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Trends in Alcohol Consumption and Associated Illnesses

Some Effects of Economic Changes

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The relation of short and long term trends in alcohol consumption to cirrhosis mortality and to fluctuations in the national economy is discussed.

Introduction

Relations between a number of sociodemographic factors and patterns of alcohol consumption are now well established. Age[1, 2] and sex,[3-5], socioeconomic level,[3] ethnic background, education, occupation, and degree of urbanization[6] all show substantial statistical correlation with the extent of drinking. At the same time it now appears to be generally accepted that the extent of alcohol-related problems, especially cirrhosis of the liver, is directly correlated with the per capita level of alcohol consumption.[6]

An apparent contradiction, however, is that while overall alcohol consumption may be directly related to a given sociodemographic factor, *heavy* drinking or alcohol-related problems may be inversely related or unrelated to the same factor. Among the more outstanding sociodemographic variables apparently showing internally contradictory relations to alcohol consumption and attendant problems are age, socioeconomic level and ethnic background.

Terris examined the trends in alcohol consumption in the United States, Canada, and England and Wales in relation to the trends in mortality attributed to cirrhosis of the liver for these areas.[7] It was found that the trends in alcohol consumption and cirrhosis of the liver mortality rates showed remarkable covariation over time. In this analysis, Terris supported the position of Jolliffe and Jellinek,[8] who drew a similar conclusion to that of Terris after noting (1) rapid decline in the cirrhosis death rate corresponding to the state and war time prohibition of 1916–1920, (2) a plateau in the cirrhosis death rate during the prohibition based on the Eighteenth Amendment, and (3) a gradual increase during 1933–1940 after repeal of the national prohibition.

The data presented by Terris appeared to obviate a major criticism [9] of the Jolliffe-Jellinek position that deaths from a chronic disease could not be expected to respond sharply to variation of an etiological agent. Terris argued that "apparently the cirrhosis death rate responds fairly rapidly to changes in availability of alcoholic beverages. This phenomenon is consistent with the clinical course of the disease . . . . In many cases the cirrhotic process can be halted and decompensation prevented by avoiding further use of alcohol. Conversely, resumption of heavy alcohol use after a period of abstinence can decompensate a previously injured liver in a relatively short period of time."[7]

From graphs presented in Terris' article it appeared that an increase in cirrhosis mortality ordinarily occurred within 1 year of an increase in alcohol consumption (i.e., Terris' data are annual and no lag is shown). Terris' explanation is that mortality could occur rapidly as a result of increase in alcohol consumption, especially when followed by a period of abstinence. This explanation is in accordance with the data presented, which showed long term trends as well as fluctuations in alcohol consumption coincident with trends and fluctuations in cirrhosis mortality.

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Major Research Issues

The present paper attempts to examine in detail the apparent discrepancy between (1) the nonexistent or inverse relations between average alcohol consumption and heavy drinking found for socioeconomic, ethnic, and age categories, and (2) the consistent findings, *over time*, in which average alcohol consumption is related to cirrhosis mortality rates. From the standpoint of trend analysis, the following major issues are raised:

- Are the effects of long term trends in alcohol consumption the same as those of short term fluctuations on alcohol-related illness?
- Why are there both substantial trend phenomena and very sharp short term fluctuations of alcohol consumption in relation to cirrhosis mortality?
- Are the effects of variation in alcohol consumption or cirrhosis mortality the same on other indicators of heavy consumption of alcohol—e.g., mental hospitalization with alcohol-related mental disorder, arrest for "drunkenness" or for driving an automobile while intoxicated?
- Are the same factors which are associated with increased alcohol consumption also operative for indicators of the outcome of heavy drinking?

Effects of Long and Short Term Trends in Alcohol Consumption on Cirrhosis Mortality

A variety of factors make it crucial to distinguish between the long and short term factors which may be involved in alcohol consumption (and, therefore, in cirrhosis mortality). Long term trends of increase in alcohol consumption may reflect, for example: (1) increasing general affluence on the part of the general population, also involving greater leisure time and "discretionary" income; (2) changed immigration patterns either deemphasizing older, and more stable, drinking habits or emphasizing the leisure time use of alcoholic beverages—especially distilled spirits; and (3) long term vogues in the use of alcoholic beverages as socializing media or as deliberate intoxicants.

In contrast, the shorter cyclical fluctuations in per capita alcohol consumption may reflect: (1) changes in the psychological state of the population (or that portion which tends to use alcohol frequently); (2) fluctuations in the ability of the population to purchase alcoholic beverages in general, or specific beverages in comparison with others; or (3) fluctuations of demand and satiation which have the effect of placing certain foods, beverages, or other consumer items on a fashion-related cyclical pattern of consumption.

Methods

Long Term Trends

First, long term linear or nonlinear trends in the alcohol consumption and cirrhosis mortality data were estimated (by least-squares methods). The secular trends were then algebraically subtracted from the raw data (Table 1) and the residuals (or short and intermediate range trends) were investigated graphically and with regression analysis. For both series of data, the trends were estimated by a procedure which determines the best-fitting mathematical equation, among several types. These equations describe the following models: (1) linear ($Y = a \pm bx$), (2) logarithmic ($Y = a \pm b \log X$, $\log Y = a \pm bx$, $\log X$), (3) reciprocal ($Y = 1/a \pm bx$), and logistic ($Y = a/1 \pm bx^{-ct}$). The curves representing each of these equations are fitted, by least-squares techniques, to the alcohol consumption and cirrhosis mortality rates. The mathematical model of the long term trend which best fits the raw rates, as judged by the product-moment correlation coefficient, is used to describe the long term trends (Table 1). So estimated, the long term trends are then algebraically subtracted from the raw rates, and what remain are the intermediate sized and short trends in these data.

By the use of multiple regression procedures, the effect on cirrhosis mortality rates of per capita alcohol consumption, lagged from 0 to 5 years, was estimated. (Multiple regression treats the alcohol consumption indicator as six independent variables in a distributed-time-lag analysis.[10] In addition, regression (zero-order) was performed at the lag at which the relationship between economic change and admission rates was most stable (transforming the data, when necessary, to control for effects of autocorrelated residuals[11, 12]).

Three- to Eleven-Year Trends

The second type of analysis compared 3- to 11-year trends in alcohol consumption and cirrhosis mortality, in fluctuations of different sizes, categorized in percentage change occurring at intervals of 3, 5, 7, 9, and 11 years. This is a "moving difference" method which allows observation, over time, of annual, 3-year, 5-year, and greater percentage changes. Thus, to obtain moving annual percentage changes, the change from each year to the next is estimated as the absolute difference between the two, divided by the value for the first year (expressed in percentages).[13] Similarly, 3-year moving changes are calculated as the percentage difference between the rate (of alcohol consumption or cirrhosis mortality) at *each* year and that of 3 years later, on a continuous basis for the entire series. In this way, the values representing every set of 4 years are expressed as the differences between the first and fourth years in 3-year percentage changes.

