Alcohol Consumption and Changes in Blood Pressure among African Americans

The Pitt County Study

Amy B. Curtis, Sherman A. James, David S. Strogatz, T. E. Raghunathan, and Siobhan Harlow

The Pitt County Study is a longitudinal investigation of anthropometric, psychosocial, and behavioral predictors of hypertension in African Americans who were aged 25-50 years at baseline in 1988. At baseline, a strong dose-response gradient was observed for alcohol consumption and blood pressure for both sexes. The current study investigated whether baseline alcohol consumption or, alternatively, changes in drinking status predicted 5-year changes in blood pressure among the 652 women and 318 men who satisfied all inclusion criteria for the longitudinal analyses. In multivariate regression analyses, baseline alcohol consumption was not significantly associated with changes in blood pressure or hypertension incidence (systolic/diastolic blood pressure >160/95 mmHg) by 1993. Change in drinking status, however, was significantly associated with changes in systolic pressure. The systolic pressure increase among individuals who initiated alcohol consumption was 6.2 mmHg (95% confidence interval (CI) 1.1-6.4) greater than abstainers, while that for individuals who reported drinking at both time points was 3.8 mmHg (95% CI 1.3-11.1) greater. Blood pressure increases for persons who discontinued drinking were comparable to those of abstainers. Results were independent of baseline age, body mass index, blood pressure, and sex. Social and economic disadvantage in 1988 was significantly associated with continuation and initiation of alcohol consumption by 1993. Am J Epidemiol 1997;146:727-33.

Received for publication February 13, 1997, and accepted for publication June 24, 1997.

Abbreviations: BMI, body mass index; CI, confidence interval.

1 Department of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, MI.
2 Survey Research Center, Institute of Social Research, University of Michigan, Ann Arbor, MI.
3 Department of Epidemiology, School of Public Health, University at Albany, SUNY, Albany, NY.
4 Department of Biostatistics, School of Public Health, University of Michigan, Ann Arbor, MI.
drinking status (e.g., initiation or discontinuance) predict these outcomes; and 3) to describe the association between baseline indicators of social and economic well-being and changes in drinking status.

MATERIALS AND METHODS

Study participants

The 1988 study population was selected through a stratified random sample of occupied black households in Pitt County, North Carolina. All 25–50 year-old African American residents were eligible to participate. Neighborhoods containing middle-class black households were oversampled in order to achieve an economically heterogeneous study population. A total of 668 men and 1,116 women (80 percent response rate) were examined. Further details on the sampling methodology were published in our earlier paper (1).

All persons with untreated baseline diastolic pressure $<$95 mmHg were eligible for follow-up in 1993. Of the 900 women who met this criterion, 780 (87 percent) were reexamined, and of the 507 men who met this criterion, 415 (82 percent) were reexamined. To reduce the likelihood of including prevalent cases of hypertension in the current study, 106 women and 88 men with baseline blood pressure $\geq$140/90 mmHg were excluded from these analyses. An additional eight women and nine men were excluded because of missing data on key study variables. Fourteen women who reported that they were currently pregnant were also excluded. After all exclusions, the analysis sample consisted of 652 women (84 percent of eligibles) and 318 men (77 percent of eligibles).

Measures

Data for both the baseline and follow-up surveys were obtained during in-home interviews (1, 13, 14). Physical measurements, taken by trained interviewers, included height, weight, and blood pressure. The protocol for obtaining these measurements was the same for 1988 and 1993. From weight and height measurements, body mass index (BMI) was calculated as weight (kg)/weight (m)$^2$. Change in BMI was determined by subtracting 1988 values from 1993 values. Approximately 15 minutes into the interview, three sitting blood pressures were taken on the right arm using a standard mercury sphygmomanometer. Systolic and diastolic blood pressure were indicated by the first and fifth Korotkoff sounds, respectively. The averages of the second and third readings were used to determine individual blood pressure values. Changes in systolic and diastolic pressure were created by subtracting the corresponding 1988 blood pressure values from the 1993 values. Hypertension incidence was defined as systolic pressure $\geq$160 mmHg or diastolic pressure $\geq$95 mmHg, or-taking antihypertensive medication in 1993.

