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Almond ingestion at mealtime reduces postprandial glycemia and chronic ingestion reduces hemoglobin A_{1c} in individuals with well-controlled type 2 diabetes mellitus

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ABSTRACT

Cohort studies are equivocal regarding a relationship between regular nut consumption and reduced risk of type 2 diabetes mellitus. Although acute trials show reductions in postprandial glycemia in healthy individuals ingesting 60 to 90 g almonds, trials have not been conducted using a single serving of almonds (28 g) in individuals with type 2 diabetes mellitus. This randomized crossover trial examined the impact of one serving of almonds at mealtime on postprandial glycemia, insulinemia, and plasma glucagon-like peptide–1 in healthy individuals and individuals with type 2 diabetes mellitus. On 2 occasions separated by at least 1 week, 19 adults (including 7 adults with type 2 diabetes mellitus) consumed a standardized evening meal and fasted overnight before ingesting the test meal (bagel, juice, and butter) with or without almonds. A small pilot study (6-7 subjects per group) was also conducted to observe whether chronic almond ingestion (1 serving 5 d/wk for 12 weeks) lowered hemoglobin A_{1c} in individuals with type 2 diabetes mellitus. A standard serving of almonds reduced postprandial glycemia significantly in participants with diabetes (–30%, $P = .043$) but did not influence glycemia in participants without diabetes (–7%, $P = .638$). Insulinemia and glucagon-like peptide–1 at 30 minutes postmeal were not impacted by almond ingestion for either group. In the pilot study, regular almond ingestion for 12 weeks reduced hemoglobin A_{1c} by 4% ($P = .045$ for interaction) but did not influence fasting glucose concentrations. These data show that modest almond consumption favorably improves both short-term and long-term markers of glucose control in individuals with uncomplicated type 2 diabetes mellitus.

Although regular nut consumption has consistently been demonstrated to reduce risk of heart disease in both men and women in large prospective cohort studies [1,2], the data are equivocal regarding a relationship between regular nut consumption and reduced risk of type 2 diabetes mellitus (T2D). In a Nurses' Health Study cohort (N = 83 818), relative risks (RRs) analysis adjusted for known confounders and dietary variables indicated that women consuming a serving of nuts at least 5 times a week were at a significantly reduced risk of T2D as compared with those who never/almost never consumed nuts (RR = 0.71) [3]. However, analyses of cohorts of the Physicians' Health Study (N = 20 224) and the Iowa

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Women's Health Study (N = 35 988) did not show significant beneficial effects of frequent nut consumption on risk of T2D (RR = 0.87 and 1.51, respectively) [4,5].

Intervention trials also show conflicting results regarding the influence of nut consumption on diabetes risk factors. Using a single-group study design, Lovejoy et al [6] examined the change in whole-body insulin sensitivity in healthy men (n = 10) and women (n = 10) instructed to consume 100 g almonds daily for 4 weeks. Following the diet intervention, insulin sensitivity was modestly improved (19%, $P = .09$) in the women only. In the same report, investigators observed no change in hemoglobin A_{1c} (HbA_{1c}) in 34 individuals with T2D consuming almond-enriched (57-113 g almonds per day) or control diets for 4 weeks in a randomized, double-blinded crossover fashion [6]. Tapsell et al [7] observed a significant group × time interaction for fasting insulin concentrations in 50 individuals with T2D randomized to receive low-fat dietary advice ±30 g walnuts daily for 1 year. Participants ingesting the walnuts displayed a 21% reduction in fasting insulin after 12 months; yet, fasting glucose and HbA_{1c} were not significantly reduced by walnut ingestion compared with control values. In acute trials, postprandial glycemia and insulinemia were reduced 40% to 50% in healthy individuals ingesting 60 or 90 g almonds with white bread [8,9].

The favorable impact of regular nut ingestion on cardiovascular risk suggests that including nuts in diabetic diet plans would benefit long-term health outcomes in these individuals, but continued investigation is required to clarify the direct impact of nut ingestion on diabetic risk factors. This investigation examined whether acute almond consumption augments the release of the incretin hormone glucagon-like peptide–1 (GLP-1) because this would provide a mechanism for the reported insulin-sensitizing effect of nut ingestion. To date, there is one report linking almond ingestion with GLP-1 release [10]. Glucagon-like peptide–1 is a 30–amino acid glucoregulatory agent that stimulates insulin secretion after meal ingestion by increasing the gene expression and biosynthesis of insulin. Glucagon-like peptide–1 is released from the L-cells of the ileum and colon within minutes of food consumption and remains in circulation for up to 3 hours [11].