These estimates of moving percentage changes include some important features of moving averages including the aggregation of several years of data (3 to 11 in this study) in order to control for the effects of "random" fluctuation within any year or very short period. Such estimates also enable assessment of the trends encompassed in intervals of different sizes. In general, the larger the time interval involved, the greater is the absolute level, or percentage, of change that tends to occur. Most important, for this study, this type of data transformation allows more precise inquiry into temporal effects of the expected relationships.

TABLE 1—Equations Describing Secular Trends in Cirrhosis of Liver Mortality Rates, Alcohol Consumption Rates, Indicators of Intoxication, and National Economic Indicators

Number	Series	Equation Describing Secular Trend
	Apparent per capita alcohol consumption (U.S. gallons)	
1	Avg monthly rate per annum, 1935–1967	$Y = 6510.365 + 1499.210 \log X$
2	Total absolute alcohol, 1934–1970	$Y = 100.025 + 36.314 \log X$
3	Distilled spirits, 1934–1970	$Y = 111.0719 + 3.2935 X$
4	Distilled spirits, absolute alcohol, 1934–1970	$Y = 52.6486 + 1.4353 X$
5	Wine, 1934–1970	$Y = 52.3964 + 3.5596 X$
6	Wine, absolute alcohol, 1934–1970	$Y = 5.8846 + 5.2680 \log X$
7	Beer, 1934–1970	$Y = 1388.134 + 291.270 \log X$
8	Beer, absolute alcohol, 1934–1970	$Y = 62.664 + 13.002 \log X$
	Cirrhosis of liver mortality rates, 1923–1968	
9	White males	$Y = 7812.941 + 206.526 X$
10	White females	$Y = 43739.176 + 850.736 X$
11	Nonwhite males	$Y = 1.0/(0.00016 - 0.0000019 X)$
12	Nonwhite females	
	Indicators of intoxication	
13	First admissions rates to state hospitals, United States, for psychosis with alcoholism, 1926–1965	No measurable trend
14	First admission rates to state hospitals, United States, with all alcohol-related mental disorders, 1926–1967	$Y = 1946.659 + 234.253 X$
15	Arrest rate, driving while intoxicated, United States, 1934–1967	$\log Y = 6.7688 + 0.0821 X$
16	Persons brought to trial, drunkenness, Massachusetts, 1911–1967	$\log Y = 10.766 - 0.275 \log X$
17	Persons brought to trial, driving while intoxicated, New York City, 1936–1970	$\Delta Y = Y_{t-1} + 0.1351\, Y_{t-1} - 0.000053 Y_{t-12}$
18	Persons brought to trial and found guilty, driving while intoxicated, Philadelphia, 1931–1967	No measurable trend
19	Persons convicted, driving while intoxicated, California, 1950–1967	No measurable trend
	Economic indicators	
20	Per capita personal income, United States, 1929–1970	$\log Y = 5.9977 + 0.05457 X$
21	Unemployment rate, United States (inverted), 1902–1968	No measurable trend

Findings

There are both long and short term positive relations between per capita alcohol consumption and cirrhosis of the liver mortality (Figure 1 and Table 2). A short term positive relationship between alcohol consumption and cirrhosis mortality occurs for whites and nonwhites and for both sexes. For males of both racial groups and for white females increases in cirrhosis mortality occur approximately 1 year after a national increase in alcohol consumption, while for nonwhite females the lag is closer to 2 years.

Cirrhosis mortality also shows a strong long term positive relationship with alcohol consumption for both racial groupings and each sex. In view of the short term relation of alcohol consumption to cirrhosis mortality within 2 years, and evidence that the condition of liver cirrhosis probably requires 10 to 15 years of previous heavy drinking,[6] it is probably not true that the long term trend relation between per capita alcohol consumption and cirrhosis mortality is directly causal within less than 3 years.

Put another way, although the long term trend in per capita alcohol consumption is related to those for cirrhosis mortality rates, it cannot be argued that a greater number of persons are *both* being exposed to alcohol consumption (or drink more heavily) and experience cirrhosis mortality within less than 2 years. Therefore, it appears that (1) only the short term fluctuations in alcohol consumption apply to proximal cirrhosis mortality, and (2) the per capita trend of increase in alcohol consumption involves new drinkers and/or increases in consumption by persons who previously drank moderately. The trend in alcohol consumption would presumably first result in a minority of consistently heavy drinkers, some of whom would develop cirrhosis within 10 to 15 years.

Alcohol Consumption and National Economic Trends

Having observed trends in fluctuations in cirrhosis mortality and concomitant trends and fluctuations in per capita alcohol consumption, the question is raised as to whether social changes, especially changes in the national economy, may influence alcohol consumption, the previous

independent variable. A major hypothesis stemming from economic reasoning (Mitchell) is that the apparent trend in per capita alcohol consumption may be real and may reflect greater long term availability of discretionary income which might lead to such "luxury" expenditures as those for distilled spirits, wine, and beer. It might similarly be argued that short term fluctuation in consumption of such beverages might be positively coincident with fluctuations in personal income.[14]

It is also possible, on the other hand, that short term increases in alcohol consumption might be related to economic recession if alcohol consumption at least partly reflected social-psychological stresses related to economic instability. The time-series analytic procedures discussed above are again (and hereafter) used to discriminate the long and short term effects of the independent variable.

It is evident from Table 3 and Figure 2 that alcohol consumption is highly correlated with long and short term changes in the state of the economy. The long term trend in per capita personal income is positively related to that in per capita alcohol consumption; however, the shorter fluctuations in per capita alcohol consumption are inversely related to those in per capita income and the inverted unemployment index. This tendency for per capita consumption of alcohol to increase during economic recession occurs within months of any given economic downturn (i.e., at zero lag, annually).

