THE EFFECTS OF CAFFEINE ON BLOOD PRESSURE AND HEART RATE: A REVIEW

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ABSTRACT

Concerns have been raised frequently about caffeine's potential for increasing blood pressure (BP) and posing a risk for cardiovascular disease. This review surveys research concerning the effects of caffeine on BP and heart rate (HR). Tolerance to caffeine, family history of hypertension, borderline hypertension, and hypertension are also examined as potential moderators. Results from epidemiological studies are inconsistent. Experimental laboratory studies have generally found that caffeine produces acute rises in systolic and diastolic BP that are additive to any stress-induced increases. Synergistic effects which might pose a more serious risk are rarely found. Heart rate data are less consistent, possibly due to the different ways HR is measured. Tolerance to the cardiovascular effects of caffeine has reliably been reported; however, overnight abstinence may be sufficient to negate tolerance effects to most levels of caffeine ingestion in typical caffeine users. Though caffeine drinkers may exhibit acute increases in BP, the long-term effects appear to be minimal. However, persons at risk for hypertension may be more vulnerable to the BP effects of caffeine.


INTRODUCTION

Caffeine, a minor stimulant, is one of the most widely consumed drugs in North America, with 80% of individuals in the United States over the age of 20 drinking coffee regularly (1). Caffeine is also ingested through tea, cola and other soft drinks, cold medications, diet pills, headache remedies, allergy tablets, antacids, and sleep prevention compounds. Despite its heavy use, concerns have been raised about caffeine as a potential risk factor for cardiovascular disease, in part because caffeine may increase blood pressure (BP) and enhance cardiovascular reactivity, defined as the increase in BP in response to a stressor. If accurate, these statements imply that caffeine may increase the incidence of coronary heart disease, kidney disease, and mortality (2–4). Concern about the effects of caffeine has increased the popularity of decaffeinated coffee and other products.

Blood pressure elevation because of caffeine seems plausible in light of several related physiological mechanisms. Caffeine affects neurotransmission at both central and peripheral sites, primarily due to its antagonistic action at adenosine receptors (5,6). Adenosine acts as an inhibitory neuromodulator of the noradrenergic neurotransmitter system. Therefore, caffeine increases norepinephrine release by decreasing the inhibitory influence of adenosine (7,8). Within the central nervous system, this action increases activity of the cerebral cortex and the locus coeruleus leading to arousal and vigilance (7). The peripheral influences of caffeine may also be mediated, in part, by increasing norepinephrine release (6). Norepinephrine serves as the neurotransmitter of the sympathetic nervous system, providing a vasoconstrictor influence on blood vessels and increasing both heart rate (HR) and contractility. Caffeine may therefore increase BP and heart rate by diminishing the inhibitory influence of adenosine at sympathetic terminals. In addition, caffeine may stimulate sympathetic drive via increased activity of the locus coeruleus, which is correlated with increased sympathetic nerve activity (9). These two mechanisms would act together to increase sympathetically-mediated vasoconstriction. Caffeine may also increase BP by directly antagonizing adenosine-mediated vasodilation (6). Thus, caffeine can act at multiple sites within the central and peripheral nervous systems to influence cardiovascular control. These mechanisms suggest that caffeine should have acute effects on BP and HR, but whether there are chronic effects is more unclear. Furthermore, complete tolerance to the cardiovascular effects of caffeine has been demonstrated, where repeated exposure to caffeine results in no BP change (10). Therefore, it is difficult to predict theoretically if cardiovascular effects of caffeine will be manifested.

This review considers four questions regarding the effects of caffeine on BP. First, does caffeine use produce acute or chronic elevations in BP and HR across individuals? Second, are individuals with cardiovascular disorders (e.g. borderline hypertension or hypertension) at increased risk (exaggerated cardiovascular response) if BP is already elevated? Third, does caffeine exaggerate the cardiovascular response to stress? Fourth, has tolerance to the cardiovascular effects of caffeine been reliably demonstrated and are regular coffee drinkers completely tolerant to the effects of caffeine such that no pressor response is manifested? The present article will attempt to answer these questions through a critical, descriptive review of the literature on caffeine and BP. Although there are several excellent previous reviews of the effects of caffeine (11–14), all are dated and/or omit a portion of relevant BP studies. In addition, none provide systematic consideration of the potentially important role of tolerance, which may obscure results. The present article provides a comprehensive, up-to-date review of the effects of caffeine on BP and HR and highlights the
importance of tolerance in understanding the empirical literature and its implications for health.

**REVIEW OF THE LITERATURE**

The papers selected for this review were identified by MEDLINE and PSYCHLIT searches from 1980 to the present. Studies were included only if the participants were healthy, with the exception that hypertensive samples were included. This review begins by surveying the epidemiological data and then summarizes laboratory experimental research concerning the effects of acute doses of caffeine on resting BP; reactivity to laboratory stressors, studies of chronic caffeine consumption, and research employing ambulatory BP measurement. Potential moderators of the effects of caffeine, such as a family history of hypertension, borderline hypertension, hypertension, and tolerance to the effects of caffeine will be discussed where relevant data are available.

