PJN
ISSN 1680-5194
ansinet.com/pjn

PAKISTAN JOURNAL OF NUTRITION

Science Alert
scialert.net

ANSInet
an open access publisher
http://ansinet.com

OPEN ACCESS Pakistan Journal of Nutrition

ISSN 1680-5194
DOI: 10.3923/pjn.2020.420.429

Review Article
The Effect of Coffee Consumption on Blood Glucose: A Review

Amal Hassan Alshawi

Department of Nutrition and Food Science, Princess Nourah Bint Abdulrahman University, Riyadh, Kingdom of Saudi Arabia

Abstract

This review describes the impact of drinking coffee on glycemic profile parameters (glycemic index [GI], glycemic load [GL], glucose tolerance and insulin sensitivity) and diabetes mellitus. Coffee is very popular in Arab communities including Saudi Arabia. Clinical research has revealed the negative effect of caffeine including a reduction in insulin sensitivity that impairs glucose tolerance. Epidemiological studies also show that drinking coffee has positive effects on both glucose tolerance and sensitivity to insulin, which might assist in reducing the risk of type 2 diabetes especially over long periods of consumption. More studies are thus needed to gain a deeper understanding of how coffee drinking is linked to type 2 diabetes.

Key words: Caffeine, diabetes mellitus, insulin sensitivity, glucose tolerance, chlorogenic acid, coffee consumption

Citation: Amal Hassan Alshawi, 2020. The effect of coffee consumption on blood glucose: A review. Pak. J. Nutr., 19: 420-429.

Corresponding Author: Amal Hassan Alshawi, Department of Nutrition and Food Science, Princess Nourah Bint Abdulrahman University, Riyadh, Kingdom of Saudi Arabia

Competing Interest: The author has declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

The word 'coffee' derives from the Arabic word '*quahweh*,' leading to the Latin botanical genus *coffea*. Of the coffee which is actually drunk, 99% comes from just two of the 103 kinds of coffee that have been discovered: *Coffea arabica* (Arabica) and *Coffea canephora* (Robusta). Coffee growing occurs in sixty tropical and subtropical nations. Around 60% of the coffee produced globally is grown on the American continent. Here, Arabica coffee with its low caffeine content and bitterness predominates[1]. Golden coffee, which comprises around 95% of all coffee exports, is produced through minimal processing of the green coffee beans[2]. Coffee is very common in many countries with the highest per capita consumption being in Europe where the average consumption is between 2.4 and 12 kg per person per year[3].

Coffee contains caffeine, which is a bioactive compound. It stimulates the central nervous system and positively affects long-term memory. Drinking coffee has historically been associated with negative health effects, more recent studies show that it may be beneficial[1]. Consuming approximately ≤400 mg of caffeine daily (3-4 cups) of brewed coffee or having five cups of tea or five caffeinated soft drinks regard as moderate seems to affect health in a neutral to positive way. Adolescents can safely consume 100-175 mg of caffeine per day while the safe level for children aged 6-12 is 45-85 mg[4]. In Saudi Arabia, the term 'Arabic coffee' is usually applied to a green coffee bean extract that is very popular. Spices such as saffron and cardamom are added to the roasted green coffee beans to enhance the color and taste of the resulting coffee product. In Saudi Arabia, as in other Arab Gulf countries, coffee is usually drunk with snacks especially soft dates.

The efficiency of consuming brewed coffee on human health has been the focus of many studies[5]. This review aims to evaluate how drinking coffee affects blood glucose and the implications for insulin sensitivity and glucose tolerance as well as its impact on diabetes.

Search strategy: Databases were searched for a randomized controlled trial that examined the effects of coffee consumption on blood glucose in healthy humans. The following keywords in the title/abstract were used: caffeine, diabetes mellitus, insulin sensitivity, glucose tolerance, chlorogenic acid and coffee consumption. The reference lists from selected reports were reviewed for further relevant studies. Studies that investigated the effect of different coffee drinks are included because coffee consumption occurs more frequently than pure caffeine intake in daily life.

A review of the literature was conducted utilizing databases such as PubMed, Google Scholar, LWW Health Library and Science Direct. Furthermore, studies cited in the selected articles were verified. Studies on association of caffeine, coffee consumption and diabetes were included.

General properties of coffee: When coffee beans are ripe, they can be processed by the 'wet' method or by drying in the sun for 3-9 days (the 'dry' method). A temperature of 200°C is required for the necessary transformation to take place during roasting. When this occurs, the coffee beans become dry, brittle and brown; they increase in size and develop their characteristic aroma and flavor. Regardless of whether coffee is prepared on an industrial scale or brewed at home, hydrous extraction of the dissolved roast and ground coffee is involved[6].

The coffee quality is determined by the chemical composition of the roasted beans, which is also affected by drying, storing, roasting and grinding after the harvest. The coffee bean quality relies on color, shape and size of the bean, the crop year, the processing methods, the roast potential and the processing method as well as the flavour[7]. Roasting is vital because the chemical compounds that are generated by the changes that happen during the roasting process are what gives coffee its characteristic flavour[8].

Recent research has demonstrated that coffee and its by-products are rich in antioxidants. They contain trigonelline and chlorogenic acids as well as caffeine, which are all bioactive compounds[9]. These compounds have a positive effect on health and this means that coffee is potentially a functional food product[2].

The coffee plant has a secondary metabolite which is caffeine, (1, 3, 7-trimethylxanthine)-a purine alkaloid. The caffeine content in the green coffee beans varies. If calculated by dry weight, it represents up to 2.3% of Robusta beans and up to 1.3% of Arabica beans. Coffee beans also contain fairly large amounts of chlorogenic acids, which are widely distributed secondary metabolites in plants with 5-O-caffeoylquinic acid being the most common[6]. After investigating the effects of various methods of brewing on the polyphenol, methylxanthine and antioxidant capacities of 13 different brews of coffee, Baeza *et al.*[10] concluded that their antioxidant capacities complied with the total phenol and chlorogenic acid derivate contents. Wachamo *et al.*[11] suggested that the high phenolic content of green coffee probably accounted for its association with the reduced threat of diseases of oxidative etiology. The impact of drinking coffee on various health consequences was reviewed by Bordenave *et al.*[12] who concluded that it benefitted carbohydrate and lipid metabolism as well as the cardiovascular system.

