Oat β-glucan lowers total and LDL-cholesterol

Sylvia Pomeroy, Richard Tupper, Marja Cehun-Aders and Paul Nestel

Abstract Several soluble polysaccharides have been shown to have cholesterol-lowering properties and to have a role in prevention of heart disease. Major sources of one such polysaccharide (β-glucan) are oats and barley. The aim of this study was to examine the effects on plasma lipid concentrations when β-glucan derived from a fractionated oat preparation was consumed by people with elevated plasma lipids. A single-blind, crossover design compared plasma cholesterol, triglycerides, high density lipoproteins and low density lipoproteins (LDLs) in 14 people; in the order of low, high and low β-glucan supplemented diets, each of three weeks duration. For the high β-glucan diet, an average intake of 7 g per day was consumed from cereal, muffins and bread. The background diet remained relatively constant over the three test periods. Differences during the interventions were calculated by one-way repeated measures analysis of variance. Where treatments were found to be significantly different, pairwise multiple comparison procedures (Tukey Test) were carried out between the high β-glucan and each of the low β-glucan phases and there was a highly significant difference between treatments for plasma cholesterol ($P = 0.009$) and for LDL-cholesterol concentrations ($P < 0.001$). The differences in plasma cholesterol (6.42 ± 0.7, 6.14 ± 0.53, 6.44 ± 0.67 mmol/L) and LDL-cholesterol (4.59 ± 0.59, 4.17 ± 0.58, 4.52 ± 0.65 mmol/L) between high β-glucan and each of the low β-glucan treatments were significant ($P < 0.05$). The effect on LDLs (9% lower) is among the highest reported. The results of this study confirm that beneficial reductions in plasma cholesterol and LDL-cholesterol concentrations can be obtained with β-glucan incorporated into a variety of foods. **(Aust J Nutr Diet 2001:58:51–55)**

Key words: plasma cholesterol, low density lipoprotein cholesterol, oats, β-glucan.

Introduction

Oats have a strong reputation as a nutritious cereal providing more protein than any other cereal as well as insoluble fibre, soluble fibre ß-glucan, minerals, vitamins, other phytochemicals, and the unsaturated fatty acids, oleic and linoleic. Interest in soluble fibres within oats has been spurred by the acceptance of approved health claims (1) based on its plasma cholesterol and its capacity to lower low density lipoprotein (LDL). The aim of this study was to determine the effects of β-glucan derived from a fractionated oat preparation in people with elevated plasma lipids with respect to the magnitude of the changes in plasma lipids.

The benefits for lowering lipids with oats and oat-based products have been reported in animal (2) and human studies (3,4). Oat gum soluble fibre has been reported to exert a greater hypocholesterolaemic effect than several other fibres tested and found to be similar to that of cholestyramine (2).

In humans, the precise effects have been difficult to determine due to the variety of dosages, differing population groups and the nature of the study supplements. Soluble dietary fibre from oats has improved lipidaemia, as reported in both metabolic ward studies (5,6) and free-living hyperlipidaemic people (7–13). In positive trials of oat concentrate, intakes varying between 25 and 106 g daily, have shown to significantly lower serum cholesterol mostly by between 5.4 and 12.8% and LDL-cholesterol by between 8.5 and 12.4% in moderately hypercholesterolaemic subjects. Larger reductions have been reported (8–13) whereas other well executed trials have proven to be negative (14–18).

Two meta-analysis studies have shed more information on this issue. One meta-analysis (19) of 23 trials provided strong support that approximately 3 g of soluble fibre from oat products per day can lower total cholesterol concentrations from 0.13 to 0.16 mmol/L and concluded that the reduction was greater in those with higher initial cholesterol concentrations. The second meta-analysis (20) of 67 trials confirmed that various soluble fibres reduced total and LDL-cholesterol by the previously reported amounts, for example 3 g of soluble fibre from oats could decrease LDL-cholesterol by approximately 0.13 mmol/L.

In the first meta-analysis, Ripson (19) based the analysis on the content of soluble fibre rather than the amount of the oat product because it was the best representation of β-glucan, the primary soluble fibre in oat bran. Beta-glucan is a soluble, viscous polysaccharide that is found mainly in the aleurone cell layer of oats. The concentration can vary due to nature of the cultivar, growing environment and preparation technique (21). This paper examines the potential of a ß-glucan rich (12–14%) oat bran to lower the plasma lipid concentrations in a group of hypercholesterolaemic subjects. The technique used in this study concentrated on retaining all essential nutrients along with ß-glucan from aluerone cell layers, whereas other processes utilised only the ß-glucan, removing or reducing other nutrients including protein, vitamins, minerals and oil.

