Short Communication

Effect of traditional Arabic coffee consumption on the glycemic index of Khalas dates tested in healthy and diabetic subjects

Juma Alkaabi MD, Bayan Al-Dabbagh PhD, Hussein Saadi MD, Salah Gariballa MD, Javed Yasin MSc

Department of Internal Medicine, Faculty of Medicine and Health Sciences, United Arab Emirates University, United Arab Emirates

The consumption of dates with coffee is common among Arabs and may affect postprandial hyperglycemia excursion. The study aimed to determine the effect of coffee on the glycemic index of a common variety of dates (Khalas) tested in healthy and type 2 diabetes mellitus individuals. Study subjects were thirteen healthy volunteers (mean age: 40.2±6.7 years) and ten diabetic participants with a mean HbA1c of 6.6±(0.7%) and a mean age of 40.8±5.7 years. Each subject participated in five days of tests with 50 g of glucose and 50 g equivalent of available carbohydrates from the dates (with/without coffee). Capillary glucose was measured in the healthy subjects at 0, 15, 30, 45, 60, 90 and 120 min, and for the diabetics at 0, 30, 60, 90, 120, 150 and 180 min. Glycemic indices were determined as ratios of the incremental areas under the response curves for the interventions. Statistical analyses were performed using the independent samples and paired t-tests. Mean±SE glycemic indices of the Khalas dates for the healthy individuals were 55.1±7.7 and 52.7±6.2 without and with coffee consumption, respectively. Similar values were observed for those with diabetes (53.0±6.0 and 41.5±5.4). Differences between glycemic indices of Khalas with or without coffee were not significant (p=0.124). There were no significant differences in glycemic index between the diabetic and healthy subjects (p=0.834 and p=0.202 without and with coffee respectively). In conclusion, at least in the short term, coffee does not adversely affect capillary glucose levels following Khalas dates consumption in healthy and diabetic volunteers.

Key Words: coffee, glycemic index, postprandial hyperglycemia excursion, dates, type 2 diabetes mellitus

INTRODUCTION

Coffee is among the most widely consumed beverages in the world; more than 50% of Americans drink coffee, and the average per capita intake is about 2 cups per day.[1] Coffee is the primary source of caffeine intake, providing approximately 210 mg/d per person in the United States. An understanding of both positive and negative health effects of coffee is important if individuals are to make informed choices regarding coffee consumption.[2] Studies so far have suggested that coffee consumption is associated with a reduction in the risk of developing Alzheimer's disease, Parkinson's disease and prostate cancer but it increases the risk of acid reflux.[3-6]

Coffee may affect postprandial hyperglycemia excursion, although conflicting results have been reported. It reportedly increases energy expenditure and thermogenesis, which can stimulate insulin sensitivity,[7] but short-term intervention studies reported lower insulin sensitivity after caffeine intake.[8,9]

Acute intake of caffeine impairs glucose homeostasis in healthy,[8,10-13] obese and diabetic individuals.[14-17] Consumption of caffeine before an oral glucose tolerance test (OGTT),[13] has been shown to reduce the insulin sensitivity index (ISI) by 20-30%,[8,14,16,18] and to decrease the glucose infusion rate during a euglycemic-hyperinsulinemic clamp by a similar extent.[12,15,18-20]

However, long-term coffee consumption may be associated with a decreased risk of type 2 diabetes.[21-26] In a systematic review of nine cohort studies, diabetes risk was lowest in subjects who drank greater than six cups daily and significantly reduced for subjects who consumed four to six cups daily, compared to those with minimal coffee consumption (less than two cups per day).[23] These associations did not differ by sex, obesity, or region including the United States, Europe, and Asia. The Nurses' Health Study, A prospective study of over 88,000 women aged 26 to 46 years found that the risk of diabetes was lower in those who consumed daily coffee even when small amounts were consumed.[27] These observational data do not prove a cause-and-effect relationship and thus

Corresponding Author: Dr Juma Alkaabi, Department of Internal Medicine, Faculty of Medicine & Health Sciences, PO Box 17666, UAE University, Al Ain, UAE.
Tel: + 9713-7137472; Fax: + 9713-7672995
Email: j.kaabi@uaeu.ac.ae
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it is difficult to recommend increasing coffee intake as a prevention strategy. The paradoxical findings of observational and clinical studies relating coffee drinking to diabetes risk may be related to the intake of sweetened coffee prior to the meals which results in reduction in the postprandial glycemia and insulinemia.[28] In addition, it remains unclear what coffee components are responsible for the beneficial effect of coffee on glucose metabolism.

The prevalence of type 2 diabetes mellitus (DM) in the United Arab Emirates (UAE) is amongst the highest in the world affecting 19% of the adult population.[29,30] Consumption of dates along with Arabic coffee is commonly practiced by the Emirati society and is indeed a routine, integral component of Arab culture. This, as with a number of other traditions, is passed from one generation to another, conveying respect and honor to guests. The tradition is to serve dates first then the coffee - normally in small cups (approximately 25 ml) with up to four cups at a sitting. Dietary control and lowering the glycemic index (GI) carbohydrates of one's diet can improve the control of diabetes.[31] Previous publications have demonstrated that dates have low to medium GI.[32-36]

In clinical practice, we commonly encounter the practical question of whether the intake of coffee with meals or snacks, and especially with dates, might precipitate postprandial excursions of the glucose levels in subjects with diabetes. Accordingly, we designed the present study to evaluate the GI of Khalas, a very common type of dates in the UAE, when consumed with and without Arabic coffee in both healthy and diabetic subjects. We hypothesized that the ingestion of coffee with dates would impair glucose tolerance compared with consumption of dates alone.

