

Habitual coffee consumption and blood pressure: An epidemiological perspective

Johanna M Geleijnse

Division of Human Nutrition,
Wageningen University,
The Netherlands

Abstract: This paper summarizes the current epidemiological evidence on coffee consumption in relation to blood pressure (BP) and risk of hypertension. Data from cross-sectional studies suggest an inverse linear or U-shaped association of habitual coffee use with BP in different populations. Prospective studies suggest a protective effect of high coffee intake (4 or more cups per day) against hypertension, mainly in women. Furthermore, the risk of hypertension may be lower in coffee abstainers. Randomized controlled trials, which are mostly of short duration (1–12 weeks), have shown that coffee intake around 5 cups per day causes a small elevation in BP ($\sim 2/1$ mmHg) when compared to abstinence or use of decaffeinated coffee. With regard to underlying biological mechanisms, most research has been devoted to BP-raising effects of caffeine. However, there are many other substances in coffee, such as polyphenols, soluble fibre and potassium, which could exert a beneficial effect in the cardiovascular system. Although the precise nature of the relation between coffee and BP is still unclear, most evidence suggests that regular intake of caffeinated coffee does not increase the risk of hypertension.

Keyword: epidemiology, coffee, blood pressure, hypertension

Introduction

Hypertension is an important risk factor for the development of stroke, coronary heart disease, and congestive heart failure (MacMahon et al 1990; Stamler et al 1993). The estimated total number of adults with hypertension in 2000 was 972 million, and this may increase by about 60% to a total of 1.56 billion in 2025 (Kearney et al 2005). A prolonged reduction of diastolic blood pressure (BP) of only 5 mmHg in the general population may reduce the number of strokes by 34% and the number of myocardial infarctions by 21% (MacMahon et al 1990; Stamler et al 1993). Many dietary and lifestyle factors have been implicated in the etiology of hypertension (Beilin et al 1999), of which overweight, physical inactivity, excessive salt consumption, and low potassium intake are the most important contributors in Western societies (Geleijnse et al 2004). The epidemiological evidence for an effect of coffee on BP is summarized in this review, with a focus on habitual ('chronic') intake because of its relevance to public health. Short-term, acute effects of coffee intake or caffeine administration on BP have been addressed by others (Nurminen et al 1999), and are beyond the scope of this review. Before discussing scientific evidence on the relation between coffee and BP, the state of the art on coffee intake in relation to cardiovascular disease is briefly discussed, and an overview of different epidemiological study designs is given with their strengths and limitations. Subsequently, cross-sectional and prospective cohort data on coffee and BP are summarized, followed by a discussion of experimental evidence from randomized controlled trials (RCTs). Finally, potential biological pathways for an effect of coffee on BP are briefly described, which is followed by an overall discussion of the total available evidence. Coffee in this review is considered to be filtered caffeinated coffee, except when stated otherwise.

Correspondence: Johanna M Geleijnse
Wageningen University, Division
of Human Nutrition, PO Box 8129, 6700
EV Wageningen, The Netherlands
Tel +31 317 482 574
Fax +31 317 483 342
Email marianne.geleijnse@wur.nl

Coffee and cardiovascular disease

Several large cohort studies reported no association between habitual coffee intake and risk of CVD (Grobbee et al 1990; Willett et al 1996; Woodward and Tunstall-Pedoe 1999; Andersen et al 2006; Lopez-Garcia et al 2006). Coffee neither influenced prognosis after myocardial infarction (Mukamal et al 2004). Sofi and colleagues (2006) performed a meta-analysis of 23 epidemiological studies on coffee, that included in total 403,631 participants. Despite a significant association between high daily coffee intake and coronary heart disease in case-control studies, no significant relation emerged from long-term follow-up prospective cohort studies (Sofi et al 2006). Among the studies that suggested an inverse relation of coffee with CVD are the Iowa Women's Health Study in over 41,000 women aged 55–69 (Andersen et al 2006), and the Scottish Heart Health Study in over 11,000 men and women aged 40–59 (Woodward and Tunstall-Pedoe 1999). Coffee may also protect against diabetes mellitus type 2. In a Dutch epidemiological study among over 17,000 men and women, subjects who drank 7 or more cups of coffee per day had only half the risk of developing diabetes compared to subjects who drank 2 cups or less (Van Dam and Feskens 2002). Van Dam and Hu (2005) conducted a meta-analysis of 15 epidemiological studies in which they showed that the risk of type 2 diabetes was 35% lower in subjects who drank over 6 or 7 cups of coffee per day, and 28% lower in subjects who drank 4–6 cups per day, compared to subjects with zero or low intake (~2 cups per day). The underlying mechanisms for a potentially protective effect of coffee on CVD and diabetes, including the possible role of BP, remain to be elucidated.

