Original Article

Beneficial effects of konjac powder on lipid profile in schizophrenia with dyslipidemia: A randomized controlled trial

Lei Zhang MPH¹, Yong Han PhD¹, Zhijun Zhao BMed¹, Xiangqun Liu MS¹, Yahui Xu MD², Guimei Cui PhD, MD², Xiangyang Zhang PhD, MD², Ruiling Zhang PhD, MD²

¹Department of Clinical Nutrition, The Second Affiliated Hospital of Xinxiang Medical University, Xinxiang, Henan, China

²Department of Psychiatry, The Second Affiliated Hospital of Xinxiang Medical University, Xinxiang, Henan, China

Background and Objectives: Konjac powder has the effect of improving blood lipids in the general population, but there is no research on schizophrenic patients who are susceptible to dyslipidemia. The aim of our study is to evaluate the effects of konjac powder on blood lipid, glucose levels, body weight, and blood pressure in schizophrenia inpatients with dyslipidemia. **Methods and Study Design:** After a two-week adaptation period, 76 people with schizophrenia were enrolled in a 30-day double-blind randomized controlled trial. The subjects in the experimental group were given a beverage containing konjac powder 30 minutes before each meal, whereas those in the control group were given a beverage containing resistant maltodextrin. **Results:** The lipid profile, plasma glucose, blood pressure, and body weight were measured at baseline and at the end of 30-day treatment. Fifty-nine subjects completed the study. There was a substantial decrease in total serum cholesterol in the experimental group but increased in the control group. Likewise, apolipoprotein B decreased in the experimental group but increased in the control group. Conclusions: We concluded that a diet supplemented with konjac powder may prevent the deterioration of dyslipidemia in people with schizophrenia, demonstrating its potential value in the treatment of metabolic disorders in schizophrenia as a new therapeutic method.

Key Words: konjac powder, schizophrenia, inpatient, dyslipidemia, randomized controlled trial

INTRODUCTION

Hyperlipidemia is a major risk factor for the development of atherosclerosis and coronary heart disease. The prevalence rate of dyslipidemia is higher in patients with schizophrenia than in the general population. According to a meta-analysis by Mitchell et al., up to 40% of patients with schizophrenia have lipid profile alterations.¹ One 3year follow-up study using Medicaid program information reported that 16.9% of schizophrenia patients developed new dyslipidemia from 2003 to 2005.² Unfortunately, dyslipidemia in patients with schizophrenia is often ignored.³ Data from the Clinical Antipsychotic Trials of Intervention Effectiveness schizophrenia trial found that approximately 88.0% of dyslipidemia cases in people with schizophrenia were untreated.⁴ According to current guidelines, such as those of the American College of Cardiology/American Heart Association (ACC/AHA) and the European Society of Cardiology/European Atherosclerosis Society (ESC/EAS),⁵ lifestyle changes should be the primary measures for the treatment of dyslipidemia, including adjusting diets, limiting caloric intake, and increasing physical activity.6

Dietary fibre is well known for its ability to decrease blood lipid levels, especially water-soluble dietary fibres such as pectin, psyllium, oat brans, guar gums, and glucomannan.⁷⁻⁹ Glucomannan, which is derived from Amorphophallus konjac, is mainly a straight-chain polymer with a few branches and the component sugars β -(1 \rightarrow 4)-linked D-mannose and D-glucose at a ratio of 1.6:1.¹⁰ The mechanism of action of glucomannan in the treatment of hyperlipidemia may be due to the inhibition of cholesterol and bile acid absorption in the gut and a reduction in the release of hydro-3-methyl-glutary-1 (HMG) CoA reductase.¹¹⁻¹³ To our best knowledge, there is currently a lack of research on the use of soluble fibre in the treatment of dyslipidemia in schizophrenia patients. Therefore, this study examined whether konjac flour containing \geq 65% glucomannan is beneficial for schizophrenia patients with dyslipidemia.

Corresponding Author: Prof Ruiling Zhang, Department of Psychiatry, The Second Affiliated Hospital of Xinxiang Medical University, Xinxiang, Henan, China.

