Original Article

Delayed effects of coffee, tea and sucrose on postprandial glycemia in lean, young, healthy adults

Jimmy Chun Yu Louie MNutrDiet[1], Fiona Atkinson MNutrDiet[1], Peter Petocz PhD[2], Jennie C Brand-Miller PhD[1]

[1]*Human Nutrition Unit, School of Molecular and Microbial Biosciences, The University of Sydney, Sydney, Australia*
[2]*Department of Statistics, Macquarie University, Sydney, Australia*

In observational studies, habitual coffee consumption has been linked to a lower risk of type 2 diabetes. We hypothesized that the mechanism may be related to delayed effects on postprandial glycemia. The aim of this study is to investigate the glycemic and insulinemic effects of consumption of caffeinated and decaffeinated coffee, sweetened and unsweetened, tea and sucrose, 1 h prior to a high carbohydrate meal. On separate occasions in random order, lean young healthy subjects (n = 8) consumed a potato-based meal 1 hour after consumption of 250 mL of black coffee (COF), black coffee sweetened with 10 g of sucrose (COF+SUC), decaffeinated coffee (DECAF), black tea (TEA), 10 g sucrose (SUC) or hot water (CON). Fingerprick blood samples were taken at regular intervals over 2 h and the glucose and insulin responses quantified as area under the curve. Compared to CON, COF caused a 28% increase in postprandial glycemia (p = 0.022). In contrast, COF+SUC decreased glycemia compared with either COF (-38%, p < 0.001) or CON (-20%, p = 0.100) but had no effect on insulin responses. DECAF, TEA and SUC had no significant effects on postprandial responses. SUC and DECAF reduced the absolute glucose concentration at the start of the meal (p < 0.01). In conclusion, only sweetened coffee significantly reduces postprandial glycemia. This observation may explain the paradoxical findings of observational and clinical studies relating coffee drinking to diabetes risk.

Key Words: coffee, tea, postprandial glycemia, insulinemia, caffeine

INTRODUCTION

There is good evidence that habitual coffee consumption reduces the risk of type II diabetes (T2DM). van Dam and Feunekes[1] found that individuals who drank at least 7 cups of coffee a day had half the risk of developing T2DM compared to those who drank less than 2 cups. Similarly, inverse relationships between coffee consumption and risk of developing T2DM,[2-9] impaired glucose tolerance,[10,11] fasting glucose concentration,[11] 2-hour insulin glucose and insulin[10,11] and insulin sensitivity,[10] have been reported. Paradoxically, caffeine itself has been found to reduce insulin sensitivity[12] and impair glucose tolerance.[13,14] Arnlov *et al*,[15] however, showed that the effects of caffeine on insulin resistance diminished with chronic consumption, while Battram *et al*[16] found that coffee had less effect than caffeine by itself. Hence other components in coffee may counteract the effects of caffeine on glucose metabolism.[16] Chlorogenic acid (CGA), for example, the chief phenolic compound in coffee, can inhibit glucose absorption from the small intestine[17] and down-regulate gluconeogenesis via the inhibition of glucose-6-phosphatase.[18,19] CGA supplements have been promulgated as an "alternative strategy" to a low glycemic index (GI) diet.[20] Tea also contains caffeine, but unlike coffee, it contains no CGA, and tea drinking has not been linked to reduced risk of diabetes.[21, 22]

We hypothesized that one mechanism which may explain the effects of coffee drinking on the risk of type 2 diabetes would be an ability to reduce postprandial glycemia. Although previous studies have found no demonstrable effect of 1-2 cups of coffee or tea when consumed *at the same time* as the meal,[23] it is possible that a lag-time exists between consumption and any effect on hepatic or peripheral metabolism. Since coffee is often consumed between meals, any delayed effect on postprandial glycemia would be physiologically relevant. The aim of the present study was therefore to explore the postprandial metabolic response to pre-feeding of normal and decaffeinated coffee, sweetened and unsweetened, 1 hour prior to a starchy meal. Tea was also studied because it represents a similarly popular caffeinated beverage that has not been linked to diabetes risk.

