



The regular consumption of coffee and development of type 2 diabetes mellitus

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Abstract

Background Drinking coffee is habitually widespread around the world. Moderate coffee consumption has a beneficial health effect on the human body. However, there are controversial effects of consuming coffee on blood glucose and insulin levels in diabetic patients, which remain to be fully elucidated.

Purpose The aim of this review was to clarify the relationship between coffee consumption and developing type 2 diabetes mellitus (T2DM).

Methods The common internet search engines were utilized to access the abstracts and full text of articles published in English in the last 20 years.

Results The association between the frequent consumption of coffee and the risk of developing type 2 diabetes has been studied intensively through epidemiologic and intervention studies. In fact, coffee is a major source of chlorogenic acids (CGAs) in the human diet. The presence of CGAs and other phenolic compounds in coffee plays a role in inhibiting glucose absorption via interference with glucose transporters and increasing insulin sensitivity. However, short-term trials confirmed the detrimental effects of heavily drinking coffee due to the presence of caffeine. Caffeine has been found to impair glucose tolerance and decrease insulin sensitivity.

Conclusion Several clinical trials have confirmed the relationship between the consumption of coffee and the development of T2DM. Moderate consumption of coffee has been suggested for its long-term benefits and positive health effects.

Keywords Blood glucose · Blood insulin · Caffeine · Chlorogenic acids · Coffee · Consumption · Diabetes

Introduction

Coffee beverage is the second most popular consumed drink in the world after water (Butt and Sultan 2011). Most people habitually enjoy consuming coffee on a daily basis for its unique aroma, flavor, mental alertness, and social engagement (Dórea and da Costa 2005). In fact, there are both beneficial and detrimental outcomes of consuming coffee that affect human health (Butt and Sultan 2011; Akash et al. 2014; Higdon and Frei 2006). The health-promoting features of coffee are often attributed to its rich phytochemicals, including caffeine and chlorogenic acids (CGAs) (Ludwig et al. 2014; Cano-Marquina et al. 2013). The biological effect

of coffee constituents must reach an organ or tissue in a sufficient concentration and be maintained for an adequate period of time in order to exhibit the beneficial or detrimental effects on the human body (Butt and Sultan 2011; Ludwig et al. 2014; Nawrot et al. 2003). Caffeine is a major pharmacologically active compound that has been shown to possess positive and negative impacts on human health, and the balance between the beneficial and detrimental effects of caffeine depend significantly on the individual's susceptibility to it (Ludwig et al. 2014). Caffeine may enhance mental alertness and memory consolidation, improve attention, help to stop a decline in brain activity with old age, and reduce risk factors involved in metabolic syndromes (Ludwig et al. 2014; Borota et al. 2014; Heckman et al. 2010; Imagawa et al. 2009; Doherty and Smith 2004). However, the potential adverse effects of excessive caffeine intake should be considered, particularly in children, pregnant women, individuals with hypertension, and diabetic patients (Nawrot et al. 2003; Temple 2009; Heckman et al. 2010).

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On the other hand, coffee contains a considerable number of phenolic constituents, such as CGAs, that display antioxidative properties (Ludwig et al. 2014). These protective compounds are linked to potential health benefits on the human body, including the prevention of several chronic diseases, such as its contribution to reducing the risk of developing type 2 diabetes mellitus (T2DM) (van Dam and Hu 2005; van Dam et al. 2006; Bidel et al. 2008; Ludwig et al. 2014).

The prevalence of diabetes in all age groups has continued to rise over the past several decades (Wild et al. 2004; Shaw et al. 2010; Guariguata et al. 2014). Diabetes and, in particular, T2DM is thought to develop due to a combination of environmental and genetic factors. There is evidence that dietary habits and lifestyle play important roles in developing or preventing diabetes. The relationship between coffee consumption and the risk of developing T2DM has been studied in epidemiologic and observational investigations (van Dam and Hu 2005; van Dam et al. 2006; Bidel et al. 2008). These studies have shown that a high consumption of coffee beverage is associated with a better glucose tolerance and a substantially lower risk of T2DM (van Dam and Hu 2005; Bidel et al. 2008). Moreover, the role of coffee constituents in reducing the risk of T2DM through decreasing body fat has also been investigated (Astrup et al. 1990; Bracco et al. 1995; Horton and Geissler 1996; Acheson et al. 2004; Higdon and Frei 2006; Lopez-Garcia et al. 2006; Thom 2007). This can be explained by the important role of some coffee constituents through direct or indirect interference with glucose absorption. Some coffee components might directly blockade the glucose transporters, increasing its rate of removal from plasma, or indirectly impair the release of glucose from disaccharides and polysaccharides (Ludwig et al. 2014). However, other clinical trials have shown that coffee consumption has been found to impair glucose tolerance and decrease insulin sensitivity (Graham et al. 2001; Johnston et al. 2003; Robinson et al. 2004; Moisey et al. 2008; Loopstra-Masters et al. 2011). These conflicting effects of coffee beverage on blood glucose and insulin levels have to be elucidated. Therefore, the focus of this article is to clarify the relationship between coffee consumption and developing T2DM.

Consumption of coffee

Coffee is a favorite beverage for many people around the world and it is frequently and widely consumed (Mitchell et al. 2014). Coffee beverage, whether it is actually prepared from *Coffea arabica* or *Coffea canephora* var. Robusta, contains a variety of chemicals that provide significant amounts of caffeine and CGAs (Briand et al. 1996; Casal et al. 2000; El-Abassy et al. 2011; Farah 2012; Garrett et al. 2012). In

addition, boiled coffee (unfiltered) is considered a source of cafestol and kahweol (Cavin et al. 2002; Farah 2012).

