Hypoglycemic and hypolipidemic effects of *samnamul* (shoot of *Aruncus dioicus* var. *kamtschaticus* Hara) in mice fed a high-fat/high-sucrose diet

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Abstract The hypoglycemic and hypolipidemic effects of *samnamul* were investigated. The α -glucosidase inhibitory activity of *samnamul* in vivo was determined in normal mice. Oral administration of *samnamul* extract (500 mg/kg) or acarbose (50 mg/kg) significantly reduced the postprandial glucose response. The effects of chronic consumption of *samnamul* on fasting hyperglycemia and dyslipidemia were determined in C57BL/6 J mice with diabetes mellitus induced by a high-fat/high-sucrose (HFHS) diet. Consumption of *samnamul* extract at 0.5% of the diet for 12 weeks decreased serum glucose, triglyceride, and cholesterol levels, the homeostasis model assessment for insulin resistance index, and activities of maltase and sucrase in the small intestine. These results suggest that *samnamul* had hypoglycemic and hypolipidemic effects in an animal model of type 2 diabetes and that the hypoglycemic effect occurred partly via the inhibition of α -glucosidase activity.

Keywords *Aruncus dioicus* var. *kamtschaticus* Hara · Glucose · Alpha-glucosidase · Triglyceride · Diabetes mellitus

Introduction

Diabetes mellitus is characterized by hyperglycemia resulting from defects in insulin secretion and/or insulin action (American Diabetes Association, 2005). Type 1 diabetes mellitus, which manifests due to impaired insulin secretion, accounts for only 5–10% of diabetes. Type 2 diabetes mellitus, which is caused mainly by insulin resistance and relative insulin insufficiency, accounts for 90–95% of diabetes. Diabetic complications related to uncontrolled hyperglycemia include retinopathy, nephropathy, neuropathy, and cardiovascular disease (CVD), which increase the morbidity and mortality of patients with diabetes (The Diabetes Control and Complications Trial Research Group, 1993). Among the complications of diabetes, CVD is the most common and the most prevalent cause of death (Matheus et al., 2013). Dyslipidemia, characterized by increased triglyceride and decreased HDL cholesterol levels, is common in diabetes mellitus (Haffner SM; American Diabetes Association, 2004). Strict control of blood glucose is one of the most important strategies in the treatment of diabetes and prevention of diabetic complications, including CVD (American Diabetes Association, 2017). In addition, the control of dyslipidemia is associated with a lower risk of cardiovascular complications.

Oral hypoglycemic drugs, which are prescribed for patients with diabetes, include biguanides, sulfonylureas, meglitinides, thiazolidinediones, and α -glucosidase inhibitors. α -Glucosidase inhibitors, such as acarbose and miglitrol, lower blood glucose by impairing the activity of α -glucosidase in the brush border cells of the small intestine, resulting in the inhibition of digestion and absorption of dietary carbohydrates (Lorenzati et al., 2010). However, undesirable side effects of acarbose, such as flatulence,

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abdominal discomfort, bloating, and diarrhea, can be a major limitation. Therefore, a strong demand exists for the development of new α -glucosidase inhibitors with no side effect (Kumar et al., 2011).

Samnamul is a young shoot of *Aruncus dioicus* var. *kamtschaticus* Hara (goat's beard), which is a perennial herbaceous plant in the family Rosaceae (Ahn et al., 2014). *Samnamul* is a vegetable used in traditional Korean cuisine. Because raw *samnamul* is collected only in spring and can be stored for only short periods of time, most *samnamul* on the market is in the form of *muknamul*, which is *samnamul* that has been blanched and then air dried after collection, enabling prolonged storage. Prior to cooking, *muknamul* must be rehydrated by soaking, boiling, and then infusion in water (Ahn et al., 2014; Lee et al., 2015). Ahn et al. (2014) reported that a 75% ethanol extract of *muknamul* form of *samnamul* had yeast α -glucosidase inhibitory activity that corresponded to 47.8% of that of acarbose in vitro. Rehydrated and cooked *samnamul* extract exerted α -glucosidase inhibition, which corresponded to 35.2% of the acarbose activity, suggesting that *samnamul* could be helpful for the control of hyperglycemia. However, the effect of *samnamul* on α -glucosidase activity has not been studied in vivo. Consumption of raw *samnamul* reduced blood glucose, triglyceride, and cholesterol levels in rats with streptozotocin (STZ)-induced diabetes, an animal model of type 1 diabetes (Shin et al., 2008). However, no beneficial effect of *samnamul* as *muknamul* on hyperglycemia or dyslipidemia in diabetes has been reported.