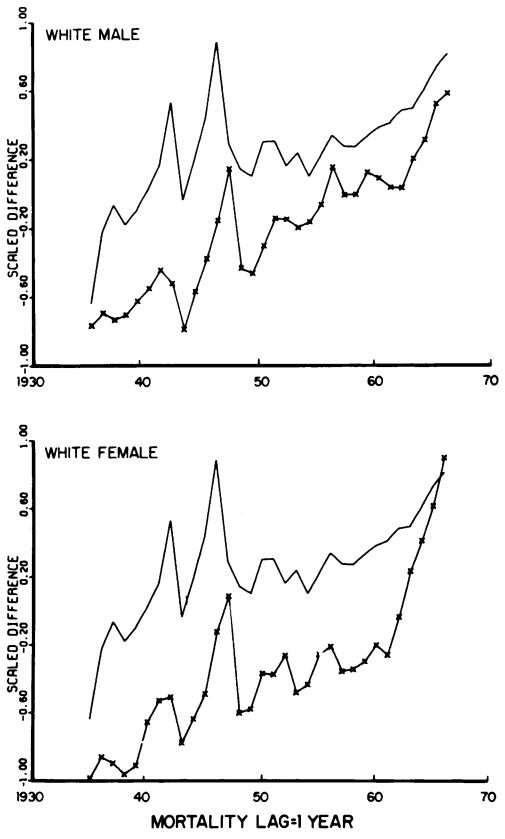

FIGURE 1 Trends in average alcohol consumption and in cirrhosis mortality rates, United States, 1934–1968. ×——×, cirrhosis mortality rates for whites by sex; ——, average alcohol consumption, United States. Fluctuations in average alcohol consumption and in cirrhosis mortality rates are scaled for viewing such that the greatest amplitude from the arithmetic mean of each series (which is set equal to zero) has been normalized to 1.0 if positive, or −1.0 if negative. Scalar range describes relative magnitude of variation in each series.

The relation of national economic changes to consumption of different alcoholic beverages is variable, however. While the secular trends in per capita consumption of distilled spirits, wine, and beer are related to the long term trend in per capita personal income, wine and beer consumption increases during recessions (Table 3). It is therefore largely per capita consumption of distilled spirits that accounts for the *inverse* relation between fluctuations in the economy and those in per capita average monthly (total) alcohol consumption.

Since cirrhosis of liver mortality rates are related to trends and fluctuations in per capita alcohol consumption, and the latter are related to national economic changes, one would predict that cirrhosis mortality rates are in turn related to economic changes. This prediction is borne out in Table 4 and Figures 3 and 4. While long term trends in cirrhosis mortality rates increase along with those in national per capita personal income, cirrhosis mortality rates also increase following recessions. National recessions in personal income and employment are consistently followed, within 2 to 3 years, by increases in cirrhosis mortality rates.

Additional tests were made to determine the combined relationship between several national economic indicators and cirrhosis mortality rates, during 1933–1973, using multiple linear regression analysis with raw (i.e., nondetrended) data. Given the previous results, it was expected that when per capita personal income and the inverted unemployment index were acting together (in the same equation), they would show essentially the same relationship to cirrhosis mortality rates that they showed separately. This was verified in Table 5, where fluctuations in employment are inversely, and per capita personal income is positively, related to cirrhosis mortality rates (Equation 1a). These data also indicate that nearly the entire variation in the cirrhosis mortality trends from 1933 to 1973 is associated with the combined effects of these two variables (e.g., R^2 = .971 in Equation 1a).

Moreover, the relations between these two variables and trends in cirrhosis mortality rates are essentially unchanged, either in direction of association or variance explained, when one adds additional lags of cirrhosis mortality behind the economic variables (Equations 2a and 3a, which include, respectively, lags one and two, and one through three). These relations are also unchanged when a measure of inflation, annual percentage changes in the consumer price index, is included in the equation (Equations 1b, 2b, and 3b). Finally, even when gross national product is substituted for per capita personal income as the measure of national wealth and the measure of inflation is included, again the basic relations are unchanged either in terms of direction or proportion of variance explained (Equations 1c, 2c, and 3c).

The relation between economic recession and alcohol

TABLE 2—Relationships between Per Capita Alcohol Consumption, United States, and Cirrhosis of Liver Mortality Rates, by Sex and Race, United States, 1935–1968 and 1948–1968

Independent Variable	Dependent Variable		Statistical Significance: Lag of Dependent Variable behind Independent Variable (Years)		
			0	1	2
				$p <$	
Avg monthly alcohol consumption rate	Cirrhosis mortality rate				
	White males	1935–1968	.001	.001	.001
		1948–1968	.001	.001*	.001
	White females	1935–1968	.001	.001*	.001
		1948–1968	.001	.001*	.001
	Nonwhite males	1935–1968	.001*	.001	.001
		1948–1968	.001*	.001	.001
	Nonwhite females	1935–1968	.001	.001*	.001
		1948–1968	.001*	.001	.001
Avg monthly alcohol consumption rate detrended	Cirrhosis mortality rate				
	White males	1935–1968	N.S.	.001*	.001
		1948–1968	N.S.	.001	.001*
	White females	1935–1968	.001	.001*	.001
		1948–1968	.001	.001	.001*
	Nonwhite males	1935–1968	.05*	N.S.	N.S.
		1948–1968	.001*	.001	.05
	Nonwhite females	1935–1968	N.S.	N.S.	N.S.
		1948–1968	.001*	N.S.	.05

* Lag at which correlation reaches maximum.

abuse may also be examined by observation of the behavior of other alcohol problem-related indicators. Such indicators include admissions to mental hospitals with alcohol-related mental disorder, and contact with the criminal justice system as a result of intoxication. Specifically, the indicators are: (1) first admission rates to state hospitals, United States, for psychosis with alcoholism; (2) first admission rates to state hospitals, United States, with all alcohol-related mental disorders; (3) arrest rate, driving while intoxicated, United States; (4) persons brought to trial, drunkenness, Massachusetts; (5) persons brought to trial, driving while intoxicated, New York City; (6) persons brought to trial and found guilty, driving while intoxicated, Philadelphia; and (7) persons convicted, driving while intoxicated, California.

In this last analysis we confine our data strictly to the short and intermediate sized trends by algebraically subtracting data points describing the long term trends (Table 1). The resulting correlations (Table 6) and graphic representations (Figures 5 and 6) show substantial inverse relations between the above indicators of alcohol abuse-related problems and the state of the economy. In this analysis, the United States unemployment index (inverted) was utilized to represent the situation of the national economy since it tends to be a slightly more sensitive indicator than per capita personal income of major loss of income during recessions. (Similar correlations are found where per capita income is the economic indicator, as is seen in the analyses of alcohol consumption and cirrhosis mortality.)