Caffeine occurs naturally in coffee beans and is rapidly absorbed in the stomach and small intestine and distributed to all tissues of the human body (Higdon and Frei 2006). The world's primary source of caffeine varies globally; however, coffee is considered the major prominent source of caffeine (Frary et al. 2005; Heckman et al. 2010; Mitchell et al. 2014). Caffeine is involved in some physiological effects, such as increased metabolic rate (Dulloo et al. 1989; Bracco et al. 1995; Doherty and Smith 2004), stimulation of the central nervous system (Smit and Rogers 2000; Ferré 2008), acute elevation of hypertension, diuresis, and reduction in cognitive decline in elderly men (Carrillo and Benitez 2000; Higdon and Frei 2006). Heavy and long-term consumption of caffeine may exhibit health problems associated with its pharmacological effects (Carrillo and Benitez 2000).

The other main constituent in coffee is CGAs. CGAs, as a phenolic compound, possess antioxidant activity (Clifford 2000; Clifford et al. 2003). In the human body, the CGAs are metabolized by microflora and hydrolyzed to caffeic acid and quinic acid. Both CGAs and caffeic acid have antioxidant activity (Clifford 2000).

The concentrations of CGAs and caffeine present in coffee beverages vary considerably based on several natural and artificial factors. It has been reported that a standard cup of coffee beverage is assumed to provide 70–350 mg of CGAs (Clifford 1999, 2000) and 58–259 mg of caffeine (McCusker et al. 2003; Higdon and Frei 2006). This variation in the concentration of CGAs and caffeine contents depends on the variety of coffee beans, methods of coffee preparation, brewing process (boiled coffee vs. filtered coffee), degree of roasting, and additives (del Castillo et al. 2002; McCusker et al. 2003; Higdon and Frei 2006; Vignoli et al. 2011; Ludwig et al. 2014). For example, the caffeine and CGAs contents were found to be higher in Robusta variety coffee beans compared to Arabica variety coffee beans (Casal et al. 2000; Vignoli et al. 2011). In addition, it is evident that individual variation in the metabolism of coffee components may increase or decrease the exposure of an individual to a bioactive compound in coffee (Urgert and Katan 1997; Higdon and Frei 2006).

It has been proven that the consumption of four cups of coffee per day is safe for most healthy adults (Higdon and Frei 2006; Mitchell et al. 2014). In fact, an average daily consumption of three to four cups of coffee beverage can provide an average of 203–906 mg of caffeine and 245–1225 mg of CGAs per day (Clifford 1999, 2000; McCusker et al. 2003; Nawrot et al. 2003; Higdon and Frei 2006). However, some groups, including people with hypertension, children, adolescents, and the elderly, may be more vulnerable to the adverse effects of caffeine and they should limit their consumption of coffee beverage (Nawrot et al. 2003; Higdon and Frei 2006; Temple 2009). In fact, the limitation of coffee consumption

during pregnancy should ensure that no more than 300 mg per day of caffeine is provided in order to exclude any increased probability of spontaneous abortion (Higdon and Frei 2006; Temple 2009; Chen et al. 2014, 2016). For children, the average intake should be restricted to 2.5 mg/kg of body weight per day, as a high level of caffeine intake is linked to some legitimate health concerns, such as anxiety, nervousness, or sleep disturbances (Nawrot et al. 2003; Higdon and Frei 2006; Temple 2009).

Health benefits of coffee consumption

This section will briefly cover the health benefits behind coffee intake. In fact, coffee beans contain several components that are definitely involved in health benefits. These constituents possess bioactive characteristics, especially caffeine and CGAs, as mentioned above. In addition, some of these constituents have potential therapeutic antioxidant and anticancer effects (Dórea and da Costa 2005; Esquivel and Jiménez 2012; Ludwig et al. 2014; Jeszka-Skowron et al. 2015). It has been concluded that CGAs might exert anticarcinogenic activity through inhibiting DNA methyltransferase (del Castillo et al. 2002; Vignoli et al. 2011). Epidemiologic researches have suggested that coffee consumption may help prevent several chronic diseases, including T2DM, Parkinson's disease, cardiovascular disease, and liver disease (Higdon and Frei 2006; Farah 2012; Cano-Marquina et al. 2013).

Moreover, regular and moderate coffee consumption may increase memory activity and improve physical performance due to the presence of caffeine (Dulloo et al. 1989; Bracco et al. 1995; Graham 2001; Doherty and Smith 2004; Imagawa et al. 2009), which acts mainly upon the central nervous system, stimulating wakefulness (Smit and Rogers 2000; Ferré 2008), increasing concentration, decreasing the sensation of fatigue, and relieving headache symptoms (Carrillo and Benitez 2000; Higdon and Frei 2006; Heckman et al. 2010).

In terms of the effect of caffeine in losing weight, there was an association between caffeine and increasing energy expenditure and, consequently, reducing weight (Dulloo et al. 1989; Astrup et al. 1990; Bracco et al. 1995; Horton and Geissler 1996; Acheson et al. 2004; Higdon and Frei 2006; Lopez-Garcia et al. 2006), suggesting that weight loss may play a role in the beneficial effect of coffee consumption on the risk of T2DM. However, other studies have not found that caffeine alone is effective in promoting weight loss (Egger et al. 1999).

Moreover, coffee beverages have some anticarcinogenic activities due to the presence of cafestol and kahweol (Cavin et al. 2002). Also, there was an inverse association between the consumption of caffeinated coffee and reduction in a type of skin cancer risk (Song et al. 2012).

