

Systematic Review of the Effect of Daily Alcohol Intake on Blood Pressure

McFadden, Christopher B., MD*
Brensinger, Colleen M., MS#
Berlin, Jesse A., ScD#
Townsend, Raymond R., MD*

* Division of Nephrology, University of Pennsylvania School of Medicine, Philadelphia, PA; # Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania School of Medicine, Philadelphia, PA

Abstract: Numerous epidemiologic investigations have found an association between a moderate intake of alcohol and increased blood pressure. Prospective studies evaluating this relationship, however, do not show reproducible findings regarding the effect of sustained alcohol intake effect on blood pressure. To further define this relationship, we performed a systematic review of studies that prospectively measured blood pressure following sustained alcohol intake (defined as daily intake of at least 1 day duration) in humans and measured blood pressure following a control period of no alcohol intake. Nine studies met entrance criteria. The review demonstrated a significant rise in systolic blood pressure (SBP) and diastolic blood pressure (DBP) of 2.7 mm and 1.4 mm Hg, respectively, following alcohol intake. The mode of blood pressure measurement, ambulatory or casual, and the duration since ethanol intake were found to be significantly associated with the changes in blood pressure. These findings may have important influences on interpreting studies measuring the effect of alcohol on blood pressure as well as regular clinical care.

Introduction:

The observation that excessive intake of ethyl alcohol is associated with a higher blood pressure is nearing its centennial mark. Lian, while caring for the French military, published his findings in 1915 demonstrating that soldiers consuming more than 2 ½ liters of wine per day were more likely to have higher blood pressure. (1) A landmark observational study published in 1977 reinforced a number of findings among smaller patient populations. (2-4) The 1977 report of the Kaiser-Permanente Multiphasic Health Examination Data, based on self-administered questionnaires from 83,947 men and women, concluded that three or more drinks per day (1 drink generally being equal to 14 grams of ethanol) was a risk factor for hypertension across races and in both sexes.(5) Other studies, challenging the threshold effect reported in the Kaiser study, suggest an effect even at lower levels of intake. (6, 7)

Attempts to evaluate the association between alcohol intake and blood pressure, in a prospective manner, are hindered by several limitations, including the difficulty in designing experimental studies of chronic alcohol use, the potential for confounding due to alcohol withdrawal, the immediate vasodepressor effect of alcohol consumption, the appropriate timing and frequency of blood pressure measurements, and the variability in type and frequency of alcohol intake. Small, prospective studies suggest that daily alcohol intake, particularly when higher than 42 grams/ day, raises blood pressure.(8-18) However, most of these studies are not randomized and are contrasted by reports of well-designed trials, using ambulatory blood pressure (ABP) monitoring, finding no discernable effect of alcohol on mean blood pressure.(19-21) Moreover, studies with more frequent BP monitoring have noted biphasic effects of ethanol on BP, suggesting

that previous, well-recognized conclusions about the hemodynamic effects of ethanol on BP may have been exaggerated due to the timing, and infrequency, of BP measurement.

We performed a systematic review of prospective, controlled human trials evaluating the influence of daily alcohol intake on blood pressure, as measured at least 24 hours after the initiation of ethanol consumption, to determine more precisely the hemodynamic effects of alcohol on blood pressure. Specifically, we looked for investigations addressing change in blood pressure following alcohol intake versus a non-ethanol control arm, during which no alcohol intake occurred, in hypertensives or non-hypertensives whose base-line alcohol intake was light (< 0.22 ounces or 3 grams/ day), moderate (0.22 to <1.0 ounces or 14 grams /day), or heavy (> 1.0 ounce or 14 grams/day) as defined by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). (22)

Methods:

A Medline based search used subject keywords “alcoholic beverages,” “ethanol,” “alcoholism,” “alcoholic intoxication,” and “temperance” to specify articles related to alcohol intake. This pool was then narrowed to articles including at least one of the following search terms: “hypertension,” “anti-hypertensive agents,” “blood pressure.” Articles were further limited to those published in English, containing abstracts, and regarding human subjects. A medical school liaison librarian assisted in the design and implementation of the search. These criteria and selected reference review produced 834 articles. Two authors (CBM, RRT) independently reviewed the abstracts or full articles to ensure compliance with the prespecified criteria. Nine articles were found to meet all criteria and included in the meta-analysis.(23-30) (31) Reasons for exclusion of the remaining 825 include observational trials (214), no ethanol intake or multiple

interventions (147), review articles (146), BP not a prespecified endpoint (100), animal or in vitro studies (96), duration of observation less than 24 hours (48), series or case reports (46), poorly described non-ethanol control phase or ethanol during non-ethanol control phase (20), and unclear ethanol dose (8).