Method

Experimental design

A single blind study design was employed in free-living hyperlipidaemic participants to test the oat preparation derived predominantly from the aleurone cell layer of oat bran through a new commercial processing technique.

The study began with a two-week control phase during which subjects were instructed to consume their normal diet and avoid barley, oat and psyllium products, as well as dietary supplements including fish oils and special mar-

Laboratory of Cardiovascular Nutrition, Baker Medical Research Institute, Melbourne, Victoria
S. Pomeroy, BSc, MPH, GradDipEd, Nutritionist
M. Cehun-Aders, RN, BEd, Research Nurse
P. Nestel, MD, FTSE, FRACP, Head, Cardiovascular Nutrition
Uncle Tobys Company, Rutherglen, Victoria
R. Tupper, BSc(hons), MAppSci, MAIFST, Product Manager
Correspondence: S. Pomeroy, Baker Medical Research Institute, PO Box 6492, St Kilda Rd Central, Melbourne, Victoria 8008. Email: sylvia.pomeroy@baker.edu.au

garines. Subjects then proceeded to a three-phase test period comprising low ß-glucan, high ß-glucan and low ß-glucan supplemented foods. Each phase lasted three weeks. Therefore the chosen test design was one in which each subject participated in a middle test phase flanked on each side by a control phase.

The habitual diets were determined by participants completing a seven-day food frequency questionnaire (22) modified to focus on energy, total fat, saturated fat, cholesterol and fibre. Because the purpose of the questionnaire was to support a constant background pattern of eating for each person, it was administered at the beginning and end of the study. The study foods (those with and those without ß-glucan) were administered in conjunction with this background diet. Subject compliance was checked half way during each phase using food check lists.

The β-glucan was incorporated into three foods—cereal, muffins and bread with approximately equal amounts in a serve of 40 g of breakfast cereal, 70 g serve of sliced bread and a 50 g serve of muffin. The low ß-glucan periods comprised the same study foods but without ß-glucan supplementation.

The foods were eaten through the day. All foods were colour-coded but only the subjects were blinded to the identity of the foods. Compliance with the protocol was assessed on the basis of counting of test foods returned at the end of the intervention period. The laboratories carrying out the various assays were ignorant of the nature of the study.

The ß-glucan was produced by the Uncle Tobys Company Limited, Rutherglen, Victoria. For this study approximately 60 g of oat bran concentrate contained 8 g of ß-glucan.

Subjects

Fifteen men and women were recruited by advertisement that sought people with known hypercholesterolaemia who had not been treated with lipid-lowering drugs. One subject clearly did not comply and was excluded. Inclusion criterion was a total cholesterol greater than 5.5 mmol/L. Exclusion criteria included smoking, alcohol intake exceeding two standard drinks per day, dietary supplements, medication likely to affect plasma lipids, bowel, liver and kidney disorders, thyroid dysfunction and diabetes mellitus.

The Human Ethics Committee of the Alfred Group of Hospitals, Melbourne, Victoria, approved the study, and volunteers gave written consent following full disclosure and explanation of the study.

Laboratory procedures

Blood was sampled twice in each test phase, one to three days apart. Samples were drawn from the subjects after overnight fasting. Plasma cholesterol and triglycerides were measured by enzymatic kits on a Cobas-Bio automated analyser (Roche, Basel). High density lipoprotein (HDL) was first precipitated selectively from plasma and cholesterol content measured. LDL was calculated using the Friedewald formula (23).

Weight and waist to hip ratios

Subjects were weighed in light clothing without shoes and result recorded to the nearest 0.2 kg. Waist measurements were made at the level of the umbilicus, and hip measurements were made 3 cm below the umbilicus, which is close to the iliac crest.

Statistical analysis

The data were expressed as means plus or minus standard deviation. Differences during the interventions were calculated by one-way repeated measures analysis of variance. Where treatments were found to be significantly different, pairwise multiple comparison procedures (Tukey Test) were carried out between high ß-glucan and each of the low ß-glucan phases. Calculations were performed by Sigma Stat 2 software (Jandel Corporation, California, SigmaStat for Windows, Version 2, 1992–1995).