At baseline, alcohol consumption was measured separately for beer, wine, and liquor as part of a food frequency questionnaire. The respondent was asked how many times per week or month he/she consumed alcohol. Respondents were also asked to report the usual number of drinks consumed and portion size (small, medium, or large) of each type of alcohol. Using visual aids, a medium-sized drink was defined as either 12 oz (355 ml) of beer, 4 oz (118 ml) of wine, or a shot glass (1.5 oz or 44 ml) of liquor. This information was then used to create a measure of the number of medium-sized drinks the respondent typically consumed per week. Alcohol consumption was subsequently represented by four exposure categories: none, $<$1 drink/week, $\geq$1 drink/week to $<$7 drinks/week, and $\geq$7 drinks/week.

The 1993 assessment of alcohol consumption followed the above measurement protocol, but the alcohol consumption questions stood alone rather than being part of a larger food frequency questionnaire. Because this difference in format could conceivably affect the comparability of reported consumption levels in 1988 and 1993 (15), the analyses focusing on changes in drinking behavior used categories indicating change or no change in drinking status rather than interval level data. Four drinking status categories were defined: never drink (did not report drinking in 1988 and 1993); discontinued drinking (reported drinking in 1988 but not in 1993); continued drinking (reported drinking in 1988 and 1993); and initiated drinking (reported drinking in 1993 only).

Data were also collected on demographic and behavioral characteristics, including age (years), smoking status (yes/no), strenuous exercise (exercise $>$3/week enough to breathe hard and perspire vs. other), and female hormone use (yes/no). Data on social well-being included scores on global perceived stress, anger, instrumental social support, emotional social support, marital status (yes/no), and active church membership (yes/no). Economic well-being was evaluated by data on socioeconomic status, a composite measure of respondent education and occupation (see reference 13), employment status (yes/no), home ownership (yes/no), and ability to pay for basic needs (very hard/other) such as housing, food, and medical care.

Baseline values of the above anthropometric, social, and economic variables were treated as potential confounders of the relation between alcohol consumption and changes in blood pressure or hypertension incidence. However, some variables (e.g., church membership and female hormone use) were only available...
Alcohol Consumption and Blood Pressure in African Americans

729

for 1993. In addition, a sex by age (years) product term was employed as a predictor in models pooling data for men and women. Finally, age (median split: >32 years vs. other) and BMI were considered as potential effect modifiers of any alcohol consumption-blood pressure relation.

Statistical analysis

Analyses of the association between change in blood pressure and the four categories of baseline alcohol consumption (abstainer, <1 drink/week, ≥1 drink/week to <7 drinks/week, and ≥7 drinks/week), as well as the change in alcohol consumption status (never, discontinued, continued, initiated), were conducted using linear regression models. Logistic regression models were used when hypertension incidence was the outcome.

The final model for baseline alcohol consumption and blood pressure change/hypertension incidence included sex, an age by sex product term, and baseline values of age, BMI, and blood pressure. All analyses were weighted for oversampling and nonresponse. SAS (16) and Gauss (17) statistical software programs were utilized; the latter was used to obtain covariance estimates for the weighted analyses (18).

RESULTS

Table 1 presents selected sex-specific baseline and follow-up characteristics for the analysis sample. At baseline, the average age of the respondents was just under 35 years, and average formal education around 13 years. Over 5 years, the BMI for women increased from 28.1 to 30.3, and that for men increased from 25.6 to 26.7. At baseline, nearly one in two men smoked (46 percent), compared with one in three women (34 percent). Smoking prevalence changed little over 5 years. Relatively few women consumed alcohol at either time point and, among drinkers, the number of drinks consumed per week declined over time for both sexes.

Table 2 summarizes the relation between baseline alcohol consumption and blood pressure change/hypertension incidence. There was little difference between drinkers and abstainers in the incidence of hypertension or change in blood pressure. Among drinkers, the more detailed analyses by the number of drinks per week (<1, ≥1 to <7, and ≥7 drinks/week) at baseline did not reveal any significant effects on blood pressure.

Tables 3 and 4 present selected baseline characteristics of the sample stratified by changes in drinking status. Among men, discontinued drinkers were less likely than never drinkers to be overweight. Never drinkers were the least likely of any other group to smoke cigarettes. Discontinued drinkers reported more economic difficulties at baseline than never drinkers, while baseline psychological distress scores were most elevated among continued drinkers. Finally, men who continued drinking were also less likely than never drinkers to have certain common social ties, including marriage and church membership.