Methodological aspects of epidemiological studies

Observational (cohort) studies, mainly cross-sectional studies, have the major advantage that they can provide insight into the long-term effects of coffee on BP. Furthermore, the large sample size allows examination of different doses of coffee and possible effect modification by gender, age, race, cardiovascular risk profile, and other characteristics. It should be noted, however, that observational evidence can never be used to demonstrate causality. Coffee drinking is part of an individual's lifestyle (Schwarz et al 1994) and is related to many other factors, such as alcohol intake, mental stress, and dietary habits, which may also influence BP. Therefore, when interpreting observational data, one should make certain that potential confounders have been adequately adjusted for. Residual confounding in the data, eg, by lack

of adjustment for physical activity, may distort the relation between coffee consumption and BP. Cohort studies often include many subjects. However, a large sample size is by no means a guarantee for the internal validity of a study and may even hamper the accurate measurement of dietary and lifestyle factors (leading to residual confounding). Inaccurate measurement of coffee intake further leads to misclassification of subjects, which could yield a diluted (attenuated) estimate for the coffee-BP association. Cross-sectional studies or surveys, in which coffee intake and BP are measured at the same point in time, have the additional problem of 'reverse causation'. Since there is no time span between the assessment of coffee intake and BP measurement, it is possible that people who are aware of having high BP have already adapted their level of coffee consumption. As a result, a biased relation may be found between low (in fact: lowered) coffee intake and high BP levels.

A main feature of experimental studies, or RCTs, is that both the intervention (eg, use of coffee or caffeine tablets) and control treatment (eg, decaffeinated coffee or placebo) are randomly assigned to participating subjects. By this means, any confounders that could distort the relation between coffee and BP are equally distributed over both groups, and a valid estimate of the effect of coffee on BP can be obtained. RCTs are therefore considered the 'gold standard' in epidemiological research, and may even provide insight into causal effects when conducted in a double-blind manner. However, compared to observational epidemiological studies, they have the disadvantage that only fixed doses of coffee or caffeine can be studied during a relatively short period of time. Furthermore, trials may suffer from poor adherence to the assigned treatments, unsuccessful randomization (in small trials), or lack of blinding of the participant and/or investigator, which could introduce bias. Also, the number of subjects that can participate in a RCT is limited.

Coffee and BP: evidence from observational studies

Cross-sectional studies

Cross-sectional studies of coffee intake and BP date from the early 1980s. Lang and colleagues (1983a) performed a cross-sectional analysis of coffee and BP in 6,321 adults from Paris. After controlling for age, sex, BMI, alcohol consumption, smoking and socioeconomic status, systolic BP appeared to be higher in coffee drinkers (Lang et al 1983a). In a similar analysis by this group among 1,491 subjects from Algiers,

diastolic BP was elevated in coffee drinkers after adjustment for sex, age, BMI, physical activity, rural versus urban residency, smoking, and tea consumption (Lang et al 1983b). BP differences in both studies were modest (2–3 mmHg). A small study in 255 Dutch elderly showed a positive relation of coffee use with BP, but only in women (Löwik et al 1991). Burke and colleagues (1992) also showed a positive relation in 843 Australian men and women aged 60–87, but only in coffee drinkers who used antihypertensive drugs. Narkiewicz and colleagues (1995) found higher systolic BP in Italian men who took 4 or more cups of coffee per day compared to nondrinkers, but only for daytime ambulatory BP and not for office BP.

In 5,147 Australian adults, caffeine consumption (mainly through coffee) within the last 3 hours was associated with higher BP in both sexes after controlling for age, adiposity, family history of hypertension, serum cholesterol, alcohol use, and smoking (Shirlow et al 1988). Average daily caffeine intake, on the other hand, was not associated with BP in this survey. Similarly, in a Swiss study in 338 young women, coffee consumption on the testing day was related to elevated BP whereas no association was found with habitual coffee intake (Höfer and Bättig 1993). In the CARDIA study of 5,115 black and white young adults (Lewis et al 1993), caffeine intake (up to 800 mg/d) through foods and beverages was not consistently related to BP when controlling for age, sex, race, physical activity, BMI, oral contraceptive use, alcohol use, smoking, and other potential confounders. An analysis of Framingham data that comprised over 6,000 middle-aged men and women neither showed a significant relation of coffee intake with BP (Wilson et al 1989).

Two large US cross-sectional studies showed a negative (inverse) correlation of coffee with BP (Prineas et al 1980; Klatsky et al 1986). This was confirmed in a survey of 500 Italian health care workers (Periti et al 1987), where BP declined by 0.8/0.5 mmHg per cup. Another Italian study by Salvaggio and colleagues (1990) among 9,601 adults showed 2–3 mmHg lower systolic BP in subjects who drank 4–5 cups of coffee per day compared to abstainers. Similarly, in 336 male self-defense officials in Japan, daily coffee drinking was associated with lower BP (around 0.5 mmHg/cup), after adjustment for green tea intake, alcohol use, smoking, body mass index (BMI), and diabetes (Wakabayashi et al 1998). In another Japanese study of 1,902 men and women aged 40+ from Tanushimaru, one of the cohorts of the Seven Countries Study, coffee consumption was inversely related to BP whereas

