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Short Communication

Acute Effect of Oral Administration of Fructose on Blood Parameters and Hepatic Glycogen in Layer Chicks

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Abstract

Objective: The aim of the present study was to investigate the effect of oral administration of fructose on blood parameters and hepatic glycogen in chickens. **Materials and Methods:** Each chick was injected orally once only with fructose solution (80 mg/0.2 mL) after 12 h fasting. The levels of plasma glucose and Free Fatty Acid (FFA) and hepatic glycogen were measured at 1, 2 or 3 h after administration and compared with levels in 12 h fasted and *ad libitum* chicks. **Results:** Although no significant differences were detected in blood parameters between the *ad libitum* and fasting condition, the level of hepatic glycogen in the fasted chicks was lower than that in the *ad libitum* chicks ($p < 0.05$). The plasma glucose level at 2 h was significantly lower than that at 1 h post-injection ($p < 0.05$). The concentration of FFA at 1 h post-injection was significantly lower than that in the fasted chicks ($p < 0.05$). **Conclusion:** These results suggest that the administration of fructose affects carbohydrate and lipid metabolism in chicks.

Key words: Chick, free fatty acid, glycogen, fructose, carbohydrate

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Both fructose and glucose, which are monosaccharides, are absorbed from the small intestine into the hepatic portal vein and metabolized mainly in the liver¹. However, it is well known that the metabolic pathway and enzymes for fructose are different from glucose in mammals^{2,3}. In addition, there are many reports that fructose intake induces dysmetabolic syndrome, such as dyslipidemia, insulin resistance and increased visceral adiposity⁴⁻⁸.

Utilization of agro-industrial by-products as feedstuffs has been of concern to animal producers because it should be considered to help develop sustainable agriculture^{9,10}. For a number of the reasons, it is important to estimate nutrients and to determine whether the component has beneficial or adverse effects on animal production. Recently, by-products derived from fruits have been used in poultry farms¹¹⁻¹³. The acute effect of fructose on the metabolism of chicks is unknown because these by-products probably contain fructose, it is necessary to investigate the fructose intake in poultry. Therefore, the objective of this study was to investigate the effect of oral injection of fructose on blood parameters and hepatic glycogen in chicks.

MATERIALS AND METHODS

Day-old male layer-type chicks (Akita Co. Ltd., Hiroshima, Japan) were kept in a windowless temperature-controlled room with 24 h lighting and maintained at a temperature of 30°C. They were given free access to a commercial starter diet (Nichiwa Sangyo Co. Ltd., Kobe, Japan) and water during the pre-experimental period. The chicks were distributed into experimental groups based on their body weight so that the average body weight was as uniform as possible for each treatment. The birds were reared individually in experimental cages and had *ad libitum* access to food up to the time of the experiments. The handling of the birds was performed in accordance with the regulations of the Animal Experiment Committee of Hiroshima University and the recommendations of the National Research Council.

Oral administration was done according to Erwan *et al.*¹⁴ and the solutions (200 mL) were administered using a small syringe with a plastic needle. D-fructose (Sigma, St. Louis, MO, USA) was dissolved in distilled water. Each chick (4 days old) was injected orally once only with fructose solution (80 mg) after 12 h fasting. At 1, 2 or 3 h after the administration, all chicks were bled by cardiac puncture and blood was collected into heparinized tubes and centrifuged for 15 min. Immediately after blood collection, they were

decapitated and their livers were removed. Similar to the fructose administered chicks, the blood and livers of the 12 h fasted (0 h) and *ad libitum* chicks were also collected. Harvested plasma and liver tissue samples were stored at -20°C until assayed. The number of chicks in each group was as follows: *Ad libitum*, six; 12 h fast (0 h), five; 1 h, six; 2 h, five; 3 h and five, respectively.

The plasma concentrations of glucose and FFA were measured using a commercial kit (Glucose C II-Test Wako and NEFA C-Test Wako, Wako Pure Chemical Industries Ltd., Osaka, Japan).

Hepatic glycogen concentrations were measured using the method of alkaline digestion and ethanol precipitation¹⁵. In brief, a tissue sample (0.5 g) was heated with 1.5 mL of 30% potassium hydroxide solution (Nacalai Tesque, Inc., Kyoto, Japan) for 30 min. After cooling for 5 min, 0.25 mL of saturated sodium sulfate (Nacalai Tesque, Inc., Kyoto, Japan) and 2 mL of 95% ethanol (Nacalai Tesque, Inc., Kyoto, Japan) were poured into the obtained solution and stirred them. The solution was separated by centrifugation at 3,500 rpm for 10 min and the supernatant water was removed. Distilled water (5 mL) was added to the obtained sediment and heated at 45°C for 5 min. Additional distilled water (5 mL) was poured into the solution and the hydrolysis-glucose assay was performed using a commercial kit (Glucose C II-Test Wako, Wako Pure Chemical Industries Ltd., Osaka, Japan).

The data were analyzed using the commercially available package, StatView (Version 5, SAS Institute, Cary, USA). In the analysis of all the data, ANOVA was used to determine statistical significance due to treatment. When a treatment effect was significant, the Tukey-Kramer test was used to compare the significance among means. Differences were declared significant at $p < 0.05$. Data were expressed as Means \pm SEM.

