The beneficial influences of xylo-oligosaccharides (XOS) obtained from alkali-pretreated corn cob and fructo-oligosaccharides (FOS) obtained from cane sugar were evaluated in experimental diabetes. These oligosaccharides were supplemented at 10% (w/w) in the basal diet of streptozotocin-induced diabetic Wistar rats, while the control rats were fed with a basal diet for a period of 6 weeks. Both the oligosaccharides exerted favourable influences in diabetic rats by significantly improving body weight and reducing hyperglycaemia and cholesterol. The characteristic diabetic complications such as severe glucosuria, proteinuria and advanced glycation end products in renal tissue, diabetic nephropathy, and blood creatinine and urea concentrations were notably reduced. Besides, these oligosaccharide supplementations significantly increased the activity of antioxidant enzymes – catalase and glutathione reductase – in the blood of diabetic rats. Supplementation of XOS and FOS resulted in a significant increase in the bifidobacteria and lactobacilli population in the caecum. The present study indicates that XOS and FOS have an ameliorating influence on metabolic abnormalities associated with diabetes, besides conferring an optimal milieu of lactobacilli and bifidobacteria, thus suggesting their potential health benefit in diabetics.

**Xylo-oligosaccharides: Fructo-oligosaccharides: Diabetes mellitus: Metabolic abnormalities: Nephropathy**

Diabetes mellitus is an endocrine disorder that affects over 100 million people worldwide, and is becoming very common with changing lifestyles. Diabetes often leads to disability from the vascular complications of coronary artery disease, cerebrovascular disease, renal failure, limb amputation and blindness, in addition to neurological complications and premature death(1,2). These diabetic complications rank high among the top ten causes of mortality throughout the world. The high fatty acid levels in plasma can lead to the development of atherosclerosis(3) in diabetic patients and a decrease in antioxidant fatty acid levels in plasma can lead to the development of athero...

Abbreviations: AGE, advanced glycation end; BW, body weight; GR, glutathione reductase; FOS, fructo-oligosaccharides; STZ, streptozotocin; XOS, xylo-oligosaccharides.

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polydipsia and elevation of serum glucose, TAG and cholesterol in diabetic rats\(^\text{16}\).  

In the above context, the present investigation evaluated the beneficial effects of two oligosaccharides – XOS and FOS – in STZ-induced diabetic rats with respect to glycaemic status, BW, hypercholesterolaemia, activities of antioxidant enzymes and also pathophysiological conditions such as advanced glycation end (AGE) products in renal tissue and kidney pathology. In the absence of any detailed information on these aspects, the present study could provide for a better understanding of the antidiabetic profile of these oligosaccharides.

**Experimental methods**

**Chemicals**

STZ was procured from ICN Biomedicals Inc. (Asse-Relegem, Belgium). Heparin, glucose oxidase, peroxidase, \(\alpha\)-diaminasidine, bovine serum albumin, Coomassie Brilliant Blue G-250, cysteine hydrochloride, thiosemiacrbazide and diacetyl monoxime were purchased from Sigma Chemical Co. (St Louis, MO, USA). Standards of FOS (1-kestose, 1-nystose and 1-fructofuranosyl nystose) were procured from Wako Pure Chemical Industries, Ltd (Osaka, Japan). Casein (refined grade) was procured from Nimesh Corporation (Mumbai, India). Salt mixture was procured from SISCO Research Laboratories Pvt. Ltd (Mumbai, India). All other chemicals and solvents used were of analytical grade.

**Production and analysis of oligosaccharides**

FOS (containing 90–93 \%(w/w) FOS and designated as FOS-90) consisting of 57 \% kestose, 30 \% nystose and 5 \% fructofuranosyl nystose were prepared by the selective removal of glucose and sucrose (our unpublished results) from FOS-56 (consisting of 56 \%(w/w) FOS), which were derived previously from cane sugar using fungal fructosyl transferase\(^\text{17}\). XOS (consisting of 90–92 \%(w/w) XOS) were prepared through a slight modification of the procedure used by Aachary & Prapulla\(^\text{18}\) under optimised conditions using commercial xylanase (Bioxyl P-40, Biocon Ltd, Bangalore, India). These two oligosaccharides were analysed by HPLC (LC-6A, Shimadzu, Kyoto, Japan) with a refractive index detector using a polar-bonded phase column (Exsil NH2, 4.6 mm x 25 cm, 5 \(\mu\)m) at an ambient temperature using acetonitrile–water (75:25) as a mobile phase at a flow rate of 1.0 ml/min. FOS were identified and quantified by comparing with standards of kestose, nystose and fructofuranosyl nystose\(^\text{17}\), while XOS were estimated according to Jeong \textit{et al.}\(^\text{19}\) and Aachary & Prapulla\(^\text{18}\). The refractive index detector used for the analysis of FOS and XOS has been standardised in our laboratory, and it was found to be satisfactory\(^\text{17,18}\).