The study purpose was to determine if acute almond ingestion altered postprandial glycemia and insulinemia, and plasma GLP-1 concentrations after meal ingestion in healthy individuals and in individuals with T2D. The impact of chronic almond ingestion (5×/wk for 12 weeks) on markers of glycemic control (HbA_{1c}, and fasting blood glucose and insulin) was also assessed in individuals with T2D in a small pilot study.

1. Research design and methods

1.1. *Subjects*

Healthy adults without T2D (2 men and 11 women) and adults with diagnosed T2D (4 men and 3 women) participated in the trial. Based on data from previous reports [8,9], 8 to 11 participants are required for 80% power using a 5% significance level to achieve a 40% reduction in postprandial glycemia. Participants were recruited from a campus population and the surrounding communities and did not report a history of peanut and/or tree nut allergy or current cigarette use, and were not pregnant or lactating. Participants with T2D had their condition diagnosed by a physician at least 1 year before the start of the trials and had not been prescribed insulin. None of the participants reported active disease conditions aside from T2D; and all prescription medication use by participants, including oral hypoglycemic agents, remained consistent during the trials. Only one individual with diabetes was being treated with incretin-based therapy (Januvia, Merck & Co., Inc, Whitehouse Station, NJ); however, this individual was also one of the 2 individuals with T2D whose GLP-1 data were not available. Written informed consent was obtained from all participants, and the study was approved by the Institutional Review Board at Arizona State University.

1.2. *Experimental design*

Participants were instructed to cease all nut consumption at least 1 week before and during the trial. Testing took place on 2 mornings 1 week apart. The day before testing, participants did not consume caffeine or perform moderate-to-intense physical activity. A standardized dinner meal (sub sandwich, chips, soda) was consumed followed by a 12-hour overnight fast. Testing was scheduled in the early morning hours. Upon arrival at the test site, fasting blood was collected; and participants were randomized to a treatment meal with or without almonds. The treatment meal without almonds was composed of a white bagel (260 kcal; 54 g carbohydrate, 1 g fat), berry juice (100 kcal; 24 g carbohydrate, 0 g fat), and butter (264 kcal; 0 g carbohydrate, 29 g fat). The treatment meal with almonds included 28 g almonds (170 kcal; 5 g carbohydrate and 15 g fat), and the butter amount was reduced to compensate for the fat content of the almonds (100 kcal; 0 g carbohydrate and 11 g fat). Hence, the 2 treatment meals were isocaloric with similar carbohydrate and fat content. Meals were consumed in 10 minutes under observation; and, when appropriate, the almonds were ingested before the consumption of the buttered bagel and juice. A venous blood draw was performed at 0 (fasting), 30, and 60 minutes postmeal; and capillary blood samples were collected at 0, 30, 60, 90, and 120 minutes postmeal. This entire protocol was repeated after 1 week for the crossover treatment. All 20 subjects completed the 2-day crossover trial with an at least 1 week washout period; however, one participant without diabetes was removed from data analyses because this participant experienced an episode of emesis during one of the test periods.

For the small pilot study, 13 individuals with T2D were screened for nut consumption and did not typically eat nuts or nut products more than 4×/wk. During the 12-week parallel-arm trial, all participants were instructed to restrict any nut consumption to less than 2×/wk with the exception of the trial almond prescription. Participants completed a 2-day diet record at baseline and at week 12. Participants were stratified by sex and body mass index (BMI), randomized to the almond group or to the cheese group (control), and instructed to consume their food prescription (1 oz almonds or 2 cheese sticks) 5 d/wk. In an attempt to address possible bias, we told the participants that the trial purpose was to examine the impact of nut and dairy ingestion on blood glucose and

cholesterol parameters because there is evidence that both foods improve these markers.

Almonds used in the pilot study were donated by the California Almond Board in 1-oz airtight packaging (1-oz portion: 173 kcal, 4.6 g carbohydrate, 14.6 g fat), and cheese sticks were purchased from a commercial grocer (Sorrento Lactalis, Buffalo, NY; 2-stick portion: 160 kcal, 0 g carbohydrate, 12 g fat). Participants were instructed to mark compliance on a 12-week calendar. A fasting venous blood sample was collected at baseline and at weeks 6 and 12.

