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Soybean β-conglycinin improves carbohydrate and lipid metabolism in Wistar rats

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The effects of dietary soybean β-conglycinin on lipid metabolism and energy consumption were studied in Wistar adult rats. Rats were fed, a diet containing casein (control group) or β-conglycinin (β-conglycinin group), for 4 weeks. Carbohydrate consumption was higher and fat consumption was lower in the β-conglycinin group than in the control group, whereas the total energy consumption was the same between the two groups. Serum adiponectin was higher in the β-conglycinin group than in the control group. Serum triacylglycerol levels in the β-conglycinin group were significantly lower than those in the control group. The secretion rate of triacylglycerols from the liver after the administration of tyloxapol, an inhibitor of lipolysis, was significantly lower in the β-conglycinin group than in the control group. These results suggest the possibility that β-conglycinin exerts hypolipidemic effects through an acceleration in carbohydrate consumption associated with an increase in adiponectin in rats.

Key words: adiponectin; β-conglycinin; energy consumption; soy protein

Soybean protein has been reported to have various physiological functions, including the reduction of serum cholesterol and triacylglycerol (TAG) levels in rodents and humans.[1–3] The blood-glucose decreasing effect of soybean protein has also been reported in diabetic KK-Ay mice.[4] Soybean protein is primarily composed of glycinin (11S globulin), lipophilic proteins, and β-conglycinin (7S globulin).[5] It has been reported that consumption of β-conglycinin results in significantly decreased blood TAG levels in rodents and humans.[6–8] Since some studies have shown that β-conglycinin feeding suppresses the activities of enzymes related to fatty acid synthesis and increases the activity of carnitine palmitoyltransferase (CPT) in the liver of rodents,[7,9] it has been suggested that suppression of fatty acid synthesis and stimulation of β-oxidation in the liver could be the cause for the reduction in serum TAG concentration. However, a reduction in the hepatic TAG concentration was not observed with feeding of β-conglycinin.[10] Furthermore, Tachibana et al. reported that β-conglycinin increased serum adiponectin levels and improved glucose tolerance in normal rats.[10] Since adiponectin has the ability to improve insulin sensitivity,[11] the results suggest that the increase in adiponectin by feeding of β-conglycinin could be a cause for the improved insulin sensitivity. These observations suggest that β-conglycinin plays a major role in the soy-protein-induced reduction in TAG concentration and the improvement in glucose metabolism. However, the precise mechanisms of the physiological functions of β-conglycinin are not fully understood. In the present study, we investigated the effects of β-conglycinin on lipid metabolism and energy consumption in male Wistar adult rats.

Materials and methods

Materials. β-conglycinin was kindly provided by Fuji Oil Co. (Osaka, Japan). Purity of β-conglycinin as protein was >90%. The contents of isoflavone and saponin were 0.4 and 0.2%, respectively.

Animals and diets. All aspects of the experiment were conducted according to the guidelines provided by the ethical committee for experimental animal care at Tohoku University (No. 2011AgA-29). Male Wistar rats (19 weeks old) were purchased from Clea Japan (Tokyo, Japan). Rats were housed individually in a temperature-controlled room (22–24 °C, lights on 08:00–20:00). They were allowed free access to a commercial chow for seven days. In both Experiments 1 and 2, the rats were divided into two groups (six rats each). The compositions of the experimental diets are

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Abbreviations: ACO, acyl-CoA oxidase; AMPK, 5′-AMP-activated protein kinase; CPT, carnitine palmitoyltransferase; FAS, fatty acid synthase; G6PDH, glucose-6-phosphate dehydrogenase; ME, malic enzyme; SREBP-1c, sterol regulatory element-binding protein 1c; TAG, triacylglycerol; VLDL, very-low-density lipoprotein.

