Randomized control trials

Effects of synbiotic food consumption on metabolic status of diabetic patients: A double-blind randomized cross-over controlled clinical trial

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SUMMARY

Background & aims: We are aware of no study indicating the effects of synbiotic food consumption on metabolic profiles, inflammation and oxidative stress among diabetic patients. The aim of the current study was to investigate the effects of synbiotic food consumption on metabolic profiles, hs-CRP and biomarkers of oxidative stress among diabetic patients.

Methods: This randomized double-blinded cross-over controlled clinical trial was performed among 62 diabetic patients aged 35–70 y. After a 2-wk run-in period, subjects were randomly assigned to consume either a synbiotic (n = 62) or control food (n = 62) for 6 weeks. A 3-week washout period was applied following which subjects were crossed over to the alternate treatment arm for an additional 6 weeks. The synbiotic food consisted of a probiotic viable and heat-resistant Lactobacillus sporogenes (1 × 10^7 CFU), 0.04 g inulin (HPX) as prebiotic with 0.38 g isomalt, 0.36 g sorbitol and 0.05 g stevia as sweetener per 1 g. Control food (the same substance without probiotic bacteria and prebiotic inulin) was packed in identical 9-gram packages. Patients were asked to consume the synbiotic and control foods three times a day. Fasting blood samples were taken at baseline and after a 6-wk intervention to measure metabolic profiles, hs-CRP and biomarkers of oxidative stress.

Results: Consumption of a synbiotic food, compared to the control, resulted in a significant decrease in serum insulin levels (changes from baseline: −1.75 ± 0.60 vs. +0.95 ± 1.09 μIU/mL, P = 0.03). Although we failed to find a significant effect of synbiotic food consumption on total- and LDL-cholesterol levels and HOMA-IR, the effects on FPG (22.3 vs. 61.2 mg/dL, P = 0.09), serum triglycerides (45.9 vs. 20.6 mg/dL, P = 0.08) and HDL-cholesterol levels (3.1 vs. −2 mg/dL, P = 0.06) tended to be significant. A significant reduction in serum hs-CRP levels (−1057.86 ± 283.74 vs. 95.40 ± 385.38 ng/mL, P = 0.01) was found following the consumption of synbiotic food compared with the control group. Supplementation with the synbiotic food led to a significant increase in plasma total GSH (319.98 vs. 19.73 μmol/L, P < 0.001) and serum uric acid levels (−0.7 vs. −0.1 mg/dL, P = 0.04) compared to the control food. No significant effect of the synbiotic food was observed on plasma TAC levels.

Conclusions: In conclusion, consumption of a synbiotic food for 6 weeks among diabetic patients had significant effects on serum insulin, hs-CRP, uric acid and plasma total GSH levels.

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1. Introduction

Type 2 diabetes (T2D) is a metabolic disorder that is characterized by high blood glucose levels, insulin resistance and relative insulin deficiency. Diabetes is highly prevalent in the world with an estimated prevalence of 8.3% in US. In Iran, it has been
estimated that 8% of adult population are affected. Several studies have reported that increased levels of inflammatory factors and biomarkers of oxidative stress can result in the development of T2D. Elevated inflammation and oxidative stress can also play a key role in the pathogenesis of both macro- and micro-vascular complications of diabetes.

Prior studies have shown that decreasing circulating levels of pro-inflammatory factors and biomarkers of oxidative stress in diabetic patients are associated with better glycemic control. The effectiveness of diet therapy and antioxidant supplementation and high fitness levels on these biomarkers has been reported. Some studies have suggested that consumption of synbiotic foods might help controlling the metabolic profile, inflammatory factors and biomarkers of oxidative stress. However, such effects have mainly been observed in animal models or non-diabetic patients. Liang et al. showed that intake of a synbiotic containing Lactobacillus acidophilus, fructose-oligosaccharide, inulin and mannitol for 8 weeks resulted in a decreased serum triglyceride, total- and LDL-cholesterol levels as well as increased HDL-cholesterol concentration in hypercholesterolemic pigs. In addition, increased levels of superoxide dismutase and glutathione, along with reduced levels of nitric oxide have been reported with a consumption of synbiotic containing L. acidophilus and inulin in a murine model. Symbiotics influence the production of short chain fatty acid (SCFA), carbon disulfide and methyl acetate and can increase the lipolytic activity. Their direct immunomodulatory effects as well as down-regulatory influence on genes involved in toll-like receptor (TLR) pathways might explain their favorable actions. Due to their effects on caveolin-1, endothelial NOS and neuronal NOS down-regulation, symbiotics might affect oxidative stress.