**Animals and dietary treatment**

The present animal study was carried out by taking all appropriate precautions and by strictly following the guidelines with regard to the use of animals for experimental purpose after due approval from the Institutional Animal Ethics Committee (CFTRI, Mysore, India). Influence of prebiotics on diabetic rats was examined using male Wistar rats (150–160 g) procured from the animal production facility of this institute. Rats were divided into the following groups: (1) control group fed with the basal diet; (2) control group fed with the basal diet containing XOS (10 \%); (3) control group fed with the basal diet containing FOS (10 \%); (4) control group fed with the basal diet containing XOS (5 \%) + FOS (5 \%); (5) diabetic control group fed with the basal diet; (6) diabetic group fed with basal diet containing XOS (10 \%); (7) diabetic group fed with the basal diet containing FOS (10 \%); (8) diabetic group fed with the basal diet containing XOS (5 \%) + FOS (5 \%). The basal diet consisted (%) of maize starch, 54; casein, 21; refined peanut oil, 10; powdered cane sugar, 10; Bernhardt-Tommarelli salt mixture, 4; and National Research Council (NRC) vitamin mixture, 1. The oligosaccharide diets contained either XOS (10 \%) or FOS (10 \%), or a combination of XOS (5 \%) and FOS (5 \%) at the 10 \% level by replacing an equivalent amount of maize starch in the basal diet (w/w).

**Induction of diabetes**

Diabetes was induced by a single administration of STZ (intraperitonally 40 mg/kg BW in 1 ml of 0.1 M-citrate buffer, pH 4.5) to overnight fasted rats. A parallel set of control rats (non-diabetic) were injected with citrate buffer only. Glucose (5 \%) was given for 48 h following the intra-peritonal injection of STZ to prevent initial drug-induced hypoglycaemic mortality. Blood was drawn from the retro-orbital plexus 1 week after STZ administration, and it was used for the determination of fasting blood glucose. Animals having at least two and half times the normal fasting blood glucose were considered as hyperglycaemic.

The groups of rats were allowed to access the respective food and water \textit{ad libitum} for 6 weeks. BW was recorded weekly. Twenty-four-hour urine samples were collected at weekly intervals from individual rats housed in metabolic cages, filtered and stored at \(-20^{\circ}\mathrm{C}\) for further analysis of urinary protein and glucose excretion. Blood glucose was monitored biweekly, while urinary excretion of glucose and protein was monitored weekly.

At the end of the experiment, rats were killed, and blood was collected in heparinised tubes (20 U heparin/ml blood) and centrifuged at 4\(^{\circ}\mathrm{C}\) at 4000 rpm for 10 min (Remi centrifuge, Mumbai, India). The separated plasma was stored at \(-20^{\circ}\mathrm{C}\) until processed. The caecum samples were collected and weighed. An aliquot from caecal content was used for microbial analysis, and the remainder was stored at \(-20^{\circ}\mathrm{C}\) for further analysis. The kidneys were excised, weighed, homogenised in phosphate buffer using a Potter–Elvehjem homogeniser, and were then centrifuged at 8000 rpm at 4\(^{\circ}\mathrm{C}\) for 20 min. The supernatant was stored at \(-20^{\circ}\mathrm{C}\) for further analysis.

**Caecal characteristics**

After dissection, the rat caecum was immediately removed, and total net weight was recorded, and then it was stored at a refrigerated temperature for further analysis. Bifidobacteria and lactobacilli were enumerated from a known quantity of suitably diluted caecal matter using spread plate method in selective Bifidobacterium iodoacetate agar (anaerobically,
37°C for 48 h) and Lactobacillus MRS (de Mann Rogosa) agar (aerobically, 37°C for 24 h), respectively. Microbial counts were expressed as log colony-forming units/g wet sample. Aseptic conditions were maintained throughout the microbial enumeration. The pH of the caecal matter was also recorded (Analab Scientific Instrument Pvt. Ltd, Vadodara, India).

