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GLYCEMIC INDEX OF SUCROSE WITH D-XYLOSE (XF) IN HUMANS

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ABSTRACT: ***The aim of this study was to evaluate sucrase inhibition by D-xylose in humans. Sucrose was administered with D-xylose to 13 healthy volunteers (5 males and 8 females), and their blood glucose levels were examined. The mean ± standard error glycemic indices (GIs) of sucrose with 5% D-xylose (XF) and sucrose alone were 59.6 ± 4.0 and 77.6 ± 3.1, respectively. The mean glycemic index of sucrose with 5% D-xylose (XF) was 23% lower than that of sucrose alone. The mean glycemic index of sucrose with 5% D-xylose (XF) in obese individuals was significantly lower than that of sucrose with 5% D-xylose (XF) in normal-weight individuals. The glycemic index of sucrose with 5% D-xylose (XF) was reduced approximately 40% when compared with sucrose alone in obese individuals. However, the reduction in the glycemic index by D-xylose was relatively less in normal-weight individuals than in obese individuals. Additionally, there was a negative correlation between the glycemic index of sucrose with 5% D-xylose (XF) and body-fat percentage. Therefore, sucrose administered with an appropriate amount of D-xylose can contribute to the reduction of problems caused by excess sucrose consumption.***

KEYWORDS: Body Mass Index (BMI), Body-Fat Percentage, D-Xylose, Glycemic Index (GI), Sucrase Inhibitor

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INTRODUCTION

Sucrose is the most abundant free sugar in plants and has a pre-eminent role as a sweetener. For this reason, sucrose is widely used as the standard reference for sweeteners in households and the food industry. However, many individuals tend to reduce sucrose consumption for personal and public health reasons because many reports and papers have raised concerns about excess sugar consumption (Young, 1993; John, 1998).

Sugar alcohols, such as sorbitol and maltitol, and aspartame can be used as substitutes for sucrose. However, these sweeteners are not perfect alternatives because they have several problems. For instance, over-consumption of sugar alcohol can cause digestive problems, and aspartame consumption is restricted for phenylketonuric (PKU) patients.

The sucrose molecule is a disaccharide derived from glucose and fructose with the molecular formula $C_{12}H_{22}O_{11}$. To form sucrose, glucose and fructose are linked via an ether bond between the glucosyl subunit and the fructosyl unit. Sucrose is rapidly hydrolyzed and converted to glucose and fructose by sucrase (sucrose α-glucosidase) on the surface of the epithelial cells of the small intestinal villi, and they are absorbed rapidly in the blood stream (Stanik and Marcus, 1980). Absorbed glucose stimulates insulin secretion, and glucose is converted to glycogen by insulin. Finally, the created glycogen is stored in the liver. This carbohydrate metabolism is very important to maintaining the energy supply in the human body. However, problems with carbohydrate metabolism, which are caused by various factors, result in obesity and metabolic syndrome (Hauri et al. 1982; Toeller, 1994; Hilmar, 1995).

The α-glucosidase inhibitors, acarbose and voglibose, inhibit the enzyme, which is located in the small intestinal villi, and reduce the rapid elevation of blood glucose after meals and the subsequent rapid increase in insulin levels (Yasunori et al. 2000). Therefore, α-glucosidase inhibition has been the main focus for controlling carbohydrate metabolism for many years (Puls et al. 1975; Saito et al. 1998). L-arabinose and D-xylose,

which are natural pentoses, inhibit intestinal sucrase (sucrose α-glucosidase) and suppresses the glycemic response after sucrose administration to rats (Kenji et al. 1996; Toshihiko et al. 1996). Additionally, Toshihiko et al. have reported that D-xylose may have the ability to suppress the elevation of blood glucose and insulin in humans. However, their results were not statistically significant.

The glycemic index (GI), first proposed in 1981 (Jenkins, 1981), is a system of classifying food items by their induced glycemic response. The GI of a food depends on the rapidity of its digestion and the absorption of its carbohydrates, which is largely determined by its physical and chemical properties (Wolever et al. 1991; Aston et al. 2008).