Tel: 86-0373-3373980; Fax: 86-0373-3373995 Email: hy_vip@126.com

Manuscript received 06 August 2020. Initial review completed 06 March 2020. Revision accepted 23 June 2020. doi: 10.6133/apjcn.202009 29(3).0009

METHODS Subjects

Subjects

Seventy-six people with schizophrenia aged between 19 and 65 years with serum total cholesterol (TC) ≥ 6.22 mmol/L or serum triglycerides (TG) ≥2.26 mmol/L were recruited from inpatients of the Second Affiliated Hospital of Xinxiang Medical University two weeks after hospitalization.^{14,15} Exclusion criteria included taking drugs other than antipsychotics that may affect lipid metabolism, switching antipsychotics or limiting diet intake due to disease development, and existing secondary causes of dyslipidemia, such as hypothyroidism or renal disease. All subjects and their families in both groups voluntarily participated in this study with informed consent. This study was approved by the human ethics committees of the Second Affiliated Hospital of Xinxiang Medical University with the Approval number 2014001 and was registered at the Chinese Clinical Trials Registry (http://www.chictr.org.cn) with the registration number ChiCTR-INR-17011863 (data: July 8, 2017).

Sample size calculation was based on the outcome of a previous similar research,¹⁶ which total cholesterol declined 0.73 mmol/L after intervention of glucomannan. At the 0.05 level with a power of 0.8, it was calculated that a total of 40 patients should be recruited for this trial. In order to reduce statistical errors and improve test efficiency, 60 effective samples were decided.

Study design

The study employed a double-blind placebo-controlled design in a 1:1 ratio. All participants in the trial were under close management with their range of activity limited, consistently keeping to the same schedule and diet. A 2-week run-in period was included at the beginning of current study in order to eliminate possible effects of changes to drug regimens, lifestyles, and diets on metabolic indices. After this baseline period, an experimental period of 4 weeks followed. During this treatment period, participants were required to ingest konjac flour or resistant maltodextrin 6 g/d.

Allocation concealment and blinding of this trial were as follows:

a) Allocation concealment: Study designer took charge of allocation concealment. Before the trial, study designer took the integers 1 to 100 as the patients' enrolment serial number, and randomly divided them into two groups by random number table. Then the results of the grouping were stored in opaque sealed envelopes and told to the dietitian. During study implementation, the investigator responsible for enrolling the patients informed the dietitian of each patient's enrolment serial number, and the dietitian prepared the patient's meal based on the grouping results.

b) Blinding: Both konjac powder and resistant maltodextrin are colourless and odourless, as well as the beverage of them are difficult to distinguish from the appearance. Researchers who were in charge of health education of patients, collecting or analyzing data, nurse and doctor of subjects, and subjects were blinded until the statistical analysis of the data was completed.

Data collection

Blood sample collection, weight measurement, and blood pressure testing were performed at the beginning and end of the treatment. Blood samples were drawn from the anterior elbow vein after one overnight fast and tested by clinical laboratory using standard techniques. Body weight (kg) and height (cm) will be measured using the Xiheng pointer height and weight scale (Wuxi, Jiangsu) to the nearest 0.1 kg and 0.1 cm without shoes, wearing lightweight clothes, barefoot and the head positioned in the Frankfurt horizontal plane. Participants were required to visit a toilet before measurements. A questionnaire was also completed to record the types and lengths of side effects throughout the trial.

Diet

The wards of patients with schizophrenia in the hospital adopt closed management - the male and female wards are separate, and no family members allowed to accompany patients. The three meals of all patients are provided by the Nutrition Department of the hospital and the foods are the same for all patients. The only other possible way for them to get food is the snacks from the visitors. Given that the subjects are adults and have a small amount of activity in closed management wards, the dietary standards of the subjects refer to the standards of adult males or females with light physical activity recommended in the Dietary Guidelines for Chinese Residents. That is to say, we considered the gender, age and activity of the subjects, but without the body size and dietary history of them. The energy intakes per day were 1800 kcal for women and 2200 kcal for men, with roughly 25% of total calories from fat, 15% from protein, and 60% from carbohydrates. A clinical nutritionist made the recipes by using the Nutrition Therapeutic System of Traditional Chinese Medicine Combining with Western Medicine (NCCW) MX 1 (Qingdao, Shandong). Subjects were not allowed to eat snacks, especially high-fat foods, such as white bread, instant noodles, and fried potatoes.