1.3. *Blood analyses and anthropometric measurements*

A calibrated OneTouch glucometer (LifeScan, Milpitas, CA) was used to determine capillary blood glucose in the crossover trial; the same glucometer was used for each subject during the study. Plasma was extracted from venous blood and analyzed for insulin (time points: 0, 30, and 60 minutes postmeal) and GLP-1 (30 minutes postmeal) using radioimmunoassays (LINCO Research, St Charles, MO). Immediately after blood was drawn, the DPP-IV inhibitor was added to the GLP-1 test tube within 30 seconds to prevent GLP-1 degradation. Hemoglobin A_{1c} (DCA200+; Siemens Healthcare Diagnostics, Deerfield, IL) was determined on fasting venous blood. In the pilot study, HbA_{1c} and plasma insulin were measured as described above; and venous blood glucose and lipids were analyzed by automated procedures (Piccolo; Abaxis, Union City, CA). Body weight (in kilograms; Tanita scale, Model TBF-300A, Tanita, Arlington Heights, IL) and height (in meters, calibrated stadiometer) were measured, and BMI was calculated (in kilograms per square meter).

1.4. *Statistical analyses*

The data were analyzed using PASW Statistics 18.0 (Predictive Analytics SoftWare Statistics package; IBM Corp., Somers, NY, 2009). Descriptive data at baseline were assessed using independent *t* tests. Postprandial glycemia and insulinemia were calculated as the incremental area under the curve (iAUC) using the trapezoidal rule. The maximum stimulated glucose concentration (C_{max}) during the 120-minute test was calculated as well as the time to maximum glucose concentration (t_{max}). Nonparametric analysis (the Wilcoxon signed rank test) was used to assess treatment differences in data that were not normally distributed; otherwise, a paired *t* test was used. For some analyses, sample sizes are reduced because of the inability to successfully collect venous blood samples (1 individual with T2D and 1 individual without diabetes), the removal of an outlying value (1 individual without diabetes for the insulin analysis only), and procedural complications with the GLP-1 assay (2 individuals with T2D). Data for the pilot study were normally distributed with the exception of HbA_{1c}, which was log transformed before analysis. General linear model repeated-measures analysis of variance was used to assess time and interaction factors controlling for sex (all analyses) and change in body weight (energy and blood factors analyses only). All results are expressed as means ± SEM, and a P value ≤ .05 was considered significant.

2. Results

Participants with T2D were older than the participants without diabetes (66 ± 3 and 53 ± 3 years); and their body weight, fasting glucose and insulin concentrations, and HbA_{1c} values were significantly higher than those for the participants without diabetes (98.9 ± 10.3 and 77.5 ± 4.2 kg, 7.3 ± 0.5 and 5.2 ± 0.2 mmol/L, 19.4 ± 3.3 and 6.3 ± 1.3 μIU/mL, and 6.7% ± 0.2% and 5.6% ± 0.1%). In addition, postprandial glycemia under control conditions was 2.5 times greater for the participants with T2D as compared with the participants without diabetes (P = .022; Table 1). Almond ingestion with the test meal reduced postprandial glycemia significantly in participants with T2D (–30%, P = .043) but did not significantly influence this parameter in participants without diabetes (–7%, P = .638) (Table 1). The maximum stimulated glucose concentration was reduced by almond ingestion in individuals with T2D but not in healthy adults (Table 1); however, the time to maximum glucose concentration was not significantly impacted by almond ingestion in either group. Mealtime insulinemia was not impacted by

Table 1 – Postmeal GLP-1 concentrations, iAUC for blood glucose and plasma insulin, C_{max}, and t_{max} under 2 treatment conditions (control and almond) in nondiabetic participants (n = 12) and participants with T2D (n = 7)

	Nondiabetic			Diagnosed T2D		
	Control	Almond	*P*	Control	Almond	*P*
iAUC glucose,[a] mmol*min/L	120 ± 21	112 ± 22	.638	429 ± 75*	299 ± 63	.043
C_{max}, mmol/L	7.3 ± 0.4	7.3 ± 0.4	1.00	12.6 ± 1.5*	11.3 ± 1.0	.046
t_{max}, min	35.0 ± 3.4	57.5 ± 10.1	.071	85.7 ± 7.8	81.4 ± 12.6	.655
iAUC insulin,[b] μIU*min/mL	52.0 ± 9.6	43.9 ± 6.8	.167	63.9 ± 10.0	57.0 ± 13.0	.430
GLP-1,[c] pmol/L	41.4 ± 9.8	43.1 ± 11.0	.790	52.3 ± 14.5	66.8 ± 22.4	.465

Means ± SE; *P* value assesses within-group differences for control and almond treatments for glycemia (Wilcoxon signed rank test), insulinemia (paired *t* test), and GLP-1 (Wilcoxon signed rank test) analyses.