Women who continued drinking were the least likely to be overweight at baseline, and, as with the men, never drinkers were much less likely to smoke than the other three groups. Persons who continued drinking or who initiated drinking were more likely than never drinkers to be of low socioeconomic status, to report high economic strain (i.e., to say that it was hard for them to pay for "basics"), and to be unemployed. Compared with never drinkers, social ties, such as marriage and church membership, were lower for women who continued to drink. Subjects who

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Men (n = 318)</th>
<th>Women (n = 652)</th>
<th>Men (n = 318)</th>
<th>Women (n = 652)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years), mean ± SE*</td>
<td>34.8 ± 0.40</td>
<td>34.4 ± 0.26</td>
<td>26.7 ± 0.24</td>
<td>30.3 ± 0.26</td>
</tr>
<tr>
<td>Education (years), mean ± SE</td>
<td>13.1 ± 0.21</td>
<td>12.9 ± 0.14</td>
<td>6.2 ± 0.59</td>
<td>2.4 ± 0.23</td>
</tr>
<tr>
<td>BMIT (kg/m²), mean ± SE</td>
<td>25.6 ± 0.22</td>
<td>28.1 ± 0.24</td>
<td>4.7 ± 0.41</td>
<td>2.4 ± 0.23</td>
</tr>
<tr>
<td>Drinks/week, mean ± SE</td>
<td>6.2 ± 0.59</td>
<td>3.4 ± 0.39</td>
<td>120.8 ± 0.55</td>
<td>116.2 ± 0.41</td>
</tr>
<tr>
<td>SBP†, mean ± SE</td>
<td>76.8 ± 0.50</td>
<td>74.6 ± 0.33</td>
<td>124.3 ± 0.77</td>
<td>121.8 ± 0.64</td>
</tr>
<tr>
<td>Married (%)</td>
<td>59.3</td>
<td>41.0</td>
<td>62.3</td>
<td>39.7</td>
</tr>
<tr>
<td>Smokers (%)</td>
<td>45.7</td>
<td>33.6</td>
<td>42.1</td>
<td>30.6</td>
</tr>
<tr>
<td>Drinkers (%)</td>
<td>57.2</td>
<td>38.3</td>
<td>46.2</td>
<td>25.9</td>
</tr>
</tbody>
</table>

* SE, standard error.
† BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.
‡ Among drinkers.
TABLE 2. Changes in systolic blood pressure (SBP) and diastolic blood pressure (DBP), and risk of hypertension* in African Americans by baseline drinking status

<table>
<thead>
<tr>
<th>Drinking status</th>
<th>No.</th>
<th>Change in SBP (mmHg)</th>
<th>95% CI‡</th>
<th>Change in DBP (mmHg)</th>
<th>95% CI</th>
<th>Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abstainers§</td>
<td>538</td>
<td>0.0§</td>
<td>0.0§</td>
<td>0.0§</td>
<td>0.0§</td>
<td>0.00§</td>
</tr>
<tr>
<td>Drinkers (drinks/week)</td>
<td>432</td>
<td>1.9</td>
<td>-0.3 to 4.2</td>
<td>0.5</td>
<td>-1.3 to 2.3</td>
<td>1.02</td>
</tr>
<tr>
<td>&lt;1</td>
<td>142</td>
<td>1.4</td>
<td>-1.7 to 4.5</td>
<td>0.4</td>
<td>-2.1 to 2.9</td>
<td>1.04</td>
</tr>
<tr>
<td>≥1, &lt;7</td>
<td>195</td>
<td>2.3</td>
<td>-0.6 to 5.1</td>
<td>0.8</td>
<td>-1.4 to 3.1</td>
<td>1.01</td>
</tr>
<tr>
<td>≥7</td>
<td>95</td>
<td>2.3</td>
<td>-1.7 to 6.9</td>
<td>-0.3</td>
<td>-3.5 to 2.9</td>
<td>0.99</td>
</tr>
</tbody>
</table>

* Defined as SBP ≥160 mmHg, or DBP ≥95 mmHg, or currently taking antihypertensive medication.
† Adjusted for baseline age, body mass index (kg/m²), blood pressure, sex, and age by sex interaction.
‡ CI, confidence interval; RR, relative risk.
§ Referent category.

initiated drinking had higher baseline anger scores than abstainers.

