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Comparative Studies of Non-digestible Polysaccharides: Wheat and Potato Resistant Starch and Pectin on Glycemic, Lipemic, Blood Urea and Intestinal Parameters in Growing Rats

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The influence of non-digestible polysaccharides on gastrointestinal parameters, serum and liver lipids, serum urea and blood glucose parameters were evaluated in growing rats during a 6 weeks experimental period. The polysaccharides consisted of either plain wheat starch (Digestible Starch, DS) control diet, or starch substituted with 12% of either Wheat Resistant Starch (WRS), Potato Resistant Starch (PRS) or Pectin (P). Body weight gain and food intake were not significantly affected by the experimental diets. The length of the small intestine was also not affected by the different diets. The caecal weight was significantly increased ($p<0.001$) in animals fed the non-digestible polysacchrides, with P and PRS showing the highest increase. There was a parallel increase in the caecal content. ($p<0.01$) The caecal material pH tended to be acidic in the groups fed non-digestible polysaccharides. The intestinal maltase activity was significantly lower ($p<0.01$) in animals fed non-digestible polysaccharides. The PRS and P groups showed significantly lower serum cholesterol concentrations ($p<0.01$) compared with the control and WRS groups. Serum triacylglycerol, liver cholesterol and triacylglycerol were not significantly affected following feeding non-digestible polysaccharides. Feeding non-digestible polysaccharides lowered the total serum lipids ($p<0.05$) without affecting the total liver lipids. The serum urea concentrations were significantly lowered by feeding WRS and PRS ($p<0.05$) The fasting blood glucose was significantly reduced in WRS and P fed groups compared with the control and PRS groups ($p<0.01$). The P fed group showed a significantly lower 1 h postprandial blood glucose ($p<0.05$). The 6 h post feeding blood glucose concentrations were significantly lowered in WRS and P fed groups compared with the control and PRS groups ($p<0.001$). The present study suggests that non-viscous resistant starch; shares some of the physiological properties of non-starch polysaccharides and have similar metabolic effects, which may be of importance in the dietary control of certain disease conditions.

Key words: Resistant starch, pectin, lipid metabolism, blood glucose, blood urea, gastrointestinal tract

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INTRODUCTION

As a new category of food ingredients, resistant starches are being evaluated for both their therapeutical implications and functional properties in food ingredients. Resistant starch has been defined as the sum of starch and products of starch degradation not absorbed in the small intestine of healthy individuals[1,2]. Three types of resistant starch have been identified[3]. RS 1 includes physically inaccessible starch, such as in seeds and legumes; RS 2 occurs in granules, non-gelatinised sources, found in banana and native potato; RS 3 is formed upon retrogradation after gelatinisation and is found in breakfast cereals, cooked and cooled potato and bread. Thus it is generated during food processing.

Resistant starch has many similarities with non-starch polysaccharides dietary fibre, which are plant polysaccharides that are not hydrolysed in the small intestine. Both resistant starch and dietary fibre are fermented by colonic microflora to products including gases and short chain fatty acids[4,5]. The type of by-product produced is largely dictated by the substrate undergoing fermentation. Beneficiary effects of indigestible carbohydrates include increased faecal bulk, lowering of colonic pH, increased short chain fatty acids, reduced intestinal transit and lower the concentrations of potentially damaging bile acids, ammonia and phenols[6-8]. The short chain fatty acids produced in the colon following fermentation of indigestible carbohydrates includes acetate, propionate and butyrate and is associated with lowering of colonic pH. Compared to dietary fibre, resistant starch produce higher proportion of butyrate which is consistent with colonic health. Butyrate is considered to be responsible for regulation of intestinal cell function and growth, suppressing tumour cells and decreasing the colonic mucosal cells proliferation. Butyrate has been demonstrated to be a potent inducer of apoptosis *in vitro*.[9] This is achieved by the induction of expression of a protein, which activates the proteases responsible for the nuclear destruction characteristics of apoptosis.