MATERIALS AND METHODS

Ethics statement

The researchers explained verbally and provided written information to all volunteers and volunteers were given the opportunity to ask questions and verify their understing. Following receiving detailed verbal and written information about the study, all participated subjects provided informed written consent. The study conformed to the requirements of the Declaration of Helsinki and was approved by Al Ain Medical District Human Research Ethics Committee (approval reference: 10/06) and registered in the ClinicalTrials.gov (ClinicalTrials.gov Identifier: NCT01422668).

Subjects

Study participants were recruited through local poster advertisements. After providing informed written consent, all volunteers completed an interviewer-administered questionnaire covering demographic data, tobacco and alcohol use, past medical and surgical history, comorbidities, medications use and current health status. In patients known to have diabetes, information on disease onset, duration, and management was elicited. Each subject underwent a complete physical examination including measurements of blood pressure, pulse rate, height and weight (for calculation of BMI), body fat composition analysis using the Tanita TBF-410 Body Composition Analyzer (Tanita Corp., Tokyo, Japan) and measurement of waist circumference. Inclusion criteria required that those in the healthy group were indeed healthy and in the diabetes group that their diabetes was controlled (HbA1c $\leq$8%) on diet with or without metformin. Exclusion criteria for both healthy and diabetic volunteers included morbid obesity (BMI >40 kg/m^2), pre diabetes, pregnancy, presence of gastroenterological disorders, alimentary tract surgery, a history of gastroenteritis in the prior six months, any alcohol intake, smoking, taking any medications (except metformin), poorly controlled diabetes (HbA1c >8%) and the presence of chronic diseases (such as bronchial asthma or rheumatoid arthritis) or acute illness (such as upper respiratory tract or urinary tract infection).

Thirteen healthy subjects (7 men and 6 women) and ten volunteers with type 2 DM (5 men and 5 women) were enrolled for the study (Table 1). These numbers were chosen based on the literature where similar numbers had provided adequate power.[37-39] In order to reduce within and between-subject variability in GI measurements, subjects were asked to refrain from changing their eating and physical activity habits until the study was completed ie subjects were advised to follow their normal diet and to avoid unusual vigorous activity. Instead of using venous samples we used capillary blood for the measurement of glucose to determine GIs and we also used oral glucose as a reference “food” three times in keeping with recommendations in the literature.[40] Patients receiving metfor-

Table 1. Demographic and other baseline characteristics of the studied subjects

Parameter	Healthy (n=13)	Type 2 DM (n=10)
	Mean ± SD	Mean ± SD
Age (yrs)	40.2 ± 6.7	40.8 ± 5.7
Body weight (Kg)	75.4 ± 16.0	83 ± 16.7
Height (cm)	166 ± 8.0	164 ± 7.8
Body mass index (Kg/m^2)	27.4 ± 4.1	30.7 ± 5.2
HbA1c (%)	5.8 ± 0.4	6.6 ± 0.7
Fasting blood glucose (mg/dL)	95.7 ± 6.5	116 ± 7.9
Waist circumference (cm)		
Men	97.3 ± 9.4	101 ± 10.9
Women	89.2 ± 12.6	98.7 ± 9.2
Body fat composition (%)		
Men	29.8 ± 8.6	30.0 ± 7.5
Women	32.3 ± 8.2	39.7 ± 6.1

min (5 patients) were asked to take their usual dose of the drug before eating the test meal.[41] Prior to the GI studies, a fasting venous blood sample was obtained from all subjects for measurements of complete blood count, glucose, hemoglobin A1c, lipids, liver function tests, urea, electrolytes and urine protein utilizing a Beckman Coulter DXC800 (Beckman Instruments, Inc, Fullerton, CA, USA) auto-analyzer at the central laboratory of Tawam hospital, a tertiary referral hospital in Al Ain.

Test dates

Khalas, a very popular variety of date in the UAE, was chosen for this study. The same batch of packaged dates (Tamer stage) obtained from a local dates processing factory (Al Saad date factory, Al Ain, UAE) was used for all tests. The composition of this type of dates has been previously described.[32]

Coffee preparation

The coffee was prepared in the traditional fashion by using five tablespoons of freshly ground medium roasted coffee beans (origin: Sri Lanka) (25 g) per 1000 ml boiled water. This was left to boil for three minutes, left at room temperature for two minutes then filtered and served with no additives such as sugar or cardamom. This method of preparation accords with the common style of making homemade coffee in the UAE. Each candidate received 100 ml of coffee followed by 150 ml of water. To ensure uniformity, all the coffee used in the study was prepared in large quantity and served on the same time of the day to each participant.

Measurement of glycemic response

The study was carried out between March and June 2010. After an overnight fast on 5 occasions in every subject, each test being separated from the next by a "washout" day. The first 3 test days utilized the reference "food" 50 g of glucose dissolved in 250 ml water (Trutol® 50, Thermo Scientific) followed sequentially by 50g carbohydrate equivalents of the Khalas dates alone and finally dates with coffee. The reference "food" (50 g of glucose) was tested on 3 alternating days in order to minimize day to day variation of glucose tolerance.