**Analytical parameters**

Fasting blood glucose and glucose excretion in urine were determined by the glucose oxidase and peroxidase method\(^{20}\). Total plasma protein and protein excretion in urine were analysed by the Bradford method\(^{21}\). The method described by Folin & Wu\(^{22}\) was used to estimate plasma and urinary creatinine. Plasma urea was estimated as described by Levine\(^{23}\), and urinary urea was measured as described by Wyebenga et al.\(^{24}\). Formation of AGE products in renal tissue was determined as reported by Monnier & Cerami\(^{25}\). Plasma total cholesterol was determined as described by Rudel & Morris\(^{26}\). Activity of catalase was assayed by the method of Takahara et al.\(^{27}\), and glutathione reductase (GR) activity was assayed using a standard procedure\(^{28}\). All the experiments were carried out in triplicate.

### Table 1. Effect of prebiotics on growth in diabetic rats (Mean values and standard deviations of eight rats)

<table>
<thead>
<tr>
<th>Animal group</th>
<th>Initial (0 d)</th>
<th>Final (6th week)</th>
<th>Weight gain (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (g)</td>
<td>SD</td>
<td>Mean (g)</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>161.1(^{a})</td>
<td>2.9</td>
<td>123.8(^{b})</td>
</tr>
<tr>
<td>Diabetic XOS</td>
<td>162.9(^{a})</td>
<td>6.9</td>
<td>142.5(^{b})</td>
</tr>
<tr>
<td>Diabetic FOS</td>
<td>162.4(^{a})</td>
<td>4.1</td>
<td>141.8(^{b})</td>
</tr>
<tr>
<td>Diabetic XOS + FOS</td>
<td>162.1(^{a})</td>
<td>8.6</td>
<td>142.6(^{b})</td>
</tr>
<tr>
<td>Normal control</td>
<td>168.9(^{a})</td>
<td>8.3</td>
<td>257.6(^{b})</td>
</tr>
<tr>
<td>Normal XOS</td>
<td>165.8(^{a})</td>
<td>9.5</td>
<td>265.7(^{c})</td>
</tr>
<tr>
<td>Normal FOS</td>
<td>164.0(^{a})</td>
<td>8.1</td>
<td>262.2(^{c})</td>
</tr>
<tr>
<td>Normal XOS + FOS</td>
<td>165.7(^{a})</td>
<td>7.4</td>
<td>263.5(^{c})</td>
</tr>
</tbody>
</table>

XOS, xylo-oligosaccharides; FOS, fructo-oligosaccharides.

\(^{a,b,c}\) Mean values within a column with unlike superscript letters were significantly different \((P<0.05)\).

### Table 2. Effect of prebiotics on total caecum weight, pH, bifidobacteria and lactobacilli population of the caecum contents (Mean values and standard deviations of eight rats)

<table>
<thead>
<tr>
<th>Animal group</th>
<th>Total caecum weight (g)</th>
<th>pH</th>
<th>Bifidobacteria*</th>
<th>Lactobacilli*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (g)</td>
<td>SD</td>
<td>Mean (g)</td>
<td>SD</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>2.05(^{a})</td>
<td>0.12</td>
<td>6.73(^{d})</td>
<td>0.16</td>
</tr>
<tr>
<td>Diabetic XOS</td>
<td>2.55(^{b})</td>
<td>0.11</td>
<td>6.49(^{b})</td>
<td>0.15</td>
</tr>
<tr>
<td>Diabetic FOS</td>
<td>2.41(^{b})</td>
<td>0.23</td>
<td>6.55(^{b})</td>
<td>0.19</td>
</tr>
<tr>
<td>Diabetic XOS + FOS</td>
<td>2.43(^{b})</td>
<td>0.16</td>
<td>6.52(^{b})</td>
<td>0.21</td>
</tr>
<tr>
<td>Normal control</td>
<td>2.92(^{b})</td>
<td>0.41</td>
<td>6.65(^{c})</td>
<td>0.11</td>
</tr>
<tr>
<td>Normal XOS</td>
<td>4.05(^{a})</td>
<td>0.21</td>
<td>6.35(^{e})</td>
<td>0.08</td>
</tr>
<tr>
<td>Normal FOS</td>
<td>3.61(^{c})</td>
<td>0.28</td>
<td>6.42(^{c})</td>
<td>0.14</td>
</tr>
<tr>
<td>Normal XOS + FOS</td>
<td>3.93(^{c})</td>
<td>0.33</td>
<td>6.38(^{d})</td>
<td>0.10</td>
</tr>
</tbody>
</table>

XOS, xylo-oligosaccharides; FOS, fructo-oligosaccharides.