Resistant starch has also been implicated as a probiotic (synbiotics) which is defined as non-digestible food ingredients that beneficially affects the host by selecting stimulating the growth and activity of one or a limited number of bacteria in the colon thereby improving host health[10]. Propionate on the other hand is mainly discussed in relation to effects on carbohydrate and cholesterol metabolism. Propionate is absorbed and metabolised in the liver. In rats, dietary supplementation with propionate appears to lower serum cholesterol[11-13]. Class 2 resistant starch was shown to lower plasma and liver lipids in rats[14]. Resistant starch from corn and rice modified glucose tolerance, colonic events and blood lipid concentrations in diabetic rats[15] Also, in humans, dietary supplementation with propionate improves glucose metabolism[16-17]. However raw and retrograded starch failed to lower fasting cholesterol concentrations in normolipidemic subjects[18].

Similarly, non-starch polysaccharides depending on the type and source may affect aspects of carbohydrate and lipid metabolism. Soluble viscous fibres such as guar gum and pectin have been shown to reduce and delay the magnitude of glycemic response[19,20]. The action was ascribed to delayed gastric emptying and intestinal absorption[21,22]. The hypolipidemic effect of non-starch polysaccharides is frequently associated with depressed plasma cholesterol and triacylgcerol concentration resulting from enhanced excretion of bile acids[23-25]. As with resistant starch, non-starch polysaccharides fermentation product, propionate mediates its action through altered hepatic metabolism[26-28]. The non-digestible polysaccharides have also been associated with changes in the structures of the small intestine. These changes include alterations in length and weight[29-30], modifications in the epithelial structure and proliferative activity[4,31-33].

Similarly, diets rich in fermentable polysaccharides have been shown to have impact on urea nitrogen disposal and faecal Nitrogen execration[34]. These effects have also been ascribed to cecal hypertrophy and this depends on their fermentability. Enlargement of the cecum provides greater surface of exchange and with elevated blood flow, fermentable polysaccharides promote a higher flux of urea towards the cecum and, concomitantly lower the ammonia in the digestive content. An elevated bacterial protein synthesis may result in a elevated faecal excretion of nitrogen, at the expense of renal excretion and this is of interest in chronic renal disease management. In contrast to soluble viscous polysaccharides resistant starch are well tolerated when administered in substantial quantities and it is therefore possible to obtain high cecal fermentation rate. It is also easy to examine those characteristics that predict physiologic actions since their structure and properties are well defined. The ability of non-digestible polysaccharides to alter aspects of carbohydrate and lipid metabolism and gastrointestinal events largely depends on their viscosity and fermentability and this is dictated by its nature and source. The objective of this study was to test the metabolic effects of two sources of non-viscous resistant starch from wheat and potato to pectin, a classical non-starch viscous polysaccharide often studied for its metabolic effects. Resistant starch type R 3 obtained

through autoclaving-cooling circle was employed in this study.

MATERIALS AND METHODS

Animals: A total of twenty-eight growing Wister strain male rats weighing between 40-50 g at the start of experiment, obtained from the animal house, Biochemistry department, University of Maiduguri, Nigeria were used for the study. They were randomly assigned into four groups of seven animals each. They were housed in an air-conditioned controlled (18°C) with 12 h light and dark cycles and allowed free access to water and formulated diets according to their test groups. Daily food consumption and weekly changes in body weights were monitored during the experimental period.

Preparation of resistant starch: Resistant starch was prepared by the method of Ranhotra *et al.*[35] wheat and potato flours were washed with distilled water to recover native starch. The starch was centrifuged to remove starch tailings and air-dried. Starch was then mixed with distilled water (20% starch suspension w/w), autoclaved (126°C, 15 psi) for 1 h for five successive days, dried and then finely ground. After each 1 h autoclaving step, samples were stored for 24 h at 4°C. Prolonged cooling after autoclaving is a prerequisite for resistance starch formation. Resistant starch was determined by the method employed by Berry[36]. Diet were prepared weekly and stored at 4°C.

Feeding and experimental design: The experimental animals were fed a formulated diet (table 1) containing identical nutrient composition except that the control diet contained digestible wheat starch (740 g kg^{-1}) and the test diets consist of 620 g kg^{-1} digestible starch plus 120 g kg^{-1} wheat resistant starch, potato resistant starch or pectin for six weeks.