The dates were weighed using an analytical scale (H110 Sartor, Sartorius AG, Goettingen, Germany), and consumed by all participants with 250 ml of water. Capillary blood glucose was monitored over 2 hrs for the healthy individuals at 0, 15, 30, 45, 60, 90 and 120 min and over 3 hrs; and for the diabetics at 0, 30, 60, 90, 120, 150 and 180 min.[37] Areas under the curve (AUC) of blood glucose concentrations resulting from glucose given orally in a dose of 50 g with a corresponding oral carbohydrate load of 50 g were compared as previously described.[37] The 50 g of glucose was used as the reference "food" (GI = 100) against which Khalas dates alone and with coffee were compared. The areas under the incremental postprandial hyperglycemia excursion -response curves for Khalas dates, with and without coffee, were expressed as a percentage of the mean area under the three glucose curves for the same subject. The resulting values for all subjects were averaged to calculate the GI for each type of intervention. For calculation of the GIs, capillary blood glucose was measured immediately after sampling in our research laboratory at the Faculty of Medicine and Health Sciences, United Arab Emirates University using one of two One Touch II® Lifescan glucometers (LifeScan, Inc, Milpitas, CA, USA), which were tested for accuracy and precision with the provided kits and against a Beckman Synchron CX7 laboratory analyzer (Beckman Instruments, Inc, Fullerton, CA, USA) which uses the glucose oxidase method. The coefficient of variation was 2.20-2.65% for both glucometers using three testing samples across the low, mid and high glucose ranges.

Statistical analysis

Data were analyzed using Microsoft Excel 2003 and SPSS version 19 (SPSS Inc, Chicago, IL, USA). For each dates meal the GI was measured by calculating the area under the curve using the formulae kindly provided by Professor Thomas Wolever from the University of Toronto-Canada. Standard descriptive statistics were used and results are presented as means and standard deviations (SD) or standard errors (SE) of the mean. In addition, for Khalas with/without coffee, the GIs were compared between DM patients and controls using independent samples t-tests. Comparisons of GIs of the Khalas dates with/without coffee were carried out using the paired t-test. In all cases the statistical significance level was set at $p<0.05$.

RESULTS

There were 13 healthy subjects (7 women and 6 men) with a mean (±SD) age of 40.2±6.7 years and BMI (±SD) of 27.4±4.1 kg/m^2, and 10 subjects with type 2 DM (F:M = 1:1) with a mean age (±SD) of 40.8±5.7 years, BMI (±SD) of 30.7±5.2 kg/m^2 and mean HbA1c (±SD) of 6.6 ±0.7 (Table 1).

The measured mean±SE GIs of the dates among healthy individuals were 55.1±7.7 and 52.7±6.2 for Khalas and Khalas with Arabic coffee, respectively. The mean±SE GIs among individuals with type 2 DM were very similar (53.0±6.0 and 41.5±5.4, respectively).

Figures 1 and 2 are graphic presentations of the GI changes in the healthy and diabetic subjects, respectively. The consumption of Khalas dates without and with Arabic coffee did not result in significant postprandial glucose excursions.

There were no statistically significant differences in the GIs, neither with nor without coffee, between the DM and control groups (t-tests +/- coffee *p*-values 0.202/0.834). Figures 3 a-c show the histograms of the distributions of the GI values without (Figure 3A) and with (Figure 3B) coffee as well as of the within subject differences (without minus with coffee Figure 3C).

DISCUSSION

Consumption of coffee with dates is very common practice in Arabic countries. There are theoretical reasons to consider that this long-established custom of combining coffee with dates ingestion might adversely alter glucose homeostasis since it has been reported that when caffeine is given during a hyperinsulinemic-euglycemic clamp, whole-body glucose disposal is decreased by 15-30%

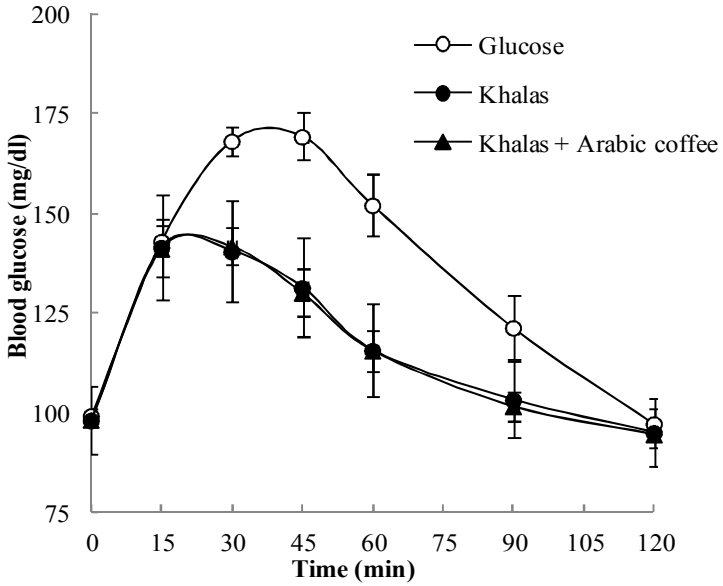

Figure 1. Capillary glucose concentrations (mean ± SEM) before and after ingestion of glucose (50 g), Khalas dates alone and dates with Arabic coffee in healthy subjects.