RESULTS

Figure 1 shows the effect of oral administration of fructose on hepatic glycogen, plasma glucose and FFA concentrations in the chicks. The level of hepatic glycogen in the fasted (0 h) chicks was significantly lower than that in the other treated chicks ($F[4,22] = 10.733$, $p < 0.0001$). Although, no significant difference was detected in FFA concentration between the *ad libitum* and fasting conditions, the level at 1 h was significantly lower than that at 0 h post-injection ($F[4,22] = 3.456$; $p < 0.05$). Although, there was no significant difference between glucose concentrations in the *ad libitum* and fasted chicks but the level at 2 h was significantly lower than that at 1 h post-injection ($F[4,22] = 3.415$, $p < 0.05$).

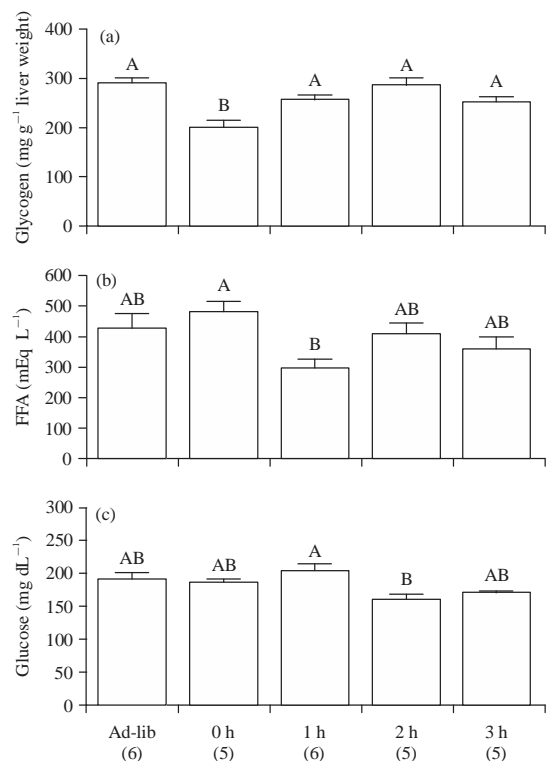


Fig. 1(a-c): Effect of fructose on liver glycogen, plasma FFA and glucose concentrations in chicks. FFA: Free fatty acid, LW: Liver weight, Ad-lib: *Ad libitum* condition. Values are Means \pm SEM of the number of chicks in parentheses. Means with different letters are significantly different at $p < 0.05$

DISCUSSION

The liver plays a crucial role in the utilization and distribution of nutrients and in particular, it is responsible for promoting gluconeogenesis and glucose production during periods of fasting¹⁶. In this study, the 12 h fasting reduced 30% of hepatic glycogen compared with the *ad libitum* condition (Fig. 1a). Consequently, the level of plasma glucose after 12 h feed deprivation appeared to be similar to the *ad libitum* condition (Fig. 1c).

Fructose is believed to enter the glycolytic pathway and accelerate activation of the glycolytic and glycogenesis pathways¹⁷⁻¹⁹. It is likely that the decreased level of FFA at 1 h postinjection (Fig. 1b) is due to promoting hepatic FFA uptake and β -oxidation of fatty acids in the liver. As results of the glucose produced via the glycolytic pathway, hepatic glycogen levels might recover to those of the *ad libitum* condition (Fig. 1a).

Hallfrisch¹ reported that oral administration of fructose does not cause a rapid increase in plasma glucose compared

with administration of glucose because there are differences in absorption and metabolism of fructose and glucose in mammals. Also, there is more moderate secretion of insulin after the fructose intake compared with glucose intake in mammals^{7,20}. In other experiment, it is found that increase of plasma insulin after fructose intake was lower than that after glucose intake in broiler chicks (control: 2.9 ± 0.1 ng mL⁻¹, fructose: 4.4 ± 0.3 ng mL⁻¹ and glucose: 5.5 ± 0.9 ng mL⁻¹) and it is reasonable that the following is possible. Increased plasma glucose via the glycolytic pathway is probably due to stimulating insulin secretion and then promoting glucose uptake in the liver and muscles. Consequently, the concentration of plasma glucose at 2 h became lower than that at 1 h post-injection (Fig. 1c).

The utilization of the agro-industrial by-products such as crop residues and these including grape¹¹, orange¹² and apple pomaces¹³ have been well studied. Although there are differences in carbohydrate metabolism between birds and mammals²¹, it is well known that a high fructose diet in mammals induces the lipid metabolism disorder dyslipidemia, or insulin-resistant diabetes^{8,22}. This study discovered the acute effect of fructose on metabolism that can be beneficial for utilization of by-products from fruits in chickens. This study will help the researcher to uncover the critical areas of carbohydrate metabolism that many researchers were not able to explore. Thus, a new theory on nutritional strategies may be arrived at. Further study on the effect of fructose on metabolism is needed to aid the research of utilization of by-products in chickens.

CONCLUSION

In conclusion, these results suggest that the administration of fructose affects carbohydrate and lipid metabolism in chicks and it appears that the acute effect of fructose on the absorption and metabolism in chicks is similar to that in mammals.

SIGNIFICANCE STATEMENT

- Fructose does not show the sharp increase in blood glucose level
- Oral administration of fructose has the decreased level of hepatic glycogen by fasting recovered in chicks
- There is a possibility that a high fructose feed stuff induces the lipid metabolism disorder in chickens

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