J Chron Dis Vol. 40, No. 3, pp. 229–236, 1987
Printed in Great Britain.

0021-9681/87 $3.00 + 0.00

ALCOHOL CONSUMPTION AND MORTALITY IN ALAMEDA COUNTY

TERRY C. CAMACHO, GEORGE A. KAPLAN and RICHARD D. COHEN

Human Population Laboratory, California Department of Health Services, 2151 Berkeley Way, Annex 2, Room 211, Berkeley, CA 94704-9980, U.S.A.

(*Received in revised form* 10 *April* 1986)

Abstract—The association between level of alcohol consumption and 15-year mortality, focusing particularly on the possible protective effect of light drinking compared to abstention, was studied in a representative population sample of 6928 residents of Alameda County, California. Because abstainers differ from light, moderate and heavy drinkers on a number of demographic, physical, and psychosocial characteristics, the role of these as confounders of the alcohol/mortality association was examined. Using multiple logistic models, the mortality experience of abstainers, moderate drinkers, heavy drinkers and very heavy drinkers was compared with that of light drinkers. Among men only, very heavy drinkers were at significantly greater risk of death from all causes than were light drinkers (OR = 2.5, $p < 0.01$). Neither abstainers nor other drinkers were at significantly higher risk of death from ischemic heart disease than were light drinkers. This pattern of results persisted with adjustment for 11 covariates of alcohol consumption in addition to age.

Alcohol Mortality Ischemic heart disease

INTRODUCTION

Considerable evidence has recently accumulated to challenge the traditional view of the relationship between alcohol consumption and health. Evidence from a number of studies now suggests that moderate consumption of alcohol may be associated with lower morbidity and mortality than either abstention or heavy drinking. Discovery of this U-shaped or J-shaped association in which moderate drinkers are at lowest risk of death, especially from coronary heart disease, has led some investigators to propose that alcohol in moderate amounts may actually exert a protective effect on health [1–11].

A careful examination of the literature reveals, however, a marked lack of consistency of results among studies. At times, especially in studies dealing with mortality from ischemic heart disease, the J-shaped curve gives way to a more nearly inverse linear association between alcohol consumption and health outcomes [9, 12–14]. Of particular interest is the fact that the alcohol/health association in a given sample often varies considerably among various subgroups defined by age, sex, smoking habits, socioeconomic status, and even type of alcoholic beverage consumed. Some examples of such variations are given in Table 1.

In the present study, we examine the association between alcohol consumption and 15-year mortality from all causes and from ischemic heart disease using data collected by the Human Population Laboratory (HPL) in its prospective study of Alameda County, California, residents. We pay particular attention to the differences between abstainers and light or moderate drinkers, hoping to shed some light on the question of whether alcohol in small amounts is actually protective of health for all causes of death and, specifically, for ischemic heart disease mortality. We hope to help clarify the currently confusing picture of the relationship between alcohol consumption and mortality in two particular ways: (1) A number of studies in this area have been limited to men only or have excluded the unemployed, poor or minority respondents. Re-

Table 1. Variation in conditions of the alcohol consumption/health associations in selected studies

Investigator	Sample	Outcome	Association with alcohol consumption	Conditions of significant association
Case-control studies				
Rosenberg *et al.*, 1981 [11]	513 female myocardial infarction (MI) patients aged 30–49 and 918 hospital controls	MI	Nondrinkers at increased risk over drinkers	For wine and total alcohol, not for beer or liquor
Salonen *et al.*, 1983 [14]	4063 Finnish males, aged 30–59	7-year MI incidence, coronary heart disease (CHD) mortality	Inverse	For spirits, but not beer (MI); for age >50, but not age <50 (CHD mortality)
Prospective studies				
Klatsky *et al.*, 1981 [7]	8060 Kaiser health plan members	10-year all-cause and CHD mortality	U-shaped	For age <50, not age >50 (all-cause); for ever-smokers only (CHD)
Cullen *et al.*, 1982 [4]	2209 males and females aged 40+	13-year mortality	Nondrinkers at increased risk over drinkers	For nonsmokers only
Kozarevic *et al.*, 1982 [9]	11,121 men aged 40+	MI, non-sudden CHD mortality	Inverse	For urban residents, not rural
Gordon and Kannel, 1983 [12]	Framingham adults aged 29–62	24-year CHD incidence	Inverse	For male nonsmokers, all females, not for male smokers
Kittner *et al.*, 1983 [13]	9150 Puerto Rican males aged 35–79	Non-sudden CHD mortality	Inverse	For age <60, not age >60; for above-median income, not for below-median income