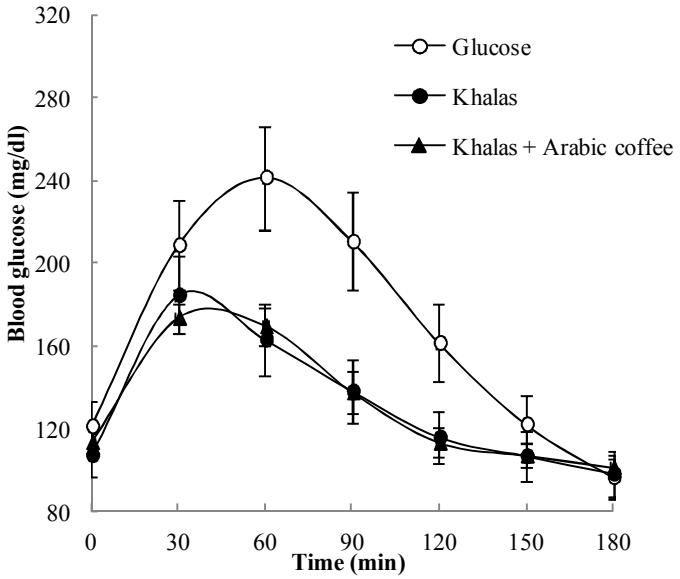

Figure 2. Capillary glucose concentrations (mean±SE) before and after ingestion of glucose 50 g, Khalas dates alone and dates with Arabic coffee in subjects with type 2 diabetes.

compared with placebo.[22,41-42] Caffeine stimulates the release of epinephrine, which exerts actions opposite to that of insulin via b-adrenergic stimulation.[43] It has been shown that intake of coffee with lipid diet or mixed meals decreases glucose tolerance.[44] Although caffeine ingestion has been shown to impair insulin sensitivity; it accounts for approximately 2% of coffee's chemical profile.[45]

This is of considerable concern in our region since the prevalence of diabetes, particularly type 2 DM, is amongst the highest in the world and in Arab countries, including the UAE.[29,30] The present study was designed to determine whether the GI of healthy volunteers and subjects with type 2 DM is adversely affected by the concomitant taking of coffee after ingestion of dates. Our observations are twofold. First, we observed that Khalas dates have a low GI. This is in keeping with previous studies.[32-36] The low GI of dates can be attributed to the presence of high level of fructose and dietary fibers. Second and most important, we demonstrate that coffee intake did not adversely affect capillary blood levels after ingestion of Khalas dates. Rather there was a trend, at least in our diabetic subjects for the postprandial hyperglycemia excursion to improve when coffee was taken along with dates versus ingestion of dates alone. Drinking coffee with dates did not significantly affect the GI values of the dates. These results are in agreement with those obtained by Aldughpassi and Wolever in 2009.[46]

Coffee has numerous biologically active compounds, including phenolic compounds (eg, chlorogenic acids, caffeic acid), polysaccharides, minerals (eg, magnesium), quinide and lipids. These compounds and others which may counteract the effects of caffeine on glucose responses,[11] affect glucose metabolism in animal or me-