\(^{a,b,c,d,e}\) Mean values within a column with unlike superscript letters were significantly different \((P<0.05)\).

\(^{a}\) Log\(_{10}\) colony-forming units/g caecal wet content.

**Histological studies**

Light microscopic observations were made with haematoxylin–eosin-stained thin sections of kidney previously fixed in 10% formalin and embedded in paraffin.

**Statistical analysis**

Values were expressed as means and standard deviations of eight rats. Statistical analysis was carried out using Origin 6.1 statistical software (Originlab Corporation, Northampton, MA, USA). Results were analysed and the significance level was calculated using the Tukey–Kramer multiple comparison test, and results are considered significant at \(P<0.05\).

**Results**

**Effect of xylo-oligosaccharides and fructo-oligosaccharides on body weight and mortality**

The influence of prebiotic supplementation on the BW of diabetic rats and normal rats is presented in Table 1. Gain in BW was markedly suppressed in diabetic rats, whereas the diabetic rats fed with XOS and FOS diets showed a significant improvement in the BW during the feeding
period in comparison with those fed with the basal diet. The BW of the control rats fed with oligosaccharide diets was not significantly increased compared with those fed with the basal diet. High mortality was generally observed in diabetic rats (as much as 50%) fed with the basal diet, but oligosaccharide supplementation reduced the mortality by up to 8, 14 and 21% in the case of FOS (10%), XOS (10%) and combination of XOS (5%) and FOS (5%) in diabetic rats, respectively (data not presented). Interestingly, the mortality was less in the case of FOS-fed diabetic rats compared with other diabetic groups.

**Effect of prebiotics on caecal characteristics**

Data on caecal wet weight, pH, bifidobacteria and lactobacilli count are presented in Table 2. Total wet weight of the caecum was significantly \( (P \leq 0.05) \) increased in diabetic and non-diabetic rats consuming XOS and FOS than in the rats fed with the basal diet. In addition, rats fed with oligosaccharides showed a significant decrease in pH from 6.73 (SD 1.6) up to 6.35 (SD 0.08) and an increase in the bifidobacteria and lactobacilli count in the caecum compared with rats fed with the basal diet. Rats fed with the XOS-containing diet, either 10% or 5%, showed a significant increase in bifidobacteria compared with rats fed with FOS (10%). The rats fed with the FOS-containing diet, either 10% or 5%, showed a significant concentration of lactobacilli compared to rats fed with XOS. Thus, the result explains the prebiotic efficacy of XOS and FOS.

**Effect of xylo-oligosaccharides and fructo-oligosaccharides on fasting glucose, cholesterol, creatinine and urea in plasma**

Hyperglycaemia was ameliorated throughout the experimental period in the case of diabetic rats fed with the XOS or FOS diet. Treatment of diabetic rats with prebiotics at the 10% dietary level for a period of 6 weeks brought down hyperglycaemia significantly \( (P \leq 0.05; \text{Fig. 1}) \), and a similar trend was observed in the urinary glucose excretion pattern in oligosaccharide-fed diabetic rats (Fig. 2). In addition, oligosaccharide diets significantly \( (P \leq 0.05) \) reduced the plasma cholesterol, creatinine and urea concentration in diabetic rats in comparison with diabetic rats fed with basal diet. Effects on plasma cholesterol, creatinine and urea are presented in Table 3.

**Effect of xylo-oligosaccharides and fructo-oligosaccharides on plasma protein, kidney weight and advanced glycation end products**