First admissions to state mental hospitals in the United States as a whole, either with alcoholic psychosis or for all alcohol-related mental disorders, show a lag in increase of approximately 2 years behind declines in the national economic situation (Figure 6). Arrest rates for the United States as a whole, persons brought to trial in New York City, those tried and found guilty in Philadelphia, and those tried and convicted in California—all for the crime of driving while intoxicated—similarly show increases within 1 to 2 years following economic recessions. Also, the same inverse relationship to economic changes is shown for persons brought to trial for the crime of "drunkenness" in Massa-

TABLE 3—Relationships between National Economic Indicators, United States: (Raw and Detrended) and Per Capita Consumption of Alcoholic Beverages, 1948–1970

Independent Variable	Dependent Variable	Statistical Significance: Lag of Dependent Variable behind Independent Variable (Years) 0	1	2
			$p <$	
Per capita personal income, United States	Monthly avg alcohol consumption rate	.001*	.001	.001
	Total alcohol	.001*	.001	.001
	Distilled spirits	.001*	.001	.001
	Distilled spirits, absolute alcohol	.001*	.001	.001
	Wine	.001	.001*	.001
	Wine, absolute alcohol	.001	.001*	.001*
	Beer	.001*	.001	.001
	Beer, absolute alcohol	.001*	.001	.001
Per capita personal income, United States, detrended	Monthly avg alcohol consumption rate	(−).001	(−).001	(−).001*
	Total alcohol	N.S.	(−).01	(−).001*
	Distilled spirits	N.S.	(−).05	(−).01*
	Distilled spirits, absolute alcohol	.01	(−).001	(−).001*
	Wine	.01	(−).001*	.001
	Wine, absolute alcohol	N.S.	N.S.	N.S.
	Beer	.001*	.001	.01
	Beer, absolute alcohol	.001*	.001	.001*
Unemployment index (inverted)	Monthly avg alcohol consumption rate	N.S.	(−).01	(−).001*
	Total alcohol	N.S.	N.S.	N.S.
	Distilled spirits	N.S.	N.S.	N.S.
	Distilled spirits, absolute alcohol			
	Wine	(−).05	N.S.	N.S.
	Wine, absolute alcohol	N.S.	N.S.	N.S.
	Beer	.001*	.001	.05
	Beer, absolute alcohol	.001*	.001	.05

* Lag at which correlation reaches maximum.

chusetts, with a 2-year lag behind the economic indicator (Figure 6 and Table 6).

Discussion

Data of the present study support the conclusions of Terris[7] and, previously, of Jolliffe and Jellinek[8] that alcohol consumption is directly related to cirrhosis of liver mortality on a short term basis. The lag between increases in per capita alcohol consumption and that in cirrhosis mortality rates lies largely between 1 and 2 years, and is clearly not within (under) 1 year as may have been assumed in earlier graphic representation. However, precisely because the relation between average alcohol consumption and cirrhosis mortality involves short term fluctuations, the long term relation between the two, while positive, is probably not direct.

The long term trend in alcohol consumption might reflect new or increased drinking in the population aggregate, but it could not simultaneously be instrumental in increasing the cirrhosis *morbidity* rate and causing *mortality* within 2 years. The reason is simply that it requires too long an interval to develop the cirrhotic condition (10 to 15 years).[6] Thus, it is probably the long term exposure to alcohol and changes in drinking patterns, as related to economic growth, on the part of the population aggregate or (at least) a large minority that is the major *secular trend* feature in cirrhosis morbidity.

The present study found, moreover, that long term trends and shorter fluctuations in the state of the economy were related to alcohol consumption, but in divergent ways. The long term trend in per capita personal income is positively related to per capita alcohol consumption, while shorter fluctuations in the two variables show a strong

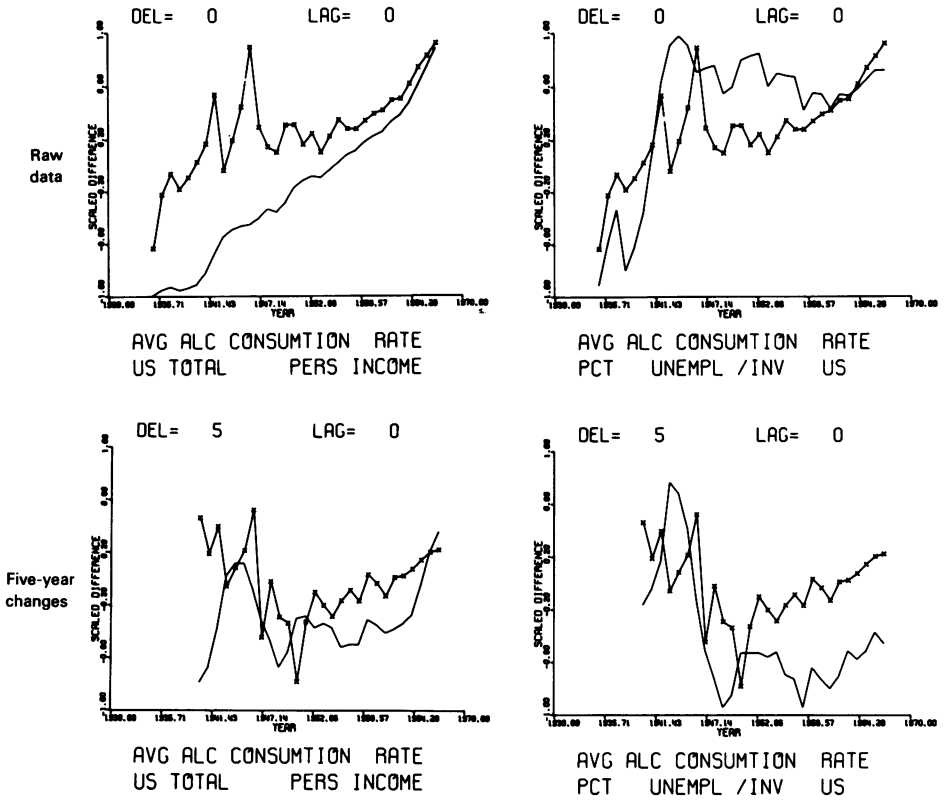

FIGURE 2 Trends in national economic indicators and in average alcohol consumption, United States, 1934–1970. ×——×, per capita personal income, unemployment index (inverted); ——, average alcohol consumption. Fluctuations in economic indicators and in average alcohol consumption rates are scaled for viewing such that the greatest amplitude from the arithmetic mean of each series (which is set equal to zero) has been normalized to 1.0 if positive, or −1.0 if negative. Scalar range describes relative magnitude of variation in each series.

TABLE 4—Relationships between National Economic Indicators, United States (Raw and Detrended) and Cirrhosis of Liver Mortality Rates, by Sex and Race, 1948–1968

Independent Variable	Dependent Variable	Statistical Significance: Lag of Dependent Variable behind Independent Variable (Years)		
		0	1	2
			$p <$	
Per capita personal income, United States	Cirrhosis mortality rate			
	White males	.001*	.001	.001
	White females	.001*	.001	.001
	Nonwhite males	.001*	.001	.001
	Nonwhite females	.001*	.001	.001
Per capita personal income, United States, detrended	Cirrhosis mortality rate			
	White males	N.S.	N.S.	(−).05*
	White females	N.S.	N.S.	(−).05*
	Nonwhite males	(−).001	(−).001	(−).001*
	Nonwhite females	(−).001	(−).001	(−).001*
Unemployment index (inverted)	Cirrhosis mortality rate			
	White males	N.S.	N.S.	(−).001*
	White females	N.S.	N.S.	(−).010*
	Nonwhite males	N.S.	(−).05	(−).001*
	Nonwhite females	N.S.	(−).010	(−).001*

* Lag at which correlation reaches maximum.