We are aware of no study indicating the effects of synbiotic food consumption on metabolic profiles, inflammation and oxidative stress among diabetic patients. The aim of the current study was, therefore, to investigate the effects of a synbiotic food on metabolic profiles, hs-CRP and biomarkers of oxidative stress in diabetic patients.

2. Subjects and methods
2.1. Participants

This randomized double-blinded crossover controlled clinical trial was carried out in Kashan, Iran, during July 2011 to January 2012. On the basis of sample size formula suggested for cross-over clinical trials, we considered the type I error of 5% (α = 0.05) and type II error of 20% (β = 0.20; Power = 80%) and serum hs-CRP levels as a key variable and reached the sample size of 23 patients for each group. Diagnosis of T2D was done based on the criteria of American Diabetes Association: those with one of the following criteria were considered as having T2D: fasting plasma glucose (FPG) ≥126 mg/dL, blood sugar (BS) 2-h pp ≥200 mg/dL and HbA1C ≥6.5%. Individuals with the above-mentioned inclusion criteria were called for participation in the study from those that attended Golabchi Diabetes Clinic affiliated to Kashan University of Medical Sciences, Kashan, Iran. Subjects were not included if they were pregnant, using insulin or vitamin supplements, or had chronic kidney disease, liver, lung and chronic or acute inflammatory disease, heart valve disease, short bowel syndrome and allergies. A total of 70 patients with T2D aged 35 to 70 y were recruited in the study and were randomly assigned to receive either a synbiotic (n = 35) or control food (n = 35) for 6 weeks. The study was conducted according to the guidelines laid down in the Declaration of Helsinki. The ethical committee of Qom University of Medical Sciences approved the study (91288-917-5) and informed written consent was obtained from all participants.

2.2. Study design

To obtain detailed information about the dietary intakes of study participants, all patients entered into a 2-wk run-in period; during which all subjects had to refrain from taking any other synbiotic and probiotic foods. During the run-in period, participants were asked to record their dietary intakes for three non-consecutive days. At the end of run-in period, subjects were randomly assigned to the initial arm of the study to receive either a synbiotic or control food for 6 weeks. A 3-week washout period was applied following which subjects were crossed over to the alternate treatment arm for an additional 6 weeks. Participants were asked not to alter their routine physical activity or usual diets and not to consume any synbiotic, probiotic and fermented products other than the one provided to them by the investigators. Symbiotic or control foods were provided to participants every month. Compliance with the consumption of foods was monitored once a week through phone interviews. The compliance was also double-checked by the use of three-day dietary records completed throughout the study in each phase of intervention. To obtain nutrient intakes of participants based on these three-day food diaries in each phase, we used Nutritionist IV software (First DataBank, San Bruno, CA) modified for Iranian foods.

2.3. Symbiotic and control foods

The synbiotic food consisted of a probiotic viable and heat-resistant Lactobacillus sporogenes (1 × 107 CFU), 0.04 g inulin (HPX) as prebiotic with 0.38 g isomalt, 0.36 g sorbitol and 0.05 g stevia as sweetener per 1 g. Patients were asked to consume the synbiotic food three times a day in a 9 g package. Therefore, they received 27 × 107 CFU L. sporogenes and 1.08 g inulin each day. Control food (the same substance without probiotic bacteria and prebiotic inulin) was packed in identical packages and coded by the producer to guarantee blinding. The synbiotic and control foods were provided by Sekkeh Gaz Company, Isfahan, Iran.

The probiotic effects of L. sporogenes have earlier been shown. Although different studies have used different dosages of the probiotics, we used it at the dosage of 107 based on prior publications that indicated the efficacy of this dosage of L. sporogenes on lipid profiles. The dosage of inulin we used in the current study was comparable to prior studies.

