Extract of *Salacia oblonga* lowers acute glycemia in patients with type 2 diabetes\(^1\)–\(^4\)

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**ABSTRACT**

**Background:** Two previous studies tested the efficacy of *Salacia oblonga* extract in healthy adults.

**Objective:** This study evaluated the effect of an herbal extract of *Salacia oblonga* on postprandial glycemia and insulinemia in patients with type 2 diabetes after ingestion of a high-carbohydrate meal.

**Design:** Sixty-six patients with diabetes were studied in this randomized, double-blinded crossover study. In a fasted state, subjects consumed 1 of the following 3 meals: a standard liquid control meal, a control meal + 240 mg *Salacia oblonga* extract, and a control meal + 480 mg *Salacia oblonga* extract. Serum glucose and insulin samples were measured at baseline and at postprandial intervals up to 180 min.

**Results:** Both doses of the *Salacia* extract significantly lowered the postprandial positive area under the glucose curve (14% for the 240 mg extract and 22% for the 480 mg extract) and the adjusted peak glucose response (19% for the lower dose and 27% for the higher dose of extract) to the control meal. In addition, both doses of the herbal extract significantly decreased the postprandial insulin response, lowering both the positive area under the insulin curve and the adjusted peak insulin response (14% and 9%, respectively, for the 240 mg extract; 19% and 12%, respectively, for the 480 mg extract) in comparison with the control meal.

**Conclusions:** The extract of *Salacia oblonga* lowers acute glycemia and insulinemia in persons with type 2 diabetes after a high-carbohydrate meal. The results from this study suggest that *Salacia* may be beneficial to this population for postprandial glucose control. *Am J Clin Nutr* 2007;86:124–30.

**KEY WORDS** *Salacia oblonga*, postprandial glycemia, herbal extract, \(\alpha\)-glucosidase inhibitor

**INTRODUCTION**

Diabetes results in both public health and financial burdens to the society. In 2005, the estimated total prevalence of diabetes in the United States was 20.8 million persons, or 7.0% of the population (1). In addition, 20.9% of persons aged \(\geq 60\) y has the disease, which was the sixth leading cause of death in 2002 (1). The financial burden of this disease translates to an estimated $132 billion in medical expenditures, disability, and lost productivity in the United States (2). Because the risk of developing long-term complications can be dramatically reduced with appropriate glycemic control (3), food ingredients that can attenuate postprandial glucose in persons with diabetes would be useful.

One such ingredient, the root extract of *Salacia oblonga*, inhibits the breakdown of oligosaccharides and polysaccharides into monosaccharides by competitive inhibition of \(\alpha\)-glucosidase activity within the lumen of the intestinal tract. Historically, the *Salacia* plant has been used as part of the traditional Ayurvedic system of Indian medicine to treat diseases such as diabetes (4); currently, extracts of *Salacia* are consumed in commercial foods and food supplements in Japan for the treatment of diabetes and obesity. Yoshikawa et al (5) isolated active components of a *Salacia* extract and concluded that their mode of action was the inhibition of \(\alpha\)-glucosidase enzymes. Two compounds isolated from *Salacia* extracts, salacinol and kotalanol, inhibit the action of the small intestinal enzymes sucrase, maltase, and isomaltase (6). Therefore, naturally derived \(\alpha\)-glucosidase inhibitors may be powerful nutritional adjuncts for the treatment of diabetes mellitus by lowering postprandial blood glucose.

The potential genotoxicity and safety of *Salacia oblonga* extract (SOE) were evaluated with a standard battery of tests (reverse mutation assay, chromosomal aberrations assay, mouse micronuclear assay) recommended by the US Food and Drug Administration for food ingredients (7), and in a 92–93-d feeding study in rats with doses of 250, 1250, and 2500 mg \(\cdot kg^{-1} \cdot d^{-1}\) by oral gavage (8). SOE was determined not to be genotoxic, and the no observable adverse effect level was determined to be 2500 mg \(\cdot kg^{-1} \cdot d^{-1}\) after daily subchronic oral gavage administrations to rats (8).

Two previous studies in healthy adults were conducted to determine the effect of SOE on postprandial glycemia and insulinemia after a control meal (9, 10). Both studies showed efficacy on postprandial glycemia with 1000 mg SOE. The primary objective of this study was to compare the effect of 2 different doses of SOE on the glycemic and insulinemic response in patients with type 2 diabetes. Because previous experiments were conducted with healthy subjects, a logical next step was to test SOE within...
the population most likely to benefit from its effects on postprandial glycemia—patients with type 2 diabetes.