The blood glucose response to foods: Currently, the focus in the glycemic response (GR) to foods is of considerable interest because it is thought to have a relationship with chronic diseases including obesity, diabetes and cardiovascular disease[13]. GR to a food or meal for a person is specified by the amount and quality of the carbohydrates[14] and this can be ascertained by the GI, GL, or glycemic impact[15]. The concept of the GI was first coined in 1981 by Augustin *et al.*[13] in their research with patients who had type 2 diabetes mellitus. The carbohydrate content of specific foods is evaluated by the GI by measuring their glycemic effects after they have been ingested[16,17]. The GI could be demonstrated as the incremental area beneath the curve of the blood glucose response to a 50 g portion of available carbohydrate of a tested sample. It is expressed as a percentage of the same person reaction after eating an identical carbohydrate portion from standard food. The variability and/or value of the GI results can be influenced by a number of methodological factors; however, variation can be minimized and reproducible findings obtained by careful use of appropriate methods[18].

The Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) outlines the most appropriate method for calculating the GI[19]. The FAO, in its consultation report on dietary carbohydrates in human nutrition, suggests using GI values as well as other data on food composition[19]. Low GI foods like legumes and low-fat animal products are recommended as part of many weight-loss diets[20]. All published data on the GI values of 600 particular foods was compiled and listed in a single table by Fiona *et al.*[20].

To assist in the creation of a local food exchange list for diabetics, Al-Mssallem[21] identified the GI value of popular Saudi foods. Fohl, gareesh, korsan, kelija eneazaa and kelija malkee are five common Saudi foods with high carbohydrate content and were evaluated for their GI values (45, 89, 61, 58 and 51%, respectively). Alkaabi *et al.*[22] assessed the GI values of five popular types of dates in both diabetic and healthy individuals. Their findings demonstrated a low GI value for all five types of dates and the authors concluded that the dates would not cause a significant rise in the concentration of postprandial glucose if eaten by diabetics. Farhat *et al.*[23] investigated the GI values of Lebanese mixed meals and desserts and found that they ranged from intermediate (50-70%) to low (≤50%). A study on the GI values of breakfast cereals in the diet of United Kingdom (UK) citizens showed that these ranged from high (≥70%) to low (≤55%); most had a moderate GI of between 60 and 65%[24].

To measure GL, the entire amount of ingested carbohydrates (in grams) is multiplied by the GI value of each food and then divided by 100[25]. When the effect of GI/GL on appetite and GR were identified, there were no significant differences in appetitive rating, plasma glucose, insulin response, or food consumption[26]. The GR is modulated by several aspects of the meal including the nutrient composition, the duration, volume/weight, energy density, rheology and palatability[27,28].

The GR is a food's capacity to raise blood sugar[29]. The way that food has been processed or cooked affects GR[30]. Because dietary fats slow the absorption of glucose, they may delay the blood glucose and insulin responses[31,32]. Bataineh investigated the glycemic and insulinemic indices of some popular Arabic sweets when ingested by healthy individuals and found that substituting olive oil for ghee in maa'moul, ghuraybah and hareesah lowered GRs without a significant effect on the insulinemic responses[33]. The blood glucose response of foods containing differing amounts of carbohydrates can be compared directly with the glycemic glucose equivalent (GGE); the glucose quantity in grams would result in a GR equal to a particular weight of a food[34,35]. Observing the glucose continuously might be used to record the GR with levels of interstitial glucose reported every 5 to 10 minutes continuously for 3-7 days for a comprehensive observation of the GR of a single meal or food[36].

Due to lack and/or resistance of insulin, type 2 diabetes mellitus is linked with the target tissues' impaired insulin response. Although increased insulin secretion overcomes insulin resistance at first, this compensation ultimately fails and results in progressively raised blood glucose levels. Before people develop type 2 diabetes, they go across a stage of impaired glucose tolerance (IGT) and increased plasma glucose levels during fasting; thus, this stage is an intermediate dysglycemia state between diabetes and normal glucose tolerance[37]. The American Diabetes Association proposed a new diagnostic criterion in 1997[38] whereby diabetes screening was carried out by testing plasma glucose levels during fasting[39]. The importance of measuring insulin sensitivity is frequently highlighted due to its important role in diabetes, cardiovascular and hypertension disease[40]; tools to measure insulin sensitivity and resistance is a key goal of many studies[41-43]. Song *et al.*[44] studied the relationship between insulin resistance and dietary behavior among healthy people in Korea: They found that a pattern based on beans and whole grains was linked with a lower prevalence of insulin resistance.

The effect of coffee consumption on the glycemic profile: If people are to make informed choices about drinking coffee and public health initiatives are to be prioritized appropriately, then it is important to know the benefits and adverse effects

on health[45]. The relationship between coffee components like antioxidant phenolic compounds, caffeine, micronutrients and fiber suggests beneficial effects[3]. According to Van Dam *et al.*[45] coffee contains a number of substances that may impact glucose metabolism.

The Hoorn[45] study was a cross-sectional and prospective study that was population-based and involved Dutch male and female adults with a range of age 50-74 years. Initially, an oral glucose tolerance test (OGTT) was conducted and a follow-up was performed during an average period of six years and four months. Some variables such as cigarette smoking, alcohol intake, body mass index (BMI), physical activity and dietary factors were adjusted. The results showed that habitual coffee-drinking can lower the threat of IGT and impact post-load rather than fasting glucose metabolism[45]. A link between habitual coffee-drinking and the incidence of increased plasma glucose levels during fasting, IGT and type 2 diabetes was assessed.