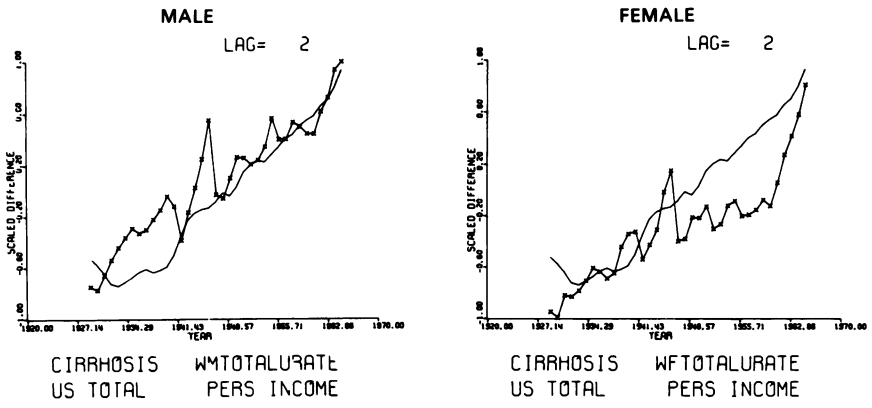

FIGURE 3 Trends in per capita personal income and in cirrhosis of liver mortality, United States, 1929–1968. ×——×, cirrhosis mortality rates for whites by sex, United States; ——, per capita personal income, United States. Fluctuations in economic indicator and in mortality rates are scaled for viewing such that the greatest amplitude from the arithmetic mean of each series (which is set equal to zero) has been normalized to 1.0 if positive, or −1.0 if negative. Scalar range describes relative magnitude of variation in each series.

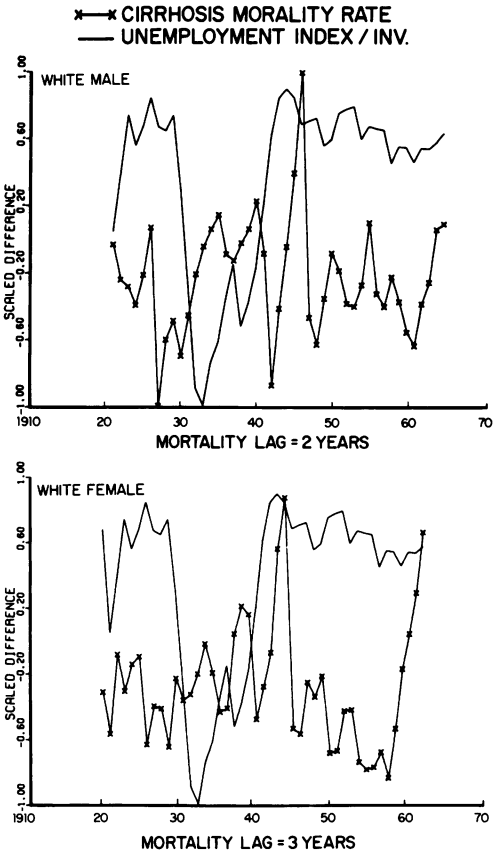

FIGURE 4 Fluctuations in employment rates and in cirrhosis of liver mortality, detrended data, United States, 1921–1968. Fluctuations in economic indicator and in mortality rates are scaled for viewing such that the greatest amplitude from the arithmetic mean of each series (which is set equal to zero) has been normalized to 1.0 if positive, or −1.0 if negative. Scalar range describes relative magnitude of variation in each series.

inverse relationship. This long term positive and short term inverse relationship is probably accounted for by consumption of distilled spirits which shows a similar relation to the state of the economy. Per capita beer and wine consumption increases with both long and short term increases in per capita income. These data support the proposition that per capita consumption of distilled spirits responds to long term economic growth and stability as well as to recessions. Thus, while wine and beer consumption, in general, reflect prosperous and stable periods, that of distilled spirits reflects long term prosperity *and* short term economic stress.

These apparently opposite movements of per capita consumption of distilled spirits may reflect the divergent activities of two populations. One of these populations experiences, and uses alcohol under conditions of, general economic prosperity and stability (as does the population aggregate over the long term). The second population, which is a minority of the aggregate, drinks distilled spirits (at least) under conditions of major economic stress. A portion of the second population become intoxicated, which often results in contact with the criminal justice system or the mental hospital. Another portion of the second population (and possibly a different portion) are long term abusers of alcohol with liver damage who show increased mortality rates within 2 years of the increased alcohol consumption.

The divergent tendencies in the general population—one toward *increased per capita* aggregate consumption under stable conditions and the other toward *heavy* alcohol consumption under unstable conditions—helps to explain inconsistencies between the earlier assumption of a direct relation between per capita consumption and cirrhosis mortality and findings of population surveys of drinking habits. In those studies based on socioeconomic status, ethnic background, and age, the presence or absence of drinking was either unrelated or inversely related to that of heavy drinking. The implication is that a larger proportion of drinkers in a given socioeconomic, ethnic, or age group did not also signify a larger proportion of *heavy* drinkers.

Thus, a national survey[3] of drinking practices in the United States (1964–1965) found that the proportion of heavy drinkers was highest at the lower socioeconomic levels. Relatively more higher income and middle class individuals reported drinking at least occasionally but few of those who did drink were heavy drinkers. The same study

TABLE 5—Least Squares Linear Multiple Regression of National Economic Indicators on Cirrhosis of Liver Mortality Rates, United States, 1933–1973

							R^2	D.W.
(1)	CDR =	C	$+\beta_1 EMPL_{t-2}$	$+\beta_2 PCI_{t-2}$	$+\beta_3 PCPI_{t-2}$	$+\beta_4 GNP_{t-2}$		
2-year lag only	(1a)	139.52	−142.68 (7.41)*	+80.27 (28.94)*			.971	1.56
	(1b)	145.18	−140.56 (6.65)*	+80.45 (28.66)*	+16.27 (0.66)		.971	1.60
	(1c)	157.20	−100.96 (4.55)*		+59.06 (2.29)*	+19.79 (27.57)*	.969	1.82
(2)	CDR =	C	$+\sum_{i=1}^{2}\beta_5 EMPL_{t-i}$	$+\sum_{i=1}^{2}\beta_6 PCI_{t-i}$	$+\sum_{i=1}^{2}\beta_7 PCPI_{t-i}$	$+\sum_{i=1}^{2}\beta_8 GNP_{t-i}$		
1- & 2-year lags	(2a)	135.05	−138.47 *(4.99)**	+80.67 *(216.44)**			.973	1.63
	(2b)	137.82	−138.28 *(4.96)**	+77.15 *(168.27)**	+64.77 *(1.50)*		.979	1.88
	(2c)	156.80	−101.37 *(4.69)**		+129.36 *(3.25)*	+19.15 *(196.45)**	.982	2.27
(3)	CDR =	C	$+\sum_{i=1}^{3}\beta_9 EMPL_{t-i}$	$+\sum_{i=1}^{3}\beta_{10} PCI_{t-i}$	$+\sum_{i=1}^{3}\beta_{11} PCPI_{t-i}$	$+\sum_{i=1}^{3}\beta_{12} GNP_{t-i}$		
1-, 2-, & 3-year lags	(3a)	136.20	−142.68 *(5.49)**	+83.10 *(142.31)**			.976	1.73
	(3b)	129.43	−129.52 *(2.71)**	+77.96 *(105.99)**	+48.98 *(0.90)*		.981	1.94
	(3c)	160.22	−105.79 *(2.61)**		+139.35 *(1.24)*	+19.66 *(132.33)**	985	2.29