Sampling and analytical procedures

Biochemical parameters: Towards the close of the feeding period animals were fasted for 18 h and then challenged with glucose at 2 g kg^{-1} body weight which was administered gastrically and tail blood sample collected after 1hr for postprandial blood glucose determination. On other occasions animals were fasted for 18 h and then gastrically administered (2 g kg^{-1}) their respective experimental diets. Blood samples were then collected 6hr postprandial for blood glucose determination. Blood glucose was determined by the glucose oxidase- peroxidase method[37]. On the last day of the feeding trial the animals were killed by decapitation.

Table 1: Composition of the experimental diets (g/kg)

Diet	DS	WRS	PRS	PEC
Digestible wheat starch[a]	740	620	620	620
Casein[a]	150	150	150	150
Soybean oil	50	50	50	50
Mineral mixture[b]	50	50	50	50
Vitamin mixture[b]	10	10	10	10
Wheat resistant starch	-	120	-	-
Potato resistant starch	-	-	120	-
Pectin[c]	-	-	-	120

DS= Digestible starch ,WRS=Wheat resistant starch PRS= Potato resistant starch; PEC= Pectin, a-Sigma, b-Pfizer product, c-BDH chemicals

Blood and liver were collected for subsequent analysis. One-gram liver was homogenized with chloroform-methanol mixture and processed for total lipids. Cholesterol in serum and tissue homogenates was assayed calorimetrically by enzymic method using cholesterol reagent kit, Randox lab. Ltd[38]. Triacyglycerol was estimated by enzymatic method[39]. Total lipids was assayed by the sulphovanillin reaction of chabrol[40] and serum urea by diacetylmonoxime procedure[41].

Intestinal parameters: After decapitation, the small intestine from the pylorus to the terminal ileum was removed. The length of the intestine was measured by suspending a 10 g weight attached[42] The cecal contents removed for determination of the wet weight per body weight ratio and the cecal tissue was rinsed, blotted dry and weighed. The pH of the caecal digesta were determined after diluting with 5 mL water[43] The small intestine was flushed with cold pH 8.0 phosphate buffered saline and split longitudinally, the mucosa then scraped with a glass slide. The Mucosal scrap was homogenised with 5 mL of phosphate buffer and maltase activity assayed according to the procedure of Dahlqvist[44].

Statistical analysis: Test of significance of differences between treatment means were carried out by the analysis of variance. Test of significance of differences between individual means were carried out by the Duncan Multiple Range Test when the F-test was significant. Results are given as means with their Standard Error of Difference (SED).

RESULTS

The final body weight gain did not differ significantly following the different treatments after six weeks period (Table 2). Feed intake were also not affected by the diets. The small intestine length of the test groups appeared to be higher although not significant. The wheat resistant starch, potato resistant starch and pectin fed group showed 19, 54 and 61% higher cecal weight compared

Table 2: Intestinal parameters following feeding of experimental diets to rats for six weeks (n=7)

Parameters	DS	WRS	PRS	PEC	SED
Body weight gain (g/d)	0.86	0.83	0.73	0.77	0.052
Feed intake (g/d)	7.28	7.18	7.12	7.4	0.180
Intestinal length (cm)	98.83	91.53	93.00	95.00	3.420
Cecal weight (g/100 g body weight)	0.43a	0.51b	0.66c	0.69	0.040
Cecal materal weight (g/100 g body weight)	0.91	0.99a	1.39ab	1.96	0.100
Cecal pH	5.97	5.61	5.66	5.00	0.020
Intestinal maltase activity*	6.55a	3.30	4.04	3.30b	0.640

Results are presented, as means with SED. Means with different superscript letters along the same row are significantly different ($p<0.05$).
*mmol L^{-1} glucose/h g/protein

Table 3: Serum and liver cholesterol, total lipids and triacylglycerols and serum urea concentrations following feeding of experimental diets for six weeks (n=7)

Parameters	DS	WRS	PRS	PEC	SED
Serum cholesterol (mmol L^{-1})	2.85a	2.79a	2.14b	2.38b	0.21
Liver cholesterol (mmol g Wet. Wt^{-1})	0.70	0.89	0.67	0.81	0.09
Serum tricacyl glucerol (mmol L^{-1})	1.99	1.98	1.64	1.51	0.32
Liver tricacyl glucerol (mmol g Wet. Wt^{-1})	1.77	1.68	1.62	1.51	0.19
Serum total lipids (mg 100 mL^{-1})	435.30a	360.20a	364.30b	333.90b	45.90
Liver total lipids (g kg Wet $Wt.^{-1}$)	138.80	105.00	136.70	136.00	11.60
Serum urea (mmol L^{-1})	6.97a	5.24b	5.54b	6.40ab	0.40

Results are presented as means with SED. Means with different superscript letter in the same row are significantly different ($p<0.05$).