SUBJECTS AND METHODS

Subjects

This was a 2-center, randomized, double-blinded, 3-period, 3-treatment crossover study. Eighty-two subjects were enrolled, 41 per site, and 66 successfully completed the study according to protocol by meeting all entry criteria, complying with meal tolerance test preparation, having nonmissing data for the primary variable at all 3 treatment visits, and consuming all test products. For the 16 subjects who were not included in the protocol analysis, 8 of the subjects had missing or mistimed blood samples, 2 were ineligible, 2 had changes to their antihyperglycemic medications during the study, 1 withdrew before treatment visits, 1 was randomly assigned incorrectly, 1 withdrew after a visit because of the subject’s complaint of shakiness, and 1 withdrew at a treatment visit after symptoms of nausea and vomiting. The 66 protocol evaluable subjects (53 men and 13 nonpregnant, nonlactating women) had the following characteristics: mean (±SEM) age of 61.3 ± 1.1 y, weight of 87.3 ± 1.5 kg, and body mass index (BMI; in kg/m²) of 28.8 ± 0.4. The self-reported ethnicity of the subjects was 1 American Indian or Alaskan native, 7 African Americans, and 58 non-Hispanic whites. All subjects were patients with type 2 diabetes mellitus based on use of antihyperglycemic medication(s), but they did not use exogenous insulin for glucose control. These subjects were free from hepatic disease, active malignancy, end-stage organ failure, chronic infectious disease, or active metabolic disease (excluding diabetes) that would interfere with nutrient absorption, metabolism, or excretion. In addition, subjects did not have a recent infection, surgery, or corticosteroid treatment and did not have a significant cardiovascular event within 12 wk of study start. The study protocol was reviewed and approved by the Schulman Associates Institutional Review Board (Cincinnati, OH), and all enrolled subjects provided informed consent before the start of the study. The trial was conducted in accordance with the principles of Good Clinical Practice and the Declaration of Helsinki.

Study procedures

The 3 meal tolerance tests were administered in random order at least 3 d apart at each of the 2 clinical research centers. The 3 treatments were as follows: 1) control was 510 g (two 8-fl oz bottles) of a liquid meal replacement (chocolate Ensure; Ross Products Division, Abbott Laboratories, Columbus, OH) and 30.0 g maltodextrin (Polydextrose; Ross Products Division, Abbott Laboratories), 2) control + 240 mg SOE (agglomerated with 960 mg maltodextrin), and 3) control + 480 mg SOE (agglomerated with 1920 mg maltodextrin). The manufacture release criteria for the SOE lot 040304 was 46 μg/mL (IC₅₀) inhibition of α-glucosidase action. The SOE ingredient is a proprietary ethanol and water extract produced at Takama Co, Ltd (Yamaguchi, Japan), supplied through Tanabe Seiyaku Co, Ltd (Osaka, Japan). The control meal consisted of 110 g carbohydrate (55 g maltodextrin, 31 g sucrose, 25 g corn syrup), 12 g fat, 18 g protein, and 620 kcal. An unblinded product coordinator opened sealed envelopes containing the randomization sequences for subjects and prepared the test meals according to the assigned order. The test meals were prepared by mixing with a blender. The product coordinator was independent from all other study procedures. Subjects prepared for each meal tolerance test by consuming an average of ≥150 g carbohydrate/d for the 3 d before test visit, which was documented on a 3-d diet record. Subjects did not consume alcohol or participate in strenuous exercise for the 24 h before the test visits. Subjects fasted (except for water and antihyperglycemic medications) for 8–16 h before the meal tolerance tests, and subjects refrained from taking oral antihyperglycemic medications on the morning of test visits. Subjects consumed the test meals within 10 min, and all postprandial blood samples were drawn based on the time of start of meal consumption, which was considered time zero. Eight venous blood samples were drawn for glucose and insulin measurements at a central laboratory (Quest Diagnostics, Collegeville, PA) at the following times: baseline (before meal consumption) and 30, 45, 60, 90, 120, 150, and 180 min after the start of meal consumption. The serum samples were allowed to clot in serum separator tubes at room temperature and centrifuged at 1000 × g for 15 min at room temperature. Glucose was measured with the use of an enzymatic method (hexokinase glucose), and insulin was measured with the use of a radioimmunoassay procedure (DPC Immulite 2000 assay; DPC Biermann, Bad Nauheim, Germany).