Where CDR = cirrhosis of liver mortality rates; C = constant; EMPL = unemployment index/inverted; PCI = per capita income; PCPI = consumer price index; GNP = gross national product; R^2 = multiple correlation squared; D.W. = Durbin-Watson statistics; *T* - statistics are in parentheses; *F* - statistics are in parentheses and italicized; and variables statistically significant at the .95 level of confidence are asterisked.

TABLE 6—Relationships between National Economic Indicators, United States, and Indicators of Alcohol Abuse

Independent Variable	Dependent Variable	Statistical Significance: Lag of Dependent Variable behind Independent Variable (Years)		
		0	1	2
			$p <$	
Unemployment index (inverted) United States, detrended	First admission rates to state hospitals, United States, for psychosis with alcoholism, 1948–1965	N.S.	(−) .001	(−) .001*
Unemployment index (inverted), United States, detrended	First admission rates to state hospitals, United States, with all alcohol-related mental disorders, 1941–1967	(−) .001*	(−) .001	N.S.
Per capita personal income, United States, detrended	Arrest rate, driving while intoxicated, United States, 1934–1967	(−) .001*	(−) .001	(−) .001
	1948–1970	(−) .01	(−) .01*	(−) .05
Per capita personal income, United States, detrended	Arrest rate, driving while intoxicated, United States, 1934–1970	(−) .001*	(−) .001*	(−) .001
	1948–1970	(−) .001	(−) .001	(−) .001
Per capita personal income, Massachusetts	Persons brought to trial, drunkenness, Massachusetts, 1929–1970	(−) .01*	(−) .001	(−) .01
	1948–1970	(−) .01*	(−) .01	N.S.
Per capita personal income, New York State	Persons brought to trial, driving while intoxicated, New York City, 1936–1970	(−) .001*	(−) .001	(−) .001
	1948–1970	(−) .001*	(−) .001	(−) .001
Per capita personal income, Pennsylvania	Persons brought to trial and found guilty, driving while intoxicated, Philadelphia, 1931–1967	(−) .001*	(−) .001	(−) .05
	1948–1967	(−) .001*	(−) .001	(−) .001
Per capita personal income, California	Persons convicted, driving while intoxicated, California, 1950	(−) .01*	N.S.	N.S.

* Lag at which correlation reaches maximum.

showed that drinking per se showed declines with age, after age 35, but heavy drinking did not show similar declines.

Again, Lolli et al.[15] found that first generation Italians in the United States, just as natives in Italy, drink frequently but have low rates of alcohol-related problems. Subsequent generations of Italian Americans have higher rates of heavier drinking.[16] Also, there have been many reports of high rates of alcoholism among Irish Americans,[17, 18] although Ireland rated 16th in apparent consumption of alcohol among 20 countries. Thus, there is no necessary relation between *widespread* drinking and a high incidence of problems related to *heavy* drinking.

Similarly, an increase in the trend of per capita alcohol consumption leads only indirectly (and not necessarily) to increases in cirrhosis mortality or other alcohol-related problems. Such an indirect relationship indicates the necessity of at least one additional intervening variable between increased general alcohol consumption and increases in the incidence of alcohol-related problems. The data of this study show that conditions of social instability and stress, particularly as related to economic conditions, probably represent one such intervening variable. The incidence of alcohol-related problems therefore becomes a multivariate function of both the long term trend in per capita alcohol consumption and social instability.

Relation to Independent Empirical Findings and Theoretical Formulations

Data of the present study are in conformity with the findings of independent studies on the importance of sociocultural factors which predispose toward heavy drinking. There is no necessary relation between widespread drinking and high incidence of alcohol-related problems.[19] However, there is evidence that among groups whose drinking habits and values are well established, alcoholism rates are low.

During periods of cultural change, especially those during which the long term increase in demand for alcoholic beverages is related to that in per capita personal income, one can anticipate social ambivalence until new norms are relatively well established. Periods of substantial long term growth in per capita personal income, as in the United States during 1941–1970, are indicative of growth in "discretionary" spending for other, perhaps "luxury," commodities and services that are not traditional in the culture.

A second issue related to secular increases in discretionary consumption, and consumption of alcohol in particular, is the matter of simple exposure of the population to alcohol as a means of reducing tension and anxiety. The smaller the proportion of persons who are abstainers in a population,

the more likely is the population aggregate to be at least acquainted with its effects on the nervous system. Thus, while widespread exposure to alcohol will not necessarily lead to heavy drinking in a given population, some exposure to the effects of alcohol is necessary for any member of the population to begin a period of heavy drinking (regardless of the precipitating causes).

The findings of this study are also consistent with the great majority of theories of alcoholism which emphasize the role of stress as a precondition of alcoholism.[20] The available evidence is that economic downturns are a very major source of economic and social stress and that they show substantial correlations with increases in suicide,[21, 22] mental hospital admissions,[23] heart disease mortality,[24] and infant mortality[25] at least for the same period as is covered by this study.

The most comprehensive explanation of alcoholism on a societal level involves the conception of such deviant behavior as being anomic. Merton offers the theory that anomie occurs as a result of discrepancy between culturally shared goals and the means for achieving them.[26] Merton argues that adaptation to the problems of this discrepancy are of four types: conformity, ritualism, retreatism, and rebellion. It has been suggested that alcoholism may represent one of the latter two.

Indeed, there are few theories which so well represent the conflict experienced by large aggregates of people during economic recessions. An operationalization of Merton's formulation is almost the precise situation of economic recession, considered as a discrepancy from the ordinarily expected sequence of continued long term advancement in economic well-being. The economic downturn, moreover, places individuals subject to it in a position from which (given loss of job or income) it is virtually impossible to conform to normal expectations, whether cultural, peer group, family, or individual.