On the other hand, some studies confirmed the positive effects of caffeine, including a negative association with the incidence of T2DM, as well as assisting in weight management (Greenberg et al. 2005; Lopez-Garcia et al. 2006; Heckman et al. 2010).

In addition, it is well known that many phenolic compounds in coffee, such as CGAs, may have potential effects on glucose and insulin levels (Johnston et al. 2003; Higdon and Frei 2006; Bidel et al. 2008). It was reported that the level of CGAs that seems to have health benefits would range from 0.5 to 2.5 g per day (Clifford et al. 2003; van Dam and Hu 2005; van Dam et al. 2006; Bidel et al. 2008; Stalmach et al. 2009). This issue has been clarified in depth in section titled [The consumption of coffee and its association with T2DM](#).

Detrimental effects of coffee consumption

The positive association between the consumption of coffee and some chronic disease risks has been more frequently investigated but there were some potential harmful effects of coffee on the human body, particularly at certain times and different stages of life. It is widely believed that coffee may cause symptoms of insomnia and restlessness for some healthy people and it is responsible for many of the central nervous system stimulant and addictive properties of caffeine (Smit and Rogers 2000). In addition, coffee consumption is associated with increases in several cardiovascular disease risk factors, including blood cholesterol, blood pressure, and plasma homocysteine (Ding et al. 2014). Some clinical studies have shown that the intake of coffee, especially unfiltered coffee, contributed significantly to the increase in triglycerides, low-density lipoprotein (LDL) cholesterol, and total cholesterol (Jee et al. 2001; Cai et al. 2012). It is evident that the presence of cafestol and kahweol in unfiltered coffee was involved in increasing serum levels of total and LDL cholesterol (Urgert and Katan 1997; Jee et al. 2001; Cai et al. 2012). Nevertheless, the caffeine content of coffee can cause a marked rise in blood pressure and irregular heartbeat, especially in those already suffering from hypertension and those who do not normally consume caffeine (Nawrot et al. 2003; Higdon and Frei 2006; Ding et al. 2014).

Moreover, a high level of caffeine intake during pregnancy is associated with slightly increased risk of miscarriage, stillbirth, premature delivery, and lower birth weight (Chen et al. 2014, 2016; Li et al. 2015; Rhee et al. 2015).

However, the most significant health concern about coffee consumption is its association with increased risk of developing T2DM, which is discussed in detail later in the section titled [The consumption of coffee and its association with T2DM](#).

Coffee consumption and diabetes mellitus

Definition and prevalence of diabetes mellitus

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia and caused primarily by a defect in insulin secretion from the islet cells of the pancreas, resulting in an inability of peripheral cells to use glucose. It is thought to develop for a variety of reasons, including several pathogenic processes ranging from autoimmune destruction of the β -cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action (American Diabetes Association, ADA 2018). The majority of cases of diabetes fall into two categories: type 1 diabetes mellitus (T1DM) and T2DM. T1DM results from β -cell destruction, leading to absolute insulin deficiency. Markers of the immune destruction of the β -cell include islet cell autoantibodies, autoantibodies to insulin, autoantibodies to glutamic acid decarboxylase, and autoantibodies to tyrosine phosphatases. Some patients with this type of diabetes may present ketoacidosis as the first manifestation of the disease. This type of diabetes commonly occurs in childhood and adolescence and accounts for only 5–10% of those with diabetes. People with T1DM present acute symptoms and markedly elevated blood glucose levels, and they need insulin for survival (ADA 2009).

T2DM accounts for 90–95% of those with diabetes and results from progressive insulin secretory defect (Olefsky and Kruszynska 2001). The cause of T2DM is thought to be due to a combination of environmental and genetic factors. Most of individuals with T2DM are obese and they do not usually need insulin treatment to survive. The risk of developing this form of diabetes increases with age, lack of physical activity, and obesity. Obesity itself or having an increased percentage of body fat distributed predominantly in the abdominal region, causes some degree of insulin resistance. Insulin resistance may improve with weight reduction and/or hypoglycemic treatment (ADA 2012).

The prevalence of diabetes mellitus is increasing at an alarming rate across many countries, and the number of people with diabetes mellitus around the world is expected to rise from 2.8% in 2000 to 4.4% in 2030 (Wild et al. 2004; Shaw et al. 2010). This doubling in cases of diabetes mellitus is thought to be for a variety of reasons, such as aging, effects of modernization, increase in the prevalence of obesity, and sedentary lifestyle (Wild et al. 2004; Shaw et al. 2010). It is expected that most people who live in developing countries will experience the largest increase in cases of diabetes due to their low and middle incomes (Shaw et al. 2010; Guariguata et al. 2014).

The consumption of coffee and its association with T2DM

Dietary habits and lifestyle play a crucial role in maintaining basic life activities and developing or preventing chronic diseases such as diabetes. The association between habitual coffee consumption and risk of developing T2DM has been intensively studied in several prospective epidemiologic studies and clinical trials. Some controlled clinical trials have found that there was a reduction in glucose tolerance after the ingestion of caffeine or caffeinated coffee, suggesting that coffee consumption could increase the risk of diabetes. On the other hand, most of the prospective epidemiologic studies have shown that the ingestion of caffeinated and decaffeinated coffee can reduce the risk of diabetes, concluding that habitual coffee consumption is associated with a lower risk of T2DM. These controversial findings will be addressed in this section, focusing on the role of coffee components in developing or preventing T2DM (Table 1).