Table 3 shows the effect of XOS and FOS on plasma protein and kidney weight in rats. Diabetic rats fed with the basal diet showed a high degree of plasma protein depletion (59.0 (SD 2.9) mg/l) compared with control rats (65.6 (SD 1.1) mg/l). In diabetic rats fed with oligosaccharides, the plasma protein destruction was reduced up to 3–4% compared with diabetic control rats. The urinary protein excretion pattern substantiates the above finding (Fig. 3). Countering of proteinuria by dietary oligosaccharides in diabetic rats was progressive with the duration of the diet regimen, and it was maximum in the FOS group. As is typical in diabetes, the increase in the kidney weight in diabetic rats was markedly high compared with the control rats. A reduction in kidney weight was observed in the case of diabetic rats fed with oligosaccharides compared with diabetic control rats. The increased kidney weight corresponds to the presence of diabetic nephropathy such as heavy AGE product formation and subsequent damage in renal tissues. XOS and FOS diets also decreased the level of AGE products in renal tissue of diabetic rats compared with those fed with the basal diet. The relative intensity of 15.3 for diabetic controls corresponds to an increased number of AGE end-products, which was found to be significantly reduced to a relative intensity of 12.83, 13.20 and 13.14 at 440 nm in the diabetic rats fed with XOS, FOS and a combination of XOS + FOS, respectively. The control rats fed with prebiotics showed a relative intensity in the range of 10.8–10.9, which
was comparable to that shown by normal control rats fed with the basal diet.

Histopathology of kidney sections

Histological examination of the kidney sections revealed pronounced glomerulosclerosis and tubular lesions and cellular infiltration in all diabetic rats maintained on the basal diet. The diabetic rats fed with XOS and FOS showed a decreased degree of renal pathology compared with diabetic rats fed with the basal diet (Fig. 4).

Effect of xylo-oligosaccharides and fructo-oligosaccharides on the activities of plasma catalase and glutathione reductase

Results showed that the activity of antioxidant enzymes, with catalase and GR, was lowered in diabetic rats compared with normal control rats, but diabetic rats fed with oligosaccharides showed improved ($P \leq 0.05$) activities of catalase and GR compared with diabetic control rats fed with the basal diet (Table 4). Such an increase, however, was not observed in normal rats fed with XOS or FOS.

Discussion

In the present study, consumption of oligosaccharides FOS and XOS showed desirable effects in STZ-induced diabetic rats. Oligosaccharide diets markedly improved the BW in diabetic rats, reduced the mortality and significantly increased the bifidobacteria and lactobacilli population in the caecum. The tested oligosaccharides at 10% dietary concentration beneficially countered fasting hyperglycaemia associated with diabetes, improved plasma albumin and significantly attenuated the rise in plasma concentrations of cholesterol, creatinine and urea. In addition, diabetes-induced reduction in the activity of antioxidant enzymes catalase and GR in plasma was ameliorated. Attenuation of diabetic nephromegaly by dietary oligosaccharides was accompanied by a reduction in AGE products in the renal tissue. Dietary levels of XOS and FOS (10%) used in the present study conform to the levels used in our previous report on the beneficial effect of FOS. Health beneficial hypocholesterolaemic and antidiabetic influences of various dietary fibres included up to 10% in the diet in animal studies have been reported by various authors without any adverse effects. Thus, we used the 10% level of oligosaccharides either individually or in combination.

While dietary XOS and FOS resulted in a similar influence on various parameters studied, a striking difference between them was their effect on microbial flora. While FOS stimulated the growth of lactobacilli, XOS stimulated the growth of bifidobacteria. Several in vitro and in vivo studies have demonstrated that diets containing oligosaccharides such as FOS and XOS selectively increase the bifidobacteria and lactobacilli population, and decrease the pH of caecal content. In the present study, their dietary supplementation at the 10% level significantly increased the caecum total weight, bifidobacteria and lactobacilli population, and decreased the pH of the caecal content. The reduction in the pH of caecal content after the supplementation of prebiotics could be attributed to an increase in the SCFA concentration.
by fermentation of bifidobacteria and lactobacilli. Among the two oligosaccharides, XOS was found to be more bifidogenic, which is in agreement with earlier reports of XOS (1 g/d) producing a selective increase in bifidobacteria\(^{30–32}\). The growth of lactobacilli was enhanced by FOS as observed previously by Campbell et al.\(^{133}\). These beneficial microflora which can utilise XOS and FOS produce SCFA such as lactate, acetate, propionate and butyrate, which in turn influence the glucose and lipid metabolism\(^{33,34}\).

The present study has shown that with the administration of XOS or FOS for a period of 6 weeks, the plasma glucose level was significantly decreased in the diabetic rats. The reduction in high glucose level was observed in diabetic rats from the second week of prebiotic supplementation, and the reduction was found to be maximum in FOS-fed diabetic rats at 6 weeks. It has been reported earlier that the intake of FOS (showing reduced renal pathology).