* Significant difference between groups for control treatment (P < .05; Mann-Whitney *U* test).

[a] iAUC calculated 2 hours postmeal.

[b] iAUC calculated 1 hour postmeal, and sample sizes are reduced: n = 10 and 6 for nondiabetic participants and diagnosed T2D participants, respectively.

[c] Means represent 30-minute postmeal concentrations, and sample sizes are reduced: n = 11 and 4 for nondiabetic participants and diagnosed T2D participants, respectively.

Table 2 – Pilot study: characteristics of participants at baseline and at trial week 12

	Control baseline	Almond baseline	Control week 12	Almond week 12	P
Sex, M/F	4/3	3/3			
Age, y	66.0 ± 3.3	66.0 ± 3.3			
Weight, kg	105.1 ± 11.2	96.1 ± 8.9	104.9 ± 11.1	93.1 ± 8.1	.083
BMI, kg/m^2	36.7 ± 3.6	32.6 ± 2.3	36.8 ± 3.6	31.3 ± 2.1	.047
Body fat, %	42.5 ± 4.5	43.8 ± 2.8	41.1 ± 4.6	42.5 ± 2.7	.853
Energy,[a] kcal/kg	20.5 ± 4.1	15.1 ± 1.2	19.2 ± 4.4	14.7 ± 2.3	.674
Fasting glucose, mmol/L	7.0 ± 0.4	7.5 ± 0.7	7.1 ± 0.4	7.1 ± 0.8	.305
Fasting insulin, μIU/mL	14.7 ± 3.3	18.8 ± 6.6	20.3 ± 4.2	20.2 ± 5.8	.610
Hemoglobin A_{1c}, %	6.6 ± 0.1	7.1 ± 0.2	6.6 ± 0.1	6.8 ± 0.3	.045
Total cholesterol, mmol/L	4.2 ± 0.2	4.0 ± 0.2	3.9 ± 0.2	4.1 ± 0.1	.681
LDL cholesterol, mmol/L	2.2 ± 0.2	1.6 ± 0.3	2.1 ± 0.1	1.8 ± 0.2	.918
Triglyceride, mmol/L	1.5 ± 0.4	2.3 ± 0.7	1.5 ± 0.3	2.1 ± 0.5	.945

Mean ± SE; means did not differ between groups at baseline (independent *t* test analyses with the exception of sex, which was assessed using χ^2 analysis). *P* values are for group × time interaction (general linear model repeated-measures analysis of variance) controlling for sex (all analyses) and change in body weight (energy and blood factors analyses only). All data were normally distributed with the exception of hemoglobin A_{1c}, which was log transformed before analysis. LDL indicates low-density lipoprotein.

[a] Energy intake at baseline and at week 12 based on 2-day diet record averages at each time point.

almond ingestion in participants with T2D or in participants without diabetes (Table 1). Glucagon-like peptide–1 concentrations at 30 minutes postmeal were not significantly impacted by almond ingestion for either group (Table 1).

In the pilot study, baseline data did not differ between treatment groups (Table 2). Adherence to the diet intervention was excellent with all subjects reporting 100% compliance. After the intervention was complete, the only blood marker that changed significantly between treatment groups over the 12-week trial was HbA_{1c} (–4% vs +1% for the almond and control groups respectively; $P = .045$) (Table 2). Chronic almond ingestion was associated with a 4% reduction in BMI as compared with no change in the control group ($P = .047$) (Table 2). Energy intakes did not differ within groups at baseline and 12 weeks.