intake of green tea was not (Hino et al 2007). Coffee was also weakly inversely correlated with systolic and diastolic BP in the Danish MONICA cohort after multivariable adjustment (Kirchoff et al 1994). In the Copenhagen Male Study of 2,975 older men, BP levels and prevalence of hypertension declined across coffee categories after multivariable adjustment, ie, 123/74 mmHg (14% hypertensive) for 1–4 cups, 121/72 mmHg (11%) for 5–8 cups, and 117/70 mmHg (7%) for 9+ cups per day (Gyntelberg et al 1995). BP data in abstainers were not presented in this study. A UK health check in 1989–1993 showed that habitual coffee intake (mainly instant) was inversely related to systolic BP in 478 men, with levels gradually declining from 134 mmHg in abstainers to 126 mmHg in subjects drinking more than 6 cups per day (Lancaster et al 1994). In 586 UK women an inverse U-shaped relation was seen, with highest systolic BP levels for 1–2 cups per day (128 mmHg) and lowest levels for >6 cups per day (117 mmHg). Data were adjusted for age, BMI, smoking, alcohol use, and physical activity (Lancaster et al 1994). A Norwegian study of almost 30,000 middle-aged men and women also showed an inverse U-shaped relation, with lowest BP values both in abstainers and in coffee drinkers who took 9 or more cups per day (Stensvold et al 1989). Lopez-Garcia and colleagues (2006) in a cross-sectional analysis of 730 healthy and 663 diabetic women from the Nurses' Health Study I cohort, studied caffeinated and decaffeinated coffee in relation to endothelial function and found no association. In these data, an inverse U-shaped relation between coffee intake and presence of hypertension was seen in healthy women, with a larger proportion of hypertensives for intakes around 1 cup per day (25%) compared to <1 cup per month (8%) and 2+ cups per day (17%). However, these data were not adjusted for confounders although mean age was similar across coffee categories (Lopez-Garcia et al 2006).

Summarizing, data from cross-sectional studies provide little support for a BP-raising effect of coffee, with the possible exception for BP measured shortly after coffee intake. Rather, the data suggest an inverse linear or U-shaped relation of habitual coffee intake with BP in populations from Europe, Asia and USA (Prineas and Jacobs 1980; Klatsky et al 1986; Periti et al 1987; Stensvold et al 1989; Salvaggio et al 1990; Kirchoff et al 1994; Lancaster et al 1994; Gyntelberg et al 1995; Wakabayashi et al 1998; Lopez-Garcia et al 2006; Hino et al 2007), although this inverse relation may be absent at young age as shown in the CARDIA study (Lewis et al 1993). Gender does not

seem to influence the relation of coffee with BP on basis of cross-sectional evidence.

Prospective studies

Only few prospective (ie, longitudinal or follow-up) studies of coffee and (change in) BP or incidence of hypertension have been performed. In an Australian cohort of working men, coffee consumption was associated with elevated BP during 6 years of follow-up (Jenner et al 1988). In a prospective study in 1,017 male medical alumni with 33 years of follow-up, coffee intake was positively associated with self-reported systolic BP (0.19 mmHg) and diastolic BP (0.27 mmHg) per cup per day, after adjustment for parental hypertension, BMI, smoking, alcohol use, and physical activity (Klag et al 2002). Compared with abstainers, coffee drinkers were at higher risk of developing hypertension, with the risk maximally increased by 49% for baseline intakes of 3–4 cups/day, but findings were no longer statistically significant after multi-variable adjustment (Klag et al 2002).

In a large study of habitual caffeine intake and risk of hypertension, Winkelmayr and colleagues (2005) analyzed data from a large prospective cohort study in 155,594 US nurses. Intake of caffeinated coffee was weakly inversely related to incident hypertension, with around 10% reduction in risk in women who drank 4 or more cups compared to women drinking 3 or less cups per day. Data were adjusted for age, BMI, family history of hypertension, physical activity, smoking, oral contraceptive use (in female subjects), and intake of alcohol, tea, and cola. Interestingly, contrary to coffee, an high intake of cola significantly increased the risk of hypertension in these women (Winkelmayr et al 2005).

Recently, a prospective analysis of coffee intake and incident hypertension has been performed in 2,985 men and 3,383 women in The Netherlands with over 10 years of follow-up (Uiterwaal et al 2007). This study showed an inverse U-shaped relation in women, with abstainers having a 49% reduced risk and women who drank more than 6 cups per day having a 33% reduced risk of hypertension compared to light coffee drinkers (>0–3 cups per day). Data were adjusted for age, height and weight, smoking, socioeconomic status, and intake of tea, alcohol, and total energy. In men, the risk of hypertension also tended to be reduced in abstainers but data were not statistically significant (Uiterwaal et al 2007). Overall, the inverse association between coffee and hypertension was mainly present in older subjects (>39 years). On the other hand, an Italian study in 800 men and 307 women with elevated BP (mean age: 33 years) showed that coffee drinkers developed sustained hypertension more frequently

than abstainers (53% vs 44%) during more than 6 years of follow-up (Hu et al 2007). The Italian ‘espresso’ coffee was the predominant type of coffee in this study, which is prepared without a paper filter. In 2,505 Finnish subjects aged 25–64 with 13 years of follow-up, the risk of initiating antihypertensive treatment was increased by 20%–30% in subjects who drank 2–7 cups daily, compared to those who drank 0–1 cups (Palatini et al 2007). Around 12% of this Finnish cohort drank unfiltered pot-boiled coffee. Interestingly in this study, the risk in subjects who drank 8 or more cups per day was only increased by 14%, and adjustment for baseline BP attenuated the associations (Palatini et al 2007).