Interventions

Trial group received konjac flour, whereas placebo control group received an equal quantity of resistant maltodextrin. Both were instructed to drink 300 mL water, in which an equal 2 g of konjac flour or resistant maltodextrin was dissolved, 30 minutes before three daily meals. Konjac powder was provided by Yuchunnong Food Co., Ltd (Dayao), which contained ≥65% glucomannan. Resistant maltodextrin powder was sourced from Xiwang Pharmaceutical Co., Ltd (Zhengzhou). Beverages were prepared by a dietician and sent to subjects by a delivery worker. To measure compliance, duty nurses were responsible for supervising and recording information about the subjects' diet and beverage intake, focusing on recording whether patients have abnormal eating behaviour, such as food refusal and overeating, or refusing to take beverage.

Statistical analyses

SPSS 22.0 software (SPSS, Chicago, IL) was used for data analysis. Continuous variables were reported as mean \pm standard deviation (SD) and Kolmogorov-

Smirnov test was applied to explore the normality of the data. The t test or rank sum test were applied to perform statistical analysis as appropriate. Categorical variables analysis adopted the Chi-square test. Two-tailed p value <0.05 was considered statistically significant.

RESULTS

Subjects' characteristics and compliance

A total of 76 patients participated in this trial from September 2015 to March 2017. Among them, 11 patients left the hospital before the end of experimental period (six patients from the treatment group, and five patients from the control group) and three patients showed poor compliance (one from the treatment group, and two from the control group). Hyperglycemia occurred in one patient, who needed to be given metformin and diet therapy. One patient appeared to have a drug allergy reaction and partial data deficiency was detected in one patient. Finally, 59 (78%) patients completed the study, 30 of whom were in the experimental group and 29 in the control group. The flow diagram of the study is shown in Figure 1. Of

the 59 subjects, 33 were men and 26 were women, with a mean age of 32.0 ± 9.8 years. The baseline characteristics were similar between the two groups (Table 1). At the end of the trial, all patients who successfully fulfilled the test did not show any apparent unusual appearance or changes to either diet or exercise, which were uniformly arranged by the hospital. During the trial, the antipsychotics and other medications remained fixed. There was no significant difference in types or chlorpromazine-equivalent doses between the 2 groups.

Outcomes of metabolic control

The outcomes of the trial on metabolic control are summarized in Table 2. Lipid profiles were improved in the glucomannan group compared with the placebo control group. TC showed substantial changes between the two groups, of which the glucomannan group nonsignificantly reduced TC by $3.90\%\pm14.38\%$ (p=0.084) throughout the trial and the placebo control group significantly increased TC by $9.19\%\pm19.59\%$ (p=0.035), respectively.

Considerable statistical changes between the two



Figure 1. Study flow diagram.

Variable	Experimental group (n=30)	Control group (n=29)	p value	
Male/Female	17/13	16/13	1.000	
Age, years (mean±SD)	32.57±9.47	31.46±10.29	0.247	
Height, centimetres (mean±SD)	167.47±6.86	166.93±6.88	0.476	
Antipsychotics			0.720	
Clozapine	11	13		
Olanzapine	9	6		
Risperidone	7	6		
Quetiapine	11	8		
Sulpiride	4	7		
Schizophrenic duration, years (mean±SD)	6.87±5.43	7.25 ± 6.00	0.082	

Table 1. Baseline characteristics of experimental and control participants (n=59)[†]

[†]All continual data complied with normal distribution and two independent t test was applied to compare the two groups.

groups were also observed in apolipoprotein (Apo) B. In the experimental group, Apo B was increased nonsignificantly by 5.05%±15.92%, while Apo B was decreased nonsignificantly in the placebo control group by 5.03%±19.79%. There were no statistical differences in high-density lipoprotein cholesterol (HDL-C), lowdensity lipoprotein cholesterol (LDL-C), TG, Apo A1, fasting blood glucose (FBG), blood pressure, and body weight changes between the two groups.

Additionally, we carried out an analysis of differences between males and females. Only the changes of TC between the two groups showed a marked difference in males but not in females. Also, major differences in Apo B and FBG changes were detected only in females. Other major end-point value differences were not found.