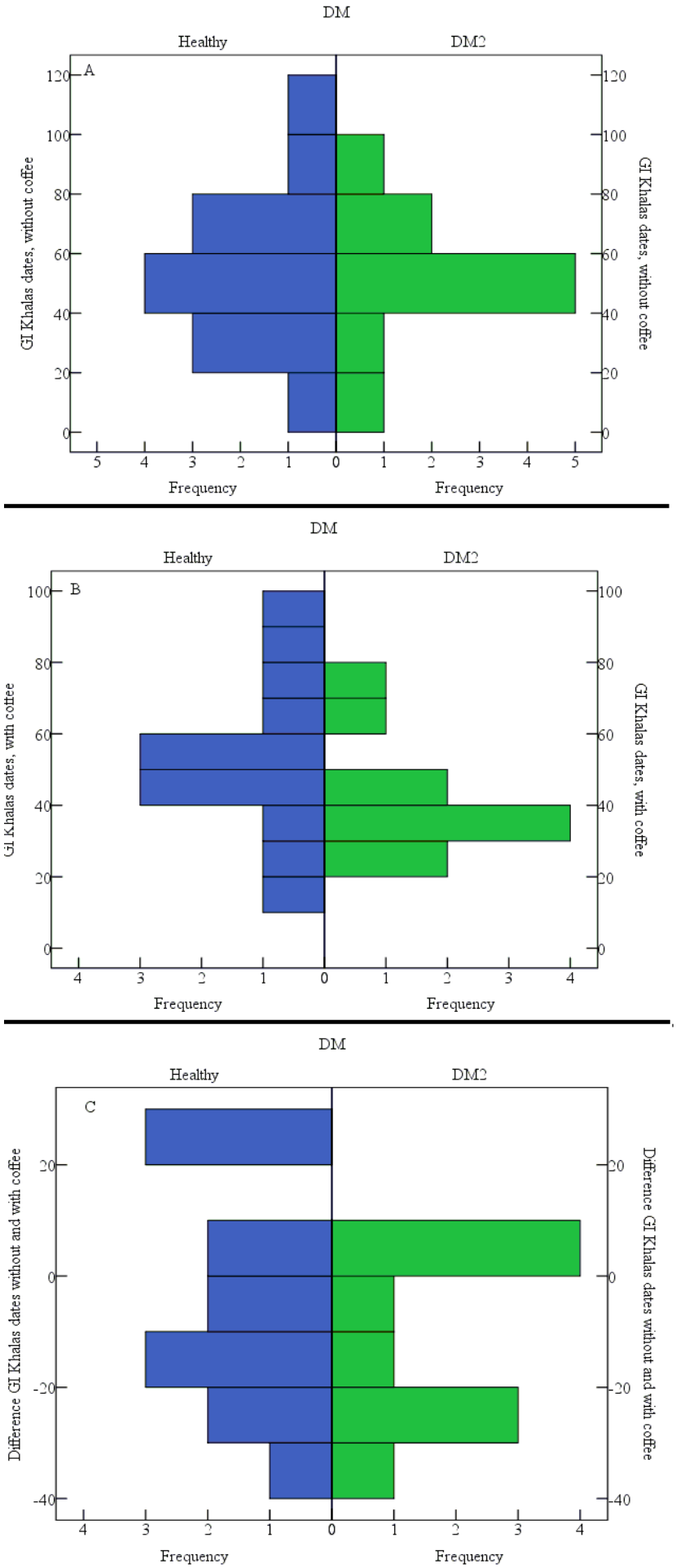

Figure 3 (A-C). Histograms of Glycemic Indices (GI) among the 13 healthy and 10 type 2 DM subjects, without (a) and with coffee (b), as well as the difference between GI without and with coffee (c).

tabolic studies and they are potent stimulants of gastric emptying.[2,8-9,16,19,47-50] In addition, coffee has antioxidant effects,[51] and there are more than 600 volatile components that have been identified in roasted coffee.[52]

In our study, we followed the norm custom of serving coffee immediately after the intake of the dates. However, in previous published literature, it has been shown that the intake of caffeine and caffeinated coffee may elicit acute insulin insensitivity when coffee was given one hour prior to the test meal.[53-55] The equivalent of 7-10 dates was used in our study, which is similar to what is maximally eaten at a single sitting by UAE nationals. The amount of coffee used in our study (100 ml) is relatively smaller if compared with the published literature.[53-55] But, this was in keeping with the maximum intake of coffee in a single traditional setting. To minimize the effect of volume of the beverage on gastric emptying, the same volume was given to all the study subjects.[56] Increased gastric emptying is a possible mechanism for the effect of coffee on glucose responses but this needs further studied. The results of this study show no evidence of increased postprandial hyperglycemia in healthy and diabetic individuals when coffee was ingested after the dates meal. Postprandial hyperglycemia excursion has been shown to increase cardiovascular risk.[57] This is reassuring to the locals with diabetes who customarily consume coffee in moderation with dates during their daily social settings.

To our knowledge, this is the first report examining the effect of coffee intake and the GIs of dates in type 2 DM subjects. Further studies are needed to study the long-term implications of coffee intake in individuals with type 1 DM and type 2 DM.

Long-term clinical trials of coffee consumption in type 2 DM subjects with measurement of insulin sensitivity, glucose metabolism, glycemic control and inflammatory markers are warranted to ensure the safety of habitual coffee consumption in the presence of diabetes. Study participants with type 2 DM were controlled on diet or metformin only and thus not representative of all persons with type 2 DM; especially at advanced stages of the disease, ie those receiving multiple oral hypoglycemic agents or insulin.

Limitations of the study

No acute insulin sensitivity testing, measurement of insulin resistance or glucose disposal were done. It is difficult to extrapolate findings from short-term metabolic studies to long-term use of coffee and thus it is premature to advocate the safety of high coffee consumption in subjects with type 2 DM. We used only Khalas dates (Tamer stage); hence the conclusions from this study may not be applied to all types of dates when consumed with Arabic coffee. It was not technically feasible to measure the amount of caffeine in the coffee.

Conclusions

The intake of traditional coffee with dates is not a risk factor for postprandial hyperglycemia and there is no evidence for discouraging people with diabetes from drinking coffee in moderation. We have determined the composition and calculated the glycemic index of Khalas, and have shown that its consumption with coffee by healthy and diabetic individuals does not result in significant postprandial glucose excursions. Our results confirm the low GI of Khalas dates in healthy and diabetic individuals when consumed with and without Arabic coffee. Nevertheless, long-term randomized clinically controlled intervention trials are needed to evaluate the effects of coffee with dates in the prevention of diabetes and the control of hyperglycemia in subjects with diabetes.

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AUTHOR DISCLOSURES

None of the authors have any undisclosed conflict of interest.