Results

The mean age of the seven men and seven women was 52 ± 10 years (range 34–69) and their mean BMI was 25 ± 3.4 kg/m^2 (range 19.3–29.8) as shown in Table 1. Their plasma lipid values on screening showed that two subjects were inadvertently entered with a cholesterol value below the inclusion criterion of 5.5 mmol/L (5.2 mmol/L, and 5.4 mmol/L.) Most were primarily hypercholesterolaemic. Nine subjects were overweight with BMI greater than 25 kg/m^2. Abdominal obesity was defined as waist to hip ratio of 1.0 or greater for men, and 0.8 or greater for women and was present in all women. On average, body mass changed less than 1 kg over the entire period. Glucose readings were within the normal range.

Dietary record

The seven-day food frequency questionnaire showed that the percentage of energy derived from total fat (35 ± 5 versus 34 ± 6) and from saturated fatty acids (13 ± 4 versus 13 ± 4) from the background diets was not significantly different at the beginning and end of the study (see Table 2). The fatty acids in the β-glucan supplements were polyunsaturated 5 g, mono-unsaturated 6 g and saturated 2 g. The fatty acid profile for the control study foods was maintained as closely as possible to the supplemented foods for amount and type of fat. This led to total fat intakes of about 76 to 79 g. Total fibre from the background diet (18 ± 5 g) remained constant. In addition

Table 1. Clinical characteristics of the 15 subjects recruited

	Mean ± sd
Age (years)	53 ± 10
Gender	8 males, 7 females
Weight (kg)	76.6 ± 16.81
BMI (kg/m$^{2)}$	25.5 ± 3.41
Waist:hip ratio	1.0 ± 0.68
Plasma cholesterol (mmol/L)	6.40 ± 0.80
Plasma triglycerides (mmol/L)	1.22 ± 0.40
Plasma glucose (mmol/L)	5.32 ± 0.34

about 20 g of fibre was provided from oat bran in the test foods, whereas the control foods contained mainly insoluble fibre from wheat. The high total fibre intake of more than 35 g per day did not appear to physically inconvenience the volunteers. Compliance was judged from self-reported data on test foods consumption and confirmed by count of test foods. The average daily β-glucan consumption was 7.4 ± 1.28 g during the supplemented phase, 92% of the 8 g target. The lower than targeted consumption of β-glucan was due to some people, especially women, being unable to eat the full ration of muffins.

Lipid changes with low and high β-glucan

Plasma cholesterol and LDL concentrations declined significantly on the high β-glucan diet and rose again on return to the control diet as shown in Table 3. The minor changes in plasma triglyceride, HDL-cholesterol, body weight, and BMI were not significant.

Discussion

This study demonstrates that the major soluble fibre of oats, ß-glucan, decreased plasma and LDL-cholesterol in a group of free-living, hypercholesterolaemic people. Whereas the majority of similar trials have reported a reduction of blood cholesterol in hypercholesterolaemic subjects consuming oat bran (5–13), other studies have found no significant effect (14–18). Several factors might account for this variability in studies: large quantities of oat bran may lead to other specified dietary modifications; low ß-glucan content of bran; and different responses among normocholesterolaemic and hypercholesterolaemic subjects.

In this study a well defined oat bran concentrate providing 8 g of ß-glucan was added to a variety of foods and compared with similar wheat-based products. All subjects had plasma total cholesterol concentrations in excess of 5.2 mmol/L and dietary records showed similar intakes of other key nutrients such as saturated fatty acids throughout.

The average plasma total cholesterol-lowering of 4% and LDL-cholesterol-lowering of 9% is consistent with results reported by Kestin et al. (9) using 95 g of oat bran concentrate and with results published by Van Horn (10) with 57 g of dry instant oats.

Conversely, Braaten et al. (24) reported almost twice the effect seen in our study (lowering of 9% versus 4.4% for serum cholesterol and 10.0 versus 9.1% for LDL-cholesterol) in subjects with similar baseline cholesterol concentrations (6.76 ± 0.13 versus 6.4 ± 0.80 in the current study) utilising only 3 g of ß-glucan derived from oat bran compared with 8 g of ß-glucan from oat bran in our study. Uusitupa et al. (3) reported similar effects to those in the current study in mild to moderate hypercholesterolaemic people with a reduction of 5.9% in LDL-cholesterol from 10 g of β-glucan.