It is well known that coffee influences glucose absorption and, thus, elicits an effect on the blood glucose levels and, subsequently, the insulin levels. It has been observed that there is an increase in the area under the glucose and insulin curves and reduction in the insulin sensitivity following the ingestion of coffee with meals (Graham et al. 2001; Johnston et al. 2003; Moisey et al. 2008). Most of the physiologic effects of coffee can be attributed to the presence of caffeine in coffee, which causes an increase by about 25–50% in the area under the glucose and insulin curves (Graham et al. 2001; Moisey et al. 2008). This effect of caffeine has been confirmed through short-term trials using decaffeinated coffee, which concluded that decaffeinated coffee may be better suited for enhancing glucose tolerance and insulin sensitivity than caffeinated coffee (Greenberg et al. 2010; Loopstra-Masters et al. 2011). An explanation of this effect is linked to the fact that caffeine is a phosphodiesterase inhibitor, which can increase the concentration of cyclic adenosine monophosphate (cAMP). Increased concentrations of cAMP have been associated with an impaired glucose tolerance after the consumption of caffeinated coffee beverage (Johnston et al. 2003). Caffeine can also inhibit muscle glucose uptake, as it acts as an adenosine receptor antagonist (Salazar-Martinez et al. 2004; Higdon and Frei 2006).

In contrast to these studies, others have not observed this association (Saremi et al. 2003). Moreover, considering the effects of caffeine on blood glucose and insulin levels that have been shown in some short-term intervention studies, these influences might be modified during long periods of coffee consumption among heavy and chronic coffee consumers (Bidel et al. 2008). Although not all prospective cohort studies have observed significant inverse associations between coffee consumption and T2DM risk, some studies confirmed the positive effects of caffeine, including a negative

Table 1 The role of coffee components in developing and preventing type 2 diabetes mellitus (T2DM)

Coffee components	Function	Outcomes	Possible explanation	References
Caffeine	Increases the concentration of cyclic adenosine monophosphate	↑ T2DM risk	Through impairing glucose tolerance	Johnston et al. (2003)
	Acts as adenosine receptor antagonist	↑ T2DM risk	Through inhibiting muscle glucose uptake	Salazar-Martinez et al. (2004); Higdon and Frei (2006)
	Increases energy expenditure, metabolic rate, and lipid oxidation	↓ T2DM risk	Through losing weight	Dulloo et al. (1989); Astrup et al. (1990); Bracco et al. (1995); Horton and Geissler (1996); Higdon and Frei (2006); Heckman et al. (2010); Acheson et al. (2004)
CGAs	Inhibits glucose transporters	↓ T2DM risk	Through reducing the amount of glucose absorbed	Bidel et al. (2008)
	Impairs releasing glucose from disaccharides and polysaccharides	↓ T2DM risk	Through lowering plasma glucose concentration	Bidel et al. (2008)
	Affects the secretion of gastrointestinal peptides	↓ T2DM risk	Through slowing intestinal glucose absorption	Johnston et al. (2003); McCarty (2005)
	Inhibits glucose-6-phosphatase	↓ T2DM risk	Through reducing plasma glucose output from the liver	McCarty (2005); Higdon and Frei (2006); Bidel et al. (2008)

↑ = increases; ↓ = decreases

association with the incidence of T2DM, as well as assisting in weight management (Lopez-Garcia et al. 2006; Heckman et al. 2010). Although caffeine ingestion may contribute to insulin resistance (Graham et al. 2001), insulin resistance may improve with weight reduction (ADA 2009). In fact, the ingestion of caffeine or caffeinated coffee was associated with weight loss through increasing energy expenditure, metabolic rate, and lipid oxidation (Dulloo et al. 1989; Astrup et al. 1990; Bracco et al. 1995; Horton and Geissler 1996; Higdon and Frei 2006; Heckman et al. 2010; Acheson et al. 2004). Consequently, moderate and regular coffee consumption could be involved in reducing the risk of developing T2DM (van Dam and Feskens 2002; Higdon and Frei 2006; Muley et al. 2012).

The relationship between coffee consumption and the risk of developing T2DM has been studied in short-term randomized controlled trials and in epidemiologic studies (van Dam and Hu 2005; van Dam et al. 2006; Bidel et al. 2008). These studies have shown that a high consumption of coffee beverage was associated with a better glucose tolerance and a substantially lower risk of T2DM (van Dam and Hu 2005; Bidel et al. 2008). In fact, participants who consumed four to six cups or more of coffee per day had a lower risk of T2DM compared with those who consumed less than two cups per day, suggesting that components in coffee other than caffeine may have protective effects (van Dam and Feskens 2002; Higdon and Frei 2006; Muley et al. 2012). In addition, habitual coffee consumption was significantly associated with lower fasting insulin concentrations and 2-h glucose concentrations. Thus, it can reduce the risk of impaired glucose test (van Dam et al. 2004). Moreover, it has been mentioned that the

prophylactic effects of coffee may also play a role on lowering the risk factors of diabetes and delaying the progress of diabetes complications as well (Oka 2007). Various mechanisms for this protective effect have been proposed, including effects on liver glucose metabolism and insulin sensitivity (Bidel and Tuomilehto 2012).

All of these beneficial health effects of coffee on lowering the risk of developing T2DM were attributed to its content of CGAs. In fact, coffee is considered as a major source of CGAs in the human diet (Clifford 1999, 2000; Thom 2007). The CGAs may have a potential effect on the glucose and insulin levels (Johnston et al. 2003; Higdon and Frei 2006; Bidel et al. 2008). The possible role of CGAs in glucose metabolism could be via its effect on inhibiting glucose transporters (Na⁺-dependent glucose transporter), which would influence the amount of glucose absorbed. Moreover, these compounds may influence α -glucosidase activity, which would lower the amount of glucose made available within the intestine and, thus, lower the plasma glucose concentration (Bidel et al. 2008). Furthermore, regular consumption of coffee may affect the secretion of gastrointestinal peptides (glucose-dependent insulinotropic polypeptide [GIP] and glucagon-like peptide-1 [GLP-1]) by decreasing GIP and increasing GLP-1, leading to slow intestinal glucose absorption (Johnston et al. 2003; McCarty 2005). In addition, the CGAs may reduce plasma glucose output from the liver by inhibiting glucose-6-phosphatase (McCarty 2005; Higdon and Frei 2006; Bidel et al. 2008).