shown in Table 1. The experimental diets contained 20% casein and β-conglycinin as the crude protein. Since different lots of β-conglycinin were used in Experiments 1 and 2, the protein content in the diets was slightly different between the two experiments. Rats were fed on one of the experimental diets for four weeks. In Experiment 1, on the morning of the last day of feeding, the rats were sacrificed by exsanguination from the aorta without fasting under ether anesthesia. The liver and white adipose tissue were excised. In Experiment 2, at the end of the feeding period, after fasting for 6 h from early in the morning, blood was collected from the tail vein. Then, rats were administered tyloxapol (480 mg/kg body weight, Sigma-Aldrich, St. Louis, USA), an inhibitor of lipolysis, via the jugular vein, and blood was collected from the tail vein after 2 h.[12] Immediately after the blood collection, the rats were sacrificed by exsanguination from the aorta under ether anesthesia. The serum was separated from the blood by centrifugation.

Measurement of energy expenditure. The measurement of energy expenditure was conducted for 24 h between days 21 and 22 of the feeding period in Experiment 1. The instruments and software used for the measurement of oxygen consumption and carbon dioxide expenditure in rats were obtained from Arco Systems (Chiba, Japan). The system consisted of 12 acrylic metabolic chambers, a mass spectrometer (model ARCO 2000), and a gas sampler (model ARCO 2000-GS). Each rat was placed into a metabolic chamber for the analysis of 24 h of respiratory gas. During the analysis, the rats had free access to the diets and water. Room air was introduced into the chambers at a rate of 1.0 L/min. Expired air was directed to a mass spectrometer. Air from each chamber was sampled for 15 s every 5 min. In addition, 24-h urine samples were collected and carbon dioxide production from protein metabolism was calculated from urinary nitrogen concentrations. Urinary nitrogen was determined with a commercial enzyme assay kit (Wako Pure Chemical Industries, Ltd, Osaka, Japan). The respiratory quotient and the consumption of carbohydrate, fat, protein, and energy were calculated by the software using the following equations:

Respiratory quotient = VCO_2/VO_2

Carbohydrate consumption (g/24 h) = $4.51 \times \{VCO_2 - (VCO_2\text{-}P)\} - 3.18 \times \{VO_2 - (VO_2\text{-}P)\}$

Fat consumption (g/24 h) = $1.67 \times \{VO_2 - (VO_2\text{-}P) - VCO_2 - (VCO_2\text{-}P)\}$

Protein oxidation (g/24 h) = urinary nitrogen (g/24 h) × 6.25

Energy consumption (cal/min) = $3.816 \times \{VO_2 - (VO_2\text{-}P)\} + 1.231 \times \{VCO_2 - (VCO_2\text{-}P)\}$

where VO_2 is the oxygen consumption, VCO_2 is the carbon dioxide production, VO_2-P is the oxygen consumption from protein metabolism, and VCO_2-P is the carbon dioxide production from protein metabolism.

Measurement of serum parameters. Serum TAG and glucose levels were measured using commercial enzyme assay kits (Wako Pure Chemical Industries, Ltd). Serum insulin and adiponectin levels were measured using commercial rat enzyme-linked immunosorbent assay kits (Shibayagi, Gunma, Japan and Otsuka Pharmaceutical Co. Ltd, Tokyo, Japan, respectively).

Measurement of TAG levels in the liver. Liver lipids were extracted according to the method of Folch et al.[13] and the liver TAG levels were measured using a commercial enzyme assay kit (Wako Pure Chemical Industries, Ltd).

Preparation of hepatic subcellular fractions. An aliquot of liver from each rat was homogenized in six volumes of a 0.25 M sucrose solution containing 1 mM ethylenediaminetetraacetic acid in 10 mM Tris-HCl buffer (pH 7.4).[14] After the nuclear fraction was precipitated, the resulting supernatant (homogenate) was centrifuged at 125,000 × *g* for 60 min to precipitate the subcellular components and the cytosol fraction was obtained as the supernatant. The protein concentration was determined according to the method of Lowry et al. with bovine serum albumin as the standard.[15]

Assays of hepatic enzyme activity. The enzyme activities of fatty acid synthase (FAS), glucose-6-phosphate dehydrogenase (G6PDH), and malic enzyme (ME) in the cytosol fraction as well as CPT and

Table 1. Dietary regimens.