Covariate-adjusted values for change in systolic and diastolic blood pressure, and hypertension incidence, as a function of changes in drinking status, are summarized in table 5. These analyses were adjusted for baseline age, BMI, blood pressure, sex, and an age by sex product term. Although baseline socioeconomic status, stress, and social ties were associated with change in selected categories of status (see tables 4 and 5), their addition to the model did not change the results and thus they were not included in the final models. Changes in drinking status were significantly associated with 5-year increases in systolic pressure. Compared with never drinkers, continued drinkers experienced a nearly 4 mmHg greater increase in systolic pressure, while subjects who initiated drinking experienced a 6 mmHg greater increase. Weaker trends in the same direction were observed for changes in diastolic pressure and hypertension incidence.

DISCUSSION

While frequency of alcohol consumption had a strong, positive association with mean blood pressure at baseline (1), in this follow-up study of the Pitt County cohort, baseline alcohol consumption did not predict 5-year hypertension incidence or changes in blood pressure. However, change in drinking status was more strongly associated with changes in blood pressure, especially systolic pressure. Relative to never drinkers, persons who initiated alcohol con-

---

* p < 0.10 for contrast with never drinkers (adjusted for age).
** p < 0.05 for contrast with never drinkers (adjusted for age).
BMI, body mass index (kg/m²).
Stated it was hard to pay for basics such as food and rent.
§ Measured in 1993 only.
TABLE 4. Baseline (1988) characteristics by women: the Pitt County Study

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Never (n = 372)</th>
<th>Discontinued (n = 111)</th>
<th>Continued (n = 139)</th>
<th>Initiated (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (&gt;40 years)</td>
<td>23.8</td>
<td>18.8</td>
<td>17.0</td>
<td>17.0</td>
</tr>
<tr>
<td>Overweight (BMI† &gt;27.8)</td>
<td>48.7</td>
<td>42.1</td>
<td>31.7**</td>
<td>31.7**</td>
</tr>
<tr>
<td>Smoker</td>
<td>14.4</td>
<td>39.3**</td>
<td>69.0**</td>
<td>51.1**</td>
</tr>
<tr>
<td>Socioeconomic status (SES)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low SES</td>
<td>31.5</td>
<td>27.9</td>
<td>41.8**</td>
<td>58.7**</td>
</tr>
<tr>
<td>Economic strain‡</td>
<td>11.2</td>
<td>13.6</td>
<td>22.3**</td>
<td>18.4</td>
</tr>
<tr>
<td>Education (years)</td>
<td>13.2</td>
<td>14.0</td>
<td>13.6</td>
<td>10.8**</td>
</tr>
<tr>
<td>Stressors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>10.0</td>
<td>15.5</td>
<td>20.0**</td>
<td>23.9**</td>
</tr>
<tr>
<td>High general stress</td>
<td>47.4</td>
<td>60.0*</td>
<td>51.6</td>
<td>54.6</td>
</tr>
<tr>
<td>High anger</td>
<td>45.3</td>
<td>50.8</td>
<td>52.5</td>
<td>80.5**</td>
</tr>
<tr>
<td>Social ties</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not married</td>
<td>49.0</td>
<td>57.4</td>
<td>69.0**</td>
<td>66.1</td>
</tr>
<tr>
<td>Church members§</td>
<td>80.5</td>
<td>72.0</td>
<td>52.0**</td>
<td>50.8**</td>
</tr>
<tr>
<td>Low emotional support</td>
<td>42.5</td>
<td>48.1</td>
<td>49.5</td>
<td>62.0*</td>
</tr>
<tr>
<td>Low instrumental support</td>
<td>40.3</td>
<td>51.0*</td>
<td>52.2**</td>
<td>57.9</td>
</tr>
</tbody>
</table>

* p < 0.10 for contrast with never drinkers (adjusted for age).
** p < 0.05 for contrast with never drinkers (adjusted for age).
† BMI body mass index (kg/m²).
‡ Stated it was hard to pay for basics such as food and rent.
§ Measured in 1993 only.

sumption had the greatest increase in blood pressure as well as the greatest risk for becoming hypertensive, while blood pressure changes for persons who discontinued drinking were similar to never drinkers. A stronger association for change in alcohol consumption than for baseline level of drinking has also been reported by other investigators (3, 12). These results are consistent with the observation of others that blood pressure elevations are an effect of one’s recent pattern of alcohol consumption, and that this effect is reversible (19, 20).