2.4. Assessment of variables

Anthropometric measurements were assessed at baseline and after 6 weeks of intervention in each separate arm. Body weight was measured in an overnight fasting status, without shoes and in a minimal clothing state by the use of a digital scale (Seca, Hamburg, Germany) to the nearest 0.1 kg. Height was measured using a non-stretched tape measure (Seca, Hamburg, Germany) to the nearest 0.1 cm. BMI was calculated as weight in kg divided by height in meters squared. Fasting blood samples (10 mL) were taken at baseline and after 6-wk intervention in each separate arm at Kashan reference laboratory in an early morning after an overnight fast. Plasma glucose levels were quantified by the use of glucose oxidase/peroxidase (GOD-POD) method with commercially available kits (Pars Azmoon Inc, Tehran, Iran). Serum insulin levels were assayed by enzyme-linked immunoassay kits (DiaMetrA, Italy). Insulin resistance was assessed using the homeostatic model assessment of insulin resistance (HOMA-IR). Serum total cholesterol and triacylglycerol concentrations were assayed using commercial kits (Pars Azmoon Inc, Tehran, Iran) by enzymatic colorimetric tests with cholesterol oxidase p-aminophenazone and glycerol phosphate oxidase, respectively. Serum HDL-C levels were assay.
measured after precipitation of the apolipoprotein B containing lipoproteins with phosphotungstic acid. Serum LDL-cholesterol levels were also measured using available kits. Serum hs-CRP was quantified by ELISA using enzyme linked immunoassay kits (LDN, Nordhorn, Germany). Plasma samples were analyzed for concentrations of TAC and total glutathione levels. Plasma TAC was assessed by the use of FRAP method developed by Benzie and Strain. The plasma total GSH was measured by the method of Beutler et al. Serum uric acid concentrations were assayed using uric acid kit (Pars Azmoon Inc, Tehran, Iran).

2.5. Statistical analysis

To ensure the normal distribution of variables, Histogram and Kolmogrov–Smirnov test were applied. For non-normally distributed variables, log-transformation was applied. Descriptive statistics (means, SEMs and range) for general characteristics of the study participants were reported. Data on dietary intakes were compared by paired t-test. To determine the effect of synbiotic food on metabolic profiles, we applied repeated measures analysis of variance. In these analyses, the treatments (synbiotic and control foods) were regarded as between-subject factors and time was considered as within-subject factor. To assess carry-over effect, we computed the average of end-of-trial values for each variable for two treatments separately and compared the means of the two treatment orders by t test. To assess if the magnitude of the change depended on the starting value, we conditioned all analyses on baseline values to avoid the potential bias that might have resulted. All statistical analyses were done using the Statistical Package for Social Science version 17 (SPSS Inc., Chicago, Illinois, USA).

3. Results

Of the 70 participants enrolled in the study, 62 patients (19 males and 43 females) completed the entire crossover study. Among individuals in symbiotic food group, 4 persons [chronic kidney disease (n = 1), need to insulin therapy (n = 1), supplement therapy (n = 1) and increased need to medications (1)] were dropped out. The exclusions in the control group were also 4 patients [need to antibiotic treatment (n = 1), need to insulin therapy (n = 2) and supplement therapy (n = 1)]. Finally, 62 participants [symbiotic group (n = 62) and control group (n = 62)] completed the trial (Fig. 1). Mean age and duration of diabetes was 53.1 ± 8.7 y and 8.0 ± 4.1 y, respectively.

No serious adverse reactions were reported following the consumption of the symbiotic food in patients with T2D throughout the study. Comparing the anthropometric measures at baseline and also after intervention, we failed to find a significant difference in weight and BMI between the two groups (Table 1).
Based on the three-day dietary records throughout the study, no statistically significant difference was seen between the two groups in terms of dietary intakes of energy, fat, saturated fatty acids (SFA), polyunsaturated fatty acids (PUFA), mono-unsaturated fatty acids (MUFA), cholesterol, dietary fiber, vitamin C and selenium (Table 2).