MATERIALS AND METHODS

Subjects

Eight (5 males, 3 females) healthy, non-smoking, regular coffee drinkers (≥ 1 cup/day) were recruited by adver-

Corresponding Author: Prof JC Brand-Miller, Human Nutrition Unit, G 08, The University of Sydney, Sydney, NSW, 2006, Australia
Tel: +61 2 9351 3759; Fax: +61 2 9351 6022
Email: j.brandmiller@mmb.usyd.edu.au
Manuscript received 4 July 2008. Initial review completed 8 December 2008. Revision accepted 12 December 2008.

tisement from the University of Sydney student population. The mean ± SEM age, weight and BMI of the subjects were 26.3 ± 1.8 y (range 20 - 35 y), 62.2 ± 3.5 kg (range 45.0 – 78.0 kg) and 20.8 ± 0.7 kg/m^2 (range 17.6 – 24.3 kg/m^2) respectively. The study protocol was approved by the Human Research Ethics Committee of the University of Sydney and volunteers gave written, informed consent.

Study design

Subjects undertook a total of 7 separate sessions in random order after an overnight fast. The test beverages represented 250 mL of the following solutions: black coffee (COF, made with 4g Nestlé® NESCAFÉ® Blend43^{TM} instant coffee powder containing ~150 mg of caffeine), black coffee sweetened with 10 g of sucrose (COF+SUC), black decaffeinated coffee (DECAF, made with 4 g Nestlé® NESCAFÉ® Decaf® instant coffee powder), black tea (TEA, made by brewing 1 Tetley® black tea bag for 3 minutes), 10 g sucrose (SUC) and hot water (CON, tested on 2 occasions). All beverages were made with just boiled water and consumed within 10 min. Artificial sweetener (Equal® Tablets) was used to sweeten the test drinks if desired, remaining constant within subjects. After 55 min, 2 fingerprick blood samples (~1 mL) were taken 5 min apart (-5 and 0 min, baseline concentration) and subjects consumed 586 g of instant mashed potato (Edgell® Instant Mash, prepared according to instructions, providing 75 g of available carbohydrate). Subjects were permitted to drink 250 mL water with the meal, remaining constant within subjects. A further 6 blood samples were collected at 15, 30, 45, 60, 90 and 120 min after the start of the meal using an automated lancet device (Accu-Chek® Safe-T-Pro Plus, Roche Diagnostics Australia, Castle Hill, Australia). Samples were collected into previously heparinized (10 IU heparin sodium salt, Sigma Chemical Co., St Louis, USA) Eppendorf micro-centrifuge tubes and centrifuged at 12,500 g for 1 min. Plasma was collected into chilled tubes and stored at -20^{o}C until analysed (< 1 month). Plasma glucose concentration was measured in duplicate using a Hitachi 912 Automatic Analyzer (Roche Diagnostics, Indianapolis, IN) employing the hexokinase/glucose-6-phosphate dehydrogenase method (Unimate 5 Gluc HK^{TM}, Roche Diagnostic Systems, Frenchs Forest, Australia). The intra-assay coefficient of variation is < 1.5% and the mean ± SEM inter-assay coefficient of variation is 11 ± 4%. Insulin was quantified using a commercial radioimmunoassay kit (Coat-a-Count, Diagnostic Products Corporation, LA, USA) in a single batch, with a mean ± SEM inter-assay CV of 15 ± 6%. Cumulative changes in plasma glucose and insulin response were expressed as the area under the 120 minute response curves (AUC) calculated according to the trapezoidal rule, truncated at the 0 minute reading (baseline concentration).[24] Areas below the baseline were ignored. A 'glucose score' was calculated for each subject using the following equation:

$$\text{Glucose Score} = \frac{\text{Area under the 120 min glucose response curve produced by prefeeding of the test drink}}{\text{Area under the 120 min glucose response curve produced by control (water)}} \times 100$$

Insulin scores were calculated in similar fashion. Results were expressed as mean ± SEM glucose or insulin score. Two-way ANOVA with drinks as fixed factor and subjects as random factor was used to determine statistical differences among the test drinks (Statistical Packages for Social Sciences version 15). A p value < 0.05 was considered marginally significant and p < 0.01 was considered statistically significant. No adjustment was made for multiple comparisons because only specific comparisons were of *a Priori* interest.