Continued and initiated drinking were associated with excess increases of 2–3 mmHg of diastolic pressure, and 4–6 mmHg of systolic pressure, despite generally low levels of alcohol consumption in both groups. Blood pressure increases of this magnitude within populations have important health implications, even when the increases occur within the normotensive range. As other investigators (21–23) have noted, the risks for stroke, coronary heart disease, and all-cause mortality increase monotonically with rising blood pressure at levels well below clinically defined hypertension. For example, it has been estimated that a reduction of 5.6 percent in coronary disease incidence in the US population as a whole could be achieved by a net reduction of 2 mmHg in diastolic pressure (22). Half of this effect would be observed among persons with diastolic pressure in the range 70–89 mmHg (22). Because alcohol consumption is associated with many other factors which in turn are associated with blood pressure, deriving causal statements, even in
prospective studies, is not a straightforward matter. Alcohol intake may affect blood pressure through multiple mechanisms with potential explanations ranging from direct adrenergic effects (24, 25) to more indirect effects involving a relation between psychosocial stress and alcohol use (26–29). Several investigators (8, 26, 27) have suggested an underlying role for psychosocial stress, i.e., the initiation or continuation of alcohol use in an otherwise low consumption population (30, 31) could be a response to stressful life conditions that tax the individual’s ability to cope. In these circumstances, alcohol may be used to try to alleviate psychosocial stress. This perspective conforms with findings in the current study which indicate a positive association between low socioeconomic status, unemployment, fewer social ties, and initiation and continuation of alcohol consumption. And, while these “social context” variables did not account for the observed association between change in drinking status and change in blood pressure, this does not exclude the possibility that the effects of alcohol consumption on blood pressure may partially mediate how background social and economic distress are related to blood pressure.

Other alternative explanations of the study findings are also possible. First, there may be potential bias due to loss to follow-up. For example, the reported findings could have been produced if subjects who continued to drink or who initiated drinking and who were lost to follow-up had smaller increases in blood pressure than their counterparts who participated in the 1993 reexamination. While it is not possible to directly assess this form of bias, differential participation among subjects who continued drinking or who initiated drinking on the basis of blood pressure change seems unlikely. Moreover, the relatively high level of participation at follow-up (85 percent) makes loss to follow-up bias an unlikely alternative explanation for the findings.

Second, the reported association could have been affected by misclassification of study participants on the change in drinking status variable. However, the decision to classify respondents as abstainers versus drinkers at both time points, rather than by change in the absolute number of drinks consumed, potentially minimized any misclassification due to the changes in questionnaire format and the inherent difficulty of quantifying “usual” consumption. Some underreporting of alcohol consumption in the Pitt County cohort might be expected, however, due to religious or other cultural proscriptions against alcohol use in many rural, southern communities (32, 33). Given the low levels of reported alcohol use by persons who continued or who initiated drinking, it is possible that the association with changes in blood pressure could be attributable to higher levels of alcohol consumption than respondents chose to report.

Third, because 4 percent of men (n = 14) and 7 percent of women (n = 46) were taking antihypertensive medication at follow-up, use of such medication could lead to misclassification on changes in blood pressure. To assess this possibility, the change in blood pressure analyses were repeated with the 60 treated hypertensives excluded. The results were unchanged, which is probably due to the relatively small number of individuals on medication.

Uncontrolled confounding is another possible explanation of the study findings. Age (years) and baseline BMI were positively associated with both abstention and blood pressure change in this study; however, because age and BMI were controlled in the analyses, the observed association is not attributable to their influence. While change in BMI was associated with blood pressure change, it was not associated with change in alcohol use, and hence did not alter results when it was included in the models. Additional adjustment for female hormone use and baseline smoking and exercise status also did not alter the reported associations. Finally, there was no evidence of confounding by sodium intake because several indicators of exposure to dietary sodium were unrelated to changes in drinking status.

To our knowledge, this is the only published longitudinal study that has investigated the association between change in drinking status (drinker vs. abstainer at two time points) and changes in blood pressure. These results indicate that in this low alcohol consumption population, those who continue to drink or who initiate drinking are at increased risk of elevated blood pressure compared with abstainers. Similar research in other populations, especially African American populations, is needed to determine the generalizability of the reported findings to US communities outside the rural South. One major objective of future research could be to clarify more fully whether a low level of alcohol consumption, in and of itself, influences blood pressure or if its apparent risk factor status is secondary to its association with social and economic distress. Additional research on this topic in African American populations may be especially important given the heightened exposure of those populations to stressful socioeconomic conditions and their well-documented elevated risk for hypertension.

REFERENCES


Am J Epidemiol  Vol. 146, No. 9, 1997
Alcohol Consumption and Blood Pressure in African Americans