Caffeine is one of the substances other than macronutrients that can affect insulin sensitivity and blood glucose concentrations in individuals with diabetes[46]. Despite research carried out on short terms with people in good health that continue to indicate that ingesting caffeine cause acute transient insulin resistance and IGT[47,48], these conclusions appear to be contradicted by epidemiological studies[46]. For an individual with existing diabetes, blood glucose concentrations may be affected by caffeine in various ways. Glucose transportation from blood to the muscles may be hindered by caffeine as it acts as an adenosine receptor antagonist to inhibit the uptake of glucose into muscle cells even when insulin is present[49]. Moreover, elevated blood glucose concentrations due to caffeine intake could be a consequence of high epinephrine (adrenaline), which in turn induces insulin resistance[50,51]. Other studies have also shown how caffeine might impact glucose metabolism. Luiz *et al.*[52] found that caffeine intake led to a significant increase in blood glucose concentrations. Robinson *et al.*[53] and Lee *et al.*[54] confirmed that ingesting caffeine increased glucose concentration and loaded when compared to a placebo. Both studies noted that caffeine caused a reduction in the insulin sensitivity index compared to placebo.

Several studies have addressed the short-term and long-term effects of coffee or caffeine consumption on glucose tolerance[55]; they concluded that coffee consumption and/or caffeine impaired glucose tolerance in the short-term[56,57]. IGT is a state of hyperglycemia where resistance to insulin sensitivity happens in peripheral tissues as a response to glucose[58]. Insulin resistance and glucose tolerance can be impaired by short-term administration of coffee, which blocks the impact of the adenosine AI receptor that regulates the uptake of glucose in skeletal muscles[59]. However, the findings from some epidemiological studies demonstrate that long-term and habitual coffee drinking might support standard glucose tolerance and enhance insulin sensitivity[60-63]. Thus, coffee may suppress insulin sensitivity in the short-term but drinking coffee regularly can stimulate glucose tolerance and insulin sensitivity[64-66]. In other words, ingesting caffeine habitually changes the negative impact of caffeine on glucose tolerance and insulin sensitivity to a positive one[55].

Consuming coffee and caffeine were linked to lower serum levels of leptin and plasminogen activator inhibitor-1[67]. In elderly individuals, low coffee consumption for short periods predicts impairment of normal glucose tolerance[68] while also lowering glucose tolerance in healthy men[69]. However, other studies report different results: Feinberg *et al.*[70] demonstrated that subjects showed a marked reduction in blood glucose concentration after drinking coffee although insulin levels were not affected. Shengxi *et al.*[71] noted that just the intake of coffee polyphenols enhances peripheral endothelial function after glucose loading among adults in good health. Thom[72] reported that instant coffee enriched with chlorogenic acid lowered glucose absorption by 6.9% versus a control.

A lowered insulin sensitivity after short-term ingestion of coffee[72-74] may be due to the caffeine-induced antagonism of adenosine receptors accompanied by a rise in levels of epinephrine[75]. For individuals with type 2 diabetes mellitus, the impact of drinking black espresso coffee does not seem to be mediated by alterations in insulin sensitivity[76]. For the study of insulin secretion and sensitivity, long-term trials on the impact of caffeine and some components of coffee could be more related. A cross-sectional study of 936 male senior citizens without type 2 diabetes revealed that drinking coffee was linked to higher insulin sensitivity but not a reduced secretion of insulin[61]; however, a cross-sectional study of 2112 healthy women found a potential decrease of insulin secretion as the result of consuming coffee. It is possible that this decrease could be relevant to a constituent in coffee other than caffeine[62].

Recent research reported no alteration in postprandial glycemic response to Lebanese mankoucheh when accompanied by Turkish coffee[77.] Post-exercise insulin concentrations and blood glucose were significantly changed by caffeine and extract from green beans of coffee versus placebo[78].

Type 2 diabetes and consuming coffee: The global increase in diabetes seems to be due to population ageing and urbanization with associated alterations in physical activity

and diet[79]. Specific criteria and diagnostic tests were used to identify people who are pre-diabetic and thus at risk of developing diabetes as well as those who have diabetes[80].

Diabetes is classified into type 1, type 2, gestational and secondary diabetes[80]. The vast majority of diabetics (90-95%) have type 2, which used to be called non-insulin-dependent or adult-onset diabetes[81]. Preventing type 2 diabetes is now a significant public health issue. Type 2 diabetes can largely be avoided by a healthy life style and a sound dietary pattern[82]. Treatment of diabetes is focused on normalizing metabolism with an emphasis on blood glucose and lipids especially low-density lipoprotein (LDL), cholesterol and blood pressure to prevent complications that are diabetes-related[81].

Coffee contains a significant amount of caffeine and chlorogenic acid and is a complex mix of chemicals. Over the last few decades, studies have identified the benefit or harm to health from drinking coffee[83]. Coffee may impact postprandial hyperglycemic excursion, although there have been conflicting results. Drinking coffee raises energy expenditure and thermogenesis to induce insulin sensitivity[84]. Coffee can produce an acute impairment of glucose tolerance or insulin resistance in healthy, non-diabetic adults and has been consistently demonstrated by a minimum of 17 short-term studies. We concluded that this effect contributes to the progression of type 2 diabetes in susceptible people[85].

