ISSN : 0974 - 7435

Volume 5 Issue 5





Trade Science Inc.

An Indian Journal SHORT COMMUNICATION

#### BTAIJ, 5(5), 2011 [290-292]

# Effects of daily intake of the rare sugar D-psicose on liver and muscle glycogen repletion with D-fructose administration after exhaustive swimming

**Tatsuhiro Matsuo** 

Faculty of Agriculture, Kagawa University, Ikenobe, Kita-gun, Miki-cho, Kagawa 761-0795, (JAPAN) E-mail : matsuo@ag.kagawa-u.ac.jp Received: 7<sup>th</sup> June, 2011 ; Accepted: 7<sup>th</sup> July, 2011

## ABSTRACT

We investigated the effects of daily intake of the rare sugar D-psicose on liver and muscle glycogen repletion with D-fructose administration after exhaustive swimming. A single dose of D-psicose with D-fructose did not affect post-exercise glycogen repletion in liver and soleus muscle. However, the rates of liver and soleus muscle glycogen repletion were faster in rats given to 3% D-psicose solution daily than in those given to water. We concluded that daily intake of D-psicose may accelerate the repletion of liver and muscle glycogen after exhaustive swimming. © 2011 Trade Science Inc. - INDIA

Adequate glycogen stores in muscle and liver are essential for high performance during prolonged exercise in both competition and training<sup>[1,2]</sup>. If the next competition or training session is scheduled to take place within a few hours after the first, it is important to restore glycogen in muscle and liver quickly. Thus, many studies have focused on maximizing the rate of glycogen repletion soon after prolonged exercise in both humans and rats<sup>[3,4]</sup>. Recently, the rare sugar D-psicose (D-ribo-2-hexulose), a C-3 epimer of D-fructose, increased hepatic glycogen accumulation in rats fed 5% D-psicose in a high-starch diet<sup>[5]</sup>. In addition, it was reported that D-psicose (10% of D-glucose) caused the disappearance of the increase in plasma glucose concentration after D-glucose loading in diabetic rats<sup>[6]</sup>. These results suggested that the administration of a small amount of D-psicose together with D-glucose after prolonged exercise increases glycogen repletion quickly. However, the administration of D-glucose is not always desirable with endurance sports, because D-glucose inhibits lipolysis, which provides an important fuel, free fatty acids (FFA), for contracting muscles<sup>[7]</sup>. On the other hand, it has been reported that D-fructose has little effect on insulin secretion compared with D-glucose<sup>[8]</sup>, and does not inhibit lipolysis in vivo<sup>[9]</sup>. The present study was performed to determined the effects of combined D-fructose and D-psicose given after exhaustive swimming on the repletion of glycogen stores in liver and muscle. We also examined the effects of daily intake of D-psicose before the administration of

## **K**EYWORDS

D-psicose; D-fructose; Liver; Muscle; Glycogen.

#### SHORT COMMUNICATION

D-fructose and D-psicose on glycogen repletion.

All procedures involving animals were approved by the Animal Care Committee of Kagawa University.

Forty-eight male Wistar rats (3 weeks old) were obtained from Japan SLC (Shizuoka, Japan). They were fed CE-2, a commercial rodent diet (CLEA, Tokyo, Japan), and water ad libitum until they were 4 weeks old. They were caged individually at 22±2°C, with lights on from 08:00 to 20:00. Each group of rats was mealfed with CE-2 from 08:00 to 09:00 and from 20:00 to 21:00. During the meal-feeding period, half of the rats were given 3% D-psicose solution (P group) and the other half were given water (W group) ad libitum. All the rats were trained by swimming for 15-30 min, 5 days per week, for 3 weeks. The swimming was performed in a plastic pool (50-cm diameter) filled with at 33-35°C (50-cm deep). Rats had a sinker corresponding to 2% of body mass, which was tied to their body during training. The rats did not exercise for 3 days after the final training session. In the P group, the drinking liquid was changed from D-psicose solution to water 2 days before the final experimental day.