DISCUSSION

The result of this randomized controlled trial showed that taking 2 g konjac powder with 300 mL water 30 minutes before each meal improved the metabolic profiles of hospitalized schizophrenic patients with dyslipidemia. We observed improvement in the metabolic parameters of TC, Apo B, and FBG relative to the placebo control treatment using resistant maltodextrin.

A number of trials have been previously undertaken to examine the effectiveness of konjac powder in the general population in the treatment of metabolic syndrome (abdominal obesity, hyperinsulinemia, dyslipidemia, and hypertension). Sood et al. performed a systematic review and meta-analysis to better characterize the effect of konjac powder on metabolic profile,17 including 14 randomized controlled studies with a total of 531 participants. Of the trials, glucomannan dosages ranged from 1.2 to 15.1 g per day. Forms of glucomannan were various, including capsules, tablets, bars, biscuits, and refined konjac meal. The combined result showed that glucomannan had a positive effect on lowering TC, LDL-C, TG, body weight, and FBG compared with control intervention, but no beneficial effectiveness in reducing BP or increasing HDL was noted. However, a recent metaanalysis, showed that glucomannan did not significantly lower body weight.18

It is generally known that HDL-C is a protective factor for atherosclerosis and LDL-C a risk factor. The determination of LDL-C can be affected by a high level of TG, requiring fasting before blood draws. As an alternative, the determination of Apo A1 and Apo B has been valued and used widely, especially in the context of coronary heart disease prediction.^{18,19} Apo A1 and Apo B are the main proteins of HDL-C and LDL-C separately, but the correlation between Apo A1 (or Apo B) and HDL-C (or LDL-C) is not consistent.^{20,21} In the present trial, LDL-C was not reduced in the glucomannan group in comparison with the placebo group, while Apo B was. This finding may be due to the high level of TG of some subjects involved in this trial.

Additionally, we carried out a subgroup analysis based on gender. The percentage change of TC showed a statistically significant difference between glucomannan group and placebo group only in male subjects. Nevertheless, significant decreases in Apo B and FBG were observed in females and not males. Many of the previous studies found significant gender differences in metabolic profiles. A crossover randomized controlled trial performed in children by Guardamagna et al. revealed that glucomannan caused a 6.1% drop in TC and a 9% drop in LDL-C in females, while the two parameters did not show significant differences in males.²² Another study, also conducted in children with dyslipidemia, compared the change percentage between sex groups and found that TC (female vs. male, 24% vs. 9%) and LDL-C (female vs. male, 30% vs. 9%) decreased more significantly in female children than in male children after 8 weeks of intervention with glucomannan in gelatine capsules. Similar discoveries also emerged in the study of the cholesterol-lowering effects of psyllium,23 a commonly available source of dietary soluble fibre.²⁴⁻²⁶ Both Jenkins et al26 and Sonia et al23 found that sex and hormonal status influence the effects of psyllium on lipid profiles by making a comparison among men, premenopausal women, and postmenopausal women.

This study also investigated FBG, blood pressure, and body weight. However, no statistical significance was observed, with the exception of FBG in female patients. The lack of significant findings might be because the participants involved in our study did not have hypertension, obesity, or hyperglycemia. Glucomannan did not affect the normal physiological variation of blood pressure, body weight, and glucose. These findings were consistent with those of earlier studies.²⁷⁻²⁹

There exist some shortcomings in this study. First, sample size of this trial is inadequate for subgroup analysis, leading to can't detect the population who gained the most benefit from konjac powder intervention. Second,