	Experiment 1		Experiment 2	
Groups	Casein (g/1000 g)	β-CG (g/1000 g)	Casein (g/1000 g)	β-CG (g/1000 g)
Casein	233	–	233	–
β-CG	–	219	–	231
α-Cornstarch	132	132	132	132
Sucrose	100	100	100	100
Cellulose	50	50	50	50
Soybean oil	70	70	70	70
AIN-93 vitamin mix.	35	35	35	35
AIN-93G mineral mix.	10	10	10	10
Choline bitartrate	2.5	2.5	2.5	2.5
t-Butylhydroquinone	0.014	0.014	0.014	0.014
Cornstarch	to 1000	to 1000	to 1000	to 1000

Note: β-CG, β-Conglycinin.

acyl-CoA oxidase (ACO) in the homogenate were determined as described elsewhere.[16)]

Analysis of mRNA expression. Total RNA was extracted from 100 mg of liver using ISOGEN (Nippon Gene, Tokyo, Japan). The mRNA levels for FAS (*Fasn*), G6PDH (*G6pd*), ME (*Me1*), sterol regulatory element-binding protein 1c (SREBP-1c; *Srebp1c*), stearoyl-CoA desaturase 1 (*Scd1*), apolipoprotein B (*Apob*), CPT 1a (*Cpt1a*), and ACO 1 (*Acox1*) in the liver were determined with a real-time PCR system (ABI Prism 7300 Sequence Detection System, Applied Biosystems, Tokyo Japan). Primers designed for *Fasn* (forward, 5′-AGCATATCCCTGGAAACAGGTGAC-3′; reverse, 5′-TCTGTGGATAGGACTGAATGCTGTG-3′), *G6pd* (forward, 5′-TTCACACCATTGCTGCACAAGA-3′; reverse, 5′-ACCCTCATACTGGAAGCCCACTC-3′), *Me1* (forward, 5′-TGGGCATCCCTGTGGGTAA-3′; reverse, 5′-GCCGCAGCCCAATATACAAG-3′), *Srebp1c* (forward, 5′-GGAGCCATGGATTGCACATT-3′; reverse, 5′-GCTTCCAGAGAGGAGCCCAG-3′), *Scd1* (forward, 5′-TGAGGCCTTTAATCATCCCAAGAA-3′; reverse, 5′-TTTATCAGGACTCGCCCAGAGTG-3′), *Apob* (forward, 5′- TAGCATGCTTGCTGACATAAATGGA-3′; reverse, 5′- ATGGAGCTGCCGGAGGTAATC-3′), *Cpt1a* (forward, 5′-TCATTGCCTGCCAGTTCCATTA-3′; reverse, 5′-TTGTCCAGCTATGCAGCCTTTG-3′), *Acox1* (forward, 5′-GAGTTCCCGATGGGCACAA-3′; reverse, 5′-TGGCTGAAAGCCTGGAGGTAAG-3′), *18S rRNA* (forward, 5′-ACTCAACACGGGAAACCTCA-3′; reverse, 5′-AACCAGACAAATCGCTCCAC-3′), and *Ef-1* (forward, 5′-GATGGCCCCAAATTCTTGAAG-3′; reverse, 5′-GGACCATGTCAACAATGGCAG-3′) were used to generate PCR-amplified probes from cDNA that had been prepared from total liver RNA.[17)] The results were quantified using a comparative method and were expressed as a relative value after normalization to the expression of *18S rRNA* or *Ef-1*.

Calculation of the secretion rate of TAGs from the liver. The secretion rate of TAGs from the liver was calculated using the following formula:

TAG secretion rate (mg/dL/h) = {serum TAG level after 2 h (mg/dL) − initial serum TAG level (mg/dL)}/2.