RESULTS

Changes in plasma glucose and insulin concentrations are shown in Figure 1 and 2 respectively. Compared with CON (hot water), COF consumed 1 h before a meal increased postprandial glycemia (+28%, p = 0.022). In contrast, sweetened coffee (COF+SUC) decreased glycemia compared with either COF (-38%, p < 0.001) or CON (-20%, p = 0.100). DECAF, TEA and SUC produced similar postprandial glucose responses to CON. Apart from COF vs. DECAF (insulin scores 122 ± 13 vs. 98 ± 6, p = 0.036), there were no significant differences in insulin

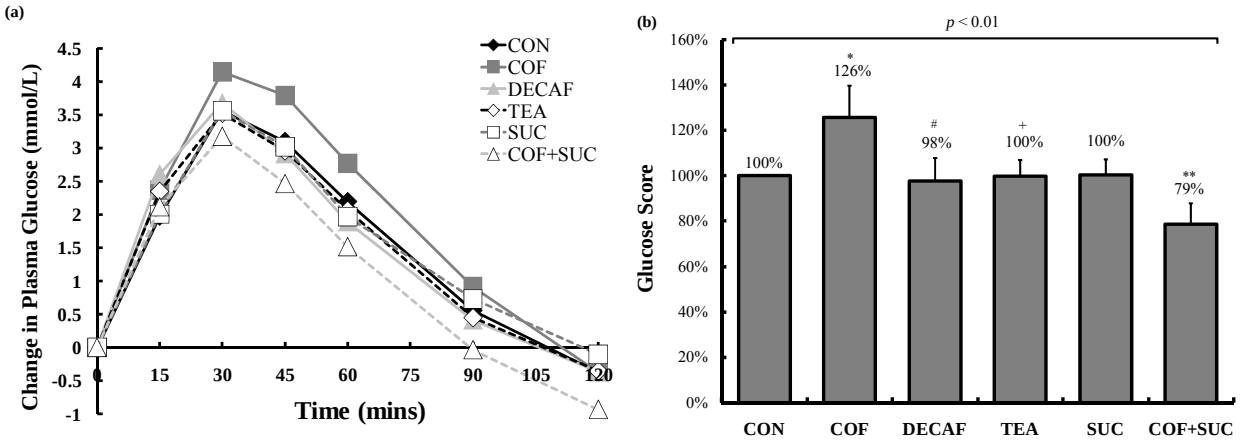

Figure 1. (a) Changes in postprandial plasma glucose concentration over time and **(b)** 'glucose scores' (AUC expressed as % of control) produced by the different test beverages. Values are expressed as mean and mean + SEM respectively (n = 8). p values were calculated by two-way ANOVA comparing CON with different test beverages. *COF vs CON, p = 0.022; #COF vs DECAF, p = 0.019; +COF vs TEA, p = 0.011; **COF vs COF+SUC, p < 0.001.

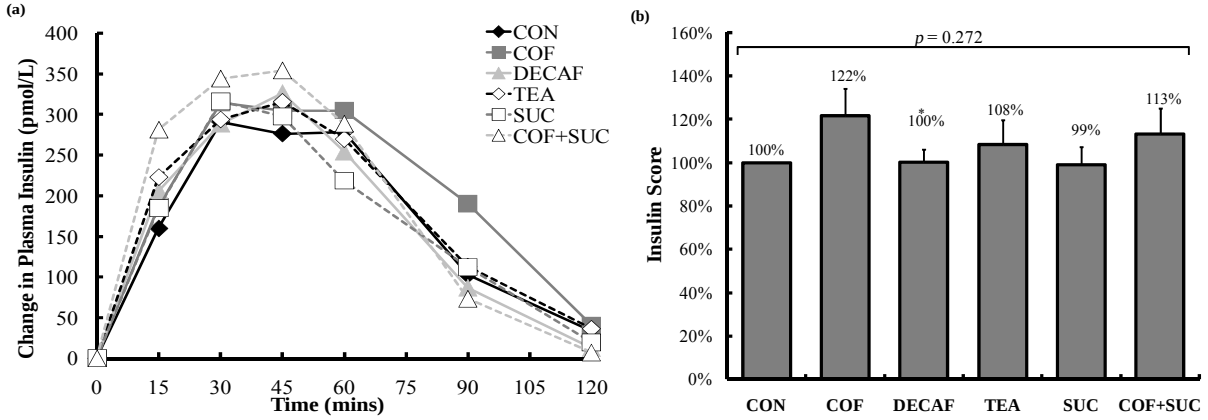

Figure 2. (a) Changes in postprandial plasma insulin concentration and **(b)** 'insulin scores' (AUC expressed as % of control) produced by different test beverages. Values are expressed as mean and mean + SEM respectively ($n = 8$). p values were calculated by two-way ANOVA comparing CON with different test beverages. *COF vs DECAF, $p = 0.036$.

responses among the beverages.