Parameter		Experimental group		Control group			
	Baseline	30 days	Change (%)	Baseline	30 days	Change (%)	- p value
TC (mmol/L)		· · · · · ·			·		
Overall	4.83 ± 0.99	4.60 ± 0.96	$-3.90{\pm}14.38$	4.64 ± 0.81	4.98 ± 0.83	9.19±19.59¶	0.005‡
Male	4.62 ± 1.01	4.47 ± 1.07	-3.11±14.69	4.67±0.62	5.05 ± 0.93	8.43±16.88	$0.044^{\$}$
Female	5.11±0.93	4.78 ± 0.80	-4.93 ± 14.49	4.61±1.03	4.91±0.71	10.13 ± 23.18	0.058
HDL-C (mmol/L)							
Overall	$0.95{\pm}0.16$	0.95±0.15	-2.13 ± 19.80	1.00 ± 0.28	1.05 ± 0.23	-7.55±17.24	0.267
Male	$0.95{\pm}0.15$	0.94±0.13	-1.99 ± 20.36	0.97±0.24	1.00 ± 0.24	-4.25±13.52	0.712
Female	$0.96{\pm}0.19$	0.96 ± 0.17	-2.31 ± 19.87	1.03 ± 0.33	1.10 ± 0.20	-11.62 ± 20.78	0.254
LDL-C (mmol/L)							
Overall	2.66 ± 0.66	2.72 ± 0.75	5.06 ± 31.01	2.61±0.63	2.90 ± 0.74	14.34±31.62¶	0.260
Male	2.55±0.71	2.63 ± 0.79	7.17±37.39	2.61±0.49	2.90±0.81	11.46±27.21	0.710
Female	$2.81{\pm}0.57$	$2.84{\pm}0.70$	2.31 ± 21.10	2.61 ± 0.80	2.90 ± 0.69	17.89 ± 37.18	0.201
TG (mmol/L)							
Overall	3.42±1.43	2.73±1.16	-17.55±30.11¶	2.85 ± 0.85	2.66±1.32	-2.75±47.42	0.157
Male	$3.54{\pm}1.80$	2.84±1.36	-14.56±37.21	$3.02{\pm}0.80$	3.09±1.29	8.70±54.35	0.159
Female	$3.27{\pm}0.78$	2.58 ± 0.86	-21.45±17.76¶	2.65 ± 0.89	2.13±1.21	-16.85±34.13	0.670
Apo A1 (g/L)							
Overall	$1.17{\pm}0.18$	1.11 ± 0.21	3.67±19.64	1.12 ± 0.24	1.14 ± 0.22	-4.21±19.92	0.132
Male	1.21 ± 0.16	1.12 ± 0.17	7.11±14.26¶	1.09 ± 0.24	1.12 ± 0.24	-4.59±19.61	0.058
Female	1.12 ± 0.20	1.11 ± 0.27	-0.83 ± 24.95	1.15 ± 0.25	1.16±0.19	-3.75 ± 21.10	0.750
Apo B (g/L)							
Overall	$1.01{\pm}0.22$	0.98 ± 0.24	-5.05 ± 15.92	0.96 ± 0.22	1.03 ± 0.24	5.03±19.79	0.035‡
Male	$0.96{\pm}0.24$	0.97 ± 0.27	0.01 ± 13.89	$0.97{\pm}0.20$	1.05 ± 0.26	4.68 ± 19.48	0.432
Female	$1.07{\pm}0.19$	0.98±0.21	-11.67±16.48¶	0.95 ± 0.25	1.02 ± 0.22	5.46 ± 20.95	0.029 [§]
FBG (mmol/L)							
Overall	5.10±0.79	5.11 ± 1.14	0.45 ± 18.57	5.08 ± 0.81	5.27 ± 0.87	4.16±10.94	0.356
Male	5.11±0.96	5.33±1.42	4.51±21.73	5.23±1.01	5.37±1.11	3.10±12.83	0.823
Female	$5.09{\pm}0.54$	4.81±0.52	-4.87 ± 12.23	4.91±0.45	5.16±0.45	5.45 ± 8.40	0.019 [§]
Systolic blood pressure (mmHg)							
Overall	114.47±13.12	117.67±8.87	3.66±10.38	112.90±11.60	116.14±11.61	3.38±10.40	0.919
Male	117.82±14.29	119.00±8.14	1.95 ± 10.22	113.56±10.81	119.69±9.34	5.99±10.36	0.268
Female	110.08 ± 10.33	115.92 ± 9.80	$5.90{\pm}10.56$	112.08 ± 12.90	111.77±12.96	0.18 ± 9.90	0.167

Table 2. Effects of glucomannan on efficacy outcomes[†]

[†]All data were shown as mean ±SD and complied with the normal distribution. [‡]Changes between experimental group and control group showed significant difference by two independent t test. [§]Changes in female or male between the two groups showed significant difference by two independent t test. [§]Changes in experimental group or control group between initial and final period of the trial significantly statistical difference by paired-sample t test