In conclusion, prospective epidemiological studies do not provide a clear picture on the role of coffee intake in the development of hypertension. Risks may be lower both in abstainers and in subjects with a relatively high intake, although this was not found in all studies. Especially in female coffee drinkers, the risk of hypertension may be reduced at higher intakes (>4–6 cups/day). The number of studies, however, is small, which hampers the drawing of conclusions.

Coffee and BP: evidence from RCTs

A large number of RCT of either coffee or caffeine intake on BP have been performed, which have been systematically reviewed by different research groups (Myers 1988; Jee et al 1999; Nurminen et al 1999; Noordzij et al 2005). Myers (1988) provided an overview of BP trials with daily caffeine doses around 100–500 mg that were published between 1978–1987 and found that caffeine did not cause any persistent increase in BP. Furthermore, he concluded that individuals who do not regularly ingest caffeine may experience an increase in BP when drinking coffee, but that tolerance develops in 2–3 days with BP returning to initial levels (Myers 1988). Jee and colleagues (1999) performed a meta-analysis of 11 RCT with a median duration of 56 days. After pooling of trials, they found a significant increase in BP of 2.4 mmHg systolic and 1.2 mmHg diastolic for a median coffee intake of 5 cups per day (Jee et al 1999). Also, they reported a dose-response relation between number of cups consumed and change in BP, and larger effects in younger subjects (Jee et al 1999).

Nurminen and colleagues (1999) performed an extensive descriptive review of both epidemiological and experimental studies, from which they concluded that regular coffee consumption may be harmful to hypertension-prone subjects. In their paper, the authors separated studies with continuous ambulatory BP monitoring from studies with

BP measurements at the research center. The 10 studies that used ambulatory BP were of shorter duration, and half of these lasted less than 1 week. Caffeinated coffee (3+ cups per day) increased ambulatory BP by around 3–6 mmHg in 7 studies, whereas BP was not affected in the remaining 3 studies (Nurminen et al 1999). In controlled trials that used office BP, caffeinated coffee caused BP elevation in one-third of the studies (Nurminen et al 1999).

Noordzij and colleagues (2005) performed a meta-analysis of caffeine and coffee trials with a median duration of 42 days, excluding studies that lasted <7 days. When caffeine trials ($n=7$) and coffee trials ($n=18$) were analyzed separately, BP elevations appeared to be 4 times greater for caffeine given as tablets (4.2 mm Hg systolic and 2.4 mm Hg diastolic) than for caffeinated coffee (1.2 and 0.5 mm Hg, respectively). These differences could not be explained by caffeine dose, since the median daily caffeine dose in caffeine trials was 400 mg per day (range, 295–750 mg) and in coffee trials was 455 mg per day (range, 225–798 mg) (Geleijnse 2006). Overall, this study showed a somewhat larger systolic BP response to coffee or caffeine in younger populations (mean age <40 years), as was also the case in the meta-analysis by Jee and colleagues (1999). It should be noted that the trials that formed the basis of the abovementioned meta-analyses were mostly conducted in normotensive Caucasian populations that were relatively young (Jee et al 1999; Noordzij et al 2005). Therefore, the BP estimates from these meta-analyses cannot be generalized to other type of populations, eg, hypertensives, elderly or Asian subjects, or to the population at large.

Recently, a number of new trials related to coffee and BP have been published. A Japanese research group repeatedly demonstrated a beneficial effect of chlorogenic acid from green bean coffee extract on vasoreactivity and BP in mild hypertensives, with reductions around 3–4 mmHg (Ochiai et al 2004; Kozuma et al 2005; Watanabe et al 2006). Another Japanese trial showed that coffee (3+ cups per day) reduced BP by 7–10 mmHg systolic and 3–7 mmHg diastolic in (pre)hypertensive men who consumed 60 ml of alcohol per day (Funatsu et al 2005). Sudano and colleagues (2005) showed that caffeinated coffee blunted the BP response to mental stress in habitual, but not in nonhabitual coffee drinkers, despite preserved muscle sympathetic nervous activation. Their findings led to the conclusion that ingredients other than caffeine could be responsible for the stimulating effect of coffee on the cardiovascular system (Sudano et al 2005).