The meta-analysis by Brown et al. (20) reported that there was no evidence to support previous findings that patients with hypercholesterolaemia were more responsive to soluble fibre than healthy individuals. Subgroup analysis of initial cholesterol concentrations showed that persons with moderate or severe hypercholesterolaemia (concentrations > 6.20 mmol/L) demonstrated only slightly greater decreases in total cholesterol than those with lower cholesterol concentrations. Nevertheless, initial LDL-cholesterol was a moderately significant predictor of changes in LDL averaging 0.02 mmol/L per

Table 2. Comparison of nutrient intakes at the beginning and end of study[a]

	Mean ± sd
Energy (kJ)	
Background beginning (without study foods)	6404 ± 2015
Background end (without study foods)	6601 ± 2235
Study foods without β-glucan	2228
Study foods with β-glucan	2282
Total fat (g)	
Background beginning	61 ± 28
Background end	62 ± 28
Background diet + study foods without β-glucan	76
Background diet + study foods with β-glucan[b]	78.9
Fibre (g)	
Background beginning	18.5 ± 5
Background end	18.5 ± 5
Fibre in study foods with β-glucan	20.24
Soluble fibre in study foods with β-glucan	8.85
Insoluble fibre in study foods with β-glucan	11.39
Cholesterol (mg)	
Background beginning	188 ± 86
Background end	180 ± 87

(a) Background values do not include the additional test foods unless indicated. No sd is given for study foods as the exact amount was given and consumed.
(b) Polyunsaturated to mono-unsaturated to saturated fatty acids ratio for oat-based supplement = 2.5:3:1

Table 3. Plasma lipids following low, high and low β-glucan dietary periods

	Mean ± sd
Total cholesterol (mmol/L)	
Low β-glucan 1	6.42 ± 0.7*
High β-glucan	6.14 ± 0.53
Low β-glucan 2	6.44 ± 0.67*
Triglycerides (mmol/L)	
Low β-glucan 1	1.25 ± 0.46
High β-glucan	1.38 ± 0.64
Low β-glucan 2	1.43 ± 0.86
Low density lipoprotein cholesterol (mmol/L)	
Low β-glucan 1	4.59 ± 0.59*
High β-glucan	4.17 ± 0.58
Low β-glucan 2	4.52 ± 0.65*
High density lipoprotein cholesterol (mmol/L)	
Low β-glucan 1	1.32 ± 0.25
High β-glucan	1.31 ± 0.25
Low β-glucan 2	1.29 ± 0.25

* Different from high, $P < 0.05$.

gram of soluble fibre. This is a conservative estimate and substantially less than in the present study.

The confounding effects of dietary modification such as displacement of dietary fat and loss of body weight are largely eliminated in studies where the type and amounts of food are controlled by the investigators. Yet, in a metabolic ward study, Beer et al. (16) failed to find a cholesterol-lowering effect in 14 normocholesterolaemic subjects despite 9 g of ß-glucan from oat gum.

In contrast, Behall et al. (25) in a less well controlled study of 23 normocholesterolaemic subjects, in which weight loss and fat displacement did occur, reported total cholesterol-lowering of 10% and 15% and LDL-cholesterol-lowering of 15 and 21% with 2 and 8 g of oat gum ß-glucan, respectively.

A possible explanation for the absence of effect in the study of Beer et al. (16) may lie in the nature of the processed ß-glucan, which had a low molecular weight (1000 kDa). By contrast the ß-glucan consumed by the subjects in Braatens' (24) study had a higher molecular weight of 1200 kDA and significantly lowered serum and LDL-cholesterol in free-living hypercholesterolaemic subjects.

Much of the physiological function of β-glucan has been attributed to its viscous nature (26). Wood et al. (27,28) found an inverse linear relationship between the mean peak blood glucose increment and log (viscosity) generated by oat gum in a glucose solution. Thus, viscosity measurement of β-glucan seems to provide an indicator of its physiological effectiveness. Much work has been conducted on viscosity measurement of β-glucan that shows that β-glucan viscosity alters depending on variety, method of fractionation or concentration, and impact of processing (shear stress, enzymic hydrolysis and, perhaps, thermal stress) (29). It is possible to produce bran-like foods that contain β-glucan with high viscosity, that theoretically would produce an improved physiological benefit (30).