strictions of this sort limit the generalizability of any findings to more broadly defined populations. The Alameda County cohort is a large representative sample of the adult population of a metropolitan area and includes persons of many backgrounds and life circumstances. (2) It is likely that abstainers, moderate drinkers and heavy drinkers are characterized by substantial differences along demographic, health and psychosocial dimensions, and these may contribute to the variance in study findings; the HPL data set includes ample health, behavioral, social and demographic data collected at two points in time and thus provides an opportunity to investigate the role of these potential confounders of the alcohol/mortality relationship.

DATA AND METHODS

The sample

The HPL panel was drawn in 1965 from the noninstitutionalized adult population of Alameda County, California, using area probability sampling methods. (See Hochstim [15] for a more detailed description of sampling methods.) Of the more than 8000 designated respondents, completed questionnaires were received from 6928, or 86%. The data collection utilized a self-administered questionnaire asking for detailed information regarding current health problems and functional disabilities; daily living habits; social activities and relationships; attitudes and feelings about self, others and the world; and demographic information. This analysis is restricted to those respondents who were 35 years of age and older in 1965 ($N = 4590$). Of these, 3128 were successfully reinterviewed in 1974, providing us with information at two times for a substantial proportion of the original sample.

Mortality follow-up

Mortality data were obtained by way of computerized death clearance procedures in which HPL files were matched with California State death records. (This procedure is described in greater detail in Belloc and Arellano [16] and Arellano *et al.* [17].) The computer search was supplemented with out-of-state death tracing as part of the 1974 and 1983 follow-ups of this cohort. To date, we have identified 1145 deaths between 1965 and 1980 in our original cohort of 6928, or about 16% over 15 years; in the older sample used here, there are 1093 deaths, or 24%.

Persons not known to be dead were consid-

ered alive for purposes of the analyses presented here. This convention is based on the fact that at the 1974 follow-up, all but 4% of those not identified as dead were located alive; at the 1983 follow-up, the equivalent figure was 6%. Ascertainment of vital status is even higher for the older portion of the sample, and in fact, only 114 persons (2.4%) of the present study sample were not positively identified as dead or located alive in 1974. Nevertheless, to check for bias due to the assumption of vital status for this small group, the major analyses of this report were replicated using only those whose vital status in 1974 was certain; the results did not differ substantially from those presented here.

Cause of death was obtained from death certificates, using codes determined by the Vital Statistics Branch of the California Department of Health Services, using the 7th, 8th and 9th revisions of the **International Classification of Diseases** [18] for the periods 1965–67, 1968–78 and 1979–80, respectively. Code equivalencies were established for the broad cause categories used here and, as cited in this report, refer to the 8th revision.

Measuring alcohol consumption

HPL respondents were asked how many times a week they drink each of three types of alcoholic beverage (beer, wine and "spirits") and, for each kind that they ever drink, how many drinks they usually take at a sitting. The alcohol measure used in this analysis is computed by multiplying frequency by number of drinks for each type of alcohol and summing the three scores to arrive at a total monthly consumption. Possible scores ranged from 0 to 270+ drinks per month.

We note here that there are some deficiencies in this measure. First, it is somewhat imprecise in terms of amount of ethanol consumed since subjects were asked only "how many drinks" of each type they consumed rather than how many ounces of alcohol. Second, questions regarding amount and frequency of drinking had precoded response categories, which imposed an artificial upper limit of reported consumption for each type of beverage (five or more drinks per sitting, two or more sittings per week). These limitations impose restraints on our ability to make fine distinctions among alcohol consumption levels and to assess the impact of extremely heavy drinking; however, they do not interfere with our primary interest, the investigation of the differences between abstainers and light or moderate drinkers. A more serious limitation, from our point of view, is the fact that the alcohol consumption measure used here describes the individual's current practice without reference to any changes this may represent from earlier habits. This problem, which is found in most alcohol studies, does have implications for the assessment of the postulated "abstainer effect"; we cannot distinguish lifelong abstainers from persons who previously drank. To the extent that the nondrinking group includes former drinkers, it is difficult to assess the long-term risks or benefits of abstention. We will attempt to assess the extent of this kind of misclassification problem in our data set using our follow-up data. However, results of the analyses presented here must be interpreted with this particular limitation in mind.