Conversely, long-term coffee intake is possibly linked with a lowered threat of type 2 diabetes[86,87]. Alkaabi *et al.*[88] suggested that eating dates with coffee was common among Arabs and that this might affect hyperglycemic excursion after meals. They studied the effect of coffee on the GI tract of type 2 diabetic and healthy subjects when eating a common variety of dates. They found that coffee did not adversely impact capillary glucose levels after the consumption of dates either in diabetic or healthy subjects at least in the short-term[88]. Information about the daily coffee consumption of a sample of 1141 American Indian males and females with a range of age 45-74 years was collected in a prospective cohort study. The sample was within the normal average of glucose tolerance at the baseline examination and was monitored with a mean of 7.6 years. The findings indicated that subjects who consumed more than 11 cups of coffee per day had a 67% lower threat of developing diabetes through the follow-up period compared to non-coffee consumers[89]. Another prospective study followed 910 adults aged ≥50 years with a mean of 8 years after their coffee consumption had been assessed. These findings indicated that both former and current coffee consumers had a reduced risk of diabetes incident versus people who never ingest coffee. The subjects with impaired baseline glucose who were former and current coffee consumers also had a lower risk of diabetes. This research strikingly demonstrates how caffeinated drinking coffee can protect against the incidence of diabetes[63].

Furthermore, according to Hamer *et al.*[90], more than fourteen cohort studies have revealed a reverse association between drinking coffee beverages and the threat of type 2 diabetes. Hamer *et al.*[90] investigated the prospective association between consuming coffee and tea beverages and the threat of type 2 diabetes mellitus. The study was conducted for 11.7 years of follow-up with a sample of 4055 males and 1768 females from the Whitehall cohort in the UK; they concluded that drinking a moderate amount of coffee and tea (more than 3 cups a day) was not prospectively linked with the incidence of type 2 diabetes[90]. A cross-sectional study of 954 non-diabetic adults who drink caffeinated coffee on a regular basis based on a food frequency questionnaire found a positive link to insulin sensitivity that was inversely related to 2 h post-load glucose levels[91]. Dutch male and female adults were followed for a mean duration of 6.4 years in another prospective cross-sectional study. They found that drinking coffee favored post-load rather than fasting glucose[45]. Research conducted with 2434 Finnish men and women reported that drinking coffee had a significant and inverse link with both fasting glucose and insulin and also with the response to a 2 h glucose tolerance test[55,92].

A review of these epidemiologic studies showed that drinking coffee can significantly decrease the threat of type 2 diabetes[93-96]. Akash *et al.*[55] found a number of mechanisms that might be involved. Ryanodine receptors are activated by caffeine and this improves the insulin secretion of β-cells of the pancreatic islets. Glucose metabolism and homeostasis are regulated by chlorogenic acid and magnesium[55]. Chu *et al.*[97]. reported numerous compounds in coffee that may produce a matrix effect by working together and providing bioactivities that lower the threat of evolving type 2 diabetes. Finally, coffee consumption is linked with the threat of diabetes mellitus, the occurrence of stroke, heart failure and some cancers in an inverse dose-dependent fashion; however, there may be harmful effects related to high anxiety, insomnia and acute myocardial infarction[97,98]. However, until the association between the threat of type 2 diabetes and long-term coffee consumption is more thoroughly understood, it would be premature to suggest coffee intake consumption as a way of preventing type 2 diabetes mellitus[55,99,100].

CONCLUSIONS AND RECOMMENDATIONS

Generally, the literature on short-term clinical trials that addressed the link between consuming coffee and type 2 diabetes mellitus shows that caffeine decreases insulin sensitivity and negatively affects glucose tolerance. Nevertheless, several prospective cohort studies in various countries have demonstrated that drinking coffee is linked with a significant decrease in the threat of developing type 2 diabetes mellitus. More studies are required to clearly understand how the main components of coffee affect glucose metabolism. Moreover, epidemiologic research needs to be carried out in Arab populations and specifically in the Saudi community to accurately identify how drinking coffee affects blood glucose and other aspects of health. Effective strategies in clinical research and related epidemiological studies are needed to clarify the effect of coffee consumption for people with type-diabetes especially in the local contest. The use of coffee drinks could be a practical, effective and environmentally friendly method to enhance insulin sensitivity.