3. Discussion

These data demonstrate that the ingestion of 1 oz (28 g) almonds before a high-starch meal lowered postprandial glycemia by 30% in individuals with T2D but did not impact this response in healthy individuals without T2D. Similarly, almond ingestion reduced the maximum stimulated glucose concentration in individuals with T2D but not in healthy individuals. To our knowledge, the antiglycemic effect of almond ingestion at mealtime has not been examined in populations with T2D. Others report significant reductions in postprandial glycemia with almond ingestion in healthy volunteers but only at high intakes of almonds (60-90 g) [8,9]. Josse et al [9] observed a significant 43% reduction in the peak 2-hour postprandial glucose concentration in healthy adults when 90 g of almonds was added to a bread meal containing 50 g carbohydrate (257 kcal). Lower amounts of almonds (30 and 60 g) did not significantly impact postprandial glycemia in this trial. These investigators attributed the antiglycemic effect of 90 g of almonds to a theoretical reduction in the gastric emptying rate, a consequence of the additional fat and protein energy contributed by the almonds (+506 kcal). Alternatively, they implicated the flavonoids in almonds that have been reported to inactivate amylase, the enzyme that hydrolyzes dietary starch.

Because the energy and fat contents of the meals were controlled in the present study, a change in mealtime energy would not have contributed to the observed 30% reduction in postprandial glycemia. Furthermore, the reduction in postprandial glycemia was noted with the ingestion of a much smaller quantity of almonds (28 vs 90 g), but only in individuals with T2D. Because salivary amylase concentrations in T2D are elevated in comparison to control concentrations by as much as 27% [12-14], it is intriguing to speculate that the flavonoids in almonds may be serving to moderate the elevated salivary amylase action in T2D. Salivary amylase accounts for a small portion of total gastrointestinal starch digestion (~15%) in healthy adults; but in the diabetic state, salivary amylase likely has a more significant role in starch digestion because pancreatic amylase activity is reduced 28% to 35% [15,16]. In healthy adults, greater quantities of almonds may be required to effectively impact starch digestion. Using molecular modeling, Lo Piparo et al [17] demonstrated that flavonoids bind to the active site of amylase, exerting varying degrees of enzyme inhibition. Almonds are source of quercetin, one of the strongest inhibitors of amylase action [17,18].

In a recent randomized, double-blind, crossover study, Li et al [19] reported significant reductions in fasting glucose and insulin concentrations in individuals with T2D adhering to a low-fat diet for 4 weeks that included 60 g almonds. In this study, the almonds were incorporated into entrées and desserts or were consumed as snacks; and participant meal plans were adjusted to maintain body weight over the course of the study. Using a similar protocol, Lovejoy et al [6] examined the effects of low-fat vs high-fat diets enriched with almonds (57 or 113 g/d) on blood lipids and glucose tolerance in men and women with T2D who were not taking insulin or medications to lower cholesterol. After 4 weeks, the almond diets did not alter fasting glucose, HbA_{1c}, or responses to an oral glucose tolerance test relative to control diets. In adults with prediabetes instructed to follow a reduced-calorie diet, almond ingestion (60 g/d) for 16 weeks reduced fasting insulin significantly compared with the control diet; but fasting glucose concentrations were not altered [20]. The

pilot data reported herein show a modest but significant reduction in HbA_{1c} for the almond diet after 12 weeks as compared with the control diet (–0.3%), but fasting glucose and insulin concentrations were not altered by diet treatment. The conflicting results from these trials may relate to differences in patient populations, differences in diet plans, differences in the amount of almonds consumed daily (28, 60, or 113 g), and/or differences in trial duration (4 or 12 weeks). Because the measurement of HbA_{1c} assesses the degree of glycemia over a period of 2 to 3 months, study durations under 10 weeks may not be of adequate duration to show changes in HbA_{1c}.

The almond-induced reduction in postprandial glycemia provides a rationale for the modest reduction in HbA_{1c} noted in the pilot study. Postprandial glycemia is responsible for 50% to 70% of the overall diurnal hyperglycemia when HbA_{1c} values are less than 8.5 [21], and postprandial glycemia is significantly correlated with HbA_{1c} in well-controlled diabetes [22]. Furthermore, because postprandial glycemia is closely associated with cardiovascular disease risk, even when HbA_{1c} is less than 7 [23], the 30% reduction in postprandial glycemia has physiological relevance beyond glucose management. Numerous reviews have detailed the research correlating nut consumption with a lower risk of cardiovascular disease [2,24]. Much of the evidence linking nuts to reduced cardiovascular disease risk centers on the favorable plasma lipid profile noted in individuals who regularly consume nuts. The data herein offer an additional mechanism to explain the link between nut consumption and reduced cardiovascular disease risk in diabetes: reduced postprandial glycemia.