Statistics

This was a randomized, double-blinded, 3-period, 3-treatment, crossover study conducted at 2 sites. A total of 82 subjects were randomly assigned to 1 of 6 treatment sequences, 1) A-B-C, 2) B-C-A, 3) C-A-B, 4) C-B-A, 5) A-C-B, and 6) B-A-C (A, control; B, control + 240 mg SOE; C, control + 480 mg SOE). Sixty-six subjects were included in the protocol analysis, which was the minimum number of subjects required in the power analysis before study start. With the use of data from a previous study, a sample size of 66 was calculated to have 83% power to detect a 20% of the control difference in treatment means with the use of a single-group repeated measures analysis of variance with a 0.05 significance level. Statistical software NQUERY ADVISOR 5.0 (Statistical Solutions, Los Angeles, CA) was used for the sample size estimation. Each variable was analyzed by using parametric or nonparametric (if declared nonnormal) 3-period, 3-treatment, crossover analysis. The parametric analysis was performed by using repeated measures analysis of variance with variance components covariance structure and Satterthwaite df with site, treatment, and period as fixed effects and subject nested within site as random effect. The 3 pairwise differences of least squares means of the treatments were tested with the use of Tukey-Kramer P value adjustments. If the parametric approach was determined to be inappropriate by the Shapiro-Wilk test for normality, then 3 pairwise treatment differences were analyzed with the use of signed rank test with stepdown Bonferroni (Holm) P value adjustments. A result was declared to be statistically significant if and only if a P value of an analysis was < 0.05. Statistical software SAS release 8.2 (SAS Institute Inc, Cary, NC) was used for the analyses.

RESULTS

Sodium

No significant differences were observed between treatments for baseline serum glucose concentrations. Baseline and postprandial values for both serum glucose and insulin are presented
TABLE 1
Serum glucose and insulin by treatment in all available subjects

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Control + 240 mg SOE</th>
<th>Control + 480 mg SOE</th>
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<td>Adjusted peak value (mg/dL)</td>
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<td>AUC (mg·min/dL)</td>
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<td>Positive AUC (mg·min/dL)</td>
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1 All values are x ± SEM; n = 61–79 (sample size differed according to data available for specific variables). SOE, Salacia oblonga extract; AUC, area under the curve. Values in a row with different superscript letters are significantly different, P < 0.05.

in Table 1 and Table 2 for all available subjects and the protocol evaluable set of subjects, respectively. Because the protocol evaluable analysis was the primary analysis and because results for both datasets are similar, only protocol evaluable data are presented in the data figures. The net changes in serum glucose concentrations are shown in Figure 1. Both doses of SOE significantly decreased the adjusted peak serum glucose response, 19% and 27%, respectively, for the 240-mg and 480-mg doses of SOE compared with the control meal alone (Figure 2). In addition, both doses of SOE significantly lowered the positive serum glucose area under the curve (AUC) for 0–180 min postprandial in comparison with the control meal, with a 14% reduction for the 240-mg dose and a 22% decrease for the 480-mg dose (Figure 3).

Serum insulin
No significant differences were observed in serum insulin across treatments at baseline. The net changes in serum insulin concentrations are shown in Figure 4. The 2 doses of SOE significantly lowered the adjusted peak serum insulin response 9% and 12%, respectively, for the 240-mg and 480-mg doses of SOE in comparison to the response by the control meal alone (Figure 5). Also, compared with the control meal, both doses of SOE significantly reduced the postprandial positive serum insulin AUC for 0–180 min, with a 14% decrease for the 240-mg dose and a 19% decrease for the 480-mg dose (Figure 6).

Safety and gastrointestinal tract tolerance
Twenty-three protocol evaluable subjects experienced adverse events during the study with all but one (moderate) event considered mild in severity. Sixteen of these subjects had symptoms related to gastrointestinal (GI) tolerance such as flatulence, belching, abdominal pain, nausea, and diarrhea which lasted ≤24 h after a test meal. Two subjects had GI symptoms after ingesting the control meal, 8 did after the 240-mg dose of SOE, and 12 subjects experienced GI symptoms after ingesting the 480-mg dose of SOE. Ten subjects had mild adverse events not related to GI symptoms, which included flu or sinus symptoms, bruised arms as a result of blood draws, study product aftertaste, vertigo, and pain or injury to the back or extremities.

DISCUSSION
This study presents the first published results on the effects of SOE on postprandial blood glucose in patients with diabetes.