When combining evidence from RCT of coffee and BP, it can be concluded that short-term (<12 weeks) intake

of caffeinated coffee, both filtered and instant, causes BP elevations around 2/1 mmHg when compared to abstinence or use of decaffeinated coffee. BP elevations were more apparent when ambulatory BP monitoring was applied (Nurminen et al 1999). Stratified meta-analyses suggest larger BP elevations in the case of prolonged coffee use (>6 weeks), a short run-in period (<1 week), younger age (<40 years) and high levels of coffee intake (>5 cups/d) (Jee et al 1999; Noordzij et al 2005). However, it should be emphasized that there is a lack of trials of coffee and BP in elderly, hypertensives and non-Caucasians and no conclusions with regard to these subgroups can be drawn. In addition, long-term trials of coffee and BP are warranted. As a suggestion, habitual noncoffee drinkers may be randomized to daily use of pills or capsules that contain filtered coffee extract or placebo for a period of >1 year. Preferably, a 2×2 factorial design should be used so that the effect of caffeine can be studied separately from other substances in coffee.

Coffee and BP: biological pathways

Most available evidence points toward caffeine as the BP-raising agent in coffee. Caffeinated and decaffeinated coffee were compared in a randomized double-blind, crossover trial in 45 habitual coffee drinkers (Van Dusseldorp et al 1989). Daily intake of 5 cups of decaffeinated coffee for a period of 6 weeks significantly reduced ambulant BP by 1.5 mmHg systolic and 1.0 mmHg diastolic, compared to a similar amount of regular coffee (Van Dusseldorp et al 1989). A number of mechanisms have been proposed by which caffeine could raise BP, including sympathetic overactivation, antagonism of adenosine receptors, increased norepinephrine release via direct effects on the adrenal medulla, renal effects, and activation of the renin–angiotensin system. These mechanisms have been discussed in detail by others (Robertson et al 1978; Myers 1988; Nurminen et al 1999). More recently, a Japanese group showed enhanced endothelium-dependent vasodilatation after acute caffeine administration in young healthy men (Umamura et al 2006), a mechanism by which caffeine could lower BP. From experimental research it has become clear the caffeine administration acutely raises BP, but tolerance to this effect develops rapidly and heavy coffee drinkers are less likely to show a BP response after caffeine intake (Robertson et al 1981; Ammon et al 1983; Myers 1988).

As shown in the meta-analysis by Noordzij and colleagues (2005) the BP-raising effect of caffeine when ingested through coffee is probably less compared to caffeine tablets. One possible explanation could be a difference

in bioavailability of caffeine from coffee and tablets. Also, coffee is a rich source of bioactive compounds that may lower BP (Clarke and Macrae 1985; Schaafsma 1989; Mazur et al 1998; Bonita et al 2007; Diaz-Rubio and Saura-Calixto 2007). The amount of caffeine and other components in coffee made in a household coffee-maker with a paper filter was quantified in the Netherlands (Schaafsma 1989). It was estimated that 5 cups of coffee per day contribute to approximately 26% of the daily intake of potassium, 12% of the daily intake of magnesium, 10% of the daily intake of manganese, and 15% of the daily intake of niacin (Schaafsma 1989). Apart from minerals and trace elements, coffee is a rich source of polyphenols, including chlorogenic acid and the lignan secoisolaricresinol (Mazur et al 1998; Bonita et al 2007). Recently, it was shown that brewed coffee (espresso, filter, instant) contains high amounts of soluble fibre and associated antioxidant polyphenols (Diaz-Rubio and Saura-Calixto 2007). It is possible that BP-lowering minerals and polyphenols in coffee outweigh potential adverse effects of caffeine. This hypothesis was confirmed by the prospective study in US Nurses (Winkelmayer et al 2005), in which caffeinated cola that is poor in polyphenols increased the risk of hypertension whereas coffee did not. Also, the meta-analysis by Noordzij and colleagues (2005) showed that caffeine intake through tablets could be more detrimental to BP than caffeine intake through coffee.

In the present review, psychosocial factors related to the habit of coffee drinking are not addressed, but it is conceivable that such factors may contribute to either beneficial or adverse effects on BP. Furthermore, interaction of coffee with smoking, alcohol consumption and mental stress may affect BP (Myers 1988), but a discussion of mechanisms that could underlie such effects is beyond the scope of this review.

In summary, there are many possible biological pathways through which a variety of bioactive substances in coffee may influence BP, either resulting in an overall BP-lowering or a BP-raising effect of coffee. Effects of coffee on BP, if any, probably result from activation or inhibition of different pathways, and not from caffeine-related pathways only.

Discussion

Epidemiological studies of long-term consumption of filtered coffee do not support the hypothesis that filtered caffeinated coffee is detrimental to BP. On the contrary, there is observational evidence that high intake (>4 cups per day) may even reduce the risk of hypertension, especially in women. The nature of the relationship between coffee and BP is not yet clear, and further investigation is needed to find out whether

abstainers are at a lower or higher risk of hypertension than occasional coffee drinkers (1–2 cups per day). Findings from experimental studies suggest a BP-raising effect of coffee of a few mmHg in the short-term (<3 months), but apparently this effect does not translate into an increased risk of hypertension in the long-term. Both observational and experimental data suggest larger BP elevations during coffee intake at younger compared to older age (ie, >40 years). However, it should be noted that studies on coffee and BP in the elderly are sparse, as are studies in several other population subgroups, and long-term intervention studies.