Parameter		Experimental group		Control group			
	Baseline	30 days	Change (%)	Baseline	30 days	Change (%)	- p value
Diastolic blood pressure (mmHg)							
Overall	76.47±9.13	77.03 ± 7.55	1.67 ± 12.61	72.52±12.44	74.14 ± 7.94	7.95±42.84¶	0.445
Male	77.65±10.14	79.18 ± 7.00	2.89±10.56	72.13±14.45	76.63 ± 8.74	15.62 ± 56.43	0.368
Female	74.92±7.75	74.23 ± 7.59	$0.07{\pm}15.18$	73.00±9.99	71.08 ± 5.77	-1.50 ± 11.26	0.768
Body weight (kg)							
Overall	73.73±11.22	74.97±11.23	1.75±3.18¶	70.41±11.93	71.24±11.42	1.39 ± 3.52	0.688
Male	76.12±12.68	77.76±12.67	2.26±3.52¶	73.88±12.70	74.25±12.79	0.51±2.03	0.093
Female	70.62 ± 8.44	71.31 ± 8.07	$1.07{\pm}2.65$	66.15±9.74	67.54±8.55	2.48±4.63	0.352
Body weight index (kg/m ²)							
Overall	26.31±3.72	26.74±3.65	1.75±3.18¶	25.23 ± 3.70	25.53 ± 3.50	1.39±3.52¶	0.688
Male	25.79±4.03	$26.34{\pm}4.00$	2.26±3.52¶	25.34±3.68	25.47±3.71	0.51±2.03	0.093
Female	26.99±3.29	27.26±3.22	1.07 ± 2.65	25.10±3.87	25.61±3.36	$2.48{\pm}4.63$	0.352

Table 2. Effects of glucomannan on efficacy outcomes[†] (cont.)

[†]All data were shown as mean ±SD and complied with the normal distribution. [‡]Changes between experimental group and control group showed significant difference by two independent t test.

[§]Changes in female or male between the two groups showed significant difference by two independent t test.

¹Changes in experimental group or control group between initial and final period of the trial significantly statistical difference by paired-sample t test.

the intervention time is short. Because of the median length of hospital stay of the hospital was 52 days, to reduce the drop-out rate, intervention time had to be limited. Therefore, future trial may consider including long-term hospitalized patients. Three, owing to lack of sufficient funds, this trial is lack of follow-up evaluation, in this regard, we are applying for research grant, in order to operate further research.

Conclusion

In conclusion, the intake of glucomannan before meals demonstrated improvement of TC, Apo B, and FBG in people with schizophrenia with dyslipidemia from our 30day intervention study. Other outcomes, such as HDL-C, LDL-C, TC, blood pressure, and body weight, were not altered significantly. The findings of this study are consistent with those reported by studies on the general population.^{22,28,30} It is an encouraging result for people with schizophrenia that glucomannan may be used in addition to drug therapy or as an alternative to drug treatment in the amelioration of dyslipidemia. Given the higher morbidity rate of dyslipidemia and other metabolic abnormalities in people with schizophrenia, more studies on the effect of diet and exercise in this population are required. To further clarify the potential benefits and to find the optimal dosage of konjac powder in people with schizophrenia with dyslipidemia, however, studies with larger sample sizes and of longer duration are warranted.

ACKNOWLEDGEMENTS

The authors thank Bingshen Hu, Zheng Zhao, Zhiying Zhang, Li Deng, Lingfeng Yue, and Fei Pan for their great supporting this study.

AUTHOR DISCLOSURES

The authors declare no conflict of interest.

This study was funded by the Department of Clinical Nutrition, the Second Affiliated Hospital of Xinxiang Medical University and the Project 51282 of Henan Province Health and Family Planning Technology Innovation Talents.

