Original Article

Systematic review and meta-analysis of soy products consumption in patients with type 2 diabetes mellitus

Bin Yang MSc¹, Ying Chen MSc¹, Tongchen Xu PhD¹,², Yinghua Yu PhD¹,³, Tao Huang PhD¹, Xiaojie Hu MSc¹, Duo Li PhD¹

¹Department of Food Science and Nutrition, Zhejiang University, Hangzhou, China
²Institute of Agro-food Science and Technology, Shandong Academy of Agricultural Science, Jinan, China
³School of Health Sciences, University of Wollongong, Australia

Clinical trials have reported the lipid-lowering effect of consuming soy products, and epidemiological studies have shown that soy intake is associated with decreased risk of type 2 diabetes mellitus (T2DM). The aim of this meta-analysis was to systematically review the effects of soy products consumption on serum lipid profiles and glycaemic control in T2DM patients. Potential papers were initially searched from PubMed (1966 to 2010) and Cochrane Library (1984 to 2010) without language limitations. All randomized controlled trials were included in which soy products supplementation was the only intervention in subjects with type 2 diabetes. Weighted mean effect size was calculated for net changes in serum lipids and fasting glucose concentrations using fixed-effect or random-effect models. Previously defined subgroup analyses were performed to identify the source of heterogeneity. Eight studies were included according to the criteria. The intake of soy products was associated with a significant reduction in serum total cholesterol (by 0.42 mmol/L; 95% confidence interval (CI): -0.70, -0.14; \( p < 0.001 \)), triacylglycerol (by 0.22 mmol/L; 95% CI: -0.38, -0.07; \( p < 0.001 \)) and low-density lipoprotein-cholesterol (by 0.30 mmol/L; 95% CI: -0.60, -0.00; \( p = 0.89 \)), and a significant increase in high-density lipoprotein-cholesterol (0.05 mmol/L; 95% CI: 0.04, 0.06; \( p = 0.89 \)). There were no significant effects on fasting glucose, insulin and glycated hemoglobin. It can be concluded that intake of soy and soy products has beneficial effects in T2DM patients in relation to serum lipids.

Key Words: soy products, type 2 diabetes mellitus, lipids, fasting glucose, meta-analysis

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most common chronic diseases, which has become a major health problem causing significant morbidity and mortality.¹ It was reported that diabetes will affect about 285 million adults around the world in 2010, and the number will increase to 439 million in 2030.² Clinical trials have shown that lifestyle interventions (which including dietary changes) could significantly reduce the risk of complications in type 2 diabetes,³ even more effective than pharmacological intervention (metformin).⁴

Soybean, a member of the legume family, is rich in protein, fiber, vitamins, minerals, polyunsaturated fatty acids and phytoestrogens etc, which are related to glucose and insulin homeostasis.⁶⁻⁸ Animal studies have demonstrated that soy protein and soy isoflavones improve glycemic control, lower insulin requirement, and increase insulin sensitivity. Epidemiological studies have shown that elevated soy protein consumption is associated with decreased serum lipid profiles.⁹ It has also been reported that soy-based diet is effective in reducing total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C) and triacylglycerol (TG), and in increasing the high-density lipoprotein-cholesterol (HDL-C),¹⁰⁻¹² suggesting that soy products may be beneficial in the management of T2DM.

However, in prospective studies, the link between elevated consumption of soy products and risk of T2DM is still inconsistent. The relationship between soy products intake and T2DM was first reported in a 20-year follow-up survey from the Finnish and Dutch cohorts of the Seven Countries Study, which found that a high consumption of legumes was related to a protective effect on the development of glucose intolerance and diabetes.¹³ Soy products consumption is generally high in Asia populations, which could strengthen the ability of epidemiologic studies to determine associations between soy products intake and T2DM. A cohort study conducted in the population of Shanghai Women’s Health Study also found that consumption of soybean was inversely associated with risk of T2DM among middle-aged Chinese women.¹⁴
Conversely, a cohort study conducted on a Japanese population, suggested that consumption of soy product and isoflavones overall were not significantly associated with the risk of T2DM in either men or women. Consistent with this, a study in Hawaii suggested that soy consumption is not protective against diabetes. Clinical trials and epidemiology studies have also documented that soy products was associated with serum lipids in subjects with obesity, hyperlipemia and in postmenopausal women. In addition, several studies have reported the beneficial effect of soy products consumption in subjects with T2DM, but the results have not been consistent. In order to systematically review the relationship between soy products consumption and risk factors in T2DM patients, we conducted this meta-analysis.