Merton's theory is fundamentally an individual-centered stress theory although it involves in the aggregate all persons subject to the discrepancy between societal goals and the means for their achievement. Nor is it necessary to postulate an industrialized society, with its own mode of major economic fluctuations, to offer support to a stress-based theory of alcoholism. Horton[27] suggests that the nearly universal occurrence of alcohol in human societies —especially primitive societies—indicates its acceptable and utilitarian function as that of reducing anxiety.

Empirical support for a stress-based theory, particularly linked to national economic fluctuations, is given by McClelland and associates.[28] It is suggested that frustrated ambitions are significant factors in the development of alcoholism. The excessive use of alcohol further compounds the problems perceived as lack of achievement by rendering the individual even less capable of achievement. The subsequent failures, in turn, reinforce a continued pattern of heavy use of alcohol.

Finally, from the standpoint of learning theory, it has

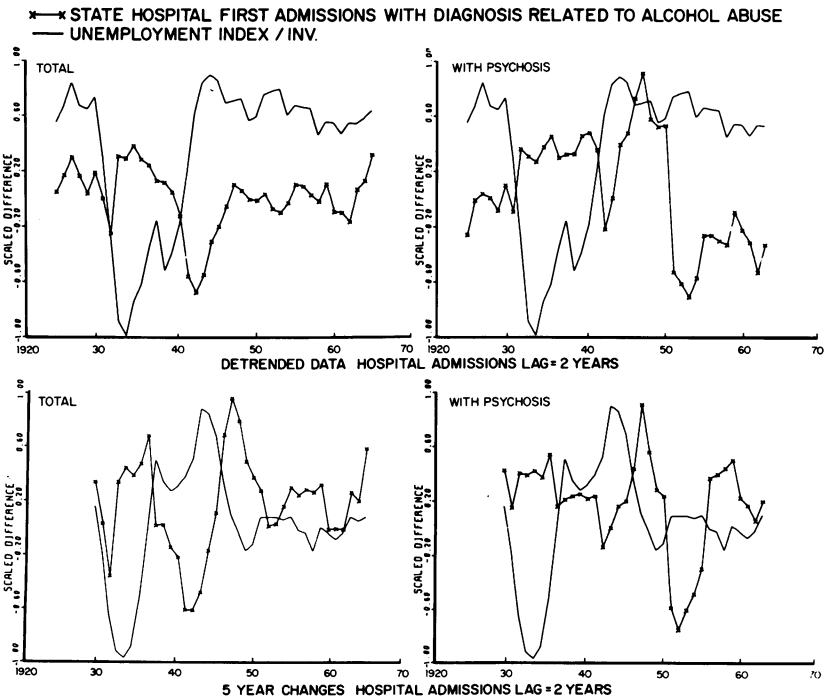

FIGURE 5 Fluctuations in employment rates, United States, and in first admission rates to state hospitals with alcohol-related mental disorders, United States, 1921–1968. Fluctuations in economic indicator and in mental hospital admission rates are scaled for viewing such that the greatest amplitude from the arithmetic mean of each series (which is set equal to zero) has been normalized to 1.0 if positive, or −1.0 if negative. Scalar range describes relative magnitude of variation in each series.

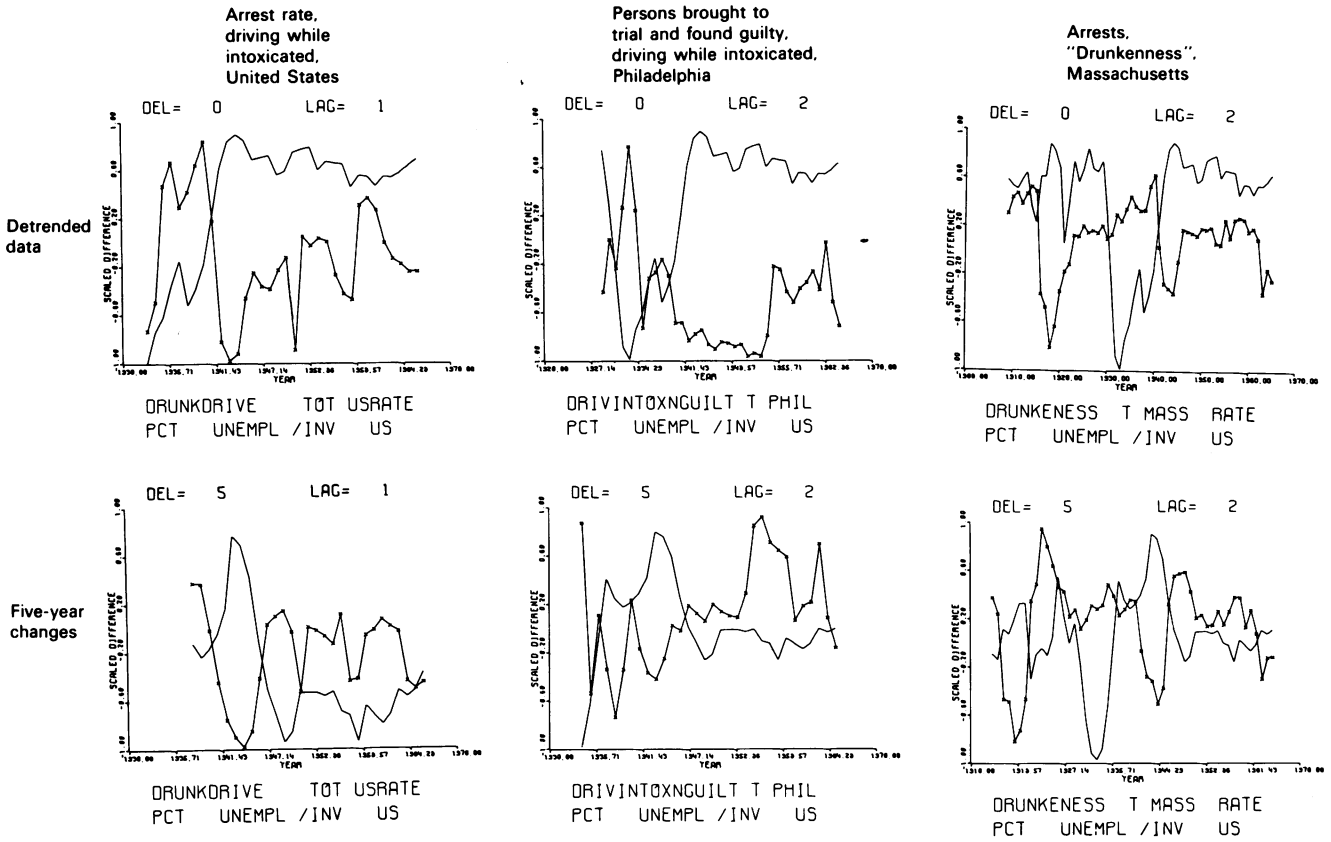

FIGURE 6 Fluctuations in national economic indicators in relation to those in criminal alcoholic intoxication, Philadelphia, Massachusetts, and (entire) United States. ×——×, indicators of criminal alcoholic intoxication; —— unemployment index (inverted), United States. Fluctuations in economic indicator and in indicators of criminal intoxication rates are scaled for viewing such that the greatest amplitude from the arithmetic mean of each series (which is set equal to zero) has been normalized to 1.0 if positive, or −1.0 if negative. Scalar range describes relative magnitude of variation in each series.

been difficult to understand why, if alcoholism is a response to anxiety, the alcoholic continues to drink in view of the punishment received by way of family discord, illness, and other sequelae.[6] The data of the present study suggest that alleviation of the effects and aftermath of economic stress itself may be a stronger stimulus to drinking than the punishing effects subsequent to heavy drinking are inhibiting.