Generally, obesity is defined as a body mass index (BMI) over 25. However, BMI is not suitable in Asian peoples because it is influenced by muscle weight. Rather, the body fat weight and body fat ratio are more able to determine obesity.

The objective of this study was to determine the effects of D-xylose on glycemic responses using a glycemic index (GI) test in humans.

MATERIALS AND METHODS

Subjects

Thirteen healthy volunteers participated in this test (5 males, 8 females). All volunteers were physically examined for their health status. Their age, sex, height, weight, body fat ratio and BMI were recorded. The physical conditions of all the subjects are presented in Table 1.

TABLE 1. Baseline characteristics of the studied subjects

PARAMETERS	SUBJECTS
Age	28.3 ± 5.5 years
BMI (Body Mass Index)	22.2 ± 3.5 kg/m^2
Body-fat percentage (%)	22.2 ± 5.8 %
Total cholesterol	176 ± 23.7 mg/dL
AST (Aspatate aminotransferase)	20 ± 6.3 IU/L
ALT (Alanine aminotransferase)	15 ± 5.6 IU/L

Glycemic index (GI) test

A capillary blood sample was used for the GI test. Glucometers (ONETOUCH Ultra, LifeScan, USA) were used to detect the blood glucose level in the capillary blood samples. These glucometers were evaluated for their accuracy and repeatability. Following an overnight fast, the fasting blood glucose levels of the volunteers were recorded. Glucose (50 g) dissolved in 250 ml of water was given to the volunteers, and their blood glucose levels were recorded at 15, 30, 45, 60, 90 and 120 minutes after its oral consumption. Two days after the glucose test, a GI test was conducted with sucrose. The test procedures followed those of the glucose test. Finally, a GI test of sucrose with 5% (w/w) D-xylose (XF) was conducted 2 days after the test with sucrose alone. Sucrose (47.5 g) and D-xylose (2.5 g) were dissolved in 250 ml of water and given to the volunteers. The other test methods followed those of the glucose and sucrose tests.

Calculation of the glycemic index (GI)

GI was calculated according to the formula provided by SUGIRS (Sydney University Glycemic Index Research Service). The formula is as follows.

Statistical analyses

The data are expressed as the means ± S.E (standard error). Student's t-test was used to compare the differences in the GI test results between groups according to body fat ratio and BMI. In all statistical analyses, $P<0.05$ was considered significant.

RESULTS

The blood glucose levels at 15, 30 and 45 min after sucrose with 5% D-xylose (XF) administration (Fig. 1A) were significantly lower than those of sucrose. The incremental area under the curve (IAUC) of XF was significantly less than the IAUC of sucrose (Fig. 2B). The calculated mean ± SE (standard error) GI of XF and sucrose alone were 59.6 ± 4.0 and 77.6 ± 3.1, respectively. The mean GI of XF was significantly lower than that of sucrose (Table 2).

TABLE 2. Mean glycemic indices of sucrose with 5% D-xylose (XF) and sucrose in subjects

SUBJECTS	XF GI	SUCROSE GI	GI RATIO (%)	BODY FAT RATIO	BMI
A	65.2	81.6	79.8%	15.5	18.3
B	57.8	80.2	72.0%	18.2	21.7
C	39.8	69.8	57.0%	35.3	25.3
D	45.5	70.1	64.8%	18.5	18.5
E	62.6	66.4	94.3%	24.8	23.3
F	40.1	72.3	55.5%	29.2	27.9
G	82.3	66.5	123.8%	18.7	23.6
H	84.6	80.6	105.0%	14.6	17.5
I	64.3	95.3	67.4%	20.4	26.1
J	57.0	104.2	54.7%	26.8	22.3
K	71.3	70.8	100.7%	22.3	19.7
L	58.3	76.7	76.0%	20.5	18.3
M	46.5	74.5	62.4%	24.2	25.8
Mean	59.6	77.6	76.8%	22.2	22.2

FIGURE 1. Changes in blood glucose levels of subjects after administration of sucrose and sucrose with D-xylose (A). The incremental area under curve of subjects calculated for 2 h after administration of sucrose and sucrose with D-xylose (B). Each value is the mean ± SE. * Statistically different from sucrose, P < 0.05.