In fact, the phenols are poorly absorbed from the human small intestine and are likely metabolized to their metabolites, where approximately a third of CGAs is only absorbed and about 67% reach the colon and may be metabolized to caffeic

acid and quinic acid (Higdon and Frei 2006). It has been reported that the beneficial health effect of lowering the risk of developing T2DM was found in people who consumed seven or more cups of coffee a day (van Dam and Hu 2005; Bidel et al. 2008). It is well known that the effects of phenols will depend on the amount consumed and also on their bio-availability (Manach et al. 2004). Indeed, randomized clinical trials to investigate the effect of Arabica coffee on blood glucose and insulin levels has not observed this effect (Al-Mssallem and Brown 2013), owing to a lower CGAs content (Louie et al. 2008; Al-Mssallem and Brown 2013). Thus, the beneficial effects of CGAs would appear to be at a higher volume of coffee beverages consumed or for long periods of frequent coffee consumption. Moreover, the differences in coffee-drinking habits, such as adding full-fat cream and sugars, should be considered, as sweetened coffee could negate any beneficial effects on glucose metabolism (Tan 2003).

Conclusion

There is an association between coffee consumption and developing type 2 diabetes mellitus (T2DM). This relationship has been confirmed through a number of controlled clinical trials. The impact of the ingestion of coffee on impairing glucose tolerance and decreasing insulin sensitivity was attributed to the effect of caffeine, which can inhibit muscle glucose uptake. However, caffeine has an effect on increasing the metabolic rate and energy expenditure. Thus, it is associated with lowering the risk of developing T2DM. On the other hand, regular and long-term consumption of coffee may have a potential effect on reducing the risk of developing T2DM due to the presence of chlorogenic acids (CGAs). CGAs play an essential role in influencing the amount of glucose absorbed, lowering plasma glucose concentration, and delaying intestinal glucose absorption. It is suggested that moderate consumption of coffee, from three to four cups per day, seems generally safe and more likely to benefit human health than harm. The influence of heavy coffee intake indicates that some detrimental effects can occur and the consumption of decaffeinated coffee may be wise.

Compliance with ethical standards

Conflict of interest The author declares no conflict of interest.

References

Acheson KJ, Gremaud G, Meirim I, Montigon F, Krebs Y, Fay LB, Gay LJ, Schneiter P, Schindler C, Tappy L (2004) Metabolic effects of caffeine in humans: lipid oxidation or futile cycling? *Am J Clin Nutr* 79:40–46. <https://doi.org/10.1093/ajcn/79.1.40>

- Akash MSH, Rehman K, Chen S (2014) Effects of coffee on type 2 diabetes mellitus. *Nutrition* 30:755–763. <https://doi.org/10.1016/j.nut.2013.11.020>
- Al-Mssallem MQ, Brown JE (2013) Arabic coffee increases the glycemic index but not insulinemic index of dates. *Saudi Med J* 34:923–928
- American Diabetes Association (ADA) (2009) Standards of medical care in diabetes—2009. *Diabetes Care* 32(Suppl 1):S13–S61
- American Diabetes Association (ADA) (2012) In: Burant CF, Young LA (eds) Medical management of type 2 diabetes, 7th edn. ADA, Alexandria, VA
- American Diabetes Association (ADA) (2018) Standards of medical care in diabetes—2018. *Diabetes Care* 41(Suppl 1):S1–S2
- Astrup A, Toubro S, Cannon S, Hein P, Breum L, Madsen J (1990) Caffeine: a double-blind, placebo-controlled study of its thermogenic, metabolic, and cardiovascular effects in healthy volunteers. *Am J Clin Nutr* 51:759–767. <https://doi.org/10.1093/ajcn/51.5.759>
- Bidel S, Tuomilehto J (2012) The emerging health benefits of coffee with an emphasis on type 2 diabetes and cardiovascular disease. *Eur Endocrinol* 9:99–106. <https://doi.org/10.17925/EE.2013.09.02.99>
- Bidel S, Silventoinen K, Hu G, Lee D-H, Kaprio J, Tuomilehto J (2008) Coffee consumption, serum γ -glutamyltransferase and risk of type II diabetes. *Eur J Clin Nutr* 62:178–185. <https://doi.org/10.1038/sj.ejcn.1602712>
- Borota D, Murray E, Keceli G, Chang A, Watabe JM, Ly M, Toscano JP, Yassa MA (2014) Post-study caffeine administration enhances memory consolidation in humans. *Nat Neurosci* 17:201–203. <https://doi.org/10.1038/nm.3623>
- Bracco D, Ferrara JM, Arnaud MJ, Jequier ER, Schutz Y (1995) Effects of caffeine on energy metabolism, heart rate, and methylxanthine metabolism in lean and obese women. *Am J Phys* 269:E671–E678. <https://doi.org/10.1152/ajpendo.1995.269.4.E671>
- Briandet R, Kemsley EK, Wilson RH (1996) Discrimination of Arabica and Robusta in instant coffee by Fourier transform infrared spectroscopy and chemometrics. *J Agric Food Chem* 44:170–174. <https://doi.org/10.1021/jf950305a>
- Butt MS, Sultan MT (2011) Coffee and its consumption: benefits and risks. *Crit Rev Food Sci Nutr* 51:363–373. <https://doi.org/10.1080/10408390903586412>
- Cai L, Ma D, Zhang Y, Liu Z, Wang P (2012) The effect of coffee consumption on serum lipids: a meta-analysis of randomized controlled trials. *Eur J Clin Nutr* 66:872–877
- Cano-Marquina A, Tarín JJ, Cano A (2013) The impact of coffee on health. *Maturitas* 75:7–21. <https://doi.org/10.1016/j.maturitas.2013.02.002>
- Carrillo JA, Benitez J (2000) Clinically significant pharmacokinetic interactions between dietary caffeine and medications. *Clin Pharmacokinet* 39:127–153. <https://doi.org/10.2165/00003088-200039020-00004>
- Casal S, Oliveira MBPP, Alves MR, Ferreira MA (2000) Discriminate analysis of roasted coffee varieties for trigonelline, nicotinic acid, and caffeine content. *J Agric Food Chem* 48:3420–3424. <https://doi.org/10.1021/jf990702b>
- Cavin C, Holzhaeuser D, Scharf G, Constable A, Huber WW, Schilter B (2002) Cafestol and kahweol, two coffee specific diterpenes with anticarcinogenic activity. *Food Chem Toxicol* 40:1155–1163. [https://doi.org/10.1016/S0278-6915\(02\)00029-7](https://doi.org/10.1016/S0278-6915(02)00029-7)
- Chen LW, Wu Y, Neelakantan N, Chong MF, Pan A, van Dam RM (2014) Maternal caffeine intake during pregnancy is associated with risk of low birth weight: a systematic review and dose-response meta-analysis. *BMC Med* 12:174–176. <https://doi.org/10.1186/s12916-014-0174-6>
- Chen LW, Wu Y, Neelakantan N, Chong MF, Pan A, van Dam RM (2016) Maternal caffeine intake during pregnancy and risk of pregnancy loss: a categorical and dose-response meta-analysis of prospective studies. *Public Health Nutr* 19(7):1233–1244. <https://doi.org/10.1017/S1368980015002463>