In part to accommodate the limitations as outlined above, as well as to make our findings comparable to other published results, we will use grouped alcohol scores in this analysis as follows: abstainers, light drinkers (1–30 drinks per month), moderate drinkers (31–60 drinks per month), heavy drinkers (61–90 drinks per month), and very heavy drinkers (91+ drinks per month). For women, the top two groups are combined since there are too few women respondents in the top category to analyze them separately.

Covariates of alcohol consumption

Previous research has identified a number of health-relevant physical, demographic, behavioral, and psychosocial variables in the HPL data set which, if associated also with alcohol consumption, could confound the association between alcohol and mortality [19–24]. The first step in our analysis, therefore, was to examine the univariate cross-sectional relationships between alcohol consumption and these potential confounders. Twelve items were found to be significantly ($p < 0.05$) associated with our measure of alcohol consumption, as follows: age, sex, race, an index of overall physical health, perceived subjective health, income, education, smoking habits, exercise, an index of social contacts, depression, and personal uncertainty. Table 2 shows the proportion of each alcohol consumption group scoring in the high-risk category of each of these variables, as well as the crude all-cause and ischemic heart disease mortality rates for each alcohol consumption group.

Table 2. Covariates of alcohol consumption

Covariates	Drinks per month				
	None ($N = 1170$)	1–30 ($N = 2467$)	31–60 ($N = 585$)	61–90 ($N = 212$)	91+ ($N = 144$)
Percentage 65 years and older	32	17	11	10	7
Percentage male	32	44	62	71	79
Percentage black	20	11	8	11	10
Percentage disabled[a]	27	19	16	10	17
Percentage rate own health "fair" or poor"	30	21	17	13	21
Percentage less than adequate income level[b]	43	24	19	14	17
Percentage less than 12 years schooling	60	41	33	39	39
Percentage never smoked	63	38	23	19	16
Percentage relatively inactive[c]	57	40	33	30	35
Percentage not married	35	23	17	18	23
Percentage with no organization memberships	46	32	27	28	29
Percentage depressed[d]	19	15	14	11	12
Percentage "uncertain"[e]	28	28	25	33	44
Percentage died (all causes) 1965–74	32	21	19	21	28
Percentage died (ischemic heart disease only) 1965–74	13	8	8	7	7

[a]Based on Physical Health Spectrum Score, Berkman and Breslow [20].
[b]Total household income adjusted for family size.
[c]Physical Activity Score based on frequency of participation in various activities. See Berkman and Breslow [20].
[d]18-item index, Roberts and O'Keefe [28].
[e]"Personal Uncertainty" Score, Berkman and Breslow [20].

Multivariate analysis

As the first step in analyzing the alcohol consumption/mortality relationship, we employ a multiple logistic model (BMDP PLR), with adjustment first for age alone and then for the covariates identified above. For this portion of the analysis, the five alcohol categories are represented by four "dummy" variables, with light drinking (1–30 drinks per month) as the reference category so that we can compare both nondrinkers and heavy drinkers to the more moderate group and thus test for the postulated curvilinear relationship between alcohol consumption and mortality.

The results of these first analyses are presented in Tables 3 and 4. The upper half of Table 3 shows the analyses for all-cause mortality separately for men and women, with alcohol consumption and age in the model. The only significant association is that for very heavy compared to light drinking for men (OR = 2.5, $p < 0.01$). Although all other odds ratios are greater than one (suggesting a J-shaped curve), the confidence intervals in all cases include 1.00.

The lower half of Table 3 presents the same analyses for mortality from ischemic heart disease. No significant patterns of increased risk are found for men or women either for heavy drinking or abstention.