MATERIALS AND METHODS

Search strategy and selection criteria
We conducted a search from PubMed (1966 to 2010) and the Cochrane Library using the following terms: soy, soya, soybeans, tofu, miso or natto and diabetes mellitus, T2DM or type 2 diabetes mellitus without time and language limitations. The references cited by the published original studies and relevant reviews were also searched. The additional articles were also conducted on Web of Sciences to minimize possible publication bias. In order to ensure the quality and validity of selected studies, the studies included in this meta analysis met the following criteria: 1) the study design was a randomized controlled trial; 2) the supplement should use soy (included soy protein combined with soy isoflavones) or soy products (tofu, miso or tofu) as the only intervention in subjects with T2DM; 3) it provided specifically tested soy-based interventions and the amount and frequency of soy products; 4) the study reported at least one of the continuous outcomes we were interested in: TC, HDL-C, LDL-C, TG, fasting glucose, fasting insulin and glycated hemoglobin (HbA1c). Studies evaluating soy fiber, soy polysaccharides, soy isoflavones alone, pinitols extracted from soybeans and soy combined foods, and the subjects with type 1 diabetes mellitus were not included in the present study.

Data collection
Two investigators (Bin Yang and Ying Chen) extracted detailed information in the predefined criteria independently to ensure consistency in data collection. Any disagreements were discussed to obtain consistency by the two investigators. These information included: author identification, year of publication, study design (cross-over, parallel, double-blind, etc.), participant characteristics (age, sex, BMI, diabetes status), sample size, duration of patient follow-up, type of intervention (soy dosage and form), and type of placebo. In addition, we also captured the metabolic parameters of the participant including those at baseline and endpoints, which were recorded as the mean ± standard deviation (SD). If a study had multiple time points for the same participants, only the last endpoint was used for analysis.

Data synthesis and statistical analysis
We converted different units to mg/dL (for lipids and glucose) and pmol/L (for insulin) by using the conversion factors 1 mg/dL = 0.0259 mmol/L for cholesterol, 1 mg/dL = 0.0113 mmol/L for TG, 1 mg/dL = 0.0556 mmol/L for glucose and 1 pmol/L = 6.945 μU/mL for insulin when these former units were reported in the studies. We calculated the mean change of TC, TG, HDL-C, LDL-C and glucose, by subtracting the mean change in the placebo group from that in the treatment group in the studies with parallel design; in studies with a crossover design, the mean changes were gained by calculating the difference in post-treatment concentrations for the treatment and placebo periods.

For the extraction of SD values for an outcome, we excluded the percentage change data in mean and SD, because the actual mean change of each patient was unknown. We calculated SD from standard errors, 95% confidence interval (CI), p-values, or t, F if necessary. The missing SD for change from baseline were imputed using the methods of Cochrane Handbook, assuming correlation coefficient of 0.5 between the beginning and end of each intervention. Sensitivity analysis was performed to test the influence of studies with imputed SD on the overall result.

Both fixed-effects models and random-effects models were used to calculate the weighted mean differences (WMD) and 95% CI for each outcome according to the level of heterogeneity. The random-effects models were presented when the significantly heterogeneity was found in the test. Otherwise, the fixed-effects models were used to calculate the effect of soy products. Statistical heterogeneity was tested by Cochrane’s Q, p-value <0.10 was considered to represent significantly statistical heterogeneity, which is a threshold to determine which effect model was conducted. Sources of heterogeneity were investigated by sensitivity analyses and subgroup analyses. Visual inspection of funnel plots and Egger’s weighted regression statistics were used to evaluate the presence of publication bias. The Trim and Fill method was used to assess the potential effect of any publication bias on the meta-analysis results for each outcomes to determine whether the results of the original analysis were affected by publication bias. All of the statistical analysis was performed by using STATA version 11.0 (StataCorp, College Station, Tex). A p-value<0.05 was considered statistically significant for all analyses.