The impact of regular almond consumption on HbA_{1c} (–0.3%) was modest but comparable to that noted for incretin-based therapies (–0.28% to –1.14%) [25] or acarbose therapies (~–0.4%) [26], 2 pharmaceutical regimens that reduce postprandial hyperglycemia. These comparisons are noteworthy because acarbose therapy reduced the progression of intima media thickness and the incidence of cardiovascular events in well-controlled clinical trials [27,28]. Although the mechanisms of action of these antiglycemic therapies are established, little data are available to explain the observed blood glucose–lowering effect of almonds. This investigation explored the possibility that almond ingestion at mealtime enhances the release of the incretin hormone GLP-1 as proposed by Cassady et al [10], who postulated that the high unsaturated fat and protein content of almonds would stimulate GLP-1 secretion. In the participants without diabetes, GLP-1 concentrations at 30 minutes postmeal were not affected by almond ingestion as compared with the control treatment (43.1 ± 11 and 41.4 ± 9.8 pmol/L, respectively; $P = .790$). Furthermore, insulin concentrations at 30 minutes postmeal were lower with almond ingestion compared with the control treatment, additional evidence that opposes a direct relationship between almond ingestion and GLP-1 secretion. In a well-controlled, 4-day inpatient cross-over trial, Brennan et al [29] was unable to show an effect of walnut ingestion (48 g daily as a breakfast shake) on GLP-1 concentrations in 15 individuals with the metabolic syndrome; however, walnut consumption significantly increased satiety on study days 3 and 4, providing a theoretical link between regular nut ingestion and reduced body weight.

The GLP-1 data from the participants with T2D were inconclusive because of the reduced sample size; however, GLP-1 concentrations at 30 minutes postmeal were not reduced compared to the healthy adult values as has been reported by others [30]. Consistent with the present data, Vollmer et al [11] did not observe any impairment in GLP-1 secretion in well-controlled T2D and suggest that impairment in postprandial GLP-1 secretion may only manifest with longer diabetes duration and poor glycemic control as indicated by elevated HbA_{1c} levels. Although these data do not show an acute effect of almond ingestion on GLP-1 secretion, future studies examining dietary strategies for promoting GLP-1 activity should use patient populations with poor glycemic control and documented reduced GLP-1 secretion.

Almonds are high in monounsaturated fatty acids and have been used successfully in clinical trials to demonstrate hypoglycemic and hypocholesterolemic effects of high–monounsaturated fatty acids diets [31]. In the 12-week pilot study, blood lipids were not impacted by almond ingestion. However, nearly one half of the participants in the chronic trial were taking prescription medications to lower cholesterol (43% and 50% for the almond and control groups, respectively); and baseline total and low-density lipoprotein cholesterol concentrations (4.0-4.2 and 1.6-2.2 mmol/L, respectively) were in the desirable ranges for participants when the study started. Fasting glucose was reduced 5% in the almond group and rose slightly in the control group after 12 weeks, but this difference between groups was not significant ($P = .305$). The pilot study was limited by the small sample size, and it is possible that uncontrolled variables impacted these results. Future trials that are adequately powered should examine the impact of chronic almond ingestion (28 g/d) on biomarkers in both well-controlled and unstable diabetes.

In conclusion, these data demonstrate beneficial effects of mealtime almond consumption for individuals with well-controlled T2D. Under randomized controlled conditions, the ingestion of 1 oz almonds immediately before a starchy meal significantly reduced postprandial glycemia 30%; moreover, when this amount of almonds was ingested 5×/wk for 12 weeks, HbA_{1c} was significantly reduced 4% compared with baseline values. These data are encouraging and relevant considering recent analyses that question the efficacy of aggressive diabetic therapies to control glucose due to reported increased occurrences of hypoglycemia and weight gain, and even mortality, among patients [32]. Simple diet strategies are needed to manage diabetes; almonds are nutritious, tasty, and easily introduced into diet plans. The epidemiologic literature suggests that moderate nut consumption (5 servings per week) is associated with reduced cardiovascular disease risk as well as lower body weight. These data indicate that regular nut ingestion may also benefit glucose control in individuals with T2D.

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