Statistical components:

There were two primary aims of the statistical analyses. The first aim was to provide an overall summary of the mean change in blood pressure in subjects whose blood pressure was measured after alcohol consumption compared with values after non-ethanol control consumption. Non-ethanol control typically comprised an isocaloric non-ethanol solution similar in volume to the ethanol consumed. The second aim was to explain differences in blood pressure changes among the studies by evaluating factors that we anticipated, *a priori*, could potentially affect study results. We also evaluated the possibility of publication bias in the observed data, i.e., the selective publication of results with statistically significant findings. All analyses were performed using STATA version 7.0 (STATA; College Station, TX). All p-values reported are two-sided.

To accomplish the first aim, we determined fixed- and random-effects summaries of the data for systolic and diastolic blood pressure. All studies were of a cross-over design, making the outcome measure of interest the mean within-subject difference in blood pressure between measurements following alcohol consumption versus following non-ethanol control consumption. In the fixed-effects summary, we took a weighted average of the within-study mean differences, with weights equal to the inverse of the variance of that within-study difference in means. In the random-effects summary we

also took a weighted average, but incorporated the among-study variability of the results into the weights.

Variance imputation

Only one study (30) reported the mean and variance of the differences in blood pressure. The remainder reported the mean and standard deviation for the blood pressures following alcohol and, separately, following non-ethanol control. Using the p-value for the statistical difference of the means reported in the first study, the variance of the remaining studies was calculated. The variance of the difference and the post-alcohol-consumption and post-non-ethanol control-consumption variances allowed for the calculation of the implied correlation between the post-alcohol and post-non-ethanol control measurements. We estimated these correlations for systolic ($R = 0.05$) and diastolic ($R = 0.39$) blood pressure from the single study, and then applied these estimates to the estimates of the post-alcohol and post-non-ethanol control from the remaining studies in order to estimate the variance of the difference in each of those studies. (32) As a sensitivity analysis, we also assumed for the remaining studies that the correlation between the post-alcohol and post-non-ethanol control values was 0.5, re-estimated the change variances under that assumption, and recalculated the summary estimates under this assumption. Because the results of the two approaches agreed very closely, we report only the main analysis involving the actual correlation estimates.

To assess the possible influence of study design on results, we used the STATA “meta reg” routine to perform meta-regressions. These analyses regress the mean difference in blood pressures against individual study characteristics. Because of the small number of studies, we assessed only one study-level covariate at a time.

Specifically, the covariates we examined were: the use of randomization (yes/no) in assigning the order of alcohol versus non-alcohol measurements, whether an ambulatory blood pressure monitor was used, and dose of alcohol (analyzed in two ways: as a continuous variable and dichotomized as <1 mg/kg/day versus = 1 mg/kg/day). We could not examine the effect of whether the subjects were fed versus fasted because all 9 studies included only fed subjects.

Three studies reported blood pressures at several times following alcohol consumption. (23, 27, 30) For these studies, the primary analyses used the average of all these measurements and are reported as “average value analyses.” One study reported results for three independent groups;(29) these groups were treated as individual studies in the analysis leading to a total of 11 groups in the final analysis.

To assess the effects of time of measurement since alcohol consumption qualitatively, we plotted the values of the change in blood pressure (between the post-consumption and non-consumption values) against time. We then performed separate summaries including the longest and shortest follow-up times for each of the three studies. The results for the longest follow-up closely resembled those using the averages over time, so we report only the results for the average and the shortest follow-up. Finally as a test of publication bias, we used the method described by Egger as implemented in the STATA “metabias” routine. (33)

Results:

Table 1 lists characteristics of each of the included studies. Importantly, the nine studies include a variety of levels of alcohol use with most studies falling into the

moderate to heavy drinking range. Limitations in baseline ethanol intake data restrict exact classification as three studies did not report baseline alcohol intake and several included ranges crossing the previously described NIAAA category levels. Baseline intake in the remaining 6 studies ranged from 10 to 70 grams/day. Spirits in the form of vodka or whiskey were the predominant form of ethanol ingested, though three studies do not report the type of alcohol used. The studies included both hypertensives and non-hypertensives. In the 4 studies including hypertensives, an antihypertensive medication washout period of at least 1 week's duration was present prior to the initiation of the study. Similarly, when reported, in these studies an ethanol-free washout period of 7 days or periods of at least 4 days of daily alcohol or non-ethanol control solution intake preceded any BP measurements in all studies (see table). Neither the baseline level of alcohol intake nor baseline blood pressure were predetermined as entrance criteria.