Measurement of amounts of crude protein and fatty acids in feces. Feces were collected from day 23 to day 25 of the feeding period in Experiment 1 and food intake was measured for 2 days. The feces were lyophilized and powdered, then the crude protein content was quantified using the Kjeldahl method.[18)] The crude protein intake was calculated from the 2-day food intake data and the crude protein excretion rate was calculated based on the calculated values. The fecal lipids were extracted[19)] and the fatty acids were quantified by the alkaline titration method after saponification and extraction of the fatty acids. Apparent fatty acid excretion rates were calculated from the fat amounts calculated from the 2-day food intake data.

Statistical analysis. All values are expressed as means ± standard errors. After the test for equality of variance, the significance of the differences between the means for two groups was determined using the Student's *t*-test. Differences were considered significant at $p < 0.05$.

Results

Food intake, body weight, body weight gain, and adipose tissue weights

Total food intake did not differ between the control and the β-conglycinin groups (Table 2). The final body weight and body weight gain were significantly lower in the β-conglycinin group than in the control group. The liver weight per 100 g of body weight was significantly lower in the β-conglycinin group when compared with the control group. The white adipose tissue weight per 100 g of body weight did not differ between the groups.

Energy consumption

Energy consumption was the same between the two groups (9.18 ± 0.13 and 9.69 ± 0.20 kcal/24 h/100 g body weight in the control and β-conglycinin groups, respectively). Carbohydrate consumption was higher and fat consumption was lower in the β-conglycinin group than in the control group (Fig. 1). The protein oxidation calculated from the urinary nitrogen excretion was higher in the β-conglycinin group than in the control group.

Table 2. Effect of dietary β-conglycinin on body weight, body weight gain, food intake, and liver and abdominal white adipose tissue weights.

	Control	β-Conglycinin
Initial body weight (g)	418 ± 5	417 ± 5
Final body weight (g)	459 ± 6	431 ± 6*
Body weight gain (g)	41.9 ± 2.6	13.9 ± 3.6*
Total food intake (g)	553 ± 10	557 ± 7
Liver weight (g/100 g body weight)	3.40 ± 0.08	3.00 ± 0.03*
Relative abdominal white adipose tissue weight		
Epididymal (g/100 g body weight)	1.47 ± 0.07	1.42 ± 0.04
Perirenal (g/100 g body weight)	2.37 ± 0.14	2.53 ± 0.13
Mesenteric (g/100 g body weight)	1.52 ± 0.13	1.65 ± 0.11
Total (g/100 g body weight)	5.36 ± 0.32	5.61 ± 0.21

Note: Data are means ± standard error of six rats.
*Significant differences at $p < 0.05$ vs. the control group.

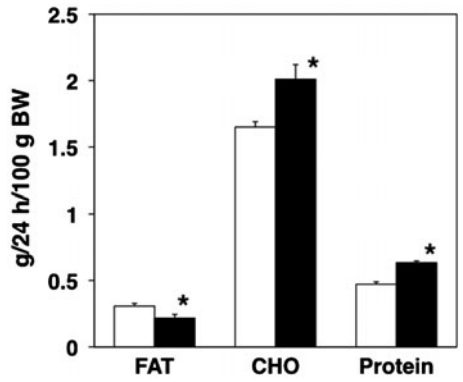

Fig. 1. Effects of dietary β-conglycinin on consumption of fat, carbohydrate, and protein. Open bars show the control group and closed bars show the β-conglycinin group.

Notes: FAT, fat consumption; CHO, carbohydrate consumption; Protein, protein consumption. Data are means ± standard error of six rats. Asterisks show significant differences at $p < 0.05$ vs. the control group.

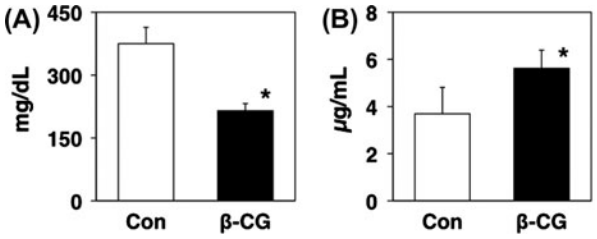

Fig. 2. Effects of dietary β-conglycinin on TAG (A) and adiponectin (B) concentrations in serum.

