11 (34.4)	6 (20)
Metabolic Syndrome[1], n (%)	15 (48.4)	15 (50)	16 (50)	15 (46.9)	15 (50)
Total Cholesterol, mg/dL; mean + SD	235.9 ± 23.0	238.0 ± 27.6	233.6 ± 22.8	235.1 ± 25.3	234.0 ± 22.7

[1] Each group was block stratified on metabolic syndrome status as defined by the NCEP ATP III guidelines.
Chi-square tests of association between groups were performed for gender and ANOVA F-tests were performed for age and BMI. All p values were not significant ($p > 0.09$).

Table 3. Average Body Weight at Baseline and at End of Treatment

	LMW		HMW		Control
	3g n = 31	5g n=30	3g n= 32	5g n= 32	n=30
Baseline (kg)	80.7 ± 14.9	80.7 ± 16.8	86.4 ± 19.4	81.7 ± 19.8	82.8 ± 15.1
End of Treatment (kg)	80.1 ± 14.9	80.5 ± 16.8	86.0 ± 19.0	81.4 ± 19.7	82.7 ± 14.8

Baseline measurements were made at week 0. End of treatment measurements were made at week 6.

Table 4. Dietary Intake During the Initial and Final Phases of Treatment

	LMW				HMW				Control	
	3g (n = 31)		5g (n=30)		3g (n= 32)		5g (n= 32)		(n=30)	
	Week 1	Week 5	Week 1	Week 5	Week 1	Week 5	Week 1	Week 5	Week 1	Week 5
Energy, kcal	1549 ± 436	1558 ± 466	1558 ± 466	1534 ± 510	1675 ± 656	1738 ± 606	1492 ± 369	1555 ± 445	1801 ± 503	1847 ± 692
Total Fat, g	58 ± 22.2	66 ± 25.4	61 ± 23	61 ± 29.5	59 ± 27.4	61 ± 25.2	58 ± 22.2	62 ± 34.4	66 ± 25.7	71 ± 27.8
Total Saturated Fat, g	17 ± 8.0	20 ± 8.8	19 ± 9.7	20 ± 11.9	18 ± 8.2	18.6 ± 8.8	18 ± 7.8	19 ± 10.6	21 ± 9.6	22 ± 9.1
Total Polyunsaturated Fat, g	13 ± 8.1	13.8 ± 5.4	13 ± 5.7	13 ± 6.5	13 ± 9.4	14 ± 8.1	13 ± 5.7	15 ± 12.2	15 ± 5.5	16 ± 7.8
Cholesterol, mg	732 ± 330	820 ± 367	184 ± 99	184 ± 80	198 ± 128	187 ± 125	211 ± 119	205 ± 126	207 ± 120	224 ± 127
Total Carbohydrates, g	185 ± 67.9	201 ± 79.6	185 ± 70.2	184 ± 66.9	208 ± 105.7	213 ± 96.7	168 ± 48.9	182 ± 64.6	221 ± 75.3	218 ± 98.4
Total Dietary Fiber, g	15 ± 6.2	16 ± 6.2	15 ± 5.0	14 ± 4.5	20 ± 13.1	19 ± 13.2	15 ± 5.4	16 ± 6.8	20 ± 7.5	18 ± 9.1
Soluble Fiber, g	4.9 ± 2.14	5.2 ± 2.1	4.9 ± 1.6	4.9 ± 1.4	6.5 ± 3.72	6.1 ± 3.7	4.9 ± 1.73	5.1 ± 2.1	6.0 ± 1.9	5.4 ± 2.2
Protein, g	70 ± 19.9	80 ± 26.5	67 ± 16.5	66 ± 16.7	73 ± 25.3	76 ± 27.1	72 ± 22.7	69 ± 23.6	84 ± 22.1	81 ± 23.6

Values: mean ± SD. Subjects kept 3-day diet records during the week 1 and week 5 of the treatment period. Pair-wise comparisons of the dietary data were made between weeks 1 and 5 and all differences were statistically insignificant ($p > 0.05$).