Table 4. Effect of prebiotics on plasma catalase and glutathione reductase activities in diabetic rats*

<table>
<thead>
<tr>
<th>Groups</th>
<th>Mean</th>
<th>sd</th>
<th>Mean</th>
<th>sd</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Catalase</td>
<td></td>
<td>Glutathione reductase</td>
<td></td>
</tr>
<tr>
<td>Diabetic control</td>
<td>3287(^{a})</td>
<td>109</td>
<td>2524(^{a})</td>
<td>13</td>
</tr>
<tr>
<td>Diabetic XOS</td>
<td>3498(^{b})</td>
<td>88</td>
<td>2606(^{b})</td>
<td>29</td>
</tr>
<tr>
<td>Diabetic FOS</td>
<td>3549(^{a})</td>
<td>75</td>
<td>2646(^{a})</td>
<td>31</td>
</tr>
<tr>
<td>Diabetic XOS + FOS</td>
<td>3508(^{b})</td>
<td>98</td>
<td>2621(^{b})</td>
<td>22</td>
</tr>
<tr>
<td>Normal control</td>
<td>4221(^{a})</td>
<td>91</td>
<td>3028(^{a})</td>
<td>97</td>
</tr>
<tr>
<td>Normal XOS</td>
<td>4319(^{b})</td>
<td>79</td>
<td>3109(^{b})</td>
<td>26</td>
</tr>
<tr>
<td>Normal FOS</td>
<td>4373(^{a})</td>
<td>81</td>
<td>3097(^{a})</td>
<td>31</td>
</tr>
<tr>
<td>Normal XOS + FOS</td>
<td>4331(^{b})</td>
<td>106</td>
<td>3152(^{b})</td>
<td>36</td>
</tr>
</tbody>
</table>

\(\text{XOS, xyl-o-oligosaccharides; FOS, fructo-oligosaccharides.}\)

\(^{a,b}\) Mean values within a column with unlike superscript letters were significantly different \((P<0.05)\).

\(^{*}\) All values are expressed as units/l.
An abnormally elevated blood glucose level causes oxidative stress and leads to the formation of AGE products. Dietary oligosaccharides reduced the formation of AGE products in the renal tissue of diabetic rats compared with those fed with the basal diet, suggesting that it would inhibit oxidative damage caused by the protein glycation reaction under diabetic conditions. These results showed that the administration of prebiotics at the 10% dietary level might effectively alleviate the pathogenesis of diabetic complications caused by impaired glucose metabolism and the glycosylation of tissue proteins, eventually resulting in an alleviation of the diabetic pathological conditions. The increased weight of kidney in diabetic rats was also countered in oligosaccharide-fed diabetic animals. Administration of these test materials for 6 weeks reduced protein depletion, nephromegaly and glycation of renal tissue proteins. Light microscopy of kidney sections revealed nephromegaly and damaged glomeruli and basement membrane in diabetic rats. The magnitude of these changes was less in FOS- or XOS-fed diabetic rats. Presumably, this beneficial ameliorating influence of dietary oligosaccharides on diabetic nephropathy is attributable to their lowering effects on blood cholesterol levels.

In the present study, the activity of catalase in the blood of diabetic rats fed with FOS or XOS was significantly (P ≤ 0.05) higher compared with diabetic rats fed with the basal diet. In contrast, supplementation of XOS (4 g/d) has been reported to reduce the activity of catalase in erythrocytes but not the activity of superoxide dismutase and glutathione peroxidase in type 2 diabetes. GR is another important oxidative defence enzyme, which converts the GSSG to GSH, an antioxidant molecule. Reduction of GSSG to GSH was found to be decreased in diabetic rats compared with diabetic rats fed with prebiotic diets. Prebiotic-mediated protection of plasma protein could be implied in improved activity of catalase and GR. The increase in GR activity in blood in turn neutralises superoxide anions and counteracts oxidative stress in diabetes.

Conclusions
The present investigation indicates that the supplementation of oligosaccharides (XOS and FOS) at the 10% dietary level confers beneficial effects in STZ-induced diabetic rats with respect to plasma glucose, cholesterol and several other metabolic parameters. The reduced hyperglycaemia and hypercholesterolaemia by oligosaccharides may have in turn contributed to a decrease in the AGE products in the renal tissue and lowered nephromegaly in diabetic rats. The present study indicates that XOS and FOS at the 10% dietary level can be used as an adjunct to dietary therapy to derive antidiabetic benefits, and to delay secondary complications. A detailed study on the mechanisms of these beneficial effects exerted by these oligosaccharides would be challenging and merits further investigation.

Acknowledgements
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References


