Effect of Cinnamon on Glucose and Lipid Levels in Non-Insulin-Dependent Type 2 Diabetes

Steve M. Blevins, md¹ Misti J. Leyva, ms, rdld² Joshua Brown, rdld² Jonelle Wright, phd^{3,4} Robert H. Scofield, md^{1,4,5} Christopher E. Aston, phd⁶

nterest in cinnamon as a potentially useful treatment for type 2 diabetes began with the discovery almost 20 years ago of cinnamon's insulin-sensitizing properties (1). Numerous in vitro and in vivo studies have elucidated cinnamon's effect on insulin signal transduction (2–6). A study in diabetic mice showed that cinnamon lowered blood glucose, total cholesterol, and triglyceride levels while raising HDL cholesterol levels (7).

The first clinical trial to evaluate the effect of cinnamon in individuals with type 2 diabetes was conducted in Pakistan (8). It showed that cinnamon powder (Cinnamomum cassia), taken over a 40day period, reduced mean fasting serum glucose (18-29%), triglyceride (23-30%), LDL cholesterol (7–27%), and total cholesterol (12-26%) levels. Three different doses of cinnamon were administered: 1, 3, and 6 g daily. All were equally effective. These findings led to widespread cinnamon use, although no study had yet evaluated the effects of cinnamon in Western diabetic populations with likely differences in diet, BMI, baseline glucose levels, and prescribed medication. We report the first U.S. study examining the effects of cinnamon on glucose and lipid levels in subjects with type 2 diabetes.

RESEARCH DESIGN AND

METHODS — Participants were recruited through e-mail announcements to campus employees and through an article in the local newspaper. Individuals of any age with type 2 diabetes, based on criteria from the American Diabetes Association (9), were eligible for the study. Exclusion criteria included insulin use, consumption of nondietary cinnamon supplements, A1C <6.0%, and acute illness. Subjects were withdrawn if any of the following medicines were initiated, discontinued, or adjusted during the study: sulfonylureas, meglitinides, metformin, thiazoledinediones, α -glucosidase inhibitors, exenatide, hydromethylglutaryl-CoA reductase inhibitors, ezetimibe, niacin, or fibric acid derivatives.

Enrolled subjects were stratified by sex and randomized to receive either cinnamon (*C. cassia*) or placebo (wheat flour). Investigators and subjects were blinded to group assignment and to capsule content. Each capsule contained 500 mg product. Subjects were instructed to ingest one capsule with breakfast and one with dinner for 3 months. Compliance was assessed by capsule count. Follow-up visits were scheduled at 1, 2, and 3 months.

Fasting glucose, cholesterol (total, LDL, and HDL), triglyceride, and insulin levels were measured at each visit. A1C

From the ¹Department of Medicine, University of Oklahoma, Oklahoma City, Oklahoma; the ²Department of Nutritional Sciences, University of Oklahoma, Oklahoma City, Oklahoma; the ³Department of Geriatrics, University of Oklahoma, Oklahoma City, Oklahoma; the ⁴Veterans Affairs Medical Center, Oklahoma City, Oklahoma; the ⁵Arthritis and Immunology Program, Oklahoma Medical Research Foundation, Oklahoma City, Oklahoma; and the ⁶Department of Pediatrics, University of Oklahoma, Oklahoma.

Address correspondence and reprint requests to Steve Blevins, MD, General Clinical Research Center, O'Donoghue Research Building, 1122 NE 13th St., Suite 150, Oklahoma City, OK 73104. E-mail: steveblevins@ouhsc.edu.

Received for publication 16 January 2007 and accepted in revised form 29 May 2007.

Published ahead of print at http://care.diabetesjournals.org on 11 June 2007. DOI: 10.2337/dc07-0098. Clinical trial reg. no. NCT00237640, clinicaltrials.gov.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

© 2007 by the American Diabetes Association.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

was measured at baseline and at 3 months. Dietary patterns were monitored monthly using a 3-day food journal.

An intention-to-treat analysis was conducted. Outcomes were analyzed using a general linear model for the threeway ANOVA (two treatments \times 2 sexes \times 4 visits) with repeated measures on the time factor. Significance was set at 0.05. We estimated that changes ranging from 11.5 (A1C) to 24.7% (triglycerides) could be detected with 80% power. For each variable, detectable change was within the range found by Khan et al. (with the exception of A1C, which was not measured by Khan et al.) (8).

RESULTS — Of 77 individuals with type 2 diabetes who visited the research center, 17 were excluded (14 had A1C <6.0%, and 3 had acute health problems). The remaining 60 were randomized. Of these, 43 completed the study. Reasons for noncompletion were change in diabetes or lipid-lowering medicine (5), initiation of nondietary cinnamon outside study protocol (1), motor vehicle accident (1), relocation (1), inconvenience (1), dissatisfaction with study (1), and unknown (7). Of the 17 who did not complete the study, 9 were in the cinnamon group and 8 were in the placebo group.

A total of 58 subjects completed the initial visit, of whom 30 had been randomized to the cinnamon group and 28 to the placebo group. One subject (in the cinnamon group) was excluded because of a major change in dietary pattern discovered before unblinding, leaving 57 subjects included in this intention-totreat analysis.

Adherence was high. The two groups differed only with respect to age (63.6 years in the cinnamon group vs. 58.0 years in the placebo group, P = 0.04). Fifty-one percent of the subjects were women. The ethnic mix was varied: 68% Caucasian, 16% Native American, 7% African American, 4% Hispanic, 2% Asian, and 3% unknown. In the cinnamon group, 77% of the subjects were taking diabetes medication, as was 91% of those in the placebo group; 55% of the cinna-

National Center for Research Resources, National Institutes of Health.