Table 3. Logistic analyses of alcohol consumption and two mortality outcomes with adjustment for age, for men and women

	Men		Women	
	Odds ratio	95% Confidence interval	Odds ratio	95% Confidence interval
All-cause mortality				
Abstain	1.3	(0.9–1.8)	1.2	(0.9–1.5)
31–60 drinks	1.1	(0.7–1.5)	1.2	(0.8–1.9)
61–90 drinks	1.5	(0.9–2.4)	1.5[a]	(0.8–2.9)
91+ drinks	2.5[b]	(1.5–4.1)		
Ischemic heart disease mortality				
Abstain	1.3	(0.8–2.0)	1.1	(0.8–1.6)
31–60 drinks	1.3	(0.8–2.1)	1.0	(0.5–2.2)
61–90 drinks	1.5	(0.8–2.9)	1.0[a]	(0.2–4.5)
91+ drinks	1.5	(0.6–2.4)		

[a]Because of the small number of women reporting heavy alcohol consumption, the two highest categories (61–90 drinks, 91+ drinks) are combined here.
[b]$p < 0.01$.

Table 4. Logistic analyses of alcohol consumption and two mortality outcomes with adjustment for 13 alcohol covariates, for men and women

	Men		Women	
	Odds ratio	95% Confidence interval	Odds ratio	95% Confidence interval
All-cause mortality				
Abstain	1.2	(0.9–1.8)	1.1	(0.9–1.5)
31–60 drinks	1.0	(0.7–1.5)	1.2	(0.7–1.8)
61–90 drinks	1.5	(0.9–2.5)	1.5[a]	(0.8–2.5)
91+ drinks	2.3	(1.4–3.9)[b]		
Ischemic heart disease mortality				
Abstain	1.2	(0.7–2.0)	1.1	(0.7–1.7)
31–60 drinks	1.2	(0.7–2.6)	0.9	(0.3–2.0)
61–90 drinks	1.6	(0.8–3.2)	0.9[a]	(0.2–4.0)
91+ drinks	1.5	(0.6–3.5)		

[a]Because of the small number of women reporting heavy alcohol consumption, the two highest categories (61–90 drinks, 91+ drinks) are combined here.
[b]$p < 0.01$.

Thus, with adjustment for age alone, we do not find a statistically significant association of alcohol consumption and either all-cause or ischemic heart disease mortality that could be described by a J- or U-shaped curve. The only significant finding is that of a positive association between all-cause mortality and very heavy vs light drinking for men only. We find no significant association between level of alcohol consumption and all-cause mortality for women and no association between alcohol consumption and ischemic heart disease mortality for either sex, although in most cases, the pattern of association between alcohol and mortality appears to suggest a curvilinear pattern.

Table 4 shows the results of logistic analyses which include adjustment for the covariates identified above as potential confounders of the relationship between alcohol consumption and mortality. Although the figures are not shown here, it is important to note that each of these covariates increases the ability of the model to predict mortality; they are associated with the outcomes of interest and with alcohol consumption. However, adjustment for them does not change the overall pattern of the alcohol/mortality relationship observed in Table 3. As before, the only significant association is that between very heavy vs light drinking and all-cause mortality among men (OR = 2.3, $p < 0.01$). The odds ratios and confidence intervals associated with the various levels of alcohol consumption in this model are nearly identical to those in the previous model (Table 3) in which the only adjustment variable is age. The postulated confounding of the relationship between alcohol consumption and mortality by related physical, psychological, and social variables was not observed.

In Table 5, we present the results of an alternative and more direct testing of the suggested but nonsignificant curvilinear relationship of alcohol consumption and mortality observed in Tables 3 and 4. We use a logistic model containing the covariates described above but enter alcohol as a single linear term rather than $N-1$ dummy variables as above (Appendix 1). The presence of a curvilinear association is tested for directly by inclusion of a quadratic alcohol term in the model. If the alcohol consumption/mortality relationship is defined by a U- or J-shaped curve, the coefficient (β) for the linear alcohol term in the model should be significantly less than zero, and the quadratic term should be significantly greater than zero (Appendix 2).