REFERENCES

- Mitchell AJ, Vancampfort D, De Herdt A, Yu W, De Hert M. Is the prevalence of metabolic syndrome and metabolic abnormalities increased in early schizophrenia? A comparative meta-analysis of first episode, untreated and treated patients. Schizophr Bull. 2013;39:295-305. doi: 10. 1093/schbul/sbs082.
- Jerrell JM, McIntyre RS, Tripathi A. Incidence and costs of cardiometabolic conditions in patients with schizophrenia treated with antipsychotic medications. Clin Schizophr Relat Psychoses. 2010;4:161-8. doi: 10.3371/csrp.4.3.2.
- Baller JB, McGinty EE, Azrin ST, Juliano-Bult D, Daumit GL. Screening for cardiovascular risk factors in adults with serious mental illness: a review of the evidence. BMC Psychiatry. 2015;15:55. doi: 10.1186/s12888-015-0416-y.
- Nasrallah HA, Meyer JM, Goff DC, McEvoy JP, Davis SM, Stroup TS, Lieberman JA. Low rates of treatment for hypertension, dyslipidemia and diabetes in schizophrenia: data from the CATIE schizophrenia trial sample at baseline. Schizophr Res. 2006;86:15-22. doi: 10.1016/j.schres.2006. 06.026.
- Graham IM, Catapano AL, Wong ND. Current guidelines on prevention with a focus on dyslipidemias. Cardiovasc Diagn Ther. 2017;7(Suppl 1):S4-10. doi: 10.21037/cdt.2017.04.04.

- Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63:2960-84. doi: 10.1016/j.jacc.2013.11.003.
- Anderson JW, Spencer DB, Hamilton CC, Smith SF, Tietyen J, Bryant CA, Oeltgen P. Oat-bran cereal lowers serum total and LDL cholesterol in hypercholesterolemic men. Am J Clin Nutr. 1990;52:495-9. doi: 10.1093/ajcn/ 52.3.495.
- Lullien V, Alary R, Guirao A, Joudrier P, Gautier MF. Isolation and nucleotide sequence of a cDNA clone encoding the bread wheat (Triticum aestivum L.) CM17 protein. Plant Mol Biol. 1991;17:1081-2. doi: 10.1007/ bf00037147.
- Haskell WL, Spiller GA, Jensen CD, Ellis BK, Gates JE. Role of water-soluble dietary fiber in the management of elevated plasma cholesterol in healthy subjects. Am J Cardiol. 1992;69:433-9. doi: 10.1016/0002-9149(92)90980d.
- Katsuraya K, Okuyama K, Hatanaka K, Oshima R, Sato T, Matsuzaki K. Constitution of konjac glucomannan: chemical analysis and 13C NMR spectroscopy. Carbohyd Polym. 2003;53:183-9. doi: 10.1016/S0144-8617(03)00039-0.
- 11. Julibert A, Bibiloni MDM, Bouzas C, Martínez-González M, Salas-Salvadó J, Corella D et al. Total and subtypes of dietary fat intake and its association with components of the metabolic syndrome in a Mediterranean population at high cardiovascular risk. Nutrients. 2019;11:1493. doi: 10.3390/ nu11071493.
- Ebihara K, Schneeman BO. Interaction of bile acids, phospholipids, cholesterol and triglyceride with dietary fibers in the small intestine of rats. J Nutr. 1989;119:1100-6. doi: 10.1093/jn/119.8.1100.
- Kiriyama S, Enishi A, Yura K. Inhibitory effect of konjac mannan on bile acid transport in the everted sacs from rat ileum. J Nutr. 1974;104:69-78. doi: 10.1093/jn/104.1.69.
- McGuire A. Diagnosing the diagnostic and statistical manual of mental disorders. Disability & Society. 2015;30: 1-4. doi: 10.1080/09687599.2015.1062233.
- Chu J. Chinese guidelines on prevention and treatment of dyslipidemia in adults. Chinese Journal of Cardiology. 2007;5:390-419. doi: 10.3969/j.issn.1672-7185.2012.16.002. (In Chinese)
- 16. Martino F, Martino E, Morrone F, Carnevali E, Forcone R, Niglio T. Effect of dietary supplementation with glucomannan on plasma total cholesterol and low density lipoprotein cholesterol in hypercholesterolemic children. Nutr Metab Cardiovasc Dis. 2005;15:174-80. doi: 10. 1016/j.numecd.2004.04.004.
- Sood N, Baker WL, Coleman CI. Effect of glucomannan on plasma lipid and glucose concentrations, body weight, and blood pressure: systematic review and meta-analysis. Am J Clin Nutr. 2008;88:1167-75. doi: 10.1093/ajcn/88.4.1167.
- Onakpoya I, Posadzki P, Ernst E. The efficacy of glucomannan supplementation in overweight and obesity: a systematic review and meta-analysis of randomized clinical trials. J Am Coll Nutr. 2014;33:70-8. doi: 10.1080/0731 5724.2014.870013.
- 19. Lamarche B, Moorjani S, Lupien P, Cantin B, Bernard P, Dagenais G, Després J-P. Apolipoprotein A-I and B levels and the risk of ischemic heart disease during a five-year follow-up of men in the Quebec Cardiovascular Study. Circulation. 1996;94:273-8. doi: 10.1161/01.CIR.94.3.273.
- 20. Sniderman A, Jungner I, Junger I, Holme I, Aastveit A, Walldius G. Errors that result from using the TC/HDL C