Therefore, this study was conducted to investigate the effects of rehydrated *muknamul* form of *samnamul* on the blood glucose level and lipid profile in mice with diabetic diet-induced type 2 diabetes. In addition, the in vivo α -glucosidase inhibitory activity of *samnamul* was studied in mice to investigate the possibility of using *samnamul* as a hypoglycemic agent.

Materials and methods

Materials and chemicals

Samnamul in the form of *muknamul* was purchased from Woolwellbeing Food Inc. (Ulleung, Korea). Casein, a mineral mixture, a vitamin mixture, and D,L-methionine were obtained from ICN Pharmaceuticals Inc. (Costa Mesa, CA, USA). *tert*-Butylhydroquinone was purchased from Fluka Co. (Milwaukee, WI, USA). Sucrose and soybean oil were purchased from Cheiljedang Co. (Seoul, Korea). Cornstarch and lard were obtained from Daesang Co. (Seoul, Korea) and Lotte Samgang Co. (Seoul, Korea), respectively. Acarbose was obtained from Bayer Korea (Seoul, Korea). The glucose, triglyceride, cholesterol, and

HDL cholesterol assay kits were products of Yeongdong Co. (Seoul, Korea). An insulin assay kit was obtained from Mercodia (Uppsala, Sweden), and an adiponectin assay kit was purchased from BioVendor Research and Diagnostic Products (Modrice, Czech Republic). Alphacel, choline bitartrate, and all other reagent-grade chemicals were purchased from the Sigma Chemical Co. (St. Louis, MO, USA).

Preparation of *samnamul* extract

Samnamul in the form of *muknamul* was rehydrated using a standard procedure, as described previously by Ahn et al. (2014) and Lee et al. (2015). Briefly, *samnamul* as *muknamul* was soaked in water (1:6, w/w) for 16 h, boiled in water (1:6, w/w) for 30 min, and re-soaked in water (1:6, w/w) for 1 h, and the excess water was then removed by squeezing. The rehydrated *samnamul* was freeze dried, milled, and then extracted using 75% ethanol (1:10, w/v) at 25 °C for 12 h. After the solution was filtered through Whatman no. 5 filter paper (Whatman, Kent, UK), the filtrate was concentrated with a rotary evaporator (Eyela, Tokyo, Japan) at 50 °C. The extraction yield was 4.6%.

Oral carbohydrate tolerance test in normal mice

Six-week-old male C57BL/6 J mice were purchased from Bio Genomics, Inc. (Seoul, Korea). The mice were fed standard rodent pellet food (Purina Inc., Seongnam, Korea) and tap water ad libitum. The mice were kept in individual cages, maintained with a 12:12-h light:dark cycle at room temperature (19–23 °C) and standard humidity (50–60%). Mice weighing 23–26 g ($n = 21$) were allocated randomly to three groups. The mice were administered maltose (2 g/kg) alone or supplied with a 75% ethanol extract of *samnamul* (500 mg/kg) or acarbose (50 mg/kg) by gastric intubation after food deprivation for 12 h. Acarbose was used as a positive control. Blood samples were taken from the tail vein at 0, 30, 60, and 120 min, and glucose levels were assessed using a glucometer (Glucotrend; Roche Diagnostics, Lewes, UK). Areas under the blood glucose response curves (AUCs) were calculated.

Assessment of fasting blood glucose levels and lipid profiles in mice fed a high-fat/high-sucrose diet

Six-week-old male C57BL/6 J mice ($n = 28$) were purchased from Bio Genomics, Inc. and allowed a 2-week adaptation period, during which time they were fed standard rodent pellet food (Purina Inc.). The mice were divided randomly into four groups and fed a basal diet (control group), a high-fat/high-sucrose diet (HFHS group), a HFHS diet containing 0.5% *samnamul* extract (*samnamul*

group), or a HFHS diet containing 0.05% acarbose (acarbose group) ad libitum for 12 weeks (Table 1).