RESULTS

Characteristics of the studies
After primary selection, ten clinical studies met the criteria for analysis. Of all ten studies, the study of Gonzalez et al. and Ble-Castillo et a. were excluded for using soy isoflavones and soy milk as intervention, which does not belong to the soy products category. Therefore, we included eight studies which represented 183 patients with T2DM. The characteristics of the selected studies are summarized in Table 1. Pipe et al and Gobert et al were thought to be derived from the same trials based on its identical characteristics. The study durations of included trials ranged from 6 weeks to 4 years. Seven studies used crossover design and only one study used parallel design, indicating that the results of this meta-analysis are robust.
Table 1. Characteristics of selected 8 randomized controlled trails of soy foods supplement in type 2 diabetes patients

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age (yr)</th>
<th>BMI (kg/m²)</th>
<th>Diabetic complication</th>
<th>T2DM durations (yr)</th>
<th>n</th>
<th>Menopausal status and gender</th>
<th>Study Design and washout</th>
<th>Duration</th>
<th>Diet dosage and dosage form</th>
<th>Diabetes therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anderson et al. (1998)</td>
<td>64.5±2.3</td>
<td>35.1±2.2</td>
<td>obesity, hypertension, proteinuria</td>
<td>&gt;5.0</td>
<td>8</td>
<td>M (4wks)</td>
<td>C</td>
<td>8 wks</td>
<td>111 g/SP</td>
<td>1 g animal protein/kg/bwt</td>
</tr>
<tr>
<td>Hermansen et al. (2001)</td>
<td>63.6±7.5</td>
<td>30.1±4.2</td>
<td>NO</td>
<td>3.0±2.7</td>
<td>20</td>
<td>14M, 6F (3 wks)</td>
<td>D, C</td>
<td>6 wks</td>
<td>50 g ISP (&gt;165 mg ISF) and 20 g SF</td>
<td>50 g casein and 20 g cellulose</td>
</tr>
<tr>
<td>Jayagopal et al. (2002)</td>
<td>62.5±6.77</td>
<td>32.2±5.0</td>
<td>NO</td>
<td>2.6±2.7</td>
<td>32</td>
<td>POM</td>
<td>D, C (2wks)</td>
<td>12 wks</td>
<td>SP 30 g/day, ISF 132 mg/day</td>
<td>30 g pure microcrystalline cellulose</td>
</tr>
<tr>
<td>Azadbakht et al. (2003)</td>
<td>62.5±12.1</td>
<td>NR</td>
<td>nephropathy</td>
<td>NR</td>
<td>14</td>
<td>10M, 4F (wks)</td>
<td>C</td>
<td>7 wks</td>
<td>20 g/SP †</td>
<td>20 g animal protein ‡</td>
</tr>
<tr>
<td>Teixeira et al. (2004)</td>
<td>53-73</td>
<td>29.8±0.8</td>
<td>nephropathy</td>
<td>NR</td>
<td>14</td>
<td>M</td>
<td>C (wks)</td>
<td>8 wks</td>
<td>0.5 g/kg/d ISP, 2.0 mg ISF</td>
<td>0.5 g/d casein and insulin</td>
</tr>
<tr>
<td>Azadbakht et al. (2008)</td>
<td>62.1±12.1</td>
<td>NR</td>
<td>nephropathy</td>
<td>10±3</td>
<td>41</td>
<td>18M, 23F</td>
<td>R, P</td>
<td>4 yrs</td>
<td>20 g/SP *</td>
<td>20 g animal protein †</td>
</tr>
<tr>
<td>Pipe et al. (2009); Gobert et al. (2009)</td>
<td>60.1±9.64</td>
<td>29.6±4.07</td>
<td>NO</td>
<td>3.4±4.8</td>
<td>29</td>
<td>16M, 13POM</td>
<td>D, C (4wks)</td>
<td>58 d</td>
<td>40 g/d SP, 88 mg/d ISF</td>
<td>40 g/d milk protein, 0 ISF</td>
</tr>
</tbody>
</table>

Abbreviations: NR, not reported; C, crossover; D, double-blind; P, parallel; SP, soy protein; ISP, isolated soy protein; ISF, isoflavones; SF, cotyledon fiber. POM, postmenopausal women
†0.8 g protein/kg body weight (35% animal proteins, 35% textured soy protein, and 30% vegetable proteins)
‡70% animal proteins and 30% vegetable proteins
§Range or mean±SD
¶Mean±SD
study used a parallel design. Four studies involved 110 T2DM patients without any complications, three studies were carried out on T2DM patients with nephropathy and one study was carried out on diabetes patients with obesity, hypertension and proteinuria. One study focused on postmenopausal women, two on men, and three on both genders. Six studies reported results of serum lipid concentrations, while two studies reported values for plasma. Four studies reported fasting glucose concentrations.