The overall effect of alcohol intake on BP was a 2.7 mm Hg SBP elevation and 1.4 mm Hg DBP elevation (Table 2). For the three studies reporting measurements of blood pressure at multiple time points after alcohol consumption, we used the average blood pressures over the entire measurement period. In general, the effects were small and the findings were quite heterogeneous across studies. Results of the overall summaries (Tables 2,3, and 4) are presented using both fixed and random effects models; the text contains fixed effects results only. Differences result from heterogeneity of findings across studies, and from the fact that small studies tended to show larger differences related to alcohol. Figures 1 and 2 display these results graphically for SBP and DBP, respectively.

Investigations of potential sources of heterogeneity revealed no association between study findings and either the use of randomization in the study or the dose of alcohol administered, for the average value analyses. However, there was a very pronounced effect of the use of ABP monitoring on study results ($p < 0.001$ for the difference between the effect of alcohol in studies using ABP compared with studies not using ABP, for both systolic and diastolic blood pressure). Table 3 presents the summary effects of alcohol separately for the ABP and non-ABP studies. Overall, ABP-based studies noted a 0.6 mm SBP *decrease* and a 0.2 mm DBP *decrease* after alcohol consumption. This contrasted sharply with non-ABP studies, with the latter showing an 8.8 mm SBP increase and a 5.9 mm DBP increase after alcohol consumption. Only non-ABP results are significant between post ethanol and post-non-ethanol control. The heterogeneity of the findings was considerably reduced after stratification on ABP use. Consequently, the results of fixed and random-effects models agree closely in these stratified analyses.

The effects of time between alcohol consumption and measurement of blood pressure are shown in Figures 3 and 4 using data from the three studies reporting multiple time points. All of these studies used ABP recordings. To quantify these findings, the effect of alcohol is summarized separately using the shortest time point measured and the longest time point measured in these three studies. For the longest time point, the results closely paralleled those using the average over the entire time period. For the shortest time points, the results are displayed in Table 4. The effect of alcohol at the short time period is significantly negative, averaging an 11.6 mm SBP decrease and a 7.9 mm DBP decrease at an average time of 5 hours in the three studies analyzed.

In the longer value analyses, the regression analyses showed a strong positive association between dose of alcohol, treated as a continuous variable, and the magnitude of the blood pressure effect. This apparent dose effect disappeared when the ABP variable was added to the models. In those same models, the association of ABP and outcome was preserved compared with the unadjusted analyses of ABP. Thus, the dose association was confounded by ABP.

There was no significant evidence of publication bias.

Discussion:

The data in this systematic review reveal important information regarding the relationship between alcohol intake and blood pressure elevation, the temporal nature of this relationship, and the manner in which measurement technique can influence these findings. Alcohol raises blood pressure in a small but statistically significant manner, averaging 2.7 mm for SBP and 1.4 mm Hg DBP across all studies using a fixed effects model.

The effect is significantly influenced by whether or not ABP measurements are taken as part of the evaluation; a major reason for this effect is the early vasodepressor effect of alcohol intake as seen in Figures 3 and 4. This phenomenon has been described previously, (21, 23) yet determining how to integrate these findings into non-ABP studies and, more importantly, clinical care is not well understood. Specifically, following the vasodepressor effect a moderate but significant *elevation* is observed. That this change occurs 10 to 15 hours after ingestion, similar to when many patients may have been seen in casual BP measurement studies, as well as regular physician visits, is of clinical concern given the common scenario of drinking alcohol during evening hours. This

potential confounder may explain the rather striking differences between the ABP and non-ABP studies observed in this study and previous publications. Alternatively, the immediate vasodepressive effect of alcohol may bias studies assessing hypertensive status in an uncontrolled setting.