The mice were sacrificed by a heart puncture procedure after an overnight fast, and blood and small intestine samples were collected. Blood samples were centrifuged (1500×g, 15 min, 5415R; Eppendorf, Westbury, NY, USA), and the serum was recovered and stored at −70 °C prior to further analyses.

Serum glucose, triglyceride, and total cholesterol, and HDL cholesterol levels were measured by enzymatic methods using commercial assay kits. Serum adiponectin levels were measured using an enzyme-linked immunosorbent assay (ELISA) kit. Serum insulin levels were measured using an ELISA kit, and the homeostasis model assessment for insulin resistance (HOMA-IR) index was calculated using the following equation (Haffner et al., 1997):

$$\text{HOMA-IR} = \text{fasting glucose (mmol/L)} \times \text{fasting insulin (ng/mL)} / 22.5$$

The mucosa was scraped from the proximal third of the jejunum-ileum of the small intestine using a glass slide, and then homogenized in cold saline (1:4, w/v). After the homogenates were centrifuged (5415R; Eppendorf) at 4000×g for 30 min, the supernatant was used to determine

maltase and sucrase activities using the method described by Dahlqvist (1984). To measure maltase activity, the supernatant diluted with distilled water (100 µL; 1:100, v/v) was mixed with 100 µL 0.1 M sodium maleate buffer (pH 6.0) containing 0.056 M maltose. To measure sucrase activity, the diluted supernatant was mixed with 0.1 M sodium maleate buffer containing 0.056 M sucrose. The reaction mixture was incubated (37 °C, 60 min; SWB-03; JEIO TECH, Daejeon, Korea) and then mixed with 0.8 mL distilled water. The reaction was stopped by heating in a boiling water bath for 2 min. The amount of glucose released from the reaction mixture was measured by the glucose oxidase method (Raabo and Terkildsen, 1960), and the protein content was determined using the method described by Lowry et al. (1951). The enzyme activity was expressed as micromoles of maltose or sucrose hydrolyzed per minute per gram of protein (U/g protein). All measurements were performed in triplicate. All animal study procedures were approved by the Institutional Animal Care and Use Committee at our University (Approval no. 2015-16 and 2016-009).

Statistical analysis

The data were presented as means ± standard errors of the mean (SEM). Statistical significance was evaluated by one-way analysis of variance followed by Tukey's test, with a significance level of 5%.

Results and discussion

Effect of *samnamul* on postprandial glucose in normal mice

The effect of *samnamul* on α-glucosidase activity in vivo was determined in normal mice. The blood glucose levels peaked at 30 min after the oral maltose dose (2 g/kg) and decreased thereafter (Fig. 1). The incremental blood glucose levels of the mice administered *samnamul* extract (500 mg/kg) were reduced significantly at 30 and 60 min after maltose dosing compared with the control group ($p < 0.05$). Administration of acarbose (50 mg/kg), a positive control, significantly lowered the incremental glucose levels at 30, 60 (both $p < 0.01$), and 90 ($p < 0.05$) min compared with the control group. The AUCs of the glucose response curves were significantly reduced in the *samnamul* group (3677 ± 357 mg·min/dL) and the acarbose group (2901 ± 314 mg·min/dL) compared with the control group (5753 ± 564 mg·min/dL; both $p < 0.01$; Fig. 1). Incremental blood glucose levels at any time-point and AUCs did not differ significantly between the *samnamul* and acarbose groups.

Table 1 Composition of experimental diets

Ingredient	Group			
	Control	HFHS ¹	<i>Samnamul</i> ²	Acarbose ³
		(%)		
Casein	20.0	20.0	20.0	20.0
Corn starch	65.0	10.1	9.6	10.05
Sucrose	–	27.0	27.0	27.0
Alphacel	5.0	–	–	–
Corn oil	5.0	3.0	3.0	3.0
Lard	–	33.0	33.0	33.0
Vitamin mixture ⁴	1.0	1.4	1.4	1.4
Mineral mixture ⁵	3.5	5.0	5.0	5.0
D,L-Methionine	0.3	0.3	0.3	0.3
Choline bitartrate	0.2	0.2	0.2	0.2
tert-Butylhydroquinone ⁶	0.001	0.007	0.007	0.007
<i>Samnamul</i> extract			0.5	–
Acarbose	–	–	–	0.05