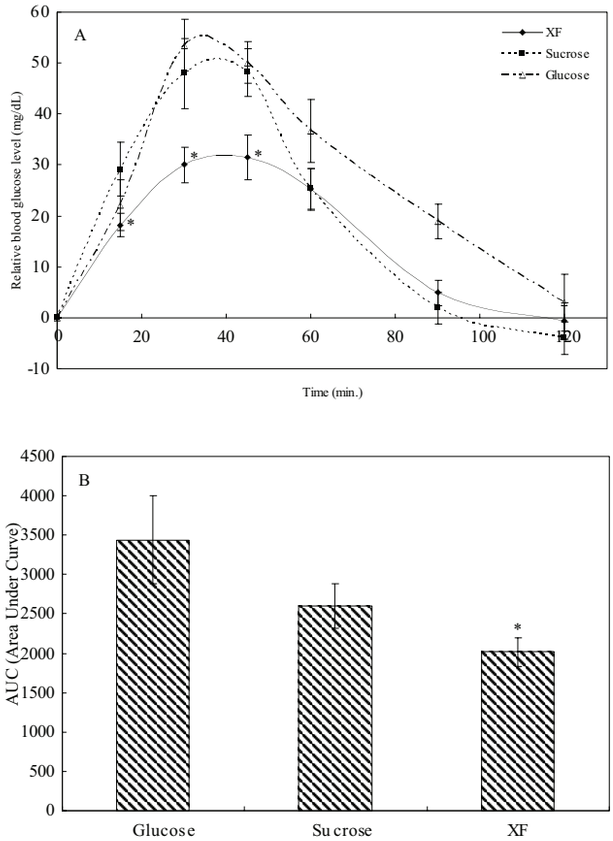
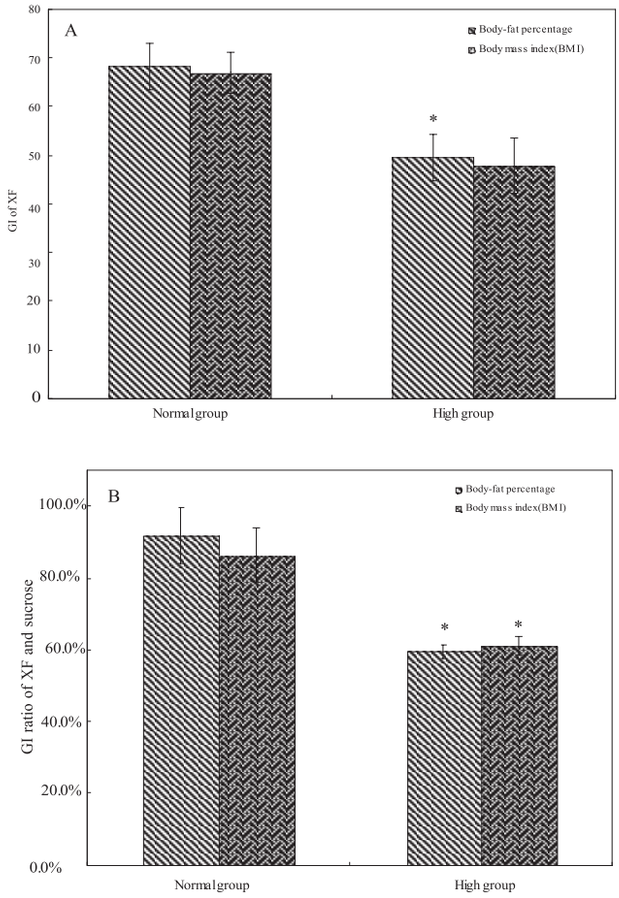


FIGURE 2. Change of GIs of XF according to body-fat percentage and body mass index (BMI) (A). The GI ratio of XF and sucrose according to body-fat percentage and BMI (B). Each value is the mean ± SE. * Statistically different from sucrose control, P < 0.05.