In our findings, the difference between the average SBP and DBP changes associated with alcohol intake, as measured by casual vs. ABP recordings, were 9.4 and 6.1 mm Hg, respectively (from Table 3). The overall average BP change, as recorded by ABP devices only, was *negative*, though not reaching statistical significance. Other studies evaluating the effect of interventions as measured by ABP versus casual office BP measurement have observed similar findings which have been ascribed to a lack of placebo and white coat hypertension effects on ABPM.(34, 35)

A unique feature described here is the comparison of blood pressure differences between shortest and longest follow-up periods in the ABP studies. SBP and DBP decreased 11.6 and 7.9 mm Hg, respectively, at the early time period in the 3 studies that presented multiple readings. The long-term readings, at an average of 20 hours, were not substantively different than the average change, which for ABP studies alone did not reach statistical significance. In contrast, non-ABP average blood pressures recorded at a similarly long follow-up did reach significance.

In our entry criteria, we required studies to have a period of alcohol abstinence as the control period. In comparison, the Prevention and Treatment of Hypertension Study (PATHS) evaluated the effect of ethanol reduction via behavioral modification in a multi-center, controlled, randomized fashion.(36, 37) Following enrollment of 641 hypertensives and non-hypertensives categorized as moderate to heavy drinkers, this

study followed subjects for 15 to 24 months with casual blood pressure monitoring. The 1.3 drink per day reduction obtained by the interventional group over the control group, from a baseline of 4.4 drinks/day, failed to significantly affect blood pressure.

Hypotheses for this failure included the inability to obtain sufficient between-group differences in alcohol intake or a reduced relationship between change in blood pressure and alcohol intake. Our review included only studies that rigorously maintained an alcohol free period in order to ensure differences in alcohol intake.

Importantly, our study is unique in that it combined distinct reports of ABP and casual blood pressure measurements. The combination of these techniques limits the potential for over or under representation of findings due to the exclusion of one technique. Since studies that used ABP measured post treatment (alcohol) or non-ethanol control consumption with the same method, this is a valid comparison and should not be thought of as mixing distinct measurement techniques. Instead, the change in blood pressure, regardless of measurement technique, was the measurement from which conclusions were drawn.

Prior literature has called attention to the period following alcohol consumption as a potentially important mediator of alcohol associated vascular damage.(21) (38) As we show in this systematic review, despite limited changes in the mean blood pressure readings, there is a significant rise in blood pressure between the 4 hour nadir in blood pressure readings and peak levels about 10 hours later (Figures 3 & 4). An extension of this finding is the potential effects of alcohol on the normal circadian rhythm of blood pressure.(19) Possible mechanisms to explain the effect of alcohol on blood pressure

include activation of the sympathetic nervous system (39) and an increase in serum cortisol levels. (10, 18) (9) (40)

The potential for weight gain associated with alcohol intake is an important issue. Studies evaluating this continue to see an influence when accounting for sodium intake and body weight.(17) In the group of studies included in our review, four studies documented equal weight during alcohol and non-ethanol control phase and four studies did not document weight. Importantly, three of the studies not documenting weight included isocaloric diets during the study interventions. One study did not describe either(28) and one study noted a significant decrease in weight during the alcohol intake phase.(27) Given that this is opposite to the expected role of weight gain confounding the positive effect of alcohol on blood pressure, (12, 13) it is likely this finding only reduced any potential effect of alcohol intake on blood pressure elevation.

Most interventional trials, whether included or not in our study, use alcohol intake over a set period of time in order to reasonably reproduce social alcohol intake. The included studies ranged from 1 to 8 hours in the evening. None exceeded an 8 hour intake period. While studies exist that evaluate a more continuous intake of alcohol in a population of alcoholic patients, in this case by regular intravenous infusion, methodological issues make generalizable conclusions from this study difficult. (8) Defining the importance of timing of intake, particularly sustained versus intermittent, is another area needing further investigation.

A wide variation in alcohol intake at baseline existed in the studies included in this review. Nonetheless, an adequate washout removed the potential for alcohol withdrawal to influence findings. Interestingly, in the one study that stratified groups by

alcohol intake,(29) the group with a higher baseline alcohol intake had higher blood pressures following controlled alcohol intake. This point is worth further investigation in a prospective fashion.