Notes: The open bars show the control group and the closed bars show the β-conglycinin (β-CG) group. Data are means ± standard error of six rats. Asterisks show significant differences at $p < 0.05$ vs. the control group.

Serum parameters

The serum TAG concentration in the β-conglycinin group was significantly lower than that in the control group (Fig. 2). The serum adiponectin concentration was higher in the β-conglycinin group than in the control group. The serum cholesterol concentration was significantly lower in the β-conglycinin group than in the control group (70.9 ± 1.7 and 104 ± 5 mg/dL, respectively). There were no significant difference in serum glucose concentration (200 ± 11 and 188 ± 8 mg/dL in the control and β-conglycinin groups, respectively) and insulin concentration (5.04 ± 1.12 and 4.57 ± 0.77 ng/mL in the control and β-conglycinin groups, respectively).

Hepatic TAG concentration

There were no significant difference in the hepatic TAG concentrations between the control and the β-conglycinin groups (18.4 and 17.1 mg/g in the control and β-conglycinin groups, respectively).

Activities of enzymes related to fatty acid synthesis and β-oxidation in the liver

The activities of FAS, G6PDH, and ME, three enzymes related to fatty acid synthesis, were markedly lower in the β-conglycinin group than in the control group (Fig. 3). The activity of CPT, a key β-oxidation enzyme in mitochondria, was significantly higher in the β-conglycinin group than in the control group. The ACO activity did not differ between the groups.

mRNA expression of genes related to lipid metabolism in the liver

The mRNA expression of the lipogenic genes *Fasn*, *G6pd*, *Srebp1c,* and *Scd1*, was markedly lower in the

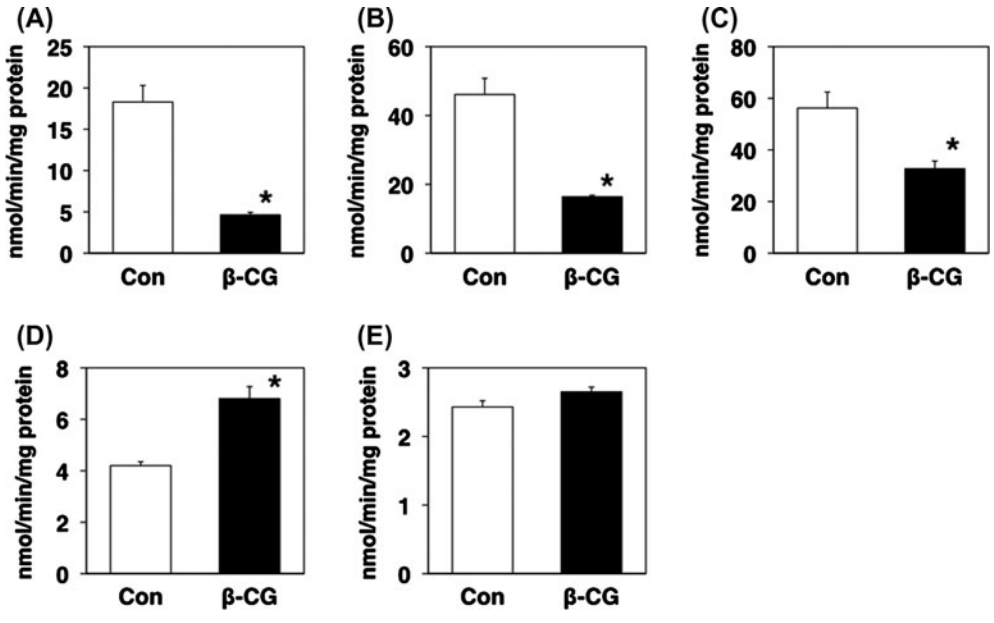

Fig. 3. Effects of dietary β-conglycinin on hepatic enzyme activities related to lipid metabolism.