REFERENCES

1. Lundsberg LS. Caffeine consumption. In: Spiller GA, editor. Caffeine. Boca Raton, FL: CRC Press; 1998. pp. 199-224.
2. Schaefer B. Coffee consumption and type 2 diabetes mellitus. Ann Intern Med. 2004;141:321. doi: 10.7326/0003-4819-141-4-200408170-00016
3. Klatsky AL, Morton C, Udaltsova N, Friedman, GD. Coffee, cirrhosis, and transaminase enzymes. Arch Int Med. 2006; 166:1190-5.
4. Eskelinen M, Ngandu T, Tuomilehto J, Soininen H, Kivipelto M. Midlife coffee and tea drinking and the risk of late-life dementia: a population-based CAIDE study. J Alzheimers Dis. 2009;16:85-91.
5. Brigas D. Gastro-oesophageal reflux disease in an obese patient. Clin Drug Investig. 2009;29:17-18. doi: 10.2165/1153123-S0-000000000-00000
6. Wilson KM, Kasperzyk JL, Rider JR, Kenfield S, Van Dam RM, Stampfer MJ, Giovannucci E, Mucci LA. Coffee Consumption and Prostate Cancer Risk and Progression in the Health Professionals Follow-up Study. J Natl Cancer Inst. 2011;103:876-84. doi: 10.1093/jnci/djr151
7. Park S, Jang JS, Hong SM. Long-term consumption of caffeine improves glucose homeostasis by enhancing insulinotropic action through islet insulin/insulin-like growth factor 1 signaling in diabetic rats. Metabolism. 2007;56:599-607. doi: 10.1016/j.metabol.2006.12.004
8. Keijzers GB, de Galan BE, Tack CJ, Smits P. Caffeine can decrease insulin sensitivity in humans. Diabetes Care. 2002; 25:364-9. doi: 10.2337/diacare.25.2.364
9. Lane JD, Barkauskas CE, Surwit RS, Feinglos MN. Caffeine impairs glucose metabolism in type 2 diabetes. Diabetes Care. 2004;27:2047-8. doi: 10.2337/diacare.27.8.2047
10. Dekker MJ, Gusba JE, Robinson LE, Graham TE. Glucose homeostasis remains altered by acute caffeine ingestion following 2 weeks of daily caffeine consumption in previously non-caffeine-consuming males. Br J Nutr. 2007;98:556-2. doi: 10.1017/S0007114507730738
11. Battram DS, Arthur R, Weekes A, Graham TE. The glucose intolerance induced by caffeinated coffee ingestion is less pronounced than that due to alkaloid caffeine in men. J Nutr. 2006;136:1276-80.
12. Norager CB, Jensen MB, Weimann A, Madsen MR. Metabolic effects of caffeine ingestion and physical work in 75-year old citizens. A randomized, double-blind, placebo-