REFERENCES

1. Mejia, E.G.d. and M.V. Ramirez-Mares, 2014. Impact of caffeine and coffee on our health. Trends Endocrinol., Metabo., 25: 489-492.
2. Ribeiro, V.S., A.E. Leitão, J.C. Ramalho and F.C. Lidon, 2014. Chemical characterization and antioxidant properties of a new coffee blend with cocoa, coffee silverskin and green coffee minimally processed. Food Res. Int., 61: 39-47.
3. Pimentel, G.D., T.O. Micheletti, R.C. Fernandes and A. Nehlig, 2019. Coffee Intake and Obesity. In: Nutrition in the Prevention and Treatment of Abdominal Obesity. Watson, R.R. (Ed.). Elsevier, UK, pp: 329-351.
4. Mitchell, D.C., C.A. Knight, J. Hockenberry, R. Teplansky, T.J. Hartmand, 2014. Beverage caffeine intakes in the U.S. Food and Chem. Toxicol., 63: 136-142.
5. Ochiai, R., Y. Sugiura, Y. Shioya, K. Otsuka, Y. Katsuragi and T. Hashiguchi, 2014. Coffee polyphenols improve peripheral endothelial function after glucose loading in healthy male adults. Nutr. Res., 34: 155-159.
6. Pimpley, V., S. Patil, K. Srinivasan, N. Desai and P.S. Murthy, 2020. The chemistry of chlorogenic acid from green coffee and its role in attenuation of obesity and diabetes. Prep. Biochem. Biotechnol., 10.1080/10826068.2020.1786699
7. Lee, L.W., M.W. Cheong, P. Curran, B. Yu and S.Q. Liu, 2015. Coffee fermentation and flavor – An intricate and delicate relationship. Food Chem., 185: 182-191.
8. Giacalone, D., T.K. Degn, N. Yang, C. Liu, I. Fisk and M. Münchow, 2019. Common roasting defects in coffee: Aroma composition, sensory characterization and consumer perception. Food Qual. Preference, 71: 463-474.
9. Muñoz, A.E., S.S. Hernández, A.R. Tolosa, S.P. Burillo and M.O. Herrera, 2020. Evaluation of differences in the antioxidant capacity and phenolic compounds of green and roasted coffee and their relationship with sensory properties. Food Sci. Technol., Vol. 128 10.1016/j.lwt.2020.109457
10. Baeza, G., M. Amigo-Benavent, B. Sarriá, L. Goya, R. Mateos and L. Bravo, 2014. Green coffee hydroxycinnamic acids but not caffeine protect human HepG2 cells against oxidative stress. Food Res. Int., 62: 1038-1046.
11. Wachamo, H.L., Vol. 6, No. 4 2017. Review on Health Benefit and Risk of Coffee Consumption. Med. Aromat. Plants, 10.4172/2167-0412.1000301
12. Bordenave, N., L.B. Kock, M. Abernathy, J.C. Parcon, A.A. Gulvady, B.J.W.v, Klinken and P. Kasturi 2015. Toward a more standardised and accurate evaluation of glycemic response to foods: Recommendations for portion size calculation. Food Chem., 167: 229-235.
13. Augustin, L.S., C.W. Kendall, D.J. Jenkins, W.C. Willett and A. Astrup *et al*., 2015. Glycemic index, glycemic load and glycemic response: An International Scientific Consensus Summit from the International Carbohydrate Quality Consortium (ICQC). Nutr. Metab. Cardiovasc. Dis., 25: 795-815.
14. Hardy, D.S., J.T. Garvin and H. Xu, 2020. Carbohydrate quality, glycemic index, glycemic load and cardiometabolic risks in the US, Europe and Asia: A dose–response meta-analysis. Nutr. Metab. Cardiovasc. Dis., 30: 853-871.
15. Salari-Moghaddam, A., A.H. Keshteli, F. Haghighatdoost, A. Esmaillzadeh and P. Adibi, 2019. Dietary glycemic index and glycemic load in relation to general obesity and central adiposity among adults. Clin. Nutr., 38: 2936-2942.
16. Schwingshackl, L. and G. Hoffmann, 2013. Long-term effects of low glycemic index/load vs. high glycemic index/load diets on parameters of obesity and obesity-associated risks: A systematic review and meta-analysis. Nutr. Metab. Cardiovasc. Dis., 23: 699-706.
17. Singh, A., P. Raigond, M.K. Lal, B. Singh and N. Thakur, 2020. Effect of cooking methods on glycemic index and *in vitro* bioaccessibility of potato (*Solanum tuberosum* L.) carbohydrates. LWT-Food Sci. Technol., Vol. 127 10.1016/j.lwt.2020.109363
18. Mann, J., J.H. Cummings, H.N. Englyst, T. Key and S. Lui *et al*., 2007. FAO/WHO scientific update on carbohydrates in human nutrition: Conclusions. Eur. J. Clin. Nutr., 61: S132-S137.
19. Gibson, N., H.C. Schönfeldt and B. Pretorius, 2011. Development of a rapid assessment method for the prediction of the glycemic index. J. Food Compos. Anal., 24: 750-754.