¹Mice fed a high-fat/high-sucrose diet

²Mice fed the HFHS diet containing 0.5% *samnamul* extract

³Mice fed the HFHS diet containing 0.05% acarbose

⁴AIN-76 vitamin mixture

⁵AIN-76 mineral mixture

⁶Antioxidative agent, 0.01 g/50 g lipids

Fig. 1 Incremental blood glucose levels (A) and area under the glucose response curve (B) of normal mice. Control group (●): Maltose (2 g/kg) was administered orally to normal mice after an overnight fast. *Samnamul* group (■): Maltose (2 g/kg) with 75% ethanol extract of *samnamul* (500 mg/kg) was administered orally to mice after an overnight fast. Acarbose group (▲): Maltose (2 g/kg) with acarbose (50 mg/kg) was administered orally to mice after an overnight fast. Values represent mean $\pm$ SEM ($n = 7$). Means that do not share a common letter are significantly different at $p < 0.05$ (*) or $p < 0.01$ (**)

Samnamul is a vegetable that has unique flavor, taste, and chewy texture, which is similar to that of beef. Because most *samnamul* is consumed as rehydrated *muknamul*, determination of the physiological functions of rehydrated *muknamul* in comparison with those of raw *samnamul* was important. The rehydrated *muknamul* form of *samnamul* displayed α -glucosidase activity in vitro (Ahn et al., 2014). In this study, the rehydrated *muknamul* suppressed the postprandial blood glucose level and the AUC, suggesting that it exerted α -glucosidase activity in vivo.

Acarbose is a widely consumed hypoglycemic agent among patients with type 2 diabetes (Lorenzati et al., 2010). Chronic consumption of acarbose reduces the risks of CVD and hypertension in patients with glucose intolerance (Chiasson et al., 2003). Postprandial hyperglycemia has been reported to increase oxidative stress (Ceriello et al., 1997), which can cause endothelial dysfunction,

contributing to the development of hypertension and CVD (Heitzer et al., 2001). Acarbose alleviated the increase in oxidative stress (Ceriello et al., 1996), resulting in its beneficial effects on the prevention of CVD and hypertension (Chiasson et al., 2003). Thus, *samnamul* is a possible candidate for the control of postprandial hyperglycemia, which can help to decrease the risk of cardiovascular complications.

Effects of chronic feeding of *samnamul* on fasting hyperglycemia and dyslipidemia in diet-induced diabetic mice

The effects of chronic consumption of *samnamul* on the fasting blood glucose level and blood lipid profile were determined in C57BL/6 J mice fed a HFHS diet for 12 weeks. These animals were reported to develop insulin resistance and type 2 diabetes after chronic consumption of the diabetogenic HFHS diet (Choi et al., 2014; Surwit et al., 1988).

Mice fed the HFHS diet displayed increased final body weight, weight gain, and feed efficiency ratios (FERs) compared with mice fed the basal diet ($p < 0.01$; Table 2). Consumption of *samnamul* extract at 0.5% of the diet or acarbose at 0.05% of the diet did not significantly affect the final body weight, weight gain, or FER compared with the HFHS group. Serum glucose and insulin levels and HOMA-IR values were significantly elevated in the HFHS group compared with the control group ($p < 0.05$; Table 3). In this study, mice fed the HFHS diet for 12 weeks developed hyperinsulinemia, elevated HOMA-IR, a surrogate marker of insulin resistance, and hyperglycemia, which are characteristics of type 2 diabetes. Yang et al. (2012) reported that consumption of the HFHS diet induced obesity and caused the downregulation of gene expression of insulin receptor substrate 2 (IRS2), protein kinase B beta (Akt2), and 5' adenosine monophosphate-activated protein kinase (AMPK) that are involved in insulin signaling pathway which results in insulin resistance in C57BL/6 J mice.