Notes: (A) fatty acid synthase; (B) glucose-6-phosphate dehydrogenase; (C) malic enzyme; (D) carnitine palmitoyltransferase; (E) acyl-CoA oxidase. The open bars show the control group and the closed bars show the β-conglycinin (β-CG) group. Data are means ± standard error of six rats. Asterisks show significant differences at $p < 0.05$ vs the control group.

Table 3. Effect of dietary β-conglycinin on mRNA expression of genes related to lipogenesis and lipolysis in the liver.

Gene	Control (Arbitrary unit)	β-Conglycinin (Arbitrary unit)
Fasn	1.00 ± 0.23	0.257 ± 0.044*
G6pd	1.00 ± 0.25	0.221 ± 0.024*
Me1	1.00 ± 0.24	0.613 ± 0.116
Srebp1c	1.00 ± 0.35	0.285 ± 0.060*
Scd1	1.00 ± 0.20	0.0463 ± 0.0072*
Apob	1.00 ± 0.06	1.04 ± 0.11
Cpt1a	1.00 ± 0.06	1.08 ± 0.19
Acox1	1.00 ± 0.08	1.00 ± 0.12

Note: Data are means ± standard error of six rats. *Fasn*, Fatty acid synthase; *G6pd*, Glucose-6-phosphate dehydrogenase; *Me1*, Malic enzyme 1; *Srebp1c*, Sterol regulatory element-binding protein 1c; *Scd1*, Stearoyl-CoA desaturase 1; *Apob*, Apolipoprotein B; *Cpt1a*, carnitine palmitoyltransferase 1a; *Acox1*, acyl CoA oxidase 1.
*Significant differences at $p < 0.05$ vs. the control group.

β-conglycinin group when compared with the control group (Table 3). The mRNA expression of *Me1* and *Apob* was the same between the two groups. The mRNA expression of the lipolytic genes *Cpt1a* and *Acox1* did not differ between the control and the β-conglycinin groups.

Fecal weight and excretion rates of crude protein and fatty acids in feces

The dry weight of feces was significantly higher in the β-conglycinin group than in the control group (Table 4). The crude protein fecal excretion rate was slightly but significantly higher in the β-conglycinin group than in the control group. The excretion rates of fatty acids in feces were the same in both the control and the β-conglycinin groups.

Table 4. Effect of dietary β-conglycinin on fecal excretion of protein and fatty acids.

	Control	β-Conglycinin
Dry weight (g/day)	1.38 ± 0.03	1.54 ± 0.04*
Apparent excretion of crude protein (%)	5.31 ± 0.22	6.70 ± 0.16*
Fatty acids (mg/day)	19.6 ± 0.8	20.1 ± 0.9
Apparent excretion of fatty acids (%)	1.54 ± 0.07	1.57 ± 0.13

Note: Data are means ± standard error of six rats.
*Significant differences at $p < 0.05$ vs. the control group.

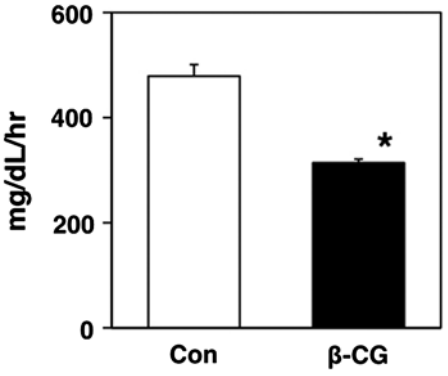

Fig. 4. Effects of dietary β-conglycinin on secretion rates of TAGs from the liver.

Notes: The open bar shows the control group and the closed bar shows the β-conglycinin (β-CG) group. Data are means ± standard error of 7–8 rats. Asterisks show significant differences at $p < 0.05$ vs. the control group.

Secretion rate of TAGs from the liver

The serum TAG levels initially and 2 h after tyroxapol administration in the β-conglycinin group were significantly lower than those in the control group (data not shown), and the secretion rate of TAGs calculated was significantly lower in the β-conglycinin group than in the control group (Fig. 4).