Table 5. Mean Lipid Levels at Baseline and End of Treatment

Lipid Variable	LMW		HMW		Control
	3g n =31	5g n=30	3g n=32	5g n=32	
TC (mg/dL)					
Baseline (week 0)	235.9 ± 23.0	238.0 ± 27.6	233.6 ± 22.8	235.1 ± 25.3	234.0 ± 22.7
End of treatment (week 6)	218.8 ± 20.1 *b	211.6 ± 20.2 *b	214.5 ± 21.6 *b	205.9 ± 25.1 *b	231.3 ± 26.9 a
% Change relative to baseline	- 7.2% *b	- 11.1% *b	- 8.2 % *b	- 12.4 % *b	- 1.2% a
% Change relative to control	- 6.0%*	- 9.9% *	- 7.0% *	- 11.2% *	
LDL-C (mg/dL)					
Baseline (week 0)	153.9 ± 15.1	154.6 ± 19.9	152.8 ± 18.1	154.5 ± 16.5	152.7 ± 13.9
End of treatment (week 6)	140.5 ± 15.1 *b	134.3 ± 12.8 *b	138.8 ± 20.3 *b	132.0 ± 11.4 *b	150.9 ± 24.3 a
% Change relative to baseline	- 8.7% *b	- 13.1% *b	- 9.2% *b	-14.6 % *b	- 1.2% a
% Change relative to control	- 7.5 % *	- 11.9 % *	- 8.0 % *	- 13.4 % *	
HDL-C (mg/dL)					
Baseline (week 0)	49.6 ± 14.8	50.4 ± 13.7	47.9 ± 10.7	50.8 ± 14.2	50.5 ± 14.4
End of treatment (week 6)	50.8 ± 15.8	49.7 ± 12.8	47.4 ± 11.2	51.9 ± 12.7	49.9 ± 13.8
% Change relative to baseline	+ 2.4%	- 1.4%	- 1.0%	+ 2.2%	- 1.2%
% Change relative to control	+ 3.6 %	- 0.2%	+ 0.2%	+ 3.4%	
Apo B (mg/dL)					
Baseline (week 0)	138.0 ± 14.9	138.2 ± 20.2	135.8 ± 18.6	139.9 ± 18.5	140.7 ± 15.5
End of treatment (week 6)	124.0 ± 13.0 *a	117.8 ± 13.8 *b	121.9 ± 21.0 *b	114.4 ± 14.5 *b	137.8 ± 28.6 a
% Change relative to baseline	- 10.1% *a	-14.8% *b	- 10.2% *b	- 18.2% *b	- 2.1% a
% Change relative to control	- 8.0%	- 12.7% *	- 8.1%*	- 16.1% *	
Apo-A1 (mg/dL)					
Baseline (week 0)	123.6 ± 33.9	122.9 ± 34.9	119.4 ± 30.4	119.6 ± 33.3	120.3 ± 34.5
End of treatment (week 6)	127.5 ± 34.6	126.5 ± 34.8	118.8 ± 30.7	124.7 ± 30.8	121.8 ± 37.0
% Change relative to baseline	+ 3.1%	+ 2.9%	- 0.5%	+ 4.3%	+ 1.2%
% Change relative to control	+ 1.9%	+ 1.7%	- 1.7%	+ 3.1%	

Lipid values: mean ± SD

Table 5. Mean Lipid Levels at Baseline and End of Treatment (cont'd.)

Lipid Variable	LMW		HMW		Control
	3g	5g	3g	5g	
TC/ HDL-C					
Baseline (week 0)	5.0 ± 1.2	5.0 ± 1.4	5.1 ± 1.2	4.9 ± 1.3	4.9 ± 1.2
End of treatment (week 6)	4.6 ± 1.3 *b	4.5 ± 1.2 *b	4.8 ± 1.1 a	4.2 ± 1.0 *b	5.0 ± 1.4 a
% Change relative to baseline	- 1.6% *b	- 2.0% *b	- 1.2% a	- 2.9% *b	+ 5.3% a
% Change relative to control	- 6.9%*	- 7.3% *	- 6.6%	- 12.2% *	
TG (mg/dL)					
Baseline (week 0)	154.9 ± 61.7	166.7 ± 91.7	164.7 ± 88.7	158.3 ± 79.2	153.9 ± 75.4
End of treatment (week 6)	142.2 ± 49.2 *a	145.7 ± 62.7 *a	152.5 ± 55.8 *a	133.7 ± 47.4 *b	158.8 ± 64.7 a
% Change relative to baseline	- 8.2% *a	- 12.6% *a	- 7.4% *a	- 15.5% *b	+ 3.2% a
% Change relative to control	- 11.4%	- 15.8%	- 10.6%	- 18.7%*	

Paired student t-tests were computed to compare baseline to week 6; * significant differences within groups ($p < 0.05$); ANOVA F-tests were done for each variable. There were no significant differences between groups at baseline. Different letters indicate significant differences between the groups after treatment ($p < 0.05$).

Table 6: LDL-C Goal Attainment at Baseline and Week 6 by Number of CHD Risk Factors[1]

	CHD Risk Factors (0-1) at Baseline	CHD Risk Factors (0-1) at Week 6	CHD Risk Factors ≥ 2 at Baseline	CHD Risk Factors ≥ 2 at Week 6
Control (n=30)	19/23	17/23	0/7	0/7
3g LMW (n=31)	11/17	14/17	0/14	2/14
5g LMW (n=30)	16/21	20/21	0/9	4/9
3g HMW (n=32)	10/20	16/20	1/12	6/12
5g HMW (n=32)	12/17	16/17	1/15	9/15
Any BBG Treatment (n=125)	48/75	66/75	2/50	21/50

[1]CHD risk factors as defined by NCEP ATP III guidelines