Table 4: Fasting blood glucose, 1 h post parandial and 6 h post feeding blood glucose concentrations following feeding experimental diets for six weeks (n=7)

Parameters	DS	WRS	PRS	PEC	SED
Fasting blood glucose (mmol L^{-1})	3.86a	2.27b	3.99a	2.44b	0.49
1 h post parandial blood glucose (mmol L^{-1})	5.90	5.63	5.65	5.00	0.19
6 h post Feeding blood glucose (mmol L^{-1})	6.15a	4.84b	6.07a	4.94b	0.67

Results are presented as means with SED. Means with different superscript letter in the same horizontal row are significantly different ($p<0.05$)

with the control group, respectively ($p<0.001$). Similarly the cecal weights were significantly higher by 8, 51 and 113% following wheat resistant starch, potato resistant starch and pectin feeding ($p<0.001$) (Table 2). The cecal material pH appeared to be more acidic in the groups fed the non-digestible polysaccharides (Table 2). The intestinal maltase activity were significantly reduced by the non-digestible polysaccharides ($p<0.001$) with wheat resistant starch and pectin exhibiting comparable response.

The total serum lipids were significant reduced in the non-digestible polysaccharide fed groups compared with the control ($p<0.001$) (Table 3). However, no significant differences were observed between the test groups. Similarly the serum cholesterol concentrations were significantly reduced in the non-digestible polysaccharide fed groups ($p<0.001$) (Table 3). However, the concentrations of liver cholestrol, triacylglycerol and total lipids were not significantly affected by the different experimental diets. The serum urea concentrations were also significantly lowered in the test groups, with wheat resistant starch exhibiting lowest response. The blood glucose response parameters are presented in Table 4. The pectin and wheat resistant starch fed groups showed significant lower fasting blood glucose concentration compared with the control and potato resistant starch fed group at the end of the study period ($p<0.01$). Pectin fed group produced a lower 1 h postprandial blood glucose concentration compared with the other experimental groups ($p<0.001$). The wheat resistant starch and pectin fed groups exhibited lower 6 h post feeding blood glucose concentrations compared with the control group ($p<0.001$).

DISCUSSION

The fraction of starch that is rendered resistant to mammalian digestive enzymes through processing is termed resistant starch and specifically classed as type III. The contents and characteristics of enzyme resistant starch not only depend on the type of starch but also affected by the process conditions and the presence of other components. From the repeated five cycles of autoclaving and cooling 12 and 11.8% resistant starch were isolated from potato and wheat flours. Retrogradation of starch after gelatinisation is believed to lead to the formation of resistant starch[36]. Miles *et al.*[45] proposed a concept of starch retrogradation encompassing two processes, a short development of the gel structure, which is governed by the crystallization of amylose and a long-term process that is due to retrogradation of amylopectin. The decrease in enzyme susceptibility could be ascribed to an increase in entanglement of the molecules and the molecular order by forming double helices of the outer chains of amylopectin and by organization of these helices in a three dimensional crystalline structure. The close contents of resistant starch isolated from the two sources are expected. The two sources share important determinant properties in resistant starch formation. Wheat has a gelatinisation temperature ranged 58-64°C and potato

59-68°C. The amylose content of wheat is 23-27% and potato starch 23%.

In the present investigation we used growing rats to get the desirable effect of growing rate on functions of gastrointestinal tract and it metabolic effects. There appeared to be no difference in the body weight gain or feed intake. Although it is expected that the digestibility of the diets investigated to vary according to the type of indigestible polysaccharide fed[46], the body weight results would suggest that the energy supply was not very different. Previous study indicated feeding high levels of corn resistant starch suppressed body weight with minimal change in the feed intake[47].