**Epidemiological Surveys**

The epidemiological studies are summarized in Table 1 which presents information about the sample size, sample characteristics such as age and gender, and unique aspects of the study. Column 4 summarizes study findings. Column 5 indicates which, if any, traditional risk factors for BP were statistically controlled for by the researchers. The final column indicates whether complete tolerance was evident.

The first part of Table 1 presents studies relying on self-reported caffeine use and assessing whether caffeine consumption chronically elevates BP. The results of these studies are inconsistent. There are reports of no association between caffeine and systolic blood pressure (SBP) and diastolic blood pressure (DBP) (15); positive associations with SBP (16) or with DBP (17,18); inverse relationships with SBP and DBP (19–22), with only SBP (23), or with only DBP (24); and even a curvilinear association with SBP and DBP (25). Periti et al. (22) and Salvaggio et al. (20) suggested that coffee consumption may alleviate anxiety, which, in turn, may lead to a reduction in BP. Although this hypothesis is interesting, numerous experimental studies have demonstrated that caffeine actually increases anxiety [(26–32) but see (33)] and a review on the withdrawal effects of caffeine (34)].

The majority of studies in the first part of Table 1 provide no evidence of tolerance to the cardiovascular effects of caffeine developing after chronic use. Even when participants are categorized on level of habitual use, there is no systematic BP difference between light and heavy caffeine consumers. However, in the best controlled study, Sharp and Benowitz (35) provided convincing evidence of tolerance comparing infrequent and habitual caffeine users. Participants were matched on blood serum level of caffeine, and after adjusting for traditional risk factors (see Table 1), infrequent users had higher average SBP and DBP readings than those of habitual users. Thus, at the same blood serum level of caffeine as infrequent users, habitual users were at least partially tolerant to the effects of caffeine.

The few epidemiological studies assessing the acute effects of caffeine (see second part of Table 1) demonstrate a consistent, positive association between caffeine use and BP (36,37) and also illustrate the importance of the method of assessing caffeine intake. Self-reported daily use is not synonymous with whether a given individual actually consumed any coffee during a set period of time (38). The importance of this point is highlighted by Höfer and Bättig (36). When women self-reported their typical coffee use, there was no relationship between coffee use and BP. However, when the same women indicated whether or not they had consumed any coffee on the day of the BP measurement, a positive association between BP and coffee use emerged (see also 37).

Overall, the epidemiological evidence for an association between caffeine use and BP is weak. In addition, epidemiological studies cannot provide evidence of causation. Any correlation between caffeine use and BP may indicate that caffeine leads to increased/decreased BP or that individuals with higher/lower BP simply have a greater affinity for coffee. There is the possibility of a third variable that causes both the change in BP and the amount of caffeine intake. An added complication is that researchers in epidemiologic studies controlled statistically for different risk factors, making direct comparison among the studies difficult.

Smits, Thein, and van’t Laar (39) are critical of the epidemiological literature on caffeine and BP because most studies adjust for smoking status. Smoking reduces the plasma half-life of caffeine (40) and a pressor response may not be manifested unless caffeine from previous use has been primarily eliminated from the body (10). Hence, correcting for smoking could lead to an underestimation of the relation between BP and caffeine.

Finally, Schreiber et al. (41) reported that simply asking individuals to estimate the number of cups of coffee they consume is a poor index of actual caffeine ingestion. In their sample, a typical self-report measure resulted in the misclassification of 40–80% of caffeine consumers when compared to an interview assessing additional sources of caffeine, asking about weekdays and weekends, and any changes in use over the course of a year. In conclusion, epidemiological surveys yield weak, inconsistent results and have methodological limitations which preclude drawing conclusions about use and BP.

**Experimental Findings**

Attempts to causally link caffeine consumption to acute changes in BP and HR have inspired numerous experimental studies. Acute changes in BP are relevant if, as some suggest, frequent rises in BP may lead to a resetting of resting levels (42,43). The first set of studies described below consists of laboratory experiments examining the effects of caffeine on resting BP and HR. This work led to subsequent research examining the effects of combining caffeine and laboratory stressors. Research using “real world” stressors, such as examinations, occupational stress, and exercise followed. Several studies have also examined the effects of repeated or chronic exposure to caffeine. The most recent advance has been studies measuring ambulatory BP, making it possible to determine whether caffeine has an effect on BP or HR under conditions of normal consumption in daily life.

**Acute Doses of Caffeine and Resting Blood Pressure:** Laboratory studies assessing the effects of an acute dose of caffeine on resting blood pressure and heart rate are described in Table 2, which follows the same format as Table 1. Again, the importance of tolerance is highlighted, as regular caffeine consumers may be tolerant to the BP effects of caffeine and experience little or no BP change after ingestion.

Numerous studies have reported that caffeine increased SBP and DBP [or mean arterial pressure (MAP)] (44) but decreased HR (39,45–49). Several other studies also reported increases in SBP and DBP [or MAP (50)] but found no effect on