We ignored the different concentrations of these outcomes in serum and plasma, because it was only slightly different, and we considered them without correction for its difference. We reported all results as serum concentrations. All studies used soy protein containing isoflavones as supplementation ranging from 20 g/d to 111 g/d. Three studies used fiber (cellulose, casein) as placebo diet, three used animal protein, and two used milk protein. Of all eight include trials, four studies reported the T2DM patients were not receiving any kind of additional drugs or diet medications during intervention and placebo periods, three reported that patients received various sorts of anti-diabetic drugs such as insulin, statins, and hypoglycemic agents.

**Effect of soy products in T2DM patients**

Figure 1 reports the forest plots for the effects of soy products on TC, TG, LDL-C and HDL-C in patients with T2DM. The mean change in TC was statistically affected in subjects with T2DM supplemented with soy products (-0.35 mmol/L; 95% CI: -0.69, -0.01) (Figure 1A) compared with placebo groups. But there was high heterogeneity ($I^2=91.5\%$, $p<0.001$). Heterogeneity was partly explained by Azadbakht et al and when this longest duration study was excluded, the heterogeneity was reduced ($I^2=28\%$, $p=0.23$) (Table 2).

The pooled estimate showed a marginal significant reduction in LDL-C, the overall effect was -0.30 mmol/L (95% CI: -0.60, -0.00) with a high heterogeneity ($I^2=94\%$, $p<0.001$) (Figure 2B). Heterogeneity was partly explained by Azadbakht et al. and when this longest duration study was excluded, the heterogeneity was reduced ($I^2=0\%$, $p=0.49$) (Table 2). The mean change in HDL-C significantly increased in subjects with T2DM supplemented with soy products, the overall effect was 0.05 mmol/L (95% CI: 0.04, 0.06) and no heterogeneity was observed ($I^2=0\%$, $p=0.89$).

The supplementation of soy products appears to significantly lower TG (-0.22 mmol/L; 95% CI: -0.38, -0.07), but a high heterogeneity was observed ($I^2=85\%$, $p<0.001$).
Soy products and type 2 diabetes mellitus patients

Table 2. Subgroup and sensitivity analysis of TC, TG, LDL-C HDL-C and fasting blood glucose using different exclusion criteria

<table>
<thead>
<tr>
<th>Subgroup and sensitivity analysis</th>
<th>TC (mmol/L)</th>
<th>TG (mmol/L)</th>
<th>LDL-C (mmol/L)</th>
<th>HDL-C (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean difference (95% CI)</td>
<td>Mean difference (95% CI)</td>
<td>Mean difference (95% CI)</td>
<td>Mean difference (95% CI)</td>
</tr>
<tr>
<td>All studies</td>
<td>N</td>
<td>p</td>
<td>N</td>
<td>p</td>
</tr>
<tr>
<td>Trim and fill</td>
<td>7</td>
<td>-0.42† (-0.70, -0.14)</td>
<td>0.00</td>
<td>7</td>
</tr>
<tr>
<td>Placebo diet</td>
<td>2</td>
<td>-0.60† (-0.92, -0.28)</td>
<td>0.00</td>
<td>3</td>
</tr>
<tr>
<td>With animal protein</td>
<td>4</td>
<td>-0.48† (-0.82, -0.14)</td>
<td>0.00</td>
<td>4</td>
</tr>
<tr>
<td>Without animal protein</td>
<td>3</td>
<td>-0.28† (-0.56, 0.00)</td>
<td>0.52</td>
<td>3</td>
</tr>
<tr>
<td>Excluded Azadbakht et al (14)</td>
<td>6</td>
<td>-0.34† (-0.49, -0.20)</td>
<td>0.23</td>
<td>6</td>
</tr>
<tr>
<td>Excluded Anderson et al (22)</td>
<td>6</td>
<td>-0.40† (-0.75, -0.04)</td>
<td>0.00</td>
<td>6</td>
</tr>
</tbody>
</table>

†p<0.05; NA: Not applicable; †p for heterogeneity

(Figure 2D). Systematic removal of each trial during sensitivity analysis and subgroup analysis did not alter the heterogeneity (Table 2). However, no heterogeneity was observed after removal of both Anderson et al and Azadbakht et al synchronal (I²=0%, p=0.42) (Data not shown).21,27

Of all eight included studies, only four studies reported serum fasting glucose concentration in a way that permitted pooling of data (Figure 2A). The pooled weighted mean difference of fasting glucose was -0.68 mmol/L (95% CI: -1.78, 0.42). High heterogeneity was observed (I²=86%, p=0.001). The pooled weighted mean difference of insulin was -0.77 pmol/L (95% CI: -4.16, 2.62). Five trials reported HbA1C data that was amenable to meta-analysis. The pooled weighted mean difference was -0.09% (95% CI: -0.50% to 0.31%).