Consumption of a synbiotic food, compared to the control, resulted in a significant decrease in serum insulin levels (changes from baseline: $-1.75 \pm 0.60$ vs. $+0.95 \pm 1.09$ μIU/mL, $P = 0.03$) (Table 3). Although we failed to find a significant effect of synbiotic food consumption on total- and LDL-cholesterol levels and HOMA-IR, the effects on FPG (22.3 vs. 4.2 mg/dL, $P = 0.08$), serum tri-glycerides (45.9 vs. 20.6 mg/dL, $P = 0.08$) and HDL-cholesterol levels (3.1 vs. $-2$ mg/dL, $P = 0.06$) were tended to be significant. A significant reduction in serum hs-CRP levels ($-1057.86 \pm 283.74$ vs. $95.40 \pm 385.38$ mg/mL, $P = 0.01$) was found following the consumption of synbiotic food compared with the control group. Supplementation with synbiotic food led to a significant increase in plasma total GSH (319.98 vs. 19.73 μmol/L, $P < 0.001$) and serum uric acid levels ($+0.7$ vs. $-0.1$ mg/dL, $P = 0.04$) compared to the control food. No significant effect of synbiotic food was observed on plasma TAC levels. When we adjusted the analyses for baseline values, no significant changes in our findings were observed (Data not shown).

### 4. Discussion

This cross-over study revealed that consumption of synbiotic food for 6 weeks among diabetic patients had significant effects on serum insulin, hs-CRP, uric acid and plasma total GSH levels; however, we failed to find any significant effect on plasma TAC levels compared to control food. To the best of our knowledge, this study is the first examining the effect of synbiotic food on metabolic status of diabetic patients.

Patients with T2D are susceptible to metabolic abnormalities, elevated systemic inflammation and oxidative stress. Several studies have shown that increased inflammation and oxidative stress in diabetic patients can contribute to the development of diabetes complications such as retinopathy, neuropathy and nephropathy. Although several attempts have been done to prevent the incidence of diabetes complications, limited data are available assessing the effects of synbiotic foods. However, earlier studies on the effects of synbiotics have mostly been assessed in vitro and in patients with multiple injuries. We demonstrated that intake of synbiotic food resulted in significantly decreased serum insulin levels as well as marginally increased serum HDL-cholesterol levels, but could not affect plasma glucose, serum total- and LDL-cholesterol levels. Earlier studies reported that supplementation with probiotics and inulin as a prebiotic led to reduced insulin levels. The use of a synbiotic food containing L. acidophilus, fructooligosaccharide, inulin and mannitol has resulted in decreased

### Table 1

General characteristics of the study participants. ($a$) Data are means ± standard deviation. ($b$) Paired $t$-test.

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 62)</th>
<th>Synbiotic food (n = 62)</th>
<th>$P^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight at study baseline (kg)</td>
<td>75.42 ± 1.96</td>
<td>74.88 ± 1.12</td>
<td>0.78</td>
</tr>
<tr>
<td>Weight at end-of-trial (kg)</td>
<td>75.39 ± 1.09</td>
<td>74.76 ± 1.13</td>
<td>0.75</td>
</tr>
<tr>
<td>Weight change</td>
<td>$-0.03 \pm 2.44$</td>
<td>$-0.12 \pm 1.57$</td>
<td>0.62</td>
</tr>
<tr>
<td>BMI at study baseline (kg/m²)</td>
<td>29.90 ± 5.18</td>
<td>29.60 ± 4.53</td>
<td>0.70</td>
</tr>
<tr>
<td>BMI at end-of-trial (kg/m²)</td>
<td>29.88 ± 5.19</td>
<td>29.55 ± 4.54</td>
<td>0.66</td>
</tr>
<tr>
<td>BMI change</td>
<td>$-0.02 \pm 1$</td>
<td>$-0.05 \pm 0.62$</td>
<td>0.78</td>
</tr>
</tbody>
</table>

### Table 2

Dietary intakes of study participants throughout the study. ($a$) Data are means ± standard deviations. ($b$) Obtained from paired $t$ test. ($c$) SFA: saturated fatty acid. ($d$) PUFA: poly-unsaturated fatty acid. ($e$) MUFA: mono unsaturated fatty acid.