Absolute glucose and insulin concentrations also varied significantly according to treatment (Table 1). Compared to CON, there was a significantly lower plasma glucose concentration at the start of the meal preceded by DECAF ($p = 0.007$) and SUC ($p < 0.001$). Compared to CON, COF+SUC tended to produce a lower 2-h glucose concentration ($p = 0.023$) but significantly higher plasma insulin concentration at the start of the meal ($p = 0.010$). Glucose and insulin scores were correlated within the COF, TEA and SUC trials (Pearson correlation coefficient = 0.66, 0.66 and 0.74 respectively), but not COF+SUC or DECAF (Pearson correlation coefficient < 0.05).

DISCUSSION

On the basis of observational studies linking coffee drinking with reduced risk of type 2 diabetes, we hypothesized that coffee drinking might attenuate postprandial glycemia. Using a realistic 1-hour pre-meal study design, one cup of normal coffee, but not decaffeinated coffee or tea, had adverse effects on carbohydrate metabolism, increasing postprandial glycemia (+28%) and insulin (+20%) to a delayed high-carbohydrate meal. Battram *et al*[16] found that caffeine, either in its pure form or in roasted coffee, reduces insulin sensitivity. In their double-blinded randomized trial, 11 healthy men consumed pure caffeine (4.46 mg/kg body weight), roasted coffee (providing the same amount of caffeine as the capsules), placebo or decaffeinate coffee on separate occasions, and underwent a 2-h oral glucose tolerance test (OGTT) one hour after that. They found pure caffeine produced a significantly higher blood glucose response and significantly lower insulin sensitivity index (ISI) during the 2-h OGTT compared to the same amount of caffeine in roasted coffee. This is consistent with our findings which showed caffeinated coffee impaired glucose metabolism and produced higher insulin response. Remarkably, the addition of a small

Table 1. The effect of pre-feeding different test drinks 1 hour before consumption of a starchy meal on plasma glucose concentrations.

	Start of meal (mmol/L)	p value†	Peak response (mmol/L)	p value†	2-hour glucose (mmol/L)	p value†	AUC (mmol/L·120 min)	p value††
CON	5.49 ± 0.09	-	9.06 ± 0.15	-	5.12 ± 0.28	-	197 ± 19	-
COF	5.27 ± 0.12	0.077	9.41 ± 0.37	0.325	4.90 ± 0.28	0.304	248 ± 31	0.022
DECAF	5.13 ± 0.13	0.007	8.80 ± 0.42	0.482	4.78 ± 0.16	0.115	193 ± 23	0.948
		0.294‡		0.096‡		0.572‡		0.019‡
TEA	5.28 ± 0.13	0.087	8.79 ± 0.27	0.463	4.92 ± 0.38	0.346	196 ± 32	0.782
		0.952§		0.091§		0.931§		0.011§
SUC	4.97 ± 0.12	< 0.001	8.53 ± 0.18	0.143	4.86 ± 0.16	0.227	198 ± 21	0.917
COF+SUC	5.54 ± 0.19	0.690	8.72 ± 0.25	0.346	4.61 ± 0.24	0.023	155 ± 19	0.100
		0.021¶		0.059¶		0.191¶		< 0.001¶

Values are presented as mean ± SEM (n = 8).