References

- 1. Khan A, Bryden NA, Polansky MM, Anderson RA: Insulin potentiating factor and chromium content of selected foods and spices. Biol Trace Elem Res 24:183-188. 1990
- 2. Karalee JJ, Anderson RA, Graves DJ: A hydroxychalcone derived from cinnamon functions as a mimetic for insulin in 3T3-L1 adipocytes. J Am Coll Nutr 20:327-336, 2001
- 3. Imparl-Radosevich J, Deas S, Polansky MM, Baedke DA, Ingebrutsen TS, Anderson RA, Graves DJ: Regulation of PTP-1 and insulin receptor kinase by fractions from cinnamon: implications for cinnamon regulation of insulin signaling. Horm Res 50:177-182, 1998
- 4. Qin B, Nagasaki M, Ren M, Bajotto G, Oshida Y, Sato Y: Cinnamon extract (traditional herb) potentiates in vivo insulinregulated glucose utilization via enhancing insulin signaling in rats. Diabetes Res Clin Pract 62:139-148, 2003
- 5. Qin B, Nagasaki M, Ren M, Bajotto G, Oshida Y, Sato Y: Cinnamon extract prevents the insulin resistance induced by a highfructose diet. Horm Metab Res 36:119-125.2004

Downloaded from http://diabetesjournals.org/care/article-pdf/30/9/2236/596984/zdc00907002236.pdf by guest on 07 August 2022

- 6. Lee JS, Jeon SM, Park EM, Huh TL, Kwon OS, Lee MK: Cinnamate supplementation enhances hepatic lipid metabolism and antioxidant defense systems in high cholesterol-fed rats. J Med Food 6:183-191, 2003
- 7. Kim SH, Hyun SH, Choung SY: Anti-diabetic effect of cinnamon extract on blood glucose in db/db mice. J Ethnopharmacol 104:119-123, 2006
- 8. Khan A, Safdar M, Khan MMA, Khattak KN, Anderson RA: Cinnamon improves glucose and lipids of people with type 2 diabetes. Diabetes Care 26:3215-3218, 2003
- 9. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 26 (Suppl. 1):S5-S20, 2003

Data are means ± SD unless otherwise indicated.

mon group and 48% of the placebo group were taking lipid-lowering medicine. The two groups did not differ with respect to BMI, A1C, cholesterol, triglyceride, or insulin levels (Table 1).

There were no significant differences between the cinnamon and placebo groups in the change in any measure from baseline to 3 months (Table 1) or from baseline to 1 and 2 months (data not shown). Similarly negative results were obtained when the analysis was restricted to the 42 participants who completed the study.

CONCLUSIONS — This is the first U.S. study to evaluate the effects of cinnamon on blood glucose and lipid levels in individuals with type 2 diabetes. We found that cinnamon taken at a dose of 1 g daily for 3 months produced no significant change in fasting glucose, lipid, A1C, or insulin levels

Khan et al. (8) showed that three different doses of cinnamon-1, 3, and 6 g daily—were equally effective at lowering fasting glucose, total cholesterol, LDL cholesterol, and triglyceride levels in subjects with type 2 diabetes. Our study population differed from theirs with respect to initial fasting glucose (139 vs. 232 mg/ dl) and triglyceride (144 vs. 215 mg/dl) levels. Roughly three-fourths of our subjects were taking metformin, more than one-third were taking a thiazoledinedione, and about one-half were taking an hydroxymethylglutaryl-CoA reductase inhibitor-medicines not taken by subjects in their study. Khan et al. did not provide data on diet, BMI, ethnic mix, or A1C, precluding further comparison of our studies.

We conclude that the effects of cinnamon differ by population. Studies should be conducted to determine how specific variables (diet, ethnicity, BMI, glucose levels, cinnamon dose, and concurrent medication) affect cinnamon responsiveness. Until then, cinnamon cannot be generally recommended for treatment of type 2 diabetes in an American population.

Acknowledgments- This study was supported solely by grant M01 RR14467 from the

Table 1—Baseline measurements and 3-month change

Variable	Cinnamon	Placebo	Р
n	29	28	_
BMI (kg/m ²)	32.5 ± 1.7	32.0 ± 1.5	0.83
$\Delta BMI (kg/m^2)$	-0.2 ± 0.1	0.0 ± 0.2	0.34
Glucose (mg/dl)	132.9 ± 9.3	144.7 ± 10.4	0.40
Δ Glucose (mg/dl)	9.8 ± 5.9	0.3 ± 9.0	0.38
A1C (%)	7.2 ± 0.3	7.1 ± 0.2	0.94
ΔA1C (%)	0.2 ± 0.1	0.1 ± 0.2	0.64
Total cholesterol (mg/dl)	170.2 ± 8.1	176.3 ± 8.0	0.60
Δ Total cholesterol (mg/dl)	0.8 ± 4.3	-2.9 ± 6.3	0.63
HDL cholesterol (mg/dl)	43.9 ± 1.5	-2.2 ± 1.7	0.45
Δ HDL cholesterol (mg/dl)	0.8 ± 1.0	-1.0 ± 1.3	0.28
LDL cholesterol (mg/dl)	101.5 ± 6.7	105.1 ± 6.7	0.70
Δ LDL cholesterol (mg/dl)	-2.0 ± 3.1	-3.0 ± 5.6	0.87
Triglycerides (mg/dl)	132.4 ± 15.5	155.7 ± 22.8	0.40
Δ Triglycerides (mg/dl)	9.6 ± 13.5	16.1 ± 19.1	0.78
Insulin (IU/ml)	12.9 ± 1.3	11.8 ± 1.6	0.61
Δ Insulin (IU/ml)	16 ± 11	57 + 32	0.25