20. Atkinson, F.S., K. Foster-Powell and J.C. Brand-Miller, 2008. International tables of glycemic index and glycemic load values: 2008. Diabetes Care, 31: 2281-2283.
21. Al-Mssallem, M.Q., 2014. The association between the glycaemic index of some traditional saudi foods and the prevalence of diabetes in Saudi Arabia: A review article. J. Diabetes Metab., Vol. 5. 10.4172/2155-6156.1000452
22. Alkaabi, J.M., B. Al-Dabbagh, S. Ahmad, H.F. Saadi, S. Gariballa and M. Al Ghazali, 2011. Glycemic indices of five varieties of dates in healthy and diabetic subjects. Nutr. J., Vol. 10. 10.1186/1475-2891-10-59
23. Farhat, A.G., S.R. Moukarzel, R.J. El-Said and C.F. Daher, 2010. Glycemic index of commonly consumed lebanese mixed meals and desserts. Asian J. Clin. Nutr., 2: 48-57.
24. Aston, L.M., J.M. Gambell, D.M. Lee, S.P. Bryant and S.A. Jebb, 2008. Determination of the glycaemic index of various staple carbohydrate-rich foods in the UK diet. Eur. J. Clin. Nutr., 62: 279-285.
25. AlGeffari, M.A., E.S. Almogbel, T.A. Homaidan, R. El-Mergawi and I.A. Barrimaha, 2016. Glycemic indices, glycemic load and glycemic response for seventeen varieties of dates grown in Saudi Arabia. Ann. Saudi Med., 36: 397-403.
26. Brouns, F., I. Bjorck, K.N. Frayn, A.L. Gibbs, V. Lang, G. Slama and T.M.S. Wolever, 2005. Glycaemic index methodology. Nutr. Res. Rev., 18: 145-171.
27. Camps, G., M. Mars, C.d. Graaf and P.A. Smeets, 2016. Empty calories and phantom fullness: a randomized trial studying the relative effects of energy density and viscosity on gastric emptying determined by MRI and satiety. Am. J. Clin. Nutr., 104: 73-80.
28. Pribic, T., L. Hernandez, A. Nieto, C. Malagelada, A. Accarino and F. Azpiroz, 2018. Effects of meal palatability on postprandial sensations. Neurogastroenterology Motil., Vol. 30, No. 2 10.1111/nmo.13248
29. Farvid, M.S., F. Homayouni, M. Shokoohi, A. Fallah and M.S. Farvid, 2014. Glycemic index, glycemic load and their association with glycemic control among patients with type 2 diabetes. Eur. J. Clin. Nutr., 68: 459-463.
30. Marques, A.M., B.S. Linhares, R.D. Novaes, M.B. Freitas, M.M. Sarandy and R.V. Gonçalves, 2020. Effects of the amount and type of carbohydrates used in type 2 diabetes diets in animal models: A systematic review. PLos ONE, Vol. 15, No. 6 10.1371/journal.pone.0233364
31. Fernandez, M.A. and A. Marette, 2020. Dairy Products and Diabetes: Role of Protein on Glycaemic Control. In: Milk and Dairy Foods: Their Functionality in Human Health and Disease. Givens, D.I. (Ed.). Academic Press, United States pp: 173-203.
32. Korat, A.V.A., Y. Li, F. Sacks, B. Rosner, W.C. Willett, F.B. Hu and Q. Sun, 2019. Dairy fat intake and risk of type 2 diabetes in 3 cohorts of US men and women. Am. J. Clin. Nutr., 110: 1192-1200.
33. Bataineh, M.F., 2002. Glycemic and Insulinemic Indices of Certain Popular Arabic Sweets with Modified Fat Content. Master Thesis, University of Jordan
34. Wallace, A.J., Eady, S.L. J.A. Willis, Scott, R.S., J.A. Monro and C.M. Frampton, 2009. Variability in measurements of blood glucose response to foods in human subjects is not reduced after a standard breakfast. Nutr. Res., 29: 238-243.
35. Wallace, A.J., J.A. Monro, R.C. Brown, C.M. Framptond, 2008. A glucose reference curve is the optimum method to determine the glycemic glucose equivalent values of foods in humans. Nutr. Res., 28: 753-759.
36. Powers, M.A., R.M. Cuddihy, D. Wesley and B. Morgan, 2010. continuous glucose monitoring reveals different glycemic responses of moderate- vs high-carbohydrate lunch meals in people with type 2 diabetes. J. Am. Diabet. Assoc., 10: 1912-1915.
37. Cai, X., L. Xia, Y. Pan, D. He, H. Zhu, T. Wei and Y. He, 2019. Differential role of insulin resistance and β-cell function in the development of prediabetes and diabetes in middle-aged and elderly Chinese population. Diabetology Metab. Syndrome, Vol. 11, No. 24 10.1186/s13098-019-0418-x
38. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2003. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care, 26: S5-S20.
39. Chia, C.W., J.M. Egan and L. Ferrucci, 2018. Age-related changes in glucose metabolism, hyperglycemia and cardiovascular risk. Circ. Res., 123: 886-904.
40. Jia, T., X. Huang, A.R. Qureshi, H. Xu and J. Ärnlöv *et al*., 2014. Validation of insulin sensitivity surrogate indices and prediction of clinical outcomes in individuals with and without impaired renal function. Kidney Int., 86: 383-391.
41. Antuna-Puente, B., E. Disse, R. Rabasa-Lhoret, M. Laville, J. Capeau and J.P. Bastard, 2011. How can we measure insulin sensitivity/resistance? [Comment mesurer la sensibilité/résistance à l'insuline?]. Diabetes Metab., 37: 179-188.
42. Kim, S.H., A. Silvers, J. Viren and G.M. Reaven, 2016. Relationship between insulin sensitivity and insulin secretion rate: not necessarily hyperbolic. Pathophysiology, 33: 961-967.
43. Tohidi, M., A. Ghasemi, F. Hadaegh, A. Derakhshan, A. Chary and F. Azizi, 2014. Age- and sex-specific reference values for fasting serum insulin levels and insulin resistance/sensitivity indices in healthy Iranian adults: Tehran lipid and glucose study. Clin. Biochem., 47: 432-438.
44. Song, S., H.Y. Paik and Y. Song, 2012. High intake of whole grains and beans pattern is inversely associated with insulin resistance in healthy Korean adult population. Diabetes Res. Clin. Pract., 98: E28-E31.