A number of methodological issues need further consideration. Firstly, it has been well established that caffeinated coffee causes an acute rise in BP shortly after exposure (Nurminen et al 1999), as is the case for other lifestyle factors such as smoking, alcohol intake, physical activity, and even talking. Such acute physiological responses are mostly transient, with BP returning to initial levels within hours. It is not yet clear whether repeated BP elevations during the day could eventually lead to persistent hypertension. For coffee, on basis of available evidence from prospective studies, this does not seem to be the case. In this respect, it should be noted that office BP measurements both in epidemiological and experimental studies are not always performed in fasting conditions. From available data, it is hard to judge to what extent coffee intake prior to BP assessment (ie, acute BP effects) influenced the outcomes of the studies. To illustrate, in a trial in young Swiss women, coffee consumption on the testing day was associated with elevated BP whereas no association was found with habitual intake (Höfer and Bättig 1993). Also, ambulatory BP monitoring that captures repeated acute BP elevations during coffee intake throughout the day may point towards a BP raising effect of coffee, while such BP elevations are probably transient. It is recommended that in future studies a sufficient time period is incorporated between the last cup of coffee and BP assessment, to avoid measurement of acute effects. Also, investigators should clearly state in their papers the time when the last cup of coffee was consumed.

Secondly, it is unclear whether occasional coffee drinkers are at higher risk of hypertension than heavy coffee drinkers. Also here, acute BP effects may blur the view on the role of coffee in the development of hypertension. There is ample evidence that habitual coffee users develop tolerance to caffeine and show no elevation in BP shortly after intake. BP responses will probably not occur in people who take many cups of coffee throughout the day ('continuous infusion' with caffeine). In studies that have no proper time period between

BP readings and the last intake of coffee or other caffeinated beverages (eg, cup of coffee or tea offered at the research center), BP may be temporarily increased in occasional, but not in heavy coffee drinkers.

Thirdly, observational epidemiological studies suggest that there may be a U-shaped relation between habitual coffee intake and BP. Such relation is less clear from RCTs, in which effects of coffee on BP are mostly in the positive direction or absent. This discrepancy may be related to the duration of trials compared to cohort studies, and it is possible that short-term BP effects are not persistent in time. However, other (methodological) aspects may also contribute to the discrepancy. Trials generally focus on one or two specific doses of coffee, mostly in the high range (around 5 cups per day), and there is a lack of experimental data on the effect of low intakes on BP. Furthermore, in many trials the control treatment comprises cessation of coffee use, which from a physiological point of view is truly different from non-use in observational studies. 'Habitual abstainers' do not drink coffee because of disliking, health problems (eg, stomach complaints), financial aspects, or other reasons. In addition, observational studies are often population-based, whereas trials are conducted in selected groups subjects. Finally, coffee consumption in trials is carefully monitored whereas observational studies make use of self-reported data. The latter could lead to misclassification of subjects. To illustrate, subjects who are aware of having high BP may find it socially desirable to report occasional use of coffee rather than higher levels of intake because of a general belief that coffee could be harmful to health. Random misclassification of coffee intake, as discussed previously, can lead to weakened associations between coffee and BP levels or hypertension.

In conclusion, it is at present unclear whether habitual coffee drinking is related to risk of hypertension, although most evidence suggest that this is not the case. At this moment, there is no reason to refrain from coffee for the prevention of hypertension. The precise nature of the relation between coffee and BP is not yet clear. More prospective studies of coffee intake and incident hypertension are needed, as are long-term randomized placebo-controlled trials. Future studies may focus on different doses of coffee, and on specific population subgroups such as the elderly, hypertensives, and non-Caucasians. With regard to underlying biological mechanisms, research should not only focus on the BP-raising properties of caffeine but also on the potentially beneficial effect of other substances in coffee, such as polyphenols, soluble fibre, and potassium.

Disclosure

The author reports no conflicts of interest in this work.