Discussion

Previous studies have reported that dietary β-conglycinin lowered serum TAG concentrations in rodents and humans.[6–8] A previous study showed that the hepatic activity of enzymes related to β-oxidation was higher and that of enzymes related to fatty acid synthesis was lower in rats fed a β-conglycinin diet than in those fed a casein diet.[9] The similar results were also observed in mice.[7] The authors of the studies suggested that β-conglycinin accelerated β-oxidation and suppressed fatty acid synthesis in the liver and that these may be causes of the reduction in serum TAG concentration.[7,9] However, in a study in mice, the effects of β-conglycinin on the activities of those enzymes were a little ambiguous.[7] In the present study, we clearly showed the increased activity of CPT and the decreased activity of enzymes related to fatty acid synthesis in the rat liver (Fig. 3). The results were similar to those reported by Fukui et al.[9] However, measurement of whole-body energy consumption revealed for the first time that fat consumption was significantly lower in rats fed β-conglycinin than those fed casein (Fig. 1). Because the liver is a major organ for the oxidation of fatty acids, our results suggest the possibility that β-conglycinin does not enhance β-oxidation in the liver. In this context, Sugano et al. reported that dietary soy protein, of which β-conglycinin comprised about 23%, did not increase ketone body production in isolated perfused rat liver when compared with casein,[20] suggesting that soy protein did not accelerate fat consumption. We also suggested that hepatic β-oxidation is not necessarily dependent on the activities of CPT and ACO in the liver, but on the amount of fatty acid substrates.[21] Therefore, it is highly possible that although β-conglycinin improves the hepatic β-oxidation ability, it does not increase β-oxidation, at least in normal feeding conditions. Yamauchi et al. showed that adiponectin stimulated ^{14}C-palmitic acid oxidation in myocytes *in vitro*.[11] In the present study, although adiponectin was higher in the β-conglycinin-fed group than in the casein-fed group (Fig. 2), fat consumption was not increased by the feeding of β-conglycinin. This discrepancy might be explained by β-oxidation being dependent on the substrate supply. *In vitro*, the supply of substrate fatty acids can be sufficient, but not *in vivo*. Further studies are necessary to determine the precise mechanisms.

In the present study, we showed that carbohydrate consumption was enhanced by feeding with β-conglycinin

(Fig. 1), suggesting that glucose oxidation in the whole body is stimulated by β-conglycinin. It is known that an increase in glucose entry into the liver stimulates glycolysis and activates carbohydrate response element-binding protein, which induces increased expression of the *FAS* gene.[22] Gene expression of enzymes related to fatty acid synthesis in the liver can also be induced by insulin stimulation. In the present study, gene expression and activities of FAS, ME, and G6PDH were suppressed in the rats fed β-conglycinin (Table 3 and Fig. 3). Gene expression of SREBP-1c, which regulates the mRNA expression of enzymes related to fatty acid synthesis, was also suppressed in the rats fed β-conglycinin. The results suggest that the entry of glucose into the liver is lower in the β-conglycinin-fed rats than in the casein-fed rats. Therefore, we think that consumption of glucose was not accelerated in the liver in rats fed β-conglycinin, but it was activated in extrahepatic tissues, such as muscle.

Like Tachibana et al. [10] we showed that the concentration of serum adiponectin was elevated in rats fed β-conglycinin (Fig. 2). Yamauchi et al. reported that adiponectin increased glucose uptake in myocytes.[11] They also showed that adiponectin stimulates phosphorylation and activation of 5′-AMP-activated protein kinase (AMPK) in myocytes. Since the activation of AMPK stimulates glucose uptake in skeletal muscle,[23,24] elevation of adiponectin by β-conglycinin feeding can cause increased uptake of glucose and acceleration of carbohydrate metabolism in muscle. Recently, Tachibana et al. reported that β-conglycinin activated phosphorylation of AMPK in the muscle of spontaneously diabetic Goto-Kakizaki rats.[25] This observation supports our view. They also observed that phosphorylation of AMPK was not activated in the liver of Goto-Kakizaki rats fed β-conglycinin.[25] Therefore, it is highly possible that acceleration of carbohydrate consumption by β-conglycinin feeding is induced by its increase in muscle, but not in liver. Because the increase in carbohydrate consumption in muscle can induce the reduction of glucose entry into the liver, gene expression, and the activity of enzymes related to fatty acid synthesis can be reduced. This could be the mechanism by which activity and gene expression of enzymes related to fatty acid synthesis and SREBP-1c mRNA were suppressed in the liver of the β-conglycinin-fed rats. Mochizuki et al. reported that β-conglycinin-derived peptides, 68% of which had a molecular mass less than 500 Da, reduced the incorporation of ^{3}H-glycerol and ^{14}C-acetate into TAGs in HepG2 cells, suggesting the possibility that peptides derived from β-conglycinin directly reduce the synthesis of fatty acids and/or TAG (26). More studies are necessary to determine the effects of β-conglycinin on fatty acid and TAG synthesis in the liver.