The small intestinal lengths were not affected by the experimental diets. Diet related changes in epithelial structure and cellular proliferation were reported[30] and depended on the site along the intestinal tract. The resistant starch diet, which is non-viscous, elicited changes that are similar to guar gum and pectin. It has been suggested that the ability of different fibres to promote mucosal growth depends on their fermentability and on their viscosity[48]. It has also been suggested that workload of absorption is the major stimulant of tissue proliferation[49]. Other workers failed to demonstrate changes in small intestinal length following feeding diets containing 5% viscous fibre derivatives after four weeks[50]. Recently Henningsson *et al.*[33] demonstrated the influences of dietary adaptation and sources of resistant on short chain fatty acids production. It would appear that these changes are time related.

Feeding indigestible polysaccharides produced changes in the cecal weight. The viscous polysaccharide pectin group showed greater changes than the non-viscous resistant starch groups. Younes *et al.*[23] reported hypertrophy of the cecum following feeding of resistant starch to rats. Earlier studies by Sourthon *et al.*[42] showed that feeding guar gum, another non-digestible viscous polysaccharide to rats increased the cecal weight. Cecal and colonic proliferative activities were reported in rats fed pectin and resistant starch[32]. Cellular proliferations were more pronounced in the cecum and less in the proximal colon and least in the distal colon. These responses are related to the fact that in rats major part of fermentation takes place in the cecum with relatively rapid transit through the mid and distal colon. The main product of indigestible polysaccharide breakdown, the short chain fatty acids are thought to be the stimulator of epithelial growth[51]. Evidence has been presented that the micro flora of the cecum and colon must adapt before fermentation proceeds most efficiently and short chain fatty acids generated in large amounts. Short chain fatty acids have been demonstrated to stimulate epithelial cell proliferation in a dose response related fashion[52]. Although, we did not examine the concentrations of short chain fatty acids in the present investigation but its makers, pH, degree of cecal enlargement and contents were determined. Indeed close relationships were observed between these parameters. Among the short chain fatty acids produced in the colon, butyrate is considered to play a role in regulating intestinal cell function and growth. Butyrate is the preferred energy source of the colonocytes[53]. Butyrate has been demonstrated to be potent inducers of apoptosis *in vitro*[9]. It activates proteases, which is responsible for the nuclear destruction. Further more, resistant starch has been implicated as a prebiotic[10].

The increase in cecal weight in rats fed non-digestible polysaccharides showed parallel increase in cecal material weight. The large quantities of materials escaping small intestinal digestion are delivered into the cecum and thereby leading to adaptive hyper tropic response[54]. It was also found that animals fed the non-digestible polysaccharide had more acidic pH. The change in pH may reflect increase in metabolic activity of the colonic micro flora producing short chain fatty acids. A close correlation was observed between the concentration of short chain fatty acids and cecal pH[51]. However, lactic and succinic acids may also contribute in regulation of lumen pH but its effect on gut functions may be different from that of short chain fatty acids[55].

Rats adapted to potato resistant starch and to pectin showed significantly lower serum cholesterol and total lipids. The results also showed general reductions in serum and liver triacylglycerols in all test groups. These results are consistent with some animals' studies. Morand *et al.*[28] observed decrease in plasma cholesterol, triglycerides and free fatty acids in rats adapted to resistant cornstarch diet during post absorptive periods. Similarly, Younnes *et al.*[6] showed resistant starch from potato depressed lipo-protein fraction triacylglycerol. There was a marked rise in cecal size and short fatty acids pool. Fatty acid synthase was also depressed. Trautewin *et al.*[24] also reported lowering of triacylglycerol by resistant starch with decrease in taurochenodeoxy cholate concentration and increase in faecal bile acid excretion. In another study, Levrat *et al.*[56] reported reduction in plasma cholesterol and triglycerides by guar gum and resistant starch in rats adapted to 0.4% cholesterol diets. Liver cholesterol was also depressed in parallel with acylcoA: cholesterol acyltransferase. Soluble non-digestible polysaccharides have been shown to lower serum cholesterol independently of alterations in fat intake[57]. Soluble non-digestible polysaccharides may reduce absorption of cholestrol or fatty acids from the gut either as a result of decreased diffusion due to the

viscous nature of soluble fibres or as a result of decreased micelle formation due to binding of bile acids and or lipids to the fibre[58]. Propionate and acetate products of colonic fermentation of soluble fibres, have been implicated in their hypocholesterolemic properties[59]. Propionate inhibits cholesterol synthesis *in vitro*[26]. It has also been suggested that insulin may have a role in cholesterol regulation. Insulin increases cholesterol synthesis[60]. Soluble fibres on the other hand reduce postprandial hyperglycaemia and are associated with insulin elevation, which may reduce cholesterol synthesis. Similarly, it is expected that peripheral uptake of triglycerides to proceed in animals fed pure digestible starch in accordance with higher insulinemia. However, some human studies failed to demonstrate the effects of resistant starch on parameters of lipid metabolism[18,61]. It is believed that the pattern and efficiency of large bowel fermentation is determined by the substrate and the micro flora.