Consumption of *samnamul* or acarbose reduced serum glucose and insulin levels, HOMA-IR values compared with the HFHS group ($p < 0.05$). Consumption of the 80% ethanol extract of raw *samnamul* at 0.3% of the diet for 5 weeks decreased the serum glucose level in STZ-treated rats (Shin et al., 2008). In this study, the extract of the dehydrated *muknamul* form of *samnamul* offered at 0.5% of the diet effectively improved insulin resistance and reduced the fasting serum glucose level, and its effects were comparable with those of acarbose offered at 0.05% of the diet in diet-induced diabetic mice. In the present study, the average daily intakes of *samnamul* extract and acarbose were 500.9 and 51.1 mg/kg body weight,

Table 2 Body weight, food intake and feed efficiency ratio in mice fed the experimental diets

Group ¹	Control	HFHS	<i>Samnamul</i>	Acarbose
Initial body weight (g)	21.9 ± 0.5 ^{ns,3,4}	21.8 ± 0.5	21.9 ± 0.5	21.7 ± 0.6
Final body weight (g)	27.0 ± 0.9 ^a	41.9 ± 1.1 ^b	40.8 ± 1.1 ^b	39.4 ± 1.0 ^b
Weight gain (mg/day)	60.2 ± 7.5 ^a	239.5 ± 14.6 ^b	225.7 ± 8.1 ^b	210.7 ± 8.8 ^b
Food intake (g/day)	3.38 ± 0.13 ^{ns}	3.17 ± 0.10	3.13 ± 0.10	3.11 ± 0.11
FER ³	1.82 ± 0.27 ^a	7.61 ± 0.56 ^b	7.22 ± 0.21 ^b	6.78 ± 0.21 ^b

¹Groups are the same as in Table 1²Feed efficiency ratio (FER, %) = (Body weight gain (g/day)/food intake (g/day)) × 100³Values are mean ± SEM (*n* = 7). Means in the same row not sharing a common letter are significantly different at *p* < 0.01⁴ns: not significant**Table 3** Serum glucose, insulin, and adiponectin levels, HOMA-IR, and maltase activity in the small intestine in mice fed the experimental diets

Group ¹	Control	HFHS	<i>Samnamul</i>	Acarbose
Glucose (mg/dL)	117.2 ± 5.6 ^{a3}	158.1 ± 5.8 ^b	133.5 ± 5.4 ^a	126.2 ± 5.4 ^a
Insulin (ng/mL)	0.79 ± 0.07 ^a	1.62 ± 0.09 ^c	1.29 ± 0.07 ^b	1.16 ± 0.10 ^b
Adiponectin (μg/mL)	9.76 ± 0.39 ^{ns4}	8.31 ± 0.52	8.09 ± 0.56	8.89 ± 0.44
HOMA-IR ²	0.225 ± 0.015 ^a	0.629 ± 0.034 ^c	0.427 ± 0.031 ^b	0.362 ± 0.033 ^b
Maltase activity (U/g protein)	291.1 ± 13.4 ^{ab}	335.9 ± 15.6 ^b	270.8 ± 14.9 ^a	259.9 ± 14.8 ^a
Sucrase activity (U/g protein)	64.3 ± 4.7 ^{ab}	78.4 ± 4.9 ^b	60.1 ± 3.4 ^a	56.4 ± 3.9 ^a

¹Groups are the same as in Table 1²Homeostasis model assessment for insulin resistance (HOMA-IR) = Fasting glucose (mmol/L) × fasting insulin (ng/mL)/22.5³Values are mean ± SEM (*n* = 7). Means in the same row not sharing a common letter are significantly different at *p* < 0.05⁴ns: not significant

respectively. Consumption of acarbose reduces the requirement for insulin to control postprandial hyperglycemia and improve insulin sensitivity (Carrascosa et al., 2001).

Samnamul extract or acarbose significantly reduced both maltase and sucrase activities of the small intestine compared with the HFHS group (*p* < 0.05). Chronic consumption of acarbose reduced small intestinal α-glucosidase activity in STZ-induced diabetic rats (Liu et al., 2010) and db/db mice, an animal model of type 2 diabetes (Kang et al., 2015; Kim et al., 2014). It was suggested that reduced intestinal activities of maltase and sucrase by chronic consumption of acarbose could contribute to a decrease of fasting blood glucose (Kim et al., 2014; Liu et al., 2010).

Consumption of *samnamul* extract did not significantly affect serum adiponectin in mice fed a HFHS diet. Adiponectin is an adipocytokine that plays a crucial role in improving insulin sensitivity (Sheng and Yang, 2008), and adiponectin levels were related inversely to insulin resistance in patients with type 2 diabetes (Aleidi et al., 2015).

Therefore, *samnamul* could improve fasting hyperglycemia via α-glucosidase inhibitory activity, rather than by influencing the adiponectin level.