There is a possibility that a reduction in fatty acid synthesis in the liver can induce a reduction in the hepatic TAG concentration. However, in the present study, hepatic TAG was not reduced in the rats fed β-conglycinin; only serum TAG was decreased (Fig. 2). The same result was also previously observed in rats.[10] We showed that feeding β-conglycinin reduced the TAG secretion rate from the liver (Fig. 4). Tachibana et al. also observed that the very-low-density lipoprotein (VLDL)–TAG concentration was lower in rats fed β-conglycinin compared with casein.[10] Mochizuki et al. showed that secretion of apo B-100, the major apoprotein in VLDL, was suppressed by the addition of β-conglycinin-derived peptides in HepG2 cells.[26] Sugano et al. reported that dietary soybean protein suppressed the secretion of VLDL-TAG from perfused rat liver.[20] The results suggest that β-conglycinin contained in soy protein has the ability to suppress VLDL-TAG secretion from the liver. On the whole, our observations suggest that suppression of VLDL-TAG secretion from the liver compensated for the decrease in hepatic TAG concentration caused by the reduction of fatty acid synthesis when fed β-conglycinin. Thus, the hepatic TAG concentration was not reduced in the rats fed β-conglycinin.

We observed that the diet containing β-conglycinin suppressed body weight gain without reducing food intake (Table 2). Other researchers also observed the suppression of body weight gain when feeding β-conglycinin to rats and mice.[7,10] We observed an increase in the apparent excretion of crude protein into feces (Table 4). It was reported that β-conglycinin has relatively lower sulfur-containing amino acids when compared with soy protein isolate,[5] in which the amino acid score is 100 as is the case in casein. These two factors could be reasons for the reduced weight gain. Although Fukui et al. observed increased fecal fatty acid excretion in rats fed β-conglycinin,[9] we did not show increased excretion of fecal fatty acids (Table 4). Therefore, the change in fecal excretion of fatty acids is not a cause of suppressed growth, at least in the present experimental conditions.

In the present study, no significant differences were observed in the serum glucose and insulin concentrations between the casein and β-conglycinin groups. The same observation was reported in Wistar rats, the same strain as in the present study, by Tachibana et al. [10] In contrast, significant reduction of the serum glucose and insulin concentrations in the feeding of β-conglycinin was reported in mice[7] and Goto-Kakizaki rats.[25] The reasons for the discrepancy could be differences in experimental conditions, such as fasting time and strains.

β-Conglycinin contained 0.4% isoflavone and 0.2% saponins in the present study. The contents were almost the same as is the case with soy protein isolate. Fukui et al. reported that the feeding of ethanol extract of soy protein isolate did not lower plasma TAG concentration in SD rats.[27] The results suggest that isoflavone and saponins contained in β-conglycinin do not influence TAG metabolism.

In conclusion, the present study suggests that β-conglycinin accelerates the consumption of carbohydrate and then induces the suppression of fatty acid synthesis in the liver. This could be a major cause of the reduction of the serum TAG concentration. The increase in adiponectin when fed β-conglycinin could be a cause of accelerated carbohydrate consumption.

Disclosure statement

No potential conflict of interest was reported by the authors.

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