†p values were calculated by two-way ANOVA comparing CON with different test beverages

‡p values were calculated by two-way ANOVA comparing DECAF with COF

§p values were calculated by two-way ANOVA comparing TEA vs COF

¶p values were calculated by two-way ANOVA comparing COF+SUC vs COF

††p values were calculated after the AUC were converted to glucose scores (see text)

amount (10 g) of sucrose to the coffee reversed this effect, and reduced the postprandial glycemic response by ~40% (COF vs. COF+SUC, $p < 0.001$). This could be explained by a significantly higher plasma insulin level at the start of meal (22.2 ± 3.2 vs. 29.5 ± 4.7, $p = 0.021$) which suggests a 'priming' effect of COF+SUC, resulting in an earlier peak insulin response to the test meal. Together, these observations might explain the conflicting literature whereby coffee drinking has been linked to reduced risk of diabetes[6,9,10] despite adverse effects on insulin sensitivity.[12,14] The common habit of sweetening coffee to mask bitterness, but not hot tea, may account for why coffee is more consistently associated with reduced risk.

The novel observation that 2 teaspoons of sucrose could not only neutralize the effect of coffee, but also have additional beneficial effects, was unexpected. A small amount of fructose, one of the products of sucrose digestion, is known to inhibit gluconeogenesis.[25] However, in the present study, sucrose alone (in hot water) did not produce separate beneficial effects. This suggests the possibility of an interaction between the components of coffee and sugar. Unfortunately, most of the epidemiological studies on the subject of coffee[1,3-5,9,26] did not examine the separate effect of consuming unsweetened coffee vs. sweetened coffee. Indeed, most studies adjusted for differences in macronutrient intake, so that any potential effect of consuming extra sucrose among coffee drinkers was removed. A study by van Dam *et al*[10] on the other hand, showed consistent protective effect of habitual coffee drinking on 2 hour postprandial blood glucose level regardless of sugar use, but the association was slightly attenuated (from -8.8% to -8.4%) with adjustment on added sugar intake, suggesting possible contribution of the use of sugar in the risk reduction.

In a systematic review, van Dam *et al*[26] showed that habitual coffee consumption, particularly of decaffeinated coffee, was linked to a substantially lower risk of developing type 2 diabetes with a RR in the highest vs. lowest quintile of consumption of coffee = 0.65. The authors suggested that components of coffee such as CGA[17,18,27] and quinides[28] improved either the postprandial glycemic response and/or insulin sensitivity, contributing to the lower risk of T2DM.[19] In the present study, we demonstrated a significantly lower baseline plasma glucose concentration ($p = 0.007$) in DECAF vs. CON, supporting the view that compounds in coffee may be producing beneficial effects. However, this did not extend to the whole postprandial period, there being no difference between the glucose and insulin scores of DECAF vs. CON. This is in contrast to the findings of Battram *et al*,[16] who showed decaffeinated coffee resulted in a significantly lower blood glucose response and significantly higher ISI. A possible explanation could be the higher volume (~500 mL) of decaffeinated coffee consumed in their study, which may have provided more active compounds. It is conceivable that CGA must be present in the small intestinal lumen simultaneously with the products of starch digestion in order to inhibit glucose absorption.[17] Our 'pre-meal' study design, together with the low volume of DECAF consumed (resulting in lower CGA content) would preclude this possibility.

A particular strength of our study was the deliberate use of fingertip blood sampling. The site of sampling has an important effect of blood glucose variability and on the ability to detect rapid changes in blood glucose as occurs after a carbohydrate-containing meal. Ellison *et al*[29] reported that finger tip blood samples may identify changes in blood glucose immediately after a meal more readily than that from forearm and thigh sites. In recent research employing fingertip sampling, we were able to document clinically useful effects of alcoholic beverages on postprandial glycemia,[30] differences that were probably undetectable in earlier studies employing venous sampling. In the present study, we recruited normal healthy young subjects using a pre-meal design that represented realistic food patterns, i.e., consuming coffee between meals. One cup of coffee or tea followed by 75 g carbohydrate portion is a physiological amount, an average person consuming 240 g carbohydrate per day. These attributes increase the generalisability of the findings but the small number of subjects ($n = 8$) and the fact that most were of Chinese ethnicity suggests caution. Young lean healthy subjects of Asian origin display higher postprandial glycemic and insulinemia than their Caucasian counterparts[31] and there may be differences in insulin sensitivity and postprandial glycemia between the two groups. Thus the findings should be confirmed in other groups.

In summary, the hypothesis that coffee consumption 1-hour before meal would reduce postprandial glycemia and insulinemia was not supported by the current findings. Indeed, prior coffee consumption increased postprandial glycemia by 28%. However, the observation that addition of a small amount of sucrose (10 g) to coffee reverses this effect is novel and noteworthy, suggesting that greater attention be given to food combinations and interactions in clinical and epidemiological research.