45. Dam, R.M.v., J.M. Dekker, G. Nijpels, C.D.A. Stehouwer, L.M. Bouter and R.J. Heine, 2004. Coffee consumption and incidence of impaired fasting glucose, impaired glucose tolerance and type 2 diabetes: the Hoorn study. Diabetologia, 47: 2152-2159.
46. Whitehead, N. and H. White, 2013. Systematic review of randomised controlled trials of the effects of caffeine or caffeinated drinks on blood glucose concentrations and insulin sensitivity in people with diabetes mellitus. J. Hum. Nutr. Diet., 26: 111-125.
47. Louie, J.C.Y., F. Atkinson, P. Petocz and J.C.B rand-Miller, 2008. Delayed effects of coffee, tea and sucrose on postprandial glycemia in lean, young, healthy adults. Asia Pac. J. Clin. Nutr., 17: 657-662.
48. Moisey, L.L., S. Kacker, A.C. Bickerton, L.E. Robinson and T.E. Graham, 2008. Caffeinated coffee consumption impairs blood glucose homeostasis in response to high and low glycemic index meals in healthy men. Am. J. Clin. Nutr., 87: 1254-1261.
49. Sacramento, J.F., F.O. Martins, T. Rodrigues, P. Matafome, M.J. Ribeiro, E. Olea and S.V. Conde, 2020. A_2 adenosine receptors mediate whole-body insulin sensitivity in a prediabetes animal model: primary effects on skeletal muscle. Front. Endocrinol., 10.3389/fendo.2020.00262
50. Shi, X., W. Xue, S. Liang, J. Zhao and X. Zhang, 2016. Acute caffeine ingestion reduces insulin sensitivity in healthy subjects: a systematic review and meta-analysis. Nutr. J., Vol. 15 10.1186/s12937-016-0220-7
51. Guarino, M.P., M.J. Ribeiro, J.F. Sacramento and S.V. Conde, 2013. Chronic caffeine intake reverses age-induced insulin resistance in the rat: effect on skeletal muscle $Glut_4$ transporters and AMPK activity. AGE, 35: 1755-1765.
52. Silva, L.A.d., J. Wouk, V.M.R. Weber, C.d.L. Eltchechem and P.d. Almeida *et al*., 2017. Mechanisms and biological effects of Caffeine on substrate metabolism homeostasis: A systematic review. J. Applied Pharm. Sci., 7: 215-221.
53. Robinson, L.E., S. Savani, D.S. Battram, D.H. McLaren, P. Sathasivam and T.E. Graham, 2004. Caffeine ingestion before an oral glucose tolerance test impairs blood glucose management in men with type 2 diabetes. J. Nutr., 134: 2528-2533.
54. Lee, S.J., R. Hudson, K. Kilpatrick, T.E. Graham and R. Ross, 2005. Caffeine ingestion is associated with reductions in glucose uptake independent of obesity and type 2 diabetes before and after exercise training. J. Diabetes Care, 28: 566-572.
55. Akash, M.S.H., K. Rehman and S. Chen, 2014. Effects of coffee on type 2 diabetes mellitus. Nutrition, 30: 755-763.
56. Campbell, B., C. Wilborn, P.L. Bounty, L. Taylor and M.T. Nelson *et al*, 2013. International society of sports nutrition position stand: energy drinks. J. Int. Soc. Sports Nutr., 10.1186/1550-2783-10-1
57. Reis, C.E.G., J.G. Dórea and T.H.M.da Costa, 2019. Effects of coffee consumption on glucose metabolism: A systematic review of clinical trials. J. Traditional Complementary Med., 9: 184-191.
58. Akash, M.S.H., K. Rehman, H. Sun and S. Chen, 2013. Interleukin-1 receptor antagonist improves normoglycemia and insulin sensitivity in diabetic Goto-Kakizaki-rats. Eur. J. Pharmacol., 701: 87-95.
59. Alagbonsi, A.I., T.M. Salman, H.M. Salahdeen and A.A. Alada, 2016. Effects of adenosine and caffeine on blood glucose levels in rats. Niger. J. Exp. Clin. Biosci., 4: 35-41.
60. Ohnaka, K., M. Ikeda, T. Maki, T. Okada and T. Shimazoe *et al*., 2012. Effects of 16-week consumption of caffeinated and decaffeinated instant coffee on glucose metabolism in a randomized controlled trial. J. Nutr. Metab., Vol. 2012 10.1155/2012/207426
61. Ärnlöv, J., B. Vessby and U. Risérus, 2004. Coffee consumption and insulin sensitivity. JAMA, 291: 1199-1201.
62. Wu, T., W.C. Willett, S.E. Hankinson and E. Giovannucci, 2005. Caffeinated coffee, decaffeinated coffee and caffeine in relation to plasma C-peptide levels, a marker of insulin secretion, in U.S. women. Diabetes Care, 28: 1390-1396.
63. Smith, B., D.L. Wingard, T.C. Smith, D. Kritz-Silverstein and E. Barrett-Connor, 2006. Does coffee consumption reduce the risk of type 2 diabetes in individuals with impaired glucose? Diabetes Care, 29: 2385-2390.
64. Kempf, K., C. Herder, I. Erlund, H. Kolb and S. Martin *et al*., 2010. Effects of coffee consumption on subclinical inflammation and other risk factors for type 2 diabetes: A clinical trial. Am. J. Clin. Nut., 91: 950-957.
65. Cherniack, E.P., N. Buslach and H.F. Lee, 2018. The potential effects of caffeinated beverages on insulin sensitivity. J. Am. Coll. Nutr., 37: 161-167.
66. Williamson, G., 2020. Protection against developing type 2 diabetes by coffee consumption: assessment of the role of chlorogenic acid and metabolites on glycaemic responses. Food and Funct., 11: 4826-4833.
67. Pham, N.M., A. Nanri, K. Yasuda, K. Kurotani and Keisuke Kuwahara *et al*., 2015. Habitual consumption of coffee and green tea in relation to serum adipokines: a cross-sectional study. Eur. J. Nutr., 54: 205-214.
68. Hiltunen, L.A., 2006. Are there associations between coffee consumption and glucose tolerance in elderly subjects? Eur. J. Clin. Nutr., 60: 1222-1225.
69. Beaudoin, M.S., L.E. Robinson and T.E. Graham, 2011. An oral lipid challenge and acute intake of caffeinated coffee additively decrease glucose tolerance in healthy men. J. Nutr., 141: 574-581.
70. Feinberg, L.J., H. Sandberg, O.D. Castro and S. Bellet, 1968. Effects of coffee ingestion on oral glucose tolerance curves in normal human subjects. Metabolism, 17: 916-922.