References

- Ammon HP, Bieck PR, Mandalaz D, et al. 1983. Adaptation of blood pressure to continuous heavy coffee drinking in young volunteers. A double-blind crossover study. *Br J Clin Pharmacol*, 15:701–6.
- Andersen LF, Jacobs DR Jr, Carlsen MH, et al. 2006. Consumption of coffee is associated with reduced risk of death attributed to inflammatory and cardiovascular diseases in the Iowa Women's Health Study. *Am J Clin Nutr*, 83:1039–46.
- Beilin LJ, Puddey IB, Burke V. 1999. Lifestyle and hypertension. *Am J Hypertens*, 12:934–45.
- Bonita JS, Mandarano M, Shuta D, et al. 2007. Coffee and cardiovascular disease. In vitro, cellular, animal, and human studies. *Pharmacol Res*, 55:187–98.
- Burke V, Beilin LJ, German R, et al. 1992. Association of lifestyle and personality characteristics with blood pressure and hypertension: a cross-sectional study in the elderly. *J Clin Epidemiol*, 45:1061–70.
- Clarke RJ, Macrae R. 1985. Coffee, Volume 1: Chemistry. London and New York: Elsevier Applied Science.
- Diaz-Rubio ME, Saura-Calixto F. 2007. Dietary fiber in brewed coffee. *J Agric Food Chem*, 55:1999–2003.
- Funatsu K, Yamashita T, Nakamura H. 2005. Effect of coffee intake on blood pressure in male habitual alcohol drinkers. *Hypertens Res*, 28:521–7.
- Geleijnse JM, Kok FJ, Grobbee DE. 2004. Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations. *Eur J Publ Health*, 14:235–9.
- Geleijnse JM. 2006. Caffeine and incident hypertension in women. *JAMA*, 295:2135–6.
- Grobbee DE, Rimm EB, Giovannucci E, et al. 1990. Coffee, caffeine, and cardiovascular disease in men. *N Eng J Med*, 323:1026–32.
- Gyntelberg F, Hein HO, Suadicani P, et al. 1995. Coffee consumption and risk of ischaemic heart disease – a settled issue? *J Intern Med*, 237:55–61.
- Hino A, Adachi H, Enomoto M, et al. 2007. Habitual coffee but not green tea consumption is inversely associated with metabolic syndrome. An epidemiological study in a general Japanese population. *Diabetes Res Clin Pract*, 76:383–9.
- Höfer I, Bättig K. 1993. Coffee consumption, blood pressure tonus and reactivity to physical challenge in 338 women. *Pharmacol Biochem Behav*, 44:573–6.
- Hu G, Jousilahti P, Nissinen A, et al. 2007. Coffee consumption and the incidence of antihypertensive drug treatment in Finnish men and women. *Am J Clin Nutr*, 86:457–64.
- Jee SH, He J, Whelton PK, et al. 1999. The effect of chronic coffee drinking on blood pressure. A meta-analysis of controlled clinical trials. *Hypertension*, 33:647–52.
- Jenner DA, Puddey IB, Beilin LJ, et al. 1988. Lifestyle- and occupation-related changes in blood pressure over a six-year period in a cohort of working men. *J Hypertens Suppl*, 6:S605–7.
- Kearney PM, Whelton M, Reynolds K, et al. 2005. Global burden of hypertension: analysis of worldwide data. *Lancet*, 365:217–23.
- Kirchoff M, Torp-Pedersen C, Hougaard K, et al. 1994. Casual blood pressure in a general Danish population. Relation to age, sex, weight, height, diabetes, serum lipids and consumption of coffee, tobacco and alcohol. *J Clin Epidemiol*, 47:469–74.
- Klag MJ, Wang NY, Meoni LA, et al. 2002. Coffee intake and risk of hypertension: The Johns Hopkins Precursors Study. *Arch Intern Med*, 162:657–62.
- Klatsky AL, Friedman GD, Armstrong MA. 1986. The relationship between alcoholic beverage use and other traits to blood pressure. A new Kaiser Permanente study. *Circulation*, 73:628–36.
- Kozuma K, Tsuchiya S, Kohori J, et al. 2005. Antihypertensive effect of green coffee bean extract on mildly hypertensive subjects. *Hypertens Res*, 28:711–18.