TABLE 2
Serum glucose and insulin by treatment in protocol-evaluable subjects

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1 All values are x ± SEM; n = 55–66 (sample size differed according to data available for specific variables). SOE, Salacia oblonga extract; AUC, area under the curve. Values in a row with different superscript letters are significantly different, P < 0.05.
Both doses of the herbal extract had significant effects on postprandial glycemia and insulinemia after a high-carbohydrate meal. The results from the current study corroborate with previous data observed in healthy adult subjects (9, 10). However, the doses of extract for this study are much lower than the 1000-mg dose found to be efficacious in the 2 previous experiments. One possible reason for the difference is that a different extraction process was used for product in the current trial. The extract used in the 2 trials with healthy subjects was made by a hot water extraction process, and the SOE for the current study was created by an ethanol and water extraction, which may result in greater concentrations of active components. It is also possible that the glycemic responses of patients with diabetes were different from that of the healthy subjects of the 2 previous studies by evidence of the prolonged glucose excursion, resulting in larger differences between treatments for positive AUC. In addition, the difference in efficacious doses of SOE may be accounted for by the different amount of carbohydrate in the 2 control meals: 82 g carbohydrate (61% of energy intake) in the healthy subject study compared with 110 g carbohydrate (71% of energy intake) in the present study with patients with type 2 diabetes.

Postprandial glycemia is important for overall glycemic control. An expert panel concluded that postprandial hyperglycemia is a risk indicator for microvascular and macrovascular complications in both patients with type 2 diabetes and persons with impaired glucose tolerance (11). In a number of prospective clinical trials, postprandial glucose excursions were linked to increased mortality from cardiovascular disease, and decreasing

![Figure 1](https://academic.oup.com/ajcn/article-abstract/86/1/124/4633153)

**FIGURE 1.** Mean (±SEM) net change from baseline in serum glucose concentrations in patients with type 2 diabetes in the 3 meal groups (n = 62–66). SOE, *Salacia oblonga* extract. No statistical analyses were conducted for these variables.

![Figure 2](https://academic.oup.com/ajcn/article-abstract/86/1/124/4633153)

**FIGURE 2.** Mean (±SEM) adjusted peak serum glucose concentrations in patients with type 2 diabetes after the control meal and after the control meal plus 2 different doses of *Salacia oblonga* extract (SOE): 160 ± 7 mg/dL (n = 55) for the control meal, 130 ± 6 mg/dL (n = 63) for the control + 240 mg SOE meal, and 116 ± 6 mg/dL (n = 63) for the control + 480 mg meal. Values with different superscript letters are significantly different. Both doses of SOE significantly reduced the adjusted peak value compared with the control meal (P < 0.0001 for both), and the 480-mg dose significantly reduced the adjusted peak value compared with the 240-mg dose of SOE (P = 0.0050). The data were analyzed with the use of a mixed-model repeated-measures ANOVA with variance components covariance structure and Satterthwaite df with site, treatment, and visit as fixed effects and subject nested within site as a random effect. The 3 pairwise differences of least-squares means of the treatments were tested with the use of Tukey-Kramer P value adjustments.
postprandial glucose by therapeutic agents will decrease the progression of retinopathy, neuropathy, and nephropathy in patients with diabetes (12). So, when designing strategies for reducing the burden of diabetic complications, both a quantitative effect of hyperglycemia (postprandial hyperglycemia and glycated hemoglobin) and a qualitative component (glucose stability throughout the day) should be considered (13).

Therapy with \( /H_9251 \)-glucosidase inhibitors can benefit patients with diabetes beyond lowering postprandial glucose. For example, in the STOP-NIDDM (Study to Prevent NIDDM) trial, the group randomly assigned to acarbose not only had a reduction in body weight, BMI, waist and hip circumferences, systolic and diastolic blood pressures, blood triacylglycerols, and 2-h postprandial glucose during a 3-y period following subjects with impaired glucose tolerance but also experienced a significantly reduced incidence of cardiovascular events and hypertension (14). A meta-analysis of 7 long-term studies showed that \( /H_9251 \)-glucosidase inhibitors significantly reduce the risk of myocardial infarction or any cardiovascular event (15). Recent research testing SOE shows that the extract improves cardiac lipid metabolism and postprandial hyperlipidemia in rats and possesses activating properties for peroxisome-proliferator activated receptor-\( \alpha \) (16, 17). In addition, a study conducted in rats shows that \( Salacia reticulata \), another species of the \( Salacia \) plant,
inhibits lipase and has a mild antiobesity effect (18). Therefore, the application for /H9251-glucosidase inhibitors in patients with type 2 diabetes may extend beyond postprandial glucose control. Of 66 subjects, only 24% had GI symptoms because of study product consumption, with 12% of all subjects having symptoms with the 240-mg dose of SOE and 18% with the 480-mg dose. It is not uncommon for /H9251-glucosidase inhibitors to cause GI symptoms because of fermentation of undigested carbohydrates in the bowel (19). The difference in the number of GI events between the 2 doses of SOE is obvious and probably dose dependent. The /H9251-glucosidase inhibitor acarbose was found to cause a dose-dependent increase in the amount of carbohydrate entering the colon, which leads to an increase in colonic fermentation and GI symptoms (20). In addition, the large amount of carbohydrate in the control meal (110 g) for this study may have caused some of the intolerance. Daily carbohydrate consumption for persons