ACKNOWLEDGEMENTS

We would like to thank Kai Lin Ek, and Karola Stockmann for their technical assistance in the testing kitchen and laboratory.

AUTHOR DISCLOSURES

Professor Jennie Brand-Miller serves on the board of directors of Glycemic Index Ltd, a non-profit company that administers a food labeling program in Australia (www.gisymbol.com.au), as director of a not-for-profit GI testing service at the University of Sydney www.glycemicindex.com and is the co-author of a series of books under the title 'The New Glucose Revolution' (Marlowe and Co., USA). Jimmy Chun-Yu Louie, Fiona Atkinson and Peter Petocz, no conflict of interests.

REFERENCES

1. van Dam RM, Feskens EJ. Coffee consumption and risk of type 2 diabetes mellitus. Lancet. 2002;360:1477-8.
2. Agardh EE, Carlsson S, Ahlbom A, Efendic S, Grill V, Hammar N, Hilding A, Ostenson C. Coffee consumption, type 2 diabetes and impaired glucose tolerance in Swedish men and women. J Intern Med. 2004;255:645-52.
3. Hu G, Jousilahti P, Peltonen M, Bidel S, Tuomilehto J. Joint association of coffee consumption and other factors to the risk of type 2 diabetes: a prospective study in Finland. Int J Obes. 2006;30:1742-9.
4. Iso H, Date C, Wakai K, Fukui M, Tamakoshi A. The relationship between green tea and total caffeine intake and risk

for self-reported type 2 diabetes among Japanese adults. Ann Intern Med. 2006;144:554-62.
5. 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 June 26, 2006;166:1311-6.
6. Rosengren A, Dotevall A, Wilhelmsen L, Thelle D, Johansson S. Coffee and incidence of diabetes in Swedish women: a prospective 18-year follow-up study. J Intern Med. 2004; 255:89-95.
7. 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.
8. Tuomilehto J, Hu G, Bidel S, Lindstrom J, Jousilahti P. Coffee consumption and risk of type 2 diabetes mellitus among middle-aged Finnish men and women. JAMA. 2004, 2004;291:1213-9.
9. 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;29:398-403.
10. van Dam RM, Dekker JM, Nijpels G, Stehouwer DA, Bouter LM, Heine RJ. Coffee consumption and incidence of impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes: the Hoorn Study. Diabetologia. 2004;47: 2152-9.
11. Yamaji T, Mizoue T, Tabata S, Ogawa S, Yamaguchi K, Shimizu E, Mineshita M, Kono S. Coffee consumption and glucose tolerance status in middle-aged Japanese men. Diabetologia. 2004;47:2145-51.
12. Keijzers GB, De Galan BE, Tack CJ, Smits P. Caffeine can decrease insulin sensitivity in humans. Diabetes Care. 2002; 25:364-9.
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. 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.
15. Arnlov J, Vessby B, Riserus U. Coffee consumption and insulin sensitivity. JAMA. 2004;291:1199-201.
16. 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.
17. Johnston KL, Clifford MN, Morgan LM. Coffee acutely modifies gastrointestinal hormone secretion and glucose tolerance in humans: glycemic effects of chlorogenic acid and caffeine. Am J Clin Nutr. 2003;78:728-33.
18. Arion WJ, Canfield WK, Ramos FC, Schindler PW, Burger HJ, Hemmerle H, Schubert G, Below P, Herling AW. Chlorogenic acid and hydroxynitrobenzaldehyde: New inhibitors of hepatic glucose-6-phosphatase. Arch Biochem Biophys. 1997;339:315-22.
19. Higdon JV, Frei B. Coffee and health: a review of recent human research. Crit Rev Food Sci Nutr. 2006;46:101-23.
20. McCarty MF. A chlorogenic acid-induced increase in GLP-1 production may mediate the impact of heavy coffee consumption on diabetes risk. Med Hypotheses. 2005;64:848-53.
21. Dufresne CJ, Farnworth ER. A review of the latest findings on the health promotion properties of tea. J Nutr Biochem. 2001;12:404-21.
22. Gardner EJ, Ruxton C.H.S., Leeds AR. Black tea - helpful or harmful? A review of the evidence. Eur J Clin Nutr. 2007; 61:3-18.
23. Brouns F, Bjorck I, Frayn K, Gibbs A, Lang V, Slama G, TMS W. Glycaemic index methodology. Nutr Rev. 2005;18: 145-71.
24. FAO/WHO. The role of glycemic index in food choice in Carbohydrates in Human Nutrition: Report of a Joint FAO/WHO Expert Consultation. Rome: WHO; 1998. p. 25-37.
25. Heacock PM, Hertzler SR, Wolf BW. Fructose prefeeding reduces the glycemic response to a high-glycemic index, starchy food in humans. J Nutr. 2002;132:2601-4.
26. van Dam RM, Hu FB. Coffee consumption and risk of type 2 diabetes: A systematic review. JAMA. 2005;294:97-104.
27. Wu T, Willett WC, Hankinson SE, Giovannucci E. Caffeinated Coffee, Decaffeinated Coffee, and Caffeine in Relation to Plasma C-Peptide Levels, a Marker of Insulin Secretion, in U.S. Women. Diabetes Care. 2005;28:1390-6.
28. 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.
29. Ellison JM, Stegmann JM, Colner SL, Michael RH, Sharma MK, Ervin KR, Horwitz DL. Rapid changes in postprandial blood glucose produce concentration differences at finger, forearm, and thigh sampling sites. Diabetes Care. 2002;25: 961-4.
30. Brand-Miller JC, Fatima K, Middlemiss C, Bare M, Liu V, Atkinson F, Petocz P. Effect of alcoholic beverages on postprandial glycemia and insulinemia in lean, young, healthy adults. Am J Clin Nutr. 2007;85:1545-51.
31. Dickinson S, Colagiuri S, Faramus E, Petocz P, Brand-Miller JC. Postprandial hyperglycemia and insulin sensitivity differ among lean young adults of different ethnicity. J Nutr. 2002;132:2574-9.