Integration of the sociocultural and stress approaches to alcoholism appears to sustain the greatest validity in view of the independent empirical support for each of the two approaches. In a review of the literature on etiology of alcoholism, Straus states that most theories emphasize the role of stress as a "precondition of alcoholism" and that the evidence indicates that "alcoholism becomes a response to stress primarily in cultures where drinking customs create exposure to frequent intoxication, where intoxication is a means of fulfilling individual rather than group functions, and where there are not culture-approved alternative modes of dealing with stress."[20]

One of the most frequently cited studies which emphasizes the integrated sociocultural-stress approach is that of Bales.[17, 29] Based on observations of differences in drinking practices and rates of alcoholism among Irish and Orthodox Jews, Bales proposed a three-factor explanation of societal influences on the rate of alcoholism: (1) culturally precipitated anxiety, (2) cultural support for drinking as a means of relieving anxiety, and (3) lack of culturally acceptable alternatives to drinking as a relief of anxiety. Thus, in contrast to Orthodox Jews, the Irish *both* experienced the stress of low socioeconomic status upon emigration to the United States, and had cultural support for intoxication as a means of temporarily moderating such stress. Subsequent studies by Snyder,[30] Lolli et al.,[15] and Jessor et al.,[16] respectively in Jewish, Italian, and Anglo-American, Spanish American, and American Indian ethnic groups tended to support Bales' integrated etiological formulation.

The findings of the present study are also consistent with the evidence that the behavior of the alcoholic in his use of alcohol is considerably different from that of the average user of alcohol in the population.[19] In particular, although drinking occurs more frequently among persons of relatively high socioeconomic status, alcoholism or serious problems related to alcohol use are found to be inversely related to socioeconomic status.[4, 31-34] Thus, on the one hand the long term trend in use of alcohol as related to increases in per capita personal income probably reflects the secular improvement of the socioeconomic status of a large proportion of the population. On the other hand, it is only following the economic downturn that the indicators of alcohol-related problems show substantial increases.

It appears, then, that among a population whose

increased discretionary income has exposed individuals to alcohol for leisure time or private use (as opposed to culturally traditional use such as in family meals or ceremonial occasions) the rate of stress-related alcohol use will also increase.[35, 36] In the United States population, especially since 1948, the stress-related use appears to be correlated with the incidence of economic recessions.

The stress-related use of alcohol during and following economic downturns involves especially the distilled spirits rather than wine or beer. In fact, the consumption of wine and beer has increased both over the secular trend coincident with the increase in per capita income and during economic upturns. This would indicate that, for the United States population in general, consumption of wine and beer is not stress-related to nearly the degree as is consumption of distilled spirits. This inference is consistent with the evidence that consumption of wine and beer poses little risk of long or short term adverse effects.[35]

Conclusions

Data of the present study support the conclusions of Terris and, previously, of Jolliffe and Jellinek that alcohol consumption is directly related to cirrhosis of liver mortality on a short term basis. The lag between increases in per capita alcohol consumption and those in cirrhosis mortality rates lies between 1 and 2 years. However, precisely because the relation between average alcohol consumption and cirrhosis mortality involves short term fluctuations, the long term relation between the two, while positive, is probably not direct. The long term trend in alcohol consumption might reflect new or increased drinking in the population aggregate, but it could not simultaneously be instrumental in increasing the cirrhosis morbidity rate and causing mortality within 2 years.

It was found, in addition, that long term trends and shorter fluctuations in the state of the economy were related to alcohol consumption, but in divergent ways. The long term trend in per capita personal income is positively related to per capita alcohol consumption, while shorter fluctuations in the two variables show a strong inverse relationship. This long term positive and short term inverse relationship is probably accounted for by consumption of distilled spirits which shows a similar relation to the state of the economy. Per capita beer and wine consumption increases with both long and short term increases in per capita income. These data support the proposition that per capita consumption of distilled spirits responds to long term economic growth and stability as well as to recessions. Thus, while wine and beer consumption in general reflects prosperous and stable periods, that of distilled spirits reflects long term prosperity *and* short term economic stress.

The opposite movements of per capita consumption of distilled spirits may reflect the divergent activities of two populations. One of these populations experiences, and uses alcohol under conditions of, general economic prosperity and stability (as does the population aggregate over the long term). The second population, which is a minority of the aggregate, drinks distilled spirits (at least) under conditions of major economic stress. A portion of the second population becomes intoxicated, which often results in contact with the criminal justice system or mental hospital. Another portion of the second population (and possibly a different fraction) represents long term abusers of alcohol with liver damage who show increased mortality rates within 2 years of the increased alcohol consumption.

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HOW I STOPPED SMOKING . . .

(Excerpts from letters received by the APHA Smoking and Health Project from former smokers.)

HOW COME YOU SMOKE CIGARETTES?

Cigarette smoking had been my recurring, fulsome habit for many years. Devastatingly, Mark Twain's quote applied: "Oh, anybody can stop smoking. I've stopped ten or a dozen times."

Following an MPH year at the School of Hygiene and Public Health, Johns Hopkins, I obtained an NIMH fellowship "to train a Public Health person in Community Psychiatry," so it was back to Boston, that bastion of Freudian training and indoctrination.

What a telltale surprise I had upon learning the depth interpretation with respect to repeated sucking and overuse of little white tubes, even those disguised with soft, succulent cork tips.

Well, it took courage to quit. An unprintable Marine Corps phrase relating to being up to a difficult task comes to mind. Apparently, I had what it took.

Of course there was an assist as follows: an excellent chest physician diagnosed and treated my persistent, low grade fever and cough. He asked whether I smoked cigarettes.

"No way, Doctor; they taste terrible. Besides I generally quit for a month or more whenever I have an upper respiratory infection."

"Why don't you stay quit?"

I did. That was in 1953. Much could be added about the resulting advantages; perhaps it is pertinent to ask, "How come *you* smoke cigarettes?"

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