controlled, cross-over study. Clin Endocrinol (Oxf). 2006;65: 223-8. doi: 10.1111/j.1365-2265.2006.02579.x
13. Petrie HJ, Chown SE, Belfie LM, Duncan AM, McLaren DH, Conquer JA, Graham TE. Caffeine ingestion increases the insulin response to an oral-glucose-tolerance test in obese men before and after weight loss. Am J Clin Nutr. 2004;80:22-8.
14. Lee S, Hudson R, Kilpatrick K, Graham TE, Ross R. Caffeine ingestion is associated with reductions in glucose uptake independent of obesity and type 2 diabetes before and after exercise training. Diabetes Care. 2005;28:566-72. doi: 10.2337/diacare.28.3.566
15. Robinson LE, Savani S, Battram DS, McLaren DH, Sathasivam P, Graham TE. Caffeine ingestion before an oral glucose tolerance test impairs blood glucose management in men with type 2 diabetes. J Nutr. 2004;134:2528-33.
16. Lane JD, Feinglos MN, Surwit RS. Caffeine increases ambulatory glucose and postprandial responses in coffee drinkers with type 2 diabetes. Diabetes Care. 2008;31:221-2. doi: 10.2337/dc07-1112
17. Thong FSL, Graham TE. Caffeine-induced impairment of glucose tolerance is abolished by beta-adrenergic receptor blockade in humans. J Appl Physiol. 2002;92:2347-52.
18. Greer F, Hudson R, Ross R, Graham TE. Caffeine ingestion decreases glucose disposal during a hyperinsulinemic-euglycemic clamp in sedentary humans. Diabetes. 2001;50: 2349-54. doi: 10.2337/diabetes.50.10.2349
19. Battram DS, Graham TE, Richter EA, Dela F. The effect of caffeine on glucose kinetics in humans: influence of adrenaline. J Physiol. 2005;569:347-55. doi: 10.1113/jphysiol.2005.097444
20. Battram DS, Graham TE, Dela F. Caffeine's impairment of insulin-mediated glucose disposal cannot be solely attributed to adrenaline in humans. J Physiol. 2007;583:1069-77. doi: 10.1113/jphysiol.2007.130526
21. Salazar-Martinez E, Willett WC, Ascherio A, Manson JE, Leitzmann MF, Stampfer MJ, Hu FB. Coffee consumption and risk for type 2 diabetes mellitus. Ann Intern Med. 2004;140:1-8. doi: 10.7326/0003-4819-140-1-200401060-00005
22. Tuomilehto J, Hu G, Bidel S, Lindström J, Jousilahti P. Coffee consumption and risk of type 2 diabetes mellitus among middle-aged Finnish men and women. JAMA. 2004; 291: 1213-9. doi: 10.1001/jama.291.10.1213
23. van Dam RM, Hu FB. Coffee consumption and risk of type 2 diabetes: a systematic review. JAMA. 2005;294:97-104. doi: 10.1001/jama.294.1.97
24. Pereira MA, Parker ED, Folsom AR. Coffee consumption and risk of type 2 diabetes mellitus: an 11-year prospective study of 28 812 postmenopausal women. Arch Intern Med. 2006;166:1311-6. doi: 10.1001/archinte.166.12.1311
25. Paynter NP, Yeh HC, Voutilainen S, Schmidt MI, Heiss G, Folsom AR, Brancati FL, Kao WH. Coffee and sweetened beverage consumption and the risk of type 2 diabetes mellitus: the atherosclerosis risk in communities study. Am J Epidemiol. 2006;164:1075-84. doi: 10.1093/aje/kwj323
26. Huxley R, Lee CM, Barzi F, Timmermeister L, Czernichow S, Perkovic V, Grobbee DE, Batty D, Woodward M. Coffee, decaffeinated coffee, and tea consumption in relation to incident type 2 diabetes mellitus: a systematic review with meta-analysis. Arch Intern Med. 2009;169:2053-63. doi: 10.1001/archinternmed.2009.439
27. van Dam RM, Willett WC, Manson JE, Hu FB. Coffee, caffeine, and risk of type 2 diabetes: a prospective cohort study in younger and middle-aged U.S. women. Diabetes Care. 2006;9:398-403. doi: 10.2337/diacare.29.02.06.dc05-1512
28. Louie JC, Atkinson F, Petocz P, Brand-Miller JC. Delayed effects of coffee, tea and sucrose on postprandial glycemia in lean, young, healthy adults. Asia Pac J Clin Nutr. 2008; 17:657-62.
29. International diabetes federation; [cited 16 March 2013]; Available from: http://www.idf.org/diabetesatlas/5e/the-global-burden.
30. Saadi H, Nagelkerke N, Al-Kaabi J, Afandi B, Al-Maskari F, Kazam E. Screening strategy for type 2 diabetes in the United Arab Emirates. Asia Pac J Public Health. 2010;22:54S-59S. doi: 10.1177/1010539510373036
31. Pastors JG, Warshaw H, Daly A, Franz M, Kulkarni K. The evidence for the effectiveness of medical nutrition therapy in diabetes management. Diabetes Care. 2002;25:608-13. doi: 10.2337/diacare.25.3.608
32. Alkaabi J, Al-Dabbagh B, Ahmad S, Saadi H, Gariballa S, Ghazali MA. Glycemic indices of five varieties of dates in healthy and diabetic subjects. Nutr J. 2011;10:59. doi: 10.1186/1475-2891-10-59
33. Miller CJ, Dunn EV, Hashim IB. Glycemic index of 3 varieties of dates. Saudi Med J. 2002;23:536-8.
34. Miller CJ, Dunn EV, Hashim IB. The glycaemic index of dates and date/yoghurt mixed meals. Are dates 'the candy that grows on trees'? Eur J Clin Nutr. 2003;57:427-30. doi: 10.1038/sj.ejcn.1601565
35. Lock DR, Bar-Eyal A, Voet H, Madar Z. Glycemic indices of various foods given to pregnant diabetic subjects. Obstet Gynecol. 1988;71:180-3.
36. Ahmed M, Al-Othaimeen A, De Vol E, Bold A. Comparative responses of plasma glucose, insulin and C-peptide following ingestion of isocaloric glucose, a modified urban Saudi breakfast and dates in normal Saudi persons. Ann Saudi Med. 1991;11:414-7.
37. Wolever TM, Jenkins DJ, Jenkins AL, Josse RG. The glycemic index: methodology and clinical implications. Am J Clin Nutr. 1991;54:846-54.
38. Aston LM, Gambell JM, Lee DM, Bryant SP, Jebb SA. Determination of the glycaemic index of various staple carbohydrate-rich foods in the UK diet. Eur J Clin Nutr. 2008;2: 279-85. doi: 10.1038/sj.ejcn.1602723
39. Brouns F, Bjorck I, Frayn KN, Gibbs AL, Lang V, Slama G, Wolever TM. Glycaemic index methodology. Nutr Res Rev. 2005;18:145-71. doi: 10.1079/NRR2005100
40. Hätönen KA, Similä ME, Virtamo JR, Eriksson JG, Hannila ML, Sinkko HK, Sundvall JE, Mykkänen HM, Valsta LM. Methodologic considerations in the measurement of glycemic index: glycemic response to rye bread, oatmeal porridge, and mashed potato. Am J of Clin Nutr. 2006;84:1055-61.
41. Thong FS, Derave W, Kiens B, Graham TE, Ursø B, Wojtaszewski JF, Hansen BF, Richter EA. Caffeine-induced impairment of insulin action but not insulin signaling in human skeletal muscle is reduced by exercise. Diabetes. 2002; 51:583-90. doi: 10.2337/diabetes.51.3.583
42. Van Dam RM, Feskens EJ. Coffee consumption and risk of type 2 diabetes mellitus. Lancet. 2002;360:1477-8. doi: 10.1016/S0140-6736(02)11436-X
43. Avogaro A, Toffolo G, Valerio A, Cobelli C. Epinephrine exerts opposite effects on peripheral glucose disposal and glucose-stimulated insulin secretion: a stable label intravenous glucose tolerance test minimal model study. Diabetes. 1996;45:1373-8. doi: 10.2337/diabetes.45.10.1373
44. Beaudoin MS, Robinson LE, Graham TE. An Oral Lipid Challenge and Acute Intake of Caffeinated Coffee Additively Decrease Glucose Tolerance in Healthy Men. J Nutr. 2011;41:574-81. doi: 10.3945/jn.110.132761