- Clifford MN (1999) Chlorogenic acids and other cinnamates—nature, occurrence and dietary burden. *J Sci Food Agric* 79:362–372. [https://doi.org/10.1002/\(SICI\)1097-0010\(19990301\)79:3<362::AID-JSFA256>3.0.CO;2-D](https://doi.org/10.1002/(SICI)1097-0010(19990301)79:3<362::AID-JSFA256>3.0.CO;2-D)
- Clifford MN (2000) Chlorogenic acids and other cinnamates—nature, occurrence, dietary burden, absorption and metabolism. *J Sci Food Agric* 80:1033–1043. [https://doi.org/10.1002/\(SICI\)1097-0010\(20000515\)80:7<1033::AID-JSFA595>3.0.CO;2-T](https://doi.org/10.1002/(SICI)1097-0010(20000515)80:7<1033::AID-JSFA595>3.0.CO;2-T)
- Clifford MN, Johnston KL, Knight S, Kuhnert N (2003) Hierarchical scheme for LC-MSn identification of chlorogenic acids. *J Agric Food Chem* 51:2900–2911. <https://doi.org/10.1021/jf026187q>
- del Castillo MD, Ames JM, Gordon MH (2002) Effect of roasting on the antioxidant activity of coffee brews. *J Agric Food Chem* 50(13):3698–3703
- Ding M, Bhupathiraju SN, Satija A, van Dam RM, Hu FB (2014) Long-term coffee consumption and risk of cardiovascular disease: a systematic review and a dose–response meta-analysis of prospective cohort studies. *Circulation* 129:643–659. <https://doi.org/10.1161/circulationaha.113.005925>
- Doherty M, Smith PM (2004) Effects of caffeine ingestion on exercise testing: a meta-analysis. *Int J Sport Nutr Exerc Metab* 14:626–646. <https://www.ncbi.nlm.nih.gov/pubmed/15657469>
- Dórea JG, da Costa THM (2005) Is coffee a functional food? *Brit J Nutr* 93:773–782. <https://doi.org/10.1079/BJN20051370>
- Dulloo AG, Geissler CA, Horton T, Collins A, Miller DS (1989) Normal caffeine consumption: influence on thermogenesis and daily energy expenditure in lean and postobese human volunteers. *Am J Clin Nutr* 49:44–50. <https://doi.org/10.1093/ajcn/49.1.44>
- Egger G, Cameron-Smith D, Stanton R (1999) The effectiveness of popular, non-prescription weight loss supplements. *Med J Aust* 171:604–608
- El-Abassy RM, Donfack P, Matemy A (2011) Discrimination between Arabica and Robusta green coffee using visible micro Raman spectroscopy and chemometric analysis. *Food Chem* 126:1443–1448. <https://doi.org/10.1016/j.foodchem.2010.11.132>
- Esquivel P, Jiménez VM (2012) Functional properties of coffee and coffee by-products. *Food Res Int* 46:488–495. <https://doi.org/10.1016/j.foodres.2011.05.028>
- Farah A (2012) Coffee constituents. In: Chu Y-F (ed) *Coffee: emerging health effects and disease prevention*, 1st edn. John Wiley & Sons, Inc. and Blackwell Publishing Ltd., New York
- Ferré S (2008) An update on the mechanisms of the psychostimulant effects of caffeine. *J Neurochem* 105:1067–1079. <https://doi.org/10.1111/j.1471-4159.2007.05196.x>
- Frary CD, Johnson RK, Wang MQ (2005) Food sources and intakes of caffeine in the diets of persons in the United States. *J Am Diet Assoc* 105:110–113. <https://doi.org/10.1016/j.jada.2004.10.027>
- Garrett R, Vaz BG, Hovell AMC, Eberlin MN, Rezende CM (2012) Arabica and Robusta coffees: identification of major polar compounds and quantification of blends by direct-infusion electrospray ionization–mass spectrometry. *J Agric Food Chem* 60:4253–4258. <https://doi.org/10.1021/jf300388m>
- Graham TE (2001) Caffeine and exercise: metabolism, endurance and performance. *Sports Med* 31:785–807. <https://www.ncbi.nlm.nih.gov/pubmed/11583104>
- Graham TE, Sathasivam P, Rowland M, Marko N, Greer F, Battram D (2001) Caffeine ingestion elevates plasma insulin response in humans during an oral glucose tolerance test. *Can J Physiol Pharmacol* 79:559–565. <https://doi.org/10.1139/cjpp-79-7-559>
- Greenberg JA, Axen KV, Schnoll R, Boozer CN (2005) Coffee, tea and diabetes: the role of weight loss and caffeine. *Int J Obes* 29:1121–1129. <https://doi.org/10.1038/sj.ijo.0802999>