Increased viscosity of gastrointestinal content induced by appropriate forms of ß-glucan may be a key factor in lowering cholesterol. Viscosity depends on the solubility of the ß-glucan and its molecular weight, which in turn is influenced by technological treatment, the cultivar and growing conditions (31). The hypothesis is that increased viscosity in the gut leads to an unstirred layer adjacent to the mucosa. This layer may serve as a physical barrier to prevent bile acid reabsorption, that leads to increased uptake of LDL-cholesterol into the liver to replenish hepatic cholesterol.

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Future events

5th Commonwealth Congress on Diarrhoea and Malnutrition

26–28 April 2001, Darwin, Northern Territory. Contact: Convention Catalysts International. Tel: (08) 8981 1875. Fax: (08) 8941 1639. Email: convention.catalysts@norgate.com.au.

20th National Dietitians Association of Australia Conference

10–12 May 2001, Adelaide Convention Centre, Adelaide SA. Contact: Elisabeth Eaton, Conference Secretariat, Festival City Conventions. Tel: (08) 8363 1307. Fax: (08) 8363 1604. Email: daa2001@fcconventions.com.au.

Speech Pathology Australia national conference—'Evidence and innovation'

20–23 May 2001, Melbourne. Contact: Gina McInnis. Tel: (03) 9642 4899. Fax: (03) 9642 4922. Email: gmcinnis@speechpathologyaustralia.org.au.

7th Annual Conference, The Australian Health Outcomes Collaboration—'Health outcomes 2001 the odyssey advances'

27–28 June 2001, Canberra ACT. Contact: Jan Sansoni or Lorna Tilley. Tel: (02) 6205 0869 or (02) 6291 7271. Fax: (02) 6205 2037. Email: jan.sansoni@act.gov.au. Website: www.health.act.gov.au/epidem/ahoc.html.

First International Scientific Congress on Nutrition and Athletic Performance

8–11 August 2001, Edmonton, Alberta, Canada. Tel: +1 780 436 5529. Fax: +1 780 437 6710. Email: srgraham@telusplanet.net. Website: www.athleticsandnutrition.com.

17th International Congress of Nutrition—'Modern aspects of nutrition: present knowledge and future perspectives'

27–31 August. 2001, Vienna, Austria. Tel: +43 1 588 00 517. Fax: +43 1 315 56 50. Email: iuns2001@verkehrsbuero.at.

New Zealand Dietetic Association 2001 Conference, 'Getting Connected'

5–7 September 2001, Christchurch, NZ. Conference Secretary, Wendy Barker. Tel: +64 3 383 1749. Email: wendybarkernz@hotmail.com or wendybarker@xtra.co.nz.

Nursing Mothers' Association of Australia International Conference

13–15 September 2001, Brisbane Qld. Contact: Elizabeth Oei. Tel: (07) 3371 6853. Fax: (07) 3871 1524. Email: t.oei@mailbox.uq.edu.au.

18th International Conference of the International Society for Quality in Health Care

2–5 October 2001, Buenos Aires, Argentina. For further information contact ISQua Conference Secretariat. Fax: (03) 9417 6851. Website: www.isqua.org.au.

Food and Nutrition Conference and Exhibition

22–25 October 2001, St Louis, Missouri, USA. Tel: +1 312 899 4855. Fax: +1 312 899 0008. Email : mtgsinfo@eatright.org.

27th Annual Scientific Meeting, Australasian Society for Parenteral and Enteral Nutrition

25–27 October 2001, Sydney. For further details contact conference convenor, Penny MacLennan. Tel: (02) 9767 8090. Fax: (02) 9767 8028. Email: mclennanp@crgmail.crg.cs.nsw.gov.au.

3rd Asian Congress of Dietetics. Harmonisation of Asian dietetics

August 2002, Kuala Lumpur, Malaysia. Contact: 3rd Asian Congress of Dietetics, Department of Nutrition and Dietetics, Faculty of Allied Health Sciences, Universiti Kebangsaan Malaysia, Jalan Raja Muda Abdul Aziz, 50300 Kuala Lumpur, Malaysia. Tel: +60 3 440 5511. Fax: +60 3 294 7621. Email: fatimah@medic.ukm.my, or, winnie@medic.ukm.my

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