Feeding the non-digestible polysaccharides reduced the serum urea levels. Younes *et al.*[6] reported an increase in cecal ammonia concentration and reduction in plasma urea in rats fed resistant starch diets. Birkett *et al.*[7] reported the effects of resistant starch on markers of colonic protein metabolism. Faecal nitrogen excretion was increased, while concentrations of phenol and ammonia decreased, in animals fed resistant starch. It was suggested that replicating bacteria use fermentable carbohydrate source as their energy substrate and act as nitrogen sink and reduce the faecal ammonia and phenols while increasing faecal execration of nitrogen. Similarly, HeiJnen and Beynen[62] showed that consumption of retrograded starch (RS3) but not cooked (RS2) resistant starch shifts nitrogen excretion from urine to faeces in cannulated piglets. It was also shown that large bowel fermentation of starch to be altered by dietary protein and non-digestible protein, namely resistant protein. It is suggested that diets rich in fermentable carbohydrates there is an enlargement of the cecum, hence a greater surface of exchange. Due to this enlargement and elevated cecal blood flow, diets containing fermentable carbohydrate promote a higher flux of urea towards the cecum and concomitantly lower the ammonia concentration in the digestive content.

Wheat resistant starch and pectin diets lowered the fasting blood glucose and 6hr post feeding blood glucose concentrations. This is consistent with the study reported by Robertson *et al.*[63], who showed prior short term consumption of resistant starch enhances postprandial insulin sensitivity and lowered blood glucose in healthy subjects. Several separate mechanisms may be involved. High proportions of acetate and butyrate are generally generated with resistant starch colonic fermentation and propionate with non-digestible viscous polysaccharides[4,51]. It was shown that utilization of propionate by extrasplanchic tissues lead to enhanced glycogen deposition and lipogenesis in rats adapted to resistant starch diet[28]. Propionate is glycogenic and glucose-6-phosphate from gluconeogenic pathway would preferentially channeled toward glycogen synthesis even in the presence of glucose. Propionate is also a potent inhibitor of lactate utilization in the liver by inhibition of pyrurate carboxylase[58]. It was also reported that in humans, dietary supplementation with propionate improves parameters of glucose metabolism[16,17]. The difference in the short chain fatty acids pattern obtained from different carbohydrates is therefore expected to dictate the overall metabolic response. However, with the viscous non-digestible polysaccharides, gastric emptying may be delayed or interaction of digestive enzymes and substrates directly affected. Intestinal absorption of nutrients may be directly affected by association with the mucosal surface and influencing the transport barrier.

Maltase activity was reduced in groups fed non-digestible polysaccharide. It has been reported that viscous fibre derivatives will inhibit the uptake of glucose by segments of rats' Jejunum and hydrolysis of maltose by mucosal enzymes[64]. Similarly, Johnson and Gee[31] found maltase activities to be reduced in the proximal segment of rats fed on guar gum. Using duodenal cannulation techniques intestinal absorption of glucose was found to be inhibited by guar gum[22]. It was also shown that diets substituted with pectin and resistant starch exhibited similar epithelial structural and cellular proliferative changes[32,65]. This evidence collectively suggests that adaptive change in intestinal structure and function might be of significance in modification of nutrient bioavailability.

The studies suggest that resistant starch shares some of the physiological actions of non-starch viscous polysaccharides and have comparable effects on some aspects of carbohydrate and lipid metabolism. How ever their mechanisms of actions may not necessarily be the same in every respect. In contrast to soluble viscous non-starch polysaccharide, resistant starch is well tolerated and with defined structure and properties are easily manipulated, which may be interesting for dietary management of carbohydrate and lipid metabolic disorders.

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