Preliminary logistic analysis using only the linear term with adjustment for the covariates yielded results identical to those described in previous analyses. When the quadratic term is added (Table 5), there is no change in these results. A significant alcohol/mortality association is found only for male all-cause mortality. In this case, the quadratic term is positive and significant, suggesting that risk increases nonlinearly with alcohol consumption. However, since the linear term is not significantly different from zero, we cannot say whether the minimum risk occurs for abstainers or for light drinkers. In the other gender-outcome groups, the values of the coefficients are consistent with a J- or U-shaped relationship, but none of the coefficients is significantly different from zero. Consequently, we cannot conclude that a J- or

Table 5. Logistic regression analyses with linear and quadratic alcohol consumption terms and adjustment for 13 alcohol covariates

	Alcohol consumption			
	Linear term		Quadratic term	
	β	β/ Standard error	β	β/ Standard error
All-cause mortality				
Men	−0.010	−1.41	0.00020	2.33[a]
Women	−0.007	−0.94	0.00010	1.61
Ischemic heart disease mortality				
Men	−0.001	−0.06	0.00004	0.44
Women	−0.020	−1.55	0.00020	1.42

[a]$p < 0.01$.

Table 6. Logistic analysis of all-cause and ischemic heart disease mortality for ex-drinkers compared to stable abstainers, adjusting for 13 alcohol covariates (odds ratios and confidence intervals for variable "quitter")

	Men		Women	
	Odds ratio	95% Confidence interval	Odds ratio	95% Confidence interval
All-cause mortality	0.7	(0.3-1.4)	1.2	(0.6-2.3)
Mortality from ischemic heart disease	0.4	(0.1-1.4)	1.8	(0.7-4.3)

U-shaped relationship is a better description of the data than a linear association.

Confusion of exdrinkers with lifelong drinkers is a possible source of error. Because many of the people who stop drinking may do so as a result of declining health, their presence in the "abstainer" category might account for any suggestion, although nonsignificant, of excess risk for abstainers compared to light drinkers. Using our 1965–74 panel sample and mortality follow-up from 1974 through 1982, we are able to address this issue by limiting our analysis to those respondents who reported no drinking at the time of the 1974 follow-up study and comparing the subsequent mortality experience of those for whom this status represented a change from 1965 (the "quitters") with that of respondents who reported abstention at both points in time. Table 6 shows the results of logistic analyses of the 1974 abstainers, adjusting for age and the other alcohol covariates, plus a term "quitter," which distinguishes those for whom abstention in 1974 represented a change from drinking habits reported 9 years earlier (coded 1) from those who reported abstention at both points in time (coded 0). The relative risk associated with this term represents the risk of exdrinkers relative to the more stable abstainers.

There are no statistically significant differences between quitters and stable abstainers as measured here for either sex for either of the two outcomes. The patterns of excess risk are different for men and women, however. Among men, those whose abstention represents a change since baseline are at lower risk of death, especially from ischemic heart disease, than those who report nondrinking at both times; for women, the opposite is true—the "quitters" show a higher risk than the stable abstainers. The same patterns obtain if we compare stable abstainers with quitters who reported drinking at least 31 drinks a month in 1965. Examination of the previously heavy drinking (61+ drinks per month) quitters in comparison with stable abstainers is not possible in this data set because the number of heavy drinkers who report abstention at follow-up is extremely small.

These results are only partially supportive of our hypothesis regarding former drinkers and are in some contradiction to the findings of others who have shown exdrinkers to be at very high risk of mortality [12, 25–27]. However, we must be extremely cautious in the interpretation of our results here since the assessment of stable or unstable drinking habits is based only on data from two points in time (some of the "stable" abstainers in our sample may have been drinkers prior to 1965 and may be more like our "quitters" in many ways) and because the number of deaths represented in this analytic group is not large.