Efforts to provide information about hypertension and “binge” drinking have been revealing. Following exposure to 2.2 g/kg alcohol in one evening, in comparison to a control evening, a group of subjects were noted to increase SBP and DBP 5 mm Hg during the period of intoxication. (41) Blood pressure subsequently fell during the periods when alcohol levels were falling. These findings suggest some threshold level whereby the previously described vasodepressor effect of alcohol is overcome, perhaps by excessive sympathetic stimulation. Better defining this dose-response relationship is one area of potential future research.

One important criterion for study inclusion was the absence of alcohol intake in the non-ethanol control phase. As a result, some well-designed studies do not show up in the analysis but deserve mention. One set of investigators prospectively evaluated the effect of reduction of chronic alcohol intake, though not to zero, through questionnaires in series of hypertensive and non-hypertensive patients. In the hypertensive group, a reduction of alcohol intake sustained for 6 weeks, from 472 ml/week (222 grams/week) to 64 ml/week (30 grams/week), was associated with reductions in blood pressure.(12) When controlled for weight loss, the reduction in alcohol intake was associated with falls in SBP and DBP of 0.8 mm Hg and 0.7 mm Hg, respectively, for each 100 ml/week (47 grams/week) decrease in alcohol intake. A group of normotensive men demonstrated similar findings.(13) These data are similar to our findings.

Limitations of this review can be considered in two regards. First, the review suffers from the strengths and weaknesses of its composite studies. An important finding is the lack of a variety of racial groups and the paucity of females included in the studies. Most studies involved Caucasian or Asian, generally Japanese, men. African-Americans are not well represented, though several studies do not describe the racial background of their participants. Similarly, women are not appropriately represented, with only one of nine studies including women.(26) Given observed differences in the risk of hypertension in African-American men in a recent cohort study, (42)ensuring adequate racial representation in further studies is essential.

Additional limitations include the relative small number of studies and sample size, the over-representation of one group's work due to our entry criteria, and the lack of long-term follow-up. In defense of these criticisms, our *a priori* entry criteria were intended to exclude settings in which partial dose reduction, particularly when quantified by retrospective questionnaires, increased variability of findings. Similarly, rigorous dose-response studies can only feasibly be accomplished in a relative short period.

The meta-analysis technique used in this review relies on assumptions about the majority of studies regarding the variance of the differences. For our primary analyses, we based these assumptions on the variance observed in one study, (30) and the correlation between post-alcohol and post-non-ethanol control blood pressures implied by those variance estimates. We assessed the sensitivity of our results to that assumption by applying a correlation of 0.5 to alcohol intake and blood pressure. Similar findings were observed, consistent with reports validating this imputation approach in the methodological literature(32). These studies also revealed little evidence of publication

bias. The use of aggregate-level (published) data also precluded examining predictors of blood pressure at the individual subject level.

Blood pressure is an objective measurement with significant intra-day variation. Nonetheless, its use as a measure of risk for future cardiovascular events is well recognized. (43, 44). Following an initial vasodepressor effect of 2-3 mm Hg for both SBP and DBP, a slightly smaller increase in both SBP and DBP occurs. Our review identifies several aspects of this relationship that should be kept in mind and deserve further attention. First, the temporal relationship is critical. This confounding issue warrants the more frequent use of ABP devices to better assess the 24 hour alcohol and blood pressure interaction. This variability likely requires the use of other surrogate cardiovascular markers besides BP in any future studies evaluating the beneficial effect of reducing alcohol intake. Further studies are needed to more closely scrutinize the timing of alcohol intake and cardiovascular risk. Additionally, future studies should include a broader racial profile and increased number of female subjects. Finally, future studies should aim to clarify the dose response effect which appears to reach a threshold at some point between 1 and 2 g/kg/day. Pending further answers, alcohol intake is certainly worth questioning about when pursuing lifestyle modifications and the treatment of resistant hypertension.

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Table Legend

Table 1: Study Characteristics

Table 2: Summary Estimates for Overall Blood Pressure Change including Fixed and Random Effects

Table 3: Summary effects separately for ABP and non-ABP studies.

Table 4: Summary Estimates for Shortest Time Period Measurements in the 3 Studies Reporting Multiple Measurements.