ratio rather than the apoB/apoA-I ratio to identify the lipoprotein-related risk of vascular disease. J Intern Med. 2006;259:455-61. doi: 10.1111/j.1365-2796.2006.01649.x.

- 21. Downs J, Clearfield M, Weis S, Whitney E, Shapiro D, Beere P et al. Primary prevention of acute coronary events with lovastatin in men and women with average cholesterol levels results of AFCAPS/TexCAPS. JAMA. 1998;279: 1615-22. doi: 10.1001/jama.279.20.1615.
- 22. Guardamagna O, Abello F, Cagliero P, Visioli F. Could dyslipidemic children benefit from glucomannan intake? Nutrition. 2013;29:1060-5. doi: 10.1016/j.nut.2013.02.010.
- Vega-López S, Conde-Knape K, Vidal-Quintanar R, Shachter N, Fernandez M. Sex and hormonal status influence the effects of psyllium on lipoprotein remodeling and composition. Metabolism. 2002;51:500-7. doi: 10. 1053/meta.2002.31342.
- Brown L, Rosner B, Willett WW, Sacks FM. Cholesterollowering effects of dietary fiber: a meta-analysis. Am J Clin Nutr. 1999;69:30-42. doi: 10.1093/ajcn/69.1.30.
- 25. Anderson JW, Davidson MH, Blonde L, Brown WV, Howard WJ, Ginsberg H, Allgood LD, Weingand KW. Long-term cholesterol-lowering effects of psyllium as an adjunct to diet therapy in the treatment of hypercholesterolemia. Am J Clin Nutr. 2000;71:1433-8. doi: 10.1093/ajcn/71.6.1433.
- 26. Jensen CD, Haskell W, Whittam JH. Long-term effects of

water-soluble dietary fiber in the management of hypercholesterolemia in healthy men and women. Am J Cardiol. 1997;79:34-7. doi: 10.1016/S0002-9149(96)00672-8.

- Zortea K, Franco VC, Francesconi LP, Cereser KM, Lobato MI, Belmonte-de-Abreu PS. Resveratrol supplementation in schizophrenia patients: A randomized clinical trial evaluating serum glucose and cardiovascular risk factors. Nutrients. 2016;8:73. doi: 10.3390/nu8020073.
- 28. Vuksan V, Sievenpiper JL, Owen R, Swilley JA, Spadafora P, Jenkins DJ et al. Beneficial effects of viscous dietary fiber from Konjac-mannan in subjects with the insulin resistance syndrome: results of a controlled metabolic trial. Diabetes Care. 2000;23:9-14. doi: 10.2337/diacare.23.1.9.
- 29. Keithley JK, Swanson B, Mikolaitis SL, DeMeo M, Zeller JM, Fogg L, Adamji J. Safety and efficacy of glucomannan for weight loss in overweight and moderately obese adults. J Obes. 2013;2013:610908. doi: 10.1155/2013/610908.
- 30. Vuksan V, Jenkins DJ, Spadafora P, Sievenpiper JL, Owen R, Vidgen E, Brighenti F, Josse R, Leiter LA, Bruce-Thompson C. Konjac-mannan (glucomannan) improves glycemia and other associated risk factors for coronary heart disease in type 2 diabetes. A randomized controlled metabolic trial. Diabetes Care. 1999;22:913-9. doi: 10. 2337/diacare.22.6.913.