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 62)</th>
<th>Synbiotic food (n = 62)</th>
<th>$P^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kcal/d)</td>
<td>2205 ± 293</td>
<td>2237 ± 215</td>
<td>0.59</td>
</tr>
<tr>
<td>Fat (g/d)</td>
<td>94.9 ± 22.2</td>
<td>98.7 ± 21.8</td>
<td>0.46</td>
</tr>
<tr>
<td>SFA ($g/d$)</td>
<td>20.9 ± 4</td>
<td>22.8 ± 5.1</td>
<td>0.08</td>
</tr>
<tr>
<td>PUFA ($g/d$)</td>
<td>43.2 ± 14.6</td>
<td>43.2 ± 11.4</td>
<td>0.99</td>
</tr>
<tr>
<td>MUFA ($g/d$)</td>
<td>24.2 ± 1.5</td>
<td>26 ± 1.5</td>
<td>0.37</td>
</tr>
<tr>
<td>Cholesterol (mg/d)</td>
<td>191.8 ± 100.5</td>
<td>192.7 ± 96.2</td>
<td>0.97</td>
</tr>
<tr>
<td>Dietary fiber (g/d)</td>
<td>16.1 ± 4</td>
<td>15.8 ± 3</td>
<td>0.70</td>
</tr>
<tr>
<td>Vitamin C (mg/d)</td>
<td>153.1 ± 57.1</td>
<td>171.4 ± 77</td>
<td>0.25</td>
</tr>
<tr>
<td>Selenium (μg/d)</td>
<td>73.60 ± 20.04</td>
<td>75.10 ± 22.03</td>
<td>0.76</td>
</tr>
</tbody>
</table>

### Table 3

Means (±standard error) of metabolic profiles, hs-CRP and biomarkers of oxidative stress at baseline and after the intervention.

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 62)</th>
<th>Synbiotic food (n = 62)</th>
<th>$P^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPG ($mg/dL$)</td>
<td>168.4 ± 8.1</td>
<td>172.6 ± 8.3</td>
<td>4.2 ± 7</td>
</tr>
<tr>
<td>Insulin (μIU/mL)</td>
<td>8.08 ± 0.59</td>
<td>9.03 ± 1.08</td>
<td>0.95 ± 1.09</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>3.27 ± 0.25</td>
<td>3.96 ± 0.60</td>
<td>0.69 ± 0.52</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>175.7 ± 6.8</td>
<td>183.5 ± 6.9</td>
<td>7.8 ± 6</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>169.9 ± 12</td>
<td>190.5 ± 14.9</td>
<td>20.6 ± 9.9</td>
</tr>
<tr>
<td>LDL-cholesterol ($mg/dL$)</td>
<td>91.4 ± 4.3</td>
<td>97 ± 5</td>
<td>5.6 ± 3.4</td>
</tr>
<tr>
<td>HDL-cholesterol ($mg/dL$)</td>
<td>50.3 ± 1.9</td>
<td>48.3 ± 1.4</td>
<td>2.2 ± 2</td>
</tr>
<tr>
<td>Total HDL cholesterol ratio</td>
<td>3.6 ± 0.9</td>
<td>3.9 ± 0.9</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>hs-CRP ($ng/mL$)</td>
<td>18977.35 ± 360.22</td>
<td>1993.15 ± 331.95</td>
<td>95.40 ± 385.38</td>
</tr>
<tr>
<td>TAC ($nmol/l$)</td>
<td>919.77 ± 28.05</td>
<td>979.83 ± 30.61</td>
<td>60.06 ± 40.76</td>
</tr>
<tr>
<td>GSH ($μmol/l$)</td>
<td>763.95 ± 36.14</td>
<td>783.68 ± 45.76</td>
<td>19.73 ± 53.20</td>
</tr>
<tr>
<td>Uric acid (mg/dL)</td>
<td>5.5 ± 0.3</td>
<td>5.4 ± 0.2</td>
<td>-0.1 ± 0.3</td>
</tr>
</tbody>
</table>

$^a$ Obtained from repeated measures ANOVA.

$^b$ HOMA-IR: Homeostasis model of assessment-insulin resistance.

$^c$ FPG: fasting plasma glucose.

$^d$ LDL-cholesterol: low density lipoprotein-cholesterol.

$^e$ HDL-cholesterol: high density lipoprotein-cholesterol.

$^f$ hs-CRP: high sensitivity C-reactive protein.

$^g$ TAC: total antioxidant capacity.

$^h$ GSH: total Glutathione.
serum triglycerides, total- and LDL-cholesterol levels as well as increased concentrations of HDL-cholesterol in hypercholesterolemic pigs after 8 weeks. A significant reduction in serum total- and LDL-cholesterol levels was also seen with intake of a synbiotic containing Lactobacillus gasseri and inulin among hypercholesterolemic patients after 12 weeks. Despite these, some reports have reached conflicting findings. Different study designs, the dosage of synbiotic used and the patients under investigation as well as duration of supplementation can explain the different findings. The underlying mechanisms of the modulation of serum lipid profiles by synbiotics have remained largely obscure. It seems that probiotics can affect serum lipid profiles through their immune-modulatory effects. Their influence on TLR4 signaling and pro-inflammatory cytokines might also explain their lipid lowering effects. The effects on insulin sensitivity might be attributed to their impact on gene expression that results in decreased inflammatory activity and adiposity.