Publication bias and sensitivity analysis

Visual inspection of funnel plots (data not shown) could not rule out publication bias for many of the analyses. Review of Egger’s weighted regression statistics suggested that publication bias was unlikely for TG, HDL-C, insulin and HbA1c, while a relatively strong suspicion of publication bias for TC (Egger’s test, p=0.01), LDL-C (Egger’s test, p=0.08) and fasting blood glucose (Egger’s test, p=0.04) was observed. However, the results of soy products on TC, LDL-C and fasting blood glucose did not change after recalculating effect size estimates using the Trim and Fill methodology. Trim and Fill analysis also suggested that two studies could potentially exist but were masked by publication bias. However, soy products had a significant, with a little reinforcement, effect when theoretically “missing” studies were imputed for TG (-0.33 mmol/L, 95% CI: -0.48, -0.17) (Table 2).

Sensitivity analysis showed that the significance in the pooled changes in TC, LDL-C, HDL-C, fasting blood glucose, insulin and HbA1C were not altered after the imputation correlation coefficient of 0.5 according to Follmann et al.20 The results of subgroup and sensitivity analysis are presented in Table 2. Exclusion of the study by Anderson et al. did not change the pooled estimate and heterogeneity in any outcomes.21 The study of Azadbakht et al could affect the heterogeneity in TC and LDL-C.27 We did a subgroup analysis by duration of ≤8 and >8 wk, with the latest data available for each study in each subgroup. The longer-term subgroup showed a more robust effect on LDL-C than the shorter-term subgroup. In addition, we did a subgroup analysis by different types of placebo diet, it appears that soy products have more robust effects on TC, TG and LDL-C in T2DM patients with animal protein as placebo diet than those with fiber as placebo diet. Sensitivity analysis with method of design
was also performed. No robust effects on any endpoints in patients with T2DM were found (data not shown).

**DISCUSSION**

In the present study, eight randomized clinical trials representing 183 T2DM patients who consumed soy products showed a significant decrease in serum TC, LDL-C, HDL-C and TG. However, no significant effect was found on fasting blood glucose, insulin or HbA1c.

Soy products have been recognized as a potential benefit but not a predictable factor in the management of T2DM. However, studies which only provided indirect relationships showed that these observed benefits of soy products in T2DM are biologically plausible. The present study is the first report to systematically assess the potential effects of soy products consumption in T2DM patients. Present data suggests that the beneficial effects of soy products in T2DM patients are attributed to
the reduction of serum TC, LDL-C and TG. Epidemiological studies have proved that soy products consumption is associated with lower risk of coronary heart disease (CHD) and ischemic stroke. This beneficial effect may partially be explained by alteration of blood lipids. Our data are also relevant to clinicians managing patients with T2DM. It was well documented that a reduction in TC and LDL-C and an increase in HDL-C is associated with a decrease in the risk of cardiovascular disease (CVD) for T2DM patients. Therefore, soy products consumption might be a potential dietary intervention to reduce the risk of CVD for patients with T2DM. Consistent with our speculation, Eastman et al found that soy products consumption could reduce the risk of CHD in the patients with T2DM. Thus, it can be concluded that soy product consumption might be useful as an adjunctive therapy for dyslipidemia in T2DM patients.

Our pooled estimates indicate that the effect of soy products on LDL-C and TC were more remarkable in T2DM patients with complications than in T2DM without complications, which may be caused by the different initial serum lipid concentrations in different subgroups. The potential mechanisms responsible for the beneficial effects of soy products on the serum lipid profiles are still being explored. Animal studies report that soy protein is a main component in soy products which may reduce the insulin/glucagons ratio, by down-regulating the expression of the hepatic transcription factor sterol regulatory element binding protein, thereby decreasing LDL-C in serum and liver. Moreover, isoflavones may regulate lipid metabolism by activation of peroxisome proliferator-activated receptors. In addition, isoflavones may also exert an effect on lipid metabolism through reducing the activity of lipoprotein lipase.