On the final day of the experiment, 6 rats from each of the P and W groups were sacrificed at 11:00 (PC and WC groups, respectively), and the remaining rats exercised for 2.5 h swimming with a load, corresponding 2% of body mass, tied to their bodies. Immediately after exercise, 6 rats from each of the P and W group were sacrificed at 13:30 (PE and WE groups, respectively). Half of the remaining P group were administered 2 g/kg D-fructose solution (PE-F group) and the other half received 2 g/kg D-fructose plus 0.2 g/kg Dpsicose solution (PE-FP group) at 13:30. Similarly, half of the remaining W group were administered 2 g/kg Dfructose solution (WE-F group) and the other half received 2 g/kg D-fructose plus 0.2 g/kg D-psicose solution (WE-FP group) at 13:30. The PE-F, PE-FP, WE-F, and WE-FP groups of rats were allowed to recover in their own cages with no drinking water for 2.5 h. All of the remaining rats were sacrificed at 16:00.

Animals were sacrificed by decapitation. Blood was collected and serum was prepared. A portion of the largest lobe of the liver and the soleus muscle were rapidly removed and snap-frozen in liquid nitrogen. All tissue samples were stored at -80°C until analysis. Tissue glycogen contents were determined according to Lo et al<sup>[10]</sup>. Serum glucose and FFA concentrations were measured enzymatically using kits (Glucose CII-test and NEFA E-test) purchased from Wako Pure Chemical Industries, Osaka, Japan. Mean values of each group were compared by two-way analysis of variance. Significance of differences between mean values was determined using Turkey's multiple comparison. Differences were considered significant at p<0.05. Statistical ana-

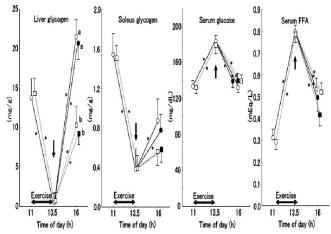


Figure 1 : Glycogen levels in the liver and soleus muscle and concentrations of serum glucose and FFA before and after exhaustive swimming. Each point represents the mean and standard error for 6 rats. The circles and squares show the P and W groups respectively. At 16:00,  $\circ$ , PE-FP group; •, PE-F group;  $\Box$ , WE-FP group; •, WE-FF group; •, WE-FF

lyzes were performed using Excel Statistics 2008 (SSRI Co., Ltd., Tokyo, Japan).

The results of this experiment are shown in Figure 1. Liver and soleus glycogen stores before swimming exercise did not differ between the P and W groups. The 2.5-hour swimming exercise resulted in significant reductions in liver and soleus muscle glycogen stores in both P and W groups but post-exercise glycogen levels in these tissues did not differ between the two groups. Liver glycogen levels in both the P and W groups increased significantly during the 2.5-hour recovery period. The rate of liver glycogen repletion was significantly faster in the PE-F and PE-FP groups than in the WE-F and WE-FP groups. The rate of soleus muscle glycogen repletion did not differ among the PE-F, PE-FP, WE-F, and WE-FP groups. Soleus muscle glycogen level was increased significantly in the PE-F and

**BioTechnology** An Indian Journal

## SHORT COMMUNICATION •

PE-FP groups, but was not altered in the WE-F and WE-FP groups during the 2.5-hour recovery period. The 2.5-hour swimming exercise resulted in a significant increase and the 2.5-hour recovery period resulted in a significant decrease in serum glucose and FFA concentrations in both the P and W groups. The daily intake of D-psicose or post-exercise D-psicose administration did not affect serum glucose and FFA concentrations.

The present study suggested that the daily intake of the rare sugar D-psicose can facilitate the repletion of liver and muscle glycogen after exhaustive swimming. However, a single dose of D-psicose did not affect postexercise glycogen repletion. D-Psicose is partly absorbed in the digestive tract and most absorbed D-psicose is excreted into the urine<sup>[11, 12]</sup>. Therefore, appropriate period might be necessary until the rat metabolism adjusts to D-psicose.