Table 1: Study Characteristics

Author	Baseline Etoh	Range of Etoh	Type of etoh	Etoh exp.	Tx.	Treatment	Placebo	Hypertensive						
	n	Intake	Intake (SD)		quantity	time	BP [^]	BP [^]	Randomized	ABP used	Multiple readings	Patients	Demographics	Non-ethanol period ^{**}
Abe, H. et al., 1994	14	33 g/day	2.8	Spirits	0.47 g/kg/day	7 days	136/83	137/83	No	Yes	Yes	Yes	Not given	7d + 4d
Howes, LG et al., 1990	11	Not described	Not described	Not described	1 g/kg/day	4 days	132/77	132/77	Yes	Yes	Yes*	No	Not given	NS + 4d
Howes, LG et al., 1986	8	10-70 g/day	Not described	Not described	66 g/day	4 days	122/70	116/62	Yes	No	No	No	Not given	NS + 5d
Howes, LG et al., 1985	10	<40 g/day	Not described	Beer or spirits	80 g/day	4 days	120/66	112/60	Yes	No	No	No	Not given#	NS + 4d
Kawano, Y. et al., 1983	16	31 g/day	10.3	Spirits	0.47 g/kg/day	7 days	137/83	138/84	No	Yes	Yes	Yes	Japanese	7d + 7d
Kumagi, Y. et al., 1993	7	3.9 g/day	2.8	Spirits	40 g/day	5 days	121/71	119/70	Yes	Yes	Yes*	No	Japanese	7d + 5d
Malhotra, H., 1985	10	<100 g/week	Not described	Spirits	1 g/kg/day	5 days	133/82	130/81	No	No	No	No	Not given	14d + 5d
	10	<100 g/week	Not described	Spirits	1 g/kg/day	5 days	170/101	163/96	No	No	No	Yes	Not given	14d + 5d
	10	up to 60 g/day	Not described	Spirits	1 g/kg/day	5 days	176/105	160/98	No	No	No	Yes	Not given	0d + 5d
Ocallaghan C.J. et al., 1985	12	9.7 g/day	10.7	Spirits	1 g/kg/day	4 days	124/68	125/70	No	Yes	Yes	No	Not given	NS + 4d
Howes, LG et al., 1992	11	Not described	Not described	Not described	1 g/kg/day	4 days	128/76	128/76	Yes	Yes	Yes*	No	Not given	NS + 4d

* Only single value reported

[^] Values reported as mean if multiple readings given

Included females

** First value is prestudy alcohol-free period, second value is duration of non-alcohol control solution intake before measurements made (NS = not stated in Methods)

Table 2: BP change overall using Average

	Pooled Est.	95% CI		p value	Number
		Lower	Upper		
SBP Fixed	2.7	0.9	4.5	0.003	11
Random	3.6	0	7.3	0.052	11
Test for heterogeneity, $p < 0.01$					
DBP Fixed	1.4	0.5	2.2	0.002	11
Random	2.2	0	4.4	0.046	11
Test for heterogeneity, $p < 0.01$					

Table 3: BP Change by Recording Method

	Pooled Est.	95% CI		p value	Number
		Lower	Upper		
ABP					
SBPFixed	-0.6	-2.8	1.6	0.6	6
Random	-0.6	-2.8	1.6	0.6	6
Test for heterogeneity, $p= 0.99$					
DBPFixed	-0.2	-1.2	0.8	0.7	6
Random	-0.2	-1.2	0.8	0.7	6
Test for heterogeneity, $p= 0.93$					
Non-ABP					
SBPFixed	8.8	5.8	11.8	<0.01	5
Random	8.3	3.5	13.1	<0.01	5
Test for heterogeneity, $p =0.04$					
DBPFixed	5.9	4.2	7.6	<0.01	5
Random	5.7	3.5	7.9	<0.01	5
Test for heterogeneity, $p =0.17$					

Table 4: Effects for shortest time periods (5 hours)

SBP

	Pooled Est.	95% CI		p value	Number
		Lower	Upper		
Fixed	-11.6	-14.2	-9	<0.01	3
Random	-9.8	-15.7	-3.8	<0.01	
Test for heterogeneity, p= 0.06					

DBP

	Pooled Est.	95% CI		p value	Number
		Lower	Upper		
Fixed	-7.9	-9	-6.8	<0.01	3
Random	-7.9	-9	-6.8	<0.01	
Test for heterogeneity, p = 0.69					

Figure Legend

Figure 1: SBP effect across all studies following ethanol intake and 95% confidence intervals. The box sizes are proportional to number of enrollees. The bottom six studies used ABP recordings for blood pressure measurements.