Studies have demonstrated that fasting serum glucose concentration significantly decreased after soy product supplementation in postmenopausal women. However, in the present study, there is no significant effect of soy products consumption on fasting serum glucose concentration in T2DM patients compared with placebo groups. One of the possible reasons is that subjects in the present study were quite different from that of previous clinical trials. Studies have reported that soy products have no effects on sex hormones in men, while increasing sex hormones in women, which is associated with diabetes in men and postmenopausal women. In addition, some of effects of soy products may influence by menopausal status. However, both men and women of different menopausal status were included in our study. Another explanation may be the duration of follow-up. The duration of studies selected in our study were relatively short (no more than 12 weeks), which is not sufficient to observe clinically significant changes in fasting blood glucose. However, we would still expect to see a trend or tendency toward beneficial changes in fasting serum glucose with soy products supplementation compared with placebo after this short time period, if in fact such a benefit truly existed.

The limitation of the present meta-analysis is the wide criteria used to select appropriate trials. Because evidence has shown that dietary fiber itself has lipid lowering effects, we excluded papers which directly mentioned soy fiber and pinitol as potential factors. The complex components of soy and various types of interventions make the effects of soy intake on T2DM patients difficult to assess. We only ensured that all subjects were diagnosed with diabetes mellitus according to the information provided by the published papers, but failed to enact a unified criteria for the selection of T2DM patients. Secondly, crossover studies are thought to have a lower internal validity than an equivalent parallel study although all of the crossover design studies in our study explicitly state the presence and duration of the washout period. No changes in any of endpoints were noted after we did sensitivity analysis, which make the inclusions of our study. In addition, the medications used by subjects was ignored, some patients did not use medications such as insulin or other hypoglycemic agents to regulate the glucose levels, which may produce a possible confounding effect on results. Because of the insufficient number of studies included in this meta-analysis, the results should be interpreted with caution and considered hypothesis-generating only. Moreover, the observed effects from meta-analysis were therefore not adjusted for other factors that could affect the outcomes. For example, the effect of soy products was influenced by the duration of follow-up because we failed to adjust for it. Therefore, we could not exclude chance as an explanation for significant results which are based on limited comparisons, although subgroup analyses were performed to identify and minimize heterogeneity. Larger and long-term randomized clinical trials are needed to make it clear which soy component is responsible for its beneficial effect in T2DM.

In summary, we found that intake of soy and soy products have beneficial effects in T2DM patients in relation to serum TC, TG, LDL-C and HDL-C. More longer-term randomized controlled trials are needed to provide further information and confirmation of these findings.

AUTHOR DISCLOSURES
Authors declare that there is no conflict of interest in this paper.

REFERENCES


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Bin Yang BSc1, Ying Chen BSc1, Tongchen Xu PhD1,2, Yinghua Yu PhD3, Tao Huang BSc1, Xiaojie Hu MSc1, Duo Li PhD1

1Department of Food Science and Nutrition, Zhejiang University, Hangzhou, China
2Institute of Agro-food Science and Technology, Shandong Academy of Agricultural Science, Jinan, China
3School of Health Sciences, University of Wollongong, Australia

二型糖尿病患者大豆制品摄入的系统综述和 meta 分析

临床干预研究发现大豆制品具有降血脂的功效。流行病学研究发现大豆摄入与二型糖尿病患病风险呈显著负相关。该 meta 分析的主要目的是系统综述大豆制品摄入对二型糖尿病患者血脂水平及血糖的影响。本文从 PubMed (1966 到 2010) 和 Cochrane Library (1984 到 2010) 数据库收集了所有的文献，并选择了关于大豆制品干预二型糖尿病患者的随机对照实验。通过固定模型和随机模型分析了血脂和血糖的加权平均效应值，同时通过亚组分析的方法检测了数据的异质性。根据本文数据的提取标准，最终获得 8 篇论文数据。结果发现，大豆制品摄入能够显著降低二型糖尿病患者血清总胆固醇(by 0.42 mmol/L; 95% confidence interval (CI): -0.70, -0.14; p<0.001)、甘油三酯(by 0.22 mmol/L; 95% CI: -0.38, -0.07; p<0.001) 以及低密度脂蛋白胆固醇的水平 (by 0.30 mmol/L; 95% CI: -0.60, -0.00; p<0.001)，同时能够显著升高高密度脂蛋白胆固醇的浓度 (0.05 mmol/L; 95% CI: 0.04, 0.06; p=0.89)。然而，对空腹血糖、胰岛素和糖基化血红蛋白没有显著作用。因此，大豆制品能够通过调节血脂水平来起到预防二型糖尿病的作用。

关键词：大豆制品、二型糖尿病、血脂、空腹血糖、meta分析