Fructokinase is a specific kinase present in liver that affects the transfer of phosphate from ATP to fructose, forming D-fructose 1-phosphate. It has also been shown to be present in the kidneys and intestine, but not in skeletal muscle<sup>[13]</sup>. This enzyme will not phosphorylate D-glucose, and unlike glucokinase its activity is unaffected by insulin, which may explain why D-fructose disappears from the blood. It seems likely that this is the major route for the phosphorrylation of D-fructose. In skeletal muscles, D-fructose is phosphorylated by another kinase, hexokinase, to D-fructose 6-phosphate<sup>[14]</sup>. Toyoda et al.<sup>[6]</sup> suggested that D-psicose can activated hepatic glucokinase by improving the translocation of glucokinase from the nucleus to the cytoplasm in the rat liver. D-Psicose may activate not only glucokinase but also fructokinase or hexokinase although the mechanism is unclear.

The results in recovering rats showed that the daily intake of D-psicose was more effective for glycogen repletion in the liver than the soleus muscle after postexercise D-fructose administration. Cori<sup>[15]</sup> and Bergstorm et al.<sup>[16]</sup> reported that D-fructose can be more effectively used as a precursor for liver glycogen than for muscle glycogen in rats and human. It has also been reported that D-fructose utilization by skeletal muscle is very small and administered D-fructose is mostly converted to D-glucose or its metabolites by the liver, and then released into the blood circulation<sup>[17]</sup>. Therefore, the above difference observed in the recovering rats between the liver and soleus muscle may be partly related to the slow supply of glycogen precursor to muscle after administration of D-fructose.

In conclusion, the daily intake of the rare sugar ppsicose may accelerate the repletion of liver and muscle glycogen after exhaustive swimming. However, detailed studies are required to clarify the underlying mechanism.

#### REFERENCES

- [1] J.Bergstrom, E.Hultman; JAMA, 221, 999 (1972).
- [2] M.J.Reed, J.T.Brozinick Jr, M.C.Lee, J.L.Ivy; J.Appl.Physiol., 66, 720 (1989).
- [3] P.C.S.Blom, A.T.Hostmark, O.Vaage, K.R.Kardel, S.Maehlum; Med.Sci.Sports Exerc., 19, 491 (1987).
- [4] J.J.Zawadzki, D.L.Costill, D.D.Pascoe, R.A.Robergs, W.J.Fink; Med.Sci.Sports Exerc., 23, 44 (1991).
- [5] T.Matsuo, K.Izumiri; Biosci.Biotechnol.Biochem., 70, 2081 (2006).
- [6] Y.Toyoda, S.Mori, N.Umemura, Y.Futamura, H.Inoue, T.Hata, I.Miwa, K.Murao, A.Nishiyama, M.Tokuda; Jpn.Pharmacol.Ther., 38, 261 (2010).
- [7] G.Ahlborg, P.Felig; Am.J.Physiol., 233, E188 (1977).
- [8] D.Hager, D.G.Pipeleers, A.Herchuelz, W.J.Malaisse; Acta.Med.Scand.Suppl., 542, 159 (1977).
- [9] P.ROzen, E.Shafrir; Israel J.Med.Sci., 8, 838 (1972).
- [10] S.Lo, J.C, Russel, A.W.Taylor; J.Appl.Physiol., 28, 234 (1970).
- [11] T.Matsuo, T.Tanaka, M.Hashiguchi, K.Izumori, H.Suzuki; Asia Pacific J.Clin.Nutr., 12, 225 (2003).
- [12] R.L.Whistler, P.P.Singh, W.C.Lake; Carbohyd.Res., 34, 222 (1974).
- [13] T.M.Cox; FASEB J., 8, 62 (1990).
- [14] P.A.Mayes; Am.J.Clin.Nutr., 58, 754S (1993).
- [15] C.F.Cori, J.Biol.Chem., 70, 577 (1926).
- [16] J.Bergstrom, P.Furst, F.Gallyas, E.Hultman, L.H.Nilsson, A.E.Rock-Norlund, E.Vinnars; Acta.Med.Scand.Suppl., 542, 57 (1972).

BioTechnolog 4n Iudian Journ