**Treatments**

**FIGURE 5.** Mean (±SEM) adjusted peak serum insulin concentrations in patients with type 2 diabetes after the control meal and after the control meal plus 2 different doses of *Salacia oblonga* extract (SOE): 65 ± 5 μIU/mL (n = 55) for the control meal, 59 ± 5 μIU/mL (n = 63) for the control + 240 mg SOE meal, and 57 ± 5 μIU/mL (n = 61) for the control + 480 mg SOE meal. Values with different superscript letters are significantly different. Both the 240-mg (n = 53) and 480-mg (n = 52) doses of SOE significantly reduced the adjusted peak value compared with the control meal (P = 0.0126 and P = 0.0021, respectively), but the values after the 2 doses of the extract did not differ significantly. The 3 pairwise treatment differences were analyzed with the use of a signed-rank test with stepdown Bonferroni (Holm) P value adjustments.

**FIGURE 6.** Mean (±SEM) positive area under the curve (AUC) for serum insulin concentration in patients with type 2 diabetes from 0 to 180 min after the control meal and after the control meal plus 2 different doses of *Salacia oblonga* extract (SOE): 8092 ± 889 μIU·min/mL (n = 65) for the control meal, 6986 ± 651 μIU·min/mL (n = 66) for the control + 240 mg SOE meal, and 6551 ± 601 μIU·min/mL (n = 66) for the control + 480 mg SOE meal. Values with different superscript letters are significantly different. Both the 240-mg and 480-mg doses of SOE significantly reduced the positive AUC compared with the control meal (P = 0.0183 and P = 0.0004, respectively), but the values after the 2 doses of the extract did not differ significantly. The 3 pairwise treatment differences were analyzed with the use of a signed-rank test with stepdown Bonferroni (Holm) P value adjustments.
with diabetes is typically <50% of energy intake (20), so the large quantity of carbohydrate, 71% of test meal calories, may have increased symptoms in study subjects. In addition to lowering the amount of carbohydrates consumed, another way to decrease GI symptoms would be to adjust the dose of SOE over a period of time, which is a common practice when administering α-glucosidase inhibitors (21).

Novel ingredients such as SOE may be ideal for medical nutritional therapy. Lifestyle modifications consisting of diet and exercise were shown to be effective for reducing macrovascular complications in patients with type 2 diabetes and for lowering relative risk of developing the disease in high-risk persons (22, 23). Although diabetes and its encompassing symptoms can be altered by diet and exercise, behavioral obstacles can prevent the occurrence of appropriate changes. Several situational obstacles for adults with diabetes were identified for dietary adherence, such as resisting temptation, eating out, feeling deprived, planning meals, social events, and so forth (24, 25). Nutritional adjuncts, including ingredients that lower postprandial glycemia, can provide more flexibility with meal planning and eating with family and friends. These adjuncts may also enable patients with type 2 diabetes to eat a greater variety of foods and carbohydrates with less restriction.

Historical and present uses of Salacia in India and Japan show that this herbal extract is used as a nutritional adjunct, either as a tea or supplement, taken with meals for its antidiabetic properties. SOE does lower postprandial glycemia in patients with type 2 diabetes. So, the long-term benefits of this herbal extract on glycemic control should be explored within this population to determine its value in the realm of nutritive therapy.

We thank the study subjects, study coordinators Pegi Deuss and Jane Gillingham, and numerous other study personnel for their dedication and invaluable contributions.

The author’s responsibilities were as follows—JAW: study design, review of the original data, and draft of manuscript; YSC: statistical design, final analysis of the data, and manuscript preparation; MJN and CJB (principal investigators): conduct of study at their clinical sites; and VAM: oversight of the study and editorial revisions of the manuscript. All authors reviewed and approved the final version of manuscript. JAW, YSC, and VAM have personal and financial conflicts of interest as employees of the sponsor company, Abbott Laboratories. MJN and CJB do not have personal or financial conflicts of interest.

REFERENCES