- Greenberg JA, Owen DR, Geliebter A (2010) Decaffeinated coffee and glucose metabolism in young men. *Diabetes Care* 33:278–280. <https://doi.org/10.2337/dc09-1539>
- Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE (2014) Global estimates of diabetes prevalence for 2013 and projections for 2035. *Diabet Res Clin Pract* 103:137–149. <https://doi.org/10.1016/j.diabres.2013.11.002>
- Heckman MA, Weil J, De Mejia EG (2010) Caffeine (1, 3, 7-trimethylxanthine) in foods: a comprehensive review on consumption, functionality, safety, and regulatory matters. *J Food Sci* 75:R77–R87. <https://doi.org/10.1111/j.1750-3841.2010.01561.x>
- Higdon JV, Frei B (2006) Coffee and health: a review of recent human research. *Crit Rev Food Sci Nutr* 46:101–123. <https://doi.org/10.1080/10408390500400009>
- Horton TJ, Geissler CA (1996) Post-prandial thermogenesis with ephedrine, caffeine and aspirin in lean, pre-disposed obese and obese women. *Int J Obes Relat Metab Disord* 20:91–97
- Imagawa TF, Hirano I, Utsuki K, Horie M, Naka A, Matsumoto K, Imagawa S (2009) Caffeine and taurine enhance endurance performance. *Int J Sports Med* 30:485–488. <https://doi.org/10.1055/s-0028-1104574>
- Jee SH, He J, Appel LJ, Whelton PK, Suh II, Klag MJ (2001) Coffee consumption and serum lipids: a meta-analysis of randomized controlled clinical trials. *Am J Epidemiol* 153:353–362. <https://doi.org/10.1093/aje/153.4.353>
- Jeszka-Skowron M, Zgoła-Grześkowiak A, Grześkowiak T (2015) Analytical methods applied for the characterization and the determination of bioactive compounds in coffee. *Eur Food Res Technol* 240:19–31. <https://doi.org/10.1007/s00217-014-2356-z>
- Johnston KL, Clifford MN, Morgan LM (2003) Coffee acutely modifies gastrointestinal hormone secretion and glucose tolerance in humans: glycemic effects of chlorogenic acid and caffeine. *Am J Clin Nutr* 78:728–733. <https://doi.org/10.1093/ajcn/78.4.728>
- Li J, Zhao H, Song JM, Zhang J, Tang YL, Xin CM (2015) A meta-analysis of risk of pregnancy loss and caffeine and coffee consumption during pregnancy. *Int J Gynaecol Obstet* 130:116–122. <https://doi.org/10.1016/j.ijgo.2015.03.033>
- Loopstra-Masters RC, Liese AD, Haffner SM, Wagenknecht LE, Hanley AJ (2011) Associations between the intake of caffeinated and decaffeinated coffee and measures of insulin sensitivity and beta cell function. *Diabetologia* 54:320–328. <https://doi.org/10.1007/s00125-010-1957-8>
- Lopez-Garcia E, Van Dam RM, Rajpathak S, Willett WC, Manson JE, Hu FB (2006) Changes in caffeine intake and long-term weight change in men and women. *Am J Clin Nutr* 83:674–680. <https://doi.org/10.1093/ajcn.83.3.674>
- Louie JC, Atkinson F, Petocz P, Brand-Miller JC (2008) Delayed effects of coffee, tea and sucrose on postprandial glycemia in lean, young, healthy adults. *Asia Pac J Clin Nutr* 17:657–662
- Ludwig IA, Clifford MN, Lean ME, Ashihara H, Crozier A (2014) Coffee: biochemistry and potential impact on health. *Food Funct* 5:1695–1717. <https://doi.org/10.1039/c4fo00042k>
- Manach C, Scalbert A, Morand C, Rémésy C, Jiménez L (2004) Polyphenols: food sources and bioavailability. *Am J Clin Nutr* 79:727–747. <https://doi.org/10.1093/ajcn/79.5.727>
- McCarty MF (2005) A chlorogenic acid-induced increase in GLP-1 production may mediate the impact of heavy coffee consumption on diabetes risk. *Med Hypotheses* 64:848–853. <https://doi.org/10.1016/j.mehy.2004.03.037>
- McCusker RR, Goldberger BA, Cone EJ (2003) Caffeine content of specialty coffees. *J Anal Toxicol* 27:520–522
- Mitchell DC, Knight CA, Hockenberry J, Teplansky R, Hartman TJ (2014) Beverage caffeine intakes in the U.S. *Food Chem Toxicol* 63:136–142. <https://doi.org/10.1016/j.fct.2013.10.042>
- Moisey LL, Kacker S, Bickerton AC, Robinson LE, Graham TE (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. <https://doi.org/10.1093/ajcn/87.5.1254>