- Lancaster T, Muir J, Silagy C. 1994. The effects of coffee on serum lipids and blood pressure in a UK population. *J Royal Soc Med*, 87:506–7.
- Lang T, Bureau JF, Degoulet P, et al. 1983a. Blood pressure, coffee, tea and tobacco consumption: an epidemiologic study in Algiers. *Eur Heart J*, 4:602–7.
- Lang T, Degoulet P, Aime F, et al. 1983b. Relation between coffee drinking and blood pressure: analysis of 6,321 subjects in the Paris region. *Am J Cardiol*, 52:1238–42.
- Lewis CE, Caan B, Funkhouser E, et al. 1993. Inconsistent associations of caffeine-containing beverages with blood pressure and with lipoproteins: the CARDIA Study: Coronary Artery Risk Development in Young Adults. *Am J Epidemiol*, 138:502–7.
- Lopez-Garcia E, van Dam RM, Willett WC, et al. 2006. Coffee consumption and coronary heart disease in men and women. A prospective cohort study. *Circulation*, 113:2045–53.
- Löwik MR, Hofman Z, Kok FJ, et al. 1991. Nutrition and blood pressure among elderly men and women (Dutch Nutrition Surveillance System). *J Am Coll Nutr*, 10:149–55.
- MacMahon S, Peto R, Cutler J, et al. 1990. Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet*, 335:765–74.
- Mazur WM, Wahala K, Rasku S, et al. 1998. Lignan and isoflavonoid concentrations in tea and coffee. *Br J Nutr*, 79:37–45.
- Mukamal KJ, Maclure M, Muller JE, et al. 2004. Caffeinated coffee consumption and mortality after acute myocardial infarction. *Am Heart J*, 147:999–1004.
- Myers MG. 1988. Effects of caffeine on blood pressure. *Arch Intern Med*, 148:1189–93.
- Narkiewicz K, Maraglino G, Biasion T, et al. 1995. Interactive effects of cigarette and coffee on daytime systolic blood pressure in patients with mild essential hypertension. HARVEST Study Group (Italy). Hypertension Ambulatory Recording VEnetia Study. *J Hypertens*, 13:965–70.
- Noordzij M, Uiterwaal CSPM, Arends LR, et al. 2005. Blood pressure response to chronic intake of coffee and caffeine: a meta-analysis of randomized controlled trials. *J Hypertens*, 23:921–8.
- Nurminen ML, Niittynen L, Korpela R, et al. 1999. Coffee, caffeine and blood pressure: a critical review. *Eur J Clin Nutr*, 53:831–9.
- Ochiai R, Jokura H, Suzuki A, et al. 2004. Green coffee bean extract improves human vasoreactivity. *Hypertens Res*, 27:731–7.
- Palatini P, Dorigatti F, Santonastaso M, et al. 2007. Association between coffee consumption and risk of hypertension. *Ann Med*, 39:545–53.
- Periti M, Salvaggio A, Quaglia G, et al. 1987. Coffee consumption and blood pressure: an Italian study. *Clin Sci*, 72:443–7.
- Prineas RJ, Jacobs DR, Crow RS, et al. 1980. Coffee, tea and VPB. *J Chron Dis*, 33:67–72.
- Robertson D, Frohlich JC, Carr RK, et al. 1978. Effects of caffeine on plasma renin activity, catecholamines and blood pressure. *N Engl J Med*, 298:181–6.
- Robertson D, Wade D, Workman R, et al. 1981. Tolerance to the humoral and hemodynamic effects of caffeine in man. *J Clin Invest*, 67:1111–17.
- Salvaggio A, Periti M, Miano L, et al. 1990. Association between habitual coffee consumption and blood pressure levels. *J Hypertens*, 8:585–90.
- Schaafsma G. 1989. [De samenstelling van koffie in Nederland: cafeïne, mineralen, spoorelementen en vitamines]. *Voeding*, 50:223.
- Schwarz B, Bischof HP, Kunze M. 1994. Coffee, tea, and lifestyle. *Prev Med*, 23:377–84.
- Shirlow MJ, Berry G, Stokes G. 1988. Caffeine consumption and blood pressure: an epidemiological study. *Int J Epidemiol*, 17:90–7.
- Sofi F, Conti AA, Gori AM, et al. 2006. Coffee consumption and risk of coronary heart disease: A meta-analysis. *Nutr Met Cardiovasc Dis*, 17:209–23.
- Stamler J, Stamler R, Neaton JD. 1993. Blood pressure, systolic, and diastolic, and cardiovascular risks. US population data. *Arch Intern Med*, 153:598–615.
- Stensvold I, Tverdal A, Foss OP. 1989. The effect of coffee on blood lipids and blood pressure: results from a Norwegian cross-sectional study, men and women, 40–42 years. *J Clin Epidemiol*, 42:877–84.
- Sudano I, Spieker L, Binggeli C, et al. 2005. Coffee blunts mental stress-induced blood pressure increase in habitual but not in nonhabitual coffee drinkers. *Hypertension*, 46:521–6.
- Uiterwaal CSPM, Verschuren WMM, Bueno-de-Mesquita HB, et al. 2007. Coffee intake and incidence of hypertension. *Am J Clin Nutr*, 85:718–23.
- Umemura T, Ueda K, Nishioka K, et al. 2006. Effects of acute administration of caffeine on vascular function. *Am J Cardiol*, 98:1538–41.
- Van Dam R, Feskens EJ. 2002. Coffee consumption and risk of type 2 diabetes mellitus. *Lancet*, 360:1477–8.
- Van Dam R, Hu F. 2005. Coffee consumption and risk of type 2 diabetes: a systematic review. *JAMA*, 294:97–104.
- Van Dusseldorp M, Smits P, Thien T, et al. 1989. Effect of decaffeinated versus regular coffee on blood pressure. A 12-week, double-blind trial. *Hypertension*, 14:563–9.
- Wakabayashi K, Kono S, Shinchi K, et al. 1998. Habitual coffee consumption and blood pressure: a study of self-defense officials in Japan. *Eur J Epidemiol*, 14:669–73.
- Watanabe T, Arai Y, Mitsui Y, et al. 2006. The blood pressure-lowering effect and safety of chlorogenic acid from green coffee bean extract in essential hypertension. *Clin Exp Hypertens*, 28:439–49.
- Willett WC, Stampfer MJ, Manson JE, et al. 1996. Coffee consumption and coronary heart disease in women. A ten-year follow-up. *JAMA*, 275:458–62.
- Wilson PW, Garrison RJ, Kannel WB, et al. 1989. Is coffee consumption a contributor to cardiovascular disease? Insights from the Framingham Study. *Arch Intern Med*, 149:1169–72.
- Winkelmayer WC, Stampfer MJ, Willett WC, et al. 2005. Habitual caffeine intake and the risk of hypertension in women. *JAMA*, 294:2330–5.
- Woodward M, Tunstall-Pedoe H. 1999. Coffee and tea consumption in the Scottish Heart Health Study follow up: conflicting relations with coronary risk factors, coronary disease, and all cause mortality. *J Epidemiol Community Health*, 53:481–7.