Original Article

Delayed effects of coffee, tea and sucrose on postprandial glycemia in lean, young, healthy adults

Jimmy Chun Yu Louie MNutrDiet[1], Fiona Atkinson MNutrDiet[1], Peter Petocz PhD[2], Jennie C Brand-Miller PhD[1]

[1]*Human Nutrition Unit, School of Molecular and Microbial Biosciences, The University of Sydney, Sydney, Australia*
[2]*Department of Statistics, Macquarie University, Sydney, Australia*

咖啡或茶與蔗糖對健康不過重的年輕人餐後血糖之延緩效應

在觀察研究中，發現長期飲用咖啡和較低的 2 型糖尿病風險相關。我們假設降低糖尿病風險的機制可能與咖啡對餐後血糖的延後效應有關。本研究目的是探討食用高碳水化合物餐點前 1 小時飲用一般咖啡(含糖或不含糖)、去咖啡因咖啡、紅茶及糖水對血糖和胰島素反應的影響。在隨機的分別場次中，8 名健康苗條的年輕人於飲用 250 毫升的咖啡(COF)、咖啡加 10 克蔗糖(COF+SUC)、去咖啡因咖啡(DECAF)、紅茶(TEA)、糖水(含 10 克蔗糖；SUC)或熱水(CON)的 1 小時後，進食了以馬鈴薯為主的餐點。餐後 2 小時期間，定時從指尖抽取血液樣本，量化血糖和胰島素的濃度曲線下面積做為血糖和胰島素的反應。跟對照組(CON)比較，COF 令餐後血糖反應上升了 28% (p=0.022)。相反地，COF+SUC 相比於 COF (-38%，p<0.001)或對照組(-20%，p=0.100)都減低了餐後血糖反應，但不影響胰島素反應。去咖啡因咖啡、紅茶及糖水對餐後血糖及胰島素反應並沒有顯著的影響。糖水和去咖啡因咖啡減低了開始用餐時的血糖濃度(p<0.01)。總括來說，只有含糖咖啡(COF+SUC)顯著地降低餐後血糖反應。本研究結果或許可以解釋有關咖啡的飲用及糖尿病風險的觀察研究及臨床實驗中矛盾的發現。

關鍵字：咖啡、茶、餐後高血糖反應、高血胰島素、咖啡因