α-Glucosidase inhibitory activities of *muknamul* form of *samnamul* during rehydration such as soaking, boiling, and re-soaking were well correlated with the contents of polyphenols (Ahn et al., 2014). Caffeic acid, one of the representative phenolic acids, was isolated from *samnamul* as an active component with antioxidant activity (Vo et al., 2014). Caffeic acid has been proven to possess strong inhibitory activity against α-glucosidase (Obloh et al., 2015). It was reported that absorption rate of caffeic acid was 95% in ileostomy subjects (Olthof et al., 2001). α-Glucosidase inhibitory effect of *samnamul* could have been partly mediated by caffeic acid.

Obesity induces oxidative stress which triggers insulin resistance (Furukawa et al., 2004; Higdon and Frei, 2003). Oxidative stress activates stress pathways involving serine/threonine kinases, which in turn impairs insulin signaling. *Samnamul* showed antioxidant activity in vitro (Ahn et al., 2014) and in rats with STZ-induced diabetes (Shin et al.,

Table 4 Serum lipid profiles in mice fed the experimental diets

Group ¹	Control	HFHS	<i>Samnamul</i>	Acarbose
Triglycerides (mg/dL)	97.2 ± 4.8 ^{a2}	127.6 ± 6.3 ^b	105.8 ± 5.2 ^a	102.1 ± 5.8 ^a
Total cholesterol (mg/dL)	94.8 ± 5.2 ^a	133.4 ± 5.9 ^c	111.6 ± 4.8 ^b	122.0 ± 6.4 ^{ab}
HDL cholesterol (mg/dL)	50.2 ± 3.0 ^{ns3}	59.7 ± 3.3	57.7 ± 3.8	61.9 ± 3.9

¹Groups are the same as in Table 1²Values are mean ± SEM (*n* = 7). Means in the same row not sharing a common letter are significantly different at *p* < 0.05³ns: not significant

2008). Therefore, Antioxidant activity of *samnamul* could also contribute to alleviation of insulin resistance in mice fed the HFHS diet in this study.

Numerous studies have been carried out to search new α -glucosidase inhibitors with no side effect from edible plants (Deguchi and Miyazaki, 2010; Fujita and Yamagami, 2001; Wang et al., 2013). Among them, extract of *Touchi*, a fermented soybean product, and guava leaf extract which showed α -glucosidase inhibition in vitro and in vivo have been accepted as health functional food in Korea (Ministry of Food and Drug Safety, 2018). *Samnamul* is the vegetable which has been used in traditional Korean cuisine. The results of this study can be useful in planning dietary guideline for diabetic patients and in developing health functional food from *samnamul*.

Serum triglyceride levels were decreased significantly by *samnamul* extract or acarbose compared with the HFHS group, with no significant influence on the HDL cholesterol level (*p* < 0.05; Table 4). Serum total cholesterol levels were significantly lowered in mice fed *samnamul* extract compared with the HFHS group. Previously, raw *samnamul* extract was found to decrease serum triglyceride and cholesterol levels in STZ-treated rats (Shin et al., 2008). In this study, the rehydrated *muknamul* form of *samnamul* had hypotriglyceremic and hypocholesterolemic effects in HFHS diet-induced diabetic mice. Insulin resistance has been associated with hypertriglyceridemia (Mooradian, 2009). Insulin resistance increases the free fatty acid flux from adipocytes to the liver, stimulating the production of triglycerides and triglyceride-rich VLDL in the liver (Taskinen, 2003). In addition, insulin resistance or deficiency decreases VLDL catabolism via the downregulation of lipoprotein lipase, leading to hypertriglyceridemia. Blood triglyceride levels are related strongly to cardiovascular complications in both type 1 and type 2 diabetes (Schofield et al., 2016). In addition, reduction of the blood cholesterol level improves cardiovascular outcomes in diabetic patients (Schofield et al., 2016). Therefore, *samnamul* may be helpful in alleviating cardiovascular complications arising from diabetes.

In conclusion, the dehydrated *muknamul* form of *samnamul* suppressed postprandial hyperglycemia in normal

mice. Chronic consumption of *samnamul* exerted hypoglycemic, hypotriglyceridemic, and hypocholesterolemic effects and improved insulin sensitivity in mice with diet-induced type 2 diabetes. These results suggest that the dehydrated *muknamul* form of *samnamul* would be beneficial in the treatment of type 2 diabetes.

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