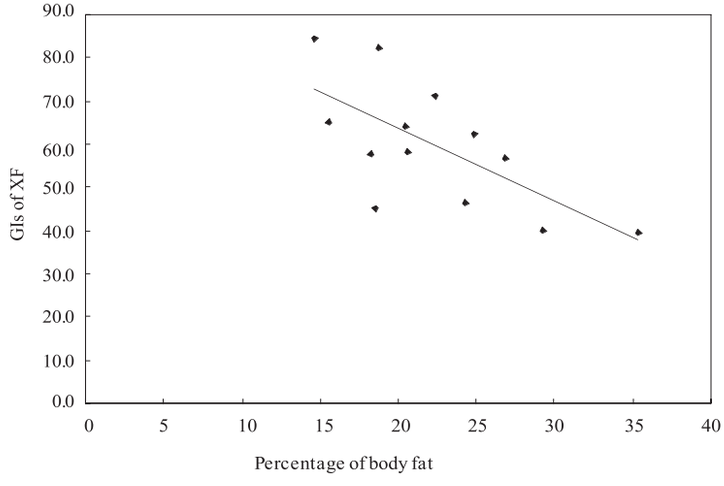


The mean GI of XF in the high body fat group was significantly lower than that in the normal body fat group whereas the GI of XF did not differ significantly from the GI of sucrose alone in the high BMI group (Fig. 2A). However,

TABLE 3. Mean glycemic indices of sucrose with 5% D-xylose (XF) and sucrose according to body fat ratio

NO.	GLYCEMIC INDICES OF XF	
	Obesity (Over 25 %)	Normal (Below 25 %)
1	65.2	39.8
2	57.8	40.1
3	45.5	64.3
4	62.6	46.5
5	82.3	
6	84.6	
7	70.8	
8	76.7	
Mean	68.2	49.5

FIGURE 3. Negative correlation between body-fat percentage and GIs of XF in 13 subjects.



the GI ratio of XF and sucrose alone in the high body fat and high BMI groups were significantly different from those in the normal body fat and normal BMI groups (Fig. 2B and Table 3).

The GI of XF and the body fat ratio were negatively correlated (Fig. 3), with a Pearson's correlation coefficient (ρ) of -0.674 (p=0.016).

DISCUSSION

Our study verified the positive effects of D-xylose on blood glucose changes in humans, and the results were statistically significant, unlike those of a previous study (Toshihiko et al. 1996). The increase in postprandial blood glucose was suppressed when sucrose with D-xylose were orally administered to the patients with D-xylose. The increase in blood glucose at 15 minutes after XF administration was lower than that after sucrose administration because sucrose was degraded more slowly due to the inhibitory effect of D-xylose on human sucrase (Fig. 1A). Additionally, the blood glucose level was changed moderately during the GI test by the co-administration of D-xylose. Consequently, the IAUC of XF was less than that of sucrose (Fig. 1B)

Additionally, D-xylose was more effective in obese individuals, particularly in the high body-fat ratio group (Fig. 2A). The GI ratio of XF and sucrose was lower in obese individuals than in normal-weight individuals; the GI of XF was significantly decreased in obese individuals, whereas the GI of sucrose was not significantly different. Differences in D-xylose absorption may account for these differences. We also observed that D-xylose absorption was delayed in obese individuals (data not shown). When a relatively large amount of D-xylose remained in the small intestine in obese individuals, residual D-xylose continuously inhibited sucrase action. Consequently, the GI of XF in obese individuals was reduced compared to the GI of XF in normal-weight individuals. D-xylose is known to be passively transported. However, there is no clear reason for the difference in the D-xylose absorption rate according to body-fat ratio and BMI. Further research is needed to determine the cause.

D-xylose, which is inexpensive and abundant, is currently used in the food industry as an ingredient. Our study confirmed that D-xylose is one of best materials to counteract sucrose's effects, particularly in obese individuals. Therefore, sucrose consumed with an appropriate amount of D-xylose can contribute to the reduction of problems caused by excess sucrose consumption.

CONCLUSION

Our study verified the sucrase inhibitory effect of D-xylose using the GI test in humans, and the results were statistically significant. In particular, D-xylose was more effective in obese individuals. Accordingly, when consumed with sucrose, D-xylose can contribute to the reduction of problems caused by excess sucrose consumption.

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