45. Spiller MA. The chemical components of coffee. In: Siller, GA editor. Caffeine. CRC Press: New York, NY; 1998. pp. 97-161.
46. Aldughpassi A, Wolever TMS. Effect of coffee and tea on the glycaemic index of foods: no effect on mean but reduced variability. Br J Nutr. 2009;101:1282-5. doi: 10.1017/S0007114508079610
47. Lien HC, Chen GH, Chang CS, Kao CH, Wang SJ. The effect of coffee on gastric emptying. Nuc Med Comm. 1995;16:923-6. doi: 10.1097/00006231-199511000-00008
48. Rodriguez de Sotillo DV, Hadley M. Chlorogenic acid modifies plasma and liver concentrations of: cholesterol, triacylglycerol, and minerals in (fa/fa) Zucker rats. J Nutr Biochem. 2002;13:717-26. doi: 10.1016/S0955-2863(02)00231-0
49. Shearer J, Farah A, de Paulis T, Bracy DP, Pencek RR, Graham TE, Wasserman DH. Quinides of roasted coffee enhance insulin action in conscious rats. J Nutr. 2003;133: 3529-32.
50. de Valk HW. Magnesium in diabetes mellitus. Neth J Med. 1999;54:139-46. doi: 10.1016/S0300-2977(99)00005-4
51. Corrêa TA, Monteiro MP, Mendes TM, Oliveira DM, Rogero MM, Benites CI et al. Medium light and medium roast paper-filtered coffee increased antioxidant capacity in healthy volunteers: results of a randomized trial. Plant Foods Hum Nutr. 2012;67:277-82. doi: 10.1007/s11130-012-0297-x
52. Gilbert RM. Caffeine consumption. In: Spiller GA, editor. The methylxanthine beverages and foods: chemistry, consumption, and health effects. New York, NY: Alan R. Liss; 1984. pp. 185-213.
53. Moisey LL, Kacker S, Bickerton AC, Robinson LE, Graham TE. Caffeinated coffee consumption impairs blood glucose homeostasis in response to high and low glycemic index meals in healthy men. Am J Clin Nutr. 2008;87:1254-61.
54. Battram DS, Arthur R, Weekes A, Graham TE. The glucose intolerance induced by caffeinated coffee ingestion is less pronounced than that due to alkaloid caffeine in men. J Nutr. 2006;136:1276-80.
55. Lane JD, Hwang AL, Feinglos MN, Surwit RS. Exaggeration of postprandial hyperglycemia in patients with type 2 diabetes by administration of caffeine in coffee. Endocr Pract. 2007;13:239-43. doi: 10.4158/EP.13.3.239
56. Young KWH, Wolever TMS. Effect of volume and type of beverage consumed with a standard test meal on postprandial blood glucose responses. Nutr Res. 1998;18:1857-63. doi: 10.1016/S0271-5317(98)00155-9
57. Aryangat AV, Gerich JE. Type 2 diabetes: postprandial hyperglycemia and increased cardiovascular risk. Vasc Health Risk Manag. 2010;24:145-55. doi: 10.2147/VHRM. S8216

Short Communication

Effect of traditional Arabic coffee consumption on the glycemic index of Khalas dates tested in healthy and diabetic subjects

Juma Alkaabi MD, Bayan Al-Dabbagh PhD, Hussein Saadi MD, Salah Gariballa MD, Javed Yasin MSc

Department of Internal Medicine, Faculty of Medicine and Health Sciences, United Arab Emirates University, United Arab Emirates

健康及糖尿病者攝取傳統阿拉伯咖啡對 Khalas 椰棗的升糖指數影響

阿拉伯人喝咖啡時，食用椰棗是常見的，這可能影響餐後高血糖波動。此研究為評估咖啡對於健康者及第二型糖尿病患者食用普及的 Khalas 椰棗的升糖指數影響。研究對象為 13 名健康志願者(平均年齡為 40.2±6.7 歲)及 10 名糖尿病參與者，其平均糖化血色素為 6.6±0.7%、平均年齡為 40.8±5.7 歲。每名參與者接受五天測試，給予 50 公克葡萄糖以及 50 公克等量碳水化合物的椰棗(有或無喝咖啡)。測量健康參與者在 0、15、30、45、60、90 及 120 分鐘與糖尿病者在 0、30、60、90、120、150 及 180 分鐘的微血管血糖濃度。升糖指數以介入後的反應曲線下面積增加率為評量依據。統計分析是使用獨立樣本與配對 t 檢定。健康者無或有攝取咖啡的椰棗升糖指數(平均數±標準誤)分別是 55.1±7.7 及 52.7±6.2。在糖尿病患者觀察到類似的數值(53.0±6.0 及 41.5±5.4)。Khalas 椰棗在有或無咖啡攝取的升糖指數差異沒有達到顯著。糖尿病跟健康者的升糖指數亦沒有顯著差異。結論是，至少就短期效應而言，咖啡對於健康及糖尿病者攝取椰棗後的微血管血糖值沒有影響。

關鍵字：咖啡、升糖指數、餐後高血糖波動、椰棗、第二型糖尿病