- Muley A, Muley P, Shah M (2012) Coffee to reduce risk of type 2 diabetes?: a systematic review. *Curr Diabetes Rev* 8:162–168. <https://doi.org/10.2174/157339912800564016>
- Nawrot P, Jordan S, Eastwood J, Rotstein J, Hugenholtz A, Feeley M (2003) Effects of caffeine on human health. *Food Addit Contam* 20: 1–30. <https://doi.org/10.1080/0265203021000007840>
- Oka K (2007) Pharmacological bases of coffee nutrients for diabetes prevention. *J Pharm Soc Japan* 127:1825–1836
- Olefsky JM, Kruszynska YT (2001) Type 2 diabetes mellitus: etiology, pathogenesis, and natural history. In: *Endocrinology*, 4th edn. WB Saunders, Philadelphia, pp 776–797
- Rhee J, Kim R, Kim Y, Tam M, Lai Y, Keum N, Oldenburg CE (2015) Maternal caffeine consumption during pregnancy and risk of low birth weight: a dose-response meta-analysis of observational studies. *PLoS One* 10:e0132334. <https://doi.org/10.1371/journal.pone.0132334>
- Robinson LE, Savani S, Battram DS, McLaren DH, Sathasivam P, Graham TE (2004) Caffeine ingestion before an oral glucose tolerance test impairs blood glucose management in men with type 2 diabetes. *J Nutr* 134:2528–2533. <https://doi.org/10.1093/jn/134.10.2528>
- Salazar-Martinez E, Willett WC, Ascherio A, Manson JE, Leitzmann MF, Stampfer MJ, Hu FB (2004) Coffee consumption and risk for type 2 diabetes mellitus. *Ann Intern Med* 140:1–8. <https://doi.org/10.7326/0003-4819-140-1-200401060-00005>
- Saremi A, Tulloch-Reid M, Knowler WC (2003) Coffee consumption and the incidence of type 2 diabetes. *Diabetes Care* 26:2211–2212. <https://doi.org/10.2337/diacare.26.7.2211>
- Shaw JE, Sicree RA, Zimmet PZ (2010) Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* 87:4–14. <https://doi.org/10.1016/j.diabres.2009.10.007>
- Smit HJ, Rogers PJ (2000) Effects of low doses of caffeine on cognitive performance, mood and thirst in low and higher caffeine consumers. *Psychopharmacology* 152:167–173. <https://www.ncbi.nlm.nih.gov/pubmed/11057520?dopt>
- Song F, Qureshi AA, Han J (2012) Increased caffeine intake is associated with reduced risk of basal cell carcinoma of the skin. *Cancer Res* 72: 3282–3289. <https://doi.org/10.1158/0008-5472.CAN-11-3511>
- Stalmach A, Mullen W, Barron D, Uchida K, Yokota T, Cavin C, Steiling H, Williamson G, Crozier A (2009) Metabolite profiling of hydroxycinnamate derivatives in plasma and urine after the ingestion of coffee by humans: identification of biomarkers of coffee consumption. *Drug Metab Dispos* 37:1749–1758. <https://doi.org/10.1124/dmd.109.028019>
- Tan DSW (2003) Coffee consumption and risk of type 2 diabetes mellitus. *Lancet* 361:702. [https://doi.org/10.1016/S0140-6736\(03\)12582-2](https://doi.org/10.1016/S0140-6736(03)12582-2)
- Temple JL (2009) Caffeine use in children: what we know, what we have left to learn, and why we should worry. *Neurosci Biobehav Rev* 33: 793–806. <https://doi.org/10.1016/j.neubiorev.2009.01.001>
- 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. <https://doi.org/10.1177/147323000703500620>
- Urgert R, Katan MB (1997) The cholesterol-raising factor from coffee beans. *Annu Rev Nutr* 17:305–324. <https://doi.org/10.1146/annurev.nutr.17.1.305>
- van Dam RM, Feskens EJ (2002) Coffee consumption and risk of type 2 diabetes mellitus. *Lancet* 360:1477–1478. [https://doi.org/10.1016/S0140-6736\(02\)11436-X](https://doi.org/10.1016/S0140-6736(02)11436-X)
- van Dam RM, Hu FB (2005) Coffee consumption and risk of type 2 diabetes: a systematic review. *JAMA* 294:97–104. <https://doi.org/10.1001/jama.294.1.97>
- van Dam RM, Dekker JM, Nijpels G, Stehouwer CD, Bouter LM, Heine RJ (2004) Coffee consumption and incidence of impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes: the Hoorn study. *Diabetologia* 47:2152–2159. <https://doi.org/10.1007/s00125-004-1573-6>
- van Dam RM, Willett WC, Manson JE, Hu FB (2006) Coffee, caffeine, and risk of type 2 diabetes. *Diabetes Care* 29:398–403. <https://doi.org/10.2337/diacare.29.02.06.dc05-1512>
- Vignoli JA, Bassoli DG, Benassi MT (2011) Antioxidant activity, polyphenols, caffeine and melanoidins in soluble coffee: the influence of processing conditions and raw material. *Food Chem* 124:863–868. <https://doi.org/10.1016/j.foodchem.2010.07.008>
- Wild S, Roglic G, Green A, Sicree R, King H (2004) Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care* 27:1047–1053. <https://doi.org/10.2337/diacare.27.10.2569>

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