The current study showed that consumption of synbiotic food for 6 weeks among diabetic patients significantly decreased serum hs-CRP. In line with our findings, the beneficial effects of a synbiotic food containing Lactobacillus casei, Bifidobacterium breve and galacto-oligosaccharides on serum hs-CRP levels have been indicated in patients undergoing hepatobiliary resection. Further, consumption of a synbiotic food containing Bifidobacterium, Lactobacillus and galacto-oligosaccharides among hepatectomized patients with or without liver cirrhosis. Several mechanisms can explain the beneficial effects of synbiotic food on serum hs-CRP levels. Consumption of a synbiotic food might result in significant changes in gut microbiota. Cut fermentation of inulin increases short-chain fatty acid (SCFA) production, which can in turn lead to decreased expression of inflammation-relevant genes including IL-6, IL-8, cyclooxygenase-2, IL-1α, and TNF-α. The up-regulation of IL-1β protein expression has also been shown by SCFA products. This protein has been associated with a reduced enzymatic synthesis of hepatic CRP. Furthermore, increased production of methylketones family (methyl-5-hept-2-one, 2-propanone, 2-butanone, 2-pentanone, 2,3-butanedione) in gut following synbiotic intake might lead to anti-inflammatory effects.

We found that supplementation with a synbiotic food could significantly increase plasma total GSH and serum uric acid levels, but did not affect plasma TAC levels. In a study by Hutt et al., 3-weeks consumption of a synbiotic food containing Lactobacillus fermentum ME-3, Lactobacillus paracasei and Bifidobacterium longum with raftilose P95 among H. pylori-colonized asymptomatic subjects resulted in a significant increase in plasma TAC and glutathione levels. Decreased levels of lipid peroxidation, increased levels of superoxide dismutase and glutathione, along with reduced levels of nitric oxide were also seen by the use of a synbiotic containing L. acidophilus as a probiotic and inulin as a prebiotic in a murine model. Similar findings in biomarkers of oxidative stress have also been indicated after intake of synbiotics in neonatal rats. The beneficial effects of synbiotics on oxidative stress might be explained by their effect on SCFA production, in particular butyrate, in the gut. Butyrate leads to NADPH provision for synthesis of GSH, apoptosis induction and up-regulation of oxidative pentose pathway activity. Furthermore, the effect of synbiotics on pro-inflammatory cytokines as well as on down-regulation of genes involved in oxidative stress and TLR pathways might provide some reasons for their effect on circulating GSH levels. Lack of significant effect of synbiotic food on TAC in the current study might be attributed to the strains of probiotics used as well as the dosage of bacterial strain and inulin.

Some points need to be considered in the interpretation of our findings. First is limiting the duration of supplementation to 6 weeks. However, even in this short time we observed a significant effect of synbiotic food on metabolic status of diabetic patients. Long period of supplementation might lead to additional benefits. We considered hs-CRP as a surrogate marker of inflammation in the current study. However, other inflammatory markers might better represent the inflammation. Further studies are required to assess the effect of synbiotics on other inflammatory biomarkers. Due to the use of different bacteria strains in different studies, the cross-study comparisons are not easy. Additional studies must explore the best composition of a synbiotic food (in terms of both bacteria strains and the appropriate prebiotic) influencing metabolic conditions in diabetic patients.

In conclusion, consumption of a synbiotic food for 6 weeks among diabetic patients had significant effects on serum insulin, hs-CRP, uric acid and plasma total GSH levels, but could not affect plasma TAC concentrations.

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Conflicts of interest

None of the authors had any personal or financial conflict of interest.

Authors’ contributions

ZA and AE contributed in conception, design, statistical analysis and drafting of the manuscript. ZT, AK-R, S-AA and HS contributed in data collection and manuscript drafting. AE supervised the study. All authors approved the final version for submission.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.clnu.2013.05.015.

References