71. Meng, S., J. Cao, Q. Feng, J. Peng and Y. Hu, 2013. Biological values of acupuncture and chinese herbal medicine: impact on the life science Evidence-Based Complementary Altern. Med., Vol. 2013 10.1155/2013/801457
72. Thom, E., 2007. The effect of chlorogenic acid enriched coffee on glucose absorption in healthy volunteers and its effect on body mass when used long-term in overweight and obese people. J. Int. Med. Res., 35: 900-908.
73. Gavrieli, A., E. Fragopoulou, C.S. Mantzoros and M. Yannakoulia, 2013. Gender and body mass index modify the effect of increasing amounts of caffeinated coffee on postprandial glucose and insulin concentrations; a randomized, controlled, clinical trial. Metabolism, 62: 1099-1106.
74. Pham, N.M., A. Nanri, T. Kochi, K. Kuwahara and H. Tsuruoka *et al.*, 2014. Coffee and green tea consumption is associated with insulin resistance in Japanese adults. Metabolism, 63: 400-408.
75. Battram, D.S., T.E. Graham, E.A. Richter and F. Dela, 2005. The effect of caffeine on glucose kinetics in humans – influence of adrenaline. J. Physiol., 569: 347-355.
76. Krebs, J.D., A. Parry-Strong, M. Weatherall, R.W. Carroll and M. Downie, 2012. A cross-over study of the acute effects of espresso coffee on glucose tolerance and insulin sensitivity in people with type 2 diabetes mellitus. Metabolism, 61: 1231-1237.
77. Kahale, K.H., C. Tranchant, S. Pakzad and A.G. Farhat, 2015. Effect of sumac spice, Turkish coffee and yerba mate tea on the postprandial glycemic response to Lebanese mankoucheh. Nutr. Food Sci., 45 : 433 -447.
78. Beam, J.R., A.L. Gibson, C.M. Kerksick, C.A. Conn, A.C. White and C.M. Mermier, 2015. Effect of post-exercise caffeine and green coffee bean extract consumption on blood glucose and insulin concentrations. Nutrition, 31: 292-297.
79. Wylie-Rosett, J. and L.M. Delahanty, 2017. The Role of Diet in the Prevention and Treatment of Diabetes. In: Nutrition in the Prevention and Treatment of Disease. Coulston, A.M., C.J. Boushey and L.M. Delahanty (Eds.). Academic Press, United States pp: 691-707.
80. American Diabetes Association, 2012. Diagnosis and classification of diabetes mellitus. Diabetes Care, 35: S64-S71.
81. American Diabetes Association, 2012. Standards of Medical Care in Diabetes. Diabetes Care, 35: S11-S63.
82. Ley, S.H., O. Hamdy, V. Mohan and F.B. Hu, 2014. Prevention and management of type 2 diabetes: Dietary components and nutritional strategies. Lancet, 383: 1999-2007.
83. Kwok, M.K., G.M. Leung and C.M. Schooling, 2016. Habitual coffee consumption and risk of type 2 diabetes, ischemic heart disease, depression and Alzheimer's disease: a Mendelian randomization study. Sci. Rep., Vol. 6, 10.1038/srep36500
84. Sarriá, B., S. Martínez-López, R. Mateos and L. Bravo-Clemente, 2016. Long-term consumption of a green/roasted coffee blend positively affects glucose metabolism and insulin resistance in humans. Food Res. Int., 89: 1023-1028.
85. Lane, J.D., 2011. Caffeine, glucose metabolism and type 2 diabetes. J. Caffeine Res., 1: 23-28.
86. Ding, M., S.N. Bhupathiraju, M. Chen, R.M. van Dam and F.B. Hu, 2014. Caffeinated and decaffeinated coffee consumption and risk of type 2 diabetes: A systematic review and a dose-response meta-analysis. Diabetes Care, 37: 569-586.
87. Dam, R.M.V., W.C. Willett, J.E. Manson and F.B. Hu, 2006. Coffee, caffeine and risk of type 2 diabetes: A prospective cohort study in younger and middle-aged U.S. women. Diabetes Care, 29: 398-403.
88. Alkaabi, J., B. Al-Dabbagh, H. Saadi, S. Gariballa and J. Yasin, 2013. Effect of traditional arabic coffee consumption on the glycemic index of khalas dates tested in healthy and diabetic subjects. Asia Pac. J. Clin. Nutr., 22: 565-573.
89. Zhang, Y., E.T. Lee, L.D. Cowan, R.R. Fabsitz and B.V. Howard, 2011. Coffee consumption and the incidence of type 2 diabetes in men and women with normal glucose tolerance: The strong heart study. Nutr. Metab. Cardiovasc. Dis., 21: 418-423.
90. Hamer, M., D.R. Witte, A. Mosdøl, M.G. Marmot and E.J. Brunner, 2008. Prospective study of coffee and tea consumption in relation to risk of type 2 diabetes mellitus among men and women: The Whitehall II study. Br. J. Nutr., 100: 1046-1053.
91. Loopstra-Masters, R.C., A.D. Liese, S.M. Haffner, L.E. Wagenknecht and A.J. Hanley, 2011. Associations between the intake of caffeinated and decaffeinated coffee and measures of insulin sensitivity and beta cell function. Diabetologia, 54: 320-328.
92. Alperet, D.J., S.A. Rebello, E.Y.H. Khoo, Z. Tay and S.S.Y. Seah, 2020. The effect of coffee consumption on insulin sensitivity and other biological risk factors for type 2 diabetes: a randomized placebo-controlled trial. Am. J. Clin. Nutr., 111: 448-458.
93. Natella, F. and C. Scaccini, 2012. Role of coffee in modulation of diabetes risk. Nutr. Rev., 70: 207-217.
94. Zaharieva, D.P. and M.C. Riddell, 2013. Caffeine and glucose homeostasis during rest and exercise in diabetes mellitus. Applied Physiol. Nutr. and Metab., 38: 813-822.
95. Bidel, S., G.Hu, J. Sundvall, J. Kaprio, J. Tuomilehto, 2006. Effects of coffee consumption on glucose tolerance, serum glucose and insulin levels - A cross-sectional analysis. Horm. Metab. Res., 38: 38-43.
96. Bhatti, K. Salman, O'Keefe, H. James, Lavie and J. Carl, 2013. Coffee and tea: perks for health and longevity? Curr. Opin. Clin. Nutr. Metab. Care, 16: 688-697.

97. Chu, Y.F., Y. Chen, R.M. Black, P.H. Brown, B.J. Lyle, R.H. Liu and B. Ou, 2011. Type 2 diabetes-related bioactivities of coffee: Assessment of antioxidant activity, NF-κB inhibition and stimulation of glucose uptake. Food Chem., 124: 914-920.
98. Asfaw, G. and M. Tefera, 2020. Total polyphenol content of green, roasted and cooked Harar and Yirgacheffee Coffee, Ethiopia. J. Applied Sci. Environ. Manage., 24: 187-192.
99. Higdon, J.V. and B. Frei, 2006. Coffee and health: A review of recent human research. Crit. Rev. Food Sci. Nutr., 46: 101-123.
100. Gao, F., Y. Zhang, S. Ge, H. Lu and R. Chen *et al*., 2018. Coffee consumption is positively related to insulin secretion in the shanghai high-risk diabetic screen (SHiDS) study. Nutr. Metab., Vol. 84 10.1186/s12986-018-0321-8