Figure 2: DBP effect across all studies following ethanol intake and 95% confidence intervals. The box sizes are proportional to number of enrollees. The bottom six studies used ABP recordings for blood pressure measurements.

Figure 3: Overall results (SBP) for studies reporting multiple readings following alcohol intake. All three studies used ABP measurements.

Figure 4: Overall results (DBP) for studies reporting multiple readings following alcohol intake. All three studies used ABP measurements.

Figure 1

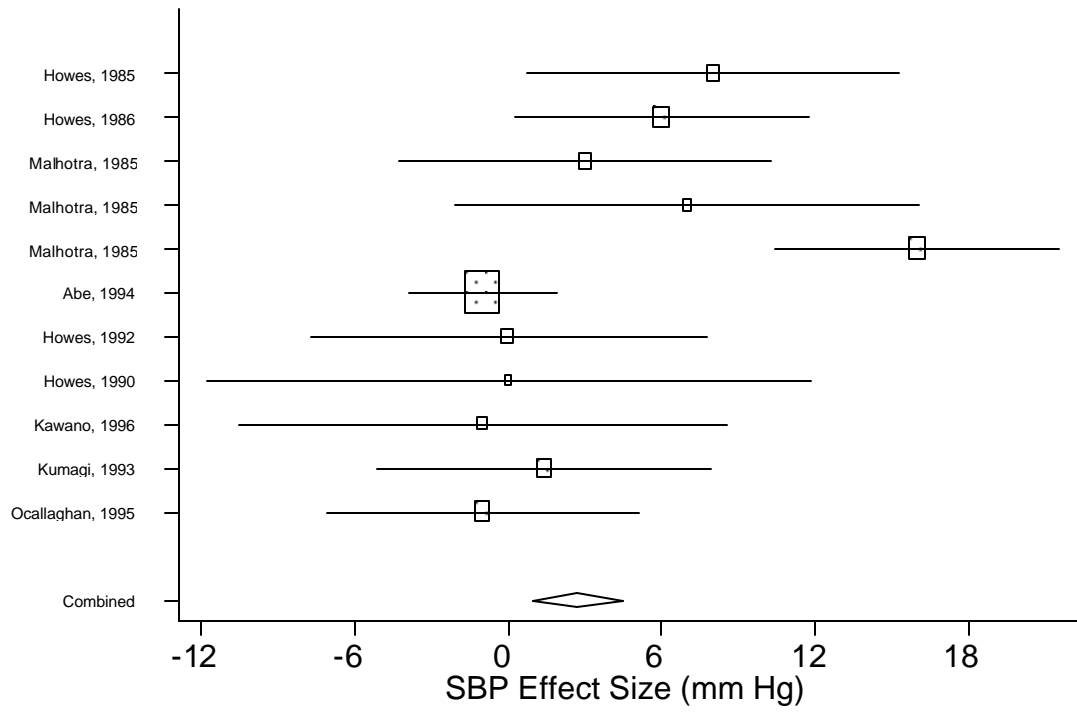


Figure 2

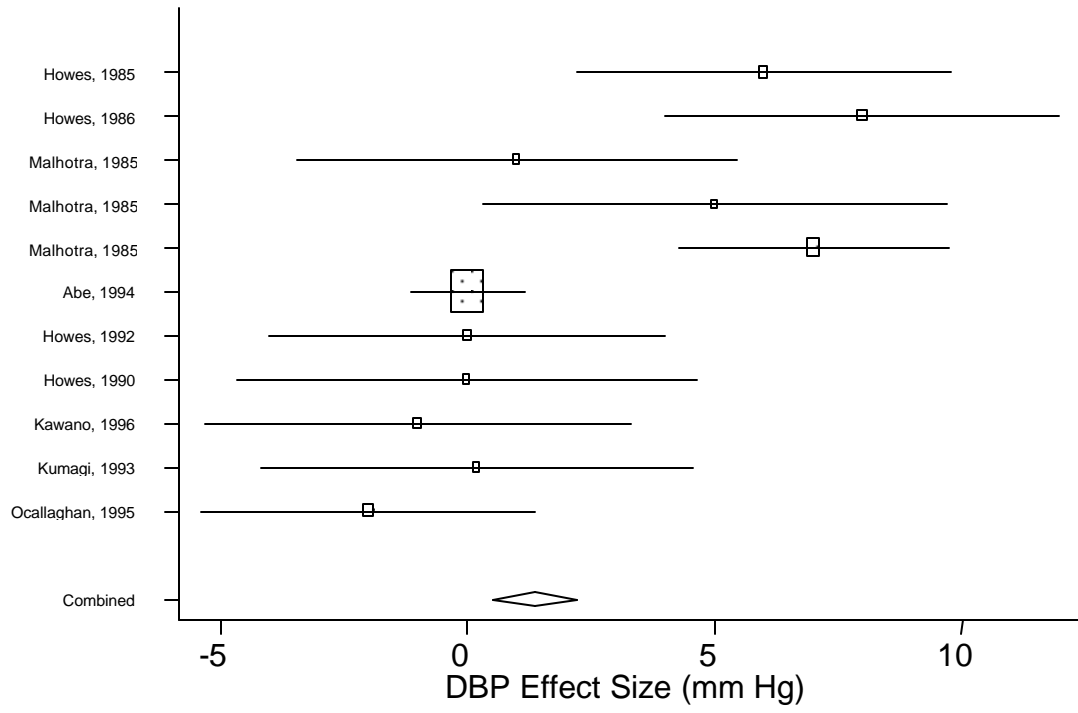


Figure 3

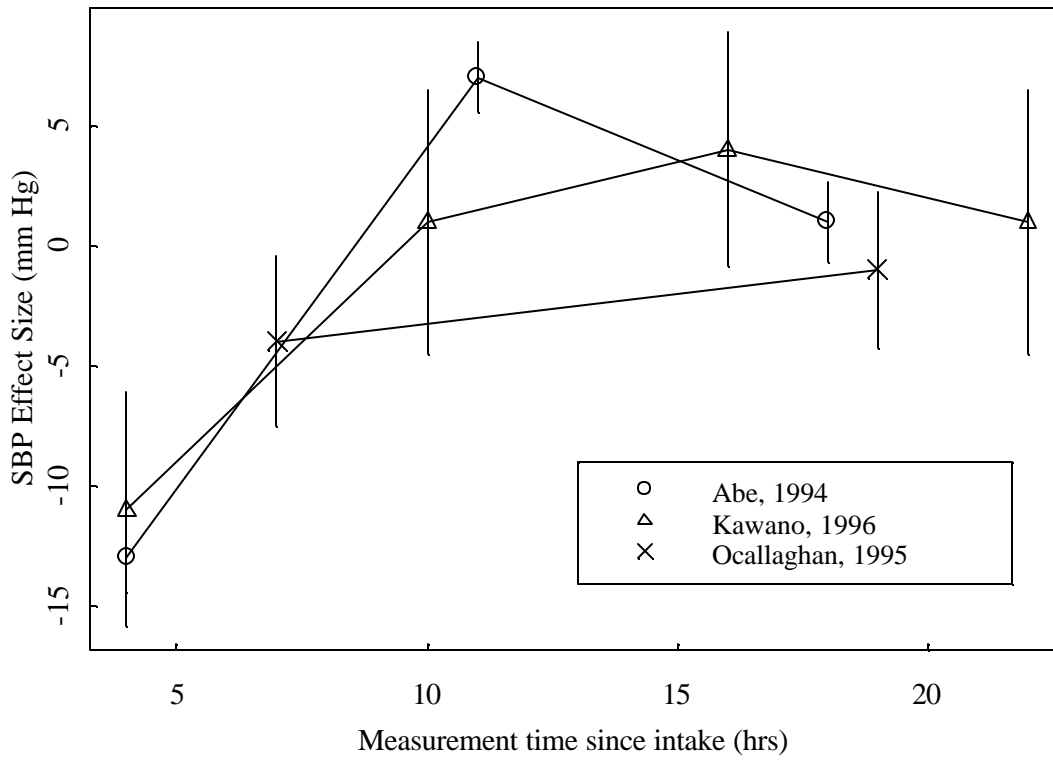


Figure 4