DISCUSSION

Using a number of methodological approaches, we have attempted to test the hypothesis that the relationship between alcohol consumption and mortality is represented by a U- or J-shaped curve in which both very heavy drinking and abstention carry greater risks than light or moderate drinking. The only portion of this hypothesis which is confirmed by the analyses is the positive association of heavy drinking with all-cause mortality for men. No such significant relationship is shown for women or for either gender when the outcome variable is ischemic heart disease mortality. No significant increase in risk is found for abstainers of either sex for either of the mortality outcomes. We have identified a number of covariates of alcohol consumption, items which are related to mortality in this data set and which might, therefore, be expected to confound the

alcohol/mortality relationship. Surprisingly, we find that adjustment for this set of 12 variables has very little impact on the results beyond adjustment for age alone. Attempts to fit a quadratic model to our data also fail to support the existence of a U- or J-shaped curve. Finally, abstainers known to be exdrinkers do not differ significantly from stable abstainers. In sum, neither the general hypothesis regarding the U-shaped curve nor the more specific hypothesis regarding the protective effects of small amounts of alcohol for all-cause or ischemic heart disease mortality are supported; the hypothesis relating heavy drinking to excess mortality is supported only in one instance.

While we thus find no significant overall relationship of alcohol to risk of death, we are nevertheless left with the persistent suggestion of a curvilinear relationship. Results for all-cause mortality for men and women and ischemic heart disease mortality for men present this pattern, and the lower limits of the confidence intervals are often rather close to 1.00.

The results presented here also suggest a possible interaction of sex with the alcohol/mortality relationship. Odds ratios tend to be somewhat stronger for men than for women, especially in terms of the effects of moderate or heavy drinking (perhaps because fewer women are in these categories). And, in some cases, the associations observed for men and women are in opposite directions.

If this subject is to be investigated further, there is a need for more careful measurement of alcohol consumption and drinking history. In addition, analysis of interactions of alcohol consumption with age, sex, other health habits, socioeconomic status and living environment may prove fruitful in addressing some of the conflicting results. In the meanwhile, it is important that the ambiguity of findings to date be recognized in order that we may avoid making premature and unwarranted conclusions regarding the potential effects of moderate intake of alcohol. It is also worth noting that, even in those studies where significant abstention/mortality associations have been demonstrated, the size of the increased risk is rather small compared to other known risk factors and the other known risks associated with alcohol intake. Therefore, it would appear prudent, from a public health perspective, to reserve judgment concerning the protective effects of moderate alcohol consumption.

REFERENCES

1. Barboriak JJ, Anderson AJ, Hoffman RG: Interrelationship between coronary artery occlusion, high-density lipoprotein cholesterol, and alcohol intake. **J Lab Clin Med** 94: 348–353, 1979
2. Barboriak JJ, Rimm AA, Anderson AJ, Schmidhoffer M, Tristani FE: Coronary artery occlusion and alcohol intake. **Br Heart J** 39: 289–293, 1977
3. Blackwelder WC, Yano K, Rhoads GG, Kagan A, Gordon T, Palesch Y: Alcohol and mortality: the Honolulu Heart Study. **Am J Med** 68: 164–169, 1980
4. Cullen K, Stenhouse NS, Wearne KL: Alcohol and mortality in the Busselton Study. **Int J Epidemiol** 11: 67–70, 1982
5. Hennekens CH, Rosner B, Cole DS: Daily alcohol consumption and fatal coronary heart disease. **Am J Epidemiol** 107: 196–200, 1978
6. Kagan A, Yano K, Rhoads GG, McGee DL: Alcohol and cardiovascular disease: the Hawaiian experience. **Circulation** 64 (Suppl. III): 27–31, 1981
7. Klatsky AL, Friedman GD, Siegelaub AB: Alcohol and mortality: a ten-year Kaiser-Permanente experience. **Ann Int Med** 95: 139–145, 1981
8. Klatsky AL, Friedman GD, Siegelaub AB: Alcohol use and cardiovascular disease: the Kaiser-Permanente experience. **Circulation** 64 (Suppl. III): 32–41, 1981
9. Kozarevic D, Demirovic J, Gordon T, Kaelber CT, McGee D, Zukel WJ: Drinking habits and coronary heart disease: the Yugoslavia Cardiovascular Disease Study. **Am J Epidemiol** 116: 748–758, 1982
10. Marmot MG, Rose G, Shipley MJ, Thomas BJ: Alcohol and mortality: a U-shaped curve. **Lancet** 1: 580–583, 1981
11. Rosenberg L, Slone D, Shapiro S, Kaufman DW, Miettinen OS, Stolley PD: Alcohol beverages and myocardial infarction in young women. **Am J Public Health** 71: 82–85, 1981
12. Gordon T, Kannel W: Drinking habits and cardiovascular disease: the Framingham Study. **Am Heart J** 105: 667–773, 1983
13. Kittner SJ, Garcia-Palmieri MR, Costas R, Cruz-Vidal M, Abbot RD, Havlik RJ: Alcohol and coronary heart disease in Puerto Rico. **Am J Epidemiol** 117: 538–550, 1983
14. Salonen JT, Puska P, Nissinen A: Intake of spirits and beer and risk of myocardial infarction and death—a longitudinal study in Eastern Finland. **J Chron Dis** 36: 533–543, 1983
15. Hochstim JR: Health and ways of living—the Alameda County, California, population laboratory. In **The Community as an Epidemiologic Laboratory,** Kessler II, Levin ML (Eds). Baltimore: Johns Hopkins University Press, 1970. pp. 149–176
16. Belloc NB, Arellano MG: Computer record linkage on a survey population. **Health Serv Rep** 88: 344–350, 1973
17. Arellano MG, Peterson GR, Petitti DB, Smith RE: The California automated mortality linkage system (CAMLIS). **Am J Public Health** 74: 1324–1330, 1984
18. U.S. Dept. of Health, Education and Welfare. **International Classification of Diseases,** 8th revision. Public Health Service Pub. No. 1693, Vol. 2. Washington, D.C.: U.S. Government Printing Office, 1968
19. Belloc NB: Relationship of health practices and mortality. **Prev Med** 2: 67–81, 1973
20. Berkman LF, Breslow L (Eds): **Health and Ways of Living: The Alameda County Study.** New York: Oxford University Press, 1983
21. Wiley JA, Camacho TC: Life-style and future health: evidence from the Alameda County Study. **Prev Med** 9: 1–21, 1980

22. Kaplan GA, Camacho T: Perceived health and mortality: a nine-year follow-up of the Human Population Laboratory cohort. **Am J Epidemiol** 117: 292–304, 1983
23. Rose G, Marmot MG: Social class and coronary heart disease. **Br Heart J** 45: 13–19, 1981
24. Wingard DL, Berkman LF, Brand RJ: A multivariate analysis of health-related practices: a nine-year mortality follow-up of the Alameda County Study. **Am J Epidemiol** 116: 765–775, 1982
25. Day N: Alcohol and mortality. In **Third Special Report to Congress on Alcohol and Health.** Washington, D.C.: National Institute on Alcohol Abuse and Alcoholism, 1977
26. Day N: **Alcohol and Mortality: Separating the Drink from the Drinker.** Thesis, University of California, Berkeley, 1979
27. Dyer AR, Stamler J, Paul O, Berkson DM, Shekelle RB, Lepper MH, McKean H, Lindberg HA, Garside D, Tokich T: Alcohol, cardiovascular risk factors and mortality: the Chicago experience. **Circulation** 64 (Suppl. III): 20–27, 1981
28. Roberts RE, O'Keefe SJ: Sex differences in depression reexamined. **J Health Soc Behav** 22: 394–400, 1981

APPENDIX 1

Given the range and dispersion of scores in the original linear alcohol consumption measure (especially at the upper end of that score), we chose to use a grouped version of the score here, assigning all members of a given group the median score for that category as follows:

Category	*Median*
1–15 drinks per month	9
16–30 drinks per month	23
31–60 drinks per month	45
61–90 drinks per month	72
91+ drinks per month	109

APPENDIX 2

It is possible to fit a logistic model in which both the linear and quadratic terms are significant but which represents a different curve than the one postulated for the alcohol/mortality risk relationship. Thus a risk curve in which the linear term is equal to zero attains its lowest point when alcohol equals zero, and risk increases as alcohol consumption increases. Similarly, if the linear term is greater than zero, the lowest point on the risk curve is moved to the left of the vertical axis, and no part of the relevant segment of the curve exhibits a downward slope. If the quadratic term is less than zero, the risk curve described has an inverted U-shape. Thus, in addition to attaining statistical significance, the linear and quadratic coefficients must have appropriate values to